

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ  
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ  
КАФЕДРА НОРМАЛЬНОЙ ФИЗИОЛОГИИ

**ОБЩАЯ ФИЗИОЛОГИЯ ВОЗБУДИМЫХ ТКАНЕЙ, ЧАСТНАЯ ФИЗИОЛОГИЯ  
КРОВИ, ЭНДОКРИННОЙ, НЕРВНОЙ И КОСТНОЙ СИСТЕМ**

**GENERAL PHYSIOLOGY OF EXCITABLE TISSUES, SPECIAL PHYSIOLOGY  
OF BLOOD, ENDOCRINE, NERVOUS, AND BONE SYSTEMS**

Практикум для студентов, обучающихся по специальности «Стоматология»

Под редакцией Ю. В. Гайкович, В. А. Переверзева

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**E-learning system:** <https://etest.bsmu.by/> → Normal physiology (in the specialty “Dentistry”)

№	TOPIC	Defended	Organization
<b>“Introduction to the academic discipline “Normal Physiology”. The basic concepts of physiology. Principles of biomedical ethics”</b>			II term (spring)
<b>“The internal environment of the human body. Physiology of the blood”</b>			The spring term includes
Session 1	Introduction. The subject and tasks of Normal Physiology. Homeostasis. Physicochemical properties of blood		2 colloquiums: – Session 8
Session 2	Physiological functions of red blood cells. Hematopoiesis. Erythrocytopoiesis. Physiological functions of platelets. Thrombocytopoiesis. Hemostasis system		– Session 14
Session 3	Physiological functions of white blood cells. Leukopoiesis. Non-specific and specific resistance of the human body. Physiological evaluation of the complete blood count		– Session 18
Session 4	Blood types. ABO system. Rhesus (RhD) and other systems. Physiological bases of blood matching for the transfusion		
<b>“Mechanism of physiological functions regulation. Humoral regulation”</b>			
Session 5	Fundamentals of information exchange of the cell with the environment. Chemical signaling. General physiology of endocrine system		
Session 6	Special physiology of endocrine system		
Session 7	Physiology of bone tissue and regulation of calcium-phosphorus metabolism		
Session 8	<b>Colloquium. Concluding session</b> on the sections “Introduction to the academic discipline “Normal Physiology”. The basic concepts of physiology. Principles of biomedical ethics”, “The internal environment of the human body. Physiology of the blood”, “Mechanism of physiological functions regulation. Humoral regulation”		
<b>“General physiology”</b>			
Session 9	Electrical signaling. Laws of excitable tissues. Biological potential. Excitability changes during excitation		
Session 10	Excitation conduction along nerve fibers. Neuromuscular synapse		

№	TOPIC	Defended	Organization
Session 11	Physiology of skeletal muscles		
Session 12	Physiology of maxillofacial region muscles. Smooth muscles. The concept of myoepithelial and glandular cells		
Session 13	General physiology of the central nervous system		
Session 14	<b>Colloquium. Concluding session</b> on the section “General physiology”		
<b>“Mechanism of physiological functions regulation. Nervous regulation”</b>			
Session 15	The role and functions of spinal cord, brainstem, and cerebellum		
Session 16	Special physiology of nervous system (mesencephalon, forebrain)		
Session 17	Autonomic nervous system: structure, functions. Autonomic reflexes		
Session 18	<b>Concluding session</b> on the section “Mechanism of physiological functions regulation. Nervous regulation”. <b>Credit</b>		
<b>PERMISSION FOR THE CREDIT IS APPROVED</b>		<i>date</i>	
		<i>signature</i>	

Credit date is the last lesson of the term.

Students are admitted to the credit if they :

- 1) passed successively the current certification (the concluding classes, or colloquiums) with two positive marks: based on the results of certification of theoretical knowledge and based on the results of certification of practical skills;
- 2) do not have absences for practical classes and lectures (or they have worked off the missed classes and lectures);
- 3) presented notes of lectures intended for the supervised student independent work;
- 4) they have not violated discipline and safety rules;
- 5) have their protocols of practical classes completed and signed.

**SECTIONS**  
**“INTRODUCTION TO THE ACADEMIC DISCIPLINE “NORMAL PHYSIOLOGY”.**  
**THE BASIC CONCEPTS OF PHYSIOLOGY. PRINCIPLES OF BIOMEDICAL ETHICS”,**  
**“THE INTERNAL ENVIRONMENT OF THE HUMAN BODY. PHYSIOLOGY OF THE BLOOD”**

**Session 1. INTRODUCTION. THE SUBJECT AND TASKS OF NORMAL PHYSIOLOGY.**  
**HOMEOSTASIS. PHYSICOCHEMICAL PROPERTIES OF BLOOD**

DATE  
 « \_\_\_ » \_\_\_\_\_ 202\_\_  
 day month year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. The subject of Normal Physiology. The significance of Normal Physiology for the system of knowledge required for higher medical education. Normal Physiology in dentistry.</li> <li>2. Physiological concept of internal environment of the body. Homeostasis as the constancy of internal environment and functions of the organism as well as the mechanisms regulating them.</li> <li>3. Basic homeostasis' constants of the blood, cardiovascular, respiratory and other systems of the organism. Relative constancy of homeostatic constants at rest and their changes at higher levels of activity of the organism.</li> <li>4. Composition of cellular membrane. Main types of membrane transport.</li> <li>5. The role of water for vital functions. The content and distribution of water in the organism. The main fluid compartments of the body.</li> <li>6. The main strict physicochemical constants of blood. Osmotic blood pressure, its role in water distribution between extracellular and intracellular compartments. The main blood substances contributing the osmotic pressure creation. Cellular dehydration and hyperhydration. Isotonic, hyper- and hypotonic solutions.</li> <li>7. Blood. Functions of the blood. Blood volume. Composition of blood, its basic physical and chemical properties. Blood plasma proteins, their functions. Hematocrit.</li> <li>8. Colloid osmotic (oncotic) blood pressure, its role in water exchange between blood and interstitial fluid (tissues). The main blood proteins contributing to the oncotic blood pressure. The interstitial edema.</li> <li>9. Hemolysis and its types, plasmolysis. Adverse consequences of erythrocytes' hemolysis in human body.</li> <li>10. H<sup>+</sup> ions concentration in blood is its index (pH) as the strict homeostatic constant. Acidosis and alkalosis. Mechanisms of normal pH maintaining in the body. Buffer systems of blood, respiratory and renal compensation of acid-base balance disturbances.</li> <li>11. Fluids of the oral cavity: oral fluid (“mixed saliva”), gingival fluid, and saliva of salivary glands. Acid-base condition of the oral cavity.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Severina, T. G.</i> Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – P. 3–13.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 1.</li> <li>4. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 33–36, 41–42, 45–52.</li> <li>5. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 3–14, 47–58.</li> </ol>
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**WORK 1.1. SAFETY REGULATIONS FOR PRACTICAL SESSIONS AT THE DEPARTMENT OF NORMAL PHYSIOLOGY**

The teaching program at the Department of Normal Physiology envisages practical works performed by the students, mastering their practical skills of operating some electric devices, computer techniques, research equipment, laboratory dishes, chemical reagents and biological fluids.

In addition, students may be allowed to do research work in the laboratories of the Department during their out-of-classes hours.

**General requirements:**

1. The student should put on a lab coat (medical gown) before entering an academic room.
2. To assign the student on duty.

**A student on duty should:**

- observe the order, rules and requirements of safety provisions while working in practical rooms;
- receive the practical rooms key and various materials necessary for carrying out practical works — in the lab assistant’s room № 103;
- in the end of practical classes — switch off the water and lights and return the received materials into room № 103.

**Safety regulations in operating electrical equipment.**

Cases of electric trauma and fires may occur while working with electric equipment.

They may be caused by:

- working with defective electric equipment (knife-switches, sockets, etc.);
- absence of electric appliances grounding;
- breaking rule of operating electric devices;
- touching current-carrying elements with hands and metal objects.

In case of revealing a defect of the electric device or electric equipment it is necessary to inform the Lecturer about it.

While operating the electric equipment and electric devices it is strictly forbidden to:

- check the presence of electric voltage with fingers and touch current-carrying parts;
- operate ungrounded electric equipment and devices if not allowed by the device instruction;
- use defected electric equipment and electric wiring;
- leave an electric circuit under tension without supervision.

**Basic rules of first aid.**

The first aid to victims should be given immediately and properly. It may affect the life, consequences of injuries, burns and poisonings. You’ll get acquainted with specific rules of rendering it at clinical departments.

In case of serious injuries, burns due to electric trauma an ambulance should be called in (telephone number 103). If the injuries are mild, the victims should be given the first aid and directed to a medical care institution. It should be kept in mind that rendering aid to a person under electric current you shouldn’t touch him with bare hands. First of all, the setting (device), which the victim touches, should be switched off or you should separate the victim from current-carrying parts using sticks, boards and other dry objects not conducting electric current or cut off wires by an axe with a dry axe handle.

*In all cases, you must call the duty laboratory assistant, who is in the room № 103, or a lecturer of the Department.*

**Actions taken in case of fire.**

In case of fire one should immediately switch off the power, call in the assistance (**room 103**) or lecturer and start extinguishing the fire. There are fire extinguishers in rooms **104, 103, 135** and **138**. For extinguishing the fire one can also use available fire hoses: unreel the hose and open the hydrant. The fire hydrants with hoses are at the end of the corridor next to room **136**, in the niche between rooms **139** and **140, 133** and **132**, and opposite room **104**.

After the completion of safety rules studying it is necessary to put your name and signature in the “Safety Register for students” in the computer class, room 104.

**With safety regulations while performing practical works has been acquainted and instructed:**

\_\_\_\_\_

Date                                  Student’s signature                                  Student’s name (completely and legibly)

**WORK 1.2. ACQUAINTANCE WITH BASIC INDICES OF BLOOD HOMEOSTASIS, CARDIOVASCULAR, RESPIRATORY AND GASTROINTESTINAL SYSTEMS OF THE ORGANISM**

The Table 1.1 should be filled with the necessary values of indices using materials of lectures, E-learning system, textbooks and the corresponding sections of this practical book.

<i>Table 1.1</i>			<i>End of the Table 1.1</i>			
<b>Some most important factors of homeostasis</b>						
<b>Factor</b>		<b>Range of normal values</b>	<b>Measurement units</b>			
<b>Blood</b>			<b>Cardiovascular system</b>			
Blood volume			liters		Heart rate (HR) at rest	beats/min
Blood viscosity			relative units		Stroke volume (SV) at rest	ml
Content of blood cells:		–	–		Ejection fraction (EF) at rest	%
Red blood cells (Erythrocytes)	in men		cells/L of blood		Cardiac output (CO) at rest	L/min
	in women		cells/L of blood		<b>Respiratory system</b>	
White blood cells (Leukocytes)			cells/L of blood		Respiration rate (RR) at rest	per minute
Platelets (Thrombocytes)			cells/L of blood		Tidal volume (TV) at rest	ml
Hematocrit:	in men		–		Minute ventilation (MV) at rest	L/min
	in women		–		Alveolar ventilation (AV) at rest	% of MV
Osmotic blood pressure			mosm/kg, mosm/L		<b>Gastrointestinal system</b>	
Oncotic blood pressure			mm Hg		pH of saliva	–
Blood pH			–		pH of pure gastric juice	–
Blood glucose content			mmol/L		pH of pancreatic juice	–
Blood protein content			g/L			

**PROTOCOL**

Give examples of the **strict homeostatic constants**:

- 1) \_\_\_\_\_
- 2) \_\_\_\_\_
- 3) \_\_\_\_\_

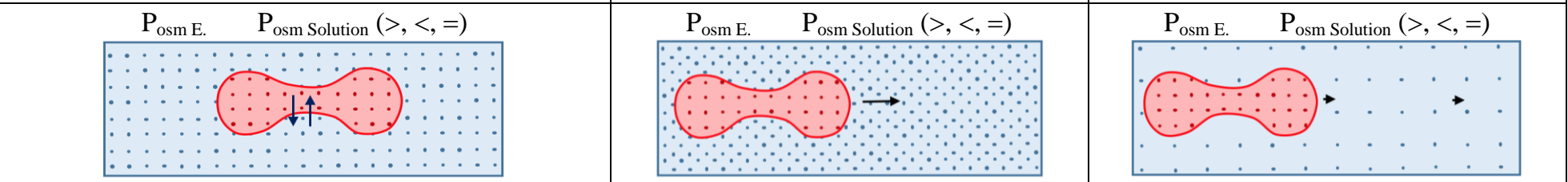
Give examples of the **soft** (changing within wide range of limits) **homeostatic constants**:

- \_\_\_\_\_
- \_\_\_\_\_

### WORK 1.3. CHANGES IN RBC' VOLUME IN SOLUTIONS OF DIFFERENT OSMOLARITY

#### Osmotic pressure — \_\_\_\_\_

Isotonic solution	Hypertonic solution	Hypotonic solution
<p>An <i>isotonic</i> solution is a solution that has the same osmotic pressure as the extracellular fluid and blood plasma. In this case, the movement of fluid between the extracellular and intracellular environments continues to exist, but the rates of transport of molecules are the same, so the movement is balanced.</p> <p><i>Examples: 0.9 % NaCl solution, 5 % glucose solution.</i></p>	<p>A <i>hypertonic</i> solution is one in which the concentration of the dissolved substance will be greater than in the extracellular fluid or plasma.</p> <p><i>Examples: 3 % NaCl solution.</i></p>	<p>A <i>hypotonic</i> solution is a solution in which the concentration of a substance is less than in the extracellular fluid or plasma.</p> <p><i>Examples: 5 % dextrose solution, 0.45 % NaCl solution.</i></p>



### WORK 1.4. HEMOLYSIS AND ITS TYPES (demonstration)

*Hemolysis* is the red blood cell membrane destruction resulting in the appearance of hemoglobin in the blood. Depending on etiology hemolysis can be **osmotic, mechanical, thermal, chemical** and **biological**. Physiological hemolysis is the result of ageing and destruction of red blood cells.

**Materials and equipment:** 4 test tubes; rat's blood; 0.9 % solution of NaCl; 5 % glucose solution; ammonium chloride; alcohol; iodine; distilled water; gauze ball; masks; rubber gloves; 3 % solution of chloramine.

**Accomplishment.** 2 ml of 0.9 % solution of NaCl are added into one test-tube and into the forth test-tube, 2 ml of 0.9 % solution of NaCl and 5 drops of ammonium chloride into the second test-tube, 2 ml of distilled water into the third test-tube and 2 ml of 5 % glucose solution into the forth test-tube. Then 2 drops of blood are added into every test-tube and the content is stirred. The forth test-tube is shaken vigorously.

1. A drops of blood in a 0.9 % NaCl solution.
2. A drops of blood in a 0.9 % solution of NaCl + 5 drops of NH<sub>4</sub>OH.
3. A drops of blood in distilled water.
4. A drops of blood in a 0.9 % NaCl solution + vigorous shaking.
5. A drops of blood in a 5 % glucose solution.

The results are evaluated after 45 min.

Test-tubes	Presence of red blood cells sediment	Color of the solution
1. 0.9 % NaCl		
2. 0.9 % NaCl + NH <sub>4</sub> OH		
3. Distilled water		
4. 0.9 % NaCl + vigorous shaking		
5. 5 % glucose		
Conclusion	Presence or absence of hemolysis	Type of hemolysis
1. 0.9 % NaCl		
2. 0.9 % NaCl + NH <sub>4</sub> OH		
3. Distilled water		
4. 0.9 % NaCl + vigorous shaking		
5. 5 % glucose		

### WORK 1.5. HEMATOCRIT ANALYSIS (demonstration)

The hematocrit index reflects the proportion of formed elements (primarily erythrocytes) in the total volume of blood. Hematocrit is determined by microcentrifugation or calculated automatically using modern hematology analyzers.

In a healthy person, hematocrit of venous and capillary blood is 0.40–0.49 (40–49 %) in men and 0.36–0.42 (36–42 %) in women.

**Materials and equipment:** hematocrit capillary, scarifier, absorbent cotton, antiseptic, centrifuge, clay or paste, scale to determine hematocrit.

**Work progress.** To do the work, open on your desktop computer (104) application “12\_Hematocrit”. Study the technique for determining hematocrit by clicking on the keywords highlighted in blue.

Capillary or venous blood is drawn into special hematocrit capillaries pre-treated with an anticoagulant (heparin or sodium citrate). Capillaries are sealed with clay or paste (a rubber cap can be used) and centrifuged for 5 minutes at 8000 rpm. After that, a particular scale is used to mark the percentage of capillary blood cells.

In modern hematology analyzers, the hematocrit index (Ht or HTC) is usually calculated by multiplying the erythrocytes count (RBC) by the mean corpuscular volume (MCV) per unit volume of blood.):  $Hct = RBC \times MCV$ .

**Instructions for writing the protocol.** Study the technique for determining hematocrit. Using the mouse to move the hematocrit capillary, determine the hematocrit value. Evaluate the result obtained.

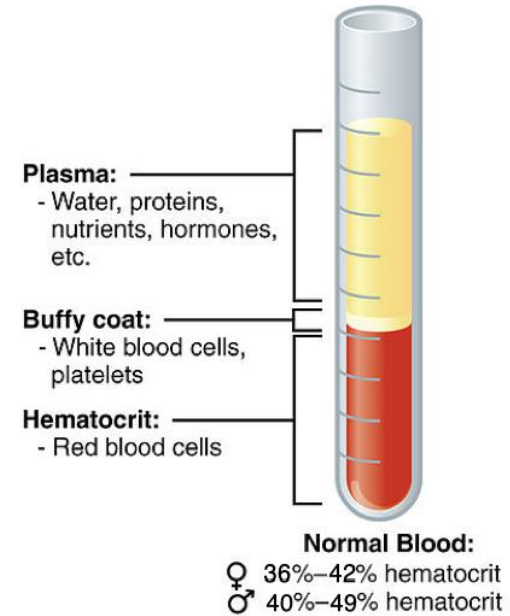


Fig. 1.1. Normal hematocrit values

### PROTOCOL

1. Hematocrit level in tested blood \_\_\_\_\_ % or \_\_\_\_\_.
2. Normal hematocrit for men: \_\_\_\_\_ % or \_\_\_\_\_.
2. Normal hematocrit for women: \_\_\_\_\_ % or \_\_\_\_\_.
3. **Conclusion:** value of hematocrit \_\_\_\_\_ (normal/higher/lower), that may be caused \_\_\_\_\_ (↑ or ↓) erythrocyte content per blood volume or \_\_\_\_\_ (↑ or ↓) circulating fluid volume.

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
Lecturer's signature

**Session 2. PHYSIOLOGICAL FUNCTIONS OF RED BLOOD CELLS. HEMATOPOIESIS.  
ERYTHROCYTOPOIESIS. PHYSIOLOGICAL FUNCTIONS OF PLATELETS.  
THROMBOCYTOPOIESIS. HEMOSTASIS SYSTEM**

DATE  
«      »      202    
day month year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Measures to prevent infection while working with blood and other biological fluids.</li> <li>2. Blood. Functions of the blood. Blood volume. Composition of blood, its basic physical and chemical properties. Blood plasma proteins, their functions.</li> <li>3. Red blood cells (erythrocytes, RBC). Peculiarities of the structure and properties of red blood cells providing their functioning. Methods of red blood cells count. Erythrocytosis and erythrocytopenia.</li> <li>4. Hemoglobin, its main types and forms. Peculiarities of the structure and properties of adult hemoglobin providing its functioning. Normal hemoglobin amount, evaluation methods.</li> <li>5. Color Index and RBC indices (MCH, MCHC, MCV, RDW), their calculation.</li> <li>6. Hematopoiesis. Erythron. Erythropoiesis. Erythropoietin, its origin, role. Role of Fe and vitamins of group B. Daily vitamin B9 and B12 requirements.</li> <li>7. Erythrocyte destruction. Red blood cells destruction products, their utilization.</li> <li>8. Platelets, their count, structure and functions. Thrombocytosis and thrombocytopenia. Thrombocytopoiesis. Role of thrombopoietin.</li> <li>9. The concept of the hemostasis system and its mechanisms. Primary (vascular-thrombocyte) and secondary (plasma-coagulation) hemostasis: significance, evaluation methods. Concept of anticoagulants and fibrinolysis.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;">Main</p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 250–254, 605–607, 613–630.</li> <li>3. <i>Severina, T. G.</i> Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – P. 22–30, 40–41.</li> </ol> <p style="text-align: center;">Additional</p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 2.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 553–554, 562–564, 567, 582, 695–706, 603–604.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 4–6, 305–320, 381–387, 389–390, 396, 397–398, 401–416, 790–793.</li> </ol>
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**WORK 2.1. TERMINOLOGY**

Red blood cells are \_\_\_\_\_  
\_\_\_\_\_

Pathological states of hemoglobin \_\_\_\_\_  
\_\_\_\_\_

Functions of RBC include:  
1) \_\_\_\_\_ 2) \_\_\_\_\_  
3) \_\_\_\_\_ 4) \_\_\_\_\_

Erythrocytosis — \_\_\_\_\_  
\_\_\_\_\_

Normal count of RBC:  
♀ \_\_\_\_\_ ♂ \_\_\_\_\_

Erythrocytopenia — \_\_\_\_\_  
\_\_\_\_\_

Hemoglobin — \_\_\_\_\_  
\_\_\_\_\_

Platelets are \_\_\_\_\_  
\_\_\_\_\_

Types of hemoglobin: \_\_\_\_\_

Normal count of PLT: \_\_\_\_\_

Physiological states of hemoglobin \_\_\_\_\_  
\_\_\_\_\_

Functions of PLT include: \_\_\_\_\_  
\_\_\_\_\_

**Self-study questions**

1. What are the conditions required for the hemocytopoiesis?
2. Which factors determines the number of red blood cells in human blood?
3. What mechanisms determine different numbers of red blood cells in men and women?
4. Why are oxygen carriers needed in the blood?
5. What is the color index for?
6. What characterizes the indicator MCH?
7. List the steps of hemostasis.
8. What is the daily requirement for iron? What percentage of iron intake is absorbed?
9. What common cause may explain an increase of erythropoiesis in blood loss, massive hemolysis, respiration at low atmospheric pressure?
10. What trace elements and vitamins are most important for erythropoiesis?

## WORK 2.2. RED BLOOD CELLS COUNT IN THE COUNTING CHAMBER UNDER THE MICROSCOPE (demonstration)

To enhance prevention of infecting with HIV, viral hepatitis and other infections transmitted through blood the work is conducted as demonstration.

To count blood cells the blood is diluted in special mixers to create an optimal concentration of cells for their count. On counting RBC under microscope the hypertonic **3 % solution of NaCl** is used as a diluter where RBC shrinkage occurs what makes their color more intensive and their counting easier. For automatic counting equipment usually various versions of isotonic solution are used for blood dilution.

The counting chamber is a thick glass, the middle part of which has **Goryaev's net**. This middle part of the glass is 0.1 mm lower than the lateral maintaining storage areas. In placing the cover glass the space of 0.1 mm is formed over the net.

Goryaev's net of the counting chamber is divided into small squares that in turn are divided into 16 small squares. A side of a small square is 1/20 mm, the area –  $1/20 \times 1/20 = 1/400 \text{ mm}^2$ ; thus the space volume over a small square is  $1/400 \times 1/10 = 1/4000 \text{ mm}^3$ .

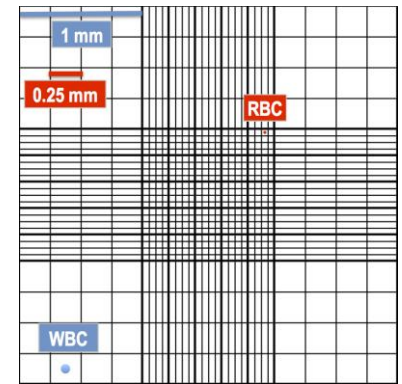
The RBC count is done by a fragment photograph of the counting chamber with red blood cells mixture. The count is done in **5 large squares** (divided into 16 small squares) **located along the diagonal or in 4 corner squares and 1 central square**.

The cells are counted according to **Egorov's rule**: this square includes all RBC inside as well as on its *left and upper* border (it may differ in different laboratories).

Let's presume that 5 large squares (80 small squares) are found to contain the total count of red blood cells equal to E. The red blood cells count in the space volume ( $1/4000 \text{ mm}^3$ ) over one small square will be E/80. To evaluate it for  $1 \text{ mm}^3$  of blood, E/80 is multiplied by 4000 and again by 200 as the blood was diluted 200-fold. To evaluate the red blood cells count in 1L of blood, the received red blood cells count in  $1 \mu\text{L}$  ( $1 \text{ mm}^3$ ) is multiplied by  $10^6$ .

### Directions for recording the protocol

Calculate the total red blood cells count in 5 large squares (divided into 16 small squares) or in 4 corner squares and 1 central square of the photograph at low magnification. Calculate the content of red blood cells in 1 liter of blood by the formula. Evaluate the received result versus the norm.



Indicate the mixer marks and bead color.



Mixer for RBC

### PROTOCOL

1. RBC count in large squares is:  
in 1 \_\_\_\_\_; in 2 \_\_\_\_\_; in 3 \_\_\_\_\_;  
in 4 \_\_\_\_\_; in 5 \_\_\_\_\_.

The total RBC count in 5 large squares is equal to \_\_\_\_\_ cells.

2. The RBC count in 1 liter of blood (X) is calculated by the formula:

$$X = \frac{E \times 4000 \times 200}{80} \times 10^6 = E \times 10^{10} \text{ cells per L}$$

X = \_\_\_\_\_  $\times 10^{12}$  cells / liter

3. RBC reference range (normal values):

in men: \_\_\_\_\_

in women \_\_\_\_\_

4. Conclusion: \_\_\_\_\_

**WORK 2.3. EVALUATION OF THE AMOUNT OF HEMOGLOBIN BY SAHLI'S METHOD (demonstration)**

The hemoglobin content in the blood of a healthy person is:  
**in men – 130–170 g/l; in women – 120–150 g/l.**

The blood hemoglobin content is evaluated by measuring the amount of the reaction product formed in the interaction of hemoglobin and various reagents.

The simplest method is a colorimetric method, based on the formation of muriatic hematin (hematin chloride) — the substance giving the solution a brown color, when hemoglobin interacts with hydrochloric acid. For this purpose Sahli's hemometer is used. It consists of a stand, the back wall of which is made of frosted glass, and 3 test-tubes. The center test-tube is graduated; it is designed for performing tests, while the lateral ones, soldered, contain the standard solution of hematin chloride. The blood used for preparing the standard contains 16.7 g% or 167 g/l of hemoglobin.

To evaluate the content of hemoglobin 0.1 N solution of HCl is added to the central test-tube, then 20 mcl of blood taken from the finger. The content of the test-tube is stirred and it is placed to the stand for 5–10 minutes. After this time muriatic hematin is formed and the solution becomes dark-brown. Then distilled water is added to the test-tube till the solution color becomes as light-brown as the color of the standard in both lateral test-tubes (the solution is stirred with a glass stick on every addition of distilled water).

The hemoglobin content is determined by the graduation on the test-tube. The digits at the level of a lower solution meniscus show the hemoglobin content in grams per 100 ml of blood (g%). For example, the tested blood contains 15.5 g% of hemoglobin, so the hemoglobin content in 1 liter is 155 g/l.



**Directions for recording the protocol**

Determine the content of hemoglobin in the tested blood. Evaluate the received result versus the norm.

**PROTOCOL**

- Hemoglobin content in tested blood = \_\_\_\_\_ g/%, or \_\_\_\_\_ g/l.
- Normal blood Hb content is:  
 in men \_\_\_\_\_ g/l;  
 in women \_\_\_\_\_ g/l.
- Conclusion:** hemoglobin content in tested blood is \_\_\_\_\_  
 (normal, increased or decreased)

**WORK 2.4. EVALUATION OF COLOR INDEX AND MCH**

To evaluate an *absolute content* of hemoglobin in every erythrocyte the **MCH** (Mean Corpuscular Hemoglobin) index is used. It's approximately equal to 30 picograms (reference range 25.4–34.6 pg). Its value is obtained by division of the hemoglobin (HGB) content in 1 liter by red blood cells count in 1 liter: **MCH = HGB / RBC**.

The **Color Index** (CI) is a relative value of hemoglobin content in red blood cells. CI is calculated by division of the hemoglobin content in g/l by the number of the first three digits of red blood cells count in 1 liter of blood with multiplication of the received value by 3:

$$CI = \frac{3 \times Hb(g/l)}{RBC \times 10^{-10}}$$

For example, the blood hemoglobin content is 152 g/l, the erythrocyte count is  $4.56 \times 10^{12}/l$ ; then CI is equal to  $3 \times 152 : 456 = 1.00$ .

CI of a healthy person is **0.8–1.05** (*normochromia*). In decreased hemoglobin content in Red Blood Cells CI is **less than 0.8** (*hypochromia* that usually occurs in iron deficiency in the organism), in **increased — over 1.05** (*hyperchromia* which is noted in insufficiency of vitamin B<sub>12</sub> and/or folic acid in the organism).

**Directions for recording the Protocol**

Calculate MCH and CI of the tested blood using the data of work 2.2 and work 2.3.

**PROTOCOL**

- Hemoglobin content in tested blood is equal to \_\_\_\_\_ g/l.  
 Red blood cells count in tested blood is equal to \_\_\_\_\_  $\times 10^{12}/l$ .

	<b>Index</b>	<b>Normal range (with units)</b>
<b>MCH =</b>	: =	
<b>CI = 3 ×</b>	: =	

- Conclusion:** \_\_\_\_\_ (normo-, hypo- or hyperchromia)

## WORK 2.5. HEMOSTATIC SYSTEM

**Hemostasis** is the mechanism that ensures the state of blood in the channel in a liquid state, stopping bleeding and preventing blood loss in case of damage to blood vessels.

**Primary hemostasis** (*microcirculatory*) — vascular and platelet response to vascular injury; It starts reactions of hemostasis in capillaries, venous and arterial vessels up to 200  $\mu\text{m}$  in diameter. This process involves platelets and endothelium of vessels. Almost 80 % of bleedings and 95 % of thrombus formations are associated with the impairment of this mechanism.

Primary (vascular-thrombocyte, microcirculatory) hemostasis means fast (within several minutes) formation of platelet clots at the site of vessel injury what is very important for stopping bleeding from small vessels with low blood pressure.

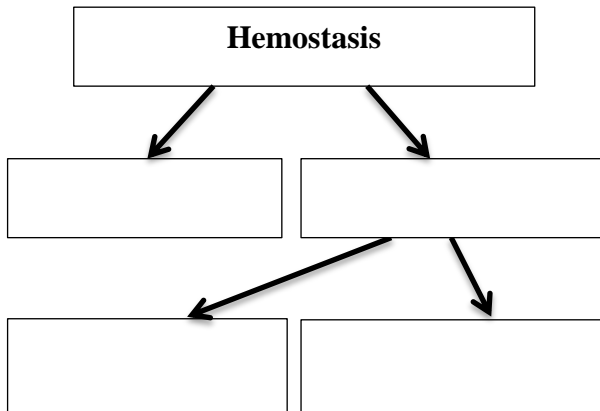
**Secondary hemostasis** (*macrocirculatory*) — blood clotting or hemostatic reaction. It starts as a rule on the basis of the primary one and follows it. It is accomplished by the blood coagulation system. Due to the secondary hemostasis a red thrombus is formed, it consists mainly of fibrin and blood cells. It provides a final stop to bleeding from injured macro vessels (**over 200  $\mu\text{m}$  in diameter**).

