GENERAL

PATHOLOGICAL PHYSIOLOGY

Minsk BSMU 2021

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ

ОБЩАЯ ПАТОФИЗИОЛОГИЯ

GENERAL PATHOLOGICAL PHYSIOLOGY

Рабочая тетрадь

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Р е ц е н з е н т ы: каф. патологической физиологии Витебского государственного ордена Дружбы народов медицинского университета; канд. мед. наук, доц., доц. каф. биологии с курсами нормальной и патологической физиологии Гомельского государственного медицинского университета Т. С. Угольник

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Предназначено для студентов 3-го курса лечебного факультета, обучающихся на английском языке, для самостоятельной подготовки к занятиям, выполнения и оформления лабораторных работ.

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LIST OF ABBREVIATIONS

AH — arterial hyperemia

BP — blod pressure

ADP — adenosine diphosphate

ATP — adenosine triphosphate

BAS — biologically active substances

VH — venous hyperemia

G-CSF — granulocyte colony stimulating factor

IL — interleukins

ABB — acid-base balance

ARS — acute radiation sikness

PUFAs — polyunsaturated fatty acids

TA — titratable acidity

HL — heat loss

HP — heat production

TPP — typical pathological processes

TNF — tumor necrosis factor

MPFFS — the mother-placenta-fetus functional system

CNS — central nervous system

RR — respiratory rate

HR — heart rate

EAMC — electronic academic methodical complex

HbO_2 — oxyhemoglobin

P_ACO_2 —partial pressure of carbon dioxide in alveolar air

P_aCO_2 — partial pressure of carbon dioxide in arterial blood

P_AO_2 — partial pressure of oxygen in alveolar air

P_aO_2 — partial pressure of oxygen in arterial blood

P_vCO_2 — partial pressure of carbon dioxide in venous blood

P_vO_2 — partial pressure of oxygen in venous blood

EAMC — electronic academic methodical complex

SECTION I
GENERAL NOSOLOGY

LESSON 1. INTRODUCTION TO THE DISCIPLINE "PATHOLOGICAL PHYSIOLOGY". SUBJECT, OBJECTIVES, METHODS OF PATHOLOGICAL PHYSIOLOGY

Date: «____» _____ 20__

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

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 pathological processes modeling, 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.

SAFETY INSTRUCTIONS

General requirements

1. Only the students that studied the instructions on labor protection and fire safety at the first lesson are allowed to attend classes at the Pathophysiology department. Every student must put the signature in a special journal to confirm that he/she understands the studied rules and will follow them.
2. The students must maintain discipline in the classroom as well as sanitary and personal hygiene.
3. It is necessary to prepare your desk in the classroom before the lesson starts. There mustn't be any materials, objects and substances that are not used in the studying process. A student must wear a laboratory coat BEFORE the beginning of the class.
4. Every group must attend classes according to the time and the number of the classroom indicated in the schedule. It's forbidden to change the working place without the permission of the teacher. It's not allowed to perform any work that is not related to the task given by the teacher and to do anything at unspecified time without the permission of the teacher.
5. While staying at the Pathophysiology department the students must be careful, especially while moving along window openings, stands, cabinets with glass construction, central heating batteries, especially after the wet cleaning of the room. Do not step on liquid or any objects that can be found on the floor.

Do not carry objects, holding them in front of you, if it makes impossible to observe your way. Do not move your back forward.

It is not allowed to sit down or stand on the windowsills of the closed or open windows; to outweigh through the windowsills of the open windows; to sit on the central heating pipes and batteries; to sit on the desks, equipment, to swing on the chairs.

6. The students must follow safety requirement while working with the electrical equipment during the class. It's necessary to get acquainted with its work principle and dangers before using. If the electrical equipment doesn't work properly, it must be disconnected. The students must inform the Head of the Department about such cases.

It is forbidden to leave unattended household electrical appliances (laptops, tablets, mobile phones, chargers) connected to the electric grid in the classrooms; to plug several devices into one outlet; to use damaged (malfunctioning) plugs, sockets, extension cords and other types of malfunctioning appliances.

7. Students must know the fire safety requirements, follow them in the classrooms, not allow actions that can lead to a fire and be able to use primary fire extinguishers.
8. At the end of laboratory classes, it is necessary to put in order your workplace, and the student who is leaving the classroom the last must turn off the light there.

Safety Instructions for Working with Electrical Appliances

When working with electrical appliances (overhead projector, power supply to the microscope, etc.), there is a danger of electric injury or fire. When working with electrical equipment and electrical appliances is strictly prohibited:

– work with faulty equipment;
– work with ungrounded devices, if this is not specified in the instructions for use;
– violate the instructions for use of the device;
– touch with a hand or metal objects to parts of devices with electric current;
– check the presence of voltage in the network without special devices;
– hang various things on sockets, wires and switches;
– strengthen the wires or the density of contacts with a rope or other improvised materials;
– to leave without supervision the included electrical appliances.

After familiarizing yourself with the safety regulations, you must sign at the end of the protocol, as well as in the department "Journal of Safety Briefing for Students" that the safety briefing has been received and learned.

Familiarized and instructed(a) with the safety regulations:

_____
(Full Name)

«_____» _____ 20____ г. _____
(date) (signature)

Responsibilities of student on duty on the laboratory classes

1. The student on duty, appointed by the head of the group before the start of the lesson, checks the sanitary condition of the audience, its readiness for the lesson. If any problems are detected, the student on duty informs the laboratory assistant or teacher about them.

2. If necessary, the student on duty receives albums, methodological recommendations, atlases, teaching aids for the current lesson in the laboratory room (room number 126). At the end of the lesson, teaching aids are returned to the laboratory.

3. If necessary, the student on duty helps the leading laboratory assistant or teacher in demonstrating slides, performing demonstration work, etc.

4. At the end of the lesson, the student on duty again checks the sanitary condition of the classroom, if necessary, helps his colleagues and laboratory assistant to clean their workplaces. Duty is considered to be over when the laboratory assistant or teacher "accepts" the practice after the lesson.

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

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

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

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

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

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

Pathological Physiology is "a basis of medical professional thinking".

From a preamble of the CART charter

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Answer the following questions:

Pathological Physiology is _____

_____

_____

The subject of Pathological Physiology — _____

_____

_____

_____

The object of Pathological Physiology — _____

_____

The goal of Pathological Physiology — _____

_____

Tasks of Pathological Physiology:

1. ____
____

2. ____
____

3. ____
____

Methods of Pathological Physiology — ____
____

2. Fill in the Table:

The main sections of Pathological Physiology

<table>
<tr><th>General Nosology</th><th>Typical Pathological Processes (TPP)</th><th>Typical forms of tissues organs and systems pathology</th></tr>
<tr>
<td>General Nosology is ____________
____
____
____
1.____________
____
____
____
____
____
2.____________
____
____
____
____
____
3.____________
____
____
____________</td>
<td>Typical pathological processes — are ______________________________
____
____
Signs of typical pathological processes
<table>
<tr><th>Signs</th><th>Characteristics</th></tr>
<tr><td>1. ____________
____
____________</td><td>TPP develop under the influence of many reasons (for example, the causes of inflammation — microorganisms, mechanical trauma, exposure to heat or cold, various chemicals, etc.)</td></tr>
<tr><td>2. ____________
____________</td><td>TPP has a standard mechanism of development (for example, the pathogenesis of inflammation includes components of alteration, exudation, and proliferation).</td></tr>
<tr><td>3. ____________
____________</td><td>TPP is a complex of adaptive, compensatory and pathological changes.</td></tr>
<tr><td>4. ____________
____
____________</td><td>TPP has standard manifestations (acute inflammation is characterized by both general (leukocytosis, fever, etc.) and local (pain, redness, swelling of the tissue, fever, etc.).</td></tr>
</table>
Examples of typical pathological processes: ______________________________
______________________________</td>
<td>Examples of typical forms of pathology:
____
____
____
____
____
____
____
____
____
____
____
____
____
____
____
____
____
____
____________</td>
</tr>
<tr><td colspan="2">General pathophysiology</td><td>Specific pathophysiology</td></tr>
</table>

PART 2. STUDY OF THE MATERIALS OF EDUCATIONAL FILM

1. Fill in the Table:

Classification of biological experiments

By object	By character	By goal
1) _____ a) _____ b) _____ 2) _____	1) _____ _____ 2) _____ _____	1) _____ _____ 2) _____ _____

2. List the basic requirements for a biological experiment:

1.	
2.	
3.	
4.	
5.	
6.	
7.	
8.	

3. What is the essence of the pathophysiological experiment? _____
_____

4. What are the main disadvantages of an acute experiment? _____
_____

5. Basic requirements for animals of the control and experimental groups are: _____
_____
_____

6. List the main features of handling with old and young experimental animals: _____
_____
_____

Control questions

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 1).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.
6. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.
7. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.
8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
9. *McPhee, S. J.* Pathophysiology of disease : an introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 2. GENERAL ISSUES OF DISEASE. GENERAL ETIOLOGY AND PATHOGENESIS

Date: «____» _____ 20__

The purpose of the Lesson: to learn basic points of the study about diseases, to consolidate and check knowledge received by the students at the lessons and while studying the section "General Nosology" by manuals.

Tasks:

– to get acquainted with the contents of the Lecture "Topical Problems of General Nosology";
– to make a conclusion on a number of thematic series of slides, illustrating various aspects of general nosology;
– solution of situational tasks;
– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Put down a definition of the notion "disease": _____
_____
_____
_____

2. Put down a definition of the concept of "*pathological reaction*", give examples: _____
_____
_____

3. Put down a definition of the concept "*pathological process*", give examples: _____
_____
_____

4. Put down a definition of the concept "*pathological condition*", give examples: _____
_____
_____

5. Incidence of the disease is caused by:

1) _____
2) _____
3) _____

6. Fill in the Table:

Disease development periods

Periods	Period boundaries	Manifestations	Period duration (give examples)
I. Hidden (latent, incubative)	_____	_____	_____
II. Prodromal	_____	_____	_____
III. Marked clinical manifestations	_____	_____ *Variants of a disease course:* ▪ _____ ▪ _____	_____
IV. Disease outcome	*Possible outcomes of a disease:* – _____ – _____ – _____ – _____ – _____		

7. Put down a definition of the concept "*etiology*": _____
_____
_____

8. Fill in the Table:

Etiology	
General	**Spesific**
Pathology section studying _____ _____ _____ _____	*Pathology section studying* _____ _____ _____ _____

9. Put down a definition of the concept "*reason*": _____
_____
_____
_____

10. Fill in the Table:

Causes of the disease	
Exogenic	**Endogenic**
1. _____ 2. _____ 3. _____	1. _____ 2. _____ 3. _____
Conditions	
Contributing Disease Emergence	**Preventing Disease Emergence**
1. _____ 2. _____ 3. _____ 4. _____ 5. _____ 6. _____	1. _____ 2. _____ 3. _____ 4. _____ 5. _____ 6. _____

11. Describe the alternative concepts in etiology:

Concept name	Characteristic	What do not reveal alternative concepts of etiology
Monocausalism		
Conditionalism		
Constitutionalism		

12. Put down a definition of the concept "*pathogenesis*": _____
_____
_____

13. Fill in the Table:

Pathogenesis	
General	**Spesific**
Pathogenesis section studing _____	*Pathogenesis section studing* _____

14. Fill in the Table:

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		

15. What is the driving force of the disease development:_____
_____

16. Fill in the table, describing the pathological and compensatory reactions.

Pathological reactions	**Compensatory reactions**
1.	1.
	2.
2.	3.
3.	4.
4.	5.

17. Put down a definition of the concept "*adaptation*": _____
_____
_____

18. Put down a definition of the concept "*compensation*": _____
_____
_____

19. Fill in the Table:

Structural basics and compensation mechanisms

<table>
<tr><th>Compensatory process</th><th>Characteristic</th></tr>
<tr><td>Regeneration</td><td>Regeneration is _____
_____
Kinds of regeneration:
<table><tr><th>intracellular</th><th>cellular</th></tr><tr><td>– _____
– _____
– _____</td><td>– _____
– _____
– _____</td></tr></table></td></tr>
<tr><td>The presence of paired organs</td><td>– _____
_____
– _____
_____
Examples: _____
_____</td></tr>
<tr><td>Activation of the duplicated systems function</td><td>_____
_____</td></tr>
<tr><td>Presence of reserve structures</td><td>_____
_____</td></tr>
</table>

20. Fill in the Table:

Types of adaptive and compensatory reactions and their characteristics

Short term	Long term
1. _____ _____ _____ 2. _____ _____ _____ 3. _____ _____ _____	1. _____ _____ _____ 2. _____ _____ _____ 3. _____ _____ _____

21. Fill in the Table:

Stages of Adaptation Reactions

Stages	Characteristic
Emergency stage	– _____ _____ _____ – _____ _____
Sustained hyperfunction stage	_____ _____ _____
Stage of progressive organ sclerosis	_____ _____ _____

22. Fill in the Table:

Cross adaptation and compensation

Kind	Positive	Negative
Characteristic	_____ _____ _____	_____ _____ _____
Examples	_____ _____ _____	_____ _____ _____

23. Put down a definition of the concepts "*adaptation price*" and "*compensation price*": _____
_____
_____
_____

24. Fill in the Table:

Periods of a terminal condition

Periods	Conscious-ness (+/–)	Corneal and pupillary reflexes (+/–)	Blood circulation state		Character of respiration	Metabolic state	Duration (+/–)
			BP	pulse			
I Preagonal							
II Agonal							
III Clinical Death							

PART 2. PRACTICAL WORK

Work 1. GET A MODEL OF ALTITUDE DISEASE IN WHITE RATS, TO IDENTIFY THE DISTURBANCES AND PROTECTIVE-ADAPTIVE REACTIONS AT THIS DISEASE IN ANIMALS

Methods. Place a white rat under the hood of Komovsky's apparatus, note the behavior of the animal, the color of the skin and mucous membranes, calculate the number of respiratory movements in 1 minute, as well as the rhythm and depth of breathing, evaluate the O_2 pressure in arterial blood. Rub the cap tightly onto the vacuum plate and pump out the air. To study the above indicators at "heights" corresponding to 0.8 atm. 0.6 atm. 0.3 atm. The results of the experiment are presented in the Table.

"Climb"		Behavior features	Breath characteristic			Skin and mucous color (degree of cyanosis)	p_aO_2, mmHg
Atmospheric pressure	Height above sea level		BH/min	Rhythm	Depth		
1.0	0 m	Normal	76	+	superficial	No signs of cyanosis (–)	96
0.8	2000 m	Anxiety, involuntary acts of defecation and urination	86	+	deep	Slight signs of cyanosis (+)	90
0.6	4000 m	Anxiety, gets up on the on hind legs, sniffs	98	+	deeper	Pronounced signs of cyanosis appear, eye color acquires a cherry hue (++)	60
0.3	8000 m	Severe anxiety, cramps, impaired coordination, inability to rise on hind legs	< 52	–	deep	Extreme manifestation of cyanosis (+++)	< 40

Conclusions:

1. What signs (symptoms) of altitude sickness can be attributed to damage phenomena, and which of them are protective and adaptive reactions of the body? _____

_____

_____

_____

2. At what altitudes did the first protective and adaptive reactions of the body and the first signs of damage appear? _____

_____

_____

_____

_____

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. Development stages of the disease, outcomes of the disease.
5. 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.
6. The notion of etiology. The significance of reasons and conditions of disease development. The essence of monocausenalism, conditionalism and constitutionalism.
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 definitions of “adaptation” and “compensation”.
11. 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.
12. The staging character of the disease. Dynamics and expressivenessof pathological and compensatory reactions of the organism in the process of the disease.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 2).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

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5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.
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9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 3. PATHOGENIC INFLUENCE OF ENVIRONMENTAL FACTORS ON THE HUMAN ORGANISM. ACTION OF ELECTRIC CURRENT ON THE ORGANISM

Date: «____» _____ 20__

The purpose of the Lesson: to study the peculiarities of the electric current damaging effects on the organism.

Tasks:

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

– to get acquainted with characteristic consequences of electrotrauma in humans (demonstration of slides);

– solution of situational tasks;

– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. List the features of electric current:

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. _____
8. _____
9. _____

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

1. _____
2. _____
3. _____

3. What frequency (Hz) and type (alternating/constant) of electric current is the most dangerous for the human body? _____

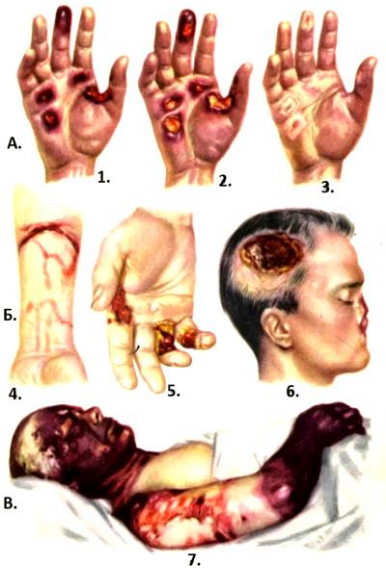
_____

_____

4. Are high frequency currents dangerous? What are the areas of their application? _____
_____
_____

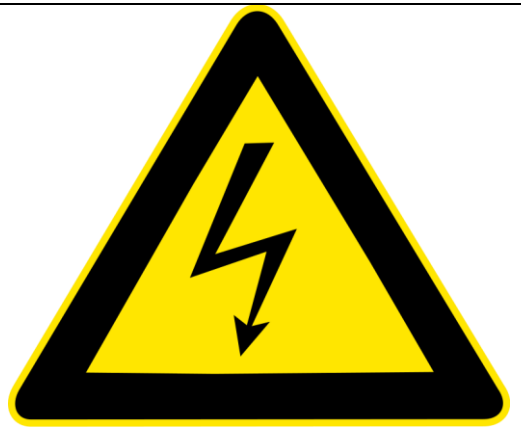
5. Fill in the Table:

Pathogenesis of electric injury

The effect of electric current on the body		
Specific		**Nonspecific**
Type	**Characteristic**	
Biological action	_____ _____ _____ _____	*The nonspecific effect of current is* _____ _____ _____ _____ *Examples:* _____ _____ _____ _____
Electrochemical (electrolytic) action	_____ _____ _____	
Electrothermal action	_____ _____ _____ *Electric labels — are* _____ _____ _____ _____	 A — in case of violation of the insulation of an electric iron (220 V): 1 — before treatment; 2 — during the treatment period; 3 — after healing; B — from the electric wire and plug of the electric iron (220 V): 4 — on the forearm; 5 — on the brush; b — on the head; B — from the electric arc during the repair of the electrical installation under voltage 380 V: 7 — on the face, neck and upper limb. **Contact electric trauma. Signs of current**
Electromechanical (dynamic) action	– _____ _____ – _____ _____ _____	

6. Fill in the Table:

Changes in the body under the influence of electric current

Changes	**Local**	**General**
Early	**Electric burns**	
	Kinds: – _____ – _____ – _____ – _____	
	4 degrees of severity: 1. _____ 2. _____ 3. _____ 4. _____	
	Features: 1. _____ 2. _____ 3. _____ 4. _____	 ***Warning symbol for electric shock***
Later	_____	

7. Fill in the Table:

Causes of death due to electrical injury

Form	Cardiac form	Respiratory form	Mixed form
Reasons	1. _____ _____ 2. _____ _____ 3. _____ _____ 4. _____	1. _____ _____ 2. _____ _____ 3. _____ _____ 4. _____	_____ _____ _____ _____ _____ _____ _____
Skin color			

8. Fill in the Table:

Electro-traumatic shock

Phase	CNS state	BP	Respiratory system	Convulsions (+/-)	Vital functions
I ( )					
II ( )					

9. Fill in the Table:

The damaging factors of atmospheric electricity

Damaging factor	Characteristic manifestations

10. Indicate the principles of electrical injury assistance:

_____
_____
_____

PART 2. PRACTICAL WORK

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

Experimental technique

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

Results of the experiment

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

Conclusions:

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

_____

_____

2. How does the reflex time depend on the duration of the electric current? _____

_____

_____

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

Experimental technique

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

- In the **1-st** mouse — to hind paws (switched on electric current will pass through hind extremities of the animal);
- In the **2-nd** — to auricles, thus providing the passage of the current through the head of the animal;
- In the **3-rd** mouse — to the fore left and hind right paws (switched on electric current will pass through the heart).

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

Results of the experiment

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

Conclusions:

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

_____

_____

_____

Control questions

1. Features of electric current as a damaging factor.
2. Factors affecting the severity of damage to the body under the influence of electric current.
3. Types of electric damage (local and general, specific and non-specific) and their characteristics.
4. Causes of death due to electrical injury and their mechanisms. “Imaginary death”.
5. Principles of first aid for electric shock.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 3).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *Электротравма* (патофизиологические аспекты) = Electric injury (pathophysiological aspects) : учеб.-метод. пособие / Д. М. Попутников [и др.]. Минск : БГМУ, 2014. 20 с.
5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
6. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.
7. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.
8. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.
9. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
10. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher’s signature: _____

LESSON 4. PATHOGENIC INFLUENCE OF ENVIRONMENTAL FACTORS ON THE HUMAN ORGANISM. HARMFUL ACTION OF IONIZING RADIATION ON THE ORGANISM

Date: «____» _____ 20__

Purpose: to study the pathophysiological aspects of radiation damage, their nature, developmental mechanism, outcomes. Give pathogenetic characteristics of various types of radiation lesions.

Tasks:

– to get acquainted with the local and general manifestations of acute radiation sickness on the basis of the teaching manual "acute radiation sickness";

– solving situational problems;

– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. According to the Law of the Republic of Belarus "On radiation safety of the population" of June 18 2019 No. 122-3 define the notion of *"Ionizing radiation"*: _____
_____
_____

2. Fill in the Table:

Types of ionizing radiation

By physical nature	1. _____ 2.
By exposure pathways	1. _____ 2. _____ 3.
By duration of exposure	1. _____ 2. _____ 3.

