

MINISTRY OF HEALTH OF UKRAINE
ZAPOROZHYE STATE MEDICAL UNIVERSITY
PATHOPHYSIOLOGY DEPARTMENT

PATHOPHYSIOLOGY PRACTICAL CLASSES MANUAL

MODULE 1 General Pathophysiology

Student of _____ group
medical faculty

Zaporozhye 2015

ЗАТВЕРДЖЕНО
на засіданні ЦМР Запорізького державного медичного університету
протокол № 5 від 24 травня 2007 р.

Edited by Yuri M. Kolesnik, full professor, M.D., Ph.D, chair of pathophysiology department.

The manual in pathophysiology was prepared by the members of pathophysiology department:

O.V. Melnikova, senior lecturer, Ph.D.

A.V. Abramov, full professor, M.D., Ph.D.

V.A. Zhulinsky, associate professor, Ph.D.

This manual was written according to the program of pathophysiology worked out by the Ministry of Health of Ukraine (Kiev, 2006). It includes plan of lectures and practical classes, instruction for every practical class, list of topics for independent studying, information about rating estimation of students' knowledge. Instruction for practical classes include: questions for preparing, description of experimental work, place for results registration and analysis. It can be used by the student of medical faculty while studying pathophysiology.

Навчально-методичний посібник до практичних занять з патофізіології за загальною редакцією завідувача кафедри професора Колесника Ю.М. підготували співробітники кафедри патофізіології Запорізького державного медичного університету: ст. викл., к.мед.н. О.В.Мельнікова, проф. А.В.Абрамов, доцент, к.мед.н. Жулінський В.О.

Посібник підготовлений у відповідності до програми з патофізіології для студентів вищих медичних навчальних закладів III—IV рівнів акредитації (2006 р). і містить плани лекцій та практичних занять, перелік тем для самостійного вивчення, питання до кожного практичного заняття, методику проведення експериментальних робіт, місце для обліку та оцінки результатів проведених експериментів, ситуаційні задачі для розв'язування.

Навчально-методичний посібник може бути використаний під час вивчення патофізіології студентами медичного факультету, які навчаються англійською мовою.

Forms of Training

The types of training activities due to the curriculum are as follows:

a) **lectures**, b) **practical classes**, B) **independent work of the students**.

The course of lectures contains the information about of the main questions on different pathophysiology sections.

Practical classes include the following:

1. The discussion on the topic of the practical class;
2. Students' study of the physiological functions and their changes in experiments on animals, isolated organs or cell, models of different pathological states. Other technologies of study can also be used (e.g. scientific movies, computer programs etc.);
3. Solving the situational tasks and tests (analysis and estimation of physiological indices, parameters of the different organs and systems functions), that is important for diagnosing and making prognosis in different clinical situations.

Each student has to fill the protocol of practical work with the experimental results and conclusions. Every protocol must be signed by the teacher.

Independent work of the student includes the following:

1. Preparing for practical classes and module control (theoretical knowledge, practical skills training);
2. Independent study of the topics which are not discussed at practical classes (working with the textbooks or other sources of information, writing of review, making reports);
3. Individual work given by the teacher (working out of clinical tasks and tests, digital programs for study, multimedia presentations and others).

THE TYPES OF THE CONTROL

Current control. Students are checked for their knowledge of theoretical information and results of independent work at practical classes by the teacher. The types of the current control are: discussion on the topic of the practical lesson, solving situational tasks and tests and practical skills control.

Sub-module control. Students are checked for their ability to use theoretical knowledge acquired at practical classes of the sub-module in order to solve clinical tasks and tests. The results of independent works of student are checked by the teacher too.

Students which have missed lessons and lectures are not allowed to pass sub-module control.

Module control is held after the study of all module topics at the last lesson of the 5th (autumn) semester.

To be allowed to pass sub-module and final module control student should to:

- attend all the lectures and practical classes according to the plan of study;
- do their independent work successfully;
- get rating grades not less that minimal.

Minimal grade (G_{\min}) is calculated with the formula $G_{\min}=3 \times N$, where N – the number of practical classes according to the plan of study (it means that each student must get at least 3 points at every practical class).

Module grade is the sum of the student's marks during current practical classes and the mark which was got at passing final module control.

The maximal quantity of grades at passing final module control can be **80**. The minimal quantity of grades during passing final module control is **50**.

The maximal quantity of grades during training of **one module is 200 grades** (120 points can be got during current practical classes and at passing of sub-modules and 80 points at final module control).

A student may increase his grade by re-passing final module control (not more than 3 times) according to permission of the head of department.

The final grade in pathophysiology is put when all the modules of the subject are successfully passed. The quantity of the obtained grades is converted into the mark according to international system – ECTS (European Credit Transfer System) and traditional Ukrainian mark.

Correlation between ECTS grades and traditional marks:

ECTS grades	Statistic index	Description	
A	The best 10% of the students	Excellent	5
B	The next 25% of the students	Very good	4
C	The next 30% of the students	Good	
D	The next 25% of the students	Satisfactory	3
E	The last 10% of the students	Sufficient	
Fx	Repeated module control	Fail/insufficient some work required to pass	
F	Obligatory second course of the subject	Fail/insufficient considerable further work required	2

Marks «F», «Fx» (insufficient) are put to those students, which have not successfully passed at least one module or sub-module of the subject:

«Fx» - student has attended all the classes and lectures of the subject,
got the minimal grades quantity,

but has not successfully passed final module control.

In this case the student is allowed to pass final module control once (not more than twice).

«F» - student has attended all the classes and lectures of the subject,

has not get the minimal grades quantity,

was not allowed to pass final module control.

This student is allowed to study pathophysiology for the second time.

MODULE 1. "GENERAL PATHOPHYSIOLOGY" STRUCTURE.

The list of topics	Lectures	Practical classes	Independent work of the students
Sub-module 1. General nosology – general etiology and pathogenesis of diseases. Pathogenic influence of the environmental factors. The role of the intrinsic factors in pathological states development.			
Topic 1. Pathophysiology as science. Its aims, methods of research activity. Definitions of the basic pathophysiology principles.	1,0	-	1
Topic 2. General etiology and pathogenesis of diseases.	1,0	2,0	-
Topic 3. Pathogenic influence of the physical factors.	-	2,0	1
Topic 4. Pathogenic influence of the chemical factors.	-	2,0	1
Topic 5. Pathogenic influence of the biological factors.	-	-	2
Topic 6. The role of heredity, constitution and age in pathology development.	-	2,0	1
Topic 7. Pathophysiology of the cell injury.	2,0	2,0	1
Topic 8. Immunological reactivity disturbances.	2,0	2,0	2
Topic 9. Pathophysiology of the allergic reaction	2,0	2,0	1
Topic 10. Checking of practical skills and test control on sub-module 1		2,0	3
Sub-module 2. Typical pathological processes			
Topic 11. Disturbances of peripheral bloodflow and microcirculation.	-	2,0	1
Topic 12. Inflammation.	2,0	2,0	1
Topic 13. Fever.	2,0	2,0	1
Topic 14. Tissue growth pathology. Tumors.	2,0	2,0	1
Topic 15. Starvation.	-	2,0	1
Topic 16. Hypoxia.	2,0	2,0	1
Topic 17. Checking of practical skills and test control on sub-module 2		2,0	3
Sub-module 3. Typical disturbances of metabolism			
Topic 18. The disturbances of energy metabolism.	-	-	1
Topic 19. The disturbances of carbohydrates metabolism.	2,0	2,0	1
Topic 20. The disturbances of lipids metabolism.	-	0,5	1
Topic 21. The disturbances of proteins metabolism.	-	0,5	1
Topic 22. The disturbances of vitamins metabolism.	-	-	1
Topic 23. The disturbances of water and salts metabolism.	-	1	1
Topic 24. Acid-base balance disturbances.	2,0	2,0	1
Topic 25. Checking of practical skills and test control on sub-module 3	-	2,0	2
FINAL MODULE CONTROL			
	-	2,0	4
General quantity of training hours - 93	20	40	35

Independent work – review of scientific literature or experimental research

LECTURE PLAN (MODULE 1)