**The primary hemostasis stages are:**

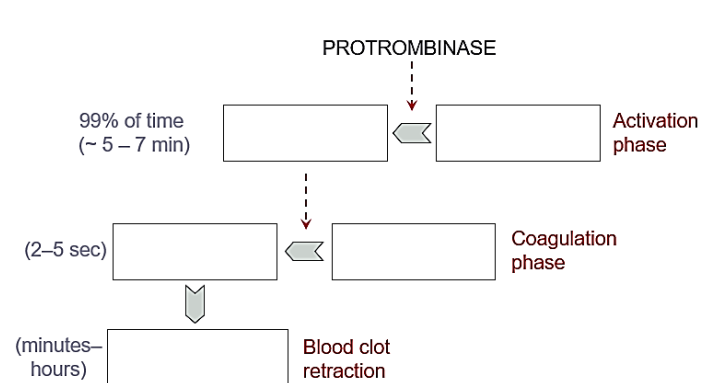
- 1) **spasm of vessels** (*local vasoconstriction*);
- 2) **platelets adhesion** (*involving Willebrand's factor*), their **activation** and secretion of platelets granules (*involving thromboxane  $A_2$  through a phospholipase mechanism*), as well as platelets **aggregation** (*at first it is reversible and then irreversible due to the action of thrombin and fibrin traces*) with the **formation of a platelets plug**;
- 3) **retraction** (*constriction and consolidation*) of the platelets plug.

The most important screening indices characterizing the primary hemostasis are: **bandage test, platelets count, bleeding time by Ivy or Duke.**

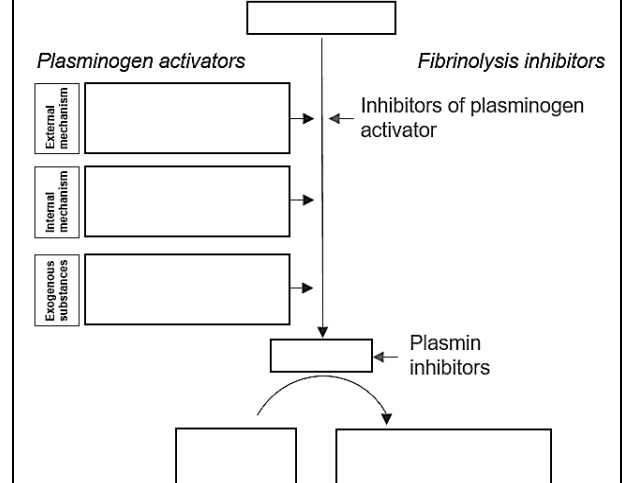
Fill in the *general scheme of hemostatic system.*



Fill in the *scheme of coagulation by Moravitz.*



Fill in the *scheme of fibrinolysis.*



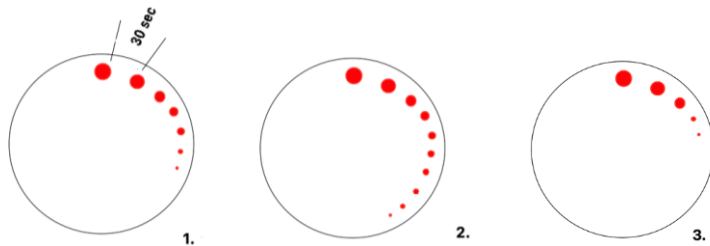
**WORK 2.6. EVALUATION AND PHYSIOLOGICAL ASSESSMENT OF PRIMARY HEMOSTASIS INDICES**

**A. Time of bleeding by Duke**

The time of bleeding evaluated by Duke’s method gives a general idea, if the primary hemostasis function is normal (and first of all it allows evaluating the function of platelets, their ability for adhesion or aggregation). An increase of bleeding time evidences the impairment of the primary hemostasis due to thrombocytopenias, thrombocytopathias, vascular wall injuries or a combination of these factors.

**Materials and methods:** a stop-watch, sterile filter paper, lancet, gauze balls, iodine, rubber gloves, masks, disinfectant solution.

**Accomplishment.** Puncture the 4th finger-cushion to the depth of 3 mm. If this is done properly, the blood is discharged spontaneously without pressure. Having made a puncture, switch on the stop-watch. Touch the first appearing blood drop with a strip of sterile filter paper that absorbs the blood. Then take off further blood drops with sterile filter paper every 30 sec. Avoid touching the skin with filter paper, as it stimulates premature stop of bleeding. Continue till blood traces are absent on the filter paper. **The normal duration of the bleeding time by Duke is 2–4 min.**

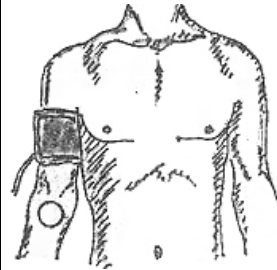


**PROTOCOL**

1. Filter-paper number \_\_\_\_\_
2. Bleeding time is \_\_\_\_\_ min \_\_\_\_\_ sec.
3. **Conclusion:** Bleeding time is \_\_\_\_\_  
(normal, increased, reduced)

**B. Bandage test** (evaluation of a vascular component of the primary hemostasis)

The method is based on the fact that dosed mechanic action (pressure) on skin capillaries of a healthy person does not cause any substantial changes. When the normal state of a capillary wall is impaired, increased vascular fragility occurs and after mechanic action at the site of the pressure multiple petechiae or hemorrhage appear manifesting the impairment of a vascular component of hemostasis.



**Materials and equipment:** a tonometer, a stop-watch, a circle of dense card-board 2.5 cm in diameter, a pen or a pencil.

**Accomplishment.** The test is done on the forearm. A circle 2.5 cm in diameter is outlined 1.5–2.0 cm from the ulnar pit. To do a test one should check if there are any hemorrhages in this circle (and their number if there are any). The blood pressure cuff is applied and the pressure of 80 mm Hg is created. The pressure is sustained at this level for 5 minutes pumping the air if necessary. The arm of the examined person should be relaxed and lie freely.

All **petechiae** that appeared in the outlined circle are counted in 10–15 minutes (taking into consideration those present before). In healthy persons petechiae are not formed or their number does not exceed 10 in the circle and their sizes are not more than 1 mm in diameter (negative bandage test). An increase of the petechiae number over 10 and petechiae sizes over 1 mm in diameter or the presence of a hemorrhage (positive bandage test) evidence the following: wall defects of micro vessels due to endocrine changes (menstrual period); infectious-toxic effect (sepsis etc.); insufficiency of vitamin C; the impairment of Willebrand’s factor formation, etc.; the presence of thrombocytopenia or thrombocytopathy etc.

**PROTOCOL**

1. Petechiae number in the circle before the test \_\_\_\_\_ (no, 1, 2, 3...)  
Petechiae number in the circle in 10–15 minutes after the test \_\_\_\_\_ (no, 1, 2, 3...).  
If petechiae are present, indicate their diameter \_\_\_\_\_ (below 1 mm or over 1 mm).
2. **Conclusion:** bandage test \_\_\_\_\_  
(negative= **without** petechiae or positive = **with** petechiae)

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
Lecturer’s signature

**Session 3. PHYSIOLOGICAL FUNCTIONS OF WHITE BLOOD CELLS. LEUKOPOIESIS.  
NON-SPECIFIC AND SPECIFIC RESISTANCE OF THE HUMAN BODY.  
PHYSIOLOGICAL EVALUATION OF THE COMPLETE BLOOD COUNT**

DATE  
«      »      202    
day month year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. White blood cells (WBC), their types. Leukocyte formula.</li> <li>2. Granulocytes, their types. Functions and properties of neutrophils. Granulocytopoiesis. Types of neutrophils depending on the degree of their maturity. Shifts of leucocyte formula.</li> <li>3. Functions and properties of basophils and eosinophils.</li> <li>4. Monocytes and tissue macrophages. Monocytopoiesis. Peculiarities of the structure and properties providing macrophages functioning. Mechanisms of phagocytosis. Concept of the complement system.</li> <li>5. T- and B-lymphocytes, peculiarities of their maturation and functions. Lymphocytopoiesis. Null and plasma cells.</li> <li>6. Concept of cellular and humoral immunity; immune response. Functions of immunoglobulins.</li> <li>7. Erythrocyte sedimentation rate (ESR), main factors affecting it, and methods of determination. Diagnostic significance of ESR.</li> <li>8. Basic indices included into the complete blood count. Physiological assessment of the complete blood count results. Diagnostic value of the complete blood count.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. By V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 254–264, 283.</li> <li>3. <i>Severina, T. G.</i> Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – P. 31–40.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 3.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 554–558.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 445–476.</li> </ol>
<p><b>WORK 3.1. TERMINOLOGY</b></p>	
<p>Erythrocyte sedimentation rate (ESR) is _____</p>	
<p>White blood cells are _____</p>	<p>Shift to the left is _____</p>
<p>Leukocyte formula is _____</p>	<p>Shift to the right is _____</p>
<p>Phagocytosis is _____</p>	

**Self-study questions**

- |  |  |
|--|--|
| <ol style="list-style-type: none"> <li>1. The count of what blood cells (erythrocytes or leukocytes) is maintained at a more constant level in blood and why?</li> <li>2. What is the leucocyte formula <i>shift to the left</i>?</li> <li>3. What is the difference between physiologic and reactive (true) leukocytosis? Causes of physiologic and reactive leukocytosis.</li> <li>4. What are the functions of T and B lymphocytes?</li> <li>5. Which cells have the function of regulating cellular immunity? Of humoral?</li> </ol> | <ol style="list-style-type: none"> <li>6. What indices of the complete blood count characterize the respiratory function of the blood</li> <li>7. What is the erythrocyte sedimentation rate?</li> <li>8. What causes an increase in erythrocyte sedimentation rate? Decrease?</li> <li>9. What are the rules for patient before taking the capillary blood for complete blood count?</li> </ol> |
|--|--|

*Make a conclusion for the further complete blood count.*

<p>Make a conclusion for complete blood count of a 20-year-old man (performed at 7.30 a.m.): RBC — <math>5 \times 10^{12}</math> cells/l; hemoglobin — 160 g/l; color index (CI), MCH — calculate; leukocytes — <math>12 \times 10^9</math> cells/l (young neutrophils — 4 %, band neutrophils — 10 %; segmented neutrophils — 59 %; basophils — 0 %; eosinophils — 1 %; lymphocytes — 20 %; monocytes — 6 %); ESR — 30 mm/h.</p>	<p>Make a conclusion for complete blood count of a 40-year-old man (performed at 4.15 p.m.): RBC — <math>2.9 \times 10^{12}</math> cells/L; hemoglobin — 90 g/L; CI, MCH — calculate; leukocytes — <math>3.1 \times 10^9</math> cells/L; platelets — <math>86 \times 10^9</math> cells/L; ESR — 20 mm/h.</p>
<p>Make a conclusion for complete blood count a woman of 35 years old (performed at 8.00 a.m.): RBC — <math>4.2 \times 10^{12}</math> cells/L; hemoglobin — 148 g/L; CI, MCH — calculate; leukocytes — <math>4 \times 10^9</math> cells/L (young neutrophils — 0 %, band neutrophils — 0 %; segmented neutrophils — 58 %; basophils — 1 %; eosinophils — 8 %; lymphocytes — 24 %; monocytes — 9 %); ESR — 2 mm/h.</p>	<p>Make a conclusion for complete blood count a woman of 25 years old (performed at 8.00 a.m.): RBC — <math>3.2 \times 10^{12}</math> cells/L; hemoglobin — 148 g/L; CI, MCH — calculate; leukocytes — <math>7 \times 10^9</math> cells/L (young neutrophils — 0 %, band neutrophils — 0 %; segmented neutrophils — 68 %; basophils — 2 %; eosinophils — 10 %; lymphocytes — 11 %; monocytes — 9 %); ESR — 17 mm/h.</p>
<p>Make a conclusion for complete blood count a woman of 18 years old (performed at 7.30 a.m.): RBC — <math>3.4 \times 10^{12}</math> cells/L; hemoglobin — 90 g/L; reticulocytes — 5.2 %; CI, MCH — calculate; leukocytes — <math>7.2 \times 10^9</math> cells/L (young neutrophils — 0 %, band neutrophils — 1 %; segmented neutrophils — 39 %; basophils — 1 %; eosinophils — 8 %; lymphocytes — 46 %; monocytes — 5 %); platelets — <math>160 \times 10^9</math> cells/L; ESR — 18 mm/h.</p>	<p>Make a conclusion for complete blood count of a 18-year-old man (performed at 8.00 p.m.): RBC — <math>4.6 \times 10^{12}</math> cells/L; hemoglobin — 150 g/L; CI, MCH — calculate; leukocytes — <math>10.3 \times 10^9</math> cells/L; platelets — <math>370 \times 10^9</math> cells/L; ESR — 20 mm/h.</p>

### WORK 3.2. ESR ANALYSIS BY PANCHENKOV'S METHOD (demonstration)

Unless the blood is not coagulated, red blood cells sediment to the test-tube bottom as their specific weight (1.096 g/ml) is higher than that of plasma (1.027 g/ml). **Normal values of erythrocyte sedimentation rate (ESR) in healthy people are: in male 1–10 mm/h; in female 2–15 mm/h.** The *most important factors affecting ESR* are the proportion of various kinds of blood plasma proteins as well as red blood cells content. An increase of globulins and fibrinogen, and/or decrease of albumins in plasma as well as decrease of red blood cells is associated with an increase of ESR. An increase of red blood cells in the blood as well as an increase of albumin and bile pigments results in a decrease of ESR. A higher value of the ESR norm in women is associated with a less red blood cells content.

Under physiological conditions an increased ESR is noted during pregnancy, in eating dry food and fasting, after vaccination (due to an increase of globulins and fibrinogens in plasma). Delayed ESR can be noted in blood thickening due to enhanced perspiration (for example, in high external temperature) or enhanced formation and content of erythrocytes in blood (for example, in Alpine residents and mountaineers).

**Materials and equipment:** Panchenkov's device, rat's blood, a watch glass, test tube, a porcelain basin, rubber gloves, masks, gauze ball, antiseptic, iodine, 3 % solution of chloramine, 5 % solution of sodium citrate.

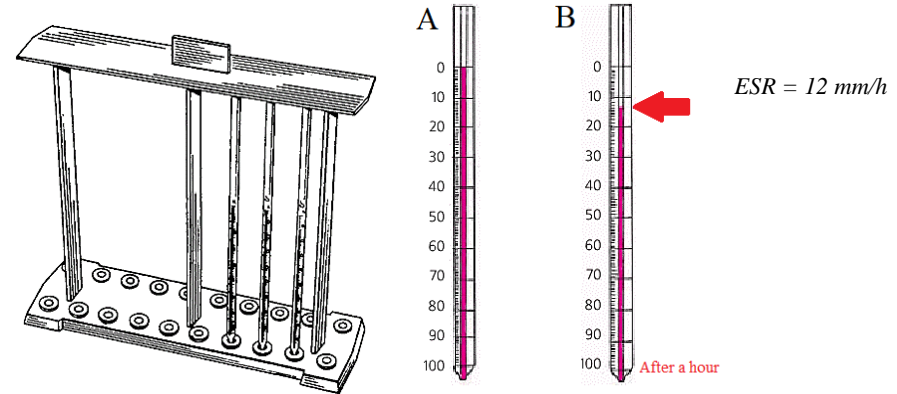


Fig. 3.1. Equipment for ESR analysis

**Accomplishment.** Panchenkov's device is used to evaluate ESR. A pipette (capillary) of the device is washed with 5 % solution of sodium citrate to prevent blood coagulation. The taken blood is carefully stirred with sodium citrate on the watch glass. The mixture is drawn into the pipette to mark 0. The pipette is placed into the stand for 1 hour in a strictly vertical position. The result is assessed by a decrease of a red column of red blood cells in the capillary from point 0 (in millimeters).

While evaluating ESR this should be followed strictly: the proportion of sodium citrate and blood 1:4; verticality of the pipette in the stand; the temperature in the room — 18–22 °C (in lower temperature ESR decreases and in higher — increases).

#### PROTOCOL

1. ESR of tested blood = \_\_\_\_\_ mm/h. Tested person sex is \_\_\_\_\_.
2. ESR reference range (normal values): for men \_\_\_\_\_ mm/h;  
for women \_\_\_\_\_ mm/h;
3. While evaluating ESR the blood is mixed with 5 % solution of Na citrate with the aim \_\_\_\_\_
4. **Conclusion:** ESR is \_\_\_\_\_  
(in norm, increased or decreased)

**WORK 3.3. WHITE BLOOD CELLS COUNT IN THE COUNTING CHAMBER UNDER THE MICROSCOPE**

The white blood cells content in the blood in norm is  $(4-9) \times 10^9/L$ .

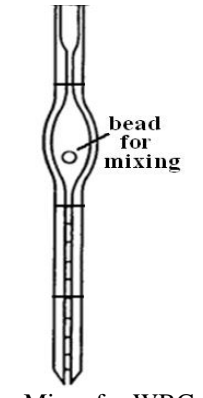
**Materials and equipment:** rat's blood; a mixer for white blood cells, a counting chamber, 5 % solution of acetic acid, a porcelain basin, gauze ball, alcohol, iodine, rubber gloves, masks, 3 % solution of chloramine.

**Accomplishment.** For white blood cells counting blood is diluted in special mixers. The blood is applied into the mixer to the mark 0.5 and then 5 % solution of acetic acid stained with methylene blue to the mark 11 (20-fold dilution of the blood) is added. The acetic acid destroys plasmatic membranes of all blood cells (chemical hemolysis), while methylene blue stains nuclei of white blood cells. The mixer is shaken for 1-2 min. The chamber is filled in from the mixer ampoule.

White blood cells (white blood cells nuclei) are counted in small magnification in 25 large squares.

**Directions for recording the Protocol:**

1. Calculate the total count of leukocytes in 25 large squares (due to image limitations, calculate it in any 5 large squares and multiply by 5).
2. Calculate the leukocyte count in 1L by the formula.
3. Assess the obtained result versus the norm.



**PROTOCOL**

1. White blood cells count (WBC) in 25 large squares is equal to \_\_\_\_\_ cells.

2. Leukocyte count (X) in 1 L of blood is calculated by the formula:  

$$X = \frac{L \times 4000 \times 20}{400} \times 10^6 = 2L \times 10^8 / L = \text{___} \times 10^8 \text{ cells/L}$$

$$X = \text{___} \times 10^9 \text{ cells/L}$$

3. Normal count of WBC: \_\_\_\_\_

4. **Conclusion:** \_\_\_\_\_

**WORK 3.4. PERCENTAGE CALCULATION OF DIFFERENT TYPES OF WHITE BLOOD CELLS IN A BLOOD SMEAR (leukocyte formula)**

White blood cells are the effector cells of the immune system and circulate throughout the bloodstream and lymphatic system. An infection or a physical injury results in an inflammatory response, which induces increased production of WBCs for resolving the injury or infection. Due to this association between WBCs and inflammatory response, WBC count is a valuable metric for diagnosis and prognosis of several diseases. Counting WBCs can be done either manually or automatically.

**Accomplishment.** Calculate the proportion of various types of white blood cells (per 100 cells) in a stained blood smear by its photograph presented on the monitor screen on the basis of the following features: size of cells, shape of the nucleus, character of the nucleus and cytoplasm staining, presence or absence of granules in the cytoplasm and type of their staining.

**Directions for recording the Protocol**

Fill in the table with obtained count data of various forms of WBC and assess the result versus the norm.

**Note!**

WBC — White blood cells

BASO — basophils, EOS — eosinophils, NEU — neutrophils

MONO — monocytes, LYMP — lymphocytes

**PROTOCOL**

Factor	Total WBC	BASO	EOS	NEU			MONO	LYMP
				young	band	segmented		
In a blood smear	cells							
	%	100	0-1	1-5	0-1	1-5	46-68	2-10

**Conclusion** on the leukocyte formula: \_\_\_\_\_

(baso-, eosino-, neutrophilia (or -penia); mono-, lymphocytosis (or -penia);  
shift to the left, shift to the right)

### WORK 3.5. PHYSIOLOGICAL ASSESSMENT OF THE COMPLETE BLOOD COUNT

Complete blood count (hematology tests) is one of the most common laboratory examinations. It includes evaluation of the following indices:

- 1) Red blood cells count per 1 liter of blood;
- 2) Hemoglobin content (g/l);
- 3) calculation of Color Index;
- 4) White blood cells count per 1 liter of blood;
- 5) Leukocyte formula (WBC differentiation);
- 6) Erythrocyte sedimentation rate (ESR).

Additional examinations include: evaluation of platelets in 1 liter of blood, count of reticulocyte percentage and some other indices. Modern hematologic analyzers allow additional evaluation of: the hematocrit, mean volumes of red blood cells, white blood cells and platelets; mean hemoglobin content in red blood cell, etc.

Using complete blood count indices the physician may assess the respiratory function of the blood (by the hemoglobin content, red blood cells count); erythropoiesis intensity (by the reticulocyte count); suggest the presence of infectious, inflammatory and autoimmune processes in the organism (by the white blood cells count, "left shift" of the leukocyte formula and ESR changes) etc.

#### PROTOCOL

Factor	Normal range	Results (male)	Conclusion
1. Red blood cells (RBC)	$(3.9-5,1) \times 10^{12}$ cells/l, m $(3.7-4.9) \times 10^{12}$ cells/l, f	$3.58 \times 10^{12}$ cells/l	
2. Hemoglobin (HGB)	130-170 g/l, male 120-150 g/l, female	100 g/l	
3. Color index (CI)	0.8-1.05	=	
4. White blood cells (WBC)	$(4-9) \times 10^9$ cells/l	$10 \times 10^9$ cells/l	
5. Leukocyte formula:	Per 100 cells (100 %)		
5.1. Basophils	0-1 %	1 %	
5.2. Eosinophils	1-5 %	2 %	
5.3. Neutrophils:			
myelocytes	0 %	0 %	
young	0-1 %	2 %	
band	1-5 %	10 %	
segmented	46-68 %	48 %	
5.4. Monocytes	2-9 %	8 %	
5.5. Lymphocytes	18-40 %	29 %	
6. Erythrocyte sedimentation rate (ESR)	1-10 mm/h, male 2-15 mm/h, female	16 mm/h	
7. Platelets	$(150-450) \times 10^9$ cells/l	$225 \times 10^9$ cells/l	

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

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
Lecturer's signature

**Session 4. BLOOD TYPES. ABO SYSTEM. RHESUS (RhD) AND OTHER SYSTEMS.  
PHYSIOLOGICAL BASES OF BLOOD MATCHING FOR THE TRANSFUSION**

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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Antigens of blood cells. Basic systems of red blood cells antigens. Human blood type systems.</li> <li>2. Antigens (agglutinogens) and antibodies (agglutinins) of ABO blood type, their characteristics.</li> <li>3. Blood typing in the ABO system using the standard and monoclonal sera.</li> <li>4. The Rh system of antigens (RhD). Consequences of mismatched blood transfusion in the Rhesus system.</li> <li>5. The concept of blood preparations and blood substitution solutions.</li> <li>6. Principles of blood matching. Tests performed before blood preparations transfusion.</li> <li>7. Risk factors for the recipient. Prevention of infecting the recipient during transfusion of donor blood or blood preparations.</li> <li>8. Donor blood preparations. Blood substituting solutions, their functions. Basic requirements to blood substituting solutions.</li> <li>9. Consequences of mismatched blood transfusion in ABO system.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 264–267, 281–292.</li> <li>3. <i>Severina, T. G.</i> Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – P. 42–52.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 4.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 558–562, 564–567.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 477–494.</li> </ol>
<p><b>WORK 4.1. TERMINOLOGY</b></p>	
<p>Blood type — _____</p>	<p>Standard sera — _____</p>
<p>Antigens — _____</p>	<p>Monoclonal sera — _____</p>
<p>Antibodies — _____</p>	<p>Recipient — _____</p>
<p>Serological test — _____</p>	<p>Donor — _____</p>
<p>Rhesus system — _____</p>	<p>Risk factors of blood transfusion: _____</p>

## WORK 4.2. BLOOD TYPING IN THE ABO SYSTEM USING STANDARD SERA (demonstration)

The ABO system blood type is determined by the presence of agglutinogens in red blood cells which is revealed by the hemagglutination reaction using standard sera. The interaction between red blood cells antigens of the tested blood and a corresponding antibodies (agglutinins) of the standard serum under-lies the bases of such reaction. As antibodies contained in standard sera are known, red blood cells antigens of the tested blood and consequently the blood type in the ABO system are determined by the presence or absence of agglutination.

**Materials and equipment:** standard sera of O (I), A (II), B (III) and AB (IV) types of two various series; pipettes for them; special plate; glass sticks; isotonic (0.9 %) solution of NaCl; lancets; gauze ball; antiseptic; iodine; rubber gloves; masks; disinfectant solution.

### Accomplishment

1. Blood typing should be done in the room with sufficient illumination and at the temperature of **15–25 °C**.

2. Determination is done on special plate. **0.1 ml** (1 large drop) of every standard serum of two series is applied to appropriate depressions of the plate.

3. The blood for the test is taken from the finger in compliance with all necessary rules. Then **0.01 ml** blood is added with glass sticks (**10 times less than the serum**) to every drop of the serum and carefully stirred. The obtained mixture is mixed again by rocking the plate.

4. The reaction of agglutination is observed during 5 minutes. Usually the agglutination reaction starts during the first *10–30 seconds*, however agglutination may be late. In case of agglutination but **no less than 3 minutes**, it is needed to add **0.9 % NaCl** to exclude false agglutination.














**The reaction in every drop may be either positive or negative.**

In a *positive* reaction there appear small red granules (agglutinates) seen with naked eye in the mixture; they consist of glued red blood cells. Step-by-step they cluster and form larger granules or flakes of irregular shape. Meanwhile the serum becomes completely or partially decolorized.

In case of a *negative* reaction the content of drops stays regularly stained in red, and agglutinates are not revealed there. The results of the reaction in both serum series should be identical.

**RULE!** Serum contains ONLY agglutinins (antibodies) !!!

**Different combinations of the reaction are possible**

Results with standard sera			The blood type is
Oαβ (I)	Aβ (II)	Bα (III)	
			O (I)
			A (II)
			B (III)
			AB (IV)
Control sample with AB (IV) sera 			

**Revealing other combinations of agglutination reactions testifies to improper blood typing!**

*Errors* while determining blood type are possible in situations, when agglutination is not revealed or a false agglutination occurs.

The absence of agglutination may be due to the following causes: 1) retardation of this reaction at high temperature of the environment > 25 °C (blood typing should be done only at the room temperature of 15–25 °C); 2) addition of an excess of tested blood to standard serums resulting in a decrease of agglutinin titer in their content (remember that a drop of the applied blood should be 5–10 times less than that of the serum); 3) weak activity of the standard serum or low agglutinin ability of red blood cells.

Revealing false agglutination in its real absence may be due to drying of a serum drop and formation of red blood cells “monetary columns” or appearance of cold agglutination at the temperature less than 15 °C. The addition of a drop of isotonic NaCl solution to the tested mixture of serum and blood and performing the test at the temperature higher than 15 °C allow avoiding the mentioned errors.

**Directions for recording the protocol:**

1. Fill in Tables 4.1 and 4.2.
2. Indicate in Table 4.2, when agglutination occurs (+) and when doesn't (-).
3. Make a drawing of obtained result.

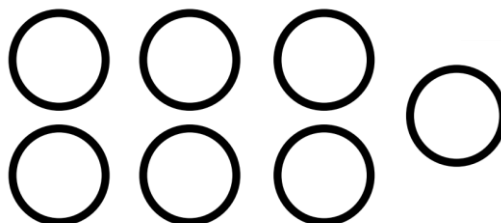
**PROTOCOL**

*Table 4.1*

Blood types	Serum agglutinins (antibodies)	RBC agglutinogens (antigens)
O (I)		
A (II)		
B (III)		
AB (IV)		

*Table 4.2*

Blood types	Standard sera			
	O (I) $\alpha\beta$	A (II) $\beta$	B (III) $\alpha$	AB (IV)
O (I)				
A (II)				
B (III)				
AB (IV)				



*Draw the experiment result*

**Conclusion:** the blood type is \_\_\_\_\_.  
 Its red blood cells \_\_\_\_\_ (contain/don't contain) agglutinogens \_\_\_\_\_.

### WORK 4.3. RHESUS (RhD) BLOOD TYPING (demonstration)

Determination of rhesus system blood types uses the same principle as ABO system blood typing. The tested whole blood (or red blood cells suspension) is mixed with the **universal anti-rhesus (anti-RhD) serum** containing antibodies to a rhesus-antigen. In case agglutination occurs, the blood is considered RhD<sup>+</sup>. The rhesus system, unlike ABO system, has no natural agglutinins, but they may appear in immunization of the organism with rhesus-incompatible blood.

**Materials and equipment:** a universal anti-rhesus reagent for the express-method; a pipette to it; a test-tube; 0.9 % solution of NaCl; lancet, gauze balls, antiseptic; iodine; rubber gloves, masks, 3 % chloramine solution.

**Accomplishment.** One drop of the universal anti-rhesus serum and one drop of the tested blood are applied to the bottom of the test-tube. The test-tube content is mixed by shaking up and then the tube is slowly bent almost to its horizontal position so that its content spreads about the walls — it makes the reaction more marked. As a rule, agglutination occurs within 1 minute, but to form a stable antigen-antibody complexes and clear agglutination and considering the possibility of retarded reaction in case of weak agglutination ability of red blood cells, the contact of blood with the reagent should be made by turning the test-tube in its horizontal position **no less than 5 minutes**. Then, to exclude non-specific red blood cells agglutination, 2–3 ml of NaCl isotonic solution are added into the test-tube and stirred, without shaking up, by 2–3-fold turning over the test-tube. The assessment is done visually.

Simultaneously with testing the whole blood a control test of standard rhesus-positive red blood cells of the same type or type I(O) in ABO system and standard rhesus-negative red blood cells of the same blood type in ABO system as the tested blood, is done.

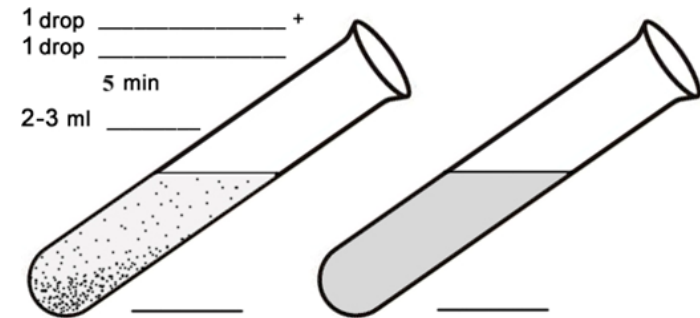
Agglutination presence manifested by flakes of red blood cells on the background of the cleared up fluid indicates that the tested blood is rhesus-positive (RhD<sup>+</sup>). Agglutination absence indicates that the tested blood is rhesus-negative (RhD<sup>-</sup>).

The result is considered authentic after checking up the control samples, i.e. in agglutination with standard rhesus-positive red blood cells and agglutination absence with standard rhesus-negative red blood cells belonging to the same blood type in ABO system as the tested blood.

#### Directions for recording the Protocol:

1. Add the view of the results of blood typing in Rhesus system to the picture of test tubes (for both cases, RhD<sup>+</sup> and RhD<sup>-</sup>).
2. Make a conclusion about Rhesus system blood type of the tested blood.

#### PROTOCOL



The tested blood is \_\_\_\_\_,  
as when it is mixed with the universal anti-Rhesus  
reagent in the test-tube, agglutination \_\_\_\_\_  
(is or is not observed)

## WORK 4.4. BLOOD TYPING IN THE ABO SYSTEM USING MONOCLONAL SERA

### Technique of blood typing using monoclonal sera

Per one large drop of anti-A, anti-B and anti-AB reagents is applied on a special plate or a porcelain dish under corresponding signs “anti-A”, “anti-B”, “anti-AB”. Next to reagent small drops of the tested blood are applied (proportion 2 : 1). The reagent is carefully mixed with the blood with glass sticks and incubated at room temperature (20–24 °C) for 5 minutes. Observation of the course of the reaction is done by rocking the plate for 1–2.5 minutes.

Agglutination with monoclonal reagents usually occurs within the first 3–5 sec. But the observation should be continued for 2.5 min due to a possibility of late agglutination with red blood cells containing weak types of antigens A and B.

### Blood types

#### Reaction of tested Red Blood Cells with monoclonal reagents

	anti-A ( $\alpha$ )	anti-B ( $\beta$ )	Anti-AB ( $\alpha, \beta$ )
O (I)	–	–	–
A (II)	+	–	+
B (III)	–	+	+
AB (IV)	+	+	+

**Accomplishment.** Work is performed using the computer program “PhysioEx”. To get started, select “Exercise 11: Blood Analysis” → “Activity 4: Blood Typing” → “Introduction (tab in the top menu)” and study the distribution of agglutinogens on the surface of red blood cells of different types, and method of determining blood types using monoclonal antibodies (video: Blood Typing wet-lab video, page 2 of 3).

