

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ  
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ  
КАФЕДРА ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ

**ОБЩАЯ ПАТОФИЗИОЛОГИЯ**  
**GENERAL PATHOLOGICAL PHYSIOLOGY**

Рабочая тетрадь



Минск БГМУ 2020

УДК 616-092(076.5)(075.8)-054.6  
ББК 52.5я73  
О-28

Рекомендовано Научно-методическим советом университета в качестве  
рабочей тетради 15.04.2020 г., протокол № 8

А в т о р ы: Ф. И. Висмонт, С. А. Жадан, С. Н. Чепелев, Е. Н. Чепелева,  
Д. М. Писарик

Р е ц е н з е н т ы: каф. патологической физиологии Витебского государст-  
венного 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

**Общая** патофизиология = General pathological physiology : рабочая тет-  
О-28 радь / Ф. И. Висмонт [и др.]. – Минск : БГМУ, 2020. – 156 с.

ISBN 978-985-21-0552-1.

Содержит задания, предназначенные для работы с учебными материалами, описания  
и протоколы оформления лабораторных работ по курсу «Общая патофизиология».

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

УДК 616-092(076.5)(075.8)-054.6  
ББК 52.5я73

ISBN 978-985-21-0552-1

© УО «Белорусский государственный  
медицинский университет», 2020

## LIST OF ABBREVIATIONS

AH — arterial hyperemia

BP — blood 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 sickness

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<sub>2</sub> — oxyhemoglobin

P<sub>A</sub>CO<sub>2</sub> — partial pressure of carbon dioxide in alveolar air

P<sub>a</sub>CO<sub>2</sub> — partial pressure of carbon dioxide in arterial blood

P<sub>A</sub>O<sub>2</sub> — partial pressure of oxygen in alveolar air

P<sub>a</sub>O<sub>2</sub> — partial pressure of oxygen in arterial blood

P<sub>v</sub>CO<sub>2</sub> — partial pressure of carbon dioxide in venous blood

P<sub>v</sub>O<sub>2</sub> — 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. \_\_\_\_\_ (signature)  
(date)

#### **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

General Nosology	Typical Pathological Processes (TPP)	Typical forms of tissues organs and systems pathology										
<p><i>General Nosology is</i></p> <p>_____</p> <p>_____</p> <p>_____</p> <p>1. _____</p> <p>_____</p> <p>_____</p> <p>2. _____</p> <p>_____</p> <p>_____</p> <p>3. _____</p> <p>_____</p> <p>_____</p>	<p><i>Typical pathological processes — are</i> _____</p> <p>_____</p> <p>_____</p> <p style="text-align: center;"><i>Signs of typical pathological processes</i></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">Signs</th> <th style="width: 70%;">Characteristics</th> </tr> </thead> <tbody> <tr> <td style="vertical-align: top;">1. _____</td> <td style="vertical-align: top;">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 style="vertical-align: top;">2. _____</td> <td style="vertical-align: top;">TPP has a standard mechanism of development (for example, the pathogenesis of inflammation includes components of alteration, exudation, and proliferation).</td> </tr> <tr> <td style="vertical-align: top;">3. _____</td> <td style="vertical-align: top;">TPP is a complex of adaptive, compensatory and pathological changes.</td> </tr> <tr> <td style="vertical-align: top;">4. _____</td> <td style="vertical-align: top;">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> </tbody> </table> <p><i>Examples of typical pathological processes:</i> _____</p> <p>_____</p>	Signs	Characteristics	1. _____	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.)	2. _____	TPP has a standard mechanism of development (for example, the pathogenesis of inflammation includes components of alteration, exudation, and proliferation).	3. _____	TPP is a complex of adaptive, compensatory and pathological changes.	4. _____	TPP has standard manifestations (acute inflammation is characterized by both general (leukocytosis, fever, etc.) and local (pain, redness, swelling of the tissue, fever, etc.).	<p><i>Examples of typical forms of pathology:</i></p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p>
Signs	Characteristics											
1. _____	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.)											
2. _____	TPP has a standard mechanism of development (for example, the pathogenesis of inflammation includes components of alteration, exudation, and proliferation).											
3. _____	TPP is a complex of adaptive, compensatory and pathological changes.											
4. _____	TPP has standard manifestations (acute inflammation is characterized by both general (leukocytosis, fever, etc.) and local (pain, redness, swelling of the tissue, fever, etc.).											
	<b>General pathophysiology</b>	<b>Specific pathophysiology</b>										



## 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) _____	1) _____	1) _____
a) _____	_____	_____
b) _____	2) _____	2) _____
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. Pavlov).
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 Pathology* / 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>I. Hidden (latent, incubative)</i>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>
<i>II. Prodromal</i>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>
<i>III. Marked clinical manifestations</i>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <p><i>Variants of a disease course:</i></p> <ul style="list-style-type: none"> <li>▪ _____</li> <li>▪ _____</li> </ul>	<hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>
<i>IV. Disease outcome</i>	<p><i>Possible outcomes of a disease:</i></p> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/> <hr/>		

7. Put down a definition of the concept “*etiology*”: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

8. Fill in the Table:

<b>Etiology</b>	
<b>General</b>	<b>Specific</b>
<i>Pathology section studying</i> _____ _____ _____ _____	<i>Pathology section studying</i> _____ _____ _____ _____

9. Put down a definition of the concept “*reason*”: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

10. Fill in the Table:

<b>Causes of the disease</b>	
<b>Exogenic</b>	<b>Endogenic</b>
1. _____ 2. _____ 3. _____	1. _____ 2. _____ 3. _____
<b>Conditions</b>	
<b>Contributing Disease Emergence</b>	<b>Preventing Disease Emergence</b>
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
<i>Monocausalism</i>	_____ _____ _____	_____ _____ _____
<i>Conditionalism</i>	_____ _____ _____	_____ _____ _____
<i>Constitutionalism</i>	_____ _____ _____	_____ _____ _____

12. Put down a definition of the concept “*pathogenesis*”:  
 \_\_\_\_\_  
 \_\_\_\_\_

13. Fill in the Table:

Pathogenesis	
General	Specific
<i>Pathogenesis section studing</i> _____	<i>Pathogenesis section studing</i> _____
_____	_____
_____	_____

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**

Compensatory process	Characteristic								
<p><i>Regeneration</i></p>	<p><b>Regeneration is</b> _____</p> <hr/> <p style="text-align: center;"><b><i>Kinds of regeneration:</i></b></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 50%; text-align: center;"><i>intracellular</i></th> <th style="width: 50%; text-align: center;"><i>cellular</i></th> </tr> </thead> <tbody> <tr> <td>— _____</td> <td>— _____</td> </tr> <tr> <td>— _____</td> <td>— _____</td> </tr> <tr> <td>— _____</td> <td>— _____</td> </tr> </tbody> </table>	<i>intracellular</i>	<i>cellular</i>	— _____	— _____	— _____	— _____	— _____	— _____
<i>intracellular</i>	<i>cellular</i>								
— _____	— _____								
— _____	— _____								
— _____	— _____								
<p><i>The presence of paired organs</i></p>	<p>— _____</p> <hr/> <p>— _____</p> <hr/> <p><b>Examples:</b> _____</p> <hr/>								
<p><i>Activation of the duplicated systems function</i></p>	<p>_____</p> <hr/> <p>_____</p> <hr/>								
<p><i>Presence of reserve structures</i></p>	<p>_____</p> <hr/> <p>_____</p> <hr/>								



20. Fill in the Table:

**Types of adaptive and compensatory reactions and their characteristics**

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

21. Fill in the Table:

**Stages of Adaptation Reactions**

Stages	Characteristic
<i>Emergency stage</i>	— _____ _____ _____ — _____ _____
<i>Sustained hyperfunction stage</i>	_____ _____ _____
<i>Stage of progressive organ sclerosis</i>	_____ _____ _____

22. Fill in the Table:

**Cross adaptation and compensation**

Kind	Positive	Negative
<i>Characteristic</i>	_____ _____ _____	_____ _____ _____
<i>Examples</i>	_____ _____ _____	_____ _____ _____

23. Put down a definition of the concepts “adaptation price” and “compensation price”:

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

24. Fill in the Table:

**Periods of a terminal condition**

Periods	Consciousness (+/-)	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<sub>2</sub> 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 <sub>a</sub> O <sub>2</sub> , 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 monocausenatism, 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 expressiveness of 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 Pathology* / 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 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? \_\_\_\_\_

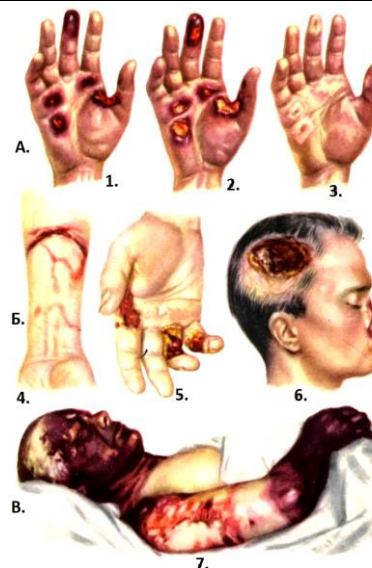
\_\_\_\_\_

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		
Type	Specific Characteristic	Nonspecific
Biological action	_____	<i>The nonspecific effect of current is</i> _____
Electrochemical (electrolytic) action	_____	_____
Electrothermal action	<i>Electric labels — are</i> _____	Examples: _____
Electromechanical (dynamic) action	_____	_____



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**





7. Fill in the Table:

**Causes of death due to electrical injury**

Form	Cardiac form	Respiratory form	Mixed form
<i>Reasons</i>	1. _____	1. _____	_____
	2. _____	2. _____	_____
	3. _____	3. _____	_____
	4. _____	4. _____	_____
<i>Skin color</i>			

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 Pathology* / 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**

<b>Level of biological organization</b>	<b>Characteristics of radiation damage</b>
<i>Molecular</i>	
<i>Subcellular</i>	
<i>Cellular</i>	
<i>Tissue, organ</i>	
<i>Organism</i>	
<i>Population</i>	

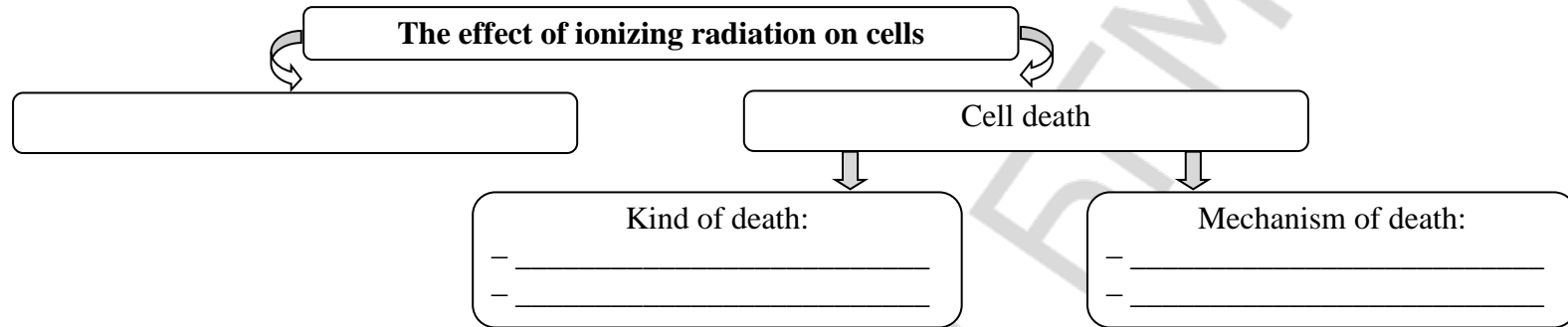
8. What are the stages of radiation damage (*structural-metabolic theory of A. M. Kuzin (1986)*):

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
<i>Skeletal</i>	
<i>Reticuloendothelial</i>	
<i>Diffuse</i>	
<i>Selective</i>	

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**

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



17. Fill in the Table:

**Possible consequences of exposure**

Effects	Stochastic	Non-Stochastic
<i>Characteristic</i>	<hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>
<i>Examples</i>	<hr/> <hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>

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 <sup>12</sup> /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 <sup>9</sup> /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 Pathology* / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

##### *Additional*

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.
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 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**

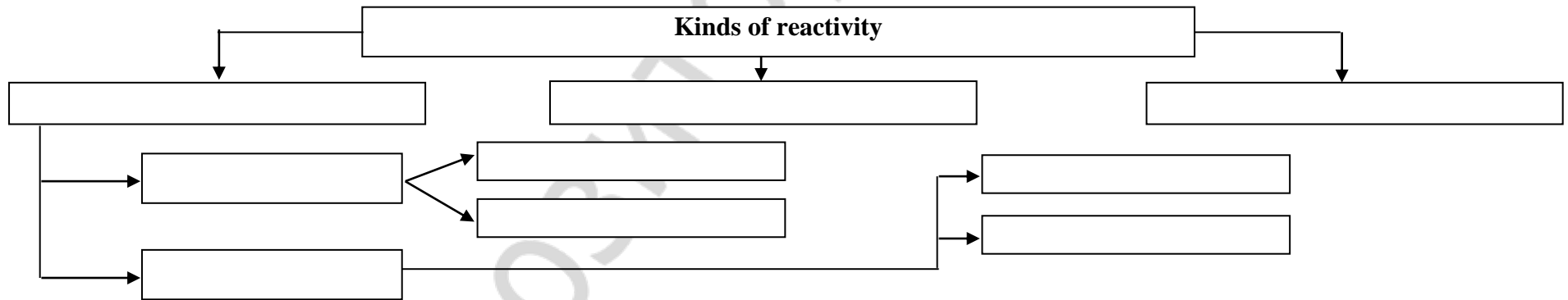
Quantitative		Qualitative	
Indicator	Characteristic	Indicator	Characteristic
1. <i>Normergy</i>	_____	_____	the most important qualitative indicator of reactivity; body resistance to pathogenic factors
	_____	_____	
	_____	_____	
2. _____	reduced reactivity, with a predominance of inhibitory processes	<i>Functional lability</i>	_____
			_____
			_____
3. <i>Hyperergy</i>	_____	_____	a common property of all living things that determines elementary reactions
	_____	_____	
	_____	_____	
4. _____	perverse reactivity	<i>Excitability</i>	_____
			_____
			_____
		<i>Sensitivity</i>	_____
			_____
			_____
		_____	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**

<b>By reducing the activity of vital processes (group 1)</b>	<b>By maintaining or increasing the level of organism vital activity (group 2)</b>	
<i>Means, techniques, methods</i>	<i>Means, techniques, methods</i>	<i>Examples</i>
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	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>
Seago	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>
E. Kretschmer	<hr/> <hr/>	<hr/> <hr/> <hr/>
M. V. Chernorutsky	<hr/> <hr/>	<hr/> <hr/> <hr/>
Eppinger and Hess	<hr/>	<hr/> <hr/> <hr/>
A. A. Bogomolets	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>
I. P. Pavlov	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/> <hr/>
W. H. Sheldon	<hr/> <hr/>	<hr/> <hr/> <hr/>

10. Fill in the Table:

**Features of aging**

<b>Feature</b>	<b>Characteristic</b>

11. Fill in the Table:

**Kinds of aging**

<b>Kinds of aging</b>	<b>Characteristic</b>

**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 mice 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<sub>2</sub> and CO<sub>2</sub> in flasks.