	TOPIC	Hours
1.	Pathophysiology as a science. Its aims, methods of research activity. Definitions of the basic pathophysiology principles. General etiology and pathogenesis.	2
2.	Pathophysiology of the cell. General mechanisms of cell injury and death. Necrosis and apoptosis mechanisms.	2
3.	Reactivity and resistance of the organism, their role in pathology development. Humoral and cellular mechanisms of primary immune response. Primary and secondary immunodeficiency.	2
4.	Allergy: classification, etiology, stages and mechanisms of development, clinical manifestations. Autoimmune processes. Hypo-sensitization.	2
5.	Inflammation: etiology and pathogenesis. Local and general signs, sequence of events in inflammation. Classification and biological importance of inflammation.	2
6.	Thermoregulation pathology. Hyperthermia and hypothermia. Fever: etiology, pathogenesis, stages of development. Biological importance of fever.	2
7.	Pathology of tissue growth. Tumor etiology, pathogenesis, stages of development. Benign and malignant tumors.	2
8.	Hypoxia: etiology, pathogenesis, classification. Protective-adaptive and compensatory reactions in hypoxia development.	2
9.	Disturbances of carbohydrates mechanisms. Diabetes mellitus: definition, etiology, pathogenesis, classification, clinical manifestation and complications.	2
10.	Anemia: etiology, pathogenesis, principles of classifications, clinical and hematological signs of different anemias.	2

PRACTICAL CLASSES PLAN (MODULE 1)

	TOPIC	Hours
1.	General etiology and pathogenesis of diseases. Radiant energy influence on the organism.	2
2.	Ionizing radiation influence on the organism	2
3.	Chemical factors influence on the organism.	2
4.	The role of heredity and constitution in pathology development.	2
5.	Pathophysiology of cell.	2
6.	Pathology of reactivity. Disturbances of immunological reactivity.	2
7.	Allergy.	2
8.	Checking of practical skills and test control on sub-module 1 “General nosology”	2
9.	Disturbances of peripheral bloodflow and microcirculation.	2
10.	Inflammation.	2
11.	Thermoregulation pathology. Fever.	2
12.	Tissue growth pathology. Tumors.	2
13.	Hypoxia.	2
14.	Checking of practical skills and test control on sub-module 2 “Typical pathological processes”	2
15.	Disturbances of carbohydrates mechanisms. Diabetes mellitus.	2
16.	Disturbance of ABB and water-salt metabolism. Edemas pathogenesis.	2
17.	Disturbance of proteins and lipids metabolism. Starvation, obesity.	2
18.	Checking of practical skills and test control on sub-module 3 “Typical disturbances of metabolism” .	2
19.	FINAL MODULE CONTROL	2
20.	Students reports on independent work. Analysis of module 1 results.	2

UNIT №1

Radiant energy influence on organism. Pathogenic effect of infra-red rays, ultra-violet rays and visible spectrum section

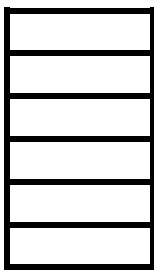
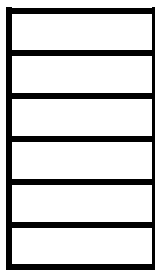
1. Subject, tasks and methods of pathophysiology. Types and planning of experiment.
2. Etiology: definition, classification of etiological factors.
3. Mechanisms of general and local effects of infra-red rays, ultra-violet rays and visible spectrum section on organism.
4. Definition of photosensitization and photosensitizers. Photosensitizers classification principles. Their mechanism of influence on human organism.
5. Pathogenesis of heatstroke and sunstroke.

Experimental work. Determine the biodose of Ultraviolet Radiation.

Smear the skin on the internal surface of forearm of one hand with the probe of cream. Put biodosimeters on the skin of both forearms – smeared and not smeared (control). Turn on mercury quartz lamp and ray the skin of forearms from a distance of 0.5 meters in the following consequence: at first one window of biodosimeter is rayed for 1 minute, then the nearest window is opened and it is rayed for 1 minute too. Irradiate all 6 windows of biodosimeter in this consequence and in these conditions.

Perform the same with another person but cover his skin with window glass instead of the cream. Compare the reaction of skin 30 minutes and 24 hours after irradiation. Identify the **biodose**, that is minimal time of irradiation in minutes on the distance of 0.5 meters from mercury quartz lamp that results weak but clearly expressed erythema.

In experimental results: draw 4 obtained biodoses (the degree of erythema), notice the difference in skin reaction during irradiation of different probationers in the same conditions and also during cream smearing and covering with glass.

	Person 1		Person 2	
				
biodose	= ____	= ____	= ____	= ____

In conclusion: explain the ultraviolet radiation effect and developing of erythema. Why does the biodose vary in different conditions for different people? What properties does the proposed probe of cream have (is it UV protecting or photosensitizing)?

CONCLUSION

Teacher's signature _____

UNIT №2

Ionizing radiation influence on organism. Radiation disease

1. Pathogenic effect of ionizing radiation: a) definition of tissues radiosensitivity; b) water radiolysis, concept of radiotoxins; c) mechanism of direct and indirect radiation damage of biological structures; d) manifestation of radiation affection at molecular, cellular, tissue, organ, system levels.

2. Acute radiation disease. Pathogenesis, classification: a) bone marrow form; b) intestinal form; c) toxemia form; d) cerebral form.

3. Stages of bone marrow form acute radiation disease. Its development, mechanisms of main clinical manifestations, blood count.

4. Chronic radiation disease. Mechanism of clinical manifestations development. Remote consequences effect of big and small doses of ionizing radiation on organism.

5. Pathophysiological basement of radioprotection. Definition of radioprotectors. Principles of their classification.

Experimental work. Determine the quantity of leukocytes in Intact and Irradiated Rats.

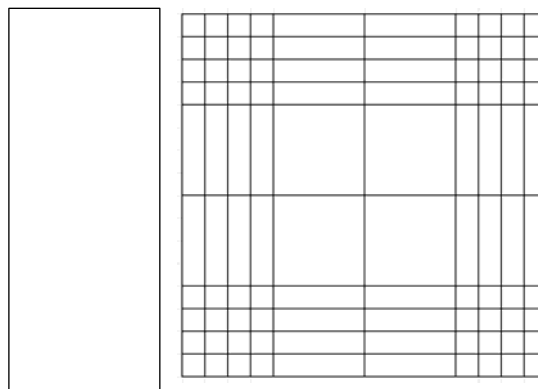
Two rats are needed for the experiment: irradiated with dose of 200 R and intact one. The rat is placed into the blood-taking machine; then you massage the tail to increase blood supply; then you cut off a little piece (1-2 mm); from the blood drop that appeared you take blood into the melangeur for leukocytes till the mark "0.5"; after this, you quickly take 3-5 % acetic acid with methylene blue into melangeur till the mark "11" before blood is coagulated. Thoroughly shuffle, shaking for 1 minute. In this case the blood is diluted 20 times. The same way you take and dilute the blood of irradiated rat in another melangeur. After this you perform the count of leukocytes in Goryaev's chamber. Before filling up you should wash the chamber and cover the glass with water and wipe dry. Then you lap the cover glass up to appearance of Neuton's rings. You release 1-2 drops of solution out of melangeur, then fill up the chamber so that all the surface where the net lies was filled with liquid without licking to grooves and without blebs. After filling-up the chamber you should leave it to stay for 1 minute for leukocytes concretion. Then you put the chamber on the microscope desk and start leukocyte count in 100 big squares with little magnification darkening the field of vision with diaphragm. The quantity of leukocytes in the unit of blood volume is determined by the formula:

$$X = (A \times 250 \times P / 100), \text{ where:}$$

A – the quantity of counted leukocytes in 100 big squares; **250** – coefficient for reduction of liquid volume over big square to 1 ml; **P** – blood dilution; **100** – the quantity of counted big squares; the result is multiplied by 10^6 for translation into **SI** system.

The same way you determine the quantity of leukocytes in irradiated rat.

In experimental results: write down the data of leukocyte count and other clinical features of radiation sickness (general condition, coverlet, quantity of breaths) in the form of table. Draw the melangeur and Goryaev's chamber net with leukocytes.



	Leukocytes quantity	Breath quantity	Skin color	General condition
Rat №1				
Rat №2				

In conclusions: to define which rat was irradiated, explain the mechanisms of leukocytes quantity change obtained; express supposition about possible stage of disease.

CONCLUSION

Task №1

The research of organism tissues radio sensitivity has such results. Muscular, nervous, bone tissues are the less radio sensitive; the most sensitive are lymphoid organs, marrow, and mucous cover of GIT organs.

1. What is tissues radio sensitivity?
2. What does radio sensitivity of tissues and organs depend on?

Task №2

Patient N., 45 years old, took radiation dose of 4 Grey. Diagnosis: acute radiation sickness.

1. What stages of acute radiation sickness can you name?
2. What does patient complain of at the beginning of the disease?
3. What laboratory features are typical to the first stage of the disease?

Task №3

Patient D., 30 years old, is a worker at nuclear power plant. He has already been 3 days at the hospital because of acute radiation sickness typical form development. He took radiation dose of 4 Grey. The patient says that he has recovered and doesn't have any complaints.

1. Has the patient recovered?
2. Why does the patient feel better if he hasn't recovered yet?
3. What blood changes are in this stage?