3. Factors determining the severity of ionizing radiation damage: _____
_____
_____

4. Give the definition of the concept *"radiosensitivity"*: _____
_____

5. Bergonier — Tribondo Rule: _____
_____

6. Define the concept of *"critical organs"* and list them: _____
_____
_____

7. Fill in the Table:

Radiation damage at different levels of biological organization

Level of biological organization	**Characteristics of radiation damage**
Molecular	
Subcellular	
Cellular	
Tissue, organ	
Organism	
Population	

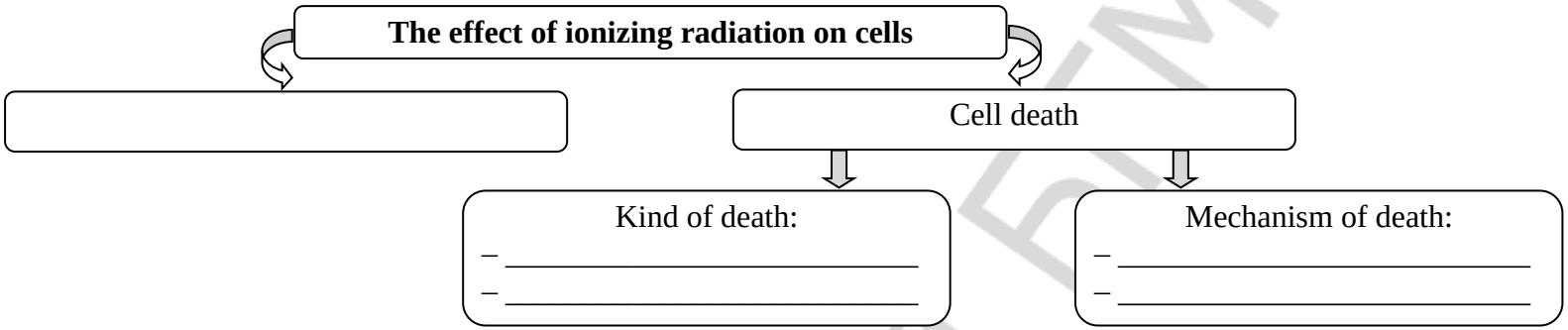
8. What are the stages of radiation damage (*structural-metabolic theory of A. M. Kuzin (1986)*):

1. _____
2. _____
3. _____

9. What are the stages of radiobiological effects development?

1. _____
2. _____
3. _____
4. _____

10. Fill in the scheme:



11. Give the definition of «*radiation sickness*»:_____
_____

12. Give the definition of *"acute radiation sickness"*: _____
_____

13. Give the definition of *"chronic radiation sickness"*:_____
_____

14. Fill in the Table:

Characteristics of radionuclides distribution in the human body

Type of distribution	**Characteristics, examples**
Skeletal	
Reticuloendothelial	
Diffuse	
Selective	

15. What are the main syndromes of *acute radiation sickness*? Give their characteristic.

1) _____;
2) _____;
3) _____.

16. Fill in the Table:

Characteristic of acute radiation sickness

<table>
<tr><th>Form</th><th>Dose (Gy)</th><th>Mortality</th><th>Manifestation</th></tr>
<tr><td>Typical
bone marrow form</td><td>_____</td><td>_____</td><td>IV degrees of severity (depending on dose):
I – _____ (_____ Gy);
II – _____ (_____ Gy);
III – _____ (_____ Gy);
IV – _____ (_____ Gy).</td></tr>
<tr><td colspan="4">Includes 3 periods:
1) Formation period:
<table>
<tr><th>Phases</th><th>Duration</th><th>Symptoms</th><th>Blood parameters</th><th>Bone marrow</th></tr>
<tr><td>Primary acute reaction</td><td>_____</td><td>_____</td><td>_____</td><td>_____</td></tr>
<tr><td>Clinical phase of imaginary well-being</td><td>_____</td><td>_____</td><td>_____</td><td>_____</td></tr>
<tr><td>Phase of expressed clinical manifestations</td><td>_____</td><td>_____</td><td>_____</td><td>_____</td></tr>
<tr><td>Early recovery phase</td><td></td><td></td><td></td><td></td></tr>
</table>
2) Recovery period _____
3) The period of outcome and consequences _____</td></tr>
<tr><td>Intestinal form</td><td>_____</td><td>_____</td><td>Main clinical manifestations: _____
_____
Changes in the blood: _____
Death occurs on the _____ day.</td></tr>
<tr><td>Toxemic form</td><td>_____</td><td>_____</td><td>Manifestations: _____
_____
Death occurs on the _____ day.</td></tr>
<tr><td>Cerebral form</td><td>_____</td><td>_____</td><td>Manifestations: _____
_____
Death occurs on the _____ day.</td></tr>
</table>

17. Fill in the Table:

Possible consequences of exposure

Effects	Stochastic	Non-Stochastic
Characteristic	_____	_____
Examples	– _____	_____

18. List the possible mechanisms for the formation of long-term effects of radiation:

1. _____
_____
2. _____
_____
3. _____
_____

PART 3. PRACTICAL PART

Work 1. STUDYING THE ACTION OF IONIZING RADIATION ON WHITE BLOOD CELLS

The conditions of the experiment. The study was carried out on two mice (Table 1.1, 1.2), one of which three days before the session was subjected to X-ray irradiation at a dose of 5 Gy (mouse No. 1), the second mouse was not exposed to radiation (mouse No. 2). Both mice were sampled from an incision in the tail tip into a leukocyte mixer and their number was counted in a Goryaev chamber.

Table 1.1

Observation Results

Pressure	Red blood cells, * 10^{12}/l	Reticulocytes, %	Respiratory rate per minute	Skin color and mucous membranes	Other changes
1 atm (before experience)	6.0	30	80	Pink	–
0.8 atm.			120	Pink	–
0.6 atm.	–	–	130	Mild acrocyanosis	Anxiety, shortness of breath
0.4 atm.	–	–	60	Expressed acrocyanosis	Reduced mobility intermittent breathing
At the end of the experiment	7.5	30	90	Pink	Normalization of state

Table 1.2

Observation Results

Object of study	X-ray dose, Gy	Total leukocyte count, * 10^9/l
Mouse № 1	5	3.2
Mouse № 2	–	13.4

Note: white blood cell count in healthy mice — $(13.4 \pm 0.4) \cdot 10^9$/l.
The results of white blood cell count in healthy and irradiated mice are compared. Assess the severity of radiation damage.

Answer the following questions:

1. What is the mechanism of leukopenia development at radiation sickness? _____
_____
2. What form of ARS developed in a mouse at a given dose of radiation?_____
_____
3. What form and phase of ARS is characterized by the most expressed leukopenia?_____
_____

Control questions

1. Ionizing radiation. The definition and general characteristic.
2. Peculiarities of ionizing radiation effect as a damaging factor.
3. Dose characteristics of ionizing radiations.

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 4).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

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4. *Жадан, С. А.* Повреждающее действие ионизирующей радиации = Damaging action of ionizing radiation : учеб.-метод. пособие / С. А. Жадан, Ф. И. Висмонт, Е. В. Меленчук. Минск : БГМУ, 2016. 26 с.
5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
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10. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 5. ROLE OF REACTIVITY, CONSTITUTION AND AGE IN PATHOLOGY DEVELOPMENT

Date: «____» _____ 20__

The purpose of the Lesson: to study factors and the mechanisms determining reactivity and resistance of the organism, the role of constitution and age 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;

– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Put down a definition «*reactivity*»: _____
_____
_____
_____
_____

2. Put down a definition *"resistance"*:_____
_____
_____
_____
_____

3. What is the interrelation between *reactivity* and *resistance* of the organism? _____
_____
_____
_____
_____

4. Fill in the missing information in the table:

Indicators of Reactivity

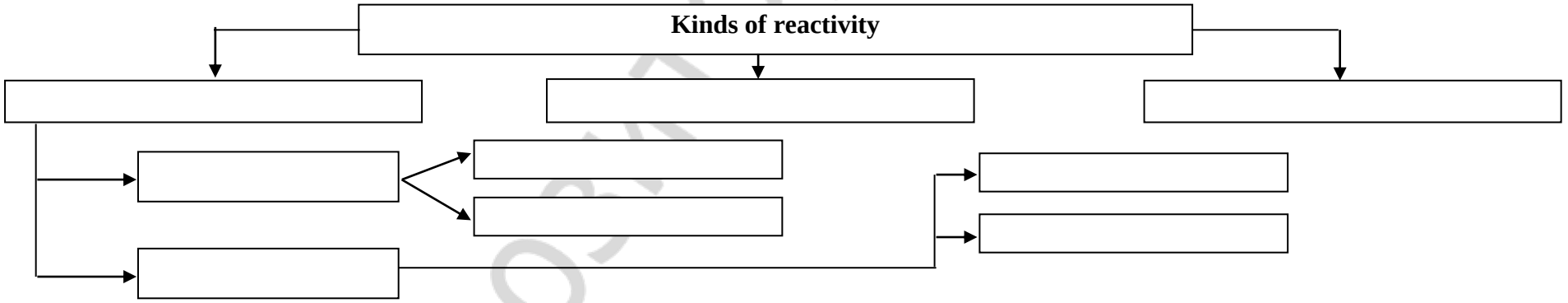
Quantitative		**Qualitative**	
Indicator	**Characteristic**	**Indicator**	**Characteristic**
1. Normergy	_____ _____ _____	_____ _____ _____	the most important qualitative indicator of reactivity; body resistance to pathogenic factors
2. _____	reduced reactivity, with a predominance of inhibitory processes	*Functional lability*	_____ _____ _____ _____
3. Hyperergy	_____ _____ _____	_____ _____ _____	a common property of all living things that determines elementary reactions
4. _____	perverse reactivity	*Excitability*	_____ _____ _____ _____ _____
		Sensitivity	_____ _____ _____ _____
		_____ _____	the shortest duration of the stimulus action of a double threshold force; which is sufficient to cause a physiological effect

5. Fill in the Table:

Ways and methods increasing nonspecific resistance of the organism

By reducing the activity of vital processes (group 1)	**By maintaining or increasing the level of organism vital activity (group 2)**	
Means, techniques, methods	*Means, techniques, methods*	*Examples*
1. _____		
2. _____		
3. _____		
4. _____		

6. Fill in the Table:



7. Put down a definition of the notion *"constitution"*:_____

_____

8. The founder of the doctrine of man constitution is _____

9. Fill in the Table:

Basic principles for the classification of constitutional types

Author	Classification Criteria	Species
Hippocrates		– – – –
Seago		– – – –
E. Kretschmer		– – –
M. V. Chernorutsky		– – –
Eppinger and Hess		– –
A. A. Bogomolets		– – – –
I. P. Pavlov		– – – –
W. H. Sheldon		– – –

10. Fill in the Table:

Features of aging

Feature	Characteristic

11. Fill in the Table:

Kinds of aging

Kinds of aging	Characteristic

PART 2. PRACTICAL PART

Work 1. DYNAMICS STUDY OF RESPIRATION AND EXCHANGE PROCESSES CHANGES IN THE DEVELOPMENT OF HYPOXIA IN MICE WITH VARIOUS FUNCTIONAL STATE OF THE CENTRAL NERVOUS SYSTEM (experiments № 1, 2)

Experiment № 1

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

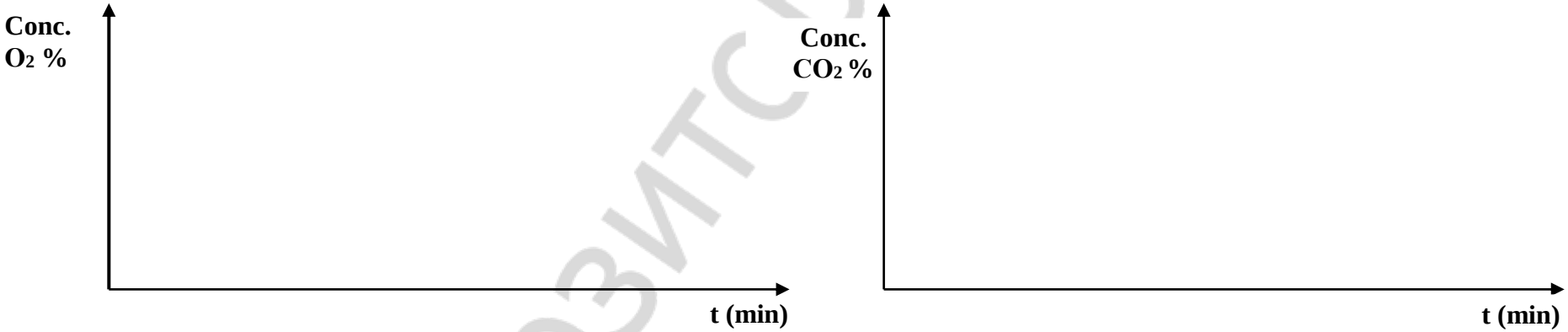
Both mice — an intact, unnarcotized one (control) and narcotized (tested) — are placed in two large-mouthed flasks of identical capacity (100 ml). The flasks are simultaneously closed by rubber corks with subsequent hermetic sealing by paraffin. We observe the behavior of mice, count respiration rate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

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

Мин	**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 is anxious. It stands up on hind paws more often. The breath has accelerated and is deeper	84	Is asleep. Respiration is calm
12	162	The anxiety of the mouse increases. It makes sharp movements. It pulls at the cork with paws. Cyanosis of ears, the nose tip, paws	72	The mouse sleeps. The respiration is regular
15	180	Sharp anxiety. Cyanosis. Breathlessness	68	No changes
18	176	Motor activity is weakened. Sharp cyanosis	62	Cyanosis signs of ears, the nose tip, paws have appeared
22	22	The mouse is lying on one side. Breath is intermittent	50	Cyanosis
23	22	Spasms, tail reaction, defecation, urination	50	Marked cyanosis
24	–	Respiratory arrest	48	–
35	–		12	–
35	–		6	–
45	–			Respiratory arrest
	Gas mixture content in the flask: O_2 = 7.1 %; CO_2 = 11.8 %		**Gas mixture content in the flask: O_2 = 3.4 %; CO_2 = 14.6 %**	

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

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



Answer the questions:

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

_____

_____

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

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

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

5. Calculate and compare an average speed (V) concentration changing of oxygen and carbon dioxide in the flasks with a control (V_1) and tested (V_2) mice, having assumed the initial concentration of O_2 and CO_2 equals to 21 % and 0.03 %, accordingly:

$V_1 = \Delta O_2 / t_1 =$

$V_1 = \Delta CO_2 / t_1 =$

$V_2 = \Delta O_2 / t_2 =$

$V_2 = \Delta CO_2 / t_2 =$

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

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

Experiment № 2

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

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	
25	168	The same condition	70	Weak cyanosis signs
28	150	The mouse has fallen down. Periodically it jumps up. Sharp cyanosis	58	No changes
31	–	The mouse is lying on one side. Sharp cyanosis. Intermittent respiration. Spasms. Agonal breathing. Respiratory arrest	50	No changes
32	–	Spasms. Agonal breathing	50	The mouse is asleep
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. Which of these two strategies of a survival in extreme conditions is used in experiments of Konstantinov and Maistrah? _____

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

_____

_____

Work 2. STUDY OF THE INFLUENCE OF THE GENDER FACTOR ON HUMAN STABILITY TO SHORT-TERM HYPOXIA

The participants in the experiment are invited to make the maximum arbitrary delay of breath after a deep breath. Measure it in seconds. Calculate the average breath holding time for men and women in your group. Compare results.

1. My breath holding time (sec.): _____

2. Fill in the table based on the data of the entire group:

	Male	**Female**
Average breath holding time, sec		

3. **Conclusion** (analyze who is more resistant to short-term hypoxia — men or women — and why?):

_____

_____

Work 3. INFLUENCE OF TYPE OF CONSTITUTION (BY CHERNORUTSKY) ON HUMAN RESISTANCE TO SHORT-TERM HYPOXIA

All students in the group are encouraged to make the maximum arbitrary breath holding after the maximum breath. Measure it in seconds. For each student, determine the type of constitution (according to Chernorutsky) using the formula:

Constitution indicator (CI) = height (cm) – (weight (kg) + chest circumference (cm)) **CI = 10–30 — normosthenic type; CI > 30 — asthenic type; CI < 10 — hypersthenic type**

My CI = is_____

To divide all subjects into three groups according to the type of constitution (CI). To calculate the average breath holding time in each group. To compare results.

	Asthenic type	**Normostenic type**	**Hypersthenic type**
Average breath holding time, sec.			

Conclusion (to analyze who was more resistant to short-term hypoxia depending on the type of constitution, and also suggest why): _____
_____

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.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 5).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.
6. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.
7. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.
8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 6. THE ROLE OF HEREDITY IN PATHOLOGY

Date: «____» _____ 20__

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

Tasks:
- to solve situational tasks in medical genetics;
- to study the genotype and clinical manifestations of hereditary pathology with tables, slides and video;
- control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Fill in the Table:

Characteristics of the diseases taking into account the role of heredity and the environmental factors in their occurrence

Group of diseases	The role of heredity and the environment	Examples of the disease
Hereditary diseases		
Single-gene diseases		
Multifactoral polygenic diseases		
Diseases caused by environmental factors		

2. Fill in the scheme:

Correlation of the concepts of “congenital diseases”, “hereditary diseases” and “phenocopies”

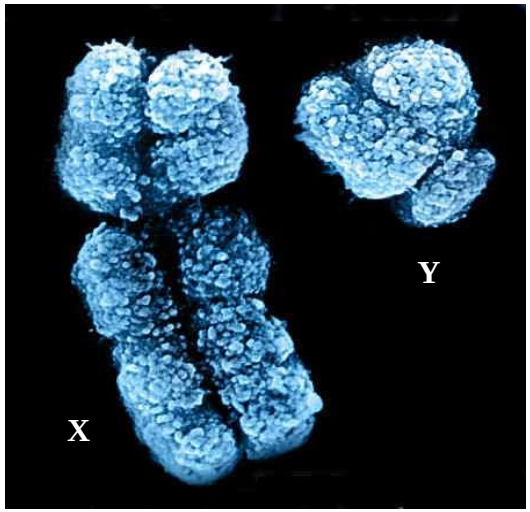
Congenital diseases

definition:

_____
_____
_____
_____
_____
_____

– _____

– _____



Hereditary diseases

definition:

_____
_____
_____
_____
_____

clinical manifestation time:

_____
_____
_____
_____

Non-Hereditary diseases

Definition: _____
_____
_____

Characteristic: _____
_____
_____
_____
_____
_____

Phenocopies

definition:

_____
_____
_____
_____
_____
_____

examples:

_____
_____
_____
_____

the reasons of occurrence:

1. _____
_____
2. _____
_____
3. _____
_____
4. _____
_____
5. _____
_____
6. _____
_____

3. Put down the definition of the notion "*mutation*": _____
_____
_____

4. Fill in the Table:

Kinds of mutations

By reasons:	1. _____ 2.
By the type of cells:	1. _____ 2. _____ 3.
Depending on the value for the organism:	1. _____ 2. _____ 3.
Depending on the extent of genome damage	1. _____ 2. _____ 3.