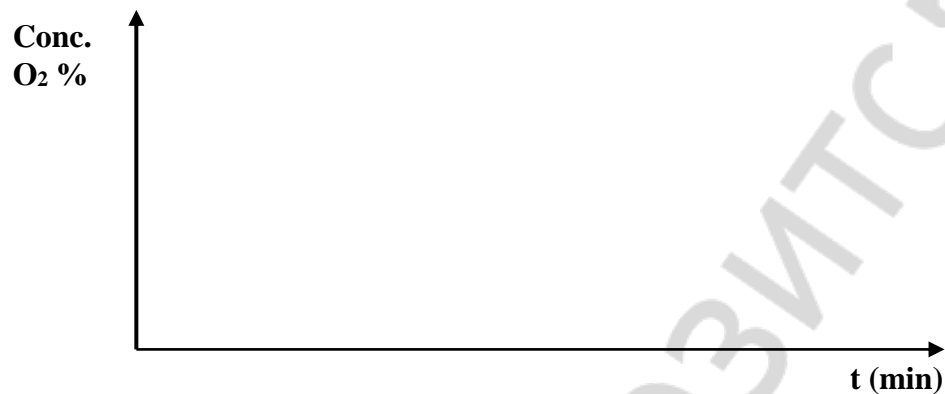
**Respiration rate (RR), the general state and life expectancy of control and tested mic**

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
<b>Gas mixture content in the flask: O<sub>2</sub> = 7.1 %; CO<sub>2</sub> = 11.8 %</b>			<b>Gas mixture content in the flask: O<sub>2</sub> = 3.4 %; CO<sub>2</sub> = 14.6 %</b>	

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<sub>2</sub> and [c] CO<sub>2</sub> 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<sub>1</sub>) and tested (V<sub>2</sub>) mice, having assumed the initial concentration of O<sub>2</sub> and CO<sub>2</sub> 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
<b>Gas mixture content in the flask: O<sub>2</sub> = 7.1 %; CO<sub>2</sub> = 11.8 %</b>			<b>Gas mixture content in the flask: O<sub>2</sub> = 5.6 %; CO<sub>2</sub> = 12.5 %</b>	

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.

	<b>Asthenic type</b>	<b>Normosthenic type</b>	<b>Hypersthenic type</b>
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 Pathology* / 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**

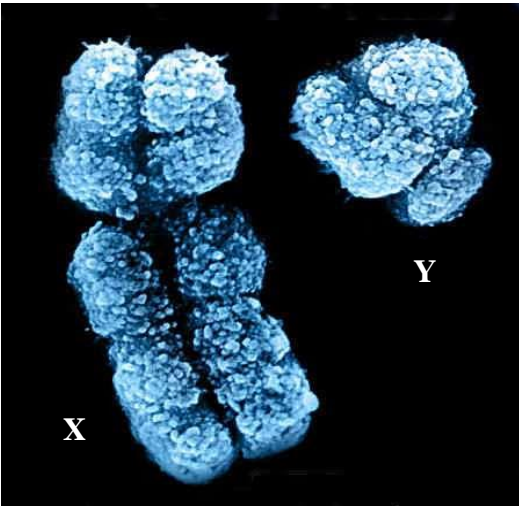
Group of diseases	The role of heredity and the environment	Examples of the disease
<i>Hereditary diseases</i>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>
<i>Single-gene diseases</i>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>
<i>Multifactoral polygenic diseases</i>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>
<i>Diseases caused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



2. Fill in the scheme:

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

<b>Congenital diseases</b>	<b>Hereditary diseases</b>	<b>Phenocopies</b>
definition: _____ _____ _____ _____ _____	definition: _____ _____ _____ _____ _____	definition: _____ _____ _____ _____ _____
- _____	clinical manifestation time: _____ _____ _____ _____	examples: _____ _____ _____ _____
- _____	<b>Non-Hereditary diseases</b> Definition: _____ _____ _____ Characteristic: _____ _____ _____ _____ _____	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**

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

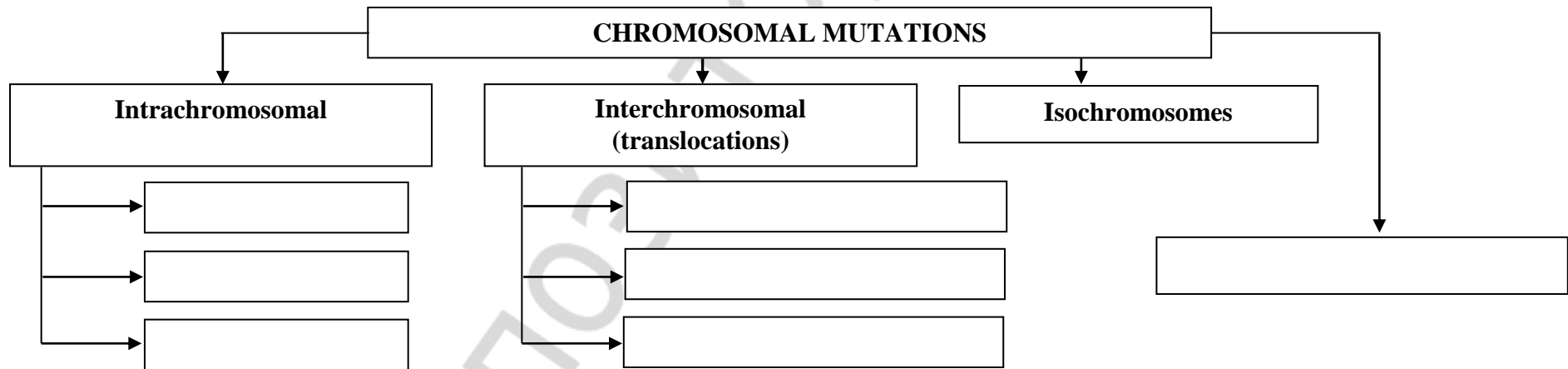
<b>Gene mutation</b>	<b>Characteristic</b>
<i>Deletion</i>	
<i>Duplication</i>	
<i>Inversion</i>	
<i>Insertion</i>	
<i>Transversion</i>	
<i>Transition</i>	

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.

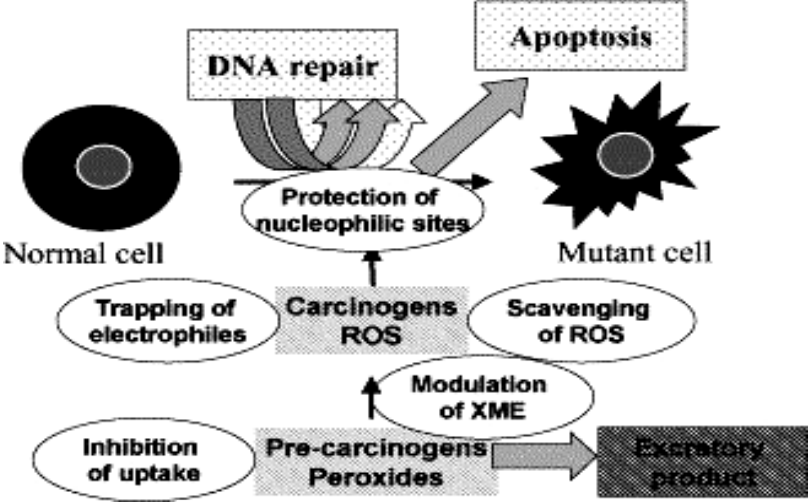
**Characteristic of some chromosomal syndromes**

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

10. Give the definition of the notion “antimutagenesis”:

11. Fill in the Table.

**Classification of antimutagens**

Exogenous	Endogenous
<p><i>Antimutagens in foods and entering the human body with food</i></p> <p>1. _____</p> <p>2. _____</p> <p>3. _____</p> <p>4. _____</p> <p>5. _____</p>	<p>1. _____</p> <p>2. _____</p> <p>3. _____</p> <p>4. _____</p>
<p><i>Antimutagens penetrating into the body through the respiratory system</i></p> <p>_____</p>	
<p><i>Antimutagens, ingested orally in the pharmacotherapy, or prophylactic uses</i></p>	
<p>1. _____</p> <p>2. _____</p> <p>3. _____</p> <p>4. _____</p>	 <p>The diagram illustrates the mechanisms of antimutagenesis. It shows a flow from 'Pre-carcinogens Peroxides' to 'Carcinogens ROS'. From 'Carcinogens ROS', three paths emerge: 1) 'Inhibition of uptake' leading to 'Excretory product'; 2) 'Modulation of XME' leading to 'Scavenging of ROS'; 3) 'Protection of nucleophilic sites' leading to 'DNA repair' and 'Apoptosis'. A 'Normal cell' is shown on the left, and a 'Mutant cell' is shown on the right. The overall process is labeled 'Mechanisms of antimutagens action'.</p>

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:** \_\_\_\_\_

**F1:** \_\_\_\_\_

**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:** \_\_\_\_\_

**F1:** \_\_\_\_\_

**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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_      **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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_      **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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_      **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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_      **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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_      **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: \_\_\_\_\_

F<sub>1</sub>: \_\_\_\_\_

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: \_\_\_\_\_

F<sub>1</sub>: \_\_\_\_\_

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: \_\_\_\_\_

F<sub>1</sub>: \_\_\_\_\_

Answer: \_\_\_\_\_

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

Character	Gene	Genotype

P: ♀ \_\_\_\_\_ x ♂ \_\_\_\_\_

G: \_\_\_\_\_

F<sub>1</sub>: \_\_\_\_\_

Answer: \_\_\_\_\_

12. Determine the birth probability of sick children with *hepatocerebral dystrophy* (*Wilson's disease*) 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: \_\_\_\_\_

F<sub>1</sub>: \_\_\_\_\_

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 blindness*, 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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_ **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: \_\_\_\_\_  
 F<sub>1</sub>: \_\_\_\_\_ **Answer:** \_\_\_\_\_

20. How many bodies of sex chromatin are there in people with genotype OX? XXY? XXX? XXXY? What is the sex of these people and what are they ill with?

Genotype	Number of sex chromatin bodies	Gender	Disease
OX			
XXY			
XXX			
XXXY			

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

Inheritance type	Pathology form	
<b>1 Autosomal-dominant (A-D)</b>	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 Polyposis of the large intestine Neurofibromatosis
<b>2 Autosomal-recessive (A-R)</b>	Crescent — cellular anemia (by incomplete domination) Galactosemia Phenylketonuria Alcaptonuria Albinism Glycogenosis	Mucoviscidosis Wilson–Konovalov Disease (hepato-cerebral dystrophy) Adrenogenital syndrome Congenital deaf-muteness Microcephaly
<b>3. Dominant X-linked (D-X)</b>	Frontal-nasal dysplasia Hypoplasia of dental enamel	Cataract Rickets, resistant to vitamin D
<b>4. Recessive X-linked (R-X)</b>	Hemophilia A and B Daltonism Hypogammaglobulinemia	Duchenne’s muscular dystrophy Hemeralopia
<b>5. Hologenic Y-linked (H-Y)</b>	Excessive hairiness of auricles	Azospemia
<b>6. Mitochondrial (M)</b>	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.

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

#### *Additional*

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.

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. 8th 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:**

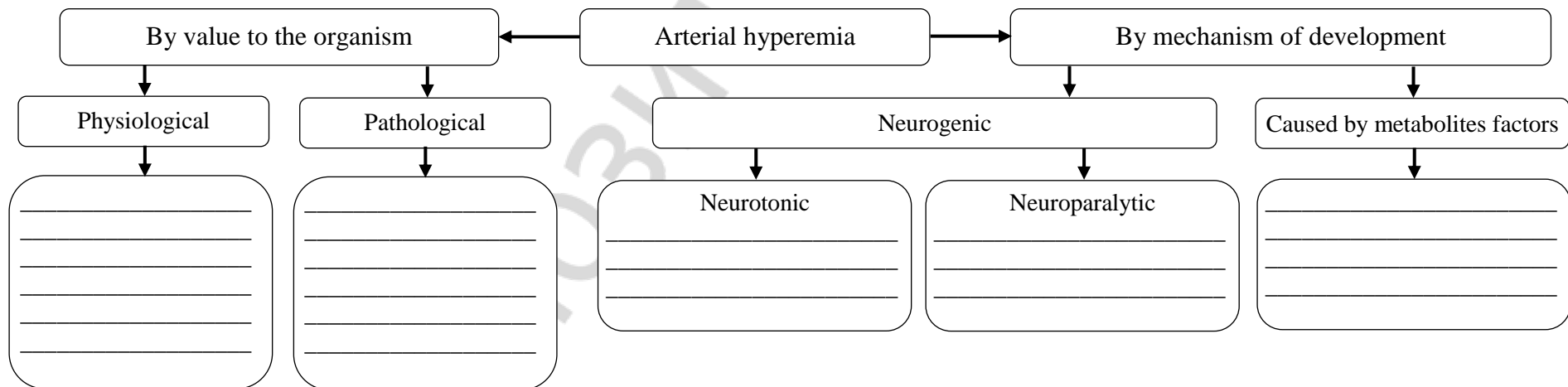
- to get acquainted with conditions of experimental work, to take part in accomplishment of experiments;
- to analyze experimental data, to present them in figures, to formulate conclusions, to give them a written substantiation;
- solving situational tasks.