Click on the “Experiment” tab. Take a tablet from the Blood Typing Slide Dispenser to determine blood types and drag it onto the worktable. After that, the wells on the tablet become denoted by the symbols A, B, and RhD. Drop the testing blood “Sample 1” into the wells, starting with “A”, then drop by drop anti-A, anti-B and anti-RhD reagents. Mix the blood with reagents with the help of Stirring Sticks of the appropriate color. Dispose of used sticks in the waste biomaterial package (Biohazard).

Place the tablet on the light table on the right side and click “Light”.

Click the “positive” on the image that appears under the drops, in which blood agglutination occurred, and “negative” under the drops in which agglutination did not occur. To record the results, click “Record Data”. Throw the tablet in a package for waste biomaterials (Biohazard).

By the same way, analyze blood samples number 2–6. To start the analysis, place the tablets on the desktop. After the finishing of the last sample analyzing, determine the test blood groups by selecting the appropriate line in the electronic protocol and clicking the “A”, “B”, “AB” or “O” and “+” or “–” buttons to specify the RhD factor. Enter the data in the laboratory protocol.

After the answering the program's question: “Why people with ABO (IV) RhD<sup>-</sup> blood type are known as universal recipients” and clicking “Check Answer” → “Submit” → “Submit”, you can, if necessary, re-see the results of blood group determination by clicking “View Experiment Results”.

#### Directions for recording the protocol:

1. Record the results. Determine the blood group in the test.
2. In the conclusion, indicate what the differences in the determination of blood types are with the help of standard isohemagglutinating sera and monoclonal anti-bodies. Explain the reason for the differences.

### PROTOCOL

Blood sample	Presence of agglutination (list “+” or “–” )			Blood type
	Anti-A	Anti-B	Anti-RhD	
1				
2				
3				
4				
5				
6				

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

## ADDITIONAL MATERIALS

### MONOCLONAL SERA: APPLICATION OF MONOCLONAL SERA ANTIBODIES IN BLOOD TYPING

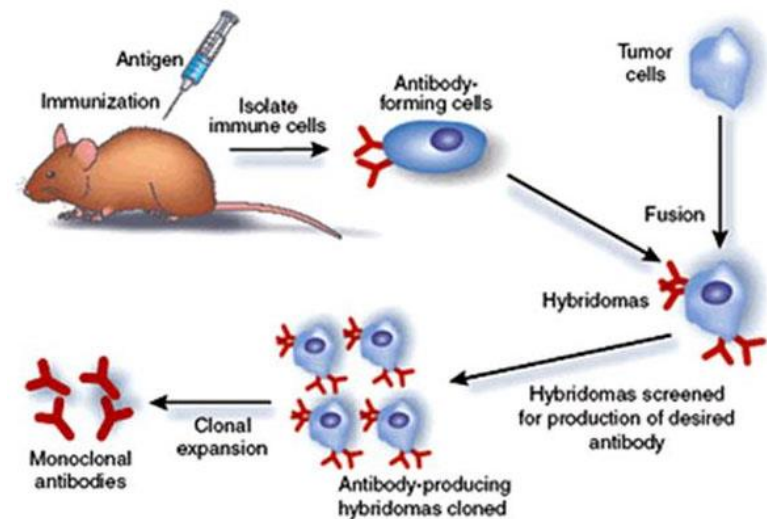
At present ABO-typing reagents produced from the human or animal serum with antibodies to red blood cells agglutinogens are still often used. These antibodies are the result of a polyclonal immune response, i.e. they come from various clones of antibody-forming cells and are the mix of immunoglobulins of various classes. To get such serums a great amount of donor blood is needed. Besides, the titer of natural antibodies in the human blood is usually low, that is why produced serums have low activity and one has to use serums obtained from specially immunized people.

Antibody-producing technology based on the fusion of a malignant myeloma cell and an antibody-forming lymphocyte of mice, becomes more and more widespread. As a result of fusion, a hybrid cell (hybridoma) is formed inheriting basic properties of its parents: immortality and the ability to constant growth — from a tumor cell, and the ability to produce antibodies — from a B-lymphocyte.

Antibodies secreted by cells-descendants of such hybrids are monoclonal, i.e. they come from one cellular clone, belong to one class of immunoglobulins, are aimed at one antigen, are standard and able to grow both in culture and in the mouse's organism as an ascite tumor producing antibodies in high concentrations, up to some tens of grams per liter.

To obtain ABO-typing monoclonal reagents it is enough to make a wash-out of tissue culture or take some ascite fluid and dilute these fluids as the titer of antibodies in them is very large (often for dilution 0.3 M solution of NaCl is used). At present ABO monoclonal reagents are commercially produced in England, Germany, Canada, Russia, Belarus, and other countries.

The benefits of monoclonal reagents are their high activity, standardization, reliability of revealing appropriate antigens, absence of false-positive reactions that is due, first of all, to the absence of antibodies of other specificity. Monoclonal reagents are not products of human cells that it excludes the possibility of transmitting viruses of hepatitis and HIV.



THE PRACTICAL WORKS ARE DEFENDED

Lecturer's signature

## SECTION

### “MECHANISM OF PHYSIOLOGICAL FUNCTIONS REGULATION. HUMORAL REGULATION”

**Session 5. FUNDAMENTALS OF INFORMATION EXCHANGE OF THE CELL WITH THE ENVIRONMENT. CHEMICAL SIGNALING. GENERAL PHYSIOLOGY OF THE ENDOCRINE SYSTEM**

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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. The concept of an autocrine, paracrine, endocrine and neuroendocrine ways of intercellular communication.</li> <li>2. Endocrine system. Its role in the regulation of physiological functions.</li> <li>3. The structures of the endocrine system (glands of internal secretion, diffuse elements) and its functions.</li> <li>4. Hormones, their chemical and functional classification, mechanisms of action. Basic ways of signal transmission. Second messengers.</li> <li>5. Methods of investigation of the endocrine system functions in humans.</li> <li>6. The structure and functions of the pituitary gland. Associations between pituitary gland the hypothalamus. Hormones of the pituitary gland (hypophysis) and hypothalamus, their role in the regulation of endocrine and not endocrine organs.</li> <li>7. The concept of the endocrine function of pineal gland (melatonin).</li> <li>8. Gonads. Male and female sex hormones and their physiological role.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 134–154, 215–249.</li> <li>3. <i>Severina, T. G.</i> Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – P. 14–21.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 5.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 299–335, 389–427.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 925–950, 1021–1054.</li> </ol>
<b>WORK 5.1. TERMINOLOGY</b>	
Hormone — _____	<p><b>Self-study questions:</b></p> <ol style="list-style-type: none"> <li>1. What types of receptors lipophilic and hydrophilic ligands bind to?</li> <li>2. What are the functions of <math>\alpha</math>-subunit of the G-protein? <math>\beta\gamma</math>-subunit?</li> <li>3. Why do the effects of thyroid and corticoid hormones develop slowly as compared to the effects of protein and peptide hormones?</li> <li>4. What are the ways of endocrine gland functional state evaluation?</li> <li>5. What are the effects of ACTH? What are the factors inhibiting its secretion?</li> </ol>
First messenger — _____	
Second messenger — _____	
Feedback system — _____	

**WORK 5.2. THE RECEPTOR MECHANISMS OF SIGNALS PERCEPTION. RECEPTORS AND THEIR TYPES**

Molecular (cellular) receptors — \_\_\_\_\_

Sensory receptors — \_\_\_\_\_

Classification of molecular (cellular) receptors	Corresponding ligands (examples)
Membrane receptors	
1.	
2.	
3.	
Intracellular receptors	
1.	
2.	

**Types of intercellular communication:**

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

**Types of signals carrying information**

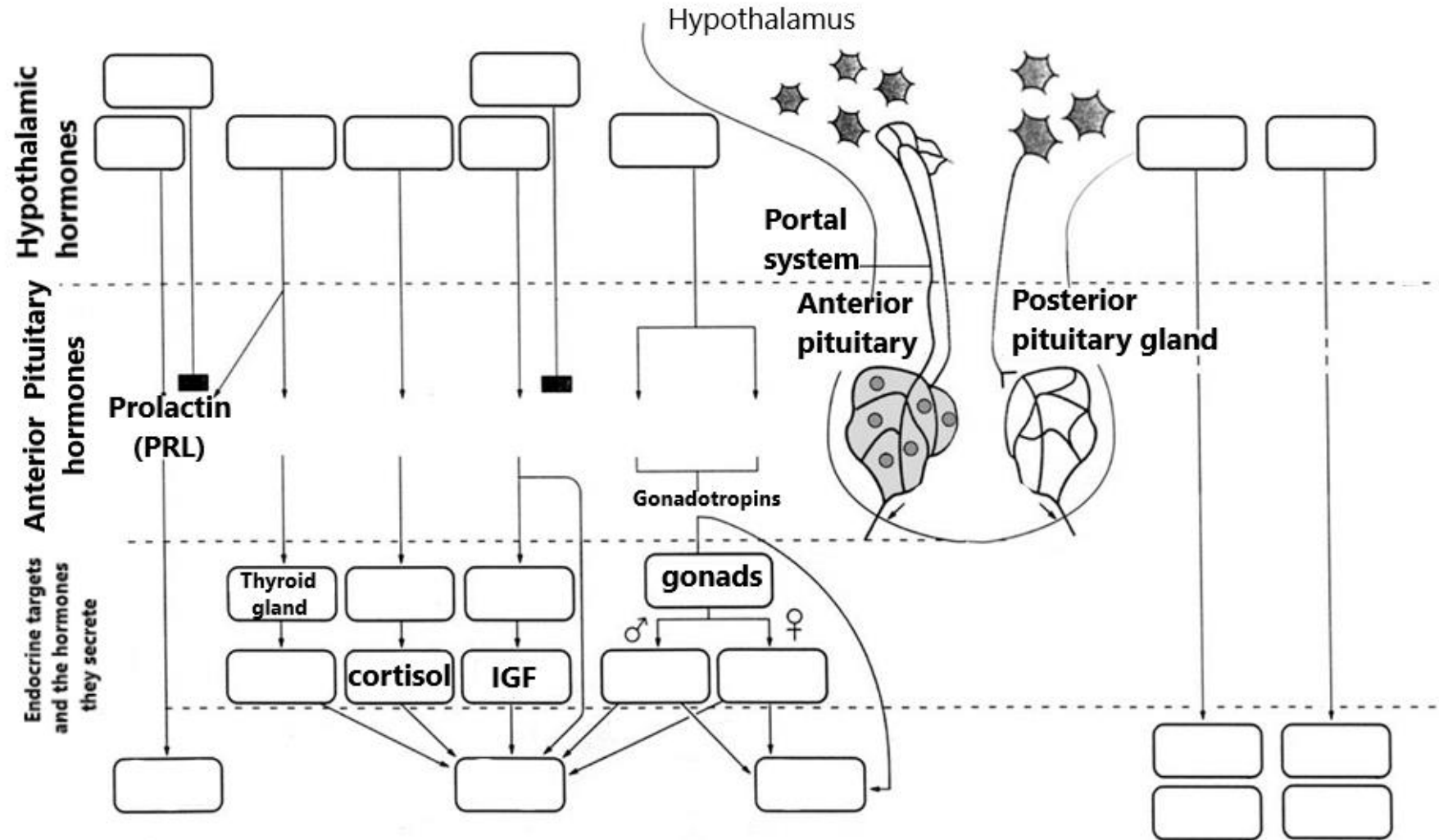
Physical nature:	
Chemical nature:	
Physicochemical nature:	
Complex signals:	

*Draw a schematic structure of membrane receptors & describe the mechanism*

7-Transmembrane receptors	1-Transmembrane receptors	Ligand-gated ion channels

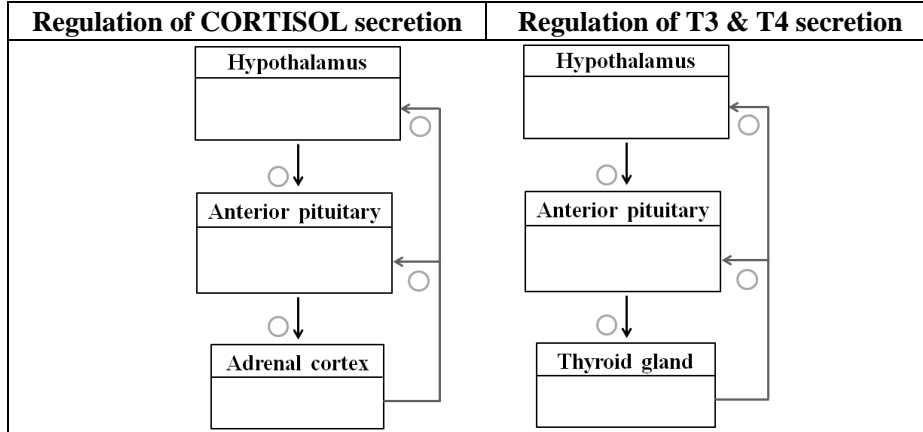
**WORK 5.3. STUDYING THE HORMONES OF THE HYPOTHALAMIC-PITUITARY SYSTEM**

Fill in the scheme (empty spaces). Use the materials of lectures, E-learning system, and textbook.

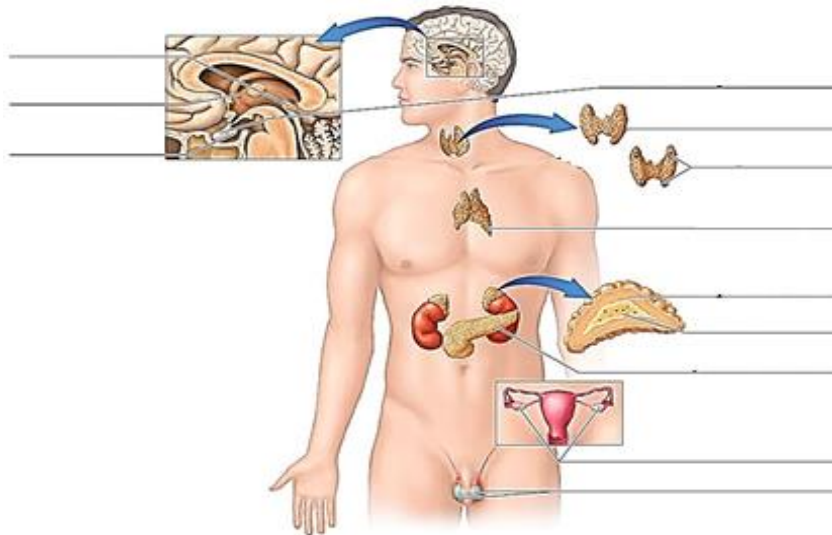


**WORK 5.4. STUDYING THE HORMONES OF THE HYPOTHALAMIC-PITUITARY SYSTEM**

Using materials of lectures and textbook fill in the names of the hormones (use abbreviations and corresponding full names) and indicate stimulating or inhibitory effects by inserting signs «+» or «-» into circles.



Fill in the scheme.



Fill in the table. Describe the main functions hormones released from pituitary gland.

Hormone	Function
<i>Anterior pituitary</i>	
1.	
2.	
3.	
4.	
5.	
6.	
<i>Posterior pituitary</i>	
1.	
2.	

## WORK 5.5. HUMAN HEIGHT MEASUREMENT

The body growth is an irregular process. Maximum growth rate is noted in newborns and infants and then it considerably decreases. Some increase of growth rate is noted in girls from 9 to 14 years and in boys from 11 to 16 years, then it decreases again. By 16 years in girls and by 18 years in boys the body growth is practically completed and in norm it does not exceed 1 cm/year. Complete ossification occurs by 20–23 years in a female organism and by 21–25 years in a male. **The height of an adult of 130–200 cm in males and 120–190 cm in females is considered normal.**

Men less than 130 cm in height and women <120 cm in height are dwarfs. People-giants are women higher than 190 cm and men higher than 200 cm.

Height is an integral factor of the effect of genetic, hormonal, tissue and external factors on the bony and other tissues of the organism. The height genetic program is realized through the endocrine system including all known hormones (thyroid, insulin, calcium-regulating, adrenal, sex), but the most important is hypothalamic-pituitary regulation of growth, the central link of which is somatotropin. Somatotropin (somatotropic hormone or growth hormone) is a basic hormone stimulating linear growth. Somatotropin stimulates growth of bones in length, growth and differentiation of internal organs, development of muscle tissue. A basic effect of somatotropin at a bony tissue level is its stimulation of cartilage growth, protein synthesis and cell mitosis induction. Somatotropin effects are mediated by insulin-like growth factors (IGF-I, IGF-II) or somatomedins that are synthesized under the action of this hormone mainly in the liver and kidneys. The linear human growth is completed, when growth zones have become closed under the effect of sex hormones.

The most simple and accessible method of studying the somatotropin function is anthropometric, i.e. the human height is evaluated versus its predicted height calculated on the basis of an average height of his parents. To determine the final height, range the following formula is used:

$$\text{Predicted final height of a male} = (\text{father's height} + \text{mother's height} + 13 \text{ cm}) : 2 + 4.5$$

$$\text{Predicted final height of a female} = (\text{father's height} + \text{mother's height} - 13 \text{ cm}) : 2 + 4.5$$

The measured height of an adult must coincide with a predicted height or deviate from a calculated value no more than 2 standard deviations (SD), i.e.  $\pm 2.6$  cm from a calculated height value. Deviations of the measured height exceeding 2 SD from a calculated height value evidence a pathologically low or high human height. In this case it is necessary to perform detailed studies of the pituitary somatotropic function to clear up the cause of growth impairment, as well as to study the state of other glands (first of all sex and thyroid glands).

**Materials and equipment:** a height meter.

**TO PERFORM THE WORK ONE SHOULD KNOW THE HEIGHTS OF THE PARENTS!**

**Accomplishment.** Height measurement is performed in standing position with the height meter. The examined should stand without shoes (in thin socks) in the right position: arms down; heels together; heels, buttocks and scapulae are pressed to the board of the height meter. The head is in position of “Frankfurt’s plane”, i.e. the lower edge of the eye and the external auditory canal should lie on one horizontal line. Measurements are performed on exhalation. The plank of the height meter is lowered to the level of the head of the examined. Measurements are performed with precision of 0.5 cm.

### PROTOCOL

1. Your height: \_\_\_\_\_ cm.  
Your sex: \_\_\_\_\_.
2. Your parents’ height:  
father’s \_\_\_\_\_ cm; mother’s \_\_\_\_\_ cm.
3. Calculate your predicted height (PH) (choose one for yourself)  
 $\text{PH } \text{♀} = (\text{father's height} + \text{mother's height} - 13 \text{ cm}) : 2 + 4.5 = \text{_____ cm.}$   
 $\text{PH } \text{♂} = (\text{father's height} + \text{mother's height} + 13 \text{ cm}) : 2 + 4.5 = \text{_____ cm.}$
4. **Conclusion.** Height of the examined is \_\_\_\_\_  
(in norm, pathologically high, pathologically low).
5. **Excess** of growth hormone in childhood or adolescence or insufficiencies of sex hormones may result in pathologically \_\_\_\_\_ height.
6. **Insufficiency** of growth hormone in childhood and adolescence or excess of sex hormones may result in pathologically \_\_\_\_\_ height.

**WORK 5.6. ASSESSMENT OF ENDOCRINE SYSTEM FUNCTIONS BASED ON CORTISOL AND ADRENOCORTICOTROPIC HORMONE CONCENTRATION IN BLOOD PLASMA**

Cortisol, a cortical adrenal hormone, is one of the essential hormones of the stress-releasing system. Cortisol secretion is controlled by the pituitary gland's **adrenocorticotrophic hormone** (ACTH). In turn, ACTH secretion is controlled by the **corticotrophin-releasing hormone** (CRH) of the hypothalamus. Increased cortisol secretion by the adrenal cortex leads to the inhibition of both ACTH and CRH secretion.

Increased plasma cortisol concentrations (hypercortisolism) resulting from adrenal tumors or administration of exogenous glucocorticoids (e.g., for treatment of rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, etc.) are called **Cushing's syndrome**. It is also sometimes called "steroid diabetes" because hypercortisolism leads to hyperglycemia. In this case, the ACTH content is usually decreased. In contrast, hypercortisolism caused by an adenohypophysis tumor is called **Cushing's disease**. In this case, there is an increase in plasma concentrations of ACTH and cortisol.

Decreased cortisol concentrations in the blood (hypocortisolism) can be observed in impaired adrenal cortex function. In primary adrenal insufficiency, known as **Addison's disease**, there is a decrease in cortisol concentration and a compensatory increase in ACTH secretion due to gradual damage to adrenal cortex cells. **Secondary adrenal insufficiency** is also characterized by decreased cortisol secretion, but the cause of this decrease is insufficient ACTH production, usually due to adenohypophysis damage. Various endocrine disorders can lead to high and low plasma levels of cortisol and ACTH (Table 5.1).

*Table 5.1*

<b>Endocrine disorder</b>	<b>Cortisol</b>	<b>ACTH</b>
Cushing's syndrome (primary hypercortisolism)	↑	↓
Iatrogenic Cushing's syndrome	↑	↓
Cushing's disease (secondary hypercortisolism)	↑	↑
Addison's disease (primary adrenal insufficiency)	↓	↑
Secondary adrenal insufficiency	↓	↓

**Materials and equipment:** plasma samples from five patients; HPLC (high-performance liquid chromatography) column with HPLC injector and detector are used for the quantitative determination of cortisol and ACTH content in blood plasma; reusable syringe.

**The progress of work.** The work is performed using the "14 PhysioEx" computer program. To start working, select "Access PhysioEx 9.0" → "Exercise 4: Endocrine System Physiology" → "Activity 4: Measuring Cortisol and Adrenocorticotrophic Hormone" → "Experiment (tab in top menu)."

1. On the HPLC detector, click on the "Cortisol" button to prepare the chromatograph column for isolation and measurement of cortisol.
2. Press the left mouse button (LMB) and place a syringe in the first tube with the plasma sample. Wait for the syringe to fill.
3. Press the LMB button and place the syringe needle in the HPLC injector. The plasma sample will enter the system and flow through the chromatography column. The first patient's plasma cortisol concentration will be displayed on the HPLC detector screen.
4. Press "Record Data" to save the result and enter the data into the protocol.
5. Press the "Clean" button under the syringe to clean it.
6. Press the "Clean Column" button on the HPLC detector to clean the chromatography column of cortisol residue.
7. Place a syringe into the tube containing the second patient's plasma sample. Wait for the syringe to fill.
8. Place the syringe needle into the HPLC injector. The plasma sample will enter the system and flow through the chromatography column. The concentration of cortisol in the blood plasma of a second patient will be displayed on the detector screen (HPLC detector).
9. Click "Record Data" to save the result and enter the data into the protocol.
10. Click the "Clean" button below the syringe to clean it.
11. Press the Clean Column button on the HPLC detector to clean the chromatography column. Answer the software question that appears on the left ("Stop & Think Question"). Then press "Submit".

**WORK 5.6. ASSESSMENT OF ENDOCRINE SYSTEM FUNCTIONS IN THE CASE OF CORTISOL CONCENTRATION AND ADRENOCORTICOTROPIC HORMONE IN BLOOD PLASMA (continuation)**

1. The plasma sample analysis will then be completed automatically. Sequentially place the syringe into the plasma samples of the remaining patients (beginning with the 3rd) and follow the above sequence.
2. After the cortisol concentration in the plasma of patient five is complete, press the "ACTH" button on the HPLC detector to prepare the chromatograph column for isolation and measurement of ACTH.
3. Repeat the steps described in 2–12 of this work.

**Directions for filling in the protocol.**

1. Record the results in the protocol (gray bars). Estimate the cortisol and ACTH concentrations of the test samples using the data from Table 5.2.

*Table 5.2*

Deviation from normal ranges in the morning hours	Cortisol level, µg/dL	ACTH level, pg/ml
Increased (↑)	≥ 23	≥ 80
Decreased (↓)	< 5	< 20

2. Conclude on the presence or absence of endocrine disorders in the examined patients and their causes.

**PROTOCOL**

Patient	Cortisol level, µg/dL	Cortisol level (↑/↓)	ACTH level, pg/ml	ACTH level (↑/↓)	Endocrine disorder
1					
2					
3					
4					
5					

*Patient #2 has rheumatoid arthritis and is receiving therapy with synthetic glucocorticoid (cortisol derivative) prednisolone tablets. How would this information change your conclusion?* \_\_\_\_\_

THE PRACTICAL WORKS ARE DEFENDED

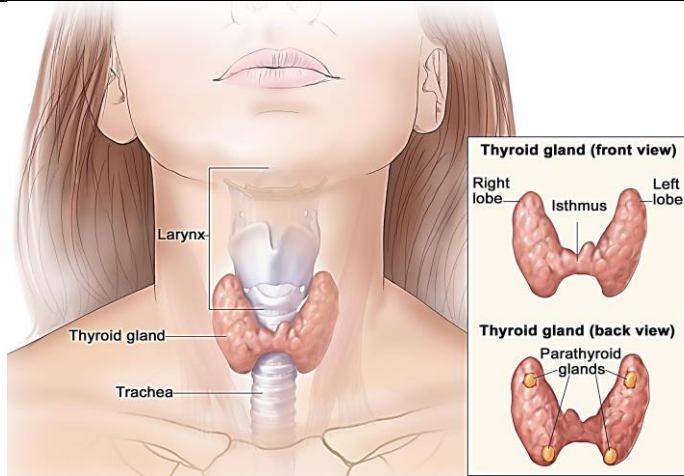
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## Session 6. SPECIAL PHYSIOLOGY OF ENDOCRINE SYSTEM

DATE  
 «      »      202    
           day                  month                  year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Thyroid gland: types of endocrine cells and their hormones. Thyroid hormones, mechanisms of their action and induced effects. Regulation of hormone secretion. Characteristic manifestations of excessive and insufficient secretion of hormones.</li> <li>2. Adrenal glands. Hormones of the adrenal cortex and medulla regulating body functions. Glucocorticoids, mineralocorticoids, sex hormones and catecholamines.</li> <li>3. The concept of stress, its mechanisms and methods of prevention.</li> <li>4. Endocrine function of the pancreas and its role in regulating carbohydrate, fat and protein metabolism. Somatostatin.</li> <li>5. Endocrine function of sex glands. Physiological role of male and female hormones.</li> <li>6. Endocrine mechanisms of water-electrolyte balance regulation (antidiuretic hormone, aldosterone, atrial natriuretic peptide).</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 155–181, 192–214.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 337–374, 429–449.</li> <li>4. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 951–1000.</li> </ol>
<p><b>WORK 6.1. TERMINOLOGY</b></p>	
<p>Sympathoadrenal system — _____</p>	<p>Metabolic action of the hormone — _____</p>
<p>Somatomedin C — _____</p>	<p>Physiological action of the hormone — _____</p>
<p>T<sub>3</sub> — _____</p>	<p>Thrombopoietin — _____</p>
<p>T<sub>4</sub> — _____</p>	<p>Erythropoietin — _____</p>
<p><b>Self-study questions:</b></p> <ol style="list-style-type: none"> <li>1. What evidences the excess and insufficiency of thyroid hormones?</li> <li>2. Why is the concentration of thyroid hormones in the peripheral blood decreased when the protein synthesizing function of the liver is impaired?</li> </ol>	<ol style="list-style-type: none"> <li>3. Which adrenal cortex hormones are vital hormones?</li> <li>4. In what way do glucocorticoids increase the blood glucose level?</li> <li>5. Which hormones are involved in maintaining water-electrolyte balance?</li> </ol>

## WORK 6.2. THE ENDOCRINE FUNCTION OF THE THYROID AND PARATHYROID GLANDS

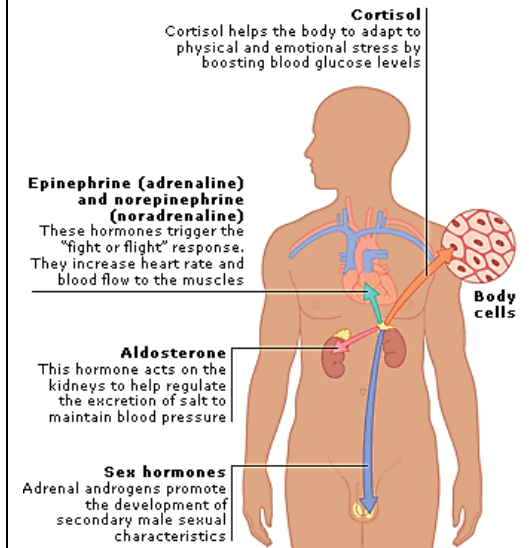
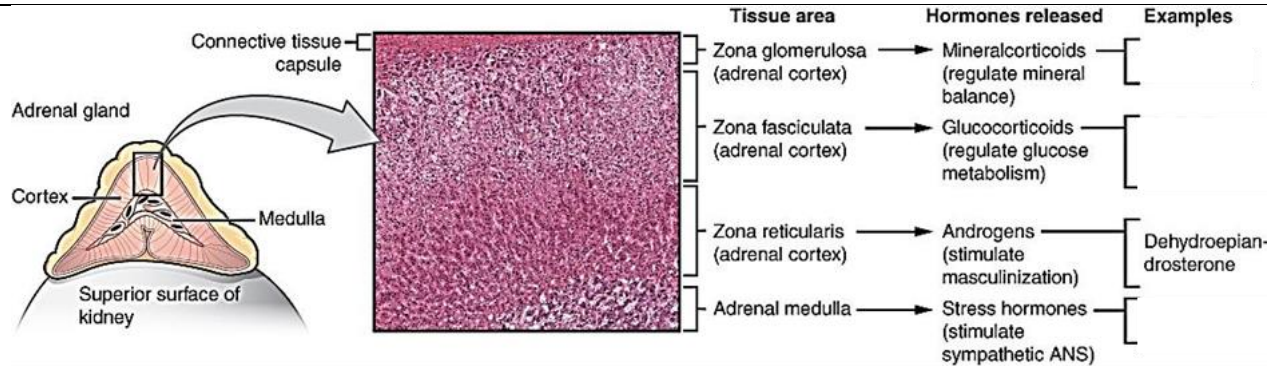


Write down the name of thyroid and parathyroid glands hormones:

- 1) \_\_\_\_\_
- 2) \_\_\_\_\_
- 3) \_\_\_\_\_

<b>In children</b>
Excess of T <sub>3</sub> and T <sub>4</sub>
Insufficiency
<b>In adults</b>
Excess of T <sub>3</sub> and T <sub>4</sub>
Insufficiency

## WORK 6.3. PHYSIOLOGY OF THE ADRENAL GLANDS



Fill in the table. Describe the main hormones of adrenal gland

Hormone	Functions
Aldosterone	
Cortisol	
Androgens	
Catecholamines	

**WORK 6.4. COMPARISON OF ARM MUSCLE STRENGTH IN MEN AND WOMEN**

Dynamometry is a method of measuring of the muscle contraction strength.

Muscle strength is an important indicator of their contractility, as well as the physical development of the human body. It is estimated by the weight of the load that can be held by the muscle at its maximum excitation, without changing its length.

Androgens are male sex hormones known to have anabolic effect on organs and tissues, especially skeletal muscle. Shoulder girdle muscles are highly sensitive to androgens. In this regard, there are marked sex differences in skeletal muscle mass and strength in man and women.

**Materials and equipment:** manual dynamometer (Fig. 6.1).

**Accomplishment:** Measure the muscle strength of the lead arm in all male and female students using hand-held dynamometer. Squeeze the handle of the dynamometer as hard as possible in your hand and record the obtained results (Fig. 6.2).

**Directions for recording the Protocol:**

1. Record the obtained results of the absolute muscle strength of the arm for all examinee.

2. Calculate the average muscle strength for men and women. Compare the results and make a conclusion based on the observed differences.