5. Put down the definition of the notion "*gene mutation*":_____
_____
_____

6. Fill in the Table.

Kinds of gene mutations depending on type of molecular changes

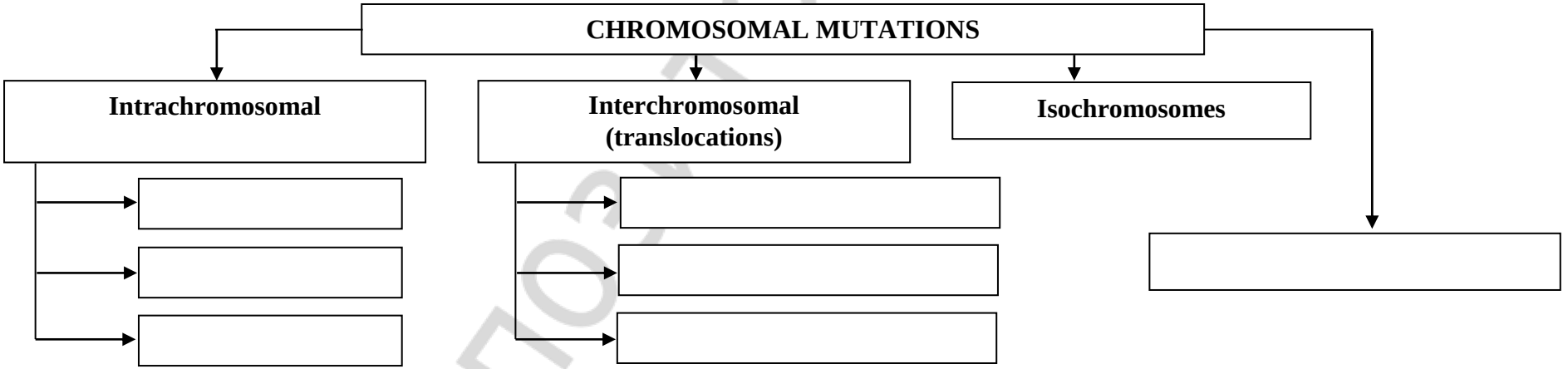
Gene mutation	**Characteristic**
Deletion	
Duplication	
Inversion	
Insertion	
Transversion	
Transition	

7. Fill in the Table.

Characteristic of some gene diseases

Disease	Type of inheritance	Characteristic
Phenylketonuria		Defect: _____ Clinical manifestations: _____
Alcaptonuria		Defect: _____ Clinical manifestations: _____
Galactosemia		Defect: _____ Clinical manifestations: _____
Hepatocerebral dystrophy		Defect: _____ Clinical manifestations: _____

8. Fill in the Scheme:



9. Fill in the Table.

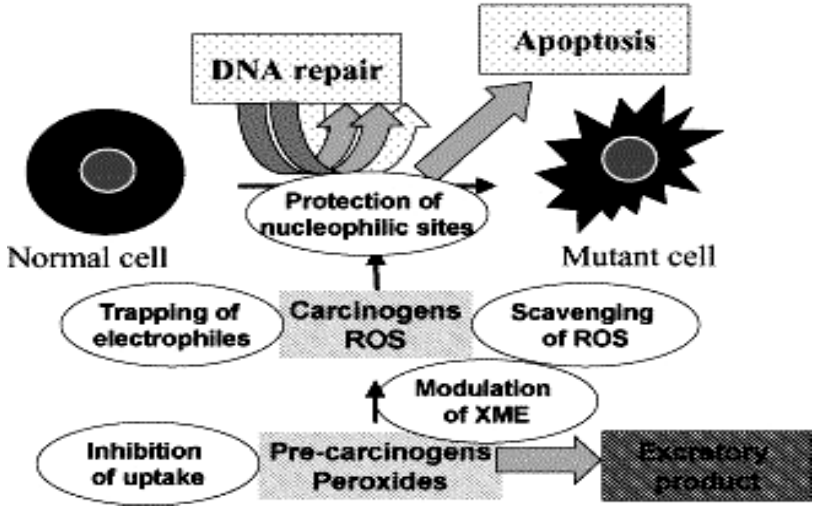
Characteristic of some chromosomal syndromes

Chromosomal mutation	Syndrome	Characteristic
Trisomy 13		Frequency: _____ Clinical symptoms: _____
Trisomy 18		Frequency: _____ Clinical symptoms: _____
Trisomy 21		Frequency: _____ Clinical symptoms: _____
XXY		Frequency: _____ Clinical symptoms: _____
XXX		Frequency: _____ Clinical symptoms: _____
XYY		Frequency: _____ Clinical symptoms: _____
XO		Frequency: _____ Clinical symptoms: _____
46,XX(XY),5p-		Frequency: _____ Clinical symptoms: _____
46,XX(XY),4p-		Frequency: _____ Clinical symptoms: _____

10. Give the definition of the notion "*antimutagenesis*":_____

11. Fill in the Table.

Classification of antimutagens

<table>
<tr><th>Exogenous</th><th>Endogenous</th></tr>
<tr><td>Antimutagens in foodsand entering the human body with food
1. _____
2. _____
3. _____
4. _____
5. _____</td><td>1. _____
2. _____
3. _____
4. _____</td></tr>
<tr><td>Antimutagens penetrating into the body through the respiratory system
_____</td><td rowspan="2"> 

Mechanisms of antimutagens action</td></tr>
<tr><td>Antimutagens, ingested orally in the pharmacotherapy, or prophylactic uses
1. _____
2. _____
3. _____
4. _____</td></tr>
</table>

12. Indicate and characterize the subsystems of mother-placenta-fetus-functional-system (MPFFS):

1) _____
2) _____
3) _____

13. Fill in the Table:

Types of fetal abnormalities

Abnormality	Terms of occurrence	Effects

PART 2. PRACTICAL PART. SOLVING OF SITUATIONAL TASKS

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____

G: _____

F_1: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____

G: _____

F_1: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

9. In genetic consultation pregnant woman C. informed, that her sister was ill with ***phenylketonuria***, but she herself didn't suffer from this pathology. C.'s husband was healthy. There were kindred marriages between close relatives in her husband's family, but none of them was ill with phenylketonuria. Is there any danger for her child to get this disease? What is the probability of it?

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

What is the role of sex? _____
Is it possible to treat such a disease? _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

16. Healthy woman H., whose father is ill with hemophilia ***A*** and mother is healthy, referred to genetic consultation with a question: whether probability for her grandsons to get this disease is great? Spouse H. and their three children — a son and two daughters — are healthy.

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

What is the type of inheritance and what is the development of hemophilia A caused by? _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

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

Character	Gene	Genotype

P: ♀ _____ **x** ♂ _____
G: _____
F₁: _____ **Answer:** _____

20. How many bodies of sex chromatin are there in people with genotype ОХ? ХХУ? ХХХ? ХХХУ? What is the sex of these people and what are they ill with?

Genotype	Number of sex chromatin bodies	Gender	Disease
ОХ			
ХХУ			
ХХХ			
ХХХУ			

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

What is the name of the mentioned syndrome? _____
What is the karyotype of the mentioned syndrome? _____

22. In patient M., height of 153 cm, there skin fold on the neck, «sphinx» neck, primary amenorrhea, sterility. There are congenital defects of the heart and kidneys.

What is the name of the mentioned syndrome? _____
What is the karyotype of the mentioned syndrome? _____

Examples of diseases with different types of inheritance

Inheriotance type	Pathology form	
1 Autosomal-dominant (A-D)	Polydactylism Brachydactylism Dactylion Curvature of fingers, nails Anonychia (underdevelopment of nails) Absence of lateral incisors Short-sightedness Far-sightedness	Astigmatism Otosclerosis Achondroplasia Family hypercholesteremia Chorea of Huntington Polyposes of the large intestine Neurofibromatosis
2 Autosomal-recessive (A-R)	Crescent — cellular anemia (by incomplete domination) Galactosemia Phenylketonuria Alcaptonuria Albinism Glycogenosis	Mucoviscidosis Wilson–Konovalov Disease (hepato-cerebral dystrophy) Adrenogenital syndrome Congenital deaf-muteness Microcephaly
3. Dominant X-linked (D-X)	Frontal-nasal dysplasia Hypoplasia of dental enamel	Cataract Rickets, resistant tovitamin D
4. Recessive X-linked (R-X)	Hemophilia A and B Daltonism Hypogammaglobulinemia	Duchenne's muscular dystrophy Hemeralopia
5. Hollandric Y-linked (H-Y)	Escessive hairiness of auricles	Azospermia
6. Mitochondrial (M)	Leber's optic atrophy Mitochondrial encephalopathy	Myoclonal epilepsy Cardiomyopathy

Control questions

1. Medical genetics, its tasks.
2. Classification of diseases taking into account the specificity of heredity and environment in their development. The concepts of penetrance and expressivity. The role in pathology.
3. Hereditary and congenital forms of pathology.
4. Phenocopies. The definition, causes of development. Examples.
5. Classification principles of hereditary forms of pathology.

6. Etiology of hereditary forms of pathology. Mutagen. Mutation, the definition of the notion. Kinds of mutation. Antimutagenesis. The mechanisms of antimutagen factor action.

7. Mono- and polygenic inherited diseases. Hereditarily determinant metabolic diseases: alcaptonuria, phenylketonuria, hepatocerebral dystrophy, galactosemia. Pathological heredity linked to sex (color blindness, hemophilia A and B, hypoplasia of tooth enamel, etc.). Type of inheritance, causes, development mechanisms, manifestations.

8. Chromosomal diseases: Down's disease, Patau's Syndrome, Edwards Syndrome, Klinefelter's Syndrome, X-chromosome Trisomy Syndrome, Shereshevsky-Turner Syndrome, "Cat's cry Syndrome". Causes of development, karyotype, symptoms.

9. Methods of hereditary forms of pathology studying: clinical-genealogical, cytogenetic, twin, biochemical, dermatoglyphics, demographic-statistical, experimental.

10. Pathology of fetal development. Gametopathy, blastopathy, embryopathy, fetopathy, stillbirth.

11. Principles for the prevention and treatment of hereditary diseases and developmental abnormalities, diseases with a hereditary predisposition.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 6).

2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.

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4. *Жадан, С. А.* Роль наследственности в патологии = The role of heredity in pathology : учеб.-метод. пособие / С. А. Жадан, Д. М. Попутников, Ф. И. Висмонт. Минск : БГМУ, 2014. 36 с

5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.

6. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.

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9. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.

10. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

SECTION II
TYPICAL PATHOLOGICAL PROCESSES

LESSON 7. PATHOPHYSIOLOGY OF REGIONAL BLOOD CIRCULATION AND MICROCIRCULATION. ARTERIAL AND VENOUS HYPEREMIA. ISCHEMIA

Date: «____» _____ 20___

The purpose of the Lesson: to study the incidence causes, development mechanisms, basic manifestations, outcomes and the significance for the organism of both arterial and venous hyperemia and ischemia.

Tasks:

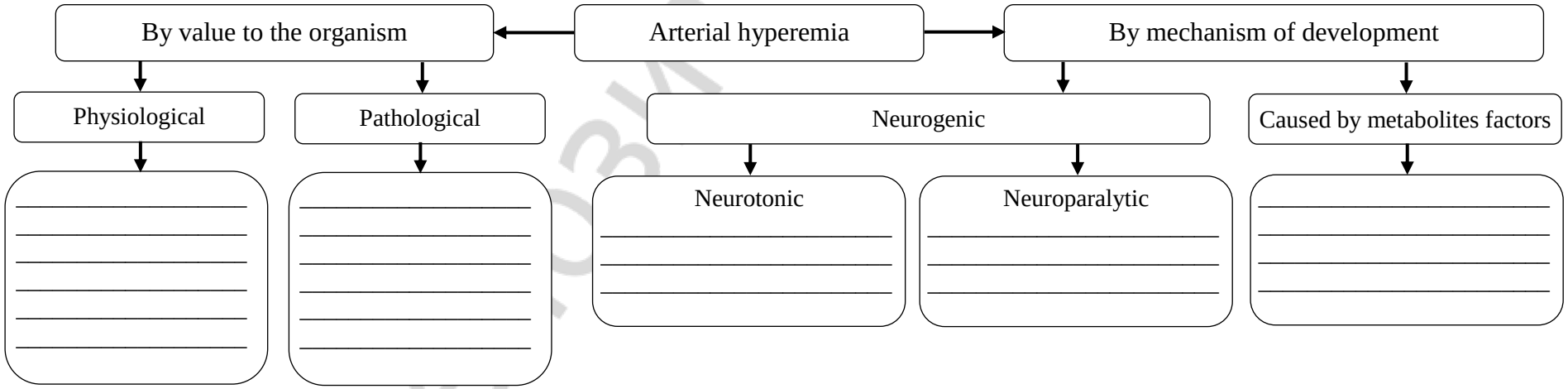
- to get acquainted with conditions of experimental work, to take part in accomplishment of experiments;
- to analyze experimental data, to present them in figures, to formulate conclusions, to give them a written substantiation;
- solving situational tasks.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Put down the concept of the notion "*arterial hyperemia*" (*AH*) — _____

_____

2. Put down the classification of *arterial hyperemia* by origin and name the main reasons for their occurrence:



3. What is the fundamental difference between *pathological* arterial hyperemia and *physiological* one? _____
_____

4. The value of VH for the body:

Positive	Negative

5. Define the concept of «*venous hyperemia*» (*VH*) — _____
_____

6. What are the main causes of venous hyperemia?

Systemic VH:	Local VH:
1.	1.
2.	2.
3.	3.

7. The value of venous hyperemia for the body:

Positive	Negative

8. Define the concept of *"ischemia"* —_____
_____

9. List and characterize the types of ischemia depending on the causes and mechanisms of its development:

Kind of ischemia	Characteristic
1.	
2.	
3.	
4.	

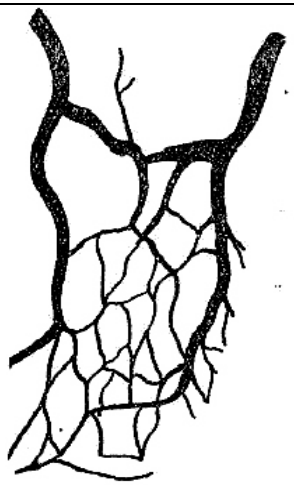
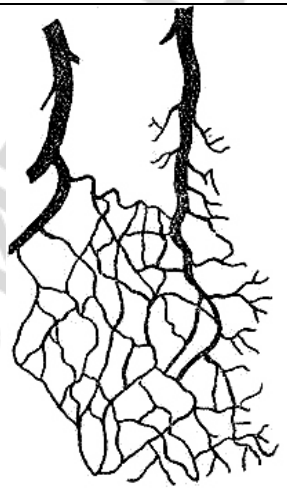
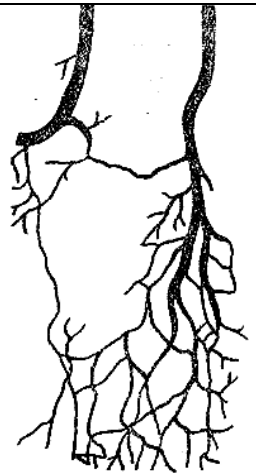
10. List the factors that determine the outcome of acute ischemia:

1. _____ 4. _____
2. _____ 5. _____
3._____

11. 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 absolutely insufficient		
3	Functionally relatively insufficient		

12. Record the names of collaterals types between the arteries in accordance with their characteristic drawings:

PART 2. PRACTICAL PART

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

We investigate the 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 the characteristic changes of blood circulation. We sketch the initial condition of vessels and the revealed changes.	 *Fig. 1.* Arterial hyperemia of a rabbit's ear: 1 — the control (an intact ear); 2 — heat exposure (arterial hyperemia)

Conclusion:
Specify the development mechanism of arterial hyperemia in this experiment:_____

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

A cork is inserted into an auricle of the rabbit so that the groove has fallen on the central artery of the ear. Then, using a ligature, the rabbit's ear is tightly fixed to the cork resulting in the impairment of blood circulation, i. e. the outflow of blood by veins is impeded. In 30–40 min we notice the signs of venous hyperemia to appear. We describe it and sketch them.	 *Fig. 1.* Venous hyperemia of a rabbit's ear: 1 — the control (an intact ear); 2 — impairment of venous outflow (VH)

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. 1.* 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 +/-)	Linear blood flow velocity	Volumetric blood flow velocity	Organ function
Arterial hyperemia										
Venous hyperemia										
Ischemia										

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

Vasodilatators:_____

Vasocontrictors:_____

Control questions

1. Typical forms of impairments of peripheral blood circulation. General characteristic.
2. The definition of the notion of arterial and venous hyperemias, ischemia; external manifestations, the reasons and development mechanisms, outcomes.
3. Changes in tissues in the area of arterial and venous hyperemias and ischemias, their significance and possible consequences.
4. The state of microcirculation in peripheral blood circulation impairments: ischemia, arterial and venous hyperemia.
5. Compensatory reactions in the impairments of local blood circulation. Post-ischemic reperfusion. Mechanisms of triggering and developing collateral blood circulation. Types of collaterals. Cerebral and cardiac steal syndromes.
6. General changes in the organism during impairments of peripheral blood circulation (arterial and venous hyperemias, ischemia) in vital organs (the heart, the brain).
7. Comparative characteristic of impairment manifestations of peripheral blood circulation: both arterial and venous hyperemias and ischemia.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 7).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
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8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 8. PATHOPHYSIOLOGY OF REGIONAL BLOOD CIRCULATION AND MICROCIRCULATION. THROMBOSIS. EMBOLISM. STASIS

Date: «____» _____ 20__

The purpose of the Lesson: to study the reasons, incidence of conditions, development mechanisms, basic manifestations and consequences for the organism of thrombosis, embolism, stasis.

Tasks:

- to study the reasons and development mechanisms of typical impairments of microcirculatory channels on the basis of textbooks;
- to get acquainted with modeling thrombosis and embolism in vessels of the tongue and intestinal mesentery of a frog;
- solving situational tasks.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Give the definition of "stasis" — _____
_____

2. List and characterize the types of stasis:

– _____
– _____
– _____

3. What are the main causes of true stasis: _____
_____

4. Specify the main mechanisms of *true* stasis development: _____
_____
_____

5. Specify the consequences of irreversible stasis: _____

6. Give the definition of *"thrombosis"* — _____
_____

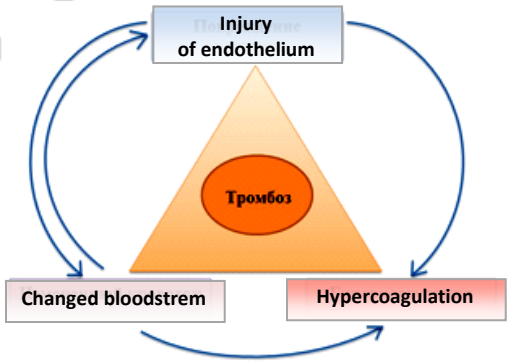
7. Specify the types of blood clots:

- *By structure:*
  1) _____ (composition: *predominantly* _____);
  2) _____ (composition: *predominantly* _____);
  3) _____ (composition: *predominantly* _____).
- *In relation to the lumen of the vessel:* 1) _____; 2) _____ .

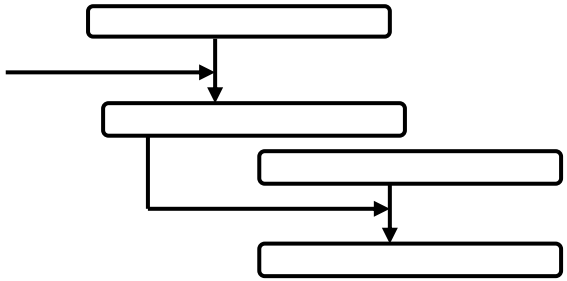
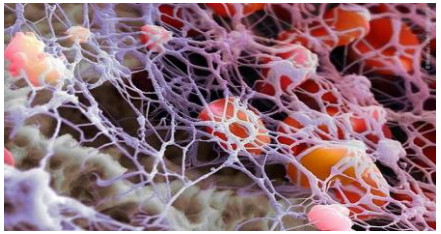
8. What parts are in a mixed thrombus: 1) _____; 2) _____; 3) _____.

9. The main factors contributing to thrombosis (the Virchow's triad) are:

1) _____
_____
2) _____
_____
3) _____
_____

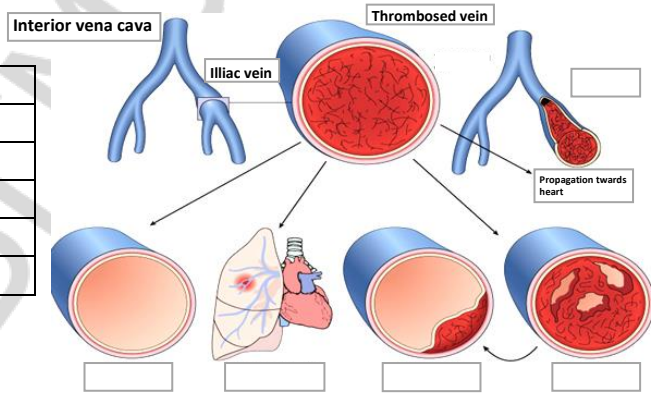


10. Fill in the Table.

Phase	Cellular	Coagulation
The essence and stages	*Fill __ with arrows* (↓, ↑): __ electrical charge of blood cells (platelets) ↓ change in the electrical potential of the vascular wall ↓ __ ATP, __ ADP in damaged areas of blood vessels and platelets ↓ __ ability of platelets to adhesive and aggregate ↓ __ antiaggregatory properties of the vascular wall in case of damage to the endothelium ↓ exposure of the subendothelium, where the adhesive proteins are	With the breakdown of platelets, enzymes (activators of the coagulation process) are released. Platelet coagulation factors (thromboplastin) enter to the plasma.
		I  II
		Under the influence of thrombastenin (retractozyme), thrombus retraction (compaction, the formation of a dense fibrin clot) occurs
	Adhesion — is _____	 ***Fibrin***
	Aggregation — is _____	
	Agglutination — is _____	
Thrombus color		

11. Name possible outcomes of thrombosis and indicate some of them on the picture.

1.
2.
3.
4.
5.
6.



12. Give the definition of the term *"embolism"* — _____

_____

13. Fill in the scheme.

Classification of emboli by the nature**:**

Kinds and their characteristic:	Kinds and their characteristic:
1. _____	1. _____
_____	_____
2. _____	2. _____
_____	_____
3. _____	3. _____
_____	_____
4. _____	4. _____
_____	_____

14. Classification of embolism:

by localization	by direction of the embolus moving
1)	1)
2)	2)
3)	3)

15. Fill in the tables.

Basic localization and signs of thromboembolism

Vascular region-source of thromboembolism	**Veins of lower extremities, small pelvis organs, right heart chambers**	**Veins of lower extremities, small pelvis organs, right heart chambers**	**Veins of lower extremities, small pelvis organs, right heart chambers**
The vascular region exposed to embolization			
The vascular region exposed to embolization			
The vascular region exposed to embolization			

PART 2. PRACTICAL PART

Work 1. THE FORMATION PROCESS OF A WHITE MURAL BLOOD CLOT IN MESENTERIC VESSELS OF A FROG

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

Use the obtained preparation for examining the picture of normal blood circulation in mesentery vessels of the frog's intestines under the microscope with small magnification. Then we find a place of fusing of two veins with uniform, not too fast blood-stream, and then a small crystal of **sodium chloride is placed near to the site of a vessel chosen earlier**. Observe the changes in the blood flow and the process of thrombosis for 10–40 min. Mark as the blood flow is gradually slowing down its course, a leukocyte-thrombocyte aggregate is being formed at the wall of a venous microvessel and subsequent loss of blood stream lamination occurs (Fig. 1).

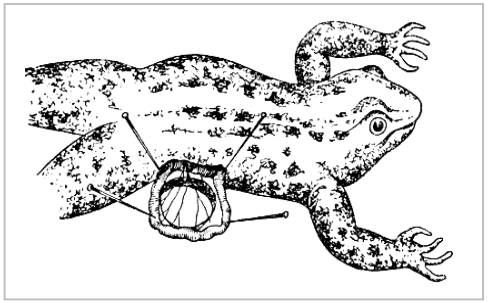


Fig. 1. Draw a mural blood clot in a mesentery vessel of the frog's intestines

Work 2. MODELING FATTY EMBOLISM OF THE FROG'S TONGUE VESSELS

An immobilized frog it is placed on a plate with its abdomen upwards. Open the thorax and expose the heart. A thin layer of cotton wool moistened with 0.65 % solution of sodium chloride is applied on the exposed heart. Turn over the frog on the plate and prepare a section of the tongue observing the blood circulation in its vessels. Then inject 0.1 ml of slightly warmed up liquid paraffin into the cavity of cardiac ventricle with a syringe. Quickly place the preparation of the tongue under the microscope. Observe the movement of emboli in the vascular lumen and the impairment of microcirculation. Similar changes can be observed in mesenteric vessels of the intestines and a swimming membrane of the frog (Fig. 2).	*Fig. 2.* Draw the fatty embolism of the frog's tongue vessels

Control questions

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 8).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.