#### 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.	

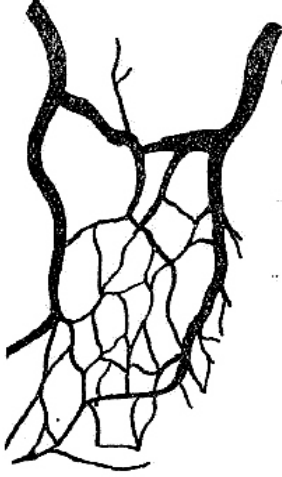
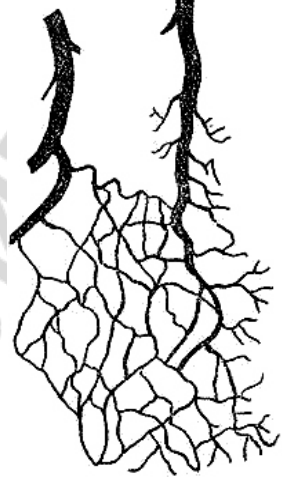
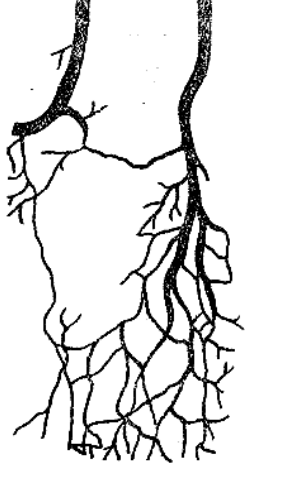
10. List the factors that determine the outcome of acute ischemia:

- |          |          |
|----------|----------|
| 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.

Vasodilators: \_\_\_\_\_

Vasoconstrictors: \_\_\_\_\_

## 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.
3. *Kumar, V. Robbins Basic Pathology* / 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. 8th 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:*

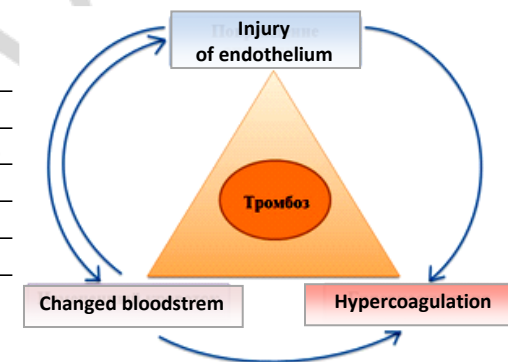
- 1) \_\_\_\_\_ (composition: *predominantly* \_\_\_\_\_);
- 2) \_\_\_\_\_ (composition: *predominantly* \_\_\_\_\_);
- 3) \_\_\_\_\_ (composition: *predominantly* \_\_\_\_\_).

➤ *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) \_\_\_\_\_

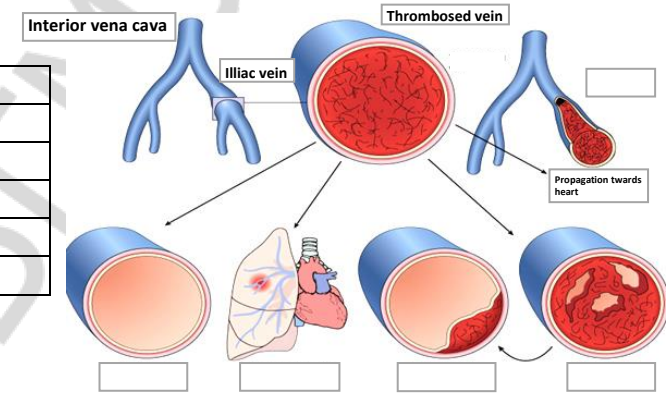


10. Fill in the Table.

Phase	Cellular	Coagulation
The essence and stages	<p style="text-align: center;"><i>Fill ___ with arrows (↓, ↑):</i></p> <p style="text-align: center;">___ 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</p> <p><b>Adhesion</b> — is _____</p> <p><b>Aggregation</b> — is _____</p> <p><b>Agglutination</b> — is _____</p>	<p>With the breakdown of platelets, enzymes (activators of the coagulation process) are released. Platelet coagulation factors (thromboplastin) enter to the plasma.</p> <div style="text-align: center;"> <pre> graph TD     A[ ] --&gt; B[ ]     A --&gt; C[ ]     B --&gt; D[ ]     C --&gt; E[ ]                     </pre> <p>I</p> <p>II</p> </div> <p>Under the influence of thrombastenin (retractozyme), thrombus retraction (compaction, the formation of a dense fibrin clot) occurs</p> <div style="text-align: center;"> <p><i>Fibrin</i></p> </div>
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:

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
4. \_\_\_\_\_



Kinds and their characteristic:

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
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**

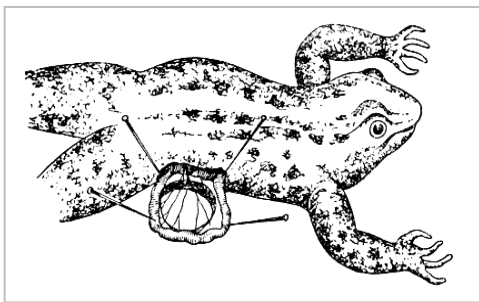
<b>Vascular region-source of thromboembolism</b>	<b>Veins of lower extremities, small pelvis organs, right heart chambers</b>	<b>Veins of lower extremities, small pelvis organs, right heart chambers</b>	<b>Veins of lower extremities, small pelvis organs, right heart chambers</b>
The vascular region exposed 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 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 Pathology* / 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 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:**

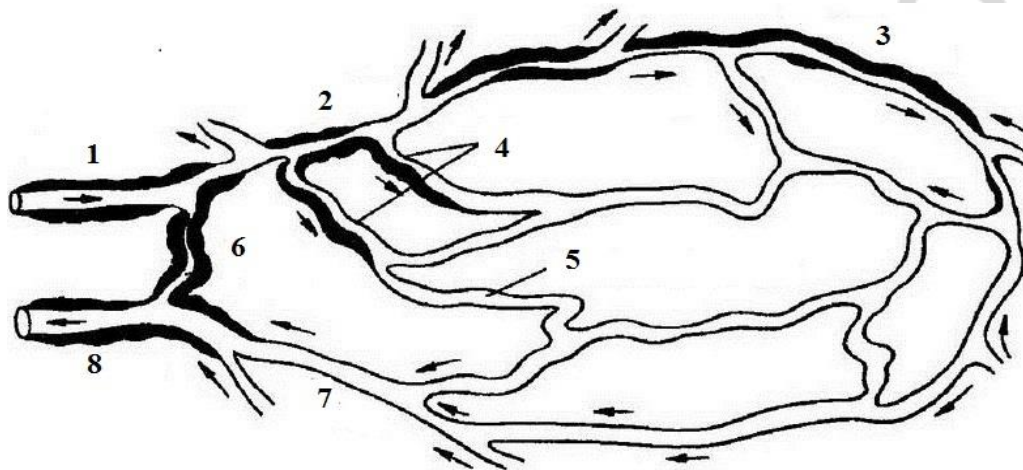
- to study the reasons, developmental mechanisms and consequences of typical impairments of the microcirculation presented in educational materials to the lesson;
- control test on the section “Typical impairments of peripheral blood circulation and microcirculation”.

### PART 1. WORK WITH TRAINING MATERIALS

1. Give the definition of “*microcirculation*”:

---

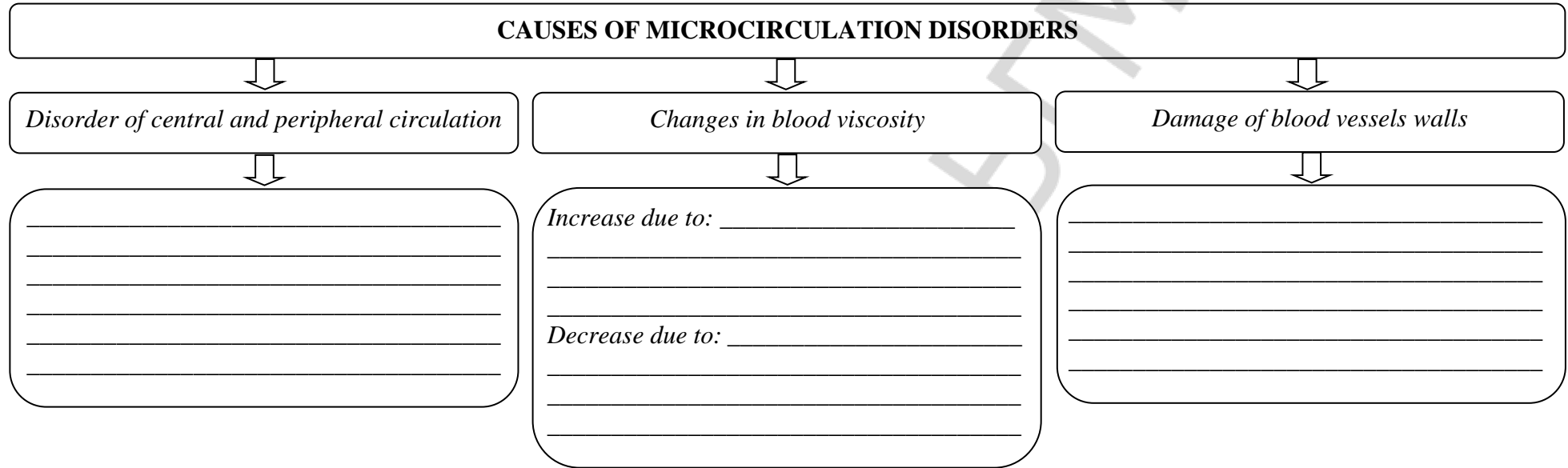
2. Identify the main components of the microcirculatory unit:



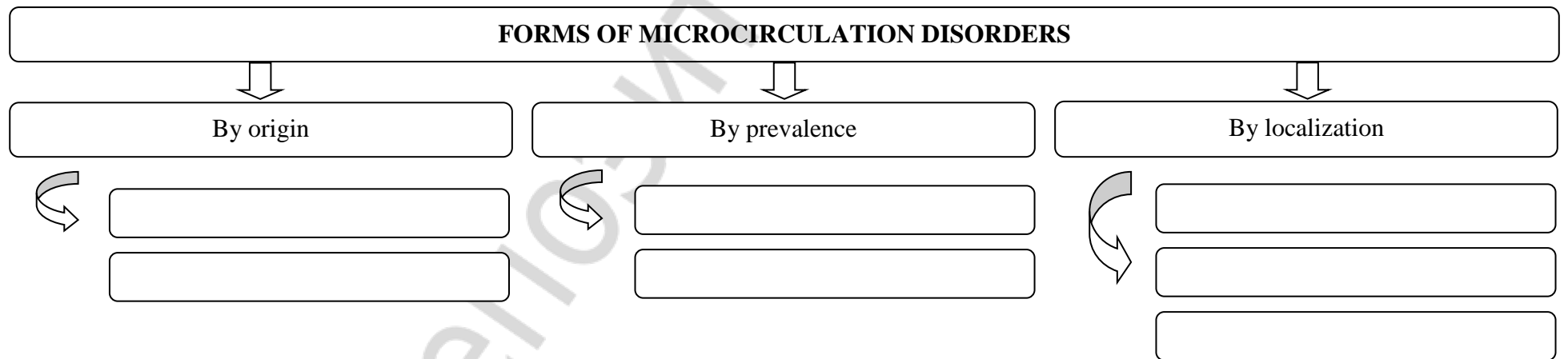
- 1 — \_\_\_\_\_
- 2 — \_\_\_\_\_
- 3 — \_\_\_\_\_
- 4 — \_\_\_\_\_
- 5 — \_\_\_\_\_
- 6 — \_\_\_\_\_
- 7 — \_\_\_\_\_
- 8 — \_\_\_\_\_

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

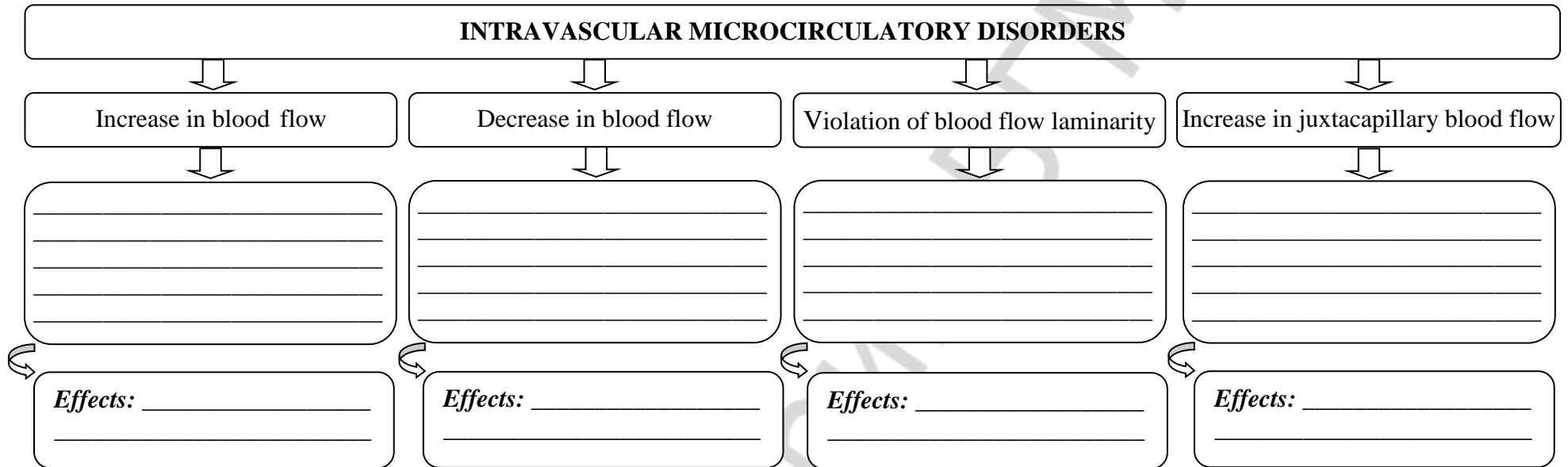
3. Fill in the Scheme:



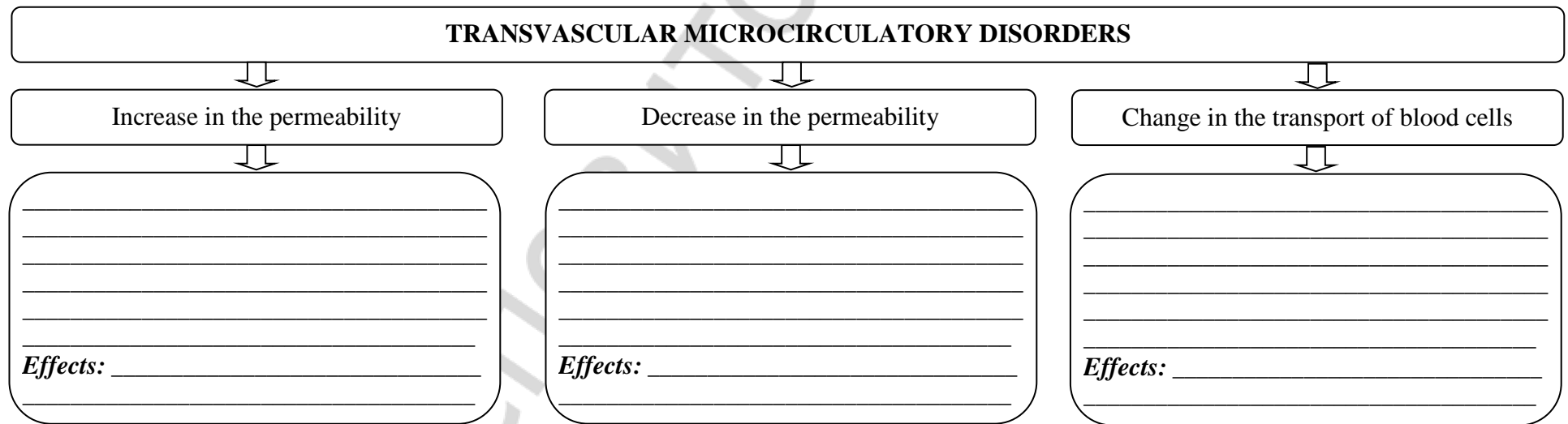
4. Fill in the Scheme:



5. Fill in the Scheme:

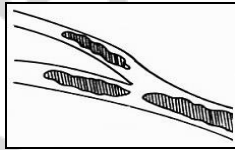
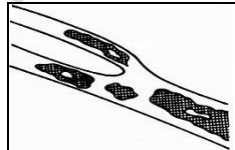
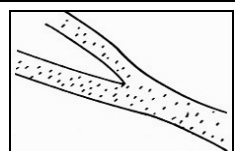
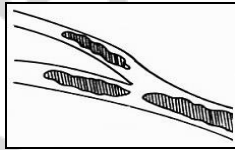
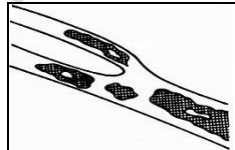
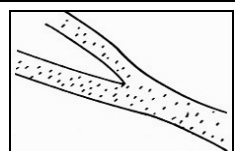
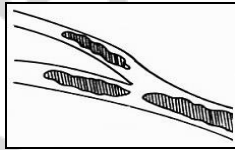
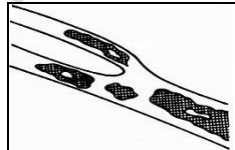
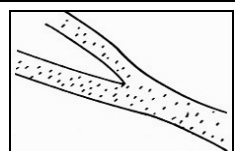


6. Fill in the Scheme:

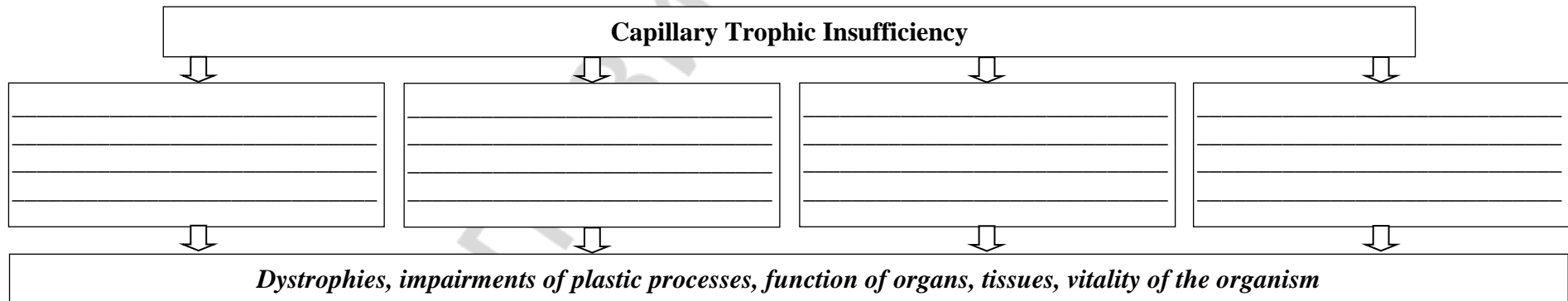




10. Fill in the Scheme:

Give the definition of <i>sludge</i> _____ _____ _____ _____ Pathogenesis of <i>sludge</i> : _____ _____ _____ _____ The consequences of <i>sludge</i> : _____ _____ _____ _____ _____	<b>Types of sludge</b>									
	By reversibility	_____ _____								
	Depending on the features of aggregates:	<table border="1" style="width: 100%;"> <thead> <tr> <th style="width: 30%;">Kind</th> <th style="width: 70%;">Characteristic</th> </tr> </thead> <tbody> <tr> <td>1) _____ </td> <td>                     _____                      _____                      _____                 </td> </tr> <tr> <td>2) _____ </td> <td>                     _____                      _____                      _____                 </td> </tr> <tr> <td>3) _____ </td> <td>                     _____                      _____                      _____                 </td> </tr> </tbody> </table>	Kind	Characteristic	1) _____ 	_____ _____ _____	2) _____ 	_____ _____ _____	3) _____ 	_____ _____ _____
	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.
3. *Kumar, V. Robbins Basic Pathology* / 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 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:

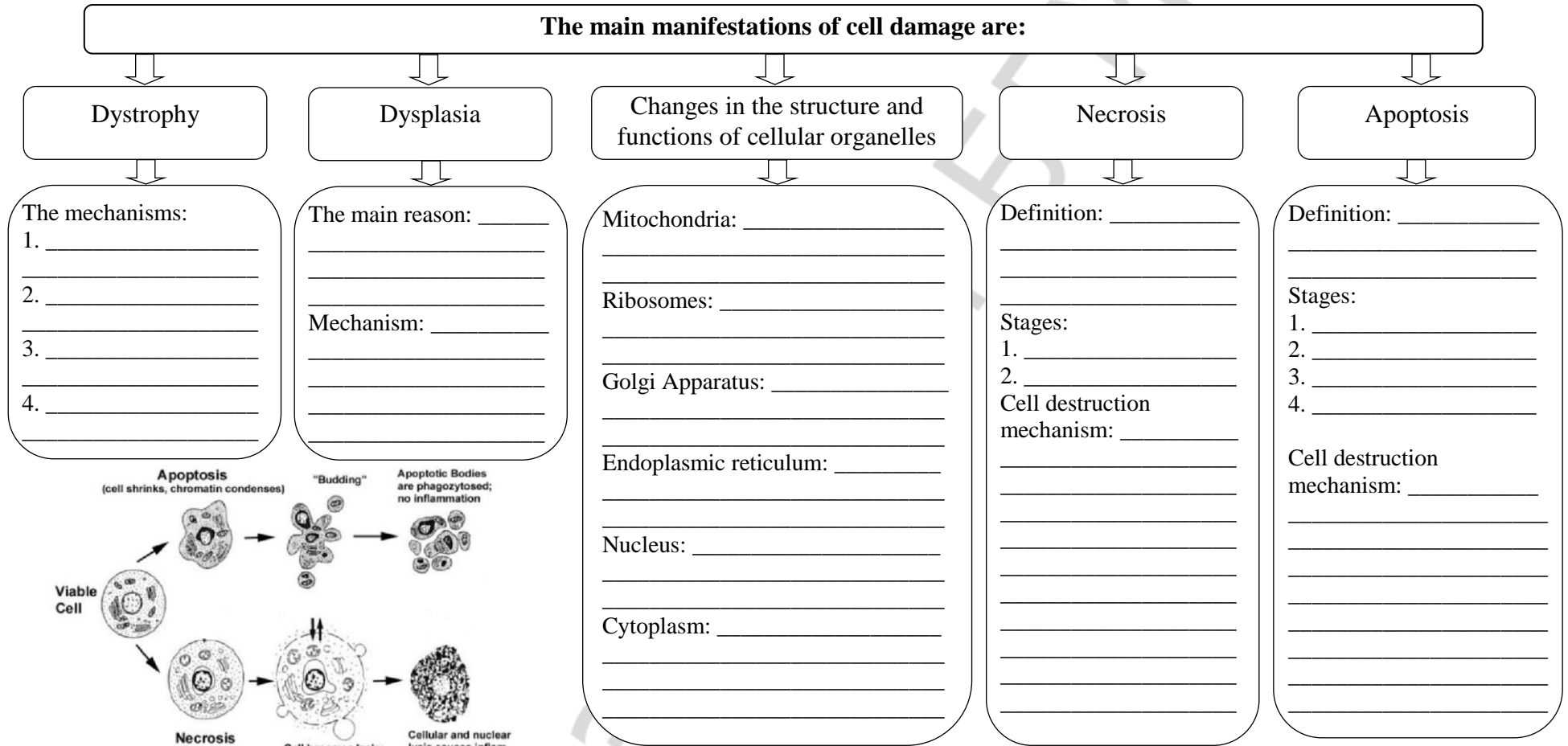
**Pathogenic cell injury variants**

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





4. Fill in the Scheme:



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) _____	<p>The diagram shows three stages of cellular adaptation. At the top, 'Normal Cells' are shown as four uniform cells on a 'Basement membrane', each with a 'Nucleus'. A red arrow labeled 'ADAPTIVE CELL CHANGES' points down to 'Hypertrophy', where the cells are larger. A second red arrow points down to 'Hyperplasia', where the number of cells has increased to six, while the individual cells are smaller than in the hypertrophy stage.</p>		
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 Pathology* / 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.
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 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):*

<b>by chemical structure</b>	➤ biogenic amines:
	➤ polypeptides:
	➤ proteins:
	➤ derivatives of PUFAs:
<b>by origin</b>	➤ cellular:
	➤ humoral:
<b>by exit from the cells</b>	➤ non-cytotoxic release:
	➤ cytotoxic release:
<b>by velocity of their release from the cells</b>	➤ immediate action:
	➤ slow action:
<b>by character of action</b>	➤ 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 leukopoiesis	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.

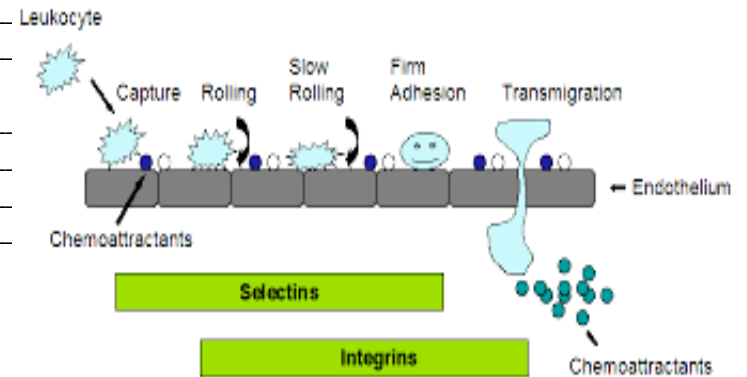
Indicate the ways of leukocyte emigration: (*granulocytes* — \_\_\_\_\_; *agranulocytes* — \_\_\_\_\_).