Fig. 6.1. Manual dynamometer

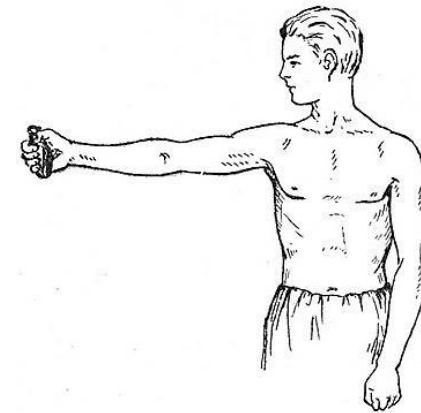


Fig. 6.2

**PROTOCOL**

Muscle strength	1	2	3	4	5	6	7	8	Average
Men									
Women									

**Conclusion:** *Higher/lower* (choose the right one) muscle strength of the arm in men compared to women depends on the high sensitivity these muscles to \_\_\_\_\_.

**WORK 6.5. ANALYSIS OF THE EFFECT OF CATECHOLAMINES AS HORMONES (OF ADRENAL MEDULLA) AND AS NEUROTRANSMITTERS (OF THE SYMPATHETIC PART OF ANS) ON CARDIOVASCULAR SYSTEM**

**Accomplishment.** The work is performed as virtual experiment on rats in the program “Physiol 2”.

**Directions for recording the Protocol:**

1. Fill in the table. Abbreviations: HR — Heart Rate, BPsyst — Systolic Blood Pressure, BPdiast — Diastolic Blood Pressure, BPmean — Mean Hemodynamic Blood Pressure.
2. Make a conclusion regarding the difference between the effects of catecholamines as neurotransmitters of sympathetic nerves and as hormones of the adrenal medulla. Indicate types of adrenoreceptors predominantly mediate the effects of noradrenaline and adrenaline on the cardiovascular system.

**PROTOCOL**

Effect on the heart	HR	BP <sub>syst</sub>	BP <sub>diast</sub>	BP <sub>mean</sub>
<b>Initial values</b>	161	98	53	66
<b>Stimulation</b> Symp. Nerves to heart T <sub>1</sub>	205	150	92	109
<b>Stimulation</b> Symp. Nerves to adrenals T <sub>6-8</sub>	213	138	74	93
Propranolol (β-adrenoblocker), 100 mg/kg + <b>stimulation</b> Symp. Nerves to heart T <sub>1</sub>	175	115	64	80
Propranolol (β-adrenoblocker), 100 mg/kg + <b>stimulation</b> Symp. Nerves to adrenals T <sub>6-8</sub>	168	142	98	111
<b>Injection</b> noradrenaline, 5 μg/kg	203	161	106	123
<b>Injection</b> adrenaline, 5 μg/kg	232	126	51	73

**Conclusions:** as the blockage of α-adrenergic receptors by phentolamine \_\_\_\_\_ (*does or does not*) prevent the sympathetic nerves effect on the heart, and the blockage of β-adrenergic receptors \_\_\_\_\_ (*does or does not*) prevent this effect, the sympathetic nerves effect on the heart is achieved through \_\_\_\_-adrenergic receptors.

Effect of *noradrenaline* is achieved mainly through \_\_\_\_\_ adrenergic receptors, while effect of *adrenaline* is achieved through both \_\_\_\_- and \_\_\_\_-adrenergic receptors.

THE PRACTICAL WORKS ARE DEFENDED

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Lecturer's signature

**Session 7. PHYSIOLOGY OF BONE TISSUE AND REGULATION OF CALCIUM-PHOSPHORUS METABOLISM**

DATE  
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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. The role of calcium and phosphate in the body, their compounds and content in bone tissue and teeth.</li> <li>2. Bone tissue: functions, features of the structure and composition, age-related changes. The concept of bone tissue remodeling.</li> <li>3. Hard tissues of teeth: types, functions. Enamel: structure, properties, functions, nutritional features.</li> <li>4. Dental formula for primary and permanent teeth.</li> <li>5. The balance of calcium and phosphate in the body and in bone tissue: age-specific features, regulatory mechanisms. The daily requirement in calcium, phosphate and fluoride.</li> <li>6. Factors for maintaining the health of bone tissue and teeth.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 181–192.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 7.</li> <li>4. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 62, 375–388.</li> <li>5. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 4. 1001–1020.</li> </ol>
<p><b>WORK 7.1. TERMINOLOGY</b></p>	
<p>Dentine — _____</p>	<p>Occlusion — _____</p>
<p>Enamel — _____</p>	<p>Dental formula — _____</p>
<p>Osteoblasts — _____</p>	<p>Daily requirements in phosphate — _____</p>
<p>Osteoclasts — _____</p>	<p>Daily requirements in calcium — _____</p>
<p>PTH — _____</p>	<p>Absorption — _____</p>



**WORK 7.3. METABOLISM OF CALCIUM AND PHOSPHORUS IN THE ORGANISM**

Please fill in the empty boxes using materials of lectures, E-learning system, textbooks and the corresponding sections of this practical book.

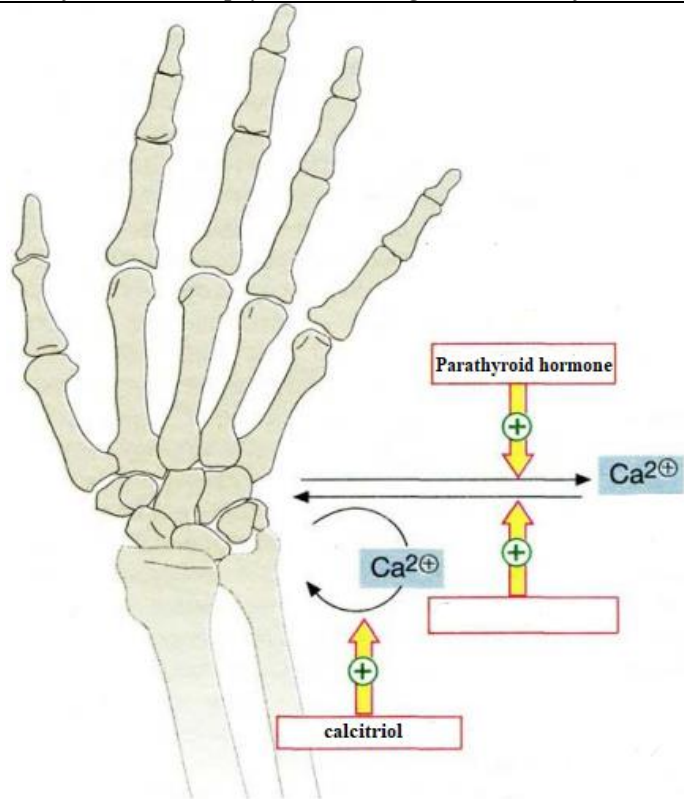


Fig. 7.2. Mechanism of Ca<sup>2+</sup> regulation

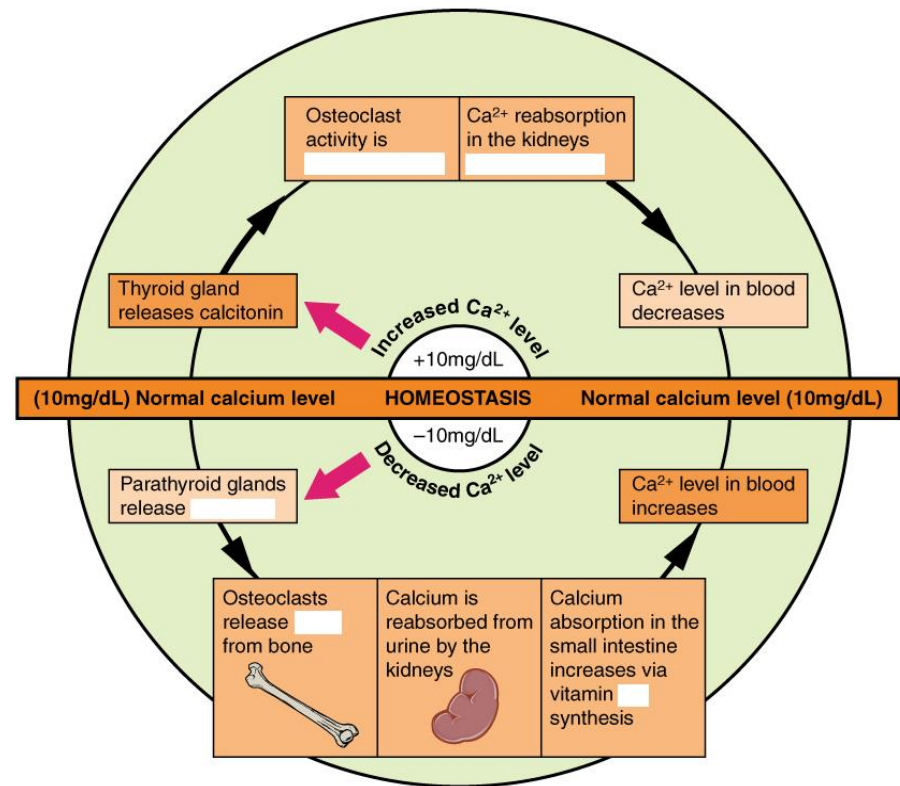


Fig. 7.3. Homeostasis of Ca<sup>2+</sup>

Fill in the table. Describe the main effects of Ca<sup>2+</sup> and P<sub>i</sub>-regulating hormones (indicate changes using symbols such as ↑↓).

Effects	Plasma Ca <sup>2+</sup> concentration	Plasma P <sub>i</sub> concentration	Intestinal Ca <sup>2+</sup> & P <sub>i</sub> reabsorption	Bone Ca <sup>2+</sup> & P <sub>i</sub> resorption	Kidney reabsorption of Ca <sup>2+</sup>	Kidney reabsorption of P <sub>i</sub>
PTH						
VitD <sub>3</sub>						
Calcitonin						

## WORK 7.4. THE EFFECT OF FEMALE SEX HORMONES ON BONE MINERALIZATION

Bone tissue is constantly being formed and resorbed. Usually, the processes of bone formation and bone resorption balance each other. The activity of osteoblasts and osteoclasts is regulated by parathyroid hormone, calcitonin, estrogens, vitamin D, cytokines, and other local factors (e.g., prostaglandins).

Bone mass in men and women peaks around the age of 30 (more in men than in women; more in the Negro race than in the Caucasoid and Mongoloid races). After peaking for about 10 years, bone mass remains constant, at which time bone resorption processes are roughly equal to its formation. It then begins to decline at about 0.3–0.5 % per year. With the onset of menopause in women, the loss of bone tissue accelerates and reaches about 3–5 % per year for about 5–7 years, then the rate of bone mass loss gradually decreases.

With the onset of menopause, the ovaries stop producing hormones, including estrogens, which is a critical cause of decreased bone density and osteoporosis in women. In severe cases, this can even lead to domestic fractures. Cases have been described of fractures of the femoral neck in older women when turning from one side to the other in bed or fractures of the radius when trying to lift a frying pan. Hormone replacement therapy is used to treat menopausal syndrome to prevent such consequences. One of the hormones that can increase bone density in women is **estrogen**. Estrogen suppresses osteoclasts' activity, allowing the bone matrix's structure to be maintained. **Calcitonin**, which stimulates bone mineralization, has a similar effect on bone.

The effects of estrogen on a woman's body are manifold, but in this paper, we will look at its effectiveness in preserving bone mass and protecting against **osteoporosis**.

Dual-energy X-ray absorptiometry is used to assess bone density (Fig. 7.4).

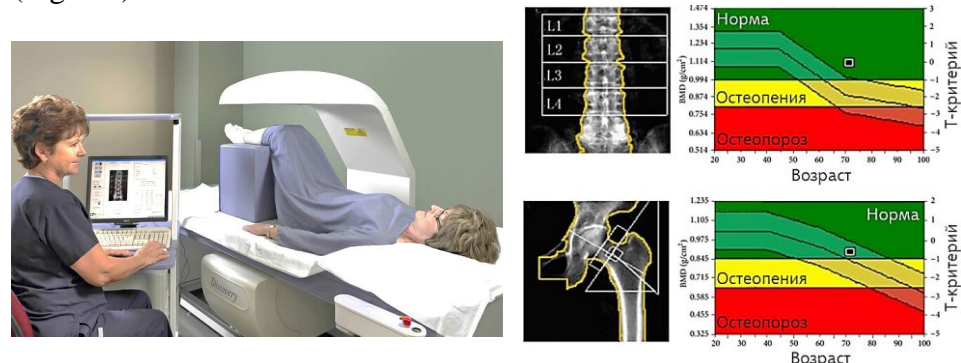


Fig. 7.4. Bone densitometry (left — methodology, right — evaluation)

A quantitative indicator of bone mineralization, the T-criterion, will be used to assess the state of bone tissue. This criterion **Остеопороз** nds to the number of standard deviations by which bone density differs from its peak value in young, healthy people of the same gender and ethnicity. The WHO has established the T-criterion limits that define osteopenia and osteoporosis (Table 7.1).

**Остеопороз** Table 7.1

Assessment of T-criterion	
Normal	+1 to -0.9
Osteopenia	-1 to -2.49
Osteoporosis	-2.5 and lower

This work uses computer models of three rats with removed ovaries. The initial T-criterion of each rat is **-2.61**, indicating osteoporosis. We are analyzing the effect of estrogen, calcitonin, and, for comparison, saline solutions on bone density under densitometry control.

### WORK 7.4. THE EFFECT OF FEMALE SEX HORMONES ON BONE MINERALIZATION (continuation)

**Progress of work.**

1. Open the “**PhysioEx**” program on your desktop.
  2. Click on the “*Access PhysioEx 9.0*” hyperlink.
  3. Click on “Exercise 4: Endocrine System Physiology”. In the open list, select “Activity 3: Hormone Replacement Therapy”. Select the “Experiment” tab in the list that appears.
  4. The laboratory simulator is in front of you. Click on the syringe with the left mouse button (LMB) and drag it onto the saline bottle. After the syringe is filled with solution, squeeze it with the left mouse button and drag it to rat #1 (signed “Control”). The saline solution will be injected intraperitoneally. The number of injections will be displayed below the rat — “1”. Clean the syringe by pressing the “Clean” button.
  5. Clamp the syringe with the LMB and drag it to the vial with the estrogen solution (“Estrogen”). After the syringe is filled with solution, squeeze it with the LMB and drag it to the rat signed “Estrogen treated”. Clean the syringe by pressing the “Clean” button.
  6. Squeeze the syringe with the LMB and drag it to the vial with the calcitonin solution (“Calcitonin”). Once the solution is in the syringe, clamp the syringe with the LMB and drag it to the rat signed “Calcitonin treated”. Clean the syringe by pressing the “Clean” button.
  7. Click on the clock at the top of the screen. The clock will scroll for one day. The new dose of solution will be injected automatically. Press the clock until the number of elapsed days of the experiment (“Elapsed days”) is 7. Pressing the clock repeatedly does not speed up the program; be patient!
- At the end of the 7 days, you will be asked to take a quiz in which you have to guess what changes will happen to the rat’s bone tissue in response to the solutions injected.
8. After completing the test, measure the T-criterion for the rats being tested. To do this, press “Anesthesia” in the upper left corner of the “Control” picture of the rat. Press and hold the LMB of the rat picture and drag it to the X-ray densitometer table (“Exam-table”). Press the “Scan” key (bottom left), then press the “Record Data” key on the right. Repeat the operation for rats that received estrogen and calcitonin replacement hormone therapy.
  9. In the table at the bottom of the screen you can find the T-criterion (“T-score”) for the tested rats.

**Directions for completing the protocol.**

Using the data obtained fill in the table and: 1) draw a conclusion about the effect of the hormones tested on bone density; 2) compare their effectiveness in rats with removed ovaries.

#### PROTOCOL

Solution	T-criterion	Conclusion:
Initial	<b>-2.61</b>	1) _____
0.9 % NaCl (control)		2) _____
Estrogen		_____
Calcitonin		

THE PRACTICAL WORKS ARE DEFENDED

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Lecturer’s signature

**Session 8. COLLOQUIUM. CONCLUDING SESSION ON THE SECTIONS “INTRODUCTION TO THE ACADEMIC DISCIPLINE “NORMAL PHYSIOLOGY”. THE BASIC CONCEPTS OF PHYSIOLOGY. PRINCIPLES OF BIOMEDICAL ETHICS”, “THE INTERNAL ENVIRONMENT OF THE HUMAN BODY. PHYSIOLOGY OF THE BLOOD”, “MECHANISM OF PHYSIOLOGICAL FUNCTIONS REGULATION”**

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**THEORETICAL QUESTIONS:**

1. The subject of normal physiology. The main stages of the development of physiology, the important discoveries and methodological approaches that contributed to its advancement as scientific discipline. Contribution of domestic scientists to the development of physiology. Physiology as a scientific basis of medicine. Application of knowledge of normal physiology by a dentist.

2. Blood system. Composition, amount, properties, basic functions of blood. Blood plasma. Organic and inorganic compounds of the blood plasma. Basic physiological constants of blood maintaining homeostasis.

3. Acid-base state of blood. Physicochemical and physiological mechanisms that ensure the constancy of blood pH. The concept of acidosis and alkalosis. Acid-base state of the oral cavity.

4. The role of water in the body, its content, distribution, balance. Electrolyte composition of blood plasma. Osmotic pressure of blood and its regulation (ADH, RAAS, etc.).

5. Proteins of the blood plasma, their classification, physiological role. Oncotic (colloid-osmotic) pressure of plasma and its role. Blood viscosity and its changes according to water balance, its influence on hemodynamics.

6. Red blood cells (erythrocytes). The amount of RBC in the blood. Features of the structure and properties of erythrocytes, ensuring the performance of their functions. The amount of hemoglobin. Hemoglobin variants at different age periods. Physiological and pathological types of hemoglobin.

7. White blood cells (leukocytes). Number and subsets of WBC. Features of the structure and properties, ensuring the performance of their functions. Distribution of WBC in the vascular bed, in tissues, its features and physiological significance. Leukocyte formula, leukocyte formula shift. Leukocytosis and leukopenia.

8. Platelets: features of structure, number, functions. The concept of the hemostasis system and its links. Primary and secondary hemostasis. The main evaluation methods of hemostasis in outpatient conditions. Duration of bleeding after tooth extraction.

9. Nervous and humoral regulation mechanisms of hematopoiesis. The role of vitamins (B12, B9, etc.) and microelements (Fe<sup>2+</sup>, etc.) to perform hematopoiesis.

**LITERATURE**

*Main*

1. Lecture & E-learning system.
2. *Moroz, V. M.* Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016.
3. *Severina, T. G.* Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. Minsk : BSMU, 2017.

*Additional*

4. <http://etest.bsmu.by/> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 8.

**Form of colloquium:**

1. *Theory*
2. *Practice (practical skill)*

10. Blood type systems (ABO, RhD, HLA, etc.). ABO system: antigens (agglutinogens) and antibodies (agglutinins) of blood types, their features. Formation of agglutinogens and agglutinins of the ABO system. The role of agglutinogens and agglutinins in determining blood type in the ABO system. Their combinations in different types of the ABO system.

11. Blood type systems. Blood type of the Rhesus (RhD) system, features of antigens and antibodies. Formation of antigens and antibodies during ontogenesis, differences between the Rh system and the ABO system. Consequences of transfusion of incompatible blood according to the Rh system. Rhesus-conflict.

12. Principles of blood transfusion. Risk factors while working with blood: for medical personnel, patients, donors. Blood substitute solutions, their classification according to the type of performing function in the body and indications for them.

13. The concept of physiological function and its regulation. Systemic principle of regulation of functions. Types of regulation of body functions. Nervous and humoral mechanisms of regulation of functions, their comparative characteristics.

14. The concept of the endocrine system. Pituitary gland, its relationship with the hypothalamus. Pituitary and hypothalamic hormones, their role in the regulation of endocrine and non-endocrine organs.

15. Endocrine function of the thyroid and parathyroid glands. Mechanisms of hormones action and their effects. Typical manifestations of excessive or insufficient hormones secretion.

16. Adrenal glands. Hormones of the outer cortex and the inner medulla. Mechanisms of hormones action and their effects. Regulation of hormone secretion. Typical manifestations of excessive or insufficient hormones secretion.

17. Endocrine function of the pancreas. The role of pancreatic hormones in the regulation of carbohydrate, fat and protein metabolism. Regulation of hormone secretion. The concept of normo-, hypo- and hyperglycemia and their causes.

18. Sex hormones. Mechanisms of hormones action and their effects. Mechanisms of regulation of hormone secretion. Typical manifestations of excessive or insufficient secretion of hormones.

19. Regulation of calcium and phosphorus metabolism in the body. Influence of calcitonin, parathyroid hormone and vitamin D3 on calcium and phosphorus metabolism. Daily needs for calcium and sources of its intake. The role of vitamin D3.

20. Hormonal mechanisms for maintaining water-electrolyte balance in the body (antidiuretic hormone, renin-angiotensin-aldosterone system, atrial natriuretic factor). Indicators of water-electrolyte balance. Sources and ways of water excretion in the human body.

21. The concept of endocrine function of the epiphysis (melatonin), heart (atriopeptides), kidneys (calcitriol, erythropoietin, etc.), salivary glands (parotin P, etc.), liver (somatomedins, thrombopoietin).

**Practical skills:**

1. Measures to prevent infection with viral hepatitis and human immunodeficiency virus (HIV) during the blood and other biological materials analysis.
2. Physiological assessment of complete blood count parameters (red blood cells count, hematocrit, hemoglobin, color index and RBC indices, white blood cells count and leukocyte formula, platelet count, Panchenkov's ESR method).
3. Assessment of primary hemostasis indices (bandage test). Features of bleeding duration from the tooth cavity.
4. Assessment of blood typing results in ABO and RhD systems using standard sera and monoclonal antibodies.
5. Measurement and evaluation of height. Evaluation of endocrine system functions (height as index of endocrine axis hypothalamus-pituitary-liver).
6. Evaluation of endocrine system functions (comparison of muscle strength of men and women, axis hypothalamus-pituitary-sex glands).
7. Dynamometry (manual) and physiological evaluation of the results.
8. Evaluation of primary and permanent teeth dental formula.

**Colloquium is** \_\_\_\_\_  
(Lecturer's signature, date)

Mark for theoretical part: \_\_\_\_\_

Mark for practical part: \_\_\_\_\_

## SECTION “GENERAL PHYSIOLOGY”

### Session 9. ELECTRICAL SIGNALING. LAWS OF EXCITABLE TISSUES. BIOLOGICAL POTENTIAL. EXCITABILITY CHANGES DURING EXCITATION

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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Electrical signaling. The concept of irritability, excitability and excitation. Basic manifestations of excitation. Types of electrical signals, their physiological significance.</li> <li>2. Stimulus parameters necessary for tissue response (thresholds of force and time, minimal gradient). The “force-duration” curve. Chronaxy, chronaximetry.</li> <li>3. Basic laws of excitable tissues response to the stimulus action.</li> <li>4. The resting membrane potential. Basic mechanisms of maintaining the resting potential.</li> <li>5. The action potential (AP) as a unit of information transfer in the nervous system. Phases and ion mechanisms of AP generation.</li> <li>6. Excitability alteration during action potential.</li> <li>7. Comparative characteristics of the receptor potential and action potential</li> <li>8. Sensory receptors. Classification, structure and functions of sensory receptors.</li> <li>9. The receptor potential, the mechanism of its origin and characteristics.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 9–27, 144–148, 635–645.</li> <li>3. <i>Severina, T. G.</i> Physiology of excitable tissues: textbook / T. G. Severina. – Minsk : BSMU, 2024. – P. 3–18.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 9.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 53–64, 89–93, 159–160.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 61–74, 577–578, 931–935.</li> </ol>
<b>WORK 9.1. TERMINOLOGY</b>	
Excitable tissues: 1) _____ 2) _____	Resting membrane potential (RMP) — _____
Excitability — _____	_____
Types of biopotentials: 1) _____ 2) _____ 3) _____	The main factors determining RMP value are: 1) _____ 2) _____ 3) _____
Action potential — _____	Graded potential — _____

Depolarization — _____	Graded potential — _____
Repolarization — _____	Receptor potential — _____
The ions permeability ratio at rest ( $P_{K^+} : P_{Na^+} : P_{Cl^-}$ ) — 1 :     :	The ions permeability ratio during the excitation ( $P_{K^+} : P_{Na^+} : P_{Cl^-}$ ) — 1 :     :
Law “all-or-none” — _____	Law of force (“strength-duration” curve) — _____
<p><b>Self-study questions:</b></p> <ol style="list-style-type: none"> <li>1. What factor allows comparing excitability in various cells? Compare the excitability of the nervous and striated muscle tissue.</li> <li>2. Why does the cardiac muscle response to stimulus according to the “all-or-none” law, while the skeletal muscle — according to the law of force?</li> <li>3. What type of membrane channels do participate in the formation of the resting potential and what type of channels are necessary for generation of the action potential?</li> <li>4. What is the effect of an increase in extracellular potassium ions concentration on the resting membrane potential value?</li> <li>5. When the myocardial blood supply is impaired, and the potassium ions concentration in the interstitial fluid increases, how this will affect on the AP generation in myocardial fibers?</li> <li>6. What is the depolarization threshold for excitable cells? What factors can change the threshold values and why?</li> </ol>	<p style="text-align: center;"><b>Self-study assignment</b></p> <p>Fill in the tables.</p> <p>The graph shows the membrane potential over time. The y-axis is labeled 'Membrane potential (mV)' with values -35, -55, E<sub>tr</sub>, E<sub>0</sub>, and -75. The x-axis is labeled 'Time (msec)'. A dashed horizontal line at E<sub>tr</sub> represents the threshold. The resting potential is at E<sub>0</sub>. The potential rises to the peak at -35 mV and then falls. Labels include 'Membrane potential difference (V<sub>m</sub>)', 'V<sub>m</sub> decreases', and 'V<sub>m</sub> increases'. Two empty boxes are provided for student input.</p>

**WORK 9.2. STUDYING CHANGES IN EXCITATION AND EXCITABILITY OF CELL MEMBRANE IN DIFFERENT PHASES OF ACTION POTENTIAL**

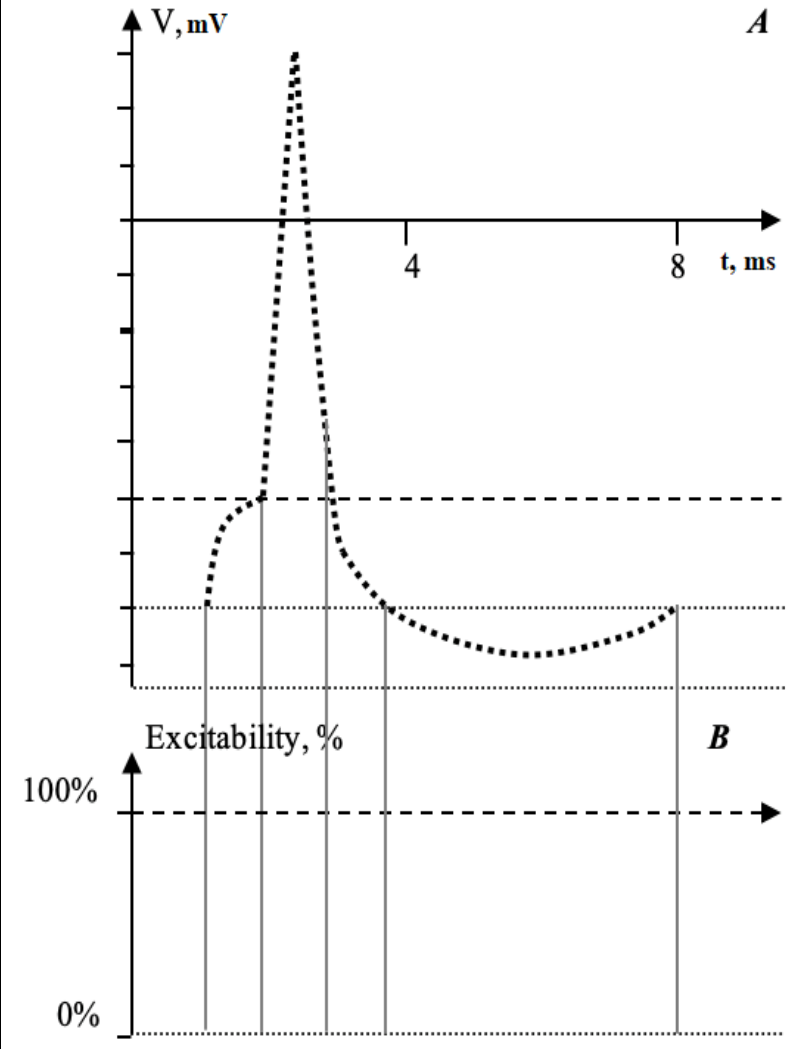
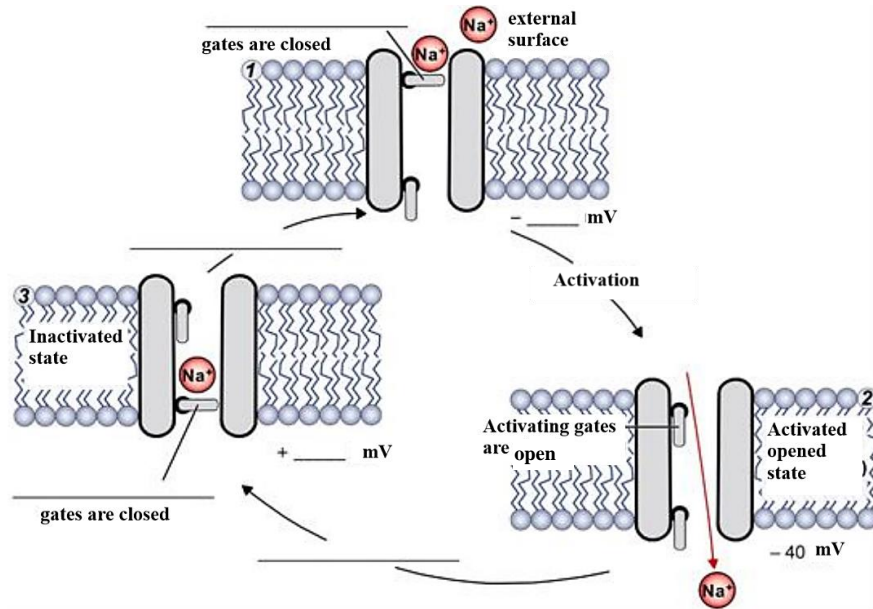
Fill in the table: Describe the mechanism of each AP phase.

AP phase	Mechanism
Fast depolarization	
Fast repolarization	
Hyperpolarization	

Fill in the table: Compare the action potential phase and changes of excitability.

AP phase	Excitability
Resting membrane potential	100 %
Graded potential change	> 100 %, Exaltation
Fast depolarization	
Fast repolarization	
Hyperpolarization	

Fill in the blankets.



**WORK 9.3. THE EFFECT OF Na<sup>+</sup> AND K<sup>+</sup> IONS ON THE RESTING MEMBRANE POTENTIAL AND ACTION POTENTIAL. VIRTUAL PROGRAM “NMJ” (“NEURO MUSCULAR JUNCTION”)**

1. Students have to perform the work in a computer class (room 104). The student uses the **NMJ** program (link on the desktop). The NMJ program is a virtual simulator of operations on an isolated neuromuscular preparation placed in Ringer's solution (Fig. 9.1). It is possible to stimulate both the muscle fiber and the nerve, and to change the concentration of ions in the solution.

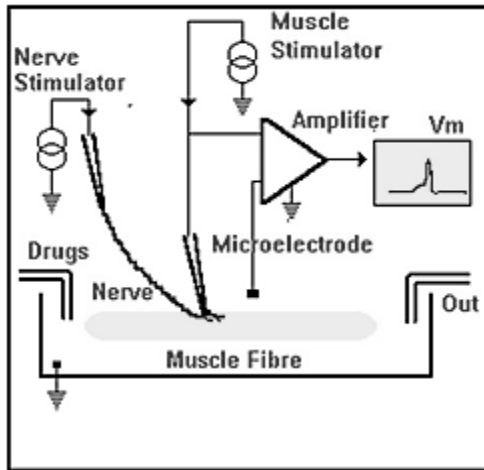


Fig. 9.1

2. Click in the upper line (menu):
- 1) Ions → potassium (K<sup>+</sup>) → 5 mM, sodium (Na<sup>+</sup>) → 120 mM;
  - 2) Stimulated → Nerve;
  - 3) Clipboard → Copy to clipboard (Fig. 9.2–9.4).