Task №4

Patient L., 65 years old went to Kiev hospital. He has already been living in the estrangement zone for 15 years (20 km zone around CNPP). He complains of: AP increase, palpitation, marked weakness, giddiness, tinnitus, weight loss, diarrhea, hemorrhages from nose and gums. Total blood count: erythrocytes $3,5 \times 10^{12}/l$, leucocytes $2,1 \times 10^9/l$; thrombocytes $80 \times 10^9/l$.

1. What was the cause of this pathology?
2. What blood analysis changes and clinic symptoms does the patient have?

Teacher's signature

UNIT №3
Chemical factors' influence on organism

1. General and local effect of chemical factors on organism.
2. Intoxication: definition, types, specific and nonspecific mechanisms. Toxins: definition, classification, direct and indirect mechanisms of influence on organism. Mechanisms of detoxication.
3. Free radicals (mechanism of their effect, systems of their neutralization).
4. Drugs and chemical addiction pathogenesis.
5. Pathogenesis of Alcoholism.

Experimental work. Investigate the Mechanisms of Organism Intoxication During Nitrite Poisoning.

Give narcosis to a rat by intraperitoneal introduction of 1,0% Thiopental Sodium solution (0,3 ml per 100 gr. of mass) or inhalation of ether fume (vapours). Then inject 3 ml of 20 % Nitrous Sodium solution under skin. Every 10 minutes you observe and write down: changes of respiration (respiration rate – counting the quantity of breaths per 1 minute and respiration rhythm), skin color changes, general condition of the animal. Development of intoxication leads to appearance of short breath and periodic respiration. After registration of periodic respiration you take 2-3 drops of blood from rat's tail, blend with 2-3 ml of distilled water in the test-tube. The color of blood from experimental animal has chocolate tint due to the presence of methemoglobin in it. With the help of spectroscope determine the presence of methemoglobin in the test-tube with diluted blood by absorption stripes in the red part of spectrum, oxyhemoglobin – in the green part of spectrum.

Arrange results as a table, draw the respiration change curve and absorption spectrum of normal and pathological hemoglobin.

	10 min	20 min	30 min	40 min
respiration rhythm				
quantity of breaths in 1 minute				
color of the skin				
general condition				

Absorption spectrum

normal hemoglobin

pathological hemoglobin.

--	--

In conclusions: explain the mechanisms of respiration changes and methemoglobin formation during Nitrite poisoning.

CONCLUSION

Teacher's signature

TESTS on chemical factors influence

1. Which stage of alcoholic and narcotic dependence is characterized by invincible pathological attraction to the substance consumption without abstinent syndrome?
 - a. physical dependence syndrome
 - b. tolerance development
 - c. sensitization development
 - d. phenomenon of cumulative effect
 - e. mental dependence syndrome
2. Which stage of alcoholic and narcotic dependence is characterized by manifestation of vegetative, somatic, psychic and neurological disorders after stopping the use of substance (alcohol, drug)?
 - a. physical dependence syndrome
 - b. mental dependence syndrome
 - c. tolerance development
 - d. sensitization development
 - e. phenomenon of cumulative effect
3. Does endogenous alcohol have physiological effect upon the organism?
 - a. yes, it is natural anti-oxidants
 - b. yes, the usage of large dosages of drugs promotes better adaptation of the organism to the environment
 - c. yes, it takes part in adaptation processes and motivation behavior
 - d. no, there is no endogenous alcohol in the organism
 - e. no, endogenous alcohol is synthesized only during pathological processes
4. What pathogenic mechanism causes the development of abstinent syndrome during drugs addiction?
 - a. decrease of endogenous opiates synthesis
 - b. development of nervous cells sensitization towards alcohol
 - c. formation of pathological tolerance of the cells in the organism
 - d. appearance of multiple organ functional insufficiency
 - e. decrease of activity of enzymes that metabolize narcotic
5. Free radicals constantly form in the healthy cell due to the effect of external and internal causes. What is the physiological role of free radicals in cell activity?
 - a. are exclusively pathogenic factors
 - b. take part in synthesis of biologically active substances
 - c. take part in processes of tissue respiration
 - d. take part in processes of cellular detoxication
 - e. take part in development of inflammation
6. A patient suffering alcoholism revealed disorders in heart, lungs, kidney and liver activity. What stage of alcoholism is characterized by multiple organ failure?
 - a. stage of psychic dependence
 - b. stage of physical dependence
 - c. terminal stage
 - d. stage of desadaptation
 - e. stage of initiation
7. What substance is responsible for the formation of multiple organ failure during alcoholism?
 - a. formaldehyde
 - b. ethanol
 - c. methanol
 - d. acetaldehyde
 - e. acetylsalicylic acid
8. What method of pathogenic therapy is the most appropriate to use during acute poisoning?
 - a. desintoxication therapy
 - b. prescription of diuretics
 - c. blood transfusion
 - d. prescription of anticonvulsants
 - e. prescription of purgatives
9. Free radicals activate lipid peroxidation in the organism and cause cellular damage. What factor promotes formation of free radicals?
 - a. hypovitaminosis D
 - b. infra-red radiation
 - c. lack of oxygen
 - d. ultraviolet radiation
 - e. excess of CO₂ (carbon dioxide)
10. Free radicals that form throughout our life have both positive and negative effects. That's why there are substances excreted in the cell that can directly or indirectly increase or decrease formation of free radicals. What is the role of antioxidants in the cells?
 - a. increase the formation of free oxygen radicals
 - b. increase the oxygen consumption in the cell
 - c. inhibit the formation of free oxygen radicals
 - d. decrease the oxygen consumption in the cell
 - e. increase ATP-formation
11. Each cell in the organism has anti-oxidation systems for protection from effect of free radicals. Which of the substances listed below can be related to the class of non-enzyme anti-oxidants?
 - a. catalase
 - b. superoxide dismutase
 - c. ceruloplasmin
 - d. beta-carotene
 - e. ferritin
12. Which of the substances listed below can be related to the class of enzyme antioxidants?
 - a. lycopene
 - b. carotin
 - c. tocopherol
 - d. ascorbic acid
 - e. glutathione peroxidase
13. What type of necrosis develops after alkali application?
 - a. coagulative
 - b. colliquative
 - c. liquifactive
 - d. caseous
 - e. fat necrosis
14. Coagulation of what substances is the leading mechanism of coagulative necrosis development?
 - a. membranes
 - b. lipids
 - c. proteins
 - d. DNA
 - e. RNA
15. The effect of toxins on the organism is determined with:
 - a. toxin chemical structure
 - b. dose of toxin
 - c. previous state of organism
 - d. duration of toxic effect
 - e. all the items are true
16. Which of the terms refers to the ability of toxins to cause defects in a developing fetus?
 - a. teratogenicity
 - b. mutagenicity
 - c. general toxicity
 - d. specific toxicity
 - e. cancerogenoicity
17. Choose the substance that causes exogenous intoxication:
 - a. scatol
 - b. ketonic bodies
 - c. phenol
 - d. indole
 - e. methanol

UNIT №4
Role of inherited factors in human pathology

1. The role of genetics in the modern medicine.
2. Definition of genotype and phenotype. Distinction between inherited forms of pathology and congenital diseases, phenocopies. Mutations and mutant genes.
3. Diseases, concerned with genetic pathology. Types of genetic defect inheritance.
4. Chromosomal diseases: classification, mechanisms of chromosome damage. Examples of autosome and sex chromosomes pathology.
5. Diagnostic methods, prophylaxis and treatment of inherited diseases.

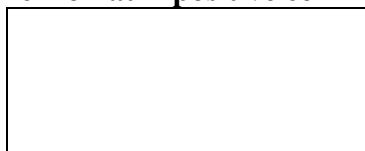
Experimental work. Determination of Barr's bodies in Epithelial Cells.

Wipe the mucous tunic of the cheek with wad of cotton wool and with the help of sterile spatula take a scrape by blunt method. Put the obtained material on object-plate in a flat layer and add 1 drop of gentian violet. Cover the preparation with a cover glass and press on it with a finger through gauze pad or filter paper for 2-3 seconds. The preparation is examined in immersion system of microscope. All three layers of mucous tunic are present in the scrape. Nuclei that fit for count are in the middle layer, the nuclei of superficial layer are usually pyknotized, and nuclei of profound layer contain many mitoses. Count 100 cells, noting the amount of those that contain genital chromatin. For count choose not damaged cells with round or oval nucleus, with even nuclear membrane. The bodies of genital chromatin are usually round- or oval-shaped, with even contours, and joint to the nuclear membrane.

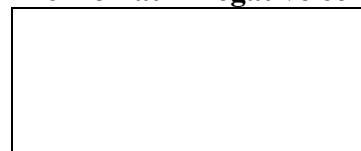
In norm the quantity of chromatin-positive nuclei varies from 20 % to 70 % in women, and from 0 to 10 % in men.