⇒ 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) \_\_\_\_\_;

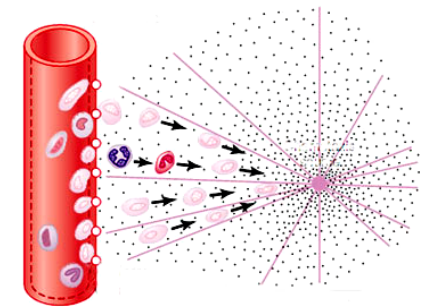
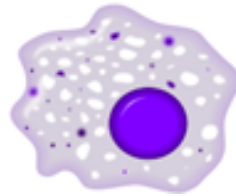
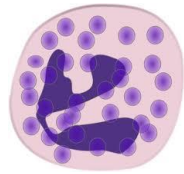


Fig. Leukocytes emigration to the inflammation focus

## 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.

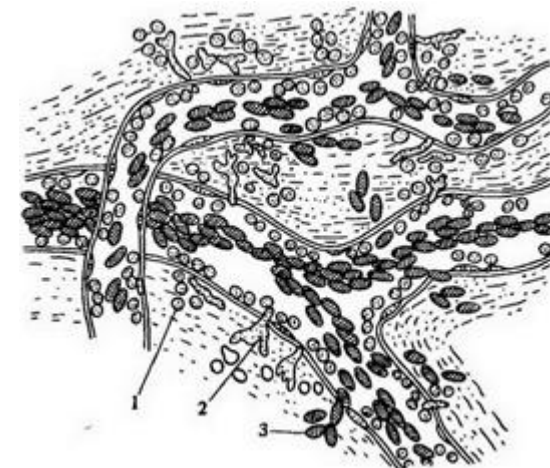


Fig. 1. The marginal standing of leukocytes in vessels





## 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 Pathology* / 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.
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 12. INFLAMMATION. PHAGOCYTE REACTION IN INFLAMMATION

Date: « \_\_\_ » \_\_\_\_\_ 20\_\_

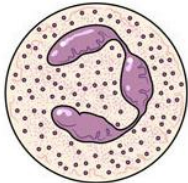
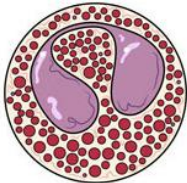
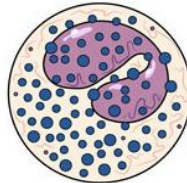
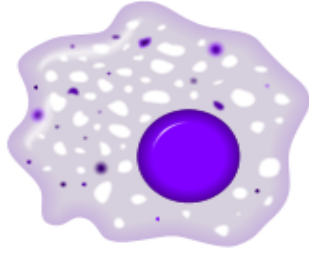
**The purpose of the Lesson:** to study phagocytosis as a protective reaction of the organism, to discuss phagocytosis stages during inflammation. To characterize the significance of inflammation as a response of the whole organism, to study the effect of the nervous system, hormonal and humoral factors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

<p style="text-align: center;"><b>Microphages Granulocytes</b></p> <div style="display: flex; justify-content: space-around; text-align: center;"> <div data-bbox="257 1037 443 1252"> <p>Neutrophil</p>  </div> <div data-bbox="555 1037 741 1252"> <p>Eosinophil</p>  </div> <div data-bbox="869 1037 1055 1252"> <p>Basophil</p>  </div> </div>	<p style="text-align: center;"><b>Macrophages (blood monocytes → tissue macrophages)</b></p> <div style="text-align: center;">  </div>
<p><b>Objects of phagocytosis:</b> _____          _____</p>	<p><b>Objects of phagocytosis:</b> _____          _____</p>



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

In 15 min about 1.0 ml of exudate with bird's erythrocytes is taken out by a syringe from the abdominal cavity of a 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:

<i>Phase of approaching</i>	<i>Phase of recognition and adherence</i>	<i>Phase of engulfment</i>	<i>Phase of digestion</i>

**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.
3. *Kumar, V. Robbins Basic Pathology* / 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.
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 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*” — \_\_\_\_\_

---



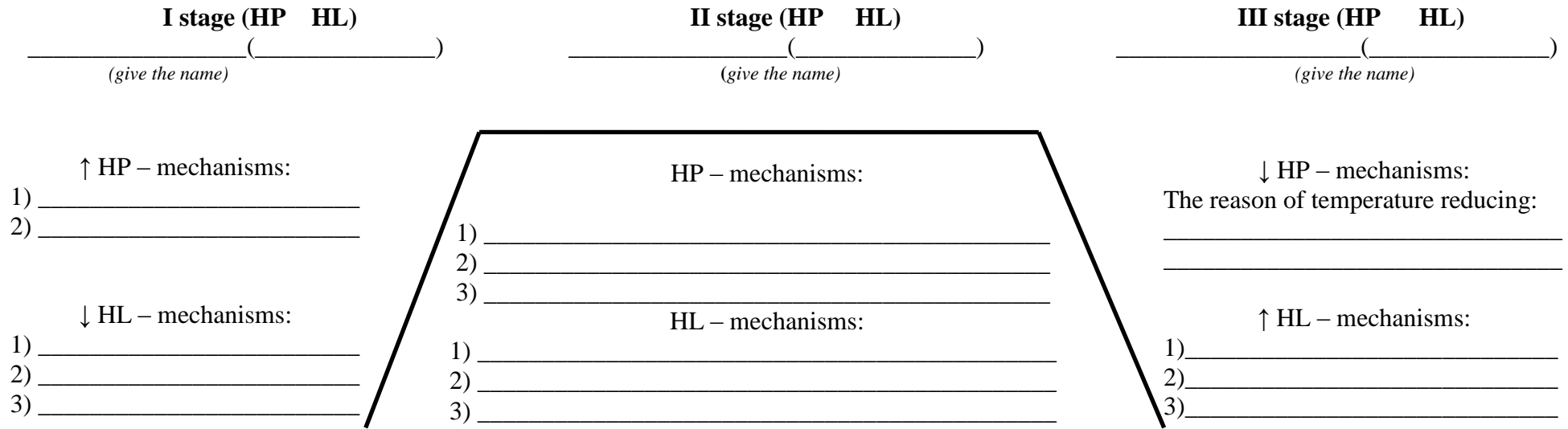
---

3. Fill in the Table:

**Classification of pyrogens**

Primary ( <i>etiological</i> )		Secondary ( <i>pathogenic</i> )	
Exogenous		Endogenous	
Infectious	Non-Infectious		
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

4. Give the characteristic of the stages of fever:



5. Заполните таблицу:

**Metabolic and organ function changes at fever**

<b>Protein exchange</b>	<hr/> <hr/> <hr/>
<b>Carbohydrate and fat metabolism</b>	<hr/> <hr/> <hr/>
<b>Water-electrolyte exchange</b>	1 <sup>st</sup> stage: <hr/> 2 <sup>nd</sup> stage: <hr/> 3 <sup>d</sup> stage: <hr/>

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

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**

<b>Mechanism</b>	<b>Centrogenic</b>	<b>Psychogenic</b>	<b>Reflexogenic</b>
<b>Cause</b>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>
<b>Mechanism</b>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>
<b>Examples</b>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>	<hr/> <hr/> <hr/>

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.

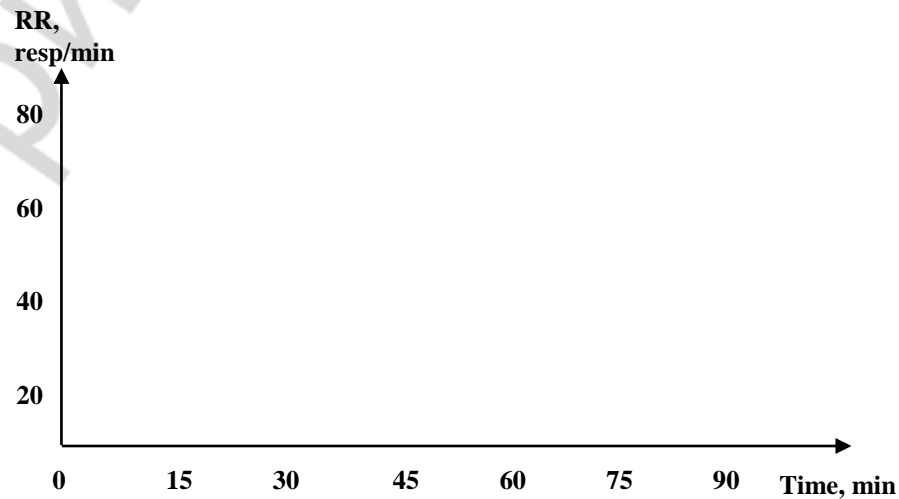
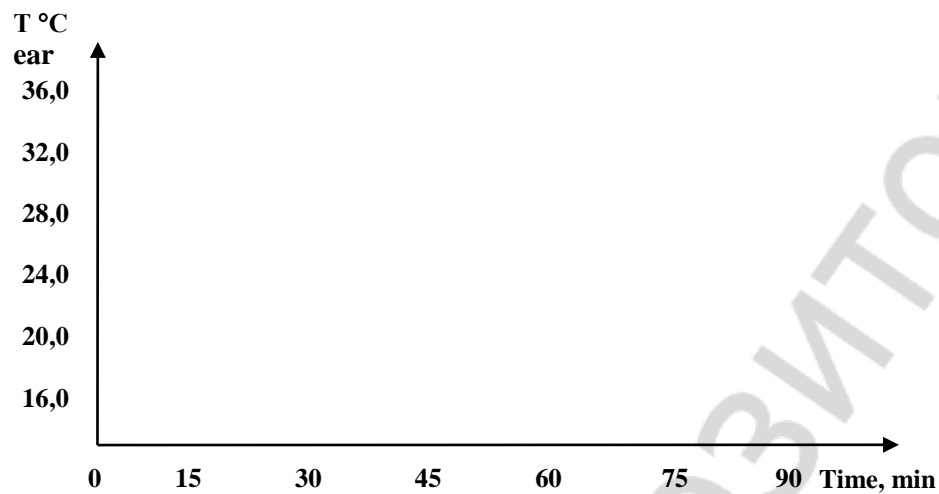
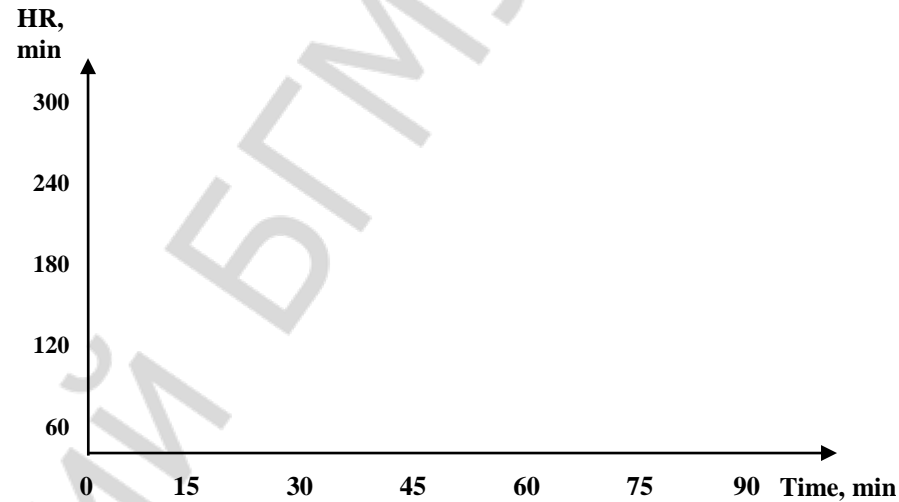
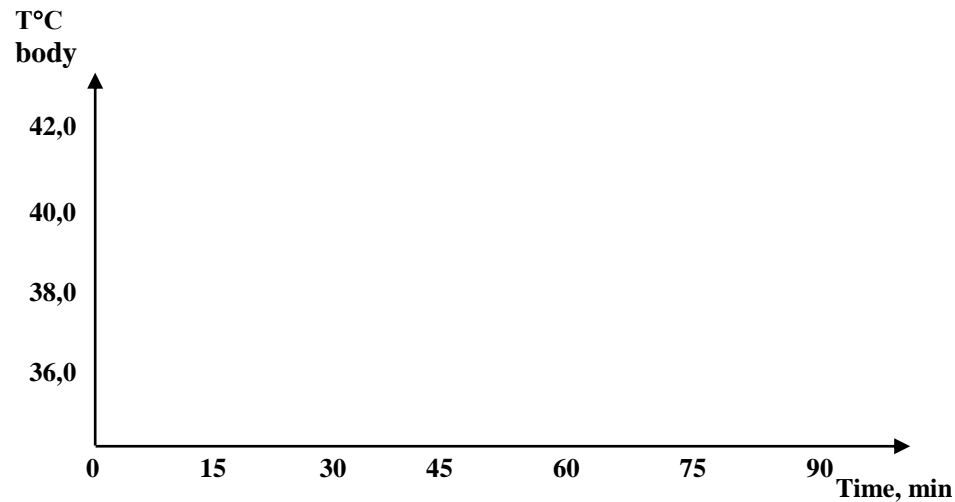
To induce experimental fever, we use endotoxin — bacterial liposaccharid pyrogenal.