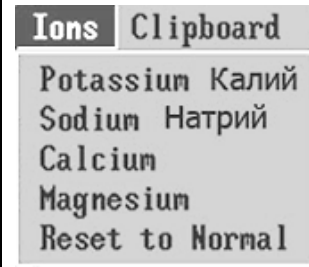


Fig. 9.2

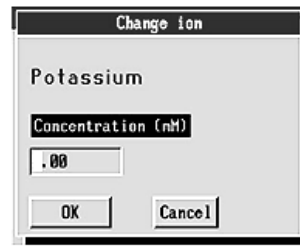


Fig. 9.3

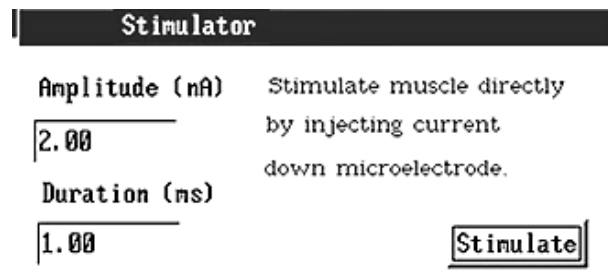


Fig. 9.4

3. You will receive an image, two graphics, as in Fig. 9.5, where the value of RMP (resting membrane potential) (arrow 1) is shown under conditions of optimal content of K<sup>+</sup> (potassium) and Na<sup>+</sup> (sodium) ions in Ringer's solution (arrow 2) (Fig. 9.5) and AP (action potential) graphs during electrical stimulation of muscles with electrical current with a force of 2 mA for 1 ms (Fig. 9.4).

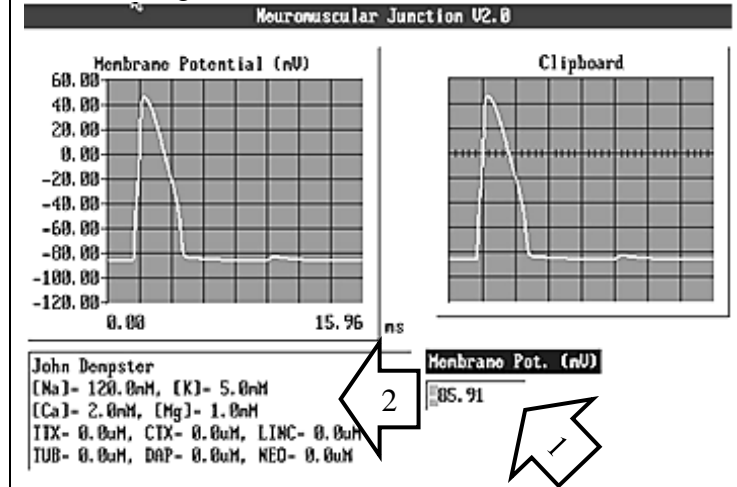


Fig. 9.5



Fig. 9.6

4. After that the program allows you to change the concentrations of electrolytes (potassium and sodium) in solution using commands “Ions” (Fig. 9.2.) and “Change ion concentration” (Fig. 9.3). You must record the values of RMP and AP (Fig. 9.6) after the indirect electrical stimulation of muscle with the same single electric current amplitude of 2 mA for 1 ms: “stimulate → nerve”.

**WORK 9.3. THE EFFECT OF Na<sup>+</sup> AND K<sup>+</sup> IONS ON THE RESTING MEMBRANE POTENTIAL AND ACTION POTENTIAL. VIRTUAL PROGRAM “NMJ” (“NEURO MUSCULAR JUNCTION”) (continuation)**

**Directions for recording the Protocol:**

1. Simulate the change in membrane potentials (RMP and AP) by indirect electrical stimulation of muscles under conditions of optimal concentration of K<sup>+</sup> and Na<sup>+</sup> ions, as well as increasing and decreasing their concentration (according to the instructions in Table 9.1) in the solution surrounding the neuromuscular preparation.
2. Record the results of RMP and AP changes in the table 9.1.
3. Draw on fig. 9.7 the resulting graphs of RMP and AP using colored pencils.
4. Explain the effect of the changes in the concentration of the K<sup>+</sup> and Na<sup>+</sup> ions on the values of RMP and AP.

5. Calculate  $\Delta E$  if  $E_{\text{threshold}} = -40 \text{ mV}$

$$\Delta E = E_{\text{threshold}} - E_0$$

1. If  $C_{K^+} = 5 \text{ mM}$  and  $C_{Na^+} = 120 \text{ mM}$ :

$$\Delta E = \underline{\hspace{2cm}}$$

2. If  $C_{K^+} = 8 \text{ mM}$  and  $C_{Na^+} = 120 \text{ mM}$ :

$$\Delta E = \underline{\hspace{2cm}}$$

3. If  $C_{K^+} = 2 \text{ mM}$  and  $C_{Na^+} = 120 \text{ mM}$ :

$$\Delta E = \underline{\hspace{2cm}}$$

4. If  $C_{K^+} = 5 \text{ mM}$  and  $C_{Na^+} = 160 \text{ mM}$ :

$$\Delta E = \underline{\hspace{2cm}}$$

**PROTOCOL**

Table 9.1

The extracellular concentration of ions			The magnitude of the potentials	
potassium	sodium		resting (RMP)	action (AP)
<b>5 mM</b>	120 mM	Copy to clipboard	-85.9 mV	+45 mV
<b>8 mM</b>	120 mM	Copy to clipboard		
<b>2 mM</b>	120 mM	Copy to clipboard		
Clipboard → clear				
5 mM	<b>120 mM</b>	Copy to clipboard	-85.9 mV	+45 mV
5 mM	<b>160 mM</b>	Copy to clipboard		
5 mM	<b>100 mM</b>	Copy to clipboard		

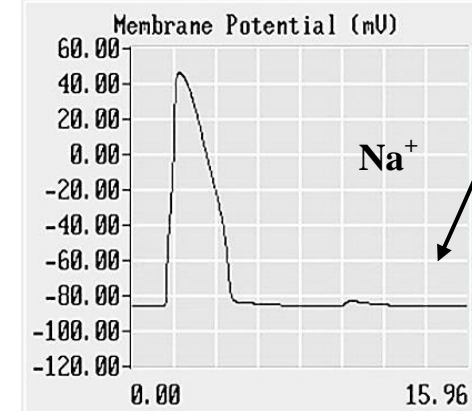
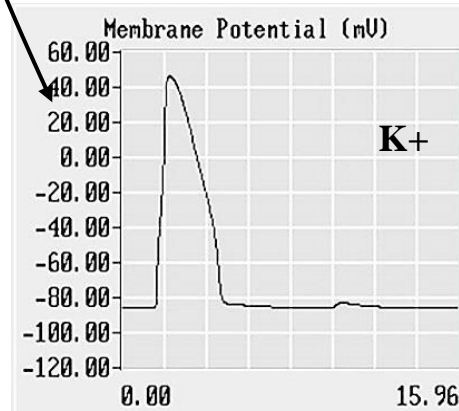


Fig. 9.7

**Conclusion:** the concentration of potassium ions in the extracellular fluid determines the *resting/action* potential, while the content of sodium ions determines the amplitude of the *resting/action* potential. Excitability increases in case of \_\_\_\_ membrane potential.

THE PRACTICAL WORKS ARE DEFENDED

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**Session 10. EXCITATION CONDUCTION ALONG NERVE FIBERS.  
NEUROMUSCULAR SYNAPSE**

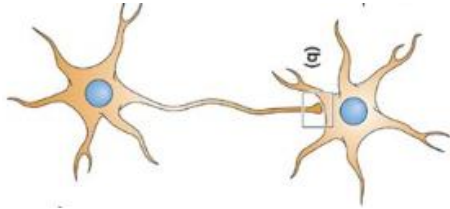
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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Physiological role of nerve fiber structural elements. Classification of nerve fibers. The role of afferent and efferent nerve fibers.</li> <li>2. Mechanism of excitation conduction along myelinated and unmyelinated nerve fibers, laws of excitation conduction.</li> <li>3. Axonal transport. Physiological basis of conduction anesthesia in dental practice.</li> <li>4. Synapse. Classification of synapses, their physiological role and functional properties.</li> <li>5. Structure of electrical and chemical synapse. Receptors of postsynaptic membrane.</li> <li>6. Mechanism of signal transmission in the neuromuscular junction. End-Plate potential (EPP). Its transformation into an action potential. Acetylcholinesterase, its role. Types of channels in synaptic membranes.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning materials.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 17–18, 47–54, 66–75.</li> <li>3. <i>Severina, T. G.</i> Physiology of excitable tissues: textbook / T. G. Severina. – Minsk : BSMU, 2024. – P. 17–29.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 10.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 85–90, 93–95, 121–135.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 69, 71–72, 89–92, 580–592.</li> </ol>
<p><b>WORK 10.1. TERMINOLOGY</b></p>	
<p>Nerve fibers are _____</p>	<p>Types of nerve fibers: 1) _____ 2) _____</p>
<p>Continuous conduction is _____</p>	<p>Saltatory conduction is _____</p>
<p>Nervous tissue consists of cell types: 1) _____ 2) _____</p>	<p>EPP is _____</p>
<p>Acetylcholinesterase is _____</p>	<p>Axon hillock is _____</p>
<p>Synapse is _____</p>	<p>Neurotransmitters of EPSP: _____ Neurotransmitters of IPSP: _____</p>

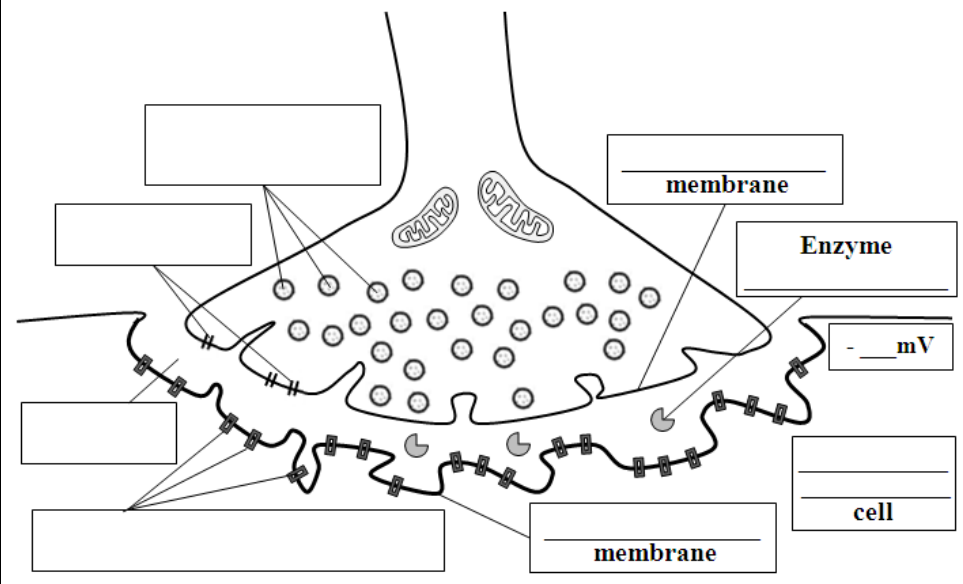
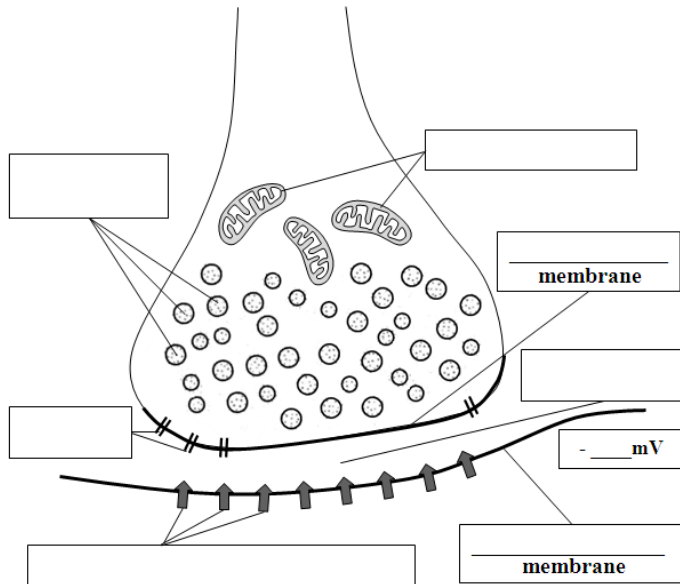
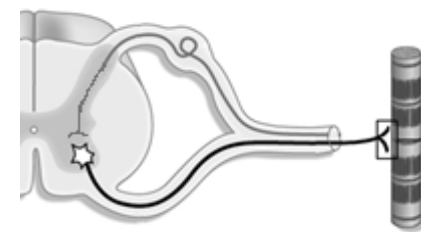
**WORK 10.2. COMPARISON OF THE CENTRAL (NEURO-NEURONAL) SYNAPSE STRUCTURE AND PERIPHERAL SYNAPSE (NEUROMUSCULAR JUNCTION)**

Fill in the names of the main synaptic structures for a neuro-neuronal central synapse and for neuromuscular junction. Indicate the main functional difference of neuromuscular junction as compared to the central synapse.

**Neuro-neuronal (central) synapse**



**Neuromuscular synapse**



**Conclusion:** the **main functional distinctive** feature of neuromuscular junction is that a single End-Plate Potential is \_\_\_\_\_ for action potential generation in this type of synapse while in the central neuro-neuronal synapse action potential generation requires at least \_\_\_\_\_ Excitatory Postsynaptic Potentials summated in a way of spatial or temporary summation. Thus, impulses from \_\_\_\_\_ neurons to the \_\_\_\_\_ muscles are always transmitted precisely according to the rate of stimulation from CNS.

### WORK 10.3. DEMONSTRATION OF LOCAL ANESTHETICS EFFECT DEVELOPMENT DEPENDING ON THE DURATION OF ACTION

Two broad categories of anesthesia exist: local and general.

Local or regional anesthesia block transmission of nerve impulses from a specific part of the body. It includes the injection and application techniques.

The local anesthetics effect develops due to blocking of an intracellular inactivation gates (h-gate) of voltage-gated sodium channels of afferent nerve fibers. Thus, local anesthetics interfere inflow of sodium ions through the membrane and its depolarization. As a result the action potential on a nerve fiber membrane is not generated. Impulses from pain receptors do not reach the central nervous system and pain sensation is not formed. Blockade of sodium channels is a process that requires certain time (usually some minutes). The time for the effect development depends on the dose of anesthetics and individual sensitivity.

Local anesthetics can block the transmission of a signal along any nerve fibers, but the sensitivity of the latter to anesthetic effects depends on its myelination, size, frequency of impulses on them, position of the fibers in the bundle.

*At first, sensory conductivity is blocked in type B and C fibers, then in A $\delta$  fibers. Thus, the pain disappears first, then other kinds of sensitivity are suppressed, and motor functions the last one.*

Myelinated fibers are blocked earlier than unmyelinated fibers of the same diameter. To stop the initiation of myelinated fibers, it is necessary for the blockade to extend to three consecutive nodes of Ranvier. The effect of anesthesia is more expressed in actively acting axons, which are more accessible to local anesthetics.

The computer program “NERVE” allows following the time dynamics of local anesthetics action.

**Accomplishment.** Open the program “Nerve”. Then choose **Nerve Physiology** → **Menu** → 7. **The effect of procaine.**

The screen shows records of action potentials produced in the experiment by direct electric stimulation of a peripheral nerve. Consequential pressing the knobs with time indication in seconds produces records of the action potential on the screen, they being obtained directly: after injection of procaine (0 s), in 1 min (60 s), 1.5 min (90 s), 2 min (120 s), 4 min (240 s) and 6 min (360 s).

#### **Direction for recording the Protocol:**

1. Observe changing of the summarized action potential amplitude of nerve fibers contained in the whole nerve, and of the depolarization velocity, fill in the protocol.
2. Make a conclusion on how many minutes it took in this case to reach the effect of local anesthesia.

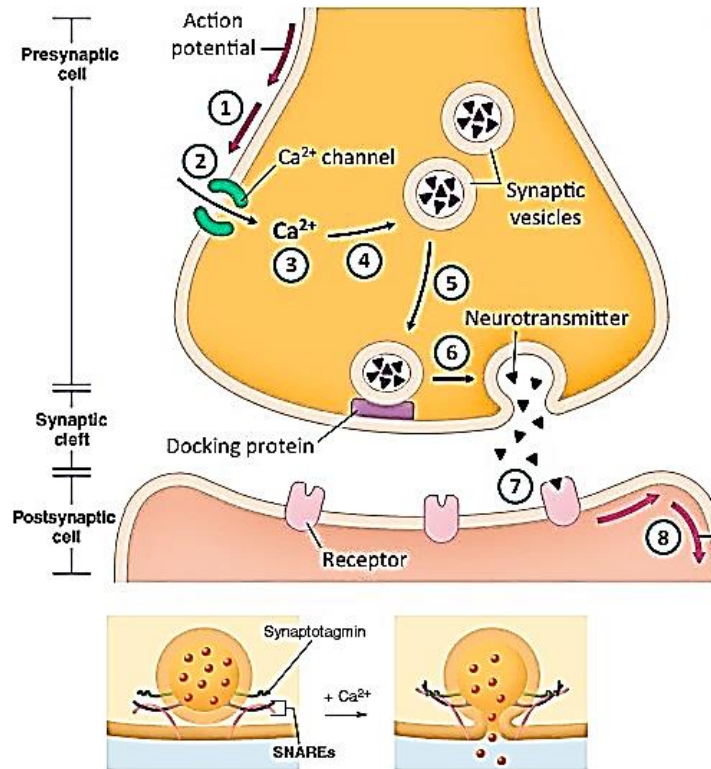
#### **PROTOCOL**

1. Amplitude of the total AP as anesthesia developed \_\_\_\_\_ ( $\uparrow$ ,  $\downarrow$ ),  
depolarization velocity \_\_\_\_\_ ( $\uparrow$ ,  $\downarrow$ ).
2. Conclusion: it took \_\_\_\_\_ min to reach the effect of local anesthesia.

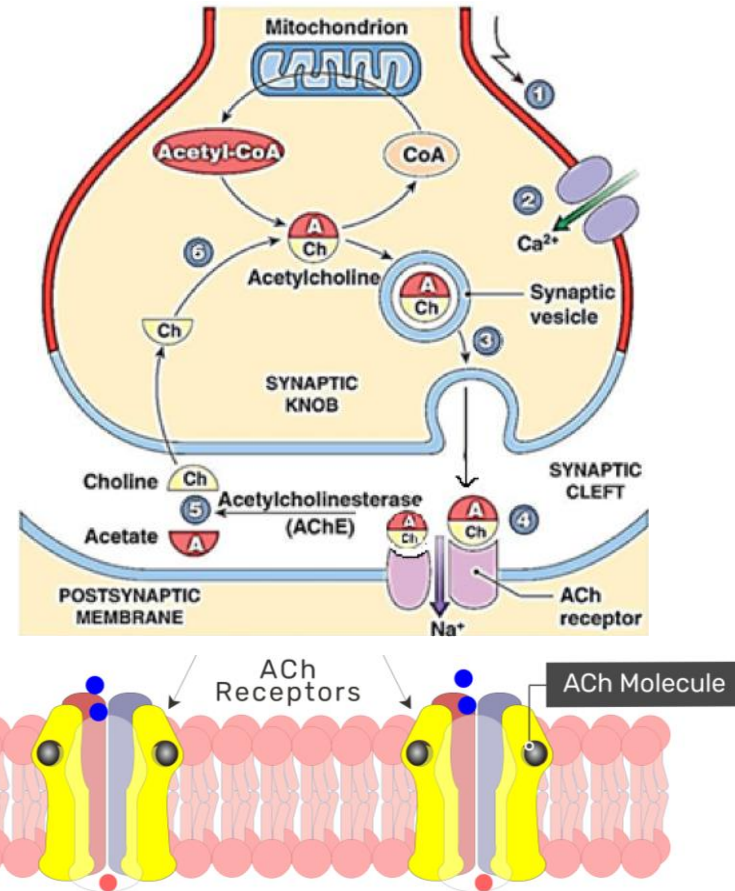
**WORK 10.4. THE MECHANISMS OF CONDUCTION IN CNS SYNAPSES**

**WORK 10.5. THE ROLE OF ACETYLCHOLINESTERASE**

Fill in the tables.



1. Action potential
2. Opening $Ca^{2+}$ voltage-gated channels
3.
4.
5.
6.
7.
8.



1. Action potential
2. Opening $Ca^{2+}$ voltage-gated channels
3.
4.
5.
6.

## ADDITIONAL MATERIALS

### The Erlanger–Gasser classification of nerve fibers

Fiber type	Myelin sheath	Diameter (µm)	Conduction velocity (m/s)	Sensitivity to anesthesia	Location	Function
A <sub>α</sub>	+	12–22	70–120	+	Efferent to muscles	Motor in skeletal muscles, afferents in muscle spindles (Ib) and tendon organs (Ib)
A <sub>β</sub>	+	8–12	40–70	++	Afferent from skin and joints	Tactile, proprioception
A <sub>γ</sub>	+	4–8	15–40	++	Efferent to muscle spindle	Muscle tone
A <sub>δ</sub>	+	1–4	5–15	++++	Afferent sensory nerves	Pain (“fast”), cold, temperature, touch
B	+ –	1–3	3–18	++++	Preganglionic sympathetic nerves	Autonomic control
C	–	0.5–1.5	0.5–3	++++	Postganglionic sympathetic nerves Afferent sensory nerves	Autonomic control Pain (“slow”), warm, temperature, touch

THE PRACTICAL WORKS ARE DEFENDED

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Lecturer’s signature

**Session 11. PHYSIOLOGY OF SKELETAL MUSCLES**

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           day                  month                  year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Physiological properties of skeletal muscles. The structure of muscle fibers.</li> <li>2. Sarcomere. Main proteins of myofilaments, their functions.</li> <li>3. Mechanisms of contraction and relaxations of a single muscle fiber and a whole muscle. Excitation-contraction coupling.</li> <li>4. Factor affecting the force skeletal muscles contraction. Tetanic muscle contractions. Muscle tone. Muscle fatigue.</li> <li>5. Motor units, their types, structural and functional properties.</li> <li>6. Dynamometry of a hand and back muscles.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 28–44, 81–83.</li> <li>3. <i>Severina, T. G.</i> Physiology of excitable tissues: textbook / T. G. Severina. – Minsk : BSMU, 2024. – P. 30–40.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>4. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 11.</li> <li>5. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 99–111.</li> <li>6. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 75–95.</li> </ol>
<p><b>WORK 11.1. TERMINOLOGY</b></p>	
<p>There are three types of muscle tissue: _____          _____</p>	<p>Sarcomere is _____          _____</p>
<p>Functions of muscular system: _____          _____</p>	<p>Physiological properties of muscle tissues: _____          _____</p>
<p>Excitation-contraction coupling is _____          _____</p>	<p>There are two main types of muscle fibers:          1. _____          2. _____</p>
<p>Tetanic contraction is _____</p>	<p>Motor unit is _____</p>
<p>There are two types of tetanus: 1. _____          2. _____</p>	<p>Stimulus that can cause skeletal muscle contraction: _____          _____</p>

**Self-study questions:**

1. The duration of muscle shortening in a single muscle contraction is 0.03 s, while the relaxation period is 0.04 s. Determine the type of this muscle contraction when the frequency of its stimulation is 10 Hz.
2. What is the difference between the processes taking place in a skeletal muscle for its tone maintenance and during its contraction?
3. What is the stimulus for skeletal muscle contraction? What factors may cause the contraction of a smooth muscle?
4. What are the sources of calcium ions for the contractions of skeletal and smooth muscles?
5. What is the type of motor units that are able to prolong contractions?
6. What are the basic types of calcium channels of smooth muscle cell membrane (1, 2, 3) and its endoplasmic reticulum (ER) (1, 2)?

**WORK 11.2. STUDYING THE MOTOR UNITS**

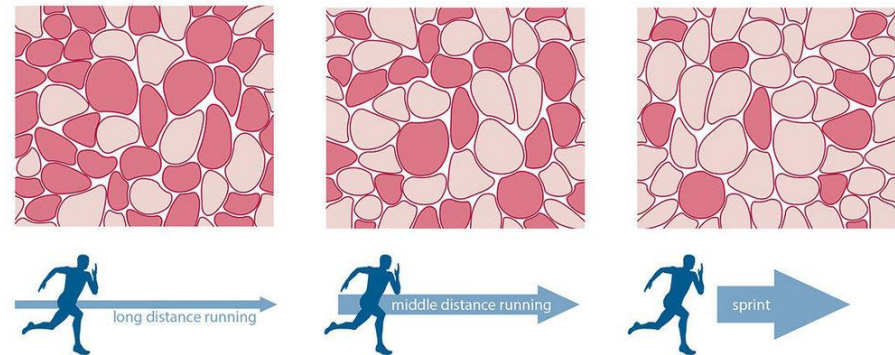
*Draw the motor unit and indicate the main structures.*

**WORK 11.3. SARCOMERE STRUCTURE**

*Draw the sarcomere and indicate main structures.*

**WORK 11.4. TYPES OF MUSCLE FIBERS**

Type	I (Slow)	Ia (FOG)	Ib (FG)
Description	Slow oxidative (SO) fibers	Fast oxidative glycolytic (FOG)	Fast-twitch glycolytic fibers
Myoglobin	high	medium	low
Mitochondria	many	moderate	few
Fatigues	slowly	moderate speed	fast
Color	red “dark meat”	red	white “white meat”
Diameter	narrow	medium	wide



*Fig. 11.1.* Distribution of white and red fibers in muscles of sprinters and stayers

**WORK 11.5. NEUROMUSCULAR SYNAPSE: MECHANISMS OF SIGNAL TRANSDUCTION**

List the mechanisms of excitation-contraction coupling (Fig. 11.2).

1. Action potential is coming down along axon to presynaptic terminal
2.
3.
4.
5.
6.
7.
8.
9. Contraction

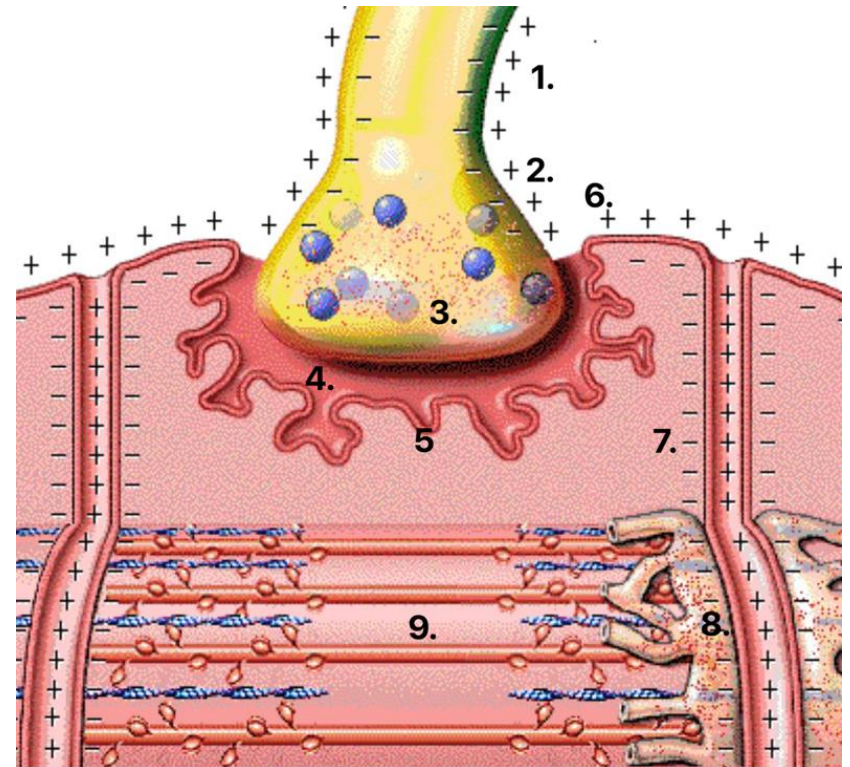
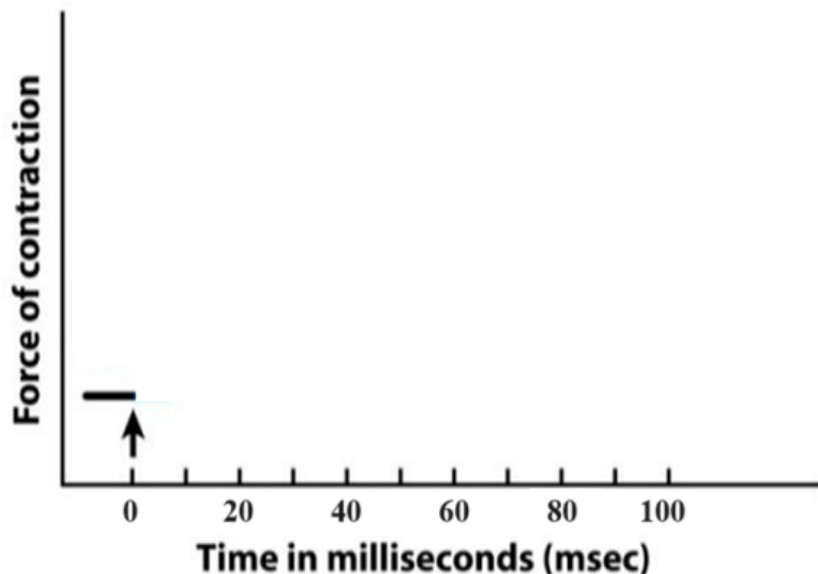


Fig. 11.2. Neuromuscular synapse

**WORK 11.6. MECHANISM OF CONTRACTION AND RELAXATION OF SINGLE AND WHOLE MUSCLE**

Open “IP” → Muscular → Contraction of whole muscle → read and go to p. 4 (muscle twitch) → draw a graph of muscle twitch and indicate contraction phases.



Fill in the table.

Phase	Description of the phase
1.	
2.	
3.	

**WORK 11.7. DYNAMOMETRY OF HANDS AND BACK MUSCLES**

Dynamometry is a method of measuring of the muscle contraction strength. Muscle strength is an important indicator of their contractility, as well as the physical development of the human body. It is estimated by the weight of the load that can be held by the muscle at its maximum excitation, without changing the length of the muscle. Muscle strength depends on its physiological cross-section, initial length, contraction rate and other factors. Muscle contraction is measured by dynamometers and expressed in absolute units (kg or N, as well as in  $\text{kg}/\text{cm}^2$  of muscle cross section (ranging from 2 to  $10 \text{ kg}/\text{cm}^2$ )) or in relative units (relative to body mass, expressed in %). Dynamometry (especially manual) is widely used in medicine and in the physiology of labor and sports activities.

**Materials and equipment:** manual dynamometer (Fig. 11.3), back muscles dynamometer (Fig. 11.4), medical scales.



Fig. 11.3. Manual dynamometer

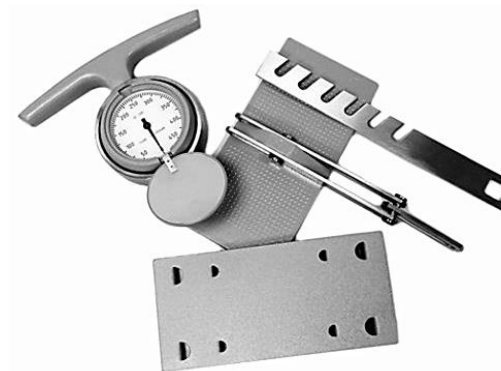


Fig. 11.4. Back muscle dynamometer

**WORK 11.8. DYNAMOMETRY OF HANDS AND BACK MUSCLES (continuation)**

**Progress of work**

The strength of the hands is determined using a manual dynamometer. The dynamometer is held parallel to the position of the floor (Fig. 11.5).

Perform maximum compression of the dynamometer with your hand. The measurement is carried out three times with each hand. Of the three dimensions (for each hand) choose the largest.