5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.

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8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.

9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 9. PATHOPHYSIOLOGY OF REGIONAL BLOOD CIRCULATION AND MICROCIRCULATION. IMPAIRMENTS OF MICROCIRCULATION

Date: «____» _____ 20__

The purpose of the Lesson: to study the reasons, incidence conditions, developmental mechanisms, basic manifestations and consequences of typical impairments of microcirculation for the organism.

Tasks:

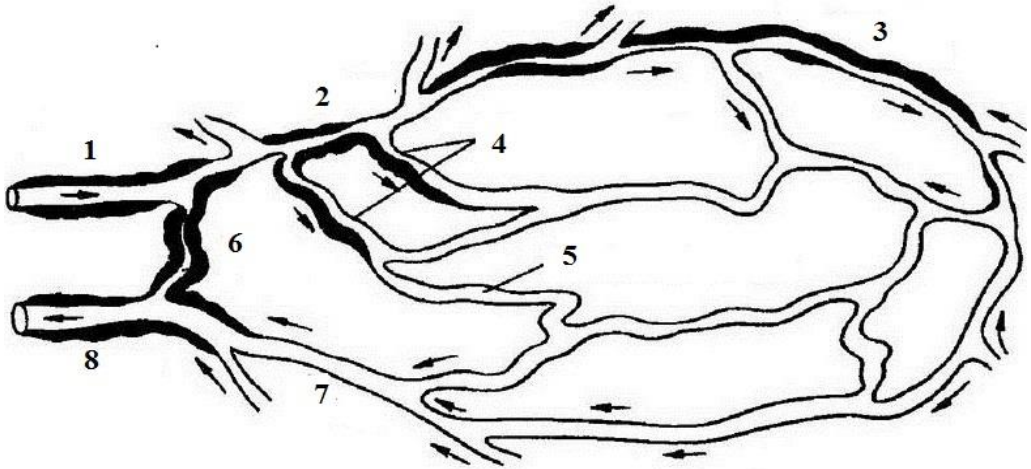
– to study the reasons, developmental mechanisms and consequences of typical impairments of the microcirculation presented in educational materials to the lesson;

– control test on the section "Typical impairments of peripheral blood circulation and microcirculation".

PART 1. WORK WITH TRAINING MATERIALS

1. Give the definition of ***"microcirculation"***:_____
_____

2. Identify the main components of the microcirculatory unit:



1 — _____
2 — _____
3 — _____
4 — _____
5 — _____
6 — _____
7 — _____
8 — _____

Schematic diagram of microcirculation (by B. W. Zweifach)

3. Fill in the Scheme:

CAUSES OF MICROCIRCULATION DISORDERS

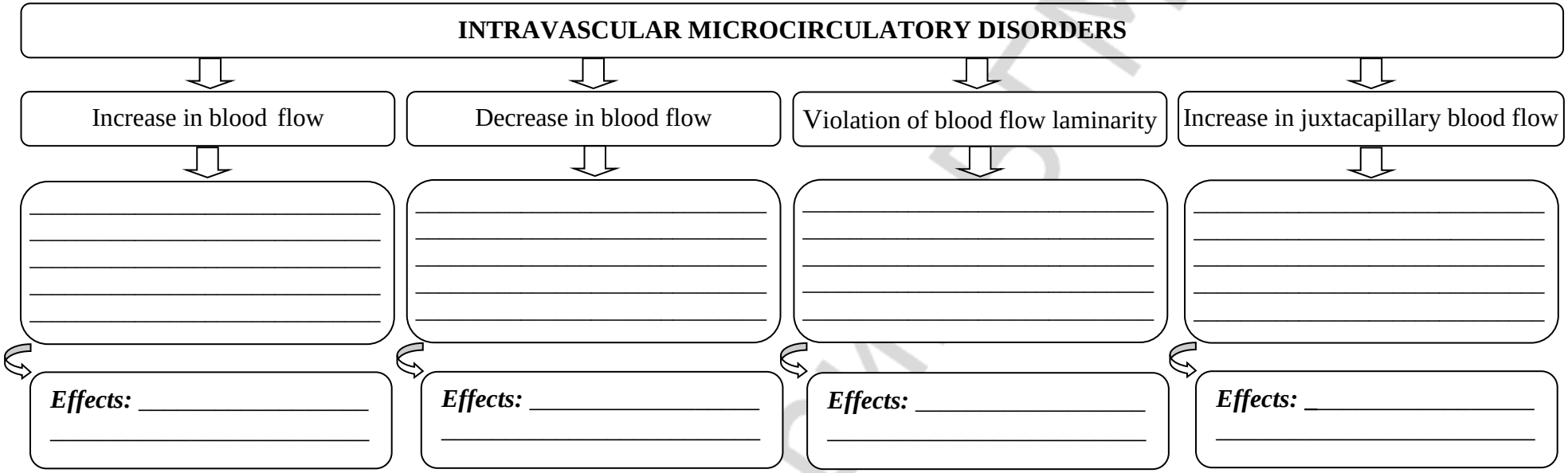
Disorder of central and peripheral circulation	*Changes in blood viscosity*	*Damage of blood vessels walls*
_____	*Increase due to:* _____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	*Decrease due to:* _____	_____
_____	_____	_____
	_____	
	_____	

4. Fill in the Scheme:

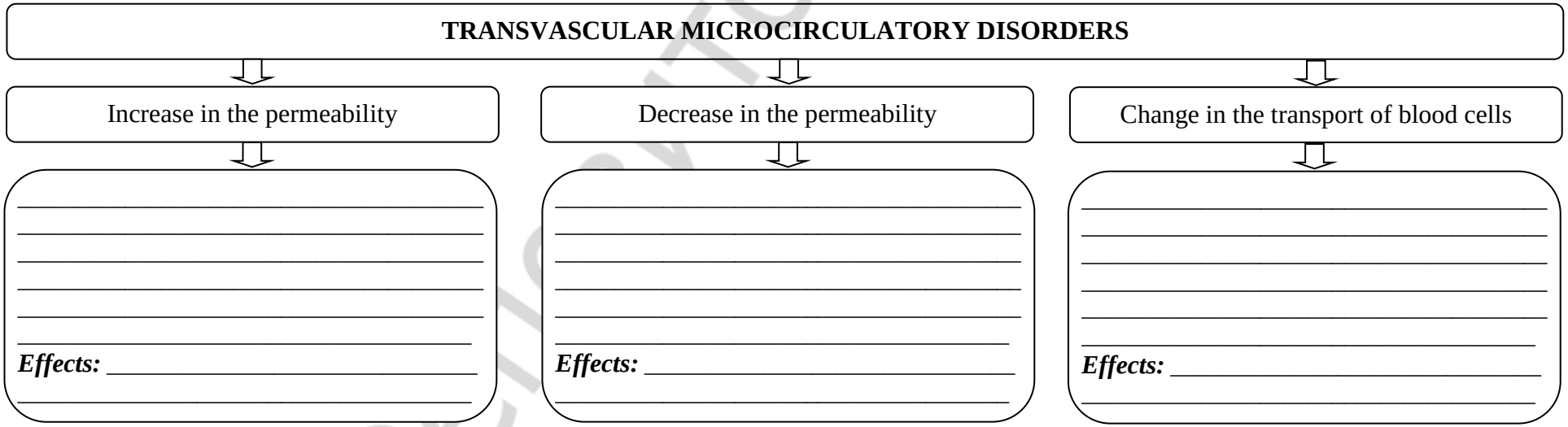
FORMS OF MICROCIRCULATION DISORDERS

By origin	By prevalence	By localization

5. Fill in the Scheme:



6. Fill in the Scheme:



7. Fill in the Scheme:

EXTRAVASCULAR MICROCIRCULATORY DISORDERS:

Increase of intercellular fluid volume

_____
_____
_____
_____
_____

Effects: _____

Decrease of intercellular fluid volume

_____
_____
_____
_____
_____

Effects: _____

8. Give the definition of the notion "***Lymphatic system insufficiency"*** _____
_____

9. Fill in the Scheme:

MECHANISMS OF THE LYMPHATIC SYSTEM INSUFFICIENCY

Mechanical circulatory failure

_____
_____
_____
_____
_____

Dynamic insufficiency

_____
_____
_____
_____
_____

Resorption deficiency

_____
_____
_____
_____
_____

Effects*: acute:* _____
chronic: _____

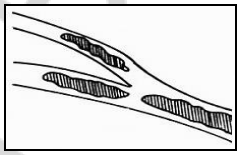
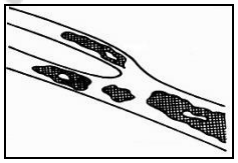
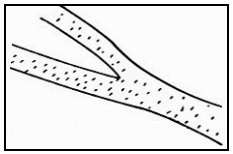
10. Fill in the Scheme:

Give the definition of ***sludge*** _____
_____
_____
_____
_____

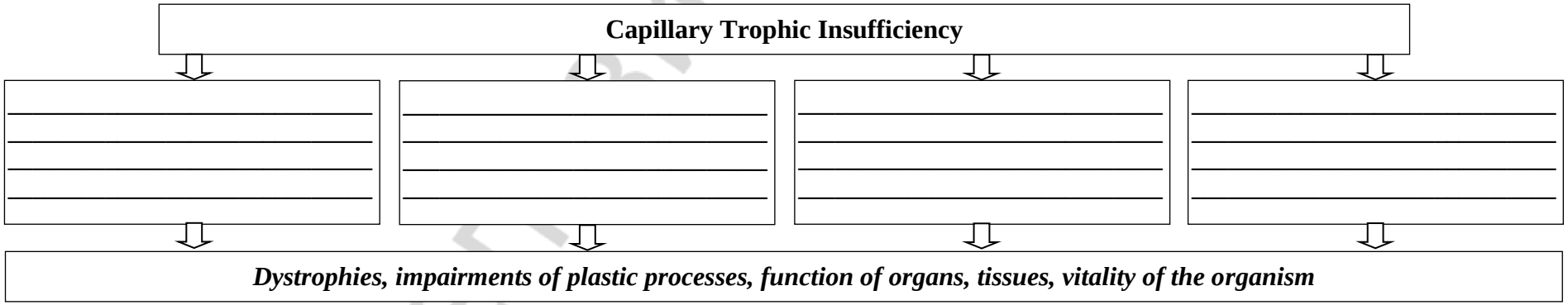
Pathogenesis of ***sludge*:** _____
_____
_____
_____
_____
_____
_____

The consequences of ***sludge*:** _____
_____
_____
_____
_____
_____
_____

Types of sludge

By reversibility	– _____ – _____	
Depending on the features of aggregates:	**Kind**	**Characteristic**
*1)*_____		_____ _____ _____ _____
*2)*_____		_____ _____ _____ _____
*3)*_____		_____ _____ _____ _____

11. Fill in the Scheme:



Control questions

1. Microcirculation. Definition of concepts: microcirculation, microcirculatory unit, their components.
2. Common causes of microcirculation disorders.
3. Forms of microcirculation disorders.
4. Intravascular microcirculation disorders. Reasons, mechanism, manifestations.
5. Transvascular microcirculation disorders. Reasons, mechanism, manifestations.
6. Extravascular microcirculation disorders. Reasons, mechanism, manifestations.
7. Typical disorders of lymph circulation. Types, manifestations.
8. The phenomenon of sludge. Reasons, mechanism, manifestations. Capillary trophic insufficiency.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 9).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
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The teacher's signature: _____

LESSON 10. PATHOPHYSIOLOGY OF THE CELL. CELL INJURY. GENERAL CELL INJURY MECHANISMS

Date: «____» _____ 20___

The purpose of the Lesson: to study the reasons and general mechanisms of damaging a cell. To characterize cell injury 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 injury, 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 set of slides "Cell injury", and also in the manual "Cell injury. Pathophysiological aspects";
– solving situational tasks;
– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Give the definition of "*Cell Injury*":_____
_____
_____
_____

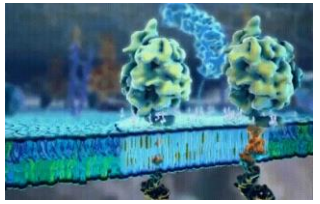
2. Fill in the Table:

Pathogenic cell injury variants

Feature	Violent	Cytopathic
The state of the cell at the time of exposure to factors		
Factors causing damage mechanisms		
Most sensitive cells		

3. Fill in the Table:

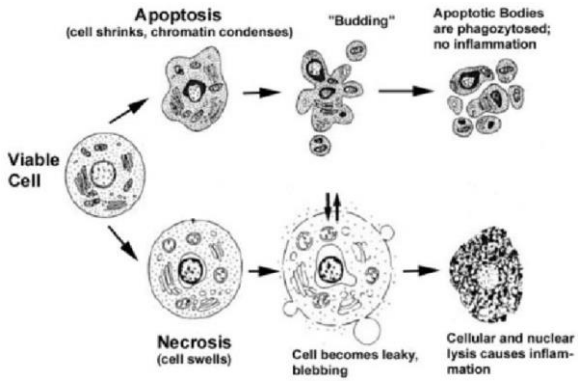
The general mechanisms of cell injury

Violation of the energy supply of cells	**Injury of cell membranes**	**An imbalance of ions and fluid**	**Violations of the genetic program**	**Disorders of intracellular processes regulation**
1) At the stage of ATP synthesis: _____ 2) At the stage of ATP transport: _____ 3) At the stage of ATP utilization: _____	*Important mechanism of cell membranes and enzymes damage:* _____ Pathogenic factors ↓ ↑ activity or hydrolase content in hyaloplasm ↓ ↓ accumulation of amphiphilic compounds ↓	*Change in membrane permeability for many ions* _____ *(list them)* ↓ **disbalance of iones** ↓ 1) _____ 2) violation of the excitation impulse ↓ **disbalance of fluid** ↓ cell volume change ↓ 1) _____ 2) _____ *(indicate the manifestations)*	*The main processes leading to a change in genetic information of the cell:* 1) _____ 2) _____ 3) _____ 4) _____	*Levels of regulatory mechanisms at which disorders may develop:* 1) _____ 2) _____ 3) _____ 

4. Fill in the Scheme:

The main manifestations of cell damage are:

Dystrophy	Dysplasia	Changes in the structure and functions of cellular organelles	Necrosis	Apoptosis
The mechanisms: 1. _____ _____ 2. _____ _____ 3. _____ _____ 4. _____ _____	The main reason: _____ _____ _____ _____ Mechanism: _____ _____ _____ _____ _____	Mitochondria: _____ _____ _____ Ribosomes: _____ _____ _____ Golgi Apparatus: _____ _____ _____ Endoplasmic reticulum: _____ _____ _____ Nucleus: _____ _____ _____ Cytoplasm: _____ _____ _____ _____	Definition: _____ _____ _____ _____ Stages: 1. _____ 2. _____ Cell destruction mechanism: _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____	Definition: _____ _____ _____ Stages: 1. _____ 2. _____ 3. _____ 4. _____ Cell destruction mechanism: _____ _____ _____ _____ _____ _____ _____ _____ _____

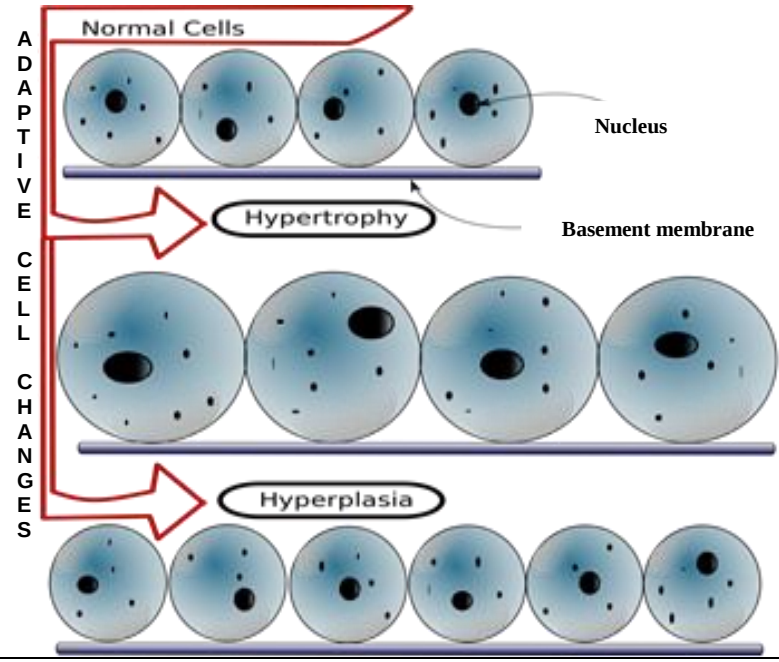


5. Specific changes in cell damage include: _____
_____
_____

6. Non-specific changes in cell damage include: _____
_____
_____

7. Fill in the Table:

The main mechanisms of cell compensation to injury

Intracellular		**Extracellular**	
Mechanisms	**Essence**	**Levels**	**Examples**
Compensation of cell energy supply violations	1) _____ 2) _____ 3) _____	Organ and tissue	_____ _____ _____
Protection of cell membranes and enzymes	1) _____ 2) _____ 3) _____	Intrasystem	_____ _____ _____
Compensation of imbalance of ions and fluid in a cell	1) _____ 2) _____	Intersystem	_____ _____
Compensation of violations in the genetic program of cells	1) _____		
Compensation of disorders caused by violation of cell regulatory functions	1) _____ 2) _____ 3) _____ 4) _____ 5) _____ 6) _____		
Decrease in functional activity of cells	1) _____ 2) _____ 3) _____ 4) _____		
Cell adaptations at the morphological level	- regeneration — _____ - hypertrophy — _____ - hyperplasia — _____ - hypotrophy — _____		

Control questions

1. The definition of the notion “cell injury”. Cell injury as a typical pathological process.
2. Principal causes and types of cell injury. Direct and indirect effect of cell damaging agents.
3. General mechanisms of cell injury.
4. The impairment of energetic supply of processes taking place in cells, as one of master mechanisms of injury.
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 of 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 injury, their mechanisms. Changes of the structure and functions of some cellular organelles at cell injury.
9. Specific and nonspecific manifestations in cell injury.
10. Intracellular mechanisms of adaptation and compensation in response to damage.
11. Integrated mechanisms of cellular injury and death (mechanisms of hypoxic necrobiosis and apoptosis).

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 10).

2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.

3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. Повреждение клетки (патофизиологические аспекты) = Cell injury (pathophysiological aspects): учеб.-метод. пособие / Е.В. Меленчук, С.А. Жадан, Ф.И. Висмонт. – Минск: БГМУ, 2016. – 23 с.

5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.

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The teacher’s signature: _____

LESSON 11. INFLAMMATION. VASCULAR REACTION IN THE FOCUS OF INFLAMMATION

Date: «____» _____ 20__

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

Tasks:

– to get acquainted with the reasons of occurrence and developmental mechanisms of the inflammatory process, impairments of peripheral blood circulation and microcirculation in inflammation on the basis of materials of education films;

– 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.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. *Inflammation* is _____
_____
_____
_____

2. The *basic local* signs of acute inflammation according to Cells-Halen are (explain their mechanisms of development):

1) _____
2) _____
3) _____
4) _____
5) _____

3. The *basic systemic* signs of acute inflammation are:

1) _____; 4) _____; 7) _____;
2) _____; 5) _____; 8) _____;
3) _____; 6) _____; 9) _____;

4. The basic stages (*components*) of inflammation are:

1) _____ 2) _____ 3) _____

5. Describe the main stages (components) of inflammation:

I. The alteration is_____

a) primary alteration — _____

_____

b) secondary alteration — _____

_____

✓ ***releasing of inflammatory mediators*** *(indicate the main of them and give the examples)*:

by chemical structure	➢ biogenic amines:
	➢ polypeptides:
	➢ proteins:
	➢ derivatives of PUFAs:
by origin	➢ cellular:
	➢ humoral:
by exit from the cells	➢ non-cytotoxic release:
	➢ cytotoxic release:
by velocity of their release from the cells	➢ immediate action:
	➢ slow action:
by character of action	➢ direct action:
	➢ indirect action:

Indicate:

– the metabolic changes in inflammation focus: _____

_____

_____

_____

– the physical and chemical changes at inflammation: _____

_____

_____

_____

– the structural and functional changes at inflammation: _____

_____

Basic effects of inflammation mediators

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

Inflammation mediator	Vascular permeability	Tone of smoothmuscular cells of vessels (↑↓)	Pain	Thrombosis	Emigration, chemotaxis of leukocytes	Opsonization	Bacteriocidity, secondary alteration	Stimulation of leukopoesis	Fever
Hystamine									
Serotonine									
Prostaglandins of group E									
Leukotrienes (LTC4, D1, E4)									
Prostacyclin (PGI2)									
Thromboxanes (TxA2)									
NO									
Lyzosomal enzymes									
Cytokines (IL-1β, TNF-α)									
Bradykinin									
Components of the complement system (C3a, C5a, C5, C9)									

II. Stage of plasma exudation and blood cells emigration:

Exudation is _____

⇨ *a) vascular reactions and changes in blood circulation in the focus of inflammation:*

- ✓ **spasm** (ischemia) — reason: _____
- ✓ **arterial hyperemia** — mechanism: _____
- ✓ **venous hyperemia** — mechanism: _____

1) blood factors (the role) — _____

2) blood vessels factors (the role) — _____

3) surrounding tissues factors (*the role)* — _____

- ✓ **stasis** — *mechanism*: _____

value: _____

⇨ *b) the actual exudation* — _____

(↑ GGB permeability → ↑ filtration process, ↑ microvesicular transport)

Result of exudation — _____

Kinds of exudates and their content:

1) fibrinous — _____

2) serous — _____

3) hemorrhagic — _____

4) purulent — _____

5) putrid — _____

6) mixed — _____

Biological sense of exudation: *1) adaptive*: _____

2) pathogenic: _____

Transudate is _____

Leukocyte emigration (leukodiapedes) is _____

_____

⇨ *1. Stage of marginal standing of leukocytes (margination) (phases):*

a) _____

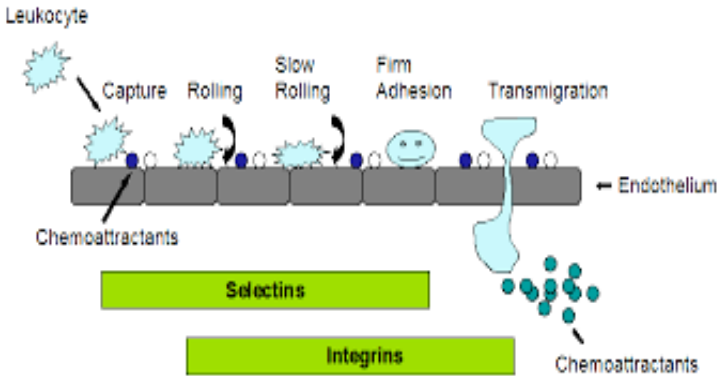
b) _____

⇨ *2. Leukocyte adhesion to endothelium occurs due to*: _____

_____

⇨ *3. Penetration of leukocytes through the vascular wall in the tissue (stages):*

- ✓ releasing of hydrolytic enzymes by leukocytes (for example, collagenases and elastases);
- ✓ hydrolysis of fibers and the main substance of a basal membrane;
- ✓ the passage of leukocytes through the basement membrane;
- ✓ exit of leukocytes from a vascular channel.