**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.	<b>Tested:</b> 0 min	38.8	33	60	220	Ears are pale, cold, vessels are narrowed
	– 15 min	39.2	24.0	72	260	
	– 30 min	39.6	24.0	30	270	
	– 45 min	39.9	27.0	46	280	
	– 60 min	40.2	28.0	58	280	
	– 75 min	40.4	28.0	60	290	
	– 90 min	40.4	30.0	70	280	
2.	<b>Control:</b> 0 min	39.2	31.0	68	220	Ears are pink, warm, vessels are moderately dilated
	– 15 min	39.2	30.0	70	242	
	– 30 min	39.0	30.0	72	236	
	– 45 min	39.0	32.0	72	230	
	– 60 min	39.2	32.0	72	230	
	– 75 min	39.3	31.0	70	220	
	– 90 min	39.2	31.0	70	220	

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

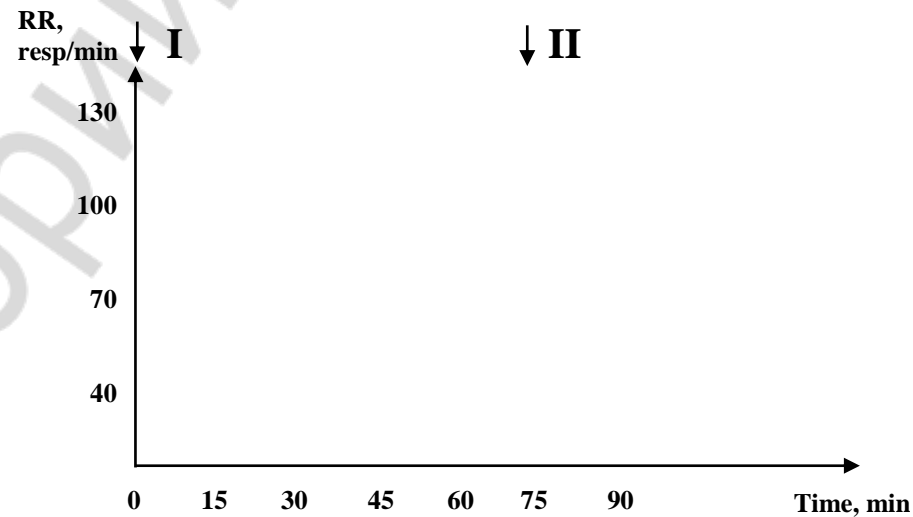
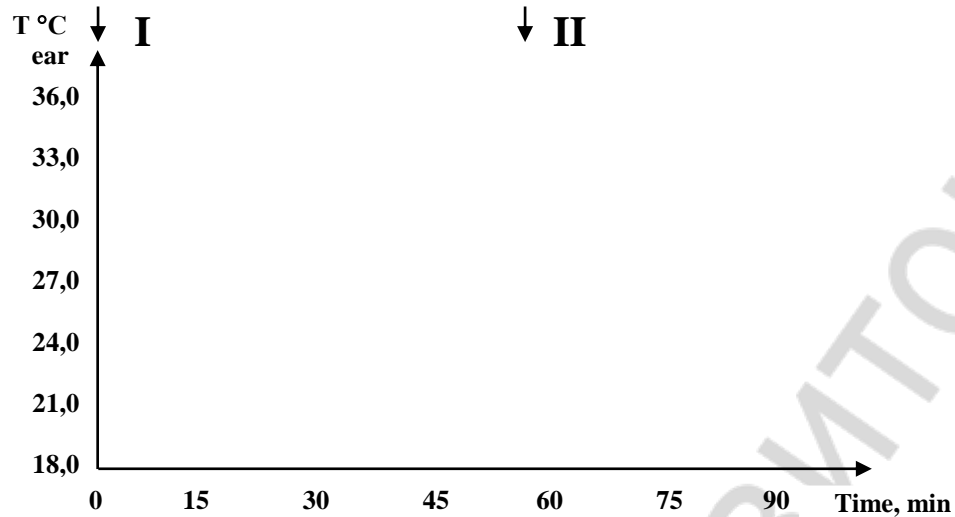
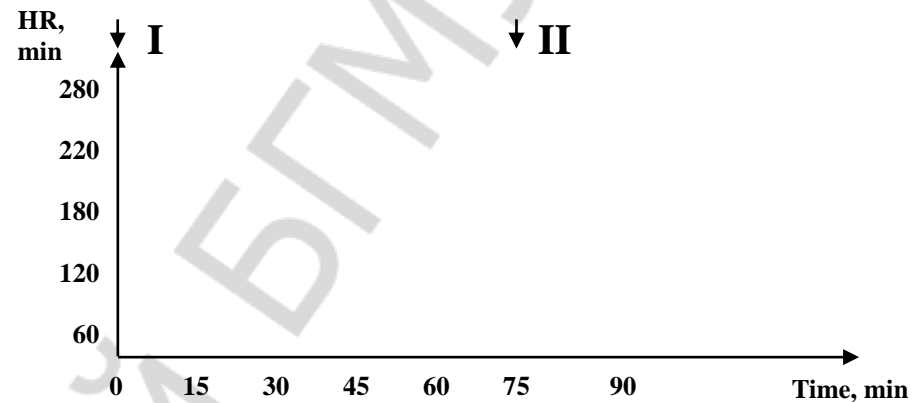
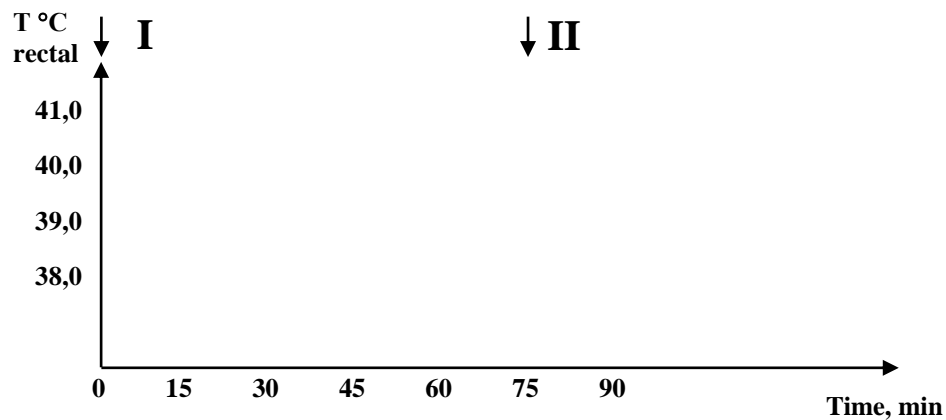
The experiment is performed on two adult rabbits of one sex with body weight of 2.0–2.5 kg. **One (tested) is injected pyrogenal (0.5 mcg/kg) on 0.5 ml of physiological solution into the marginal vein of the ear, to the other (control) — 0.5 ml of physiological solution.** Immediately after injections the animals are placed in **the thermo-chamber 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 thermo-chambers and measurement of body temperature, respiratory rate and heart rate are continued every 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. <i>Tested:</i>	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. <i>Control:</i>	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																							

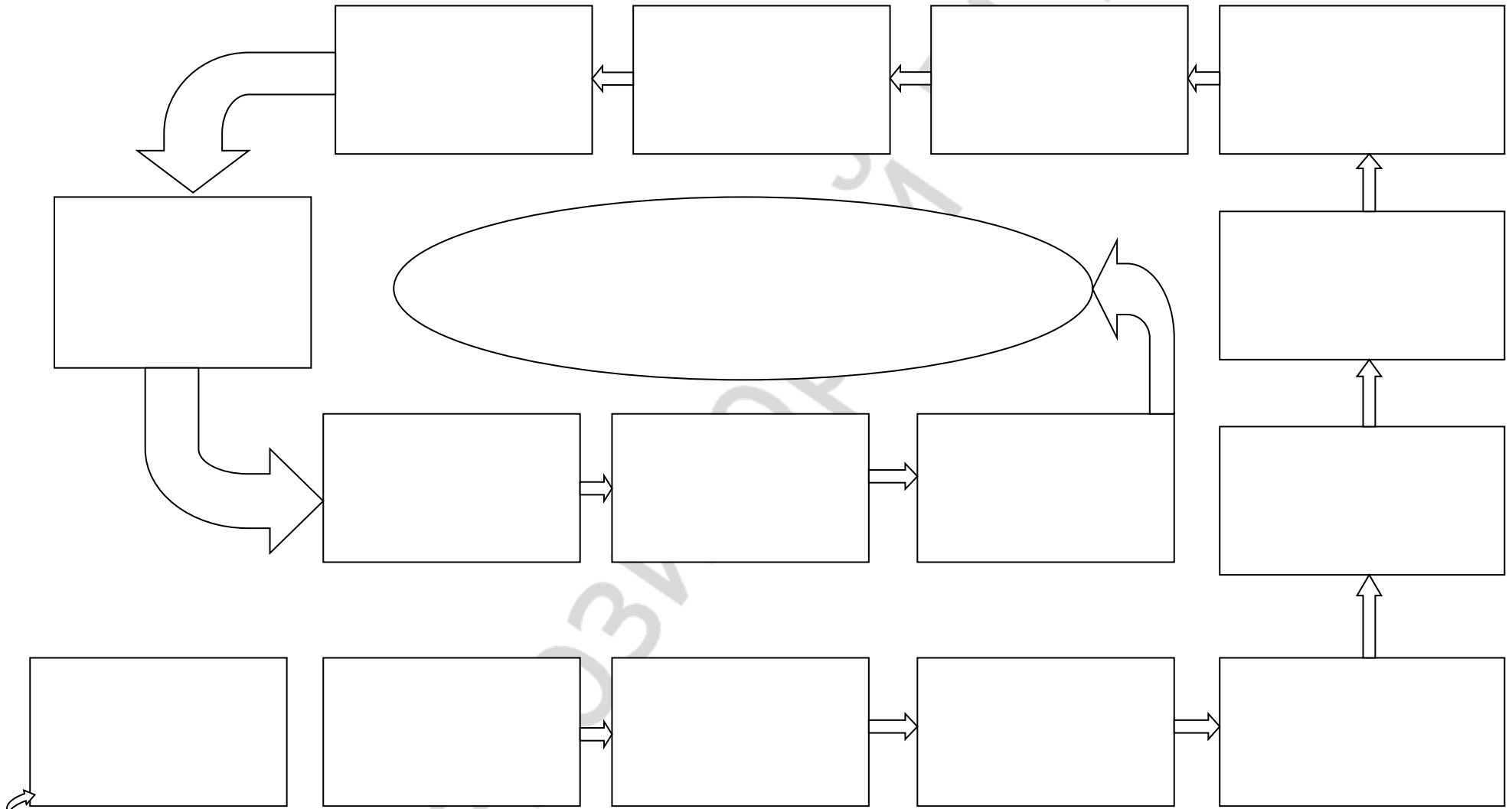
Temperature curve														Fever name	Daily temperature variations	Occurs in diseases							
1		2		3		4		5		6		7					8		9		10		
m	e	m	e	m	e	m	e	m	e	m	e	m	e				m	e	m	e	m	e	
41																					Remittent		
40																							
39																							
38																							
37																							
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 Pathology* / 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 14. TYPICAL VIOLATIONS 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**

<b>Blood parameters</b>	<b>Value</b>
pH	
p <sub>a</sub> CO <sub>2</sub>	
HCO <sub>3</sub> <sup>-</sup>	
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
<b>Blood plasma electrolytes (mmol/L)</b>	
Na <sup>+</sup>	135–145
K <sup>+</sup>	3.5–5.0
Ca <sup>2+</sup>	2.23–2.57
Cl <sup>-</sup>	96–108
<b>Additional ABB indicators</b>	
Titrateable 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**

<b>Chemical buffer systems</b>	<b>The essence of the compensatory system</b>
1)	
2)	
3)	
4)	

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

**Long-term (physiological) compensation mechanisms**

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

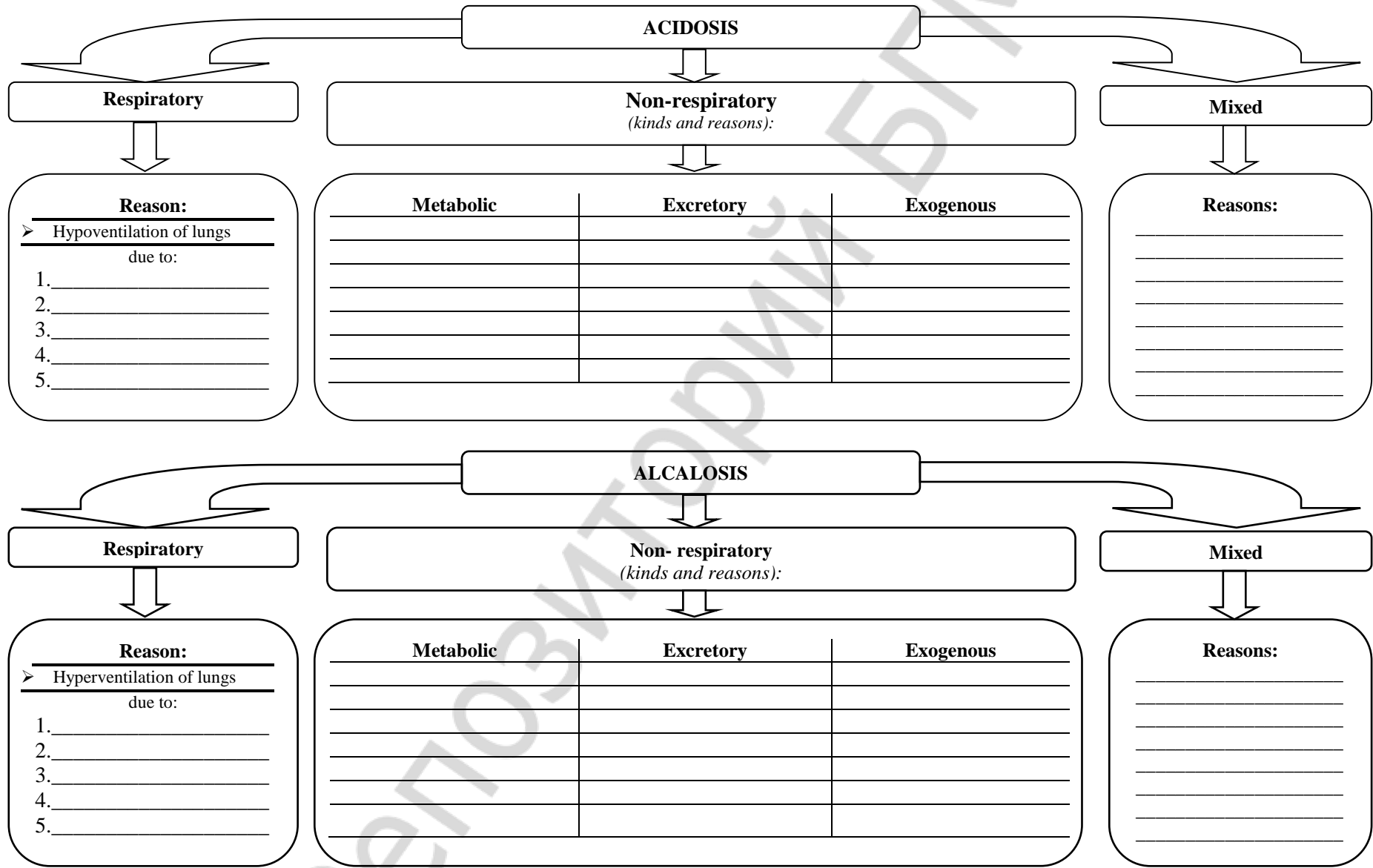
9. Fill in the Tables:

**ABB disorders classification**

<b>Criterion</b>	<b>Kinds</b>
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 alkalosis	acute		
	chronic		
4. Non-Respiratory alkalosis	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
pH <sub>arterial blood</sub>	7.46	
p <sub>a</sub> CO <sub>2</sub>	32 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	22 mmol/l	
BE	-1 mmol/l	
TK urine	20	
pH urine	6.0	
Bicarbonates in the urine	-	

In a week	Indicator characteristic
7.41	
30 mm Hg	
17 mmol/l	
-6 mmol/l	
0	
7.2	
+	

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 <sub>arterial blood</sub>	7.37	
p <sub>a</sub> CO <sub>2</sub>	56 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	32 mmol/l	
BE	+7.5 mmol/l	
Na <sup>+</sup>	142 mmol/l	
K <sup>+</sup>	88 mmol/l	
Cl <sup>-</sup>	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 <sub>arterial blood</sub>	6.95	
p <sub>a</sub> CO <sub>2</sub>	20 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	5.5 mmol/l	
BE	-20 mmol/l	
SB	4 mmol/l	
Ketone bodies in blood plasma	10 mmol/l	
K <sup>+</sup>	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 <sub>arterial blood</sub>	7.27	
p <sub>a</sub> CO <sub>2</sub>	27 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	15.5 mmol/l	
BE	-10 mmol/l	
SB	15 mmol/l	
Concentration of trace anions in plasma	15 mmol/l	
K <sup>+</sup>	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 <sub>arterial blood</sub>	7.0	
p <sub>a</sub> CO <sub>2</sub>	80 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.22	
p <sub>a</sub> CO <sub>2</sub>	55 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.29	
p <sub>a</sub> CO <sub>2</sub>	56 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.26	
p <sub>a</sub> CO <sub>2</sub>	28 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 electrilyte balance the following data are received.