Calculate the Hand Strength Index (HSI) for right and left hands by the formula:

$$HSI = \frac{\text{Muscle strength in kg} \times 100 \%}{\text{Body mass in kg}}$$

$$HSI_r = \frac{\text{Muscle strength in kg} \times 100}{\text{Body mass in kg}} = \frac{\quad \times 100}{\quad} = \quad; \quad HSI_l = \frac{\text{Muscle strength in kg} \times 100}{\text{Body mass in kg}} = \frac{\quad \times 100}{\quad} = \quad.$$

**Satisfactory HSI** for men is **55 units**, for women — **50 units**.

The strength of the extensor muscles of the back is measured by a back muscle dynamometer (Fig. 11.6) three times, and the highest value is selected.

Calculate the Back Strength Index (BSI) by the formula:

$$BSI = \frac{\text{Muscle strength in kg}}{\text{Body mass in kg}} = \frac{\quad}{\quad} = \quad$$

**Satisfactory BSI** for men is **2 units**, for woman — **1.5 units**.

**Directions for recording the Protocol:**

1. Put down the obtained data into the Protocol.
2. Calculate HSI and BSI.
3. Evaluate muscle strength of the tested person and make a conclusion.

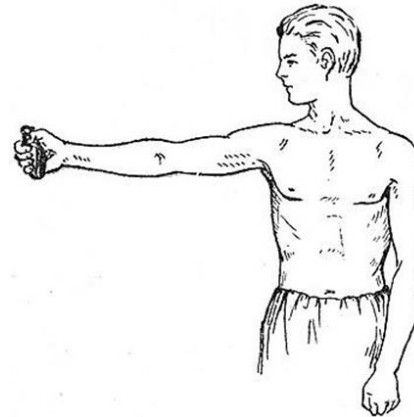


Fig. 11.5. Position for measuring hand muscle strength

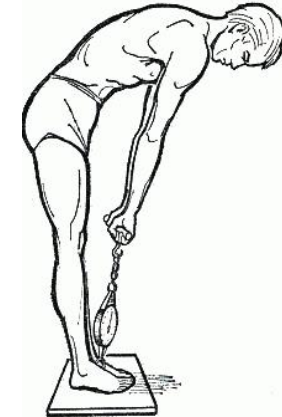


Fig. 11.6. Position for measuring Back muscle strength

**PROTOCOL**

Body mass \_\_\_\_\_ (kg), sex \_\_\_\_\_

Muscles	Muscle strength	Muscle strength index (in units)
Right hand		HSI <sub>r</sub> =
Left hand		HSI <sub>l</sub> =
Back extensors		BSI =

**Conclusion:** Right hand strength index is \_\_\_\_\_, left hand strength index is \_\_\_\_\_ (satisfactory, unsatisfactory). Back strength index is \_\_\_\_\_ (satisfactory, unsatisfactory).

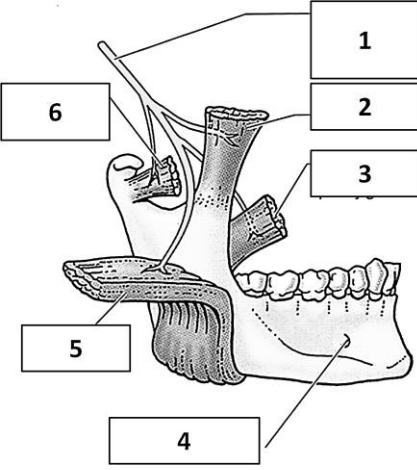
THE PRACTICAL WORKS ARE DEFENDED

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**Session 12. PHYSIOLOGY OF MAXILLOFACIAL REGION MUSCLES. SMOOTH MUSCLES.  
THE CONCEPT OF MYOEPIHELIAL AND GLANDULAR CELLS**

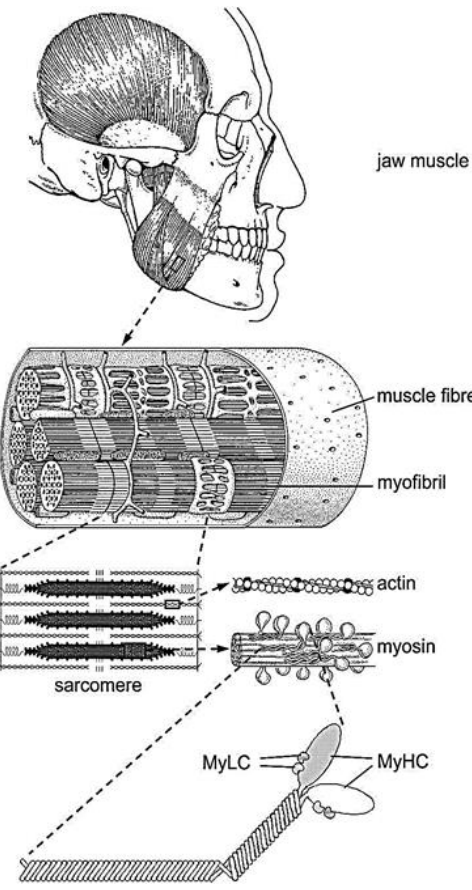
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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. The concept of the components of the masticatory system and their functional interaction. Movement of the mandible. Physiological occlusion.</li> <li>2. Muscles of the maxillofacial region and their functions. Functional features of different masticatory muscles.</li> <li>3. Contraction of masticatory muscles, regulation of this process.</li> <li>4. Periodontium, its endurance to the pressure developed by the masticatory muscles. Gnathodynamometry.</li> <li>5. Smooth muscles. Classification, physiological properties and features. Factors causing smooth muscle contraction.</li> <li>6. Membrane receptors and ion channels involved in triggering contraction. Role of calcium. Mechanism of contraction and relaxation of smooth muscle.</li> <li>7. The concept of myoepithelial cells (salivary and other exocrine glands) and their functions.</li> <li>8. Glandular epithelium, glands: functions, properties.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 28–46, 496–501.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 115–118.</li> <li>4. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 97–105.</li> </ol>
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<b>WORK 12.1. TERMINOLOGY</b>	<b>WORK 12.2. MUSCLES OF MASTICATION</b>															
Masticatory system — _____	<div style="display: flex; align-items: center; justify-content: center;">  </div> <p style="text-align: center;"><i>Fig. 12.1</i></p>															
Physiological occlusion — _____																
Centric occlusion — _____																
Centric relation — _____																
Intercuspal position (2–4 mm) — _____																
<i>Fill in the table.</i>																
<table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 50%; text-align: center;">Structure</th> <th style="width: 50%; text-align: center;">Function</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> <tr> <td>4.</td> <td></td> </tr> <tr> <td>5.</td> <td></td> </tr> <tr> <td>6.</td> <td></td> </tr> </tbody> </table>			Structure	Function	1.		2.		3.		4.		5.		6.	
Structure	Function															
1.																
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3.																
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6.																

### WORK 12.3. ELECTROMYOGRAPHY OF THE MASTICATORY MUSCLES

*Electromyography of mastication muscles* is a method of recording of the total electrical activity of the chewing muscles. When chewing food, the lower jaw with respect to the upper one makes movements in six directions due to the complex structure of the temporomandibular joint and the location of the chewing muscles.



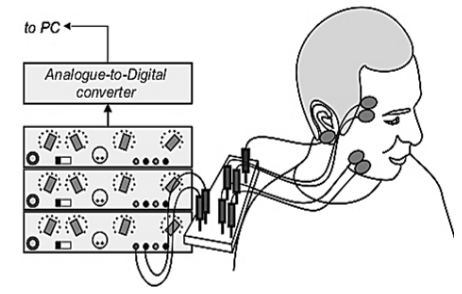
The diagram illustrates the structure of masticatory muscles. At the top, a lateral view of a human head shows the jaw muscle. Below it, a cross-section of a muscle fiber is shown, containing myofibrils. A detailed view of a sarcomere shows actin and myosin filaments, with MyLC (Myosin Light Chain) and MyHC (Myosin Heavy Chain) components.

Chewing muscles, especially the masseter and medial pterygoid muscles, refers as a muscle of power. The mandibular branch of the trigeminal nerve performs innervation of the muscles of mastication. Development of pathological processes in the lower jaw leads to impaired digestion in the oral cavity. Pain reflex reduces contractility and electrical activity of the masticatory muscles. Currently, the quantitative analysis of EMG is carried out using special instruments that allow one to measure the frequency of oscillations, to carry out spectral analysis and to estimate the average amplitude of the pulses. One of the most common methods of EMG analysis is its integration (determining the average amplitude value per unit time, i.e., dividing the total amplitude by the number of measurements, for example, per second). This indicator is proportional to the size of the developed muscular effort. In a state of functional rest, low amplitude electrical activity of the muscles is recorded, which reaches a maximum with arbitrary compression of teeth.

**Fig. 12.2. Muscles of mastication**

**Accomplishment.** The examined person seats in a chair; the skin is degreased at the points where the electrodes are applied. Above the masseter and digastric muscles of the face, glue on two electrodes, after having previously smeared them with a paste. The common electrode is fixed on the earlobe with a clip, and then the electrical activity of the muscles is recorded in various functional states of the oral cavity:

- a) rest: the facial muscles are relaxed, the jaws and teeth are open, the lower jaw is slightly lowered;
- b) opened mouth: jaws are wide opened;
- c) closed mouth: jaws are tightly closed;
- d) chewing a standard chewing gum.



**Fig 12.3**

#### PROTOCOL

- 1. Fill in the table.

*Table 12.1*

#### Obtained results of electromyography of mastication muscles

Recorded EMG	Rest	Open mouth	Closed mouth	Chewing
Masseter				
Digastric				

- 2. **Conclusion.** At rest, the electrical activity of the masseter and digastric muscles is \_\_\_\_ (↓ or ↑). With an open mouth electrical activity increases in the \_\_\_\_\_ muscle. When the mouth is closed, electrical activity increases in the \_\_\_\_\_ muscle. When chewing, the frequency and amplitude of the impulses in the studied muscles \_\_\_\_ (↓ or ↑).

## WORK 12.4. MANDIBULAR MOVEMENTS IN DIFFERENT PLANES. GOTHIC ARCH

In lateral movements, the condyle appears to rotate with a slight lateral shift in the direction of the movement. This movement is called the **Bennett movement** and may have both immediate and progressive components. By the use of recording equipment such as a pantograph or kinesiograph, it is possible to record mandibular movements in relation to a particular plane of reference (e.g., sagittal, horizontal, or frontal planes). If a point (the incisive point) located between the incisal edges of the two mandibular central incisors is tracked during maximal lateral, protrusive, retrusive, and wide opening movements, such movements are seen to take place within a border or envelope of movements. Functional and parafunctional movements occur within these borders. However, most functional movements such as those associated with mastication occur chiefly around centric. Border movements in the horizontal plane are shown in Fig. 12.4.

At Fig. 12.4, right mandibular movement with schematic representation of movement at the incisal point in the horizontal plane (*CR, LL, P, RL*) and at the condyle (*W, C, B, P*) made by a pantograph are presented. Teeth are not in occlusion. *CR*, Centric relation; *LL*, left lateral; *P*, protrusive; *RL*, right lateral; *CO*, centric occlusion; *IEC*, incisal edge contact. On the right side, the condyle moves from *C* (centric) to right working (*W*). On the balancing side, the left condyle moves from *C* along line *B* and makes an angle *BG*, called the *Bennett angle*. *C* to *P*, Straight protrusive movement.

The **maximum opening movement** is 50 to 60 mm, depending on the age and size of the individual. An arbitrary lower limit for normal of 40 mm may be in error, since as some individuals may have no difficulty incising a large apple and have no history of TMJ muscle dysfunction. The **maximum lateral movement** in the absence of TMJ muscle dysfunction, including pain, is about 10 to 12 mm. The **maximum protrusive movement** is approximately 8 to 11 mm, again depending on the size of the subject and skull morphology.

**Materials and equipment:** millimeter ruler.

**Accomplishment.** Suggest to the tested person to open his mouth as wide as possible. Measure the distance between the upper and lower incisors with an accuracy of 1 mm. Normally, it is 40–60 mm. Ask the subject to insert between the incisors 3 middle fingers of his non-working hand. Normally, with the maximal lowering of the mandible, the distal phalanges of the 3 middle fingers should fit between the incisors of the jaws. Ask the tested person to describe the “**gothic arch**” with the lower jaw, first without contact, and then in contact with the teeth of the upper jaw. In the case of the normal function of the chewing system, the mandible evenly (right to left or left to right) describes the “gothic arch” within the scope of its movements both without contact and in contact with the teeth of the upper jaw.

**Directions for recording the Protocol:**

1. Measure the distance between the incisors of the upper and lower jaw at the maximum opening of the mouth.
2. Make a visual assessment of whether the mandible of the tested person describes the “gothic arc” while moving.
3. Make a conclusion about the amount of movement of the lower jaw.

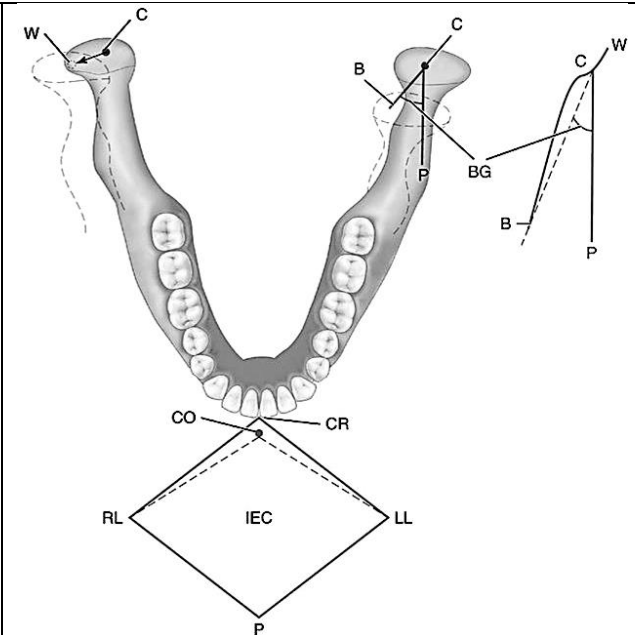


Fig. 12.4

### PROTOCOL

1. The distance between the incisors of the upper and lower jaw at the maximum opening of the mouth is \_\_\_\_\_ mm (normal range is from \_\_\_\_\_ to \_\_\_\_\_ mm).
2. During the movement of the mandible, the “gothic arch” is described \_\_\_\_\_ (completely or interrupted).
3. **Conclusion.** The amount of movements of the mandible of the tested person is \_\_\_\_\_ (full or limited).

## WORK 12.5. ASSESSMENT OF INTEROCCLUSAL SPACE. THE OCCLUSION

Basic jaw positions are usually described as **centric occlusion**, **intercuspal position**, **centric relation**, retruded contact position, and rest position of the mandible. **Centric occlusion or intercuspal position** is defined as maximum intercuspation of the teeth. Centric relation is a position of the mandible (or path of opening and closing without translation of the condyles) in which the condyles are in their uppermost position in the mandibular fossae and related anteriorly to the distal slope of the articular eminence. Because the mandible appears to rotate around a transverse axis through the condyle in centric relation movement, guidance of the jaw by the clinician in opening and closing movements that do not have translation is referred to as **hinge axis movement**. In this position, the condyles are considered to be in the terminal hinge position. Under physiological conditions of the masticatory system, centric relation is used to transfer the position of the mandible (in relation to the maxilla) to an articulator. Fig. 12.5 schematic representation of mandibular movement envelope in the sagittal plane. CR, Centric relation; CO, centric occlusion; F, maximum protrusion; R, rest position; E, maximum opening; B to CR, opening and closing on hinge axis with no change in radius. In the natural dentition, centric occlusion is, in the majority of people, anterior to centric relation contact on the average by approximately 1 mm. Centric occlusion (or **acquired** or **habitual centric** as it is sometimes called) is a tooth-determined position, whereas centric relation is a jaw-to-jaw relation determined by the condyles in the fossae. Closure into occlusion occurs usually anterior to centric relation; however, a coincidence of centric relation contact and the intercuspal position is evident in about 10 % of the population.

Rest position is a postural position of the mandible determined largely by neuromuscular activity and to a lesser degree by the viscoelastic properties of the muscles. Thus, because tonicity of muscles may be influenced by the central nervous system as a result of factors such as emotional stress and by local peripheral factors such as a sore tooth, the rest position of the mandible is not consistent.

The **interocclusal space** with the mandible in rest position and head in upright position is about 2–4 mm at the incisors but has considerable normal variance even from 1 up to 8–10 mm without evidence of dysfunction.

**Materials and equipment:** pencil (or handle), ruler, caliper.

**Accomplishment.** Mark two points on the skin, one at the tip of the nose, the other — on the chin along the midline of the face. Ask the tested person to sit up straight, close his lips and completely relax the muscles of the face. With complete relaxation of muscles, the lower jaw occupies the physiological **rest position**. Using a caliper measure the distance between the marked points. It is the **centric occlusion**. Then ask the subject to clench the teeth. Measure the distance between the same points on the skin. It is the **centric occlusion**. The difference between the **rest position** and a **centric occlusion** is **interocclusal space**.

**Directions for recording the protocol:**

1. Indicate values of the **rest position** and **centric occlusion**.
2. Calculate the magnitude of the **interocclusal space**.
3. Make a conclusion about the size of the **interocclusal space**.

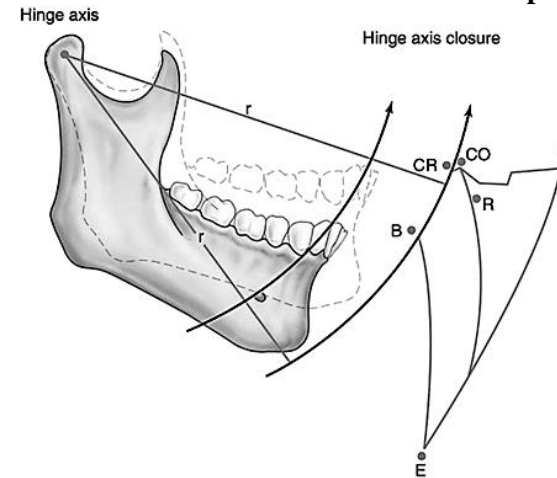
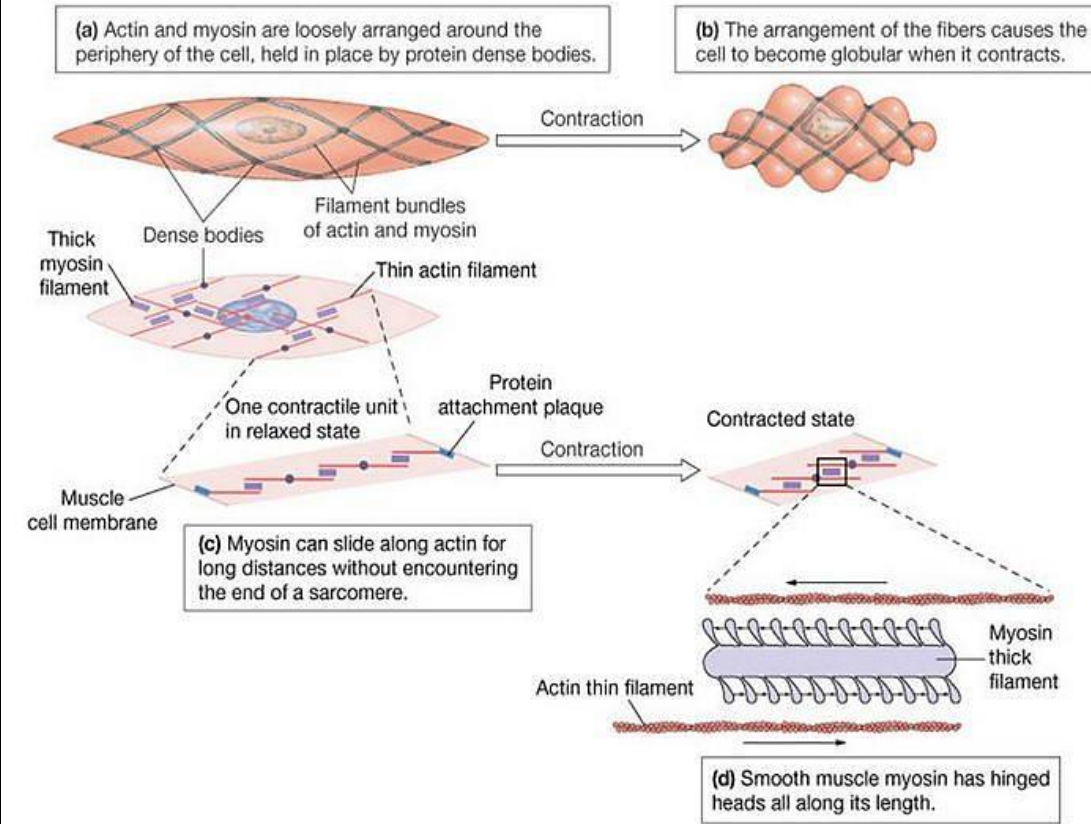


Fig. 12.5

### PROTOCOL

1. **Rest position** \_\_\_\_\_ mm; **centric occlusion** \_\_\_\_\_ mm.
2. **Interocclusal space**= \_\_\_\_\_ - \_\_\_\_\_ = \_\_\_\_\_ mm.
3. **Conclusion.** The size of the **interocclusal space** is \_\_\_\_\_  
(normal, increased, reduced).

**WORK 12.6. SMOOTH MUSCLES. CONTRACTION OF SMOOTH MUSCLE**



*Fig. 12.6. Mechanism and characteristics of contraction*

**WORK 12.7. TYPES OF SMOOTH MUSCLE UNITS**

*Draw a single-unit smooth muscle*

*Draw a multi-unit smooth muscle*

**WORK 12.8. REGULATION OF SMOOTH MUSCLE CONTRACTION**

*List the factors that may cause the smooth muscle contraction*

1.
2.
3.
4.

THE PRACTICAL WORKS ARE DEFENDED

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Lecturer's signature

## Session 13. GENERAL PHYSIOLOGY OF THE CENTRAL NERVOUS SYSTEM

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           day                    month                    year

<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Central nervous system. Its functions and role in maintaining the vital activity of an integral organism and its relationship with the environment.</li> <li>2. Neuron: structure, functions, properties, relationship with glial cells. The role of neuroglia.</li> <li>3. Features of the structure and CNS synapses functions in comparison with neuromuscular synapses. Neurotransmitters, their classification, the main types of receptors.</li> <li>4. Reflex principle of the nervous system functioning. Reflex. Types of reflexes.</li> <li>5. Structure of the reflex arch. Feedback system, its significance.</li> <li>6. Inhibition in the CNS, forms of its manifestation, types and role. Mechanisms of central inhibition.</li> <li>7. Primary (postsynaptic and its types, presynaptic) and secondary (pessimal, inhibition after excitation) inhibition. Inhibitory neurotransmitters. Mechanism of functioning of the inhibitory synapse. Inhibitory postsynaptic potential.</li> <li>8. Principles of CNS coordination activity: divergence, convergence, reciprocal inhibition, common final pathway, dominance, feedback. Excitatory and inhibitory neurotransmitters, receptor mechanisms of their action.</li> <li>9. Mechanisms of interaction of excitation and inhibition processes in a neuron. Integrative activity of a neuron.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 54–79.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 13.</li> <li>4. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 123–130, 137–155.</li> <li>5. <i>Hall, J. E. Guyton</i> and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 577–580, 595–606, 790–793.</li> </ol>
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### 13.1. TERMINOLOGY

Reflex — _____	Feedback — _____
Coordination in CNS — _____	EPSP — _____
Inhibition — _____	IPSP — _____

### Self-study questions

1. What are the functions of the nervous system?	4. What is the role of inhibition in the central nervous system?
2. What are the functions of neuroglia?	5. How is reciprocal inhibition organized?
3. What is the difference between sensory and motor neurons?	6. What is the role of the common final pathway?

### WORK 13.2. EXCITATORY AND INHIBITORY POTENTIALS

#### Characteristics of EPSP or IPSP

All postsynaptic potentials have certain characteristics in common. Importantly, the amplitude of graded potential is proportional to the size of the stimulus. Measurement of a graded potential uses the membrane resting potential as its baseline. The potential change is one of decreasing negativity (or of *depolarization* = excitatory postsynaptic potential EPSP), but it could also be one of increasing negativity (or of *hyperpolarization* = inhibitory postsynaptic potential IPSP).

Excitatory synapse	Inhibitory synapse
Postsynaptic potential (choose one)	
IPSP / EPSP	IPSP / EPSP
Neurotransmitters (write examples):	
Choose the changes of membrane potential:	
-5, -30, -90, -120 mV	-5, -30, -90, -120 mV
Is it depolarization or hyperpolarization?	

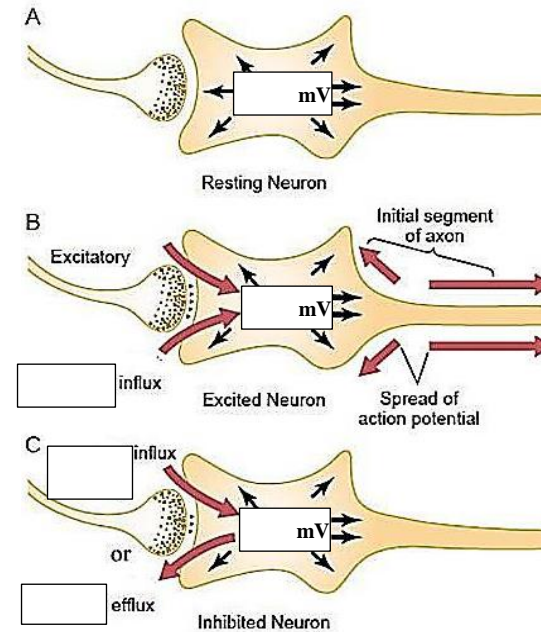
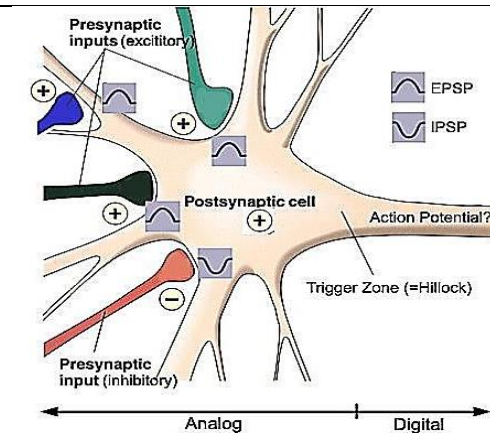


Fig. 13.1. EPSP and IPSP

### WORK 13.3. STUDYING THE MECHANISMS OF SUMMATION AT CENTRAL SYNAPSES



Will the action potential develop?

\_\_\_\_\_ (yes/no)

Draw the scheme of temporal summation of graded potentials

Draw the scheme of spatial summation of graded potentials

**WORK 13.4. STUDYING OF A KNEE (TENDON) REFLEX**

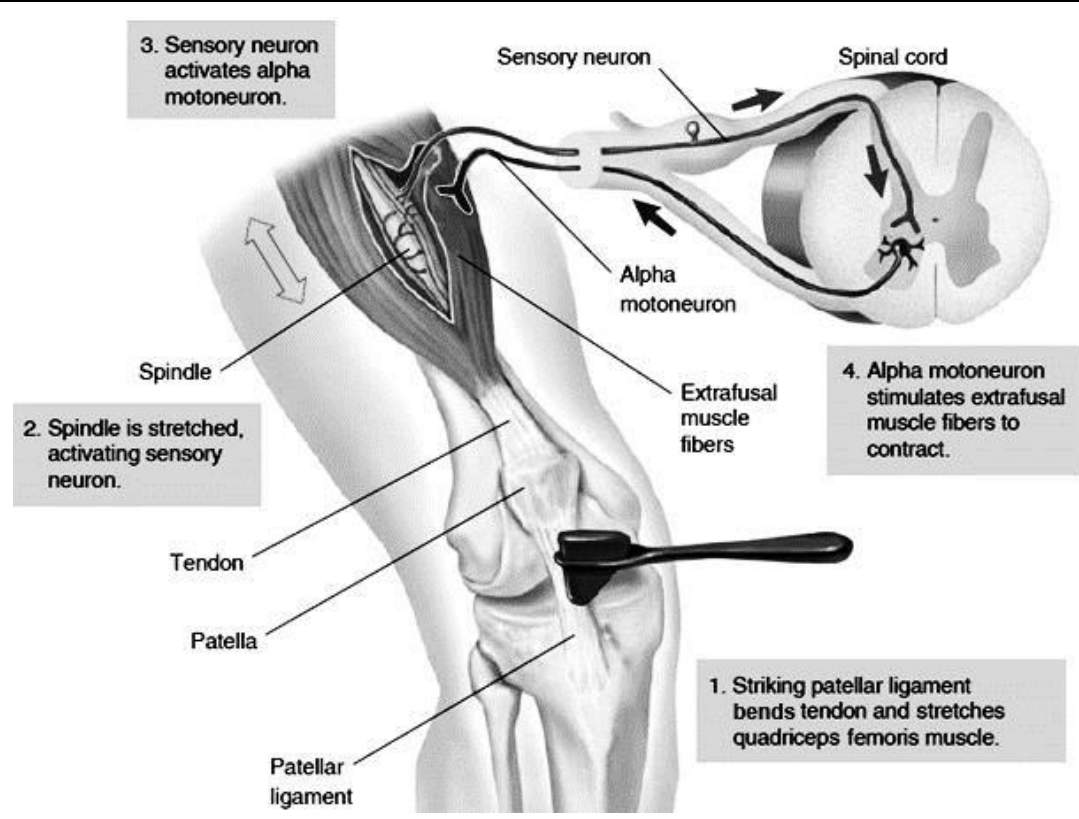


Fig. 13.2. Knee (tendon) reflex

List the parts of reflex arch:

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

**Myotatic reflexes** participate in regulation of muscle tone and support of body posture. Fast stretching of muscle by mechanical hit on the tendon results in contraction of whole muscle and movement reaction. Realization of such reflexes are available only in case of total relaxation of antagonist muscle.

In clinical practice tendon reflexes are studied to determine the functional state of different parts of the reflex arch and for the topic diagnosis of some CNS diseases.

During the accomplishment to prevent voluntary inhibition of reflex there are several techniques:

- 1) Ask the tested person to count from 200 in 7 (for example, 200, 193, 186 etc.)
- 2) Ask the tested person to clasp hands together.

**Materials and equipment.** A percussion hammer.

The examined person should sit down on the chair and put one his leg on the other. Hit the tendon of a quadriceps muscle of the hip below the patella with the percussion hammer. Observe the extension movement of the leg in the knee joint. Compare the reflex reaction on both extremities.

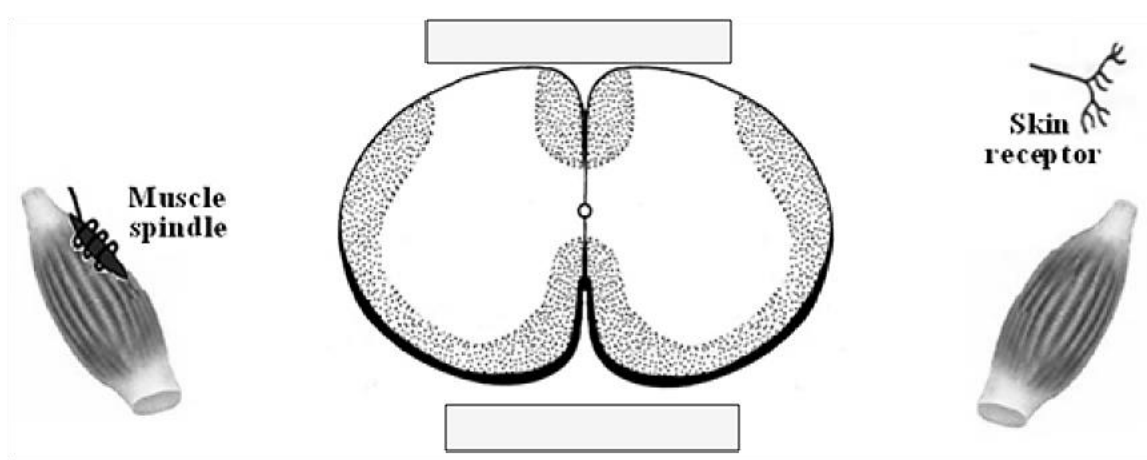
**PROTOCOL**

1. Knee reflex is \_\_\_\_\_ (marked, absent) on \_\_\_\_\_ (one or both extremities).
2. **Conclusion:** the reflex reaction is \_\_\_\_\_ (in norm, asymmetric, absent).