In experimental results: sketch chromatin-positive and chromatin-negative cells, describe the variations of genital chromosomes pathology and corresponding percentage of genital chromatin, note the data obtained from experiment.

chromatin-positive cell



chromatin-negative cell



Percentage of genital chromatin in the norm and genital chromosomes pathology

genotype	phenotype	Barr bodies quantity
XX		
XY		
XO		
XXY		
XXX		

In conclusions: explain possible changes in genital chromatin amount and the mechanism of appearance of Barr's bodies in the cell.

CONCLUSION

HEREDITARY PATHOLOGY TASKS

Task №1

Mary N., 25 years old, applied to genetic consultation in order to clear up her future child pathology development probability. She has a 4 old daughter. Daughter has 6 fingers at the right arm and 7 fingers at the right leg. Father and grandmother had the same pathology.

1. What hereditary pathology does Mary N. daughter have?
 2. Define the probability of this pathology development in second child.
-
-
-
-

Task №2

Healthy parents have a child with inherited disease – phenylketonuria. Parents applied to genetic consultation in order to clear up their future child pathology development probability.

1. What type of inheritance is typical for phenylketonuria?
 2. Define the probability of this pathology development in second child.
 3. What methods are used for diagnostic and treatment of this disease?
-
-
-
-

Task №3

Sergey D., 3 years old, has increased bleeding sickness from his birth. He often has hemorrhages in big joints (knee-joint and hip-joint). Diagnosis: hemophilia A.

1. What type of inheritance is characteristic for this pathology?
 2. Who is a carrier of pathologic gene?
-
-
-
-

Task №4

Family with inherited pathology child was prescribed medical genetic consultation in order to prevent birth of second child with inherited disease. Clinic genealogy, cytogenetic and biochemical methods were used in order to fulfill complete diagnostics.

1. Describe this diagnostic methods.
-
-
-
-
-
-

Task № 5

Karyotype study of patient G., revealed 47 chromosomes, three sex chromosomes (XXY).

1. Define hereditary disease with such karyotype.
2. Give the description of the patient with this disease.

Task № 6

A child 8 years old has low hearing, convulsions attacks, mental retardation. Wassermann test is positive in child and in his mother.

1. Is this disease inherited one?
2. Prove your answer

Teacher's signature

UNIT №5

Cell pathology

1. Definition, main types and causes of cell damage.
2. Morphological and functional features of cell damage.
3. Mechanisms of cellular damage.
4. Mechanisms of cellular membrane damage. The role of peroxide lipid oxidation in membranes damage.
5. Types of cell death: necrosis and apoptosis.
6. Mechanisms of protection during cellular damage.

Experimental work. Study the morphological and functional features of cell damage under the effect of physical and chemical factors.

Put 1-2 drops of Infusorium culture on three numerated object-plates. On the first object-plate add a chip of kitchen salt to the edge of culture drop. Irradiate the culture on the second object-plate for 10 minutes from distance of 20 cm using mercury quartz lamp. Microscope the culture on the third object-plate as a control one first, and then expose to the effect of lower temperature ($+5^{\circ}\text{C}$ for 10 min), then high temperature ($+30^{\circ}\text{C}$ for 10 min) valuating the mobility of Infusorii.

In experimental results: during microscoping of Infusorium culture, value, sketch and describe morphological and functional features of cell damage that rise under the influence of effects applied.

Cell damage features

morphological	functional

In conclusions: explain the mechanisms of rise of revealed disorders under different effects.

CONCLUSION

Task № 1

Patient was made blood biochemical test in order to confirm hepatitis. Increased level of adenylateaminotrensferase and aspartateaminotrensferase was found.

1. What cellular changes could lead to this situation? Substantiate your answer.
2. Explain possible mechanism of these ferments appearance in organism.
3. What clinical importance does this test have?

Task № 2

Patient with gastritis and increased acidity had fibrogastroscopy, during which the tissue was taken from the place of mucous coat erosion. Observed tissue histology: increased size cells with their form and coloring changing without features of cell organoids and membranes damage.

1. What features of cell violation (morphological of functional) are described here?
2. Point out cell adaptation feature to damage.
3. What clinic importance does this analysis have?

Task № 3

During ECG examination of the patient there were found the features of intoxication (sinus bradycardia, negative asymmetric T wave, ventricle extrasystoles, atrioventricular transmission delay). The patient was treated with heart glycosides (Strophanthinus).

1. What features of heart muscle violation does the patient have? Point out.
2. What mechanism of heart muscle violation does the patient have?

Teacher's signature

UNIT №6

The importance of reactivity in pathology. Immunological reactivity. Immunopathology

1. Reactivity and resistance: definition, their interrelation, types. Mechanisms of reactivity and resistance formation (general, nonspecific, specific).
2. Definition of human constitution: its role in pathology, classification of constitutional types according to Hippocrates, Sigo, Krechmer, Chernorutsky. Definition of diathesis, its types.
3. Role of mononuclear phagocytes system in organism resistance to the pathogenic agents effect. Phagocytosis: stages and mechanisms. Exogenous and endogenous hemotaxins. Phagocytosis violation: cause, mechanisms, consequences.
4. Organs and tissues of immune system. Specific and nonspecific immunity mechanisms.
5. Immunological reactivity: humoral and cellular mechanisms of primary immune response. Types of violation. Immunological tolerance.
6. Primary immunodeficiencies. Clinical manifestation, examples of diseases.
7. Secondary immunodeficiencies: causes and mechanisms of development. Acquired immunodeficiency syndrome (AIDS): Etiology, pathogenesis, main manifestations

Experimental work. Determine permeability of skin capillaries.

On the internal surface of forearm put plastic funnel connected with vacuum manometric plate and Kamovsky's pump by rubber tubes. With the help of pump create negative pressure of about 0,4 atm by manometer and hold this depression for 5 minutes. Valuate the results of experiment by the degree of permeability changing:

1 degree – small punctulated hemorrhages (in quantity of 10-15) are observed against a background of reddening of skin surface in the place of cuvette application.

2 degree – middle sized hemorrhages (30-40) are observed against a background of intense reddening.

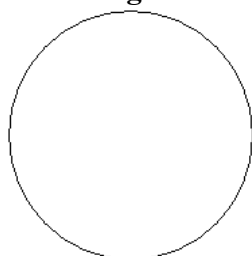
3 degree – entire hemorrhage.

The 1 degree of reaction is observed during normal level of reactivity and resistance of the organism (normergy). Hold the experiment on 3-4 students-volunteers.

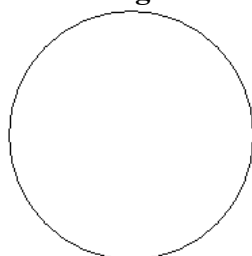
Arrange results as a table. Sketch the samples of skin capillaries reaction

Student	sex	age	Capillary permeability	
			Number of hemorrhages	degree
1				
2				
3				

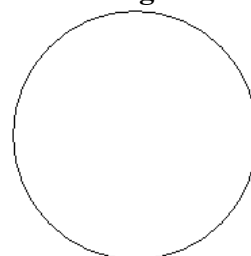
1 degree



2 degree



3 degree



In conclusions: estimate the level of individual reactivity of the organism and explain possible mechanisms of revealed disorders.

CONCLUSION

Task1

The prophylaxis of some infectious diseases includes vaccination – injection of weak microorganisms in order to stimulate the synthesis of antibodies to the given microorganisms.

1. What immune cells are able to synthesize antibodies?
2. What is the mechanism of primary immune response humoral type?
3. What types of antibodies do you know? Describe their function.

Task 2

Patient S, 15 y.o., from the early childhood is frequently ill with infectious diseases, caused by staphylococci; chronic purulent inflammation on the skin . During immunological examination the primary deficiency in phagocytes system was revealed.

1. What type of immunity do the phagocytes take part in?
2. What body cells are phagocytes? Describe all their functions.

Teacher's signature

Unit 7

The importance of reactivity in pathology. Allergic reactivity

1. Classification of allergic reactions according to Coombs and Gell. Etiology of allergy, types of allergens. Distinction between allergy and immunity.
2. Type 1 allergic reactions (anaphylactic): mechanisms, main clinical forms.
3. Type 2 allergic reactions (cytotoxic): mechanisms of development, main clinical forms.
4. Type 3 allergic reactions (immunocomplex): mechanisms, main clinical forms. Immune complexes pathogenic effect. Serum disease.
5. Type 4 allergic reactions (delayed hypersensitivity): mechanisms, main clinical forms. The role of lymphokines.
6. Pseudoallergic reactions
7. Autoallergic diseases. Causes and mechanisms of development.

Experimental work. Determine reaction of degranulation of peritoneal mast cells to antigen in sensitized rat.