Parameters	Value	Characteristics
pH <sub>arterial blood</sub>	7.15	
p <sub>a</sub> CO <sub>2</sub>	25 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sup>+</sup> 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
pH <sub>arterial blood</sub>	7.26	
p <sub>a</sub> CO <sub>2</sub>	62 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	26 mmol/l	
BB	43 mmol/l	
SB	22 mmol/l	
BE	1 mmol/l	

In a week	Characteristics
7.46	
30 mm Hg	
18 mmol/l	
40 mmol/l	
20 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 <sub>arterial blood</sub>	7.47	
p <sub>a</sub> CO <sub>2</sub>	29 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.39	
p <sub>a</sub> CO <sub>2</sub>	27 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 relieve it he constantly takes baking soda.

The investigation of ABB parameters revealed:

Parameters	Value	Characteristics
pH <sub>arterial blood</sub>	7.5	
p <sub>a</sub> CO <sub>2</sub>	43 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.29	
p <sub>a</sub> CO <sub>2</sub>	30 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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<sub>2</sub>?**

**№ 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 <sub>arterial blood</sub>	7.55	
p <sub>a</sub> CO <sub>2</sub>	60 mm Hg	
HCO <sub>3</sub> <sup>-</sup>	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 <sub>arterial blood</sub>	7.28		7.34		7.44	
p <sub>a</sub> CO <sub>2</sub>	20 mm Hg		36 mm Hg		49 mm Hg	
BB	31 mmol/l		39 mmol/l		51 mmol/l	
HCO <sub>3</sub> <sup>-</sup>	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 2<sup>nd</sup> day: \_\_\_\_\_



c) on the 3<sup>rd</sup> 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 Pathology* / 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 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 _____		
<b>Kinds</b>	<b>Reasons</b>	<b>Manifestations</b>
Isoosmolar	- _____ - _____ - _____ - _____ -	- _____ - _____ - _____ - _____ -
Hypoosmolar	- _____ - _____ - _____ - _____ -	- _____ - _____ - _____ - _____ -

Hyperosmolar	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>
<b>Hyperhydration is</b> _____		
<b>Kinds</b>	<b>Reasons</b>	<b>Manifestations</b>
Isoosmolar	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>
Hypoosmolar	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>
Hyperosmolar	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>	<ul style="list-style-type: none"> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> <li>- _____</li> </ul>

3. Define the concept of “*edema*”: \_\_\_\_\_

4. Put down the classification of edema:

<b>Criteria</b>	<b>Kinds of edemas</b>	<b>Definition</b>	
<b><i>By localization</i></b>	anasarca		
	dropsy		
	ascites		
	hydrothorax		
	hydropericardium		
	hydrocele		
	hydrocephalus		
		<b>Reason</b>	<b>Mechanism</b>
<b><i>By pathogenesis</i></b>	hydrodynamic		
	lymphogenous		
	oncotic		
	osmotic		
	membranogenic		

		Reason	Mechanism
<i>By etiology</i>	cardial		
	renal		
	endocrine		
	cachexic		
	inflammatory		
	allergic		
	toxic		
<b>Characteristic</b>			
<i>By rate of development</i>	fulminant		
	acute		
	chronic		
<b>Characteristic</b>			
<i>By spreading</i>	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 vertebrae. After death of the animals open the thorax of both rats, apply a ligature at the trachea, take out the lungs, weigh them and do a pathomorphological examination.

Results of the experiment.

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

Type of effect	Respiratory rate (resp/min)	General state	Pathomorphological changes in lungs
i/p injection of 0.1 % Adrenaline solution			
– Initial	120	Normal	Weight of the lungs — 5.8 g, pulmonary weight factor — 0.029. Foamy liquid in the trachea. The lungs volume is enlarged, looks like marble, foamy discharge on the section
– 1 min	160	General excitation, impairment of motor coordination	
– 2 min	Rare deep respiration	Foamy discharge from the mouth	
– 3 min	Terminal respiration	– // – // – // –	
– 4 min	Respiratory arrest	Death of the animal	
i/p injection of 0.9 % solution of NaCl			
– Initial	130	General state without visible changes	Weight of the lungs — 1.2 g, pulmonary weight factor — 0.006. The trachea is freely passable. Lungs are collapsed, of light pink color
– 1 min	–		
– 2 min	–		
– 4 min	–		

#### Conclusion:

Explain the development mechanism of 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 vertebra. 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
<b>Unnarcotized rat + injection of NH<sub>4</sub>Cl</b>	– Initial	128	Normal	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
	– 15 min	150	Impairment of movements coordination	
	– 30 min	20	The rat is motionless, is lying on one side	
	– 45 min	Rare deep respiration	Neck and mouth muscles take part in respiration	
	– 55 min	Terminal respiration Respiratory arrest	Foamy discharge from the mouth Death of the animal	
<b>Hexenal narcosis + injection of NH<sub>4</sub>Cl</b>	Initial	100	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
	15 min	103		
	30 min	102		
	55 min	102		
		Quiet, rhythmic respiration		

**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 Pathology* / 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. 8th 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 severity	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**

<b>Exogenous</b>		<b>Endogenous</b>	
<b>Types</b>	<b>Reasons and pathogenesis</b>	<b>Types</b>	<b>Reasons and pathogenesis</b>
<b>Hypoxic</b>		1)	
1)		2)	
2)		3)	
<b>Hyperoxic</b>		4)	
1)		5)	
2)		6)	
		7)	

Table 3

**Stages of Hypoxia**

<b>Stages</b>	<b>Manifestations</b>
Hidden	
Compensated	
Expressed	
Severe not compensated	
Terminal	

Table 4

**Mechanisms for short-term adaptation**

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

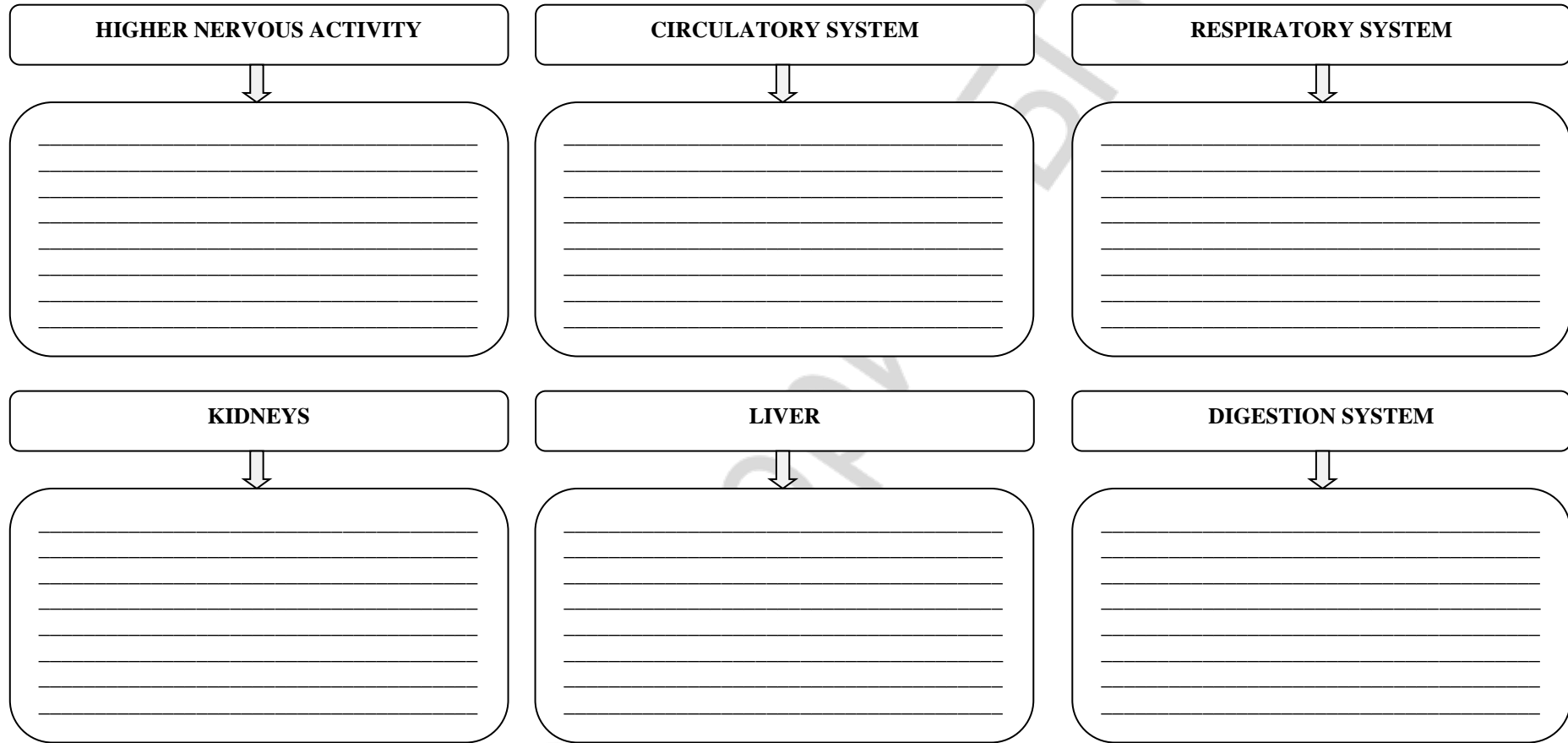
Table 5

**Mechanisms for long-term adaptation**

<b>Organ and systems</b>		<b>Effects</b>		<b>Mechanisms of effects</b>
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,  $t\ 37\ ^\circ\text{C}$  (the temperature of water corresponds to the body temperature). At pumping out the air from under the bell there occurs stretching of the rubber glove and at the "altitude" corresponding to 19 kms — "boiling" of water in the glass — a model of decompression disease (expansion of gases in cavities, gas embolism and tissue emphysema).

**Answer the questions:**

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

a) stretching of a rubber glove: \_\_\_\_\_

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	$P_{AO_2}$	$P_aO_2$	$P_vO_2$	$\Delta a-vO_2$	HbO <sub>2</sub> 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 <sub>2</sub> 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 Pathology* / V. Kumar, A. K. Abbas, J. C. Aster. 10th ed. Canada : Elsevier, 2018. 952 p.

### *Additional*

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.
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 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: \_\_\_\_\_

\_\_\_\_\_



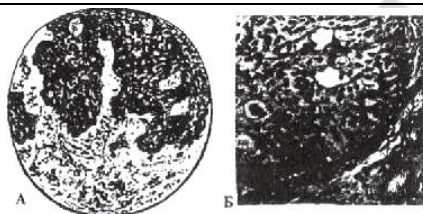
8. Specify the types of human malignant neoplasms the viral etiology of which is proved: \_\_\_\_\_

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

10. Fill in the Table.

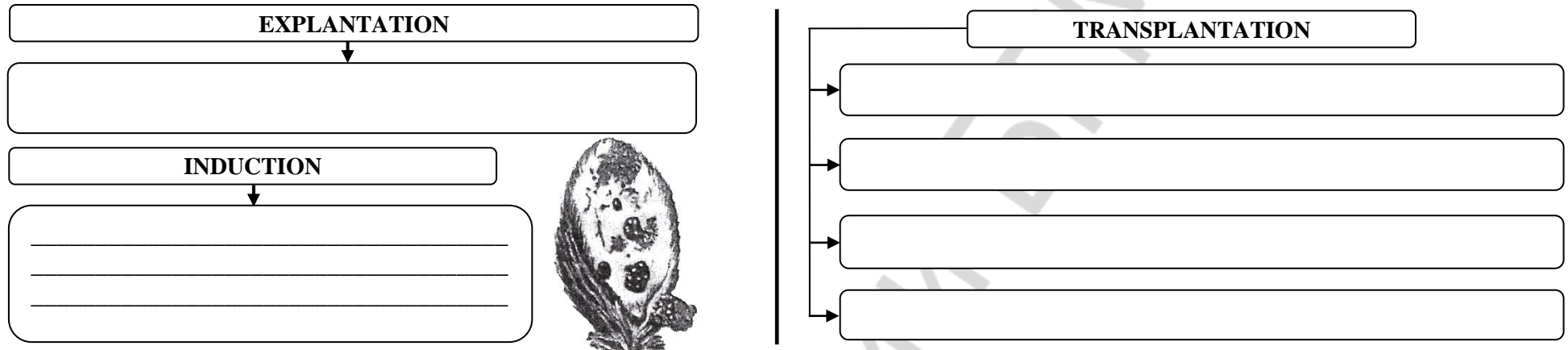
**Biological peculiarities of malignant tumors**

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 <i>(indicate specific tumor antigenic markers)</i>		
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:

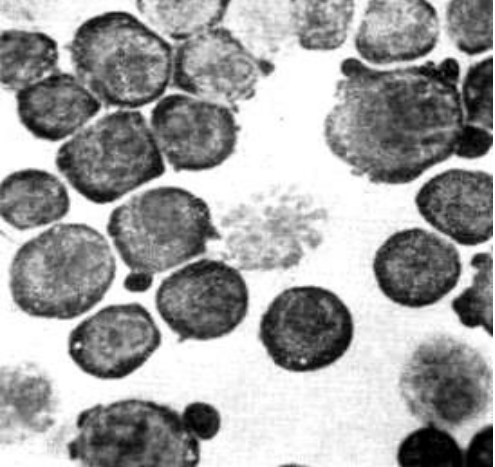
	<p>1 — gigantic multinuclear cells;                  2 — cell with 3–4 polar pathologic mitoses;                  3 — cells with stuck chromosomes in pathologic mitosis;                  4 — cells with chromosomal bridges in pathologic mitosis</p>
--	---

*Fig. 1. Cells of the CaVe line*

### Studying a micropreparation of Ehrlich's ascite carcinoma

Take out some ascite liquid with a thin needle of a 5 mm syringe from a narcotized mouse with an intertwined ascite tumor of 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 ( $10 \times 90$ ).