Indicate the ways of leukocyte emigration: (*granulocytes* — _____; *agranulocytes* — _____).

⇨ *4. Directional movement of leukocytes to the affected area is provided by: chemotaxis and electrotaxis.*

- ➢ ***chemotaxis*** is supplied by _____ — *chemotaxis-inducing substances*.

Types of chemoattractants:

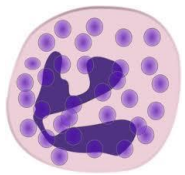
1) endogenous: _____

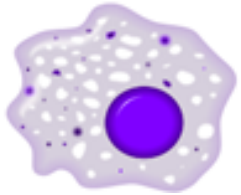
2) exogenous:_ _____

- ➢ ***electrotaxis*** is _____

The order of leukocytes emigration to the inflammation focus:

1) _____; ⟶ 2) _____; ⟶ 3) _____;





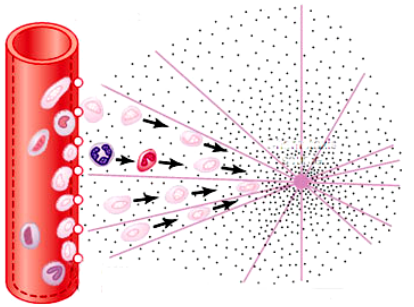


Fig. Leukocytes emigration to the inflammation focus

PART 2. PRACTICAL PART

Work 1. STUDYING VASCULAR REACTIONS AND LEUKOCYTES EMIGRATION IN INFLAMMATION OF THE FROG'S INTESTINAL MESENTERY (KONGAME'S EXPERIMENT)

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

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

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

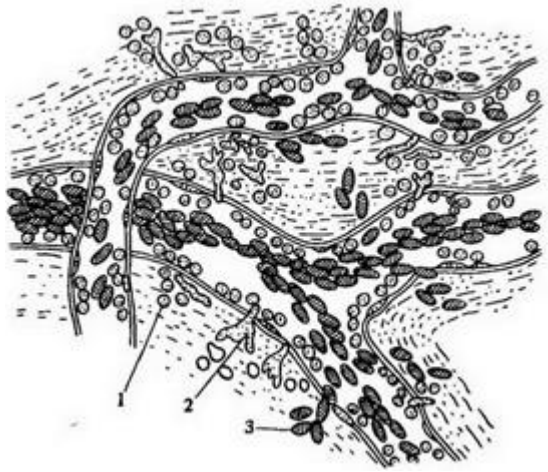


Fig. 1. The marginal standing of leukocytes in vessels

We sketch the observed vascular phenomena (hyperemia) and mural standing of leukocytes. Draw the marginal standing of leukocytes in vessels of the frog's intestinal mesentery at inflammation	Conclusions: 1. What factors causes the inflammation of the frog's intestinal mesentery in the given experiment? _____ _____ _____ _____ _____ 2. What factors provide adhesion and margination of leukocytes to a vessel wall in inflammation? _____ _____ _____ _____

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, significance in the development of secondary alteration.
5. Metabolic changes in the focus of inflammation.
6. Physical and chemical changes in the focus of inflammation, mechanisms of their development and significance.
7. Functional element of the organ as a substrate of alteration and formation of inflammatory reaction.
8. Impairment stages of peripheral blood circulation in the focus of inflammation and mechanisms of their development.
9. The reasons and mechanisms of increasing the permeability of a vascular wall in the focus of inflammation.
10. The definition, mechanism and significance of exudation in inflammation.
11. Types of exudates, their distinctions from transudate.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 11).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *Жадан, С. А.* Воспаление (патофизиологические аспекты) = Inflammation (pathophysiological aspects) : учеб.-метод. пособие / С. А. Жадан, Е. В. Меленчук, Ф. И. Висмонт. Минск : БГМУ, 2015. 35 с.
5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
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7. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.
8. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.
9. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
10. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 12. INFLAMMATION. PHAGOCYTE REACTION IN INFLAMMATION

Date: «____» _____ 20__

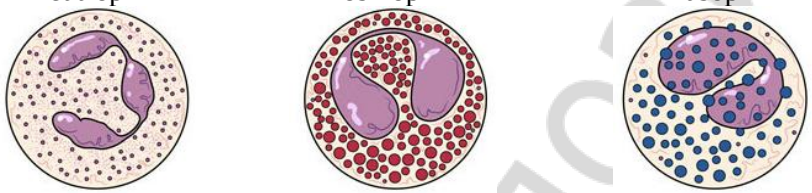
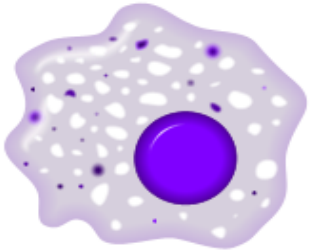
The purpose of the Lesson: to study phagocytosis as a protective reaction of the organism, to discuss phagocytosis stages during inflammation. To characterize the significance of inflammation as a response of the whole organism, to study the effect of the nervous system, hormonal and humoral factors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

Tasks:

– to get acquainted with the role of granulocytes for the development of a phagocyte (protective) reaction of the organism in inflammation on the basis of materials presented in the teaching video "The role of the granulocyte colony-stimulating factor (G-CSF) in regulating phagocytosis";
– to study phagocytosis stages of bird's erythrocytes by leukocytes of a guinea-pig on micropreparations;
– to study the role of superficial tension in the process of phagocytosis in Danilevsky's modeling experiment;
– solving situational tasks;
– control test of the topic "Inflammation".

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. **Phagocytosis** is_____
_____
_____

Microphages **Granulocytes** Neutrophil Eosinophil Basophil 	**Macrophages** **(blood monocytes → tissue macrophages)** 
Objects of phagocytosis: _____ _____	**Objects of phagocytosis: _____** _____

2. Phagocytosis phases (stages) are (give their characteristic):

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

a) endogenous: _____

b) exogenous: _____

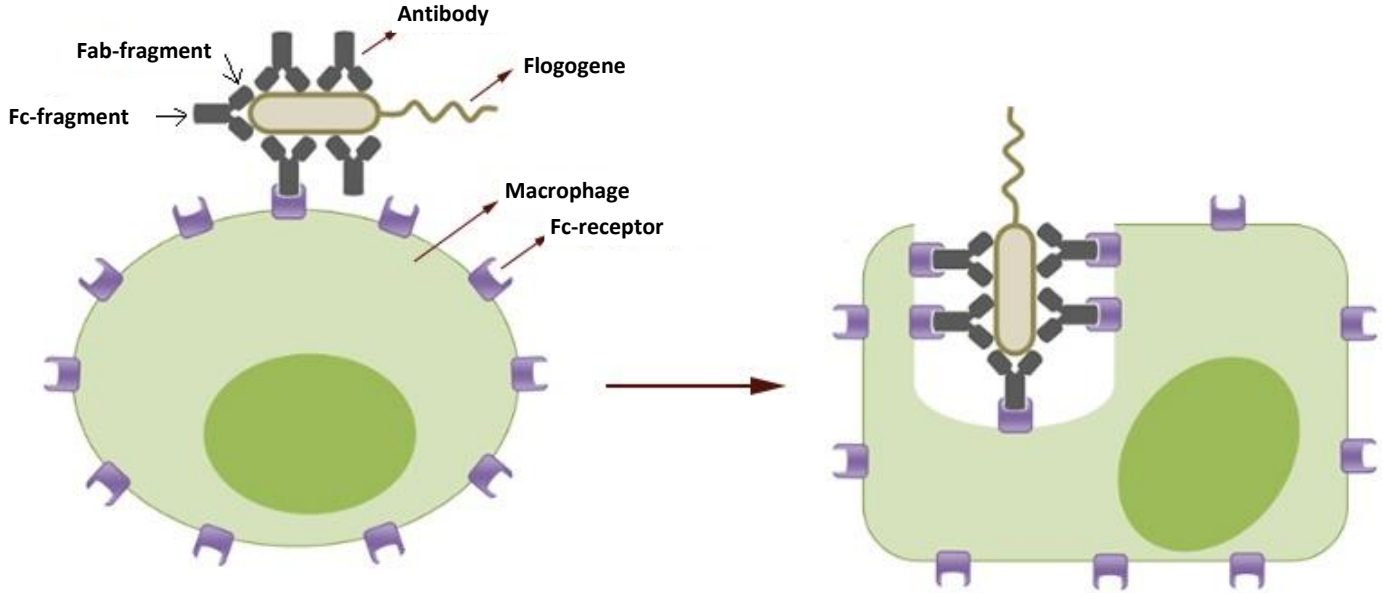
B) Phase of recognition and attachment of phagocytosis object:

a) non-immune mechanism: _____

b) immune mechanism: _____

Opsonization is _____

_____

Opsonins:	

C) Phase of engulfment with formation of a phagocytic vacuole: _____

_____

D) Phase of killing or degradation of the ingested material (digestion): _____

_____

3. **The proliferation stage of inflammation is** _____

_____

4. What are the functions of the main effectors cells at proliferation stage?

a) monocytes — _____

b) fibroblasts — _____

c) endotheliocytes — _____

5. List major factors causing bacteriocidity of phagocytes _____

_____

_____

6. Specify principal causes of incomplete phagocytosis:

a) _____

b) _____

c) _____

d) _____

7. 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			

8. Fill in the Table.

Inflammation theory	**The founder**	**The essence of the theory**

PART 2. PRACTICAL PART

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 ºC (erythrocytes, containing a nucleus, serve as an object of phagocytosis).

In 15 min about 1.0 ml of exudate with bird's erythrocytes is taken out by a syringe from the abdominal cavity of a guinea-pig and smear cultures are prepared. Then, every 15–20 min after the first sample the second and a third samples of exudates are taken and smear cultures are prepared too. The smear cultures are stained according to Romanowsky–Giemsa and then they are investigated under the microscope.

Draw the phagocytosis stages:

Phase of approaching	*Phase of recognition and adherence*	*Phase of ingulfment*	*Phase of digestion*

Conclusion:

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

_____

_____

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

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 12).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
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8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 13. PATHOLOGY OF THERMAL REGULATION. FEVER

Date: «____» _____ 20__

The purpose of the Lesson: to study the incidence reasons, development mechanisms and general biological significance of fever.

Tasks:
- to study the state of heat exchange processes in the development of feverish reaction in rabbits after injection of bacterial endotoxin;
- to study the character of thermoregulatory reactions of rabbits with endotoxic fever under overheating;
- to construct the most typical temperature curves in various kinds of fever;
- solving situational tasks;
- control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Put down the definition of the notion "*fever"* — _____
_____
_____
_____

2. Put down the definition of the notion "*pyrogen*" — _____
_____
_____

3. Fill in the Table:

Classification of pyrogens

Primary *(etiological)*			**Secondary** *(pathogenic)*
Exogenous		**Endogenous**	
Infectious	**Non-Infectious**		
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
			_____

4. Give the characteristic of the stages of fever:

I stage (HP HL)
_____(_____)
(give the name)

↑ HP – mechanisms:
1) _____
2) _____

↓ HL – mechanisms:
1) _____
2) _____
3) _____

II stage (HP HL)
_____(_____)
(*give the name)*

HP – mechanisms:
1) _____
2) _____
3) _____

HL – mechanisms:
1) _____
2) _____
3) _____

III stage (HP HL)
_____(_____)
(give the name)

↓ HP – mechanisms:
The reason of temperature reducing:
_____
_____

↑ HL – mechanisms:
1)_____
2)_____
3)_____

5. Заполните таблицу:

Metabolic and organ function changes at fever

Protein exchange	_____ _____ _____
Carbohydrate and fat metabolism	_____ _____ _____
Water-electrolyte exchange	1st stage: _____ _____ 2nd stage: _____ _____ 3d stage: _____

The cardiovascular system	Heart rate: _____ _____ Heart rhythm: _____ _____ Vascular tone: _____ 1st stage: _____ 2nd stage: _____ 3d stage:
Respiratory system	_____ _____
Digestive system	_____ _____
Nervous system	_____ _____ _____
Endocrine system	_____ _____ _____

6. What factors determine the level of body temperature rising in a febrile state?

_____
_____
_____

7. What is the protective and adaptive value of fever?

_____
_____
_____

8. What is the pathogenic significance of fever?

_____
_____
_____

9. Fill in the Table:

Endogenous hyperthermia

Mechanism	**Centrogenic**	**Psychogenic**	**Reflexogenic**
Cause	_____ _____ _____ _____	_____ _____ _____ _____	_____ _____ _____ _____
Mechanism	_____ _____ _____ _____	_____ _____ _____ _____	_____ _____ _____ _____
Examples	_____ _____ _____ _____	_____ _____ _____ _____	_____ _____ _____ _____

10. Indicate the scope of pyrotherapy: _____
_____
_____
_____
_____
_____

PART 2. PRACTICAL PART

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.

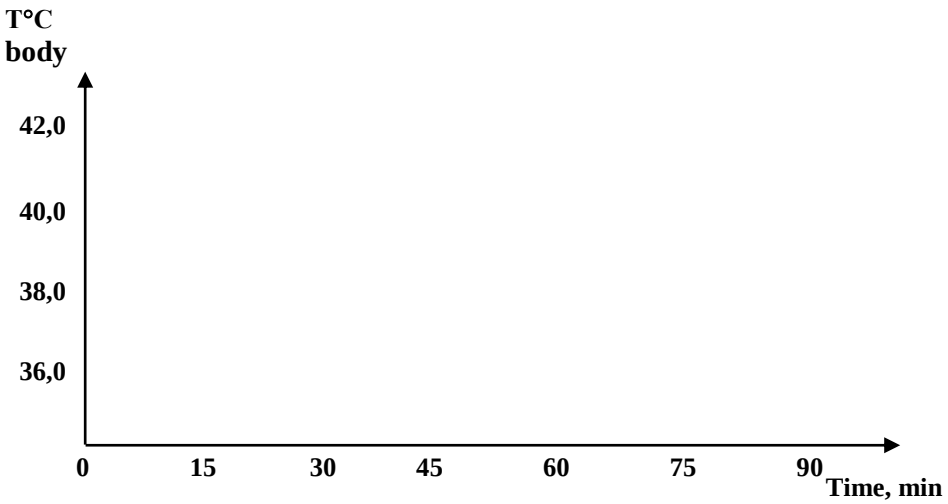
To induce experimental fever, we use endotoxin — bacterial liposaccharid pyrogenal.

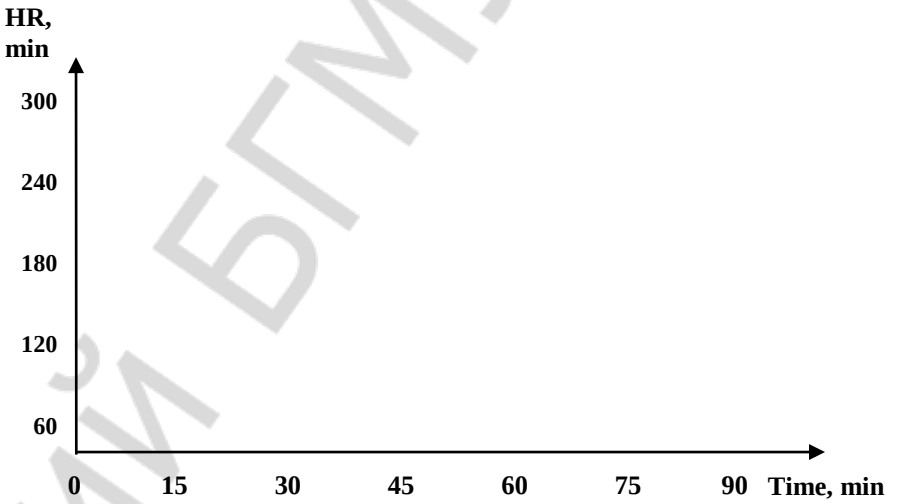
The first rabbit (tested) is injected pyrogenal (0.5 mcg/kg) in 0.5 ml of physiological solution **into the marginal vein of the ear**, and **the second (control) — 0.5 ml of physiological solution**. Then observe the condition and behavior of the animals. In every 15 min after injections take rectal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

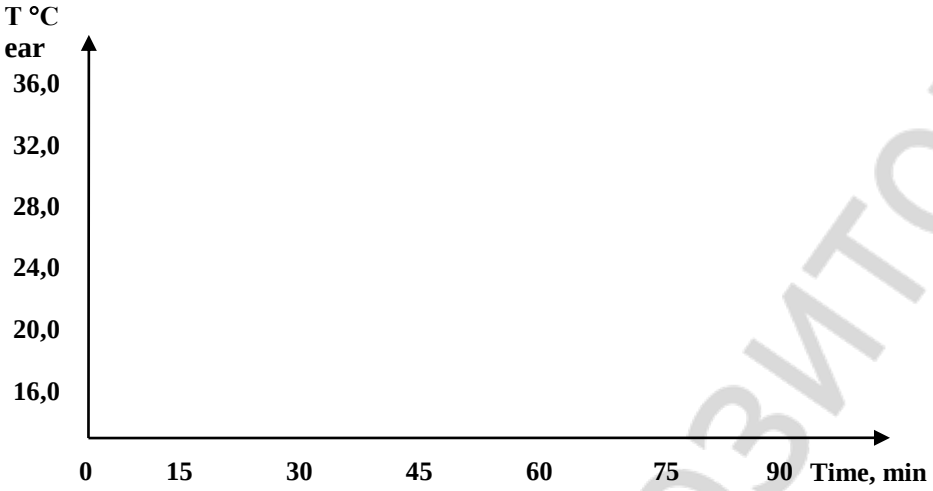
Experiment Results

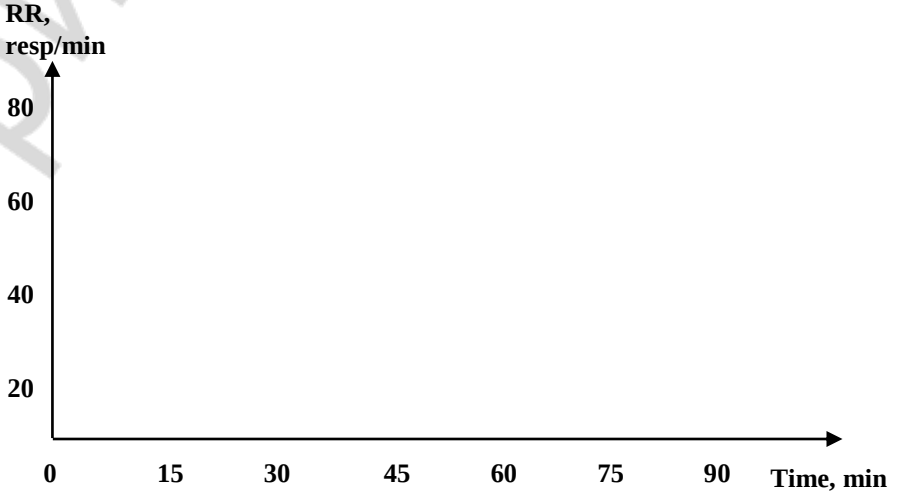
№	Group of animals. Time since the beginning of the experiment		Temperature, °C		Respiratory rate (resp/min)	Heart rate (beats/min)	Notes
			rectal	ear skin			
1.	***Tested*:**	0 min	38.8	33	60	220	
		– 15 min	39.2	24.0	72	260	
		– 30 min	39.6	24.0	30	270	
		– 45 min	39.9	27.0	46	280	Ears are pale, cold, vessels are narrowed
		– 60 min	40.2	28.0	58	280	
		– 75 min	40.4	28.0	60	290	
		– 90 min	40.4	30.0	70	280	
2.	***Control*:**	0 min	39.2	31.0	68	220	
		– 15 min	39.2	30.0	70	242	
		– 30 min	39.0	30.0	72	236	Ears are pink, warm, vessels are moderately dilated
		– 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	

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.