Inject 10,0 ml of Tirode's solution warmed up to 37⁰ C intraperitoneally to previously sensitized rat under deep etheric narcosis. Massage the anterior wall of the belly for 15-20 seconds. Make an incision 1,5 cm long along the centerline of mesogastrium region through all layers of anterior abdominal wall. Turn the rat over, belly up, so that the content of abdominal cavity flew down to the test-tube along the intestine. The walls of the test-tube should be previously moistened with Heparin. Carefully mix the obtained peritoneal dredge with anti-coagulant for 2-3 minutes to prevent its coagulation. Using the pipette, put 2 separate drops of peritoneal dredge on the object-plate stained with Neutral Red and dried up. Add bovine serum to one of the drops, and horse serum - to the other.

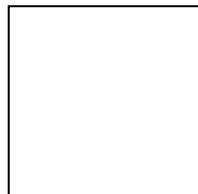
Intact mast cells are about 1,5 times bigger than leukocytes in diameter. Are proper round- or oval-shaped. Cytoplasm is filled with granules stained into purple-red color. Fading is observed in the place of nucleus. Membrane is uninterrupted, is clearly contoured during microscope screw rotation.

Degranulated mast cells have uneven external contour. The cytoplasm is vacuolized. The stoma is observed on the external contour of cells. The granules flow out through this stoma.

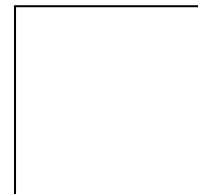
Examine each drop under microscope and count the percentage of degranulated mast cells out of 50. Sketch mast cells, intact and degranulated.

	General mast cells quantity	Degranulated mast cells quantity	% degranulation	Sensitization degree
Rat №1				
Rat №2				

intact mast cell



degranulated mast cell



In conclusions: explain the mechanisms of degranulation, evaluate the clinical significance of reaction performed.

CONCLUSION

Task 1.

Patient was injected with Novocaine solution in order to provide local anesthesia during tooth extraction . In 1 minute after injection the patient turned pale and unconscious. ABP – 90/60 mmHg, heart rate – 128 bpm.

1. What has happened to the patient?
2. What type of allergic reaction can be the reason of the patient's state?
3. Describe the pathogenesis of the described clinical signs.

Task 2.

Patient complains of the skin rashes, which appear after cooling the skin (cold water or cold air exposure). After returning from the street in the winter the opened areas of the skin turn red (hyperemia) and itches appear.

1. Is it an allergic reaction, or no? Prove your answer.
2. Explain the pathogenesis of the described clinical signs.

Task 3

A nurse complains of the rashes on the skin of the hands. Her usual work is to do injection of antibiotics and other medicines to the patients. The symptoms of skin irritation usually disappear after summer vacations. In 7-10 after working with the solutions of medicines the symptoms of rashes appear again.

1. Is it an allergic reaction, or no? Prove your answer.
2. Explain the pathogenesis of the described clinical signs.
3. What biologically active substances provide the development of the described clinical signs?

Teacher's signature _____

Unit 8

SUB-MODULE 1 CONTROL Checking of practical skills and theoretical knowledge «GENERAL NOSOLOGY. PATHOGENIC INFLUENCE OF THE ENVIRONMENTAL FACTORS. THE ROLE OF THE INTRINSIC FACTORS IN PATHOLOGICAL STATES DEVELOPMENT».

The final practical class in sub-module 1 consists of estimating student's rating grade. To be allowed to pass sub-module 1 control the student should:

1. get no less than 18 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes' protocols signed by the teacher and show the lectures notebook.

Students have an opportunity to pass sub-module control once more during two weeks. If sub-module control was not successfully passed in two weeks they will not be allowed to the following lectures and practical classes and rating mark for sub-module 1 control will be "zero".

SUB-MODULE 1 PRACTICAL SKILLS

1. Students should be able to analyze:

- basic definitions of the general nosology (health, disease, remission, relapse complication, pathological reaction, pathological process, pathological state, etiology, pathogenesis, reactivity, resistance, adaptation, compensation);
- etiological factors influence on the organism (causes, risk factors, conditions of diseases development);
- mechanisms of pathogenic and sanogenic influence of physical, chemical and biological factors;
- causality-effective relations in pathogenesis (to define local and general changes, pathogenic and adaptive, specific and non-specific events, the leading event in the pathogenesis of disease);
- causes, mechanisms of development, signs and consequences of cell's injury;
- disturbances of immune system function (immune deficiency, allergy);
- mechanisms of cells, tissues and organs allergic injury (due to Gell and Coombs classification).

2. To obtain practical skills:

- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in such states:
electrical current affection, burns and frostbites, excessive influence of ultraviolet and infrared rays (sunstroke and heat stroke), radiation sickness, decompression and compression sickness, intoxication, congenital and inborn diseases, immune deficiency, allergy, autoimmune diseases, pseudoallergic reactions.
- to describe mechanisms of pathogenesis of the given states, to reveal the leading event in the pathogenesis, to make a prognosis of development.
- to explain the main principles of diseases prophylaxis and treatment on the basis of analysis of etiology, pathogenesis and clinical manifestation of the disease.

UNIT №9

Violation of peripheral blood circulation

- 1.Arterial hyperemia: definition, types, mechanisms of development, consequences.
- 2.Venous hyperemia: definition, types, mechanisms of development, consequences.
- 3.Ischemia: definition, causes, types, pathogenesis, consequences. Infarction: types and mechanisms of development
- 4.Stasis: definition, causes, pathogenesis, consequences. Types and mechanisms of stasis.
- 5.Thrombosis: definition, types, mechanism of thrombus formation..
- 6.Embolism: definition, types of embolism. Embolism of big and small circles of circulation of the blood, system of portal vein.

Experimental work. Demonstration of peripheral blood circulation changes in human.

Put the rubber tourniquet on the arm of student-volunteer until pulse disappearance on arteria radialis. Fixate in such position for 30 seconds. Observe the development of ischemia. Then gradually loosen the tourniquet until appearance of pulse and fixate again. Observe the development of venous hyperemia. After removal of tourniquet observe the development of arterial hyperemia.

In experimental results: consequently describe the signs of peripheral blood circulation disturbance modeled in experiment.

	color of the skin	temperature of the skin
ischemia		
venous hyperemia		
arterial hyperemia		

In conclusions: explain the mechanisms of appearance of disturbances.

CONCLUSION

Task 1

The driver T. has got a trauma of the neck with the damage of jugular vein.

1. What kind of embolism will develop in the patient?
2. Explain the mechanism of this embolism development.
3. How can you prevent this embolism?

Task 2

The sportsman felt his extremities hot after body-building exercises, the color of the skin was red and hot to the touch.

1. What kind of peripheral blood circulation disturbance had developed in sportsman?
2. Are these signs normal?
3. Explain the mechanism of development of this condition.

Task 3

The experiment was held on the rabbit: sympathetic nerves that innervate the left ear were cut.

1. What violation of peripheral blood circulation will occur in the experiment?
2. Explain the mechanism of its development.
3. What other types of this violation of peripheral blood circulation can you name?

Task 4

Patient A., 50 years, is suffering from diabetes mellitus for 5 years. He has been working as a salesman for 30 years. The patient complains about pains in the legs. Clinical examination of the legs: edema, skin is cyanotic and cool, small subcutaneous hemorrhages.

1. What violation of peripheral blood circulation has developed in patient?
2. Explain the mechanism of its development.
3. Explain the mechanism of edema, low skin temperature and subcutaneous hemorrhages?

Task 5

Patient G. has got a wound of the arm. The nurse dress a wound with a tight bandage. In a few hours he felt pain in the wounded arm, the skin color turn pale, its temperature decreased.

1. What violation of peripheral blood circulation has developed in patient?
2. Explain the mechanism of its development.

3. What other negative consequences can occur if the bandage will not be removed?

Task 6

Patient B., 30 years old, has got a fracture of right femur bone. During reposition of bone's breaks patient felt pain in the left side of chest, which was enhanced with breathing; palpitation, short breath and feeling of faint.

1. What kind of peripheral blood circulation disturbance had developed in patient?
2. Explain the mechanism of its development.
3. What possible complication can develop in this clinical case?

Task 7

The development of thrombosis was provided in the experiment on laboratory animal.

1. What conditions are favorable for thrombus formation?
2. What type of vessels do thrombi usually form in?
3. What complications of thrombosis do you know?