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:

		<ul style="list-style-type: none"><li>1 — dwarfish cells;</li><li>2 — gigantic cells,</li><li>2a — gigantic multinuclear cells;</li><li>3 — irregular-shaped cells;</li><li>3a — cell with spherical cytoplasmic processes;</li><li>4 — dark cells with hyperchromous nuclei and sharp basophilic cytoplasm;</li><li>5 — large light cell with a clearly marked structure of nuclear chromatin;</li><li>6 — cellular mitosis;</li><li>7 — pathological mitosis;</li><li>8 — nuclear division without division of cytoplasm.</li></ul>
<p>Fig. 2. Cells of Ehrlich's Ascite Carcinoma:</p>		

#### Answer 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.
3. *Kumar, V. Robbins Basic Pathology* / 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 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 studying 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).

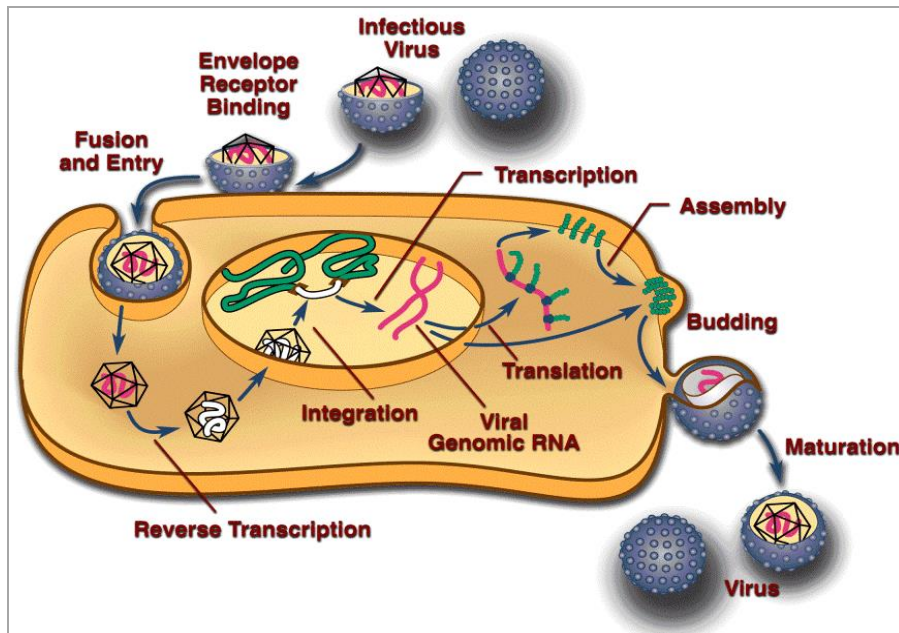
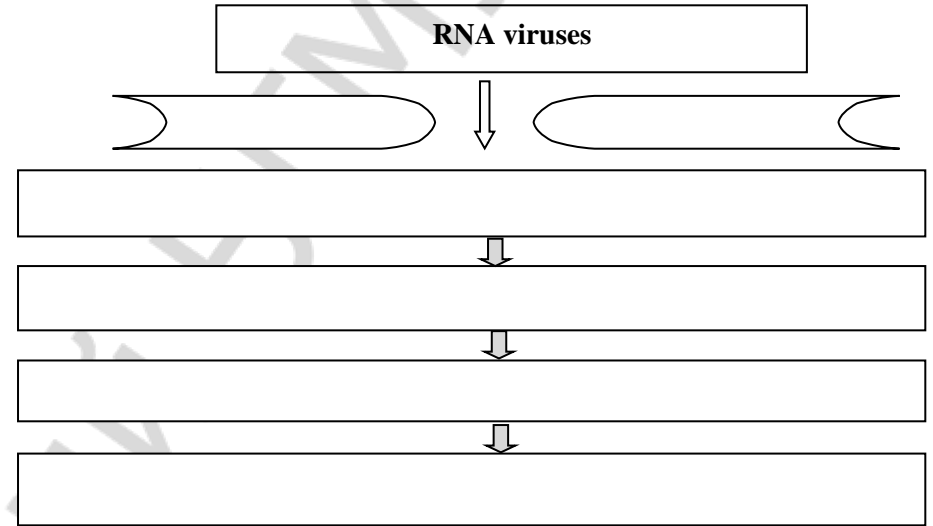
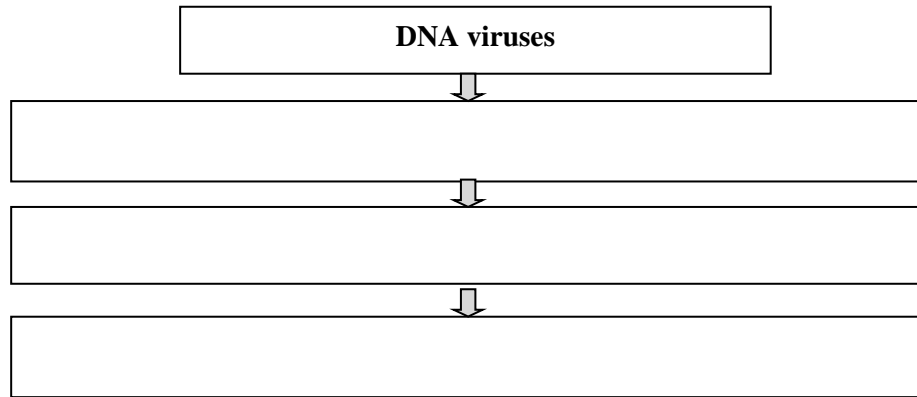
**PART 1. WORK WITH EDUCATIONAL MATERIALS**

1. Fill in the Table:

**Characterization of the basic theories of carcinogenesis**

<b>Theory</b>	<b>Essence of the concept</b>
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 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 host's 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"? — \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

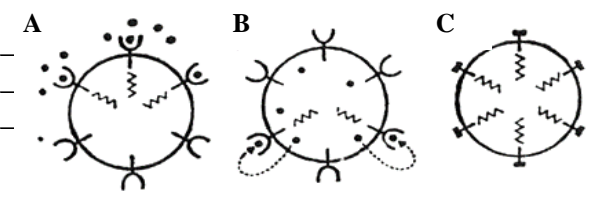
5. What functions do proteins, products of a proto-oncogen, perform? \_\_\_\_\_  
\_\_\_\_\_

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 — \_\_\_\_\_



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 “antineoplastic resistance” — \_\_\_\_\_

15. Fill in the Table:

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

16. Put down the definition of the term “precancerous conditions” — \_\_\_\_\_

17. Fill in the Table:

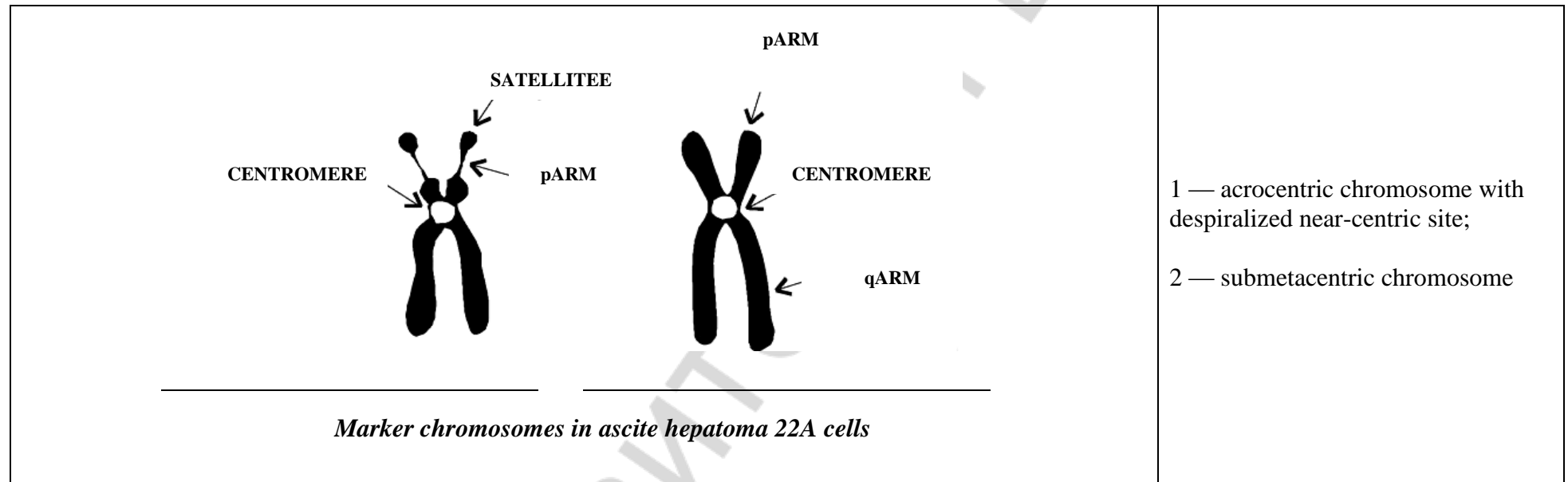
Type of precancerous condition	Probability of malignancy	Examples
Facultative		
Obligate		



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

In the inhomogeneous 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 paracentromer area 1–2 subcentromer ones.

Designate the corresponding chromosomes in the figure below:



### 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 Pathology* / 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 necessary to work out them in accordance with the applicable requirements).
2. Attendance at all lectures (if there are missing lessons, it is necessary 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:** \_\_\_\_\_

**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

List of abbreviations.....	3
SECTION I. GENERAL NOSOLOGY.....	4
Lesson 1. Introduction to the discipline “Pathological physiology”. Subject, tasks, methods of pathological physiology.....	4
Lesson 2. General questions of the doctrine of the disease. General etiology and pathogenesis.....	11
Lesson 3. Pathogenic effect of environmental factors on the human body. The effect of electric current on the body.....	22
Lesson 4. Pathogenic effect of environmental factors on the human body. The damaging effects of ionizing radiation to the body.....	29
Lesson 5. The role of reactivity, constitution and age in the development of pathology.....	36
Lesson 6. The role of heredity in the development of pathology.....	48
SECTION II. TYPICAL PATHOLOGICAL PROCESSES.....	61
Lesson 7. Pathophysiology of regional blood circulation and microcirculation. Arterial and venous hyperemia. Ischemia.....	61
Lesson 8. Pathophysiology of regional blood circulation and microcirculation. Thrombosis. Embolism. Stasis.....	67
Lesson 9. Pathophysiology of regional blood circulation and microcirculation. Microcirculatory Disorders.....	73
Lesson 10. Pathophysiology of the cell. Cell damage. Common damage mechanisms.....	79
Lesson 11. Inflammation. Vascular reaction at the focus of inflammation.....	84
Lesson 12. Inflammation. Phagocytic reaction in the focus of inflammation.....	91
Lesson 13. Pathology of thermoregulation. Fever.....	97
Lesson 14. Typical violation of metabolism. Acid-base balance impairments.....	110
Lesson 15. Typical metabolic disorders. Pathological physiology of water metabolism. Swelling and dropsy.....	122
Lesson 16. Hypoxia.....	129
Lesson 17. Pathophysiology of tissue growth. Tumors. Biological features of tumor growth. Methods of experimental reproduction of tumors. Etiology of tumors.....	136
Lesson 18. Pathophysiology of tissue growth. Tumors. The mechanisms of carcinogenesis. The relationship of the tumor and the organism. Principles for the treatment and prevention of tumors.....	142
Credit. Abstract defense.....	150
Appendix. Sample title page abstract.....	154

Учебное издание

**Висмонт Франтишек Иванович**  
**Жадан Светлана Анатольевна**  
**Чепелев Сергей Николаевич и др.**

## **ОБЩАЯ ПАТОФИЗИОЛОГИЯ**

## **GENERAL PATHOLOGICAL PHYSIOLOGY**

Рабочая тетрадь

На английском языке

Ответственный за выпуск Ф. И. Висмонт  
Переводчик С. А. Жадан  
Компьютерный набор С. А. Жадан  
Компьютерная вёрстка Н. М. Федорцовой

Подписано в печать 12.06.20. Формат 60×84/8. Бумага офсетная.

Ризография. Гарнитура «Times».

Усл. печ. л. 18,13. Уч.-изд. л. 7,0. Тираж 196 экз. Заказ 220.

Издатель и полиграфическое исполнение: учреждение образования  
«Белорусский государственный медицинский университет».

Свидетельство о государственной регистрации издателя, изготовителя,  
распространителя печатных изданий № 1/187 от 18.02.2014.

Ул. Ленинградская, 6, 220006, Минск.