For the works 13.5–13.6. An example representing connections between different neurons.



**WORK 13.5. COMPARISON OF MONOSYNAPTIC AND POLYSYNAPTIC REFLEX ARCH**

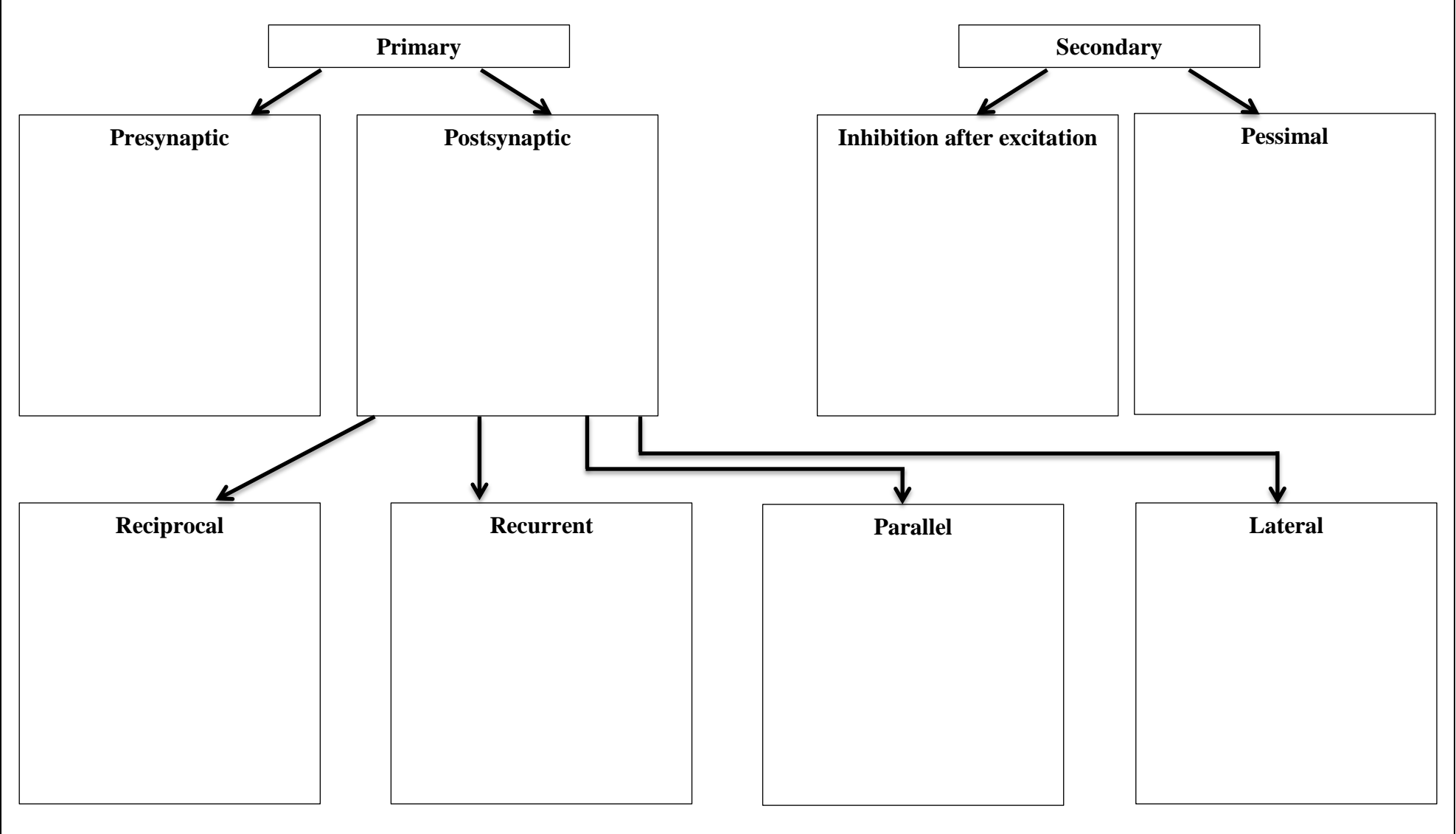


**Monosynaptic reflex arch**

1. Receptor part is presented by \_\_\_\_\_ and located in \_\_\_\_\_
  2. Afferent neuron is presented by \_\_\_\_\_ neuron, its body (soma) is located in \_\_\_\_\_
  3. Interneuron is \_\_\_\_\_
  4. Efferent neuron is presented by \_\_\_\_\_ or \_\_\_\_\_ neurons, its body (soma) is located in \_\_\_\_\_
  5. Effector organs are \_\_\_\_\_ and \_\_\_\_\_ muscle fibers.
- Velocity of signal transmission (action potential) for A $\alpha$  type is \_\_\_\_\_ m/sec, for A $\gamma$  type is \_\_\_\_\_ m/sec in efferent fibers. These fibers have \_\_\_\_\_ sheath.
- Neurotransmitter in neuro-muscular junction (synapse) is \_\_\_\_\_. It can bind with \_\_\_\_\_ receptor (muscle subtype).

**WORK 13.6. THE CENTRAL INHIBITION MECHANISMS**

*Fill in the table based on textbook, lectures, E-materials.*



## ADDITIONAL MATERIALS

### THEORIES OF THE MORPHOFUNCTIONAL ORGANIZATION OF THE NERVOUS SYSTEM

At the end of the 19th century, there were two hypotheses about the structure of the nervous system: the network (reticular) theory and the neural theory.

The reticular theory was developed and proposed in 1871 by the German histologist and anatomist Josef von Gerlach (1820–1896) and supported by the Italian scientist C. Golgi. According to the reticular theory, nerve tissue is a kind of syncytium in which the cells are to some extent deprived of individuality since their outgrowths are interconnected, continuously passing one into another, as a result of which a continuous diffuse network “rete nervosa diffusa” is formed.

In 1873, Camillo Golgi, studying the structure of brain gray matter, invented the method of impregnating neurons with silver nitrate. Using his preparations, Camillo Golgi demonstrated that the nervous system consists of a vast network formed by ramifications of free endings of dendrites and inseparably connected by a “diffuse nervous network”.

**Gradually scientists have defined the main points of the theory of reticularism:**

1) Neurons are not single independent cells but are connected in a “diffuse network”.

2) The fibers of this network are interconnected cytoplasmically (forming syncytium) and electrically. Nerve impulses can propagate in both directions from the point of contact of neurons.

3) The main functional centers of the nervous system are not neurons but “diffuse nerve networks”.

4) Neurons do not function independently but perform cooperative activities.

The neuronal theory — the cellular theory of the nervous system structure was formed at the end of the XIX century due to the works of several scientists of that time — S. Ramon-i-Cahal, W. Gis, A.-G. Forel, W. von Waldeyer, A. Van Heuchten. The Spanish histologist Santiago Ramon y Cajal was particularly zealous in presenting the neural theory.

Unlike Camillo Golgi, Ramon y Cajal believed that the nervous system consists of individual nerve cells, which form connections among themselves — contacts through which information is transmitted between neurons.

It should be noted that he was helped to develop the “neural theory” by the method of nerve tissue staining invented by his opponent K. Golgi. Among other things, Ramon y Cajal made another fundamental contribution to neuroscience: impulse transmission always occurs in one direction: from the dendrite to the neuron body and from it to the axon.

In 1897, Charles Scott Sherrington, a British scientist in the field of physiology and neurobiology, described contacts between nerve cells in the form of a small gap between neurons, which he called synapses (from the Greek word “clasp”). The final proof of the existence of the synapse was obtained in the 1950s using an electron microscope.

**The main points of the neural theory described by Ramon-y-Kahal:**

1) Nerve stimulus can propagate in only one direction: from sensory endings and cells through the brain to motor terminals (e.g., knee reflex).

2) Neurons are connected by chemical synapses capable of one-way mediator conduction.

3) Any neuroplasmic (syncytial) connections in neurons are absent, unlike in other cells.

4) Neurons develop and function independently.

5) Binuclear neurons are the result of amitosis.

Although their different ideas about the organization of the structure of nervous tissue K. Golgi and Ramon-i-Kahal both received the Nobel Prize in Physiology and Medicine (1906) “in recognition of their work on the structure of the nervous system”, it was one of the most controversial chapters in the history of the Nobel Committee, as both scientists interpreted the same histological phenomenon differently. Thus, even in his Nobel lecture, Camillo Golgi actively attacked the ideas supported by Ramon-i-Kahal and made arguments supporting the reticular theory.

In 1893, Russian scientist A. S. Dogel for the first time, described cytoplasmic anastomosis not only between cell bodies (which would be logical in the case of origin of binuclear neurons by amitosis) but also between nerve fibers (Fig. 13.2). Since then, neurophysiologists have increasingly observed such a pattern in the brain and peripheral nervous system of experimental animals. Moreover, it was noticed that these cytoplasmic anastomoses between neurons could not only form but also disappear, reflecting the plasticity of nerve processes.

The above data testify that the syncytial network of neurons (by the reticularism principle) is an integral part of the nervous system structure and is entirely compatible with the neuronal theory, supplementing it. This makes it possible to supplement the provisions of the neuronal theory with new data and try to formulate a unified neuron-reticular concept of the nervous system organization.

**The main provisions of the unified neuron-reticular theory are as follows:**

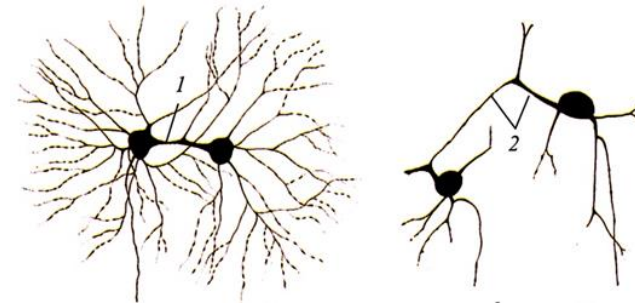
1) The action potential in the reflex arc spreads in one direction: from sensory endings and cells through insertion neurons to motor neurons and along their axons to target cells (myocytes and others).

2) Most neurons are connected by chemical synapses capable of one-way information transfer only. However, in some cases, there are also electrical synapses with the formation of functional syncytium between the cells and two-way signal conduction between them, including for reverberation of excitation along closed neural pathways in brain structures (involved in the processes of learning, memory, etc.).

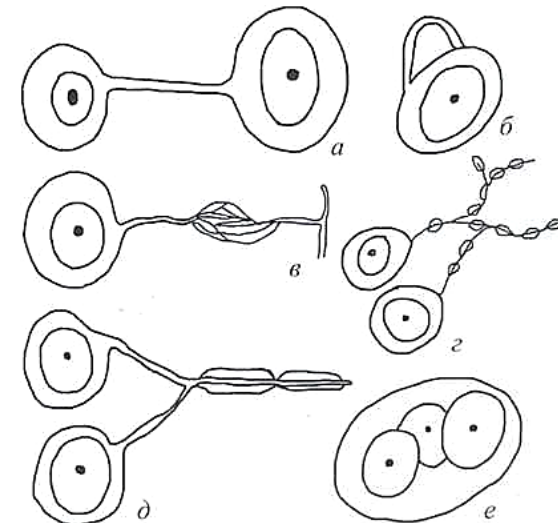
3) In vivo cine- and video-microscopic studies allowed us to establish the presence of neuroplasmic (true syncytial) connections between neurons (with the formation of a multinucleated cell and a common axon).

Unlike other true syncytial structures (e.g., skeletal myosimplasts), multinucleated neurons can not only form from separate single-nucleated neurons but also split back into single-nucleated cells.

4) Binuclear (or more) neurons can be both the result of amitosis and their formation by complete syncytial fusion of the processes (axons) and bodies of two (or more) neurons.



*Fig. 13.3. Cytoplasmic anastomoses between retinal reticulum neurons according to A. S. Dogel*



*Fig. 13.4. Variants of syncytial fusion of neurons described in the Laboratory of Functional Morphology and Physiology of the Institute of Physiology named after I. P. Pavlov of the Russian Academy of Sciences*

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
Lecturer's signature

**Session 14. COLLOQUIUM. CONCLUDING SESSION ON THE SECTION  
“GENERAL PHYSIOLOGY”**

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**THEORETICAL QUESTIONS:**

1. General properties of excitable tissues. Excitation and forms of its manifestation. Parameters of excitability. Change of excitability in the process of excitation. Refractoriness, its causes and physiological significance. Laws of excitable tissues response to the stimuli. Chronoximetry. Force-duration curve. Reaction of excitable tissues to the direct current.
2. Modern concepts of the structure and functions of membranes. Transport of substances through the cell membrane.
3. The concept of cell receptors and their functions.
4. Biopotentials as carriers of information in a living organism. Types of electrical signals in the organism, their comparative characteristics. Resting potential and graded potentials. Factors determining the value of membrane potential. The concept of galvanism.
5. Sensory receptors: definition, classification, role, basic properties. Receptor and generator potentials. The concept of information coding principles in sensory receptors.
6. Action potential as a carrier of information. Generation of action potential, phases and mechanisms of its development. Features of the structure and functioning of potential-dependent sodium channels.
7. Neuron: structure, functions, properties, relationship with glial cells. The role of neuroglia.
8. Physiological role of the structural elements of the nerve fiber. The role of afferent and efferent nerve fibers. Classification of nerve fibers. The role of nerve fibers of different types. Mechanism of excitation conduction along myelinated and unmyelinated nerve fibers, laws of excitation conduction. Axonal transport. Physiological basis of conduction anesthesia in dental practice.
9. Synapse. Classification of synapses, their physiological role. Structure of electrical and chemical synapse. Receptors of postsynaptic membrane. Mechanism of signal transmission in neuromuscular synapse. Endplate potential. The role of acetylcholinesterase. Mechanisms of neurotransmitter recovery. Mechanism of action potential generation at the postsynaptic cell.
10. Physiological properties of skeletal muscles. Single contraction (a twitch), its phases. Summation of contractions, tetanic contraction. Types and modes of skeletal muscle contraction. Motor units and their features in different muscles. Types of muscle fibers.
11. Structure of skeletal muscle fibers. Sarcomere. Mechanism of contraction and relaxation of a single muscle fiber and muscle as whole. Functional role of muscles of mastication.

**LITERATURE**

*Main*

1. Lecture & E-learning system.
2. *Moroz, V. M.* Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 7–82, 144–148, 635–646.
3. *Severina, T. G.* Physiology of excitable tissues: textbook / T. G. Severina. – Minsk : BSMU, 2024.

*Additional*

4. <http://etest.bsmu.by/> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 14.
5. *Ganong, W. F.* Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016.
6. *Hall, J. E.* Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016.

**Form of colloquium:**

1. *Theory*
2. *Practice (practical skill)*

12. Smooth muscles. Classification, physiological properties and features. Factors causing contraction of smooth muscle cells. Membrane receptors and ion channels involved in triggering contraction. Role of calcium, mechanisms of its concentration increase in sarcoplasm. Mechanism of contraction and relaxation of smooth muscle.
13. Central nervous system. Its functions and role in maintaining the vital activity of an integral organism and its relationship with the environment. Features of the structure and functions of CNS synapses in comparison with neuromuscular synapses. Neurotransmitters, their classification, the main types of receptors.
14. Reflex principle of the nervous system functioning. Reflex. Types of reflexes. Structure of the reflex arc. Feedback system, its significance.
15. Inhibition in the CNS, forms of its manifestation, types and role. Mechanisms of central inhibition. Primary (postsynaptic and its types, presynaptic) and secondary (pessimal, inhibition after excitation) inhibition. Inhibitory neurotransmitters. Mechanism of functioning of the inhibitory synapse. Inhibitory postsynaptic potential.
16. Principles of CNS coordination activity: reciprocal inhibition, final common pathway, dominance, reverse afferentation. Excitatory and inhibitory neurotransmitters, receptor mechanisms of their action. Mechanisms of interaction of excitation and inhibition processes in a neuron. Integrative activity of a neuron.

**Practical skills:**

1. Assessment of extracellular concentration of K<sup>+</sup> and Na<sup>+</sup> shifts on membrane potential values.
2. Possibility of pharmacological effect on process of signal transmission in synapses (example of neuromuscular junction).
3. Features of innervation of skeletal and smooth muscles and impact of neurotransmitters.
4. Dynamometry (manual and standing) and physiological evaluation of the results.
5. Study of the main tendon reflexes on the example of the knee reflex (morphological basis [reflex arch]). Physiological assessment of the obtained data.

**Colloquium is** \_\_\_\_\_

*(Lecturer's signature, date)*

Mark for theoretical part: \_\_\_\_\_

Mark for practical part: \_\_\_\_\_

## SECTION “MECHANISM OF PHYSIOLOGICAL FUNCTIONS REGULATION. NERVOUS REGULATION”

### Session 15. THE ROLE AND FUNCTIONS OF SPINAL CORD, BRAINSTEM, AND CEREBELLUM

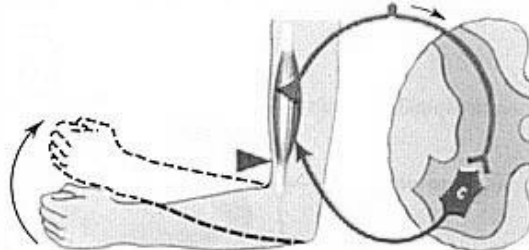
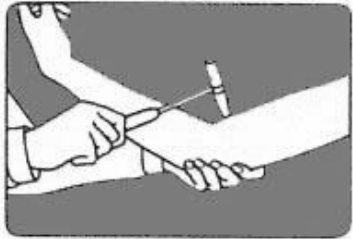
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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Spinal cord. Functions of the spinal cord.</li> <li>2. Spinal level of regulation of muscle tone, posture and movement. Basic spinal reflexes.</li> <li>3. Functions of the main ascending and descending conductive pathways of the spinal cord.</li> <li>4. Consequences of spinal cord injury. Spinal shock.</li> <li>5. The medulla oblongata and the pons. Sensory, somatic and autonomic functions.</li> <li>6. Vital centers, reflex activity. Functional interaction with other parts of the CNS. Defense reflexes.</li> <li>7. Functions of the cerebellum. Consequences of cerebellar damage.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 80–118.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 15.</li> <li>4. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 227–253, 263–273.</li> <li>5. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 6–10, 695–714, 721–745, 751–761.</li> </ol>
<b>WORK 15.1. TERMINOLOGY</b>	
The general division of the brain includes: 1) _____ 2) _____ 3) _____	Vital centers: 1) _____ 2) _____ 3) _____
Reflex is _____ _____	Spinal cord functions: 1) _____ 2) _____ 3) _____ 4) _____
Muscle spindles are _____	Spinal shock is _____
Alpha-motoneuron is _____	Cerebellum functions: 1) _____ 2) _____ 3) _____ 4) _____

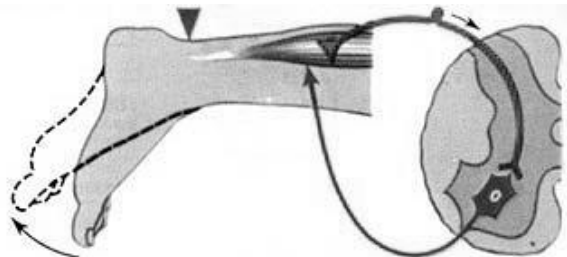
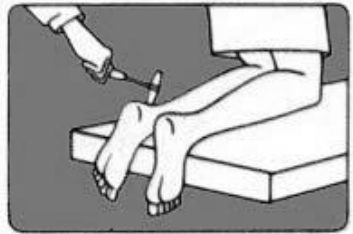
**WORK 15.2. STUDYING THE TENDON REFLEXES (MYOTATIC REFLEXES)**

**Accomplishment**

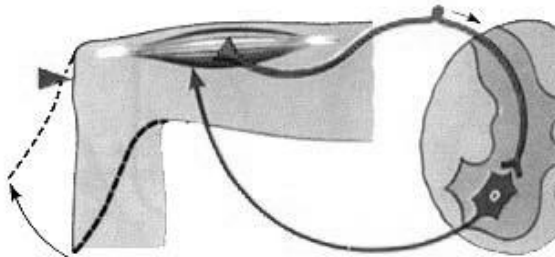
*Study the myotatic reflexes. Indicate the level of regulation in spinal cord. Compare the symmetry of reflexes. Record the results in protocol.*



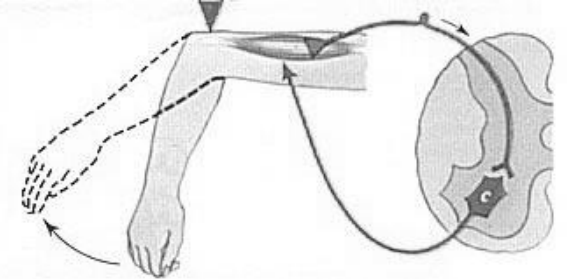
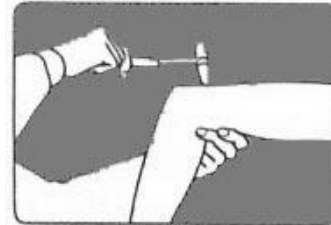
1. Tendon flexion reflex of the upper extremity (elbow reflex), C<sub>4</sub>-C<sub>5</sub>



2. \_\_\_\_\_



3. \_\_\_\_\_



4. \_\_\_\_\_

**PROTOCOL**

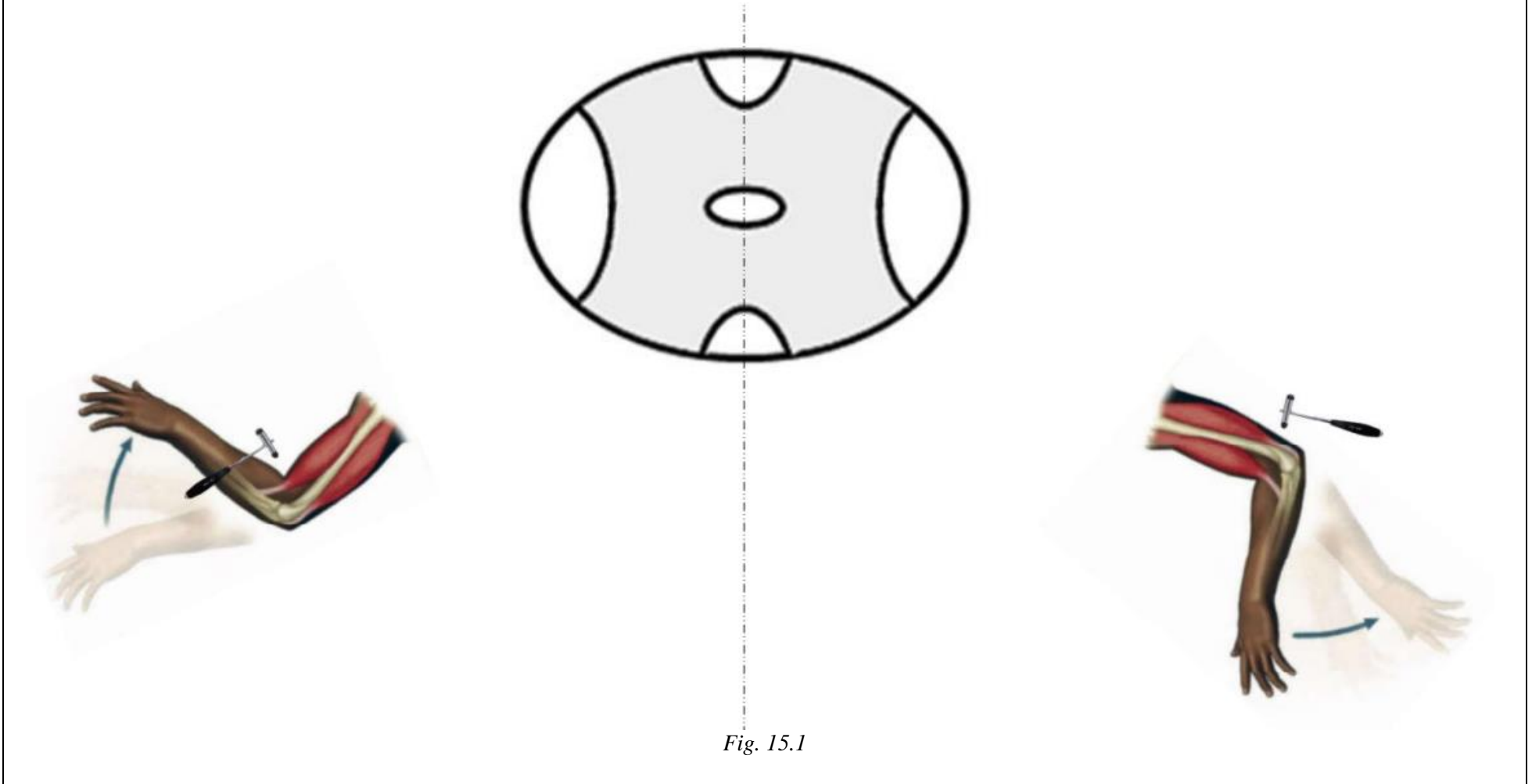
1. The myotatic reflexes are \_\_\_\_\_

2. The myotatic reflexes are \_\_\_\_\_ (marked or absent) and \_\_\_\_\_ (symmetric or asymmetric)

3. **Conclusion:** the reflex reaction is \_\_\_\_\_ (in norm or impaired)

**WORK 15.2. STUDYING THE STRETCH REFLEXES (OR MYOTATIC REFLEXES) (continuation)**

*Draw the schemes of reflex arches, maintaining the integration processes of contraction and relaxation. Indicate the level of spinal cord closure, parts of reflex arches, neurotransmitters.*



**WORK 15.3. STUDYING THE PUPILLARY REFLEXES**

Iris muscles, by contracting, are able to change the size of the pupil and thus regulate the flow of light to the retina. Normally, the diameter of the pupil is 2–8 mm, and the pupils are equal in size and have a regular round shape. When illuminated, the pupil contracts (miosis) and dilates when darkened (mydriasis). Impaired regulation of pupil size leads to anisocoria (unequal pupils), pupil deformation, and impaired pupil response to light.

**Materials and equipment:** neurological hammer.  
**Progress of work.**

**Direct pupil response to light**

The subject should sit facing the light source and cover one eye with his hand. Alternately cover the subject's other eye with the screen and open it. Observe the change in pupil size.

**Consensual reflex**

A) Under twilight (curtains), additionally illuminate one eye of the subject and observe the pupil diameter of the other (unilluminated) eye;

B) Close one eye of the subject and observe the pupil diameter of the open eye.

**Pupil response during accommodation and convergence**

Ask the subject to observe the tip of the pen moving gently toward and away from the bridge of the nose. Observe the reaction of the pupils.

**Directions for filling in the protocol:**

1. Evaluate the condition of the pupils and the degree of pupillary reflexes.
2. Conclude on the state of the pupillary reflexes.
3. Draw the reflex arcs of the direct and common pupillary reflexes (in Fig. 15.2).

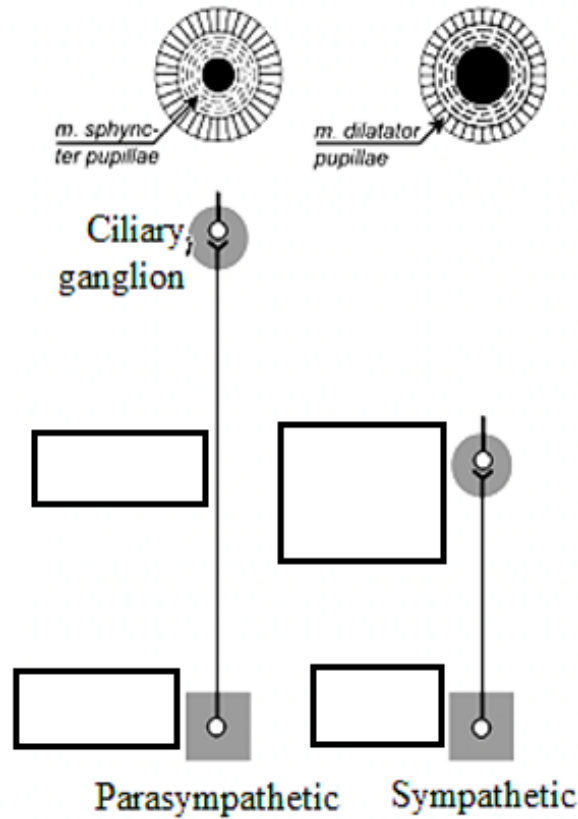


Fig. 15.2

**PROTOCOL**

The **coincident** reaction of the pupil to additional lighting or closing of the pupil of the other eye is caused by \_\_\_\_\_ at the chiasma and at the midbrain level and bilateral involvement of structures of the autonomic nervous system that regulate pupil diameter and lead to their contraction or dilation.

*The diameter of the pupils* varied from 2 mm to 5 mm. Their shape is \_\_\_\_\_ (rounded, irregular), size is \_\_\_\_\_ (same, different). In convergence the pupils of the eyes \_\_\_\_\_, and in divergence — \_\_\_\_\_.

**Conclusion.** Pupillary reflexes are \_\_\_\_\_ (expressed/disturbed). Pupil diameter is controlled by \_\_\_\_\_.

**WORK 15.4. STUDYING THE CEREBELLUM FUNCTIONS**

Efferent signals from the cerebellum regulate neuronal activity of vestibular (Deiters') and red nuclei, the thalamus nuclei, and through them the activity of peripheral ( $\alpha$ - and  $\gamma$ -motor neurons of the spinal cord and nuclei of cranial nerves) and central (cortical) motor neurons. Through these pathways efferent signals from the cerebellum *regulate strength of muscle contractions* ensuring the ability for prolonged *tonic muscle contraction*, relate the *volume of a voluntary movement* with the distance to the aim of this movement, and *quickly change flexing to extending and vice versa*. The cerebellum provides the synergy of contractions in complex movements.

Cerebellum functions disorder is manifested by: decrease of muscle contraction force (asthenia); loss of the ability to prolonged muscle contraction that makes standing, sitting difficult (astasia); involuntary change of muscle tone (dystonia); finger trembling at rest (tremor); movement impairment revealed as excessive or insufficient movement (dysmetria); coordination impairment (ataxia) that is manifested as "drunk" (swaying) gait and etc.; speech motor disorders (dysarthria); swinging rhythmic twitching of eyeballs (nystagmus); impairment of alternating opposite movements (adiadochokinesis), etc.

**Directions for recording the protocol:**

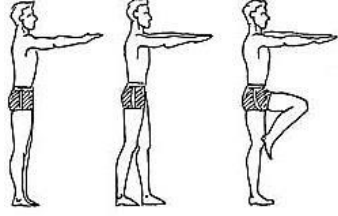

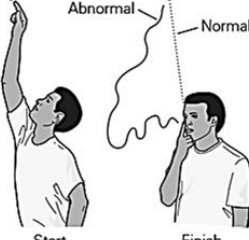
1. Point out, if the examined succeeded correctly (without faults) to perform the offered tests.
2. Make a conclusion about the quality of the cerebellum control of motor activity.