Teacher's signature

UNIT № 10

Inflammation. Alteration, vascular reactions and exudation under inflammation. Exudation mechanisms studying in experiment

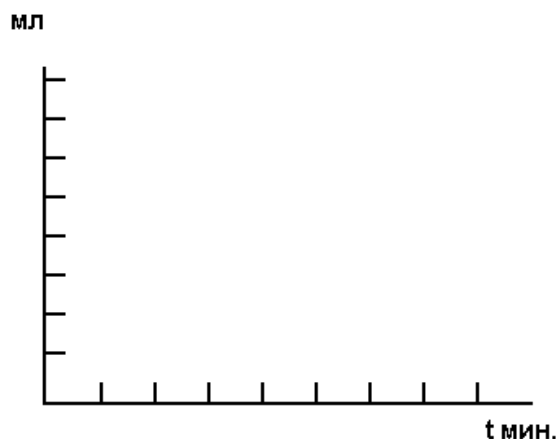
1. Inflammation: definition, etiology, characteristic, stages.
2. Primary and secondary alteration: causes and mechanisms. Physical and chemical changes in inflammation center.
3. Inflammation mediators, their origin, mechanisms of action. Alteration importance.
4. Local blood circulation violation under inflammation. Vascular reactions order under inflammation, their mechanisms and importance.
5. Exudation mechanisms in inflammation center. Causes and mechanisms of blood vessels penetrations extension. Inflammatory edema pathogenesis. Exudation importance.
6. Leucocytes emigration mechanisms in inflammation center. Leucocytes marginal standing, its mechanisms. Leucocytes role in development of inflammation local and general manifestation. Phagocytosis importance.
7. Regeneration and repair mechanisms. Difference between regeneration and repair
8. Inflammation classification principles. Role of reactivity, neural and hormonal factors in inflammation development. Inflammation outcomes.

Experimental work. Investigation of the inflammatory edema development in experiment.

The rat is given narcosis (0,7 ml of 1 % Thiopental Sodium solution intraperitoneally). Measure the volume of hind legs putting them one by one into measuring test-tube. Measure the volume of liquid forced out by leg adding water from burette to the test-tubes. Inject 0.1 ml of physiological solution into the control leg, and 0.1 ml of 1 % formalin solution – into the experimental leg. Measure the volume changes of both legs every 5 min during 30 min, valuating the degree of their edema, hyperemia and functional disturbances. Pay attention to development of main signs of inflammatory reaction.

Register obtained data in a table and build a diagram that depict dependence of changes in legs volume on time that passed from the beginning of experiment. Make conclusions about mechanisms of exudation development.

	Volume of the leg, ml	
before injection		
injection	0.1 ml of 0,9% NaCl solution	0.1 ml of 1 % formalin solution
5 min after injection		
10 min after injection		
15 min after injection		
20 min after injection		
25 min after injection		



CONCLUSION

Task 1

Patient C., 51 years old, with diagnosis “peritonitis”. After paracentesis (puncture of abdominal cavity) liquid was obtained which had the following signs: yellow color with green tint, density 1,029; proteins content – 0,39 g/L. In sediment: high amount of neutrophils with degenerative forms presence and purulent bodies.

1. What type of liquid was obtained after paracentesis?
2. Explain the mechanism of liquid appearance in abdominal cavity.

Task 2

Patient A., 35 years old, has got a burn of arm, its size was 2×2 cm, III stage. During repeated examination 2 days later it was obtained that the size of inflammation site increased 4×3 cm, color around site was cyanotic, painful, with high tension.

1. Why site of inflammation had been increased?
2. What factors do take part in this process?

Task 3

Patient B., 65 years old, with diagnosis “heart failure”. During patient’s examination it had been revealed accumulation of liquid in abdominal cavity (ascites). After diagnostic puncture liquid had the following signs: transparent, color was light-yellow, density – 1,014; proteins content 0,2 g/L. In sediment: single cells (most of them are lymphocytes).

1. What type of liquid was obtained after paracentesis?
2. Does the patient have signs of inflammation?

Task 4

Some drops of turpentine got on mucosal membrane of the eye of laboratory animal. 15 minutes later expressed inflammation appeared: redness of conjunctive, dilatation of mucosal membrane’s capillaries, swelling and pain.

1. Will the inflammation develop if you’ll quickly apply anesthetics on eye’s mucosal membrane?
2. What is the mechanism of swelling development?

Task 5

Patient F., 30 years old, had made tuberculin skin test – Mantoux reaction (intracutaneous injection 2 TU of tuberculin) – for diagnostic purpose. 24 hours later in site of injection painful red infiltration appeared, its size was 25mm. Body temperature was increased to 37,2 °C. A doctor estimates such reaction as positive hyperergic.

1. What signs are the evidence of inflammation's development in patient? Prove your answer.
 2. What is mechanism of alteration in this case?
 3. Explain the mechanism of inflammatory site increase.
-
-
-
-
-
-
-
-

Task 6

Patient U. was hospitalized to the hospital with the suspicion of diphtheria. The following signs were observed after examination: the mucosal membrane of throat was red and covered with grey thin coating, which was tightly connected with underlying tissues. Body temperature was 39°C, tachycardia, tachypnea, skin was pale and humid.

1. What kind of inflammation (alterative, exudative, proliferative) is characterized in this case? Prove your answer.
 2. Explain the mechanism of thin coating formation on the mucosal membrane of the throat.
-
-
-
-
-
-
-
-

Teacher's signature _____

UNIT № 11
Thermoregulation pathology. Fever

1. Normal thermoregulation.
2. Definition of fever. Etiology and types of fever. Primary and secondary pyrogens.
3. Fever pathogenesis. Stages of fever.
4. Importance of fever
5. Hyperthermia: stages, mechanism of development. The distinction from fever
6. Hypothermia stages, mechanism of development.

Experimental work. Investigate hypothermia in normal and narcotized rats.

Count the number of breaths and measure the rectal temperature of 2 rats. Pay attention to the color of visible coverlet, eyes, general condition of the animal. Inject Thiopental Sodium to one rat intraperitoneally (0.5 ml of 1 % solution per 100 gr. of body mass). Both animals are placed into desiccator with ice after the beginning of narcosis. Measure the temperature and count the quantity of breaths repeatedly every 10 minutes.

Arrange obtained results as a table

RESULTS

Time (min)	Breaths in 1 min	Temperature	color of skint	color of eyes	general condition
0					
10					
20					

In conclusions: explain the mechanisms of obtained results, determine the role of nervous system in mechanisms of heat regulation.

CONCLUSION

Task 1

Patient M., 52 years old. Diagnosis: bronchial asthma. Patient is treated with glucocorticoids. Fever reaction appeared as a result of post-injective abscess development. Subfebrile temperature didn't correspond to heaviness of inflammatory process.

1. Why patient has low fever reaction?

Task 2

Patient T, 47 years old, delivered to the hospital by ambulance complains for increasing pain behind sternum and in the epigastrical area during a day. During patient's examination were found: paleness of skin, hyperhydrosis, acrocyanosis, body's temperature 38,0°C, ABP 100/65 mmHg, heart rate – 100 bpm. The analysis of ECG revealed myocardial infarction.

1. What is the cause of temperature increase in this case?
2. Should this patient be prescribed antipyretic medicines?

Task 3

Patient V., 32 years old, complaints about headache, weakness, myalgia, pain in extremities, stuffiness in nose, fever in the morning. Temperature rose to 39.2°C. Diagnosis: influenza.

1. What is starting devices of fever development in this case?
2. What protective features of the fever can you name?

Task 4

Patient T, 27 years old, was operated for mitral insufficiency. Systemic controlled hypothermia was conducted and his body's temperature was decreased to 34°C after narcosis. The operation on the dry heart, which last for 40 minutes, was effective.

1. Explain, why hypothermia was conducted to the patient?
2. Explain, why organism not so sensitive for intoxication, infection, hypoxia, current defeat during hypothermia.

Teacher's signature _____

UNIT № 12

Tissue growth pathology. Tumors.

1. General characteristic of tissue growth violation main types.
2. Definition of “tumor”. Biological peculiarities of tumor growth.
3. Anaplasia. Features of malignant neoplasms.
4. Tumors etiology.
5. Pathogenesis of tumors.
6. Invasion and metastasis of malignant tumor cells

Experimental work. Determine anti-trypsin properties of serum of healthy person and oncologic patient.

The principle of anti-trypsin reaction: mix trypsin, casein and probationer’s serum by the scheme:

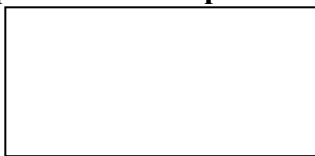
Serum	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Trypsin	1.0	1.2	1.4	1.6	1.8	2.0	2.2	2.4	2.6	2.8	3.0
Physiologic solution	2.0	1.8	1.6	1.4	1.2	1.0	0.8	0.6	0.4	0.2	0
Casein	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0

Shake the liquid in test-tubes and place into thermostat for 30 min. After this, carry out bedding of 30 % nitric acid in each test-tube. Determine the bounds of protein digestion in both sets of test-tubes by the appearance of a circle.

The degree the casein digestion delay depends on the quantity of anti-trypsin substances in the serum. Compare results with data obtained in the same conditions but with normal serum.