Make conclusions, answer the following questions:

1. Explain the reasons for lowering the temperature of auricle, lowering RR and increasing HR in an experienced rabbit: _____

_____

2. What are the possible mechanisms of the next changes on the first stage of pyrogenal-induced fever?

– increasing heat production_____

– reduction of heat emission_____

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

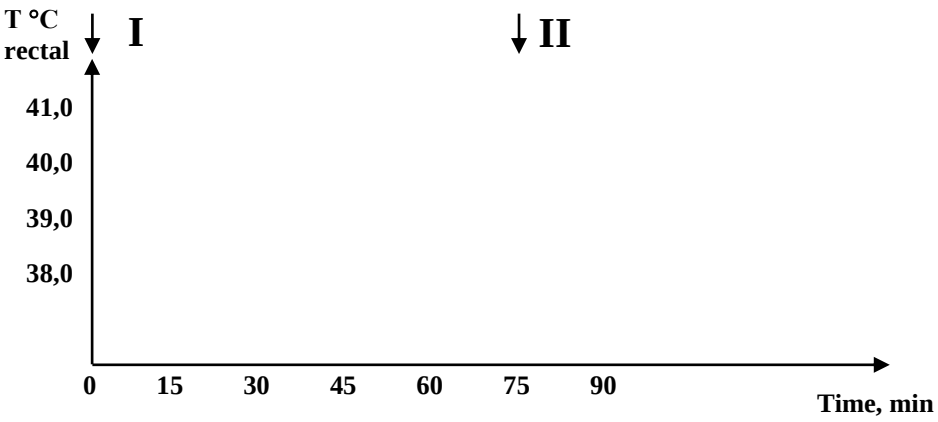
The experiment is performed on two adult rabbits of one sex with body weight of 2.0–2.5 kg. **One (tested) is injected pyrogenal (0.5 mcg/kg) on 0.5 ml of physiological solution into the marginal vein of the eat, to the other (control) — 0.5 ml of physiological solution**. Immediately after injections the animals are placed in **the thermochamber with dry air** and overheating at temperature of air **40–42 °C** is performed. Thermometry, as well as registration of respiratory rate and heart beat are performed every 15 min within one hour, according to a technique described in work 1. Then the animals are taken from thermochambers and measurement of body temperature, respiratory rate and heart rate are continued everyone 15 min during their stay in **thermo-neutral conditions (20–21 °C)**.

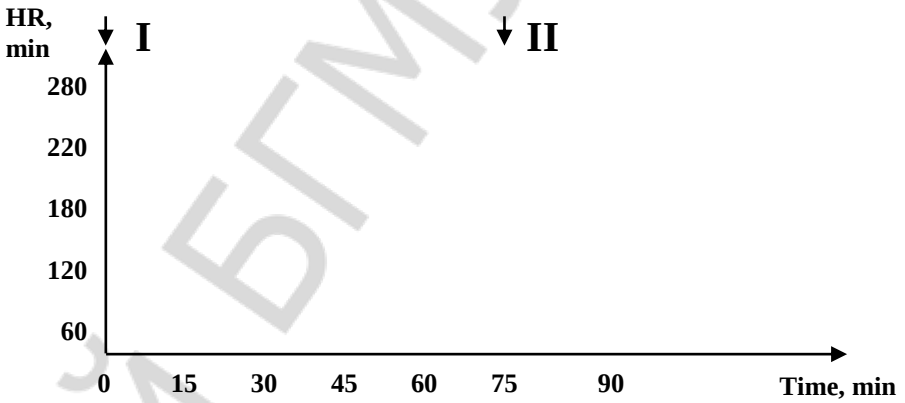
The data received are recorded into the table.

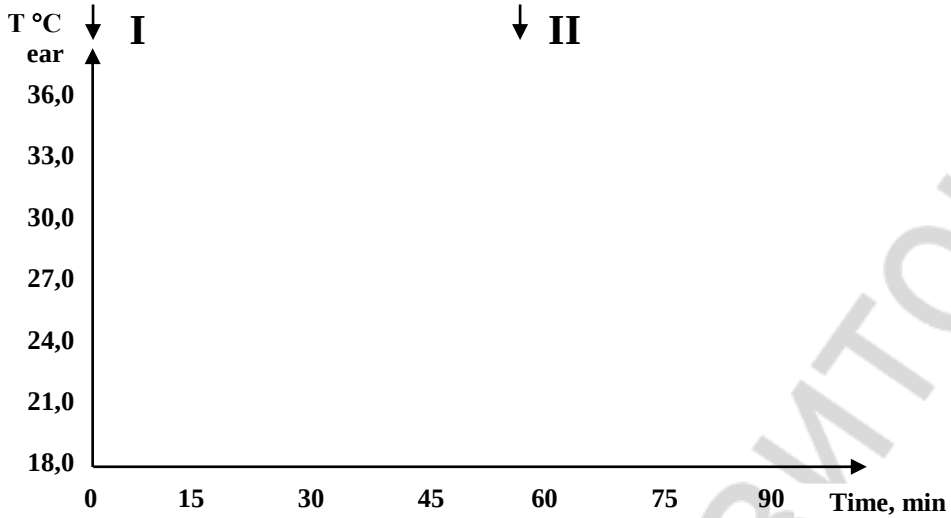
Experiment Results

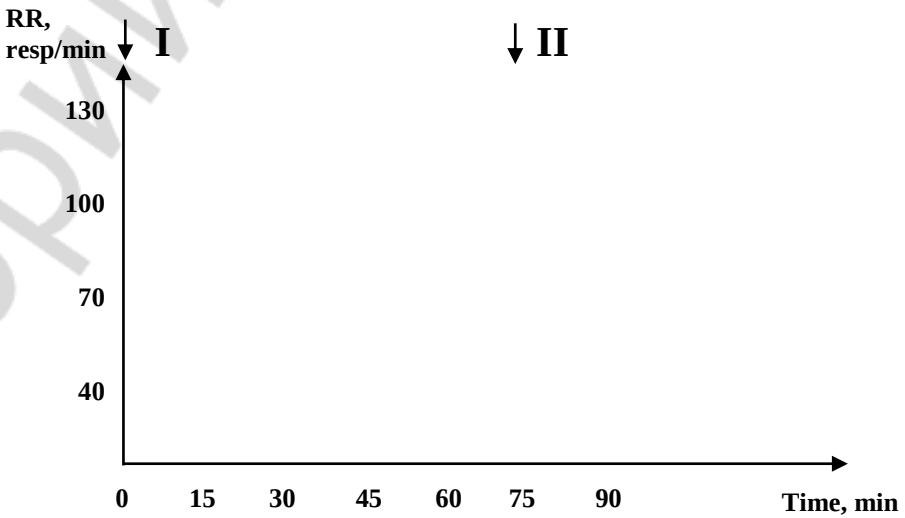
№	Group of animals. Time since the beginning of the experiment	Temperature, °C		Respiratory rate (resp/min)	Heart rate (bpm)
		rectal	ear skin		
1.	***Tested*:** 0 min	38.8	33.0	62	220
	– 15 min	39.0	31.0	68	220
	– 30 min	39.2	26.0	78	242
	– 45 min	39.6	28.0	48	260
	– 60 min	40.0	32.0	92	272
	Placing 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*:** 0 мин	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

Make conclusions, answer the following 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 over-heating? _____

_____

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

1. Indicate the Latin name of the fever depending on fluctuations in daily temperatures:

Constant → _____
Relapsing → _____
Intermittent → _____
Exhausting (hectic) → _____
Remittent → _____
Atypical → _____
Perverted → _____

2. Give a classification of fevers by the degree of temperature rise:

Kind of the fever	Deviations of the temperature

3. Draw the temperature curves for the indicated types of fevers: * m — morning, e — evening:

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

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

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

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

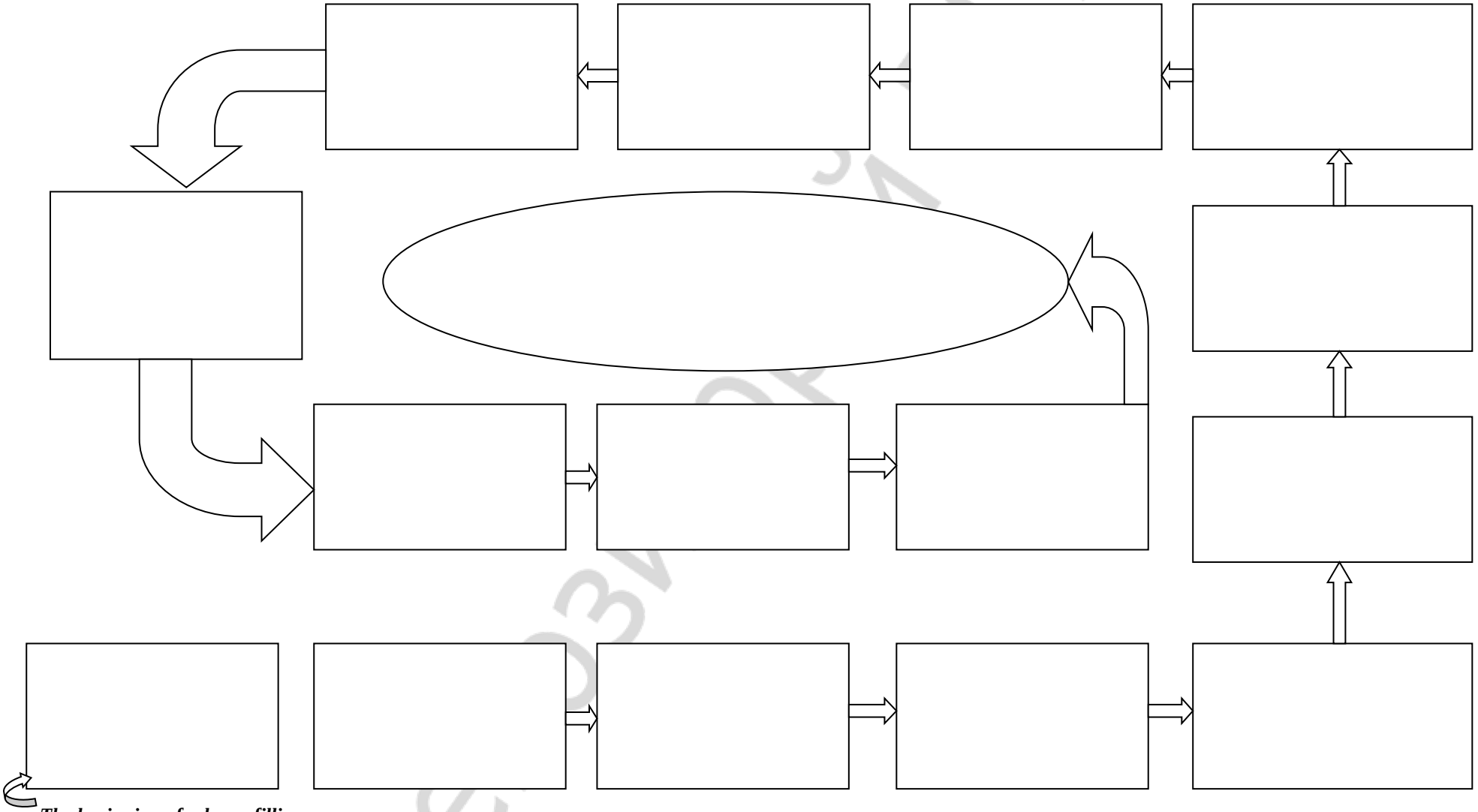
Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																					Remittent		
40																							
39																							
38																							
37									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

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

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

Work 4. PATHOGENESIS OF FEVER

Fill out the scheme:



The beginning of scheme filling

Control questions

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 13).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

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5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.
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7. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.
8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.
9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher’s signature: _____

LESSON 14. TYPICAL VIOLAYIONS OF METABOLISM. ACID-BASE BALANCE IMPAIRMENTS

Date: «____» _____ 20__

The purpose of the Lesson: to study typical forms of acid-base impairments of the internal environment of the organism, their kinds, reasons, development mechanisms, manifestations and compensatory mechanisms, basic laboratory parameters, correction principles of the acid-base balance.

Tasks:

– to get acquainted with laboratory parameters of the acid-base balance;

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

– solving situational tasks;

– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Define the concept of *"acid-base balance"* — _____
_____
_____

2. Formula for calculation pH:

pH=

3. Fill in the Table:

pH shift compared to physiological norm by:	Consequences for the body
± 0.1	
± 0.3	
± 0.4	

4. What products (acidic or alkaline) are formed more during the life of the organism (how many times)? _____
_____
_____

5. Due to what compensation mechanisms does the body maintain a normal pH value? _____
_____
_____

6. Fill in the table, indicating normal ABB parameters (arterial blood).

ABB indicators in a norm

Blood parameters	Value
pH	
p_aCO_2	
HCO_3^-	
SB (standard bicarbonate)	
BB (buffer bases)	
BE (excess / deficiency of buffer bases)	
Lactic Acid (Lactate)	0.5–2.2 mmol/l
Ketone bodies	0.43–1.033 mmol/l
Blood plasma electrolytes (mmol/L)	
Na^+	135–145
K^+	3.5–5.0
Ca^{2+}	2.23–2.57
Cl^-	96–108
Additional ABB indicators	
Titratable acidity (TK) of daily urine	20–40 mmol/l
Ammonia daily urine	20–50 mmol/l
Urine pH	4.5–8.0

7. Fill in the table, indicating the **urgent** compensation mechanisms:

Urgent compensation mechanisms

Chemical buffer systems	The essence of the compensatory system
1)	
2)	
3)	
4)	

8. Fill in the table with long-term compensation mechanisms.

Long-term (physiological) compensation mechanisms

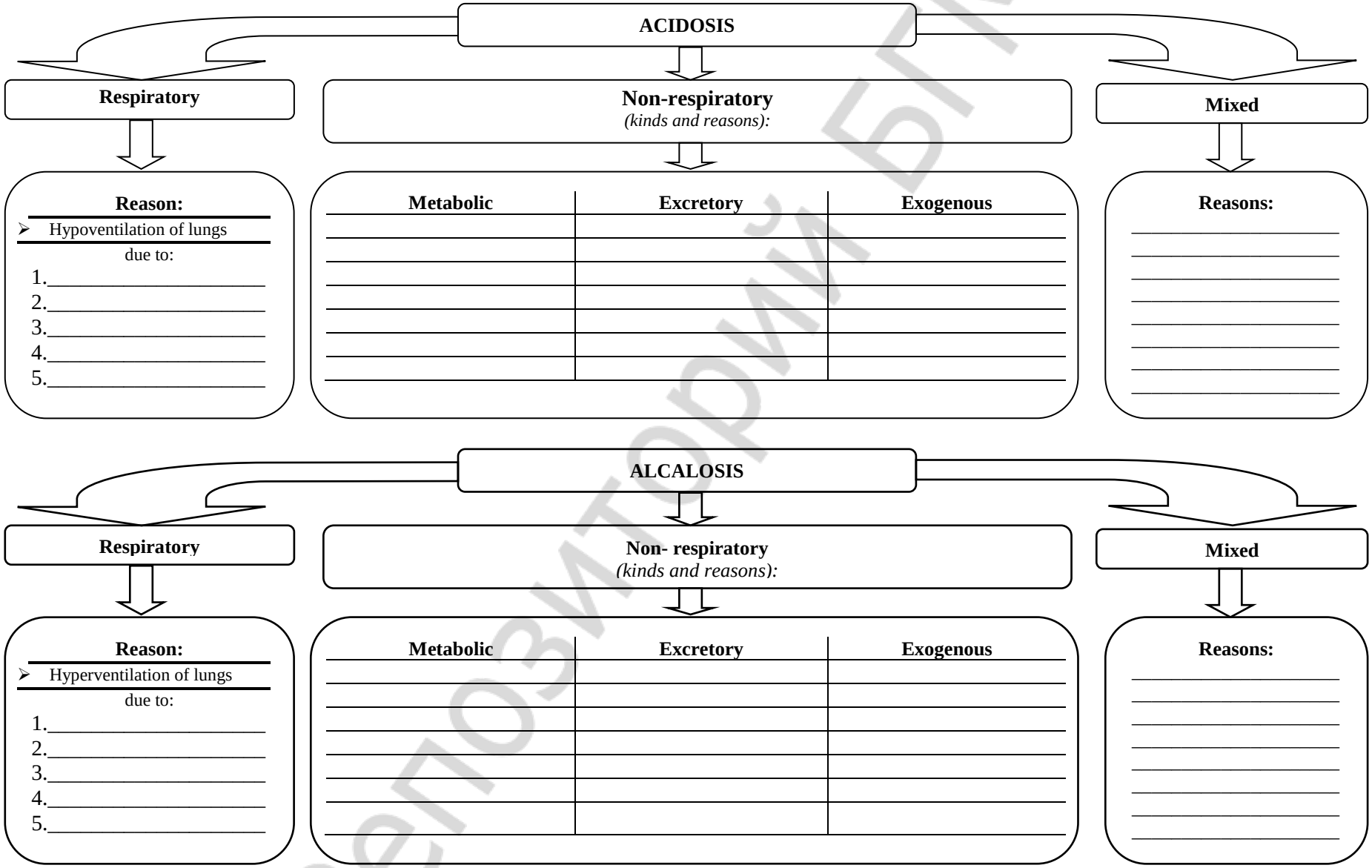
Physiological components	The essence of the compensatory system
1) Lungs	
2) Kidneys	
3) Liver	
4) Stomach	
5) Bowels	
6) Pancreas	
7) Bones	
8) Skin	

9. Fill in the Tables:

ABB disorders classification

Criterion	Kinds
According pH	1) _____ is _____ 2) is
According reason	1) 2)
According compensation degree	1) _____ pH _____ 2) _____ pH _____ 3) pH
By mechanism of development	1)_____ 2) non-respiratory: 1) _____; 2) _____; 3) _____ 3)
By course	1) 2)
By primary of violation	1) 2)

10. Fill in the schemes:



11. Fill in the table.

Changes in respiratory and metabolic components at typical ABB disorders

Type of ABB violations		Primary violation	Expected Compensation
1. Respiratory acidosis	acute		
	chronic		
2. Non-Respiratory acidosis	acute		
	chronic		
3. Respiratory alcalosis	acute		
	chronic		
4. Non-Respiratory alcalosis	acute		
	chronic		

PART 2. PRACTICAL PART

Situational Tasks

№ 1

A group of tourists from the middle region of the European part of the CIS is delivered by air to a tourist camp on Pamir, 2500 m over the sea level. Several persons began complaining of tiredness, weakness, early fatigue. While examining one of those on the 2nd day of staying in the camp the following parameters of the acid-base state were revealed.

Parameters	2nd day	Indicator characteristic	In a week	Indicator characteristic
$pH_{\text{arterial blood}}$	7.46		7.41	
p_aCO_2	32 mm Hg		30 mm Hg	
HCO_3^-	22 mmol/l		17 mmol/l	
BE	–1 mmol/l		–6 mmol/l	
TK urine	20		0	
pH urine	6.0		7.2	
Bicarbonates in the urine	–		+	

1. Make a conclusion about ABB violation on the 2nd day of stay: _____

2. Give a conclusion on the nature of ABB violations in a week: _____

№ 2

The patient of 56 years suffers from pulmonary emphysema and respiratory insufficiency:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.37	
p_aCO_2	56 mm Hg	
HCO_3^-	32 mmol/l	
BE	+7.5 mmol/l	
Na^+	142 mmol/l	
K^+	88 mmol/l	
Cl^-	7.37	

Make a conclusion about ABB violation:

№ 3

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	6.95	
p_aCO_2	20 mm Hg	
HCO_3^-	5.5 mmol/l	
BE	–20 mmol/l	
SB	4 mmol/l	
Ketone bodies in blood plasma	10 mmol/l	
K^+	7.5 mmol/l	
Ketone bodies in urine		
TK urine	60 mmol/l	

Make a conclusion about ABB violation:

What are the possible approaches to the correction of ABB in this patient?

№ 4

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.27	
p_aCO_2	27 mm Hg	
HCO_3^-	15.5 mmol/l	
BE	–10 mmol/l	
SB	15 mmol/l	
Concentration of trace anions in plasma	15 mmol/l	
K^+	5.8 mmol/l	

Make a conclusion about ABB violation:

№ 5

The patient was admitted to the first aid hospital in the condition of asphyxia. The blood test revealed:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.0	
p_aCO_2	80 mm Hg	
HCO_3^-	19 mmol/l	
BE	–8 mmol/l	
SB	18 mmol/l	
BB	37 mmol/l	
Lactate	4.5 mmol/l	

Make a conclusion about ABB violation:

№ 6

The patient was admitted to clinic in a severe condition. Extensive infarction of anterior lateral walls of the left ventricle, acute left-ventricular cardiac insufficiency and pulmonary edema was diagnosed. While estimating ABB parameters the following data were received:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.22	
p_aCO_2	55 mm Hg	
HCO_3^-	20 mmol/l	
BE	–5 mmol/l	
Lactate	4.76 mmol/l	

Make a conclusion about ABB violation:

№ 7

Patient M., 37 years, was delivered to the intensive care department with acute poisoning with sleeping draughts:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.29	
p_aCO_2	56 mm Hg	
HCO_3^-	25 mmol/l	
BE	+1 mmol/l	

Make a conclusion about ABB violation:

Is there a need for the appointment of sodium bicarbonate in this case to correct the disturbed acid-base state?

№ 8

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.26	
p_aCO_2	28 mm Hg	
HCO_3^-	5.5 mmol/l	
BE	–20 mmol/l	
SB	14 mmol/l	
Lactate	6.8 mmol/l	

Make a conclusion about ABB violation:

№ 9

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.15	
p_aCO_2	25 mm Hg	
HCO_3^-	12 mmol/l	
BE	–20 mmol/l	
SB	15 mmol/l	
Lactate	6.2 mmol/l	
Ketonic bodies in blood plasma	3.7 mmol/l	
Potassium	6.5 mmol/l	
Concentration of trace anions in plasma	26 mmol/l	
Reduced content of K^+ in RBC		

Make a conclusion about ABB violation:

№ 10

Patient B., 13 years, with acute poliomyelitis on the 4th day of the disease noted the difficulty of respiration, due to which he was administered artificial pulmonary ventilation (APV). Investigation results of ABB are presented in the table:

Parameters	Value	Characteristics	In a week	Characteristics
$pH_{\text{arterial blood}}$	7.26		7.46	
p_aCO_2	62 mm Hg		30 mm Hg	
HCO_3^-	26 mmol/l		18 mmol/l	
BB	43 mmol/l		40 mmol/l	
SB	22 mmol/l		20 mmol/l	
BE	1 mmol/l		–2.2 mmol/l	

1. What form of ABB disturbance took place in a child before artificial ventilation of the lungs: _____

2. Give a conclusion on the nature of the ABB violation 2 hours after artificial ventilation of the lungs: _____

3. Is the volume of pulmonary ventilation set correctly during ventilation? _____

№ 11

Patient Z., 16 years, was admitted to clinic with acute pneumonia. The condition is heavy. The body temperature is 39.8 °C. Expressed breathlessness. The anamnesis revealed no pulmonary pathology. The investigation of ABB parameters revealed:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.47	
p_aCO_2	29 mm Hg	
HCO_3^-	22 mmol/l	
BE	–1.8 mmol/l	

Make a conclusion about ABB violation:

What is the reason?