**PROTOCOL**

1. The tests for ataxia in the examined were \_\_\_\_\_ (+ or -), as in Romberg's pose one \_\_\_\_\_ (*kept or didn't*) balance, his gait was \_\_\_\_\_ (*normal or impaired*); tests for dysmetria and tremor were \_\_\_\_\_ (+ or -); dysarthria \_\_\_\_\_ (*was or wasn't*) revealed.
2. **Conclusion.** The cerebellum control of motor activity in the examined was \_\_\_\_\_ (*in norm or impaired*)

**Materials and equipment:** a glass, a book.

**Cerebellum control of skeletal muscles motor activity**

Type of experiment	Technique
Romberg's pose (coordination assessment of movements or <i>abasia</i> test)	The examined should stand with feet close and hands stretched forward, at first with open and then with closed eyes. In norm the person keeps the balance in Romberg's pose (i.e. the abasia test is negative) 
Gait (assessment of movements coordination or <i>ataxia</i> test)	Examined should walk about the room forward and backward with open and closed eyes. In norm the gate of a healthy person is usual, without swaying to the sides and broad placing his feet (i.e. the ataxia test is negative) 
<i>Dysmetria</i> test	The examined should take from the table and put back some object (a book, a glass). In norm the person puts the subject to the same place with an error $\pm 2$ cm (i.e. the dysmetria test is negative)
Speech ( <i>dysarthria</i> test)	The examined should repeat some words difficult for pronunciation ( <i>adiadochokinesis, atrioventricular, deoxyhemoglobin</i> etc.). Note, if there is slowed down, irregular or discontinuous speech
Finger-nose test (for <i>dysmetria</i> and <i>tremor</i> )	The examined should point with his index finger (at first of the left and then of the right hand) to the tip of his nose with open and closed eyes. In norm the person touches his nose tip with accuracy of $\pm 1$ cm without tremor of fingers (i.e. the test for dysmetria and tremor is negative) 

**WORK 15.5. STUDYING OF MOTOR FUNCTIONS OF SOME CRANIAL NERVES**

Motor functions are performed by nine cranial nerve pairs. Among them five pairs are motor (IV, V, VI, XI, XII) and four pairs are mixed (III, VII, IX, X). The motor nuclei of the trigeminal nerve (V pair) are located in the tegmentum at the level of the bridge and innervate the masticatory muscles. Neurons of the motor nuclei of the facial nerve (VII) located in the bridge innervate the facial mimic muscles. The motor nucleus of the glossopharyngeal (IX) and vagus (X) nerves is common and lies in the medulla oblongata, and the neurons of this nucleus innervate the muscles of the pharynx, soft palate, larynx and epiglottis, as well as the vocal folds. Finally, the muscles of the tongue are innervated by the nuclei of the hypoglossal nerve (XII).

Examination of motor functions of V, VII, IX, X and XII nerves		Guidelines for the filling in the protocol: 1. Indicate whether the examinee was able to complete all the tests and whether the results were within the norm. 2. Make a conclusion about the state of cranial nerves motor functions.
Cranial nerve	Technique	
V (trigeminal nerve)	The student is asked to open and close their mouth, then make several chewing movements. The researcher's hands are on the student's chewing muscles, determining the degree of their tension. Normally there is no lateral movement of the lower jaw, the muscles are tensed on both sides equally.	<p style="text-align: center;"><b>PROTOCOL</b></p> <p>1. The student _____ (<i>did / didn't</i>) _____ (<i>all / some / none</i>) tests. The results obtained _____ (<i>corresponded / didn't corresponded</i>) the norm.</p> <p>2. <b>Conclusion:</b> motor functions of studied V, VII, IX, X, and XII cranial neurons _____ (<i>norm / impaired</i>). <i>If impaired, indicate the pair of cranial nerves whose function was impaired.</i> Impaired motor function of _____ cranial nerves was detected.</p>
VII (facial nerve)	The student is asked: 1) raise the eyebrows upward (with the crease in the forehead expressed equally on both sides); 2) tightly close and then squeeze the eyes (normally they close equally); 3) smile and puff out your cheeks (the movements should be the same on both sides); 4) blow out the fire of a match or lighter (with the lips stretched forward).	
IX and X (lingual-pharyngeal and vagus nerves)	The student is asked: 1) stand at the window, open your mouth and say "a" loudly (with the tongue of the soft palate in the middle line); 2) say out loud several phrases of your choice (there should be no nasal tone of voice); 3) drink a few sips of water (swallowing should be free)	
XII (hyoid nerve)	The student is asked to stick out their tongue (normally, the tongue should be positioned in the midline).	

THE PRACTICAL WORKS ARE DEFENDED

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Lecturer's signature

**Session 16. SPECIAL PHYSIOLOGY OF NERVOUS SYSTEM  
(MESENCEPHALON, FOREBRAIN)**

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<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Electrophysiological methods of CNS research. Electroencephalography (EEG).</li> <li>2. Midbrain (mesencephalon). Thalamus, metathalamus, epithalamus.</li> <li>3. Functional features of thalamic nuclei. Participation of the thalamus in the formation of pain sensation and in the performance of higher integrative functions of the brain.</li> <li>4. Hypothalamus. Centers and functions of the hypothalamus. Neurosecretory cells. Sensory neurons (osmo-, thermo-sensitive, etc.) Integration of somatic, autonomic and endocrine functions.</li> <li>5. Basal nuclei. Morpho-functional organization. Integrating function of basal nuclei in the organization and realization of complex movements.</li> <li>6. Limbic system: structural and functional organization, role in formation of motivations and emotions, organization of memory.</li> <li>7. Cerebral cortex: morpho-functional organization, sensory and motor functions, participation in organization of movements.</li> <li>8. Current concepts of localization of functions in the cortex. Consequences of damage to different areas of the cerebral cortex.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 80–118.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <a href="http://etest.bsmu.by/">http://etest.bsmu.by/</a> – For English Medium Students – Dentistry – Normal Physiology (Dent) – Session № 16.</li> <li>4. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 227–253, 263–273.</li> <li>5. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 6–10, 695–714, 721–745, 751–761.</li> </ol>
<p><b>WORK 16.1. TERMINOLOGY</b></p>	
<p>Electroencephalography is _____</p>	<p>Basal nuclei are _____</p>
<p>Thalamus is _____</p>	<p>Motor cortex is _____</p>
<p>Hypothalamus is _____</p>	<p>Associative cortex is _____</p>
<p><b>Self-study questions</b></p>	
<ol style="list-style-type: none"> <li>1. In what way does the animal feeding behavior change in case of damage of lateral or ventro-medial nuclei of the hypothalamus?</li> <li>2. List the main centers of hypothalamus and the functions that it organizes and regulates.</li> <li>3. What is the difference between efferent projections of specific and unspecific nuclei of the thalamus?</li> </ol>	<ol style="list-style-type: none"> <li>4. The formation of what CNS mediator is impaired in parkinsonism?</li> <li>5. List basic functions of the limbic system.</li> <li>6. Which rhythm of EEG is considered synchronized? What is the desynchronization reaction?</li> <li>7. Which gyrus of the cerebral cortex is the somatosensory cortical area?</li> </ol>

**WORK 16.2. STUDYING THE TACTILE SENSITIVITY**

The examined is lying with his eyes closed. Touch the symmetric parts of the head, body and extremities of the examined with gauze ball. In norm he senses every touch and confirms his sensation with words.

**Directions for recording the protocol:**

1. Describe sensations of the examined.
2. Make a conclusion about the state of tactile sensitivity in the examined.

**PROTOCOL**

1. The examined \_\_\_\_\_ (*sensed or didn't sense*) touching with gauze ball and \_\_\_\_\_ (*correctly or with a mistake*) localized it.
2. **Conclusion:** the state of tactile sensitivity in the examined \_\_\_\_\_

**WORK 16.3. STUDYING THE MUSCLE-JOINT SENSATION (KINESTHESIA)**

**Accomplishment.** The examined is lying with his eyes closed. Perform mild flexing and extending movements of the hand fingers of the examined, starting with end finger-cushions. In norm the examined should correctly distinguish all performed actions, giving correct answers what finger is performing a passive movement at the given moment, if flexing or extending is performed.

**Directions for recording the protocol:**

1. Describe if the examined distinguishes performed actions correctly.
2. Make a conclusion about the state of the muscle-joint sensation in the examined.

**PROTOCOL**

1. The examined distinguishes the performed actions \_\_\_\_\_ (*correctly, incorrectly*)
2. **Conclusion:** \_\_\_\_\_

**WORK 16.4. STUDYING THE ROLE OF THE DIENCEPHALON AND TELEENCEPHALON IN THE FORMATION OF SENSORY MODALITIES**

The formation of sensory sensation occurs as a result of integration of sensory information at different levels of the CNS. In particular, the integration of information from the gustatory, olfactory, and somatosensory systems, which takes place at the level of the thalamus and the associative orbitofrontal cortex (where the conscious sense of taste and smell is formed), is necessary for a complete sense of taste.

**Materials:** hard pieces of fruit cut into cubes or chewing gum (lollipop), a nose clip or gauze ball.

**Progress of work.** The test person covers his or her nose with a clamp or gauze ball, and closes his or her eyes. Then he/she is asked to determine the taste of a piece of fruit or chewing gum. Remove the clamp from the nose and ask to re-determine the taste of the test stimulus.

**Directions for recording the protocol:**

1. Note the change in taste after inclusion in the olfactory analysis.
2. Explain the mechanism of the observed phenomenon.

**PROTOCOL**

1. The examined with the nose closed identified the taste \_\_\_\_\_ (*correctly, incorrectly*). After the nasal breathing was restored, taste \_\_\_\_\_ (*did, did not*) change and \_\_\_\_\_ (*was, was not*) correctly identified.
2. **Conclusion:** the important role of the formation of sensory modalities is played by \_\_\_\_\_ afferent information. The level of regulation is \_\_\_\_\_. The sensory information about taste comes to the thalamic nuclei from \_\_\_\_\_.

## WORK 16.5. ELECTROENCEPHALOGRAPHY

*Electroencephalography* is a method for recording the total bioelectric activity of the brain.

**Accomplishment.** To record an EEG the examined is seated in the armchair in a shielded, grounded chamber with light and sound isolation.

In points intended for electrodes application the head skin is swabbed with mixture of alcohol and ether for removing fat. Four pairs of electrodes are attached symmetrically to the occipital, parietal, temporal and frontal head regions on both sides.

During EEG recording the examined should sit quietly with maximum relaxation of muscles and eyes closed. At first a calibrating signal is recorded, and then the background electric activity of various parts of the brain cortex is registered. Then the examined is asked to open his eyes, electric activity of the brain being observed.

The examined is asked again to relax his muscles and close the eyes. Some minutes later, when a clearly marked alpha rhythm appears on the record, sudden sound is made and EEG changes are being observed. Alpha rhythm is replaced by beta rhythm on eyes opening, on sudden action of sound and other stimuli as well as during doing mental arithmetic, thinking about answers to questions, etc.

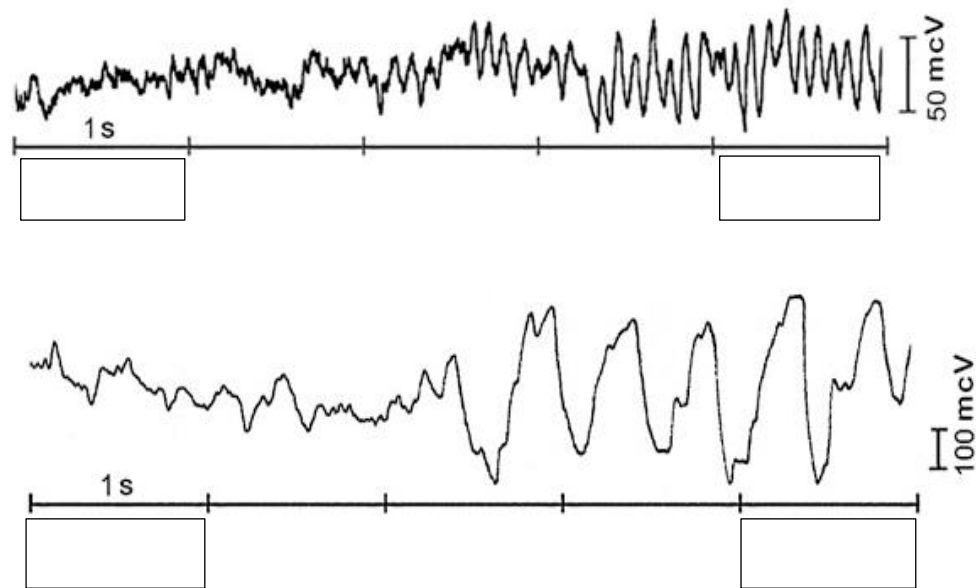


Fig. 16.1 Electroencephalography

### Directions for recording the Protocol:

1. Sign the types of EEG rhythms in the given EEG fragment (Fig. 16.1).
2. Fill in the Table 16.1.

Table 16.1

### Properties of rhythm of EEG

Rhythm	Frequency (Hz)	Amplitude (mcV)
Gamma ( $\gamma$ )		
Beta ( $\beta$ )		
Alpha ( $\alpha$ )		
Theta ( $\theta$ )		
Delta ( $\delta$ )		

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
Lecturer's signature

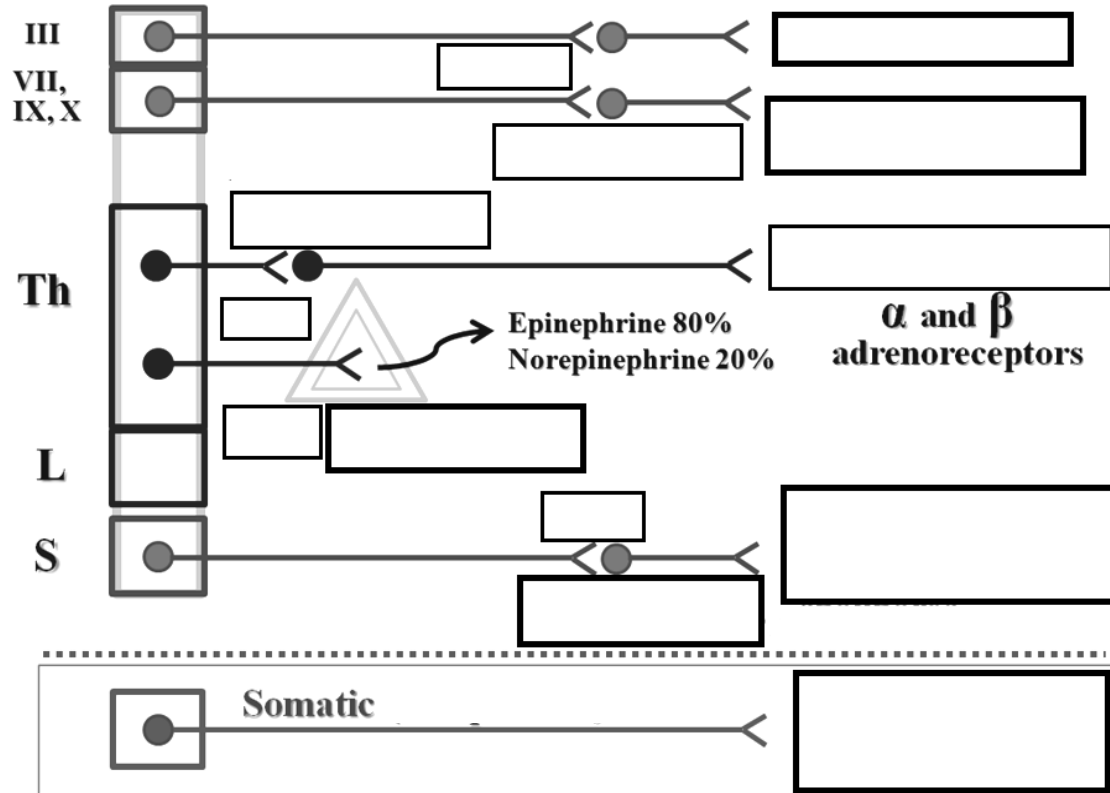
**Session 17. AUTONOMIC NERVOUS SYSTEM: STRUCTURE, FUNCTIONS.  
AUTONOMIC REFLEXES**

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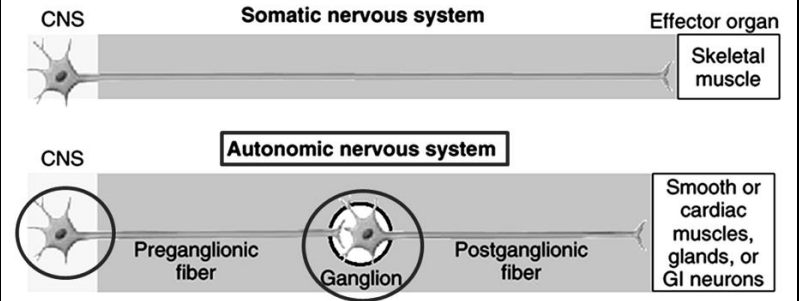
<p><b>BASIC QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. The role and functions of the autonomic nervous system (ANS).</li> <li>2. Comparative characteristics of somatic and autonomic nervous system (sensory receptors, afferent, efferent, and intercalary divisions, effector organs).</li> <li>3. Differences of neuroeffector connections of smooth muscle and neuromuscular junctions.</li> <li>4. Comparative characteristics of the structure and neurochemical mechanisms of the sympathetic and parasympathetic parts of ANS, as well as their influence on the effector organs. Relative antagonism and synergism of their effect.</li> <li>5. The concept of metasymphathetic part of ANS.</li> <li>6. Basic objective and subjective indices for evaluation the functional state of ANS parts.</li> <li>7. The concept of the principles of the autonomic functions' correction (for example, salivation) by affecting the neurotransmitter-receptor mechanisms in ANS ganglia and of the effector cells.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>Main</i></p> <ol style="list-style-type: none"> <li>1. Lecture &amp; E-learning system.</li> <li>2. <i>Moroz, V. M.</i> Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016. – P. 119–133.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <i>Ganong, W. F.</i> Review of medical physiology / W. F. Ganong. – 25th ed. – McGraw-Hill Companies, Inc., 2016. – P. 255–267.</li> <li>4. <i>Hall, J. E.</i> Guyton and Hall textbook of medical physiology / J. E. Hall. – 13th ed. – Elsevier, 2016. – P. 773–785.</li> </ol>
<p><b>WORK 17.1. TERMINOLOGY</b></p> <p>Autonomic nervous system (ANS) is _____</p> <p>Division of ANS: 1) _____ ; 2) _____</p> <p>Metasymphathetic part is _____</p> <p>The higher autonomic center is _____</p> <p>Neuroeffector connection is _____</p> <p>The general purpose of Sympathetic system is: _____</p> <p>The general purpose of Parasympathetic system is: _____</p>	<p><b>Self-check questions:</b></p> <ol style="list-style-type: none"> <li>1. What are the peculiarities of ANS innervation of the adrenal glands' medulla?</li> <li>2. What are the peculiarities of sweat glands innervation by ANS?</li> <li>3. Why can sympathetic nerves produce opposite effects on vascular tone?</li> <li>4. What are the metabolic effects of the sympathetic nervous system?</li> <li>5. List possible effects of atropine taking into account that this medicine is an antagonist of muscarinic cholinergic receptors.</li> <li>6. What action do sympathetic nerves produce on: the diameter of the pupil; heart function; bronchi; GIT (gastrointestinal tract) sphincters tone; skin vessels; vessels of skeletal muscles; secretion of gastric juice; adipose tissue; sweat glands; CNS activity?</li> </ol>

**WORK 17.2. STUDYING THE AUTONOMIC NERVOUS SYSTEM**

Fill in the name of neurotransmitters and their receptors in ANS.



Comparison of somatic and autonomic nervous system.



Fill in the spinal cord segments:

The divisions of autonomic nervous system:

**Parasympathetic:**

Midbrain (*nerve III*)

Hindbrain (*VII, IX, X*)

Spinal cord (\_\_\_\_\_ segments)

**Sympathetic:**

Spinal cord (\_\_\_\_\_ segments)

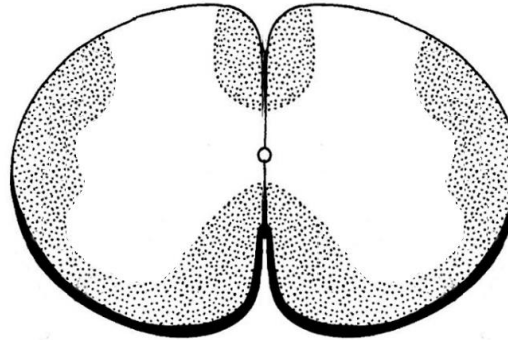
Spinal cord (\_\_\_\_\_ segments)

**WORK 17.3. DESCRIPTION OF SPINAL REFLEXES OF THE SYMPATHETIC AND SOMATIC NERVOUS SYSTEM**

**PROTOCOL**

**Somatic (polysynaptic) reflex arch**

**Autonomic (sympathetic) reflex arch**



Indicate the reflex arch structural parts of a somatic reflex:	Indicate the reflex arch structural parts of a sympathetic reflex:
1. Receptor part is presented by _____ receptors.	1. Receptor part is presented mainly by _____
2. Afferent part is presented by _____, which are located in _____	2. Afferent part is presented by _____, which are located in _____
3. Interneurons are located in _____	3. Interneurons are located in _____
4. Efferent part is presented by _____ or _____ motor neurons, that are located in _____	4. Efferent part consists of 2 neurons, which are located in: _____ and _____
5. Working organs. They are _____ and _____ muscle fibers of skeletal muscles.	5. Working organs. They are _____ muscle cells; cardiomyocytes; gland cells, myoepithelial cells.
6. Signal (AP) transmission rate is from _____ m/sec to _____ m/sec in efferent fibers, as they have _____ sheath and are referred to the type _____	6. Signal (AP) transmission rate is from _____ m/sec to _____ m/sec in efferent postganglionic fibers, as they do not have _____ sheath and are referred to the type _____
7. Neurotransmitter of the neuromuscular junction is _____, which binds to the _____ type of _____ receptors.	7. Main neurotransmitter in neuroeffector junction is _____, which binds to the _____ and _____ types of _____ receptors.

**WORK 17.4. CLINOSTATIC REFLEX**

Reflex study allows determining the functional state of parasympathetic and sympathetic centers regulating the heart function. When a man passes from standing to lying position, the heartbeat rate **decreases by 4–6 beats/min**. Pulse retardation over 6 beats/min evidences the increased tone of the parasympathetic part of ANS that regulates the heart functioning. The absence of reaction or its paradox character — pulse acceleration — evidences the prevalence tone of the sympathetic part of ANS regulating heart functioning.

**Materials and equipment:** a couch, a stopwatch.

**Accomplishment.** At first the pulse of the examined is counted (per 15 sec and multiplied by 4), when he is standing. Then, in 10–25 seconds after the examined lay down, the pulse is again calculated in the same way.

**Directions for recording the Protocol:**

1. Put down the pulse rate (PR) in standing position and then in lying position, count the pulse difference.
2. Make a conclusion of the tone of the sympathetic and parasympathetic departments of ANS regulating the heart functioning of the examined.

**PROTOCOL**

Pulse Rate, beats/min		
In standing position	In lying	Pulse difference [PR lying – PR standing]

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

**WORK 17.5. ORTHOSTATIC REFLEX**

Reflex study allows determining the functional state of sympathetic and parasympathetic centers regulating the heart functioning. When a man passes from lying to standing position, the heartbeat rate **increases normally 6–24 beats/min**. Pulse acceleration over 24 beats/min evidences the tone dominance of the sympathetic department of ANS, under 6 beats/min — that of the parasympathetic department of ANS.

**Materials and equipment:** a coach, a stopwatch.

**Accomplishment.** The pulse of the examined is counted when he is lying (the man is lying quietly for 4–6 min before the count starts). Then he is asked to stand up and his pulse is counted in 15–25 sec again.

**Directions for recording the Protocol:**

1. Put down the pulse rate (PR) in lying and standing position, calculate the pulse difference.
2. Make a conclusion of the tone of the sympathetic and parasympathetic departments of ANS regulating the heart functioning in the examined.

**PROTOCOL**

Pulse Rate, beats/min		
In lying position	In standing	Pulse difference [PR standing – PR lying]

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

**WORK 17.6. HERING’S RESPIRATORY-CARDIAC REFLEX**

Reflex study allows determining the functional state (tone) of the parasympathetic center regulating the heart functioning. When respiration is held on after a deep inhalation, the tone of *n. vagus* nuclei and heartbeat rate **decreases normally by 4–6 beats/min**. Pulse retardation by 8–10 beats/min and over evidences the parasympathetic ANS part tone increase, under 4 beats/min — tone decrease.

**Materials and equipment:** a stopwatch.

**Accomplishment.** The pulse is counted when the examined is sitting, then he is asked to make a deep inhalation and hold on the breath and the pulse is counted again.

**Directions for recording the Protocol:**

1. Put down the pulse rate (PR) before the breath is held on and when breath is held on during inspiration. Calculate the pulse difference.
2. Make a conclusion about the tone of the ANS parasympathetic department regulating the heart function in the examined.

**PROTOCOL**

Pulse Rate, beats/min		
Before breath holding	During breath holding after inspiration	Pulse difference [PR breath holding – PR before BH]

**Conclusion:** the tone of the ANS parasympathetic department \_\_\_\_\_

**WORK 17.7. ASSESSMENT OF NEUROTRANSMITTER MECHANISMS OF THE EFFECT OF SYMPATHETIC AND PARASYMPATHETIC PARTS OF ANS ON THE HEART FUNCTION**

**Accomplishment.**

The program “**Physiol 2**” is used; it allows to perform various virtual experiments on rats. The description of work with the program is given in work 1.3.

For making the experiment in menu:

1. Choose Help → Preparation.
2. Help → Drugs
3. Drugs → Injected, or Stimulate
4. New Rat

**Directions for recording the Protocol:**

1. Make an experiment and analyze the data from the table of the protocol.

Abbreviations: HR — Heart Rate, BP<sub>syst</sub> — Systolic Blood Pressure, BP<sub>diast</sub> — Diastolic Blood Pressure, BP<sub>mean</sub> — Mean Hemodynamic Blood Pressure.

2. Make a conclusion about the effect of the ANS sympathetic and parasympathetic parts on the force of heart contraction and heart rate as well as about neurotransmitter mechanisms of these effects.

**PROTOCOL**

<b>Effects of the heart</b>		<b>HR</b>	<b>BP<sub>syst</sub></b>	<b>BP<sub>diast</sub></b>	<b>BP<sub>mean</sub></b>
1.	Initial values (baseline)	161	98	53	66
2.	Stimulation Symp. Nerves to heart T <sub>1</sub>	210	130	95	106
3.	New Rat + Injection of noradrenaline, 5 µg/kg	212	130	95	106
4.	New Rat + Phentolamine (α-adrenoblocker), 100 mg/kg	161	98	53	66
5.	New Rat + Phentolamine (α-adrenoblocker), 100 mg/kg + Stimulation Symp. Nerves to heart T <sub>1</sub>	210	114	98	106
6.	New Rat + Propranolol (β-adrenoblocker), 100 mg/kg	161	98	53	66
7.	New Rat + Propranolol (β-adrenoblocker), 100 mg/kg + Stimulation Symp. Nerves to heart T <sub>1</sub>	170	99	65	75
8.	New Rat + Stimulation Vagus Nerve to heart	112	42	30	40
9.	New Rat + Acetylcholine, 5 µg/kg	115	31	19	28
10.	New Rat + Atropine (M-cholinoblocker), 10.0 mg/kg	161	98	53	66
11.	New Rat + Atropine (M-cholinoblocker), 10.0 mg/kg + Stimulation Vagus Nerve to heart	152	82	44	57

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

THE PRACTICAL WORKS ARE DEFENDED

\_\_\_\_\_  
 Lecturer’s signature

**Session 18. CONCLUDING SESSION ON THE SECTION “MECHANISM OF PHYSIOLOGICAL FUNCTIONS REGULATION. NERVOUS REGULATION”. CREDIT**

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<p><b>THEORETICAL QUESTIONS:</b></p> <ol style="list-style-type: none"> <li>1. Principles of CNS coordination activity: reciprocal inhibition, final common pathway, dominance, reverse afferentation. Excitatory and inhibitory neurotransmitters, receptor mechanisms of their action. Mechanisms of interaction of excitation and inhibition processes in a neuron. Integrative activity of a neuron.</li> <li>2. Comparative characteristics of somatic and autonomic nervous system (sensory receptors, afferent, inter and efferent sections, effector organs). Differences between neuroeffector junction of smooth muscles and neuromuscular synapses of skeletal muscles.</li> <li>3. Spinal cord. Functions of the spinal cord. Spinal level of regulation of muscle tone, posture and movement. Basic spinal reflexes. Functions of the main ascending and descending conductive pathways of the spinal cord. Consequences of spinal cord injury. Spinal shock.</li> <li>4. The medulla oblongata and the pons. Sensory, somatic and autonomic functions. Vital centers, reflex activity. Functional interaction with other parts of the CNS. Defense reflexes.</li> <li>5. Functions of the cerebellum. Consequences of cerebellar damage.</li> <li>6. Midbrain (mesencephalon). Thalamus, metathalamus, epithalamus. Functional features of thalamic nuclei. Participation of the thalamus in the formation of pain sensation and in the performance of higher integrative functions of the brain. Hypothalamus. Centers and functions of the hypothalamus. Neurosecretory cells. Sensory neurons (osmo-, thermo-sensitive, etc.) Integration of somatic, autonomic and endocrine functions.</li> <li>7. The role of the autonomic nervous system (ANS) in maintaining the vital activity of the organism. Functions of ANS. Comparative characteristic of structure and physiological properties of ANS and somatic nervous system (afferent, central, efferent sections).</li> <li>8. Comparative characterization of the structure and functions of sympathetic and parasympathetic parts of ANS. Synergism and relative antagonism of influences of sympathetic and parasympathetic parts of ANS. Influence of sympathetic ANS on effector organs, sensory functions. Mechanisms of their performance. The concept of metasymphathetic nervous system.</li> </ol>	<p style="text-align: center;"><b>LITERATURE</b></p> <p style="text-align: center;"><i>The main</i></p> <ol style="list-style-type: none"> <li>1. Lecture.</li> <li>2. <i>Moroz, V. M.</i> Physiology : Textbook / V. M. Moroz ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnytsia : Nova Knyha, 2016.</li> </ol> <p style="text-align: center;"><i>Additional</i></p> <ol style="list-style-type: none"> <li>3. <i>Ganong, W. F.</i> Review of Medical Physiology / W. F. Ganong – 23th ed. – McGraw-Hill Companies, Inc., 2010.</li> <li>4. <i>Guyton, A. C.</i> Textbook of Medical Physiology / A. C. Guyton, J. E. Hall. – 12th ed. – WB Saunders, 2005.</li> </ol> <p><b>Form of colloquium:</b></p> <ol style="list-style-type: none"> <li>1. <i>Theory</i></li> <li>2. <i>Practice (practical skill)</i></li> </ol>
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**PRACTICAL SKILLS:**

1. Study of the main tendon reflexes on the example of the knee reflex (morphological basis [reflex arc]). Physiological assessment of the obtained data.
2. Comparative characteristic of MONO- and POLY-synaptic reflex arches.
3. Evaluation of EEG rhythms in different functional states of the CNS.
4. Assessment of tone and reactivity of sympathetic and parasympathetic parts of ANS by heart rate on the example of clinostatic and orthostatic reflexes. Necessity of knowledge of these reflexes for a dentist.

Permission for the credit is approved by \_\_\_\_\_  
(Lecturer's name, signature)

**The credit is passed** \_\_\_\_\_  
(mark, date, Lecturer's signature)

**LITERATURE***Main*

1. Lecture & E-learning system.
2. *Moroz, V. M.* Physiology : textbook / V. M. Moroz [et al.] ; ed. by V. M. Moroz, O. A. Shandra. – 2nd ed. – Vinnitsia : Nova Knyha, 2016.
3. *Severina, T. G.* Physiology of excitable tissues: textbook / T. G. Severina. – Minsk : BSMU, 2024. – 59 p.

*Additional*

4. *Severina, T. G.* Physiology of blood. Lecture notes / T. G. Severina. – 2nd ed. – Minsk : BSMU, 2017. – 52 p.
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**ОБЩАЯ ФИЗИОЛОГИЯ ВОЗБУДИМЫХ ТКАНЕЙ, ЧАСТНАЯ ФИЗИОЛОГИЯ КРОВИ, ЭНДОКРИННОЙ,  
НЕРВНОЙ И КОСТНОЙ СИСТЕМ**

**GENERAL PHYSIOLOGY OF EXCITABLE TISSUES, SPECIAL PHYSIOLOGY OF BLOOD, ENDOCRINE,  
NERVOUS, AND BONE SYSTEMS**

Практикум для студентов, обучающихся по специальности «Стоматология»

На английском языке

Под редакцией Ю. В. Гайкович, В. А. Переверзева

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