In experimental results: sketch the test-tubes with positive and negative anti-trypsin reaction.

positive anti-trypsin reaction



negative anti-trypsin reaction



In conclusions: describe possible mechanism of accumulation of anti-trypsin substances in the serum of oncologic patient, explain the specificity of given reaction, evaluate its importance for clinics.

CONCLUSION

Task 1

Patient R., 52 years old, complained about weakness, abdomen pains, digestion disorder, and 40% weight waste. Laparotomy: pancreas tumor with metastases in mesentery of intestines, liver and spleen. Carbohydrate, protein and lipid metabolisms disorder were found. Blood analysis: hypoglycemia, hyperlipidemia, hyponitrogenemia, negative nitrogenic balance.

1. How weight waste can be explained, and what are possible mechanisms of this effect?
2. What mechanisms causes carbohydrate, protein and lipid metabolisms disorder?

Task 2

Patient G., 35 years old. Leukemia was found in 3 months after he was rayed by ionizing radiation. He had 15 kg weight waste, there were a lot of hemorrhages of different size on the skin. Roentgenogram revealed pneumonia.

1. What is a possible mechanism of patient's tumor development?
 2. What is cell division limit (Hayflick's limit)? How does it change in tumor cells?
-
-
-
-

Task 3

Patient E. Right mamma tumor without pain was found, skin around it wasn't hyperemied, there was no temperature rise. Histology research: tumor is in capsule, there is no inflammation around the tumor, expansive growth, tumor cells are equal in size and form, tumor metastases in other organs and tissues were not found.

1. What type of tumor does patient have? Prove your answer.
 2. Why this type of tumor is surrounded with capsule?
-
-
-
-

Task № 5

Cells clone was cultivated in vitro (in Petri dish). If oxygen is added, lactate is secreted, if glucose solution is added, glucose concentration in the clone equals to 0.

1. Determine the character of cellular clone, which was cultivated in the experiment.
 2. What was a cause of glucose absence and lactate formation in the cellular clone?
-
-
-
-

Teacher's signature _____

UNIT № 13

Hypoxia

1. Hypoxia as pathogenic factor of different diseases. Types of hypoxia
2. Etiology and pathogenesis of different types of hypoxia.
 - a) hypoxic hypoxia
 - b) hemic hypoxia
 - c) tissue hypoxia
 - d) circulatory hypoxia
3. Protective adaptive and compensatory reactions under hypoxia
4. Clinical application of adaptation to hypoxia.

Experimental work. Ber-Sechenov's experiment.

Build a machine consisting of vacuum plate, Kamovsky's pump and oxygen bag. Count rat's respiratory movements, note coverlet color, behavior and general condition of the animal. Arrange results as a table. Then place the rat under the bell-glass of vacuum plate. Imitate "rising to height" of 4, 9 and 11 km, evacuating air from under the bell-glass. Count rat's respiratory movements, note coverlet color, behavior and general condition of the animal on each stage of "rising". Calculate the partial pressure of oxygen. Arrange results as a table. Watch the development of altitude sickness symptoms, continuing to evacuate the air: the animal gets weaker, lays down, possibly develops convulsions on the peak of anoxaemia. After appearance of clearly expressed signs of altitude sickness, slowly let the air under the bell-glass and watch the recovery of disturbed functions.

Repeat the experiment considering the changes of atmospheric air composition under the bell-glass (50-70 % of oxygen). Determine the "height" when the altitude sickness symptoms appear during the second raising with changed atmospheric air composition. Arrange results as a table.

RESULTS

1st experiment

Altitude	Atmosphere pressure	p O ₂ 21%	Breaths in 1 min	Skin color	General condition
0	760				
4 km	463				
9 km	231				
11 km	170				

2nd experiment

Altitude	Atmosphere pressure	p O ₂ 70%	Breaths in 1 min	Skin color	General condition
0	760				
4 km	463				
9 km	231				
11 km	170				

In conclusions: analyze experimental results and clarify the reason of altitude sickness appearance. Explain the mechanisms of development of respiration changes, coverlet changes, general condition of the animal.

CONCLUSION

Task № 1

Patient K., 43 years old, complaints of breathlessness during physical work. Clinical examination: pale skin, acrocyanosis, hard breathing, dry, dissipated crepitation is heard over the lungs. Roentgenogram: diffuse pneumosclerosis. Arterial blood saturation by oxygen is 74%. Anamnesis: the patient has been working at the asbestosis plant during 10 years.

1. What type of hypoxia does the patient have?
2. What mechanism underlies patient hypoxia development? Prove your answer.

Task № 2

Geologist went to the mountains. He felt worse in the 2nd day of being there. Mountain disease symptoms: breathlessness, skin cyanosis, tachycardia, headache, appetite loss, general weakness, insomnia occur.

1. What factor causes mountain disease?
2. What pathogenic mechanism underlies symptoms which developed in the patient?

Task № 3

Patient K., 45 years old. Diagnosis: prolonged stomach ulcer. He was hospitalized because of stomach bleeding. The patient complaints of weakness, nausea, giddiness, tinnitus, flashing in the eyes. He is pale and has expressed breathlessness, moderate pain in epigastric region. Arterial pressure – 100/70 mm of mercury. Pulse – 95 per minute.

1. What hypoxia features does this patient have?
2. What type of hypoxia does patient have? Prove your answer.

Task № 4

Patient K., 32 years old, was intoxicated by carbon monoxide. His complaints about headache, nausea, cough. Clinical examination: mental confusion, red skin, respiration is frequent and superficial, tachycardia, arterial pressure – 145/100 mm of mercury.

1. What type of hypoxia does patient have? Prove your answer.

2. Why patient's skin is red?
3. What clinic features are the manifestation of organism protective adaptive reactions to hypoxia?
Explain their importance and mechanism of development.

Teacher's signature

UNIT 14

SUB-MODULE 2 CONTROL Checking of practical skills and theoretical knowledge «TYPICAL PATHOLOGICAL PROCESSES».

The final practical class in sub-module 2 consists of estimating student's rating grade. To be allowed to pass sub-module 2 control the student should:

1. get no less than 15 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes protocols signed by the teacher and show the lectures notebook.
4. successfully pass sub-module 1

Students have an opportunity to pass sub-module control for the second time during two weeks. If sub-module control was not successfully passed in two weeks student will not be allowed to attend the lectures and practical classes and rating mark for sub-module 2 control will be "zero"..

SUB-MODULE 2 PRACTICAL SKILLS

1. Students should be able to analyze:

- the role of reasons and conditions in typical pathological processes onset and development;
- typical pathological processes due to the principles of their classification, clinical manifestation and outcomes;
- the role of typical pathological processes in different diseases onset, development and outcomes;
- stages of typical pathological processes development (inflammation, fever, tumor);
- local and general events, pathogenic and adaptive mechanisms of development, specific and non-specific changes, leading pathogenic mechanism of typical pathological processes;
- urgent (adaptive) and long-term (compensatory) mechanisms in typical pathological processes development.

2. To obtain practical skills:

- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in the case of: arterial and venous hyperemia, ischemia, thrombosis, embolism, stasis, sludge syndrome, inflammation, fever, tumor, hypoxia.
- to describe mechanisms of pathogenesis of the given states, to reveal the leading event in the pathogenesis, to make a prognosis of development, to explain the main principles of diseases prophylaxis and treatment on the basis of etiology, pathogenesis and clinical manifestation of the disease analysis.

UNIT № 15
Carbohydrate metabolism. Diabetes mellitus

1. Disturbance of nervous and hormone regulation of carbohydrate metabolism. Definition of insulin-dependent and insulin-independent tissues of the organism.

2. Diabetes mellitus. Classification. Etiology of insulin-dependent (1st type) and insulin-independent (2nd type) diabetes mellitus. The difference between these types.

3. Disturbance of carbohydrate, protein, lipid, water-electrolyte exchanges and acid-base balance during diabetes mellitus.

4. Mechanisms of development of main clinical features and complications of diabetes mellitus.

5. Pathogenesis of diabetic comas: hyperglycemic, hypoglycemic, hyperosmolar, hyperlactacidemic.

Experimental work.

1. Measurement of glucose concentration by glucometer Super Glucocard II in intact and diabetic rats.

1. Insert lancet into Multi-Lancet.

2. Open the package with the test strip. Insert the disposable test strip into the test meter. Test strip contains electro-chemical compound which generates electrical impulse proportionally to blood glucose concentration.

3. To get a blood drop with the help of Multi-Lancet and apply it on a test strip.

4. 3 μ L of blood will be used for measurement of blood glucose level. The measurement is completed in 30 seconds and the test result appears on the display.

The normal blood glucose plasma is 3,3-5,5 mmol/L.

You need to measure blood glucose concentration in 2-3 students.