№ 12

Child D., 4 years, was delivered to hospital due to elevation of the body temperature and frequent loose stool (8–10 times a day). On examination moderate dehydration and breathlessness were noted. The investigation of ABB parameters revealed:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.39	
p_aCO_2	27 mm Hg	
HCO_3^-	17 mmol/l	
BE	–8 mmol/l	

Make a conclusion about ABB violation:

What is the reason?

№ 13

Patient K., 38 years, is delivered to hospital with an attack of titanic spasms. Questioning of the patient revealed that about half a year ago he got into a car accident. He has received an open fracture of the right humeral bone. Fracture knitting occurred in usual terms. But since then he had been suffering from strong heartburn and to relive it he constantly takes baking soda.

The investigation of ABB parameters revealed:

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.5	
p_aCO_2	43 mm Hg	
HCO_3^-	32 mmol/l	
BE	+12 mmol/l	

Make a conclusion about ABB violation:

What is the direct cause of the violation of the acid-base balance in this case?

Can these changes in ABB lead to development of tetany, if so, how? _____

_____

_____

№ 14

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.29	
p_aCO_2	30 mm Hg	
HCO_3^-	18 mmol/l	
BE	–11 mmol/l	

Make a conclusion about ABB violation:

What is the probable cause of the ABB violation in this case?

How to explain the decrease in pCO_2?

№ 15

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

Parameters	Value	Characteristics
$pH_{\text{arterial blood}}$	7.55	
p_aCO_2	60 mm Hg	
HCO_3^-	50 mmol/l	
BE	18 mmol/l	

Make a conclusion about ABB violation:

What is the possible cause of ABB disturbance in this patient?

№ 16

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

Parameters	Before treatment	Characteristics	On the 2nd day	Characteristics	On the 3rd	Characteristics
$pH_{\text{arterial blood}}$	7.28		7.34		7.44	
p_aCO_2	20 mm Hg		36 mm Hg		49 mm Hg	
BB	31 mmol/l		39 mmol/l		51 mmol/l	
HCO_3^-	12 mmol/l		18 mmol/l		29 mmol/l	
BE	–18 mmol/l		–9 mmol/l		6 mmol/l	

1. Indicate the type of ABB violations:

a) before treatment: _____

b) on the 2nd day: _____

c) on the 3rd day:_____

2. Is there a need for further administration of diseased sodium bicarbonate?_____

Control questions

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 14).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
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The teacher's signature: _____

LESSON 15. TYPICAL VIOLATIONS OF METABOLISM. PATHOLOGICAL PHYSIOLOGY OF WATER METABOLISM. EDEMA AND DROPSY

Date: «____» _____ 20__

The purpose of the Lesson: to study the reasons and development mechanisms of water balance impairment in the organism, pathogenesis of cardiac, renal, toxic, inflammatory, cachectic, allergic and other kinds of edema and dropsy.

Tasks:

– studying developmental mechanisms of pulmonary edema in experimental acute cardiac insufficiency induced by injection of adrenaline;
– studying developmental mechanisms of a toxic pulmonary edema in experiment, when the central nervous system plays pathogenic role;
– solving situational tasks;
– control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Typical forms of impaired water metabolism include: 1) _____; 2) _____

2. Fill in the Table:

Hypohydration is _____		
Kinds	**Reasons**	**Manifestations**
Isoosmolar	- _____ - _____ - _____ - _____ -	- _____ - _____ - _____ - _____ -
Hypoosmolar	- _____ - _____ - _____ - _____ - _____	- _____ - _____ - _____ - _____ - _____ -

Hyperosmolar	- _____ - _____ - _____ - _____ - _____	- _____ - _____ - _____ - _____ - _____ -

Hyperhydration is _____

Kinds	**Reasons**	**Manifestations**
Isoosmolar	- _____ - _____ - _____ - _____ - _____	- _____ - _____ - _____ - _____ - _____
Hypoosmolar	- _____ - _____ - _____ - _____ - _____	- _____ - _____ - _____ - _____ - _____ -
Hyperosmolar	- _____ - _____ - _____ - _____ - _____	- _____ - _____ - _____ - _____ - _____ - _____ - _____

3. Define the concept of "*edema*": _____
_____

4. Put down the classification of edema:

Criteria	Kinds of edemas	Definition	
By localization	anasarca		
	dropsy		
	ascites		
	hydrothorax		
	hydropericardium		
	hydrocele		
	hydrocephalus		
		Reason	**Mechanism**
By pathogenesis	hydrodynamic		
	lymphogenous		
	oncotic		
	osmotic		
	membranogenic		

		Reason	Mechanism
By etiology	cardial		
	renal		
	endocrine		
	cachexic		
	inflammatory		
	allergic		
	toxic		
Characteristic			
By rate of development	fulminant		
	acute		
	chronic		
Characteristic			
By spreading	local		
	general		

PART 2. PRACTICAL WORK

Work 1. ADRENALINE PULMONARY EDEMA IN A RAT

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

Results of the experiment.

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

Type of effect	Respiratory rate (resp/min)	General state	Pathomorphological changes in lungs
i/p injection of 0.l % Adrenaline solution – Initial – 1 min – 2 min – 3 min – 4 min	 120 160 Rare deep respiration Terminal respiration Respiratory arrest	 Normal General excitation, impairment of motor coordination Foamy discharge from the mouth – // – // – // – Death of the animal	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
i/p injection of 0.9 % solution of NaCl – Initial – 1 min – 2 min – 4 min	 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

Conclusion:

Explain the development mechanism of adrenaline-induced pulmonary edema _____

_____

_____

_____

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

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

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

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

Type of effect		Respiratory rate (resp/min)	General state	Pathomorphological changes in lungs
Unnarcotized rat + injection of NH_4Cl	– Initial – 15 min – 30 min – 45 min – 55 min	128 150 20 Rare deep respiration Terminal respiration Respiratory arrest	Normal Impairment of movements coordination The rat is motionless, is lying on one side Neck and mouth muscles take part in respiration Foamy discharge from the mouth Death of the animal	Lungs weight — 6.0 g, pulmonary weight factor — 0.03. Foamy liquid in the trachea. Lungs volume is enlarged, they remind marble, foamy discharge on dissection
Hexenal narcosis + injection of NH_4Cl	Initial 15 min 30 min 55 min	100 103 102 102 Quiet, rhythmic respiration	General condition without visible changes	Lungs weight — 1.4 g, pulmonary weight factor — 0.007. The trachea is freely passable. The lungs collapsed, are of light pink color

Conclusions: Explain the protective action mechanism of hexanol narcosis on the development of toxic pulmonary edema: _____

_____

_____

_____

Control questions

1. Regulation mechanisms of water exchange and their impairment (hypo- and hyperhydrations).
2. Edemas and dropsies (definition).
3. Kinds of edemas.
4. Pathogenic factors of edema development.
5. Pathogenesis of cardiac, renal, toxic, cachectic 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.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 15).

2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.

3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.

5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.

6. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.

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8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.

9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

LESSON 16. HYPOXIA

Date: «____» _____ 20___

The purpose of the Lesson: to study etiology and pathogenesis of hypoxic conditions, their types, basic manifestations, urgent and long-term mechanisms of compensatory-adaptive reactions in response to hypoxia.

Tasks:
- to study pathogenic action of the lowered barometric pressure on the organism (dysbaric phenomena) in experiment;
- to study the reasons and developmental mechanisms of some kinds of hypoxia using the educational materials;
- solving situational tasks;
- control test.

PART 1. WORK WITH EDUCATIONAL MATERIALS

1. Put down the definition of the notion *"hypoxia"* — _____
_____

2. The essence of oxygen homeostasis is _____
_____

3. The main components of oxygen homeostasis are: _____
_____

4. Fill in the Table:

Table 1

Kinds of Hypoxia

Degree of seviety	Rate of development	Localization	Etiology
1) _____	1) _____	1) _____	1) _____
2) _____	2) _____	2) _____	2) _____
3) _____	3) _____		
4)	4)		

Table 2

Types of hypoxia depending on the cause of their development

Exogenous		**Endogenous**	
Types	**Reasons and pathogenesis**	**Types**	**Reasons and pathogenesis**
Hypoxic		1)	
1)		2)	
2)		3)	
Hyperoxic		4)	
1)		5)	
2)		6)	
		7)	

Table 3

Stages of Hypoxia

Stages	**Manifestations**
Hidden	
Compensated	
Expressed	
Severe not compensated	
Terminal	

Table 4

Mechanisms for short-term adaptation

Organ and systems		Effects		Mechanisms of effects
System of external breathing	→		→	
Heart	→		→	
Circulatory System	→		→	
Blood System	→		→	
Biological oxidation system	→		→	

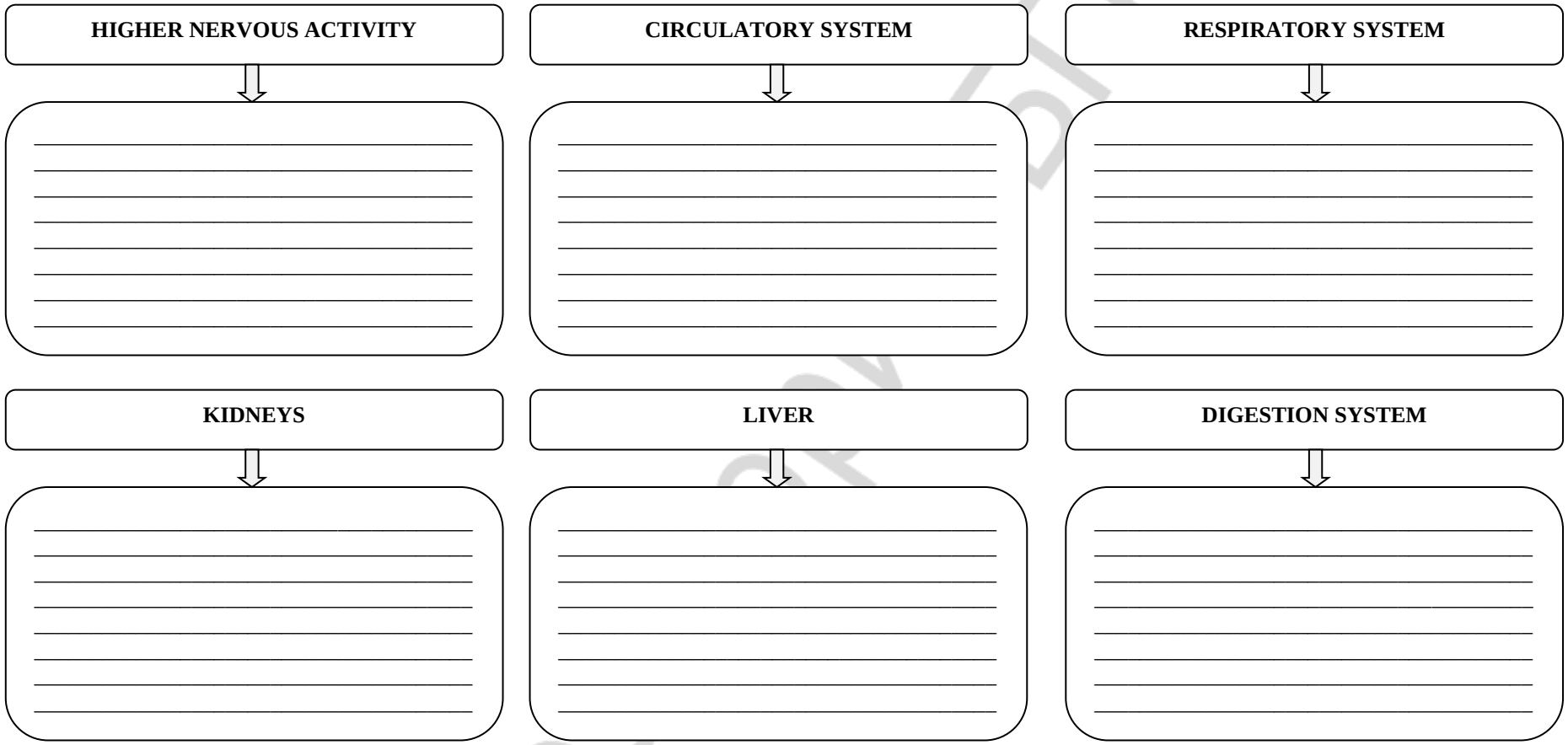
Table 5

Mechanisms for long-term adaptation

Organ and systems		Effects		Mechanisms of effects
System of external breathing	→		→	
Heart	→		→	
Circulatory System	→		→	
Blood System	→		→	
Biological oxidation system	→		→	

5. Fill in the scheme:

Disorders manifestations of organ and tissue functions at hypoxia



6. Give the definition of *"disbarism"* — _____
_____
_____

PART 2. PRACTICAL PART

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

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

Kind of animal	**General condition while "raising the altitude", km**					
	3–4		**3–4**		**3–4**	
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	–//–

Answer the questions:

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

_____

_____

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

_____

Work 2. STUDYING "DYSBARIC" PHENOMENA (MODELING EXPERIMENT)

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

Answer the questions:

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

а) 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 OF THE REASONS AND MECHANISMS OF SOME TYPES OF HYPOXIA; CHANGES IN THE BLOOD AND TISSUES

1. Fill in the Table:

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

Type of hypoxia	P_AO_2	P_aO_2	P_vO_2	$\Delta a\text{-}vO_2$	HbO_2 content	P_aCO_2	Type of hypoxia
1. Hyperbaric							
2. Normobaric							
3. Respiratory							
4. Circulatory							
5. Hemic							
6. Tissue							
7. Loading							

2. Describe the pathological forms of hemoglobin. Fill in the Table:

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

Control questions

1. The definition of the notion “hypoxia”. Hypoxia as a typical pathological process.
2. Principles of classification of hypoxic conditions. Types of hypoxia.
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.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 16).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

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4. Hypoxia (pathophysiological aspects): Teaching manual by Melenchuk E.V., Zhadan S.A., Vismont F.I. – Minsk: BSMU, 2014. – 24 p
5. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.
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10. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher’s signature: _____

LESSON 17. TYPICAL IMPAIRMENTS OF TISSUE GROWTH. TUMORS. BIOLOGICAL PECULIARITIES OF TUMOR GROWTH. METHODS OF EXPERIMENTAL REPRODUCTION OF TUMORS. ETIOLOGY OF TUMOURS

Date: «____» _____ 20__

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

Tasks:

– studying the methods of experimental oncology, issues of epidemiology and etiology of malignant neoplasms, biological peculiarities of a tumor cell on the basis of the educational materials;

– studying manifestations of cellular atypism of tumors on micropreparations of Ehrlich's ascite carcinoma and cellular line of human gastric cancer cave (carcinoma ventricular);

– solving situational tasks.

PART 1. WORK WITH EDUCATIONAL MATERIALS

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

2. Define the concept of ***"tumor"*** — _____
_____
_____

3. List the risk factors for developing of tumors:_____
_____

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

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

6. List the basic cancerogenic effects of ***physical*** origin:_____
_____

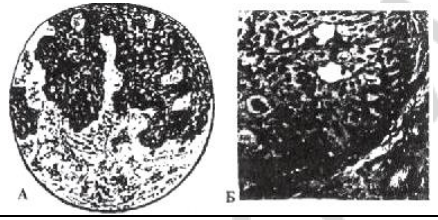
7. List the basic ***biological*** carcinogens: _____
_____

8. Specify the types of human malignant neoplasms the viral etiology of which is proved: _____
_____

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

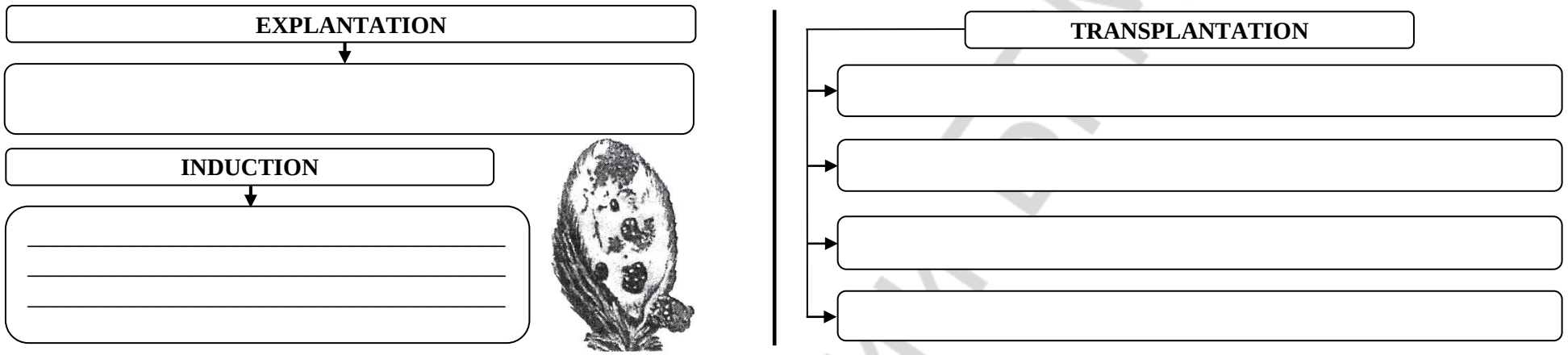
10. Fill in the Table.

Biological peculiarities of malignant tumors

Biological peculiarities	**Characteristic of neoplasm features**	
	Benig	**Malignant**
1. Relative autonomy and unregulated growth (indicate the reasons for the uncontrolled and unlimited proliferation of cells with tumor growth)		
2. Inheritance of changes		
3. Recurrence ability		
4. Immortalization — the immortality of the tumor population		
5. Growth character of tumor 		
6. Metastasis (stages)		

7. Morphological atypism:		
– tissue		
– cellular		
8. Functional atypism:		
– hypo-		
– hyper-		
– dysfunction		
9. Biochemical atypism		
10. Energetic atypism		
11. Antigenic (AG) atypism:		
– antigenic simplification		
– antigenic divergence		
– antigenic reversion *(indicate specific tumor antigenic markers)*		
12. Tumor progression		
13. The systemic effect of the tumor on the body		

11. List the methods for experimental reproduction of tumors:



PART 2. PRACTICAL PART

Work 1. 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 the cellular line of gastric cancer CaVe

The cellular line CaVe was discovered by J. V. Dobrynin and R. P. Dirlugjanom in 1959 from a solid cancer of the antral department of the stomach. The cellular line is presented by large polygonal or slightly elongated epithelium-like cells with light transparent cytoplasm. The cell borders are clearly visible. The nuclei are round, with 3–7 nucleoli of irregular shape. Overgrown cultures look as a continuous epithelial layer or as fusing cellular membranes with narrow slits. Sometimes the tubular formations remaining iron elements are observed among a continuous layer of cells. Examining the preparation fixed and stained in hematoxilin-eosin under large magnification (10 × 90), observe and sketch morphological peculiarities of tumor cells:

Fig. 1. 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

Studying a micropreparation of Ehrlich's ascite carcinoma

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

During microscopic investigation observe cellular atypism (dwarfish and gigantic cells of various form), prevalence of round cells with an extremely hypochromous nucleus and sharp basophilic cytoplasm (so-called dark cells), the presence of large cells with clearly outlined chromatin structure and pale-stained cytoplasm ("light" tumor cells); frequent mitosis and amitosis, pathologic mitosis, division of nuclei without division of cytoplasm:

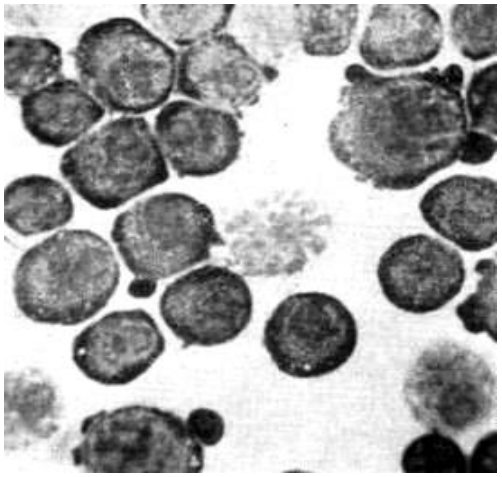


Fig. 2. Cells of Ehrlich's Ascite Carcinoma:

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

Answear on the questions:

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

_____
_____

2. What division abnormalities are characteristic of tumor cells? _____
_____
_____

Control questions

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

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 17).
2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.
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9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher’s signature: _____

LESSON 18. PATHOPHYSIOLOGY OF TISSUE GROWTH. TUMORS. MECHANISMS OF CARCINOGENESIS. RELATIONSHIPS OF THE TUMOR AND ORGANISM. PRINCIPLES OF TREATMENT AND PREVENTION OF TUMORS

Date: «____» _____ 20__

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

Tasks:

– to study mutational, epigenomic, viral-genetic theories of tumor pathogenesis, modern conceptions of tumoral transformation mechanisms (the theory of an oncogen); interaction problems of a tumor and major regulatory systems of the organism — neuro-endocrine and immune; mechanisms of systemic tumor effect of on the organism on the basis of studing materials "Pathological physiology of tumor growth". Studying cytogenetic peculiarities of cells in ascite hepatoma 22A;

– solving situational tasks;

– final control test on the topic "Pathological physiology of tissue growth" (Lesson 17, 18).

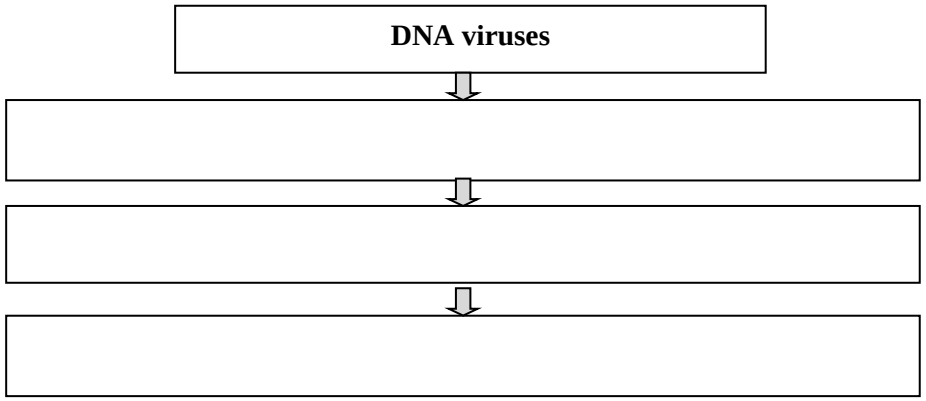
PART 1. WORK WITH EDUCATIONAL MATERIALS

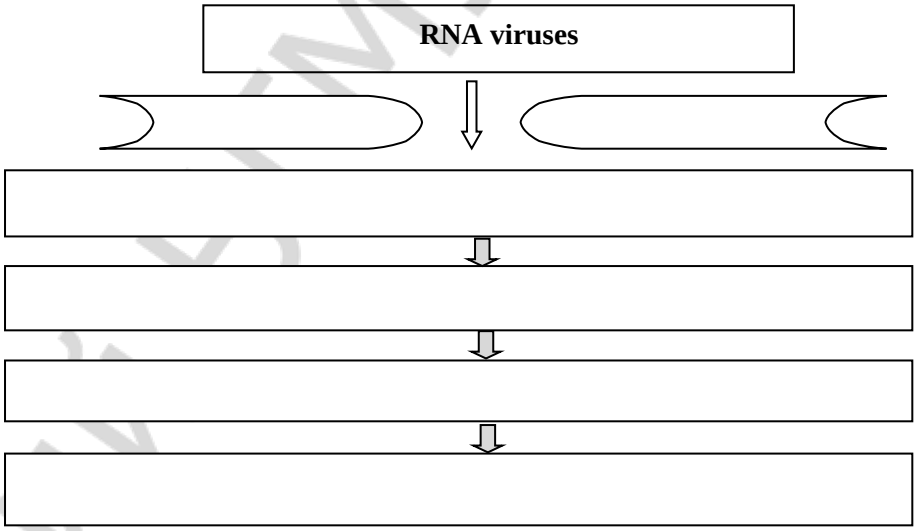
1. Fill in the Table:

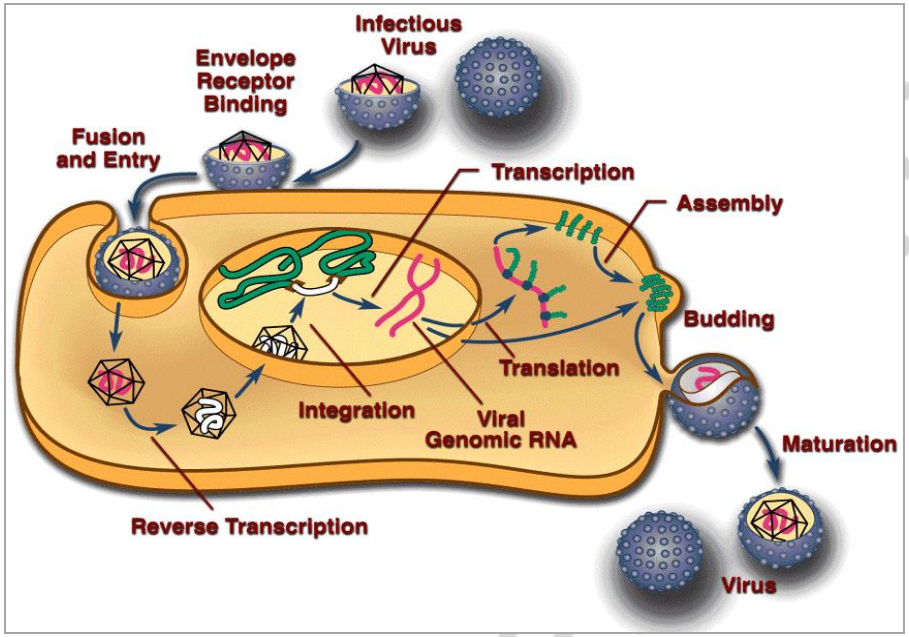
Characterization of the basic theories of carcinogenesis

Theory	Essence of the concept
Somatic Mutation	
Epigenetic	
Virus	
Modern	

2. Complete the scheme of oncovirus action







An oncovirus is a virus that can cause cancer. In the 1950–60s, these acutely transforming retroviruses were often called *oncornaviruses* to show their RNA virus origin. Now it refers to any virus with a DNA or RNA genome causing cancer and can be used synonymously with "tumor virus" or "cancer virus". However, the majority of animal and human viruses to do not cause cancer; this is probably because of the coevolution between the virus and its host. In most viruses, DNA is transcribed into RNA, and then the RNA is translated into a protein through protein synthesis. However, retroviruses (mentioned above) are a single-stranded RNA virus that stores its nucleic acid in the form of mRNA and then targets a host cell as an obligate parasite. Once it gets inside the hosts cells cytoplasm, the virus transcribes differently than most viruses. The virus uses its own reverse transcriptase enzyme to produce DNA from its RNA genome, in reverse of the usual pattern. When the new DNA is incorporated into the host cell's genome by an integrate enzyme, the retroviral DNA is referred to as a provirus. This DNA becomes integrated into the host cell's genome and then undergoes the usual transcriptional and translational processes to express the genes carried by the virus, producing the proteins required to assemble new copies of the virus. It is difficult to detect the virus until it has infected the host, which makes research an early detection hard.

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

_____

4. What is a *"proto-oncogen"*? — _____

_____

_____

_____

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

_____

_____

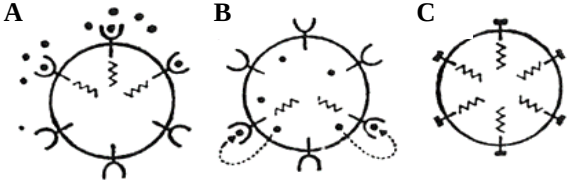
6. List the transformation mechanisms of a proto-oncogen into an oncogen:

1) _____
2) _____
3) _____
4) _____
5) _____
6) _____

7. List the basic functions of oncogen — *oncoproteins*:

A — _____
B — _____
C — _____

A **B** **C**



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

_____

_____

_____

9. List the main stages of carcinogenesis and their stages:

1) _____ (*phase: a)* _____; *b)* _____; *c)* _____);
2) _____ (*phase: a)* _____; *b)* _____);
3) _____ (*phase: a)* _____; *b)* _____).

10. Explain the reasons for the ineffectiveness of immune responses to the tumor:

1) _____
2) _____
3) _____
4) _____

11. The main mechanisms of immunosuppression in cancer:

1) _____
2) _____
3) _____
4) _____
5) _____

12. Fill in the Table:

Basic manifestations of systemic tumor effect on the organism (Paraneoplastic syndrome)

Syndrome	Development mechanism	Basic manifestations
Cachexia		
Immunopathological		
Psychoneurological		
Paraneo-endocrine		

Syndrome	Development mechanism	Basic manifestations
Thrombo-hemorrhagic		
Anemic		

13. Indicate the main causes of pain development in the malignant tumors: _____
_____

14. Put down the definition of the term *"antineoplasytic resistance"* — _____
_____

15. Fill in the Table:

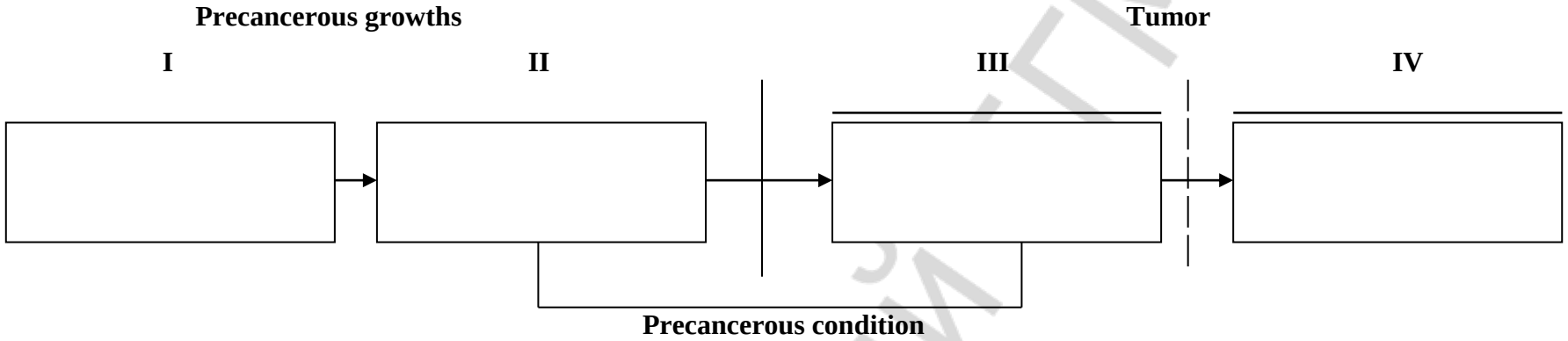
Mechanisms of antineoplasytic resistance	Essence	Examples
Anticarcinogenic effects		
Antitransformation effects		
Anticellular effects		

16. Put down the definition of the term *"precancerous conditions"* —_____
_____

17. Fill in the Table:

Type of precancerous condition	Probability of malignancy	Examples
Facultative		
Obligate		

18. Fill out the scheme reflecting the staged development of malignant tumors:



19. The main ways to prevent malignant neoplasms:

Clinical prophylaxis	Hygiene prophylaxis

20. List the basic principles for treatment of tumor disease: _____
_____
_____

PART 2. PRACTICAL PART

Work 1. STUDYING CYTOGENETIC PECULIARITIES OF A CELL IN ASCITE HEPATOMA 22A

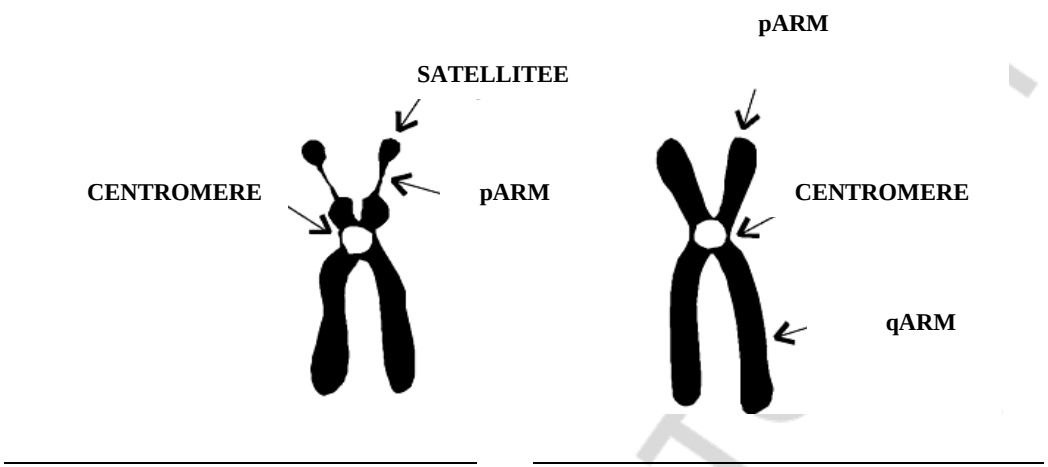
The cellular karyotype is examined by studying metaphasal plates under the light microscope. For this purpose, cells of ascite hepatoma 22A are processed with colchicine resulting in the arrest of cellular division at a metaphase stage by suppressing the formation of spindles. Then, the cells are applied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

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

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

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

Designate the corresponding chromosomes in the figure below:



Marker chromosomes in ascite hepatoma 22A cells

1 — acrocentric chromosome with despiralized near-centric site;

2 — submetacentric chromosome

Control questions

1. The evolution of ideas about tumors pathogenesis. The role of mutational, epigenomic, and virusogenetic mechanisms in carcinogenesis.
2. Modern ideas about the molecular genetic foundations of malignant transformation. Oncogen concept; nature of products of oncogen activity and possible mechanisms of their action.
3. The concept of anti-oncogenes (protective genes or suppressor genes).
4. Stages of carcinogenesis.
5. The relationship of the tumor and the body:

- mechanisms of anti-blastoma resistance;
- the role of the nervous system in tumor development;
- the role of the endocrine system in tumor development; the concept of dishormonal tumors;
- the role of the immune system in tumors development; modern ideas about antitumor immunity.

6. Manifestations, mechanisms of systemic action of tumor on the body, causes and mechanism of cancer cachexia development.
7. The concept of precancerous conditions, types of precancer.
8. Principles of the prevention and treatment of tumors.

RECOMMENDED LITERATURE

Basic

1. EAMC (http://etest.bsmu.by/ → Courses → For Students with Training in English → General Medicine → Pathological Physiology → Lesson 18).

2. *Pathophysiology* : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. V. Krishtal [et al.] ; ed. by N. V. Krishtal, V. A. Mikhnev. Kyiv : AUS Medicine Publishing, 2017. 656 p.

3. *Kumar, V.* Robbins Basic Pathlogy / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

Additional

4. *General* and clinical pathophysiology : textbook for students of higher educational institutions, of IV th level of accreditation / A. V. Kubyshkin [et al.] ; ed. by A. V. Kubyshkin, A. I. Gozhenko. 2nd ed. Vinnytsya : Nova Knyha Publishers, 2016. 656 p.

5. *Litvitsky, P. F.* Pathophysiology : textbook for students / P. F. Litvitsky, S. V. Pirozhkov, E. B. Tezikov. Moscow : GEOTAR-Media, 2016. 432 p.

6. *Simeonova, N. K.* Pathophysiology : textbook for students of higher medical educational institutions of the III–IV accreditation levels / N. K. Simeonova ; ed. by V. A. Mikhnev. 2nd ed. Kyiv : AUS Medicine Publishing, 2015. 544 p.

7. *Gozhenko, A. I.* Pathophysiology / A. I. Gozhenko, I. P. Gurkalova. Odessa : The Odessa State Medical University, 2005. 325 p.

8. *Mufson, M. A.* Pathophysiology : PreTest Self-Assessment & Review / M. A. Mufson, C. A. Heck, S. M. Nesler. 3th ed. Chicago : Medical Publishing Division, 2002. 268 p.

9. *McPhee, S. J.* Pathophysiology of Disease: An Introduction to Clinical Medicine [Electronic resource] / S. J. McPhee. 8nd ed. NY : McGraw-Hill Education, 2019.

The teacher's signature: _____

CREDIT. PROTECTION OF ABSTRACT

Date: «____» _____ 20__

The purpose of the lesson: during the preparation of the essay, to deepen, systematize and consolidate the theoretical knowledge obtained in the discipline "Pathological physiology". Writing an essay allows you to consolidate the acquired by students the ability to search for the necessary information, orientation in modern scientific literature.

A credit lesson takes the form ***of defense the abstract*** by student.

Abstract writing

The structure of the abstract includes:

• **Title page** (a sample title page is presented in the appendix);

• **A table of contents** indicating the work plan, which should contain an introduction, the name of the main sections (subsections) of the work, a conclusion, a list of used literature;

• **Introduction** which defines the purpose and objectives of the study, its relevance, theoretical and practical significance, the basic questions studied, as well as not fully disclosed questions on the topic under study, the object and subject of the study are determined, and statistical methods, if applicable, are indicated;

• **The main text** which reveals the main content of the plan. The text should contain at least two sections (subsections are allowed);

• **Conclusion** where evidence is formed on the basis of the content of the material studied by the author;

• **List of used literature and other sources**. Literature is drawn up in accordance with the requirements of GOST 7.1-2003 "Bibliographic record. Bibliographic description. General requirements and compilation rules". References to the literature are printed inside the article in square brackets after the quotation according to the alphabetical order declared in the list of references. The number of references in the work should be at least ten.

The text of the work should be printed on one side of a white sheet of A4 paper (orientation — portrait) at 1.5 intervals, in Times New Roman font, size 14 (cover sheet — 16 font). Each page of text and applications should have margins: left — 30 mm, right — 10 mm, top and bottom 15 mm. The first line indent (paragraph indent) is 1.25 cm. Line spacing is one and a half. Do not do hyphenation. Pagination at the bottom center: all but the title page. Abstract volume: not less than 15 pages. **The student's signature is placed** on the last page of the abstract.

When writing a work, **compliance with copyright requirements is required**. A report is attached to the abstract to check the text of **the work for plagiarism** (the uniqueness of the work (introduction, main part and conclusion) should be at least 60 %). Checking the uniqueness of the work is carried out on one of the following sites: https://www.text.ru or https://www.antiplagiat.ru.

The abstract should be drawn up according to the requirements.

The abstract is protected by a short (6–8 min) presentation to the audience on the topic of work and answers to questions. When speaking, the use of multimedia devices (projector, computer, TV) is allowed.

Early defense of the abstract is possible at one of the practical classes during the semester, provided that the work is performed in accordance with all the requirements presented and the content of the abstract corresponds to the topic of the practical lesson.

Selection of the topic of the abstract

The topics of the essay are determined by the requirements of the curriculum, the program for the study of the discipline "Pathological physiology" at the Belarusian State Medical University, cover sections submitted to an independent form of training. The independent choice of the topic of the essay by the student is also allowed, provided that it is agreed with the teacher.

Themes of abstracts

1. Experimental modeling in medicine.
2. Postresuscitation pathology.
3. The pathogenic effect of meteorological factors on the body.
4. Hypothermia and its effect on the human body.
5. Infectious process as a form of interaction of micro- and macroorganisms.
6. Immunodeficiency conditions.
7. Allergy as a form of pathological reactivity.
8. Diseases of autoimmune aggression and their importance in human pathology.
9. Pathology of carbohydrate metabolism, hereditary and acquired forms.
10. Damage to the lysosomal apparatus of cells as a key link in the pathogenesis of certain diseases.
11. Non-ionizing radiation and their pathogenic effect.
12. Small doses of radiation. Modern ideas about biological effects and mechanisms of their action.
13. The concept of microelements. Biological system of microelement homeostasis.
14. Therapeutic fasting.
15. Proteins of heat shock and their role in pathology.
16. To the question of the possibility of reversing malignant growth (the problem of "normalization" of the tumor cell).
17. Diabetes mellitus. Pathophysiological aspects. The mechanisms of insulin resistance.
18. Molecular mechanisms of the development of angiopathy in diabetes.
19. Hypo- and hyperglycemic conditions.
20. Disorders of protein metabolism. Primary and secondary dysproteinemia.
21. Disorders of lipid metabolism. Primary and secondary dyslipidemia.
22. Obesity, its types. Etiology and pathogenesis.

23. Disorders of cholesterol metabolism. Modern ideas about the pathogenesis of atherosclerosis.
24. Alimentary insufficiency. Starvation and its types.
25. Pathophysiology of the exchange of macro- and microelements.
26. Extreme states. Etiology and pathogenesis.
27. Stress and its role in pathology.
28. Inherited determinations of collagen metabolism.
29. Mucopolysaccharidoses. Etiology and pathogenesis.
30. The role of Kupffer cells in normal and pathological conditions.
31. The main causes of violations in the functional system of the mother – placenta – fetus (FSMPP). Factors that have a damaging effect on the embryo and fetus.
32. The concept of gestational dominant (dominant of pregnancy), its importance in pathology.
33. Critical (sensitive) periods in the life of the embryo and fetus, their significance in pathology.
34. Features of the functional integration of homologous organs of the fetus and the maternal organism in the conditions of pathology (examples).
35. Placental insufficiency. Definition, reasons, criteria.
36. Anomalies of development, their types, characteristics, timing of occurrence, consequences.
37. Hypoxia of the fetus and newborn. Causes, types, consequences.
38. Alcoholic disease of the fetus and newborn.
39. The concept of transplant and blastomogenesis.
40. Aging of the body. The main laws and mechanisms.
41. Physiological and pathological aging. Progeria. Reasons, types, manifestations.
42. The main component of aging is changes at the cellular, subcellular, molecular levels.
43. The main adaptive mechanisms in aging.
44. Aging and disease. The main types of pathology, their anatomical foundations.
45. Features of the manifestation and course of disease in the elderly and senile.
46. Social aspects of aging. The struggle for active longevity. Possible approaches to increase life expectancy.
47. The main adaptive mechanisms in complete fasting.
48. Alimentary dystrophy. The reasons are the main manifestations.
49. The concept of therapeutic fasting.
50. Philo- and ontogenesis of reactivity and resistance. Features of reactivity of early childhood.
51. Diathesis. Definition of a concept. Modern View on the pathogenesis of diathesis. The main clinical forms of diathesis in children.
52. Philo- and ontogenesis of the inflammatory reaction.
53. Phylo- and ontogenesis of a febrile reaction.

The student in the credit lesson receives a test in the discipline "Pathological physiology" when all the above conditions are met:

1. Attendance at all practical classes (if there are missing lessons, it is neesesary to work out them in accordance with the applicable requirements).
2. Attendance at all lectures (if there are missing lessons, it is neesesary to work out them in accordance with the applicable requirements).
3. Submission and defense of the abstract.
4. The presence of the teacher(s) signature in the workbook after each lesson.

Abstract topic: _____

_____

The teacher's signature: _____

Appendix

Sample title page abstract

Ministry of Health of the Republic of Belarus

Belarusian State Medical University

Department of Pathological Physiology

ESSAY

The topic: **"Modern ideas about mediators of fever and their role in pathology"**

Performed:
3rd year student
Faculty of General Medicine
Group 6301
Ivanov Ivan Ivanovich

Scientific adviser:
MD, Professor
Vismont F.I.

CONTENT

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GENERAL PATHOLOGICAL PHYSIOLOGY

Рабочая тетрадь

На английском языке

2-е издание

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