Student's name.	Glucose concentration	Normal value	Conclusion

2. Holding a glucose tolerance test in intact and diabetic rats.

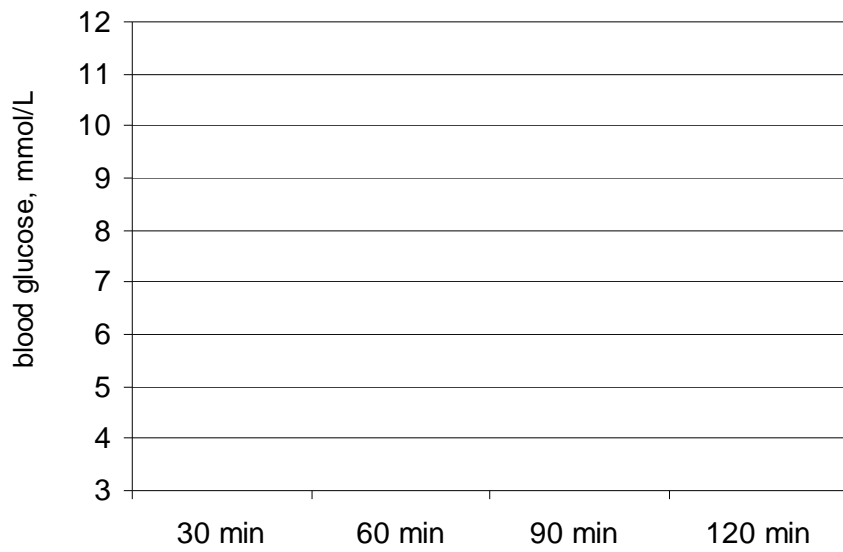
After measurement of glucose concentration in intact and diabetic rats, inject glucose (dose 1,75 gr. per 1 kg.) intraperitoneally to both rats. In 30, 60, 90 min and in 2 hour determine glucose concentration in the blood repeatedly. Arrange obtained data as a table.

Glucose tolerance test interpretation

	<i>capillary blood glucose (mmol/l)</i>		
	<i>On an empty stomach</i>	<i>30, 60, 90 min after sugar load.</i>	<i>120 min after sugar load.</i>
Norm	< 5.6	< 11,1	< 7.8
Impaired glucose tolerance	> 5.6	\geq 11.1	7.8 – 11.1
Diabetes mellitus	\geq 6.1	\geq 11.1	\geq 11.0

Write the sample profiles for every type of glucose tolerance and built a diagram.

	<i>capillary blood glucose (mmol/l)</i>		
	<i>On an empty stomach</i>	<i>30, 60, 90 min after sugar load.</i>	<i>120 min after sugar load.</i>
Norm			
Impaired glucose tolerance			
Diabetes mellitus			



Explain mechanisms of glucose metabolism disturbance in diabetes development.

CONCLUSION

Teacher's signature

Unit 16

Acid-base balance pathology (ABB) and water-salt metabolism disturbances

1. Mechanisms of ABB maintenance in the organism: blood buffer systems, physiological mechanisms of ABB regulation.
2. Typical disturbances of ABB: acidosis and alkalosis. Classification, definitions.
3. Gas acidosis, causes and mechanisms of development, compensation mechanisms.
4. Metabolic acidosis, kinds, causes and mechanisms of development, compensation mechanisms.
5. Gaseous and non-gaseous alkalosis, causes and mechanisms of development, compensation mechanisms.
6. Edema: definition, classification, etiology, pathogenesis of different edema types.

Normal indices of ABB:

pH of arterial blood	7,35-7,45 (7,36-7,42)
pH of venous blood	7,26-7,38 (7,26-7,36)
pH of capillary blood	(7,35-7,44)
pO ₂ of arterial blood	85-95 mm of mercury.
pO ₂ of venous blood	40-45 mm of mercury.
pCO ₂ of arterial blood	35-45 mm of mercury.
Standard bicarbonate (SB)	20-24 mmol/L
Buffer base(BB)	44-46 mmol/L
Buffer base shift (BE)	±2 mmol/L

Kinds of ABB disorders	Hydrogen ion exponent - pH	Partial pressure in blood pCO ₂	Standard bicarbonate - SB
norm	7,37 – 7,43	35 – 45 mm of mercury	22 – 26 mmol/L
Gas acidosis			
compensated	N	↑↑	↑↑
partly compensated	↓	↑↑	↑
decompensated	↓↓	↑↑	N
Metabolic acidosis			
compensated	N	↓↓	↓↓
partly compensated	↓	↓	↓↓
decompensated	↓↓	N	↓↓
Gas alkalosis			
compensated	N	↓↓	↓↓
partly compensated	↑	↓↓	↓
decompensated	↑↑	↓↓	N
Metabolic alkalosis			
compensated	N	↑↑	↑↑
partly compensated	↑	↑	↑↑
decompensated	↑↑	N	↑↑

Task 1

Patient C, is suffering from lungs emphysema and respiratory insufficiency. ABB indices: pH=7,36; pCO₂=56 mmHg; BB=50 mmol/L; SB=29 mmol/L; BE=+8 mmol/L.

1. Estimate ABB and explain the reason of possible changes?

Task 2

Patient Z, 26 y.o, was delivered to the hospital with acute pneumonia. The patient is in the hard state, body temperature 39,8⁰C, short breath is present. ABB indices:

pH=7,47; pCO₂=29 mmHg; BB=40,2 mmol/L; SB=16,5 mmol/L; BE=-1,8 mmol/L.

1. Estimate ABB and explain the reason of possible changes?

Task 3

A child 4 years old was delivered to the hospital on the suspicion of acute intestinal infection: high body temperature, frequent watery stool (8-10 times a day). The child is slightly dehydrated, short breath is observed. ABB indices:

pH=7,39; pCO₂=28 mmHg; BB=34 mmol/L; SB=16 mmol/L; BE=8 mmol/L.

1. Estimate ABB and explain the reason of possible changes?

Task 4

Patient R, 54 y.o. was delivered to the hospital in a hard state. He complains of the excessive fatigue and severe weight loss. For the last week he feels the pain in the stomach after every meal. The attacks of pain result in vomiting with gastric content. ABB indices:

pH=7,55; pCO₂=60 mmHg; BB=54 mmol/L; SB=29 mmol/L; BE=+18 mmol/L.

1. Estimate ABB and explain the reason of possible changes?

Teacher's signature

Unit 17

Disturbances of protein and lipids metabolism. Starvation, obesity.

1. Mechanisms of protein metabolism maintenance. Laboratory indices of protein metabolism.
2. Gout: etiology, pathogenesis, clinical manifestation, complications, diagnosis and treatment principles.
3. Mechanisms of lipid metabolism maintenance. Laboratory indices of lipid metabolism.
4. Obesity: etiology, pathogenesis, types, clinical manifestation, complications, diagnosis and treatment principles.
5. Starvation: etiology, pathogenesis, types, clinical manifestation, complications. Medical starvation.

Experimental work. To calculate body mass index.

The body mass index (BMI) uses height and weight to determine healthy weight. It is calculated by dividing the weight in kilograms by the height in meters squared ($BMI = \text{weight [kg]} / \text{height [m]}^2$)

Classification of Overweight and Obesity by BMI, Waist Circumference, and Associated Disease Risk

	BMI(kg/m ²)	Obesity Class	Disease Risk Relative to Normal Weight and Waist Circumference	
			Men ≤ 102 cm Women ≤ 88 cm	Men > 102 cm Women > 88 cm
Underweight	<18.5			
Normal	18.5–24.9			
Overweight	25.0–29.9		Increased	High
Obesity	30.0–34.9	I	High	Very high
	35.0–39.9	II	Very high	Very high
Extreme obesity	≥ 40	III	Extremely high	Extremely high

Increased waist circumference also can be a marker for increased risk, even in persons of normal weight.

Experimental results

Student name	Height, kg	Weight, m	BMI	Conclusion

CONCLUSION

Situational Problem

A 25-year-old woman is 165 centimetres tall and weighs 136 kilograms. She works as a receptionist in an office, brings her lunch to work with her, spends her evenings watching television, and

gets very little exercise. She reports that she has been fat ever since she was a little girl, she has tried “every diet under the sun,” and when she diets she loses some weight, but gains it all back again.

1. How would you classify patient’s obesity?
2. What are her risk factors for obesity?
3. What would be one of the first steps in helping her develop a plan to lose weight?

Teacher’s signature

Unit 18

SUB-MODULE 3 CONTROL Checking of practical skills and theoretical knowledge «TYPICAL METABOLISM DISTURBANCES».

The final practical class in sub-module 3 consists of estimating student's rating grade. To be allowed to pass sub-module 3 control the student should:

1. get no less than 9 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes protocols signed by the teacher and show the lectures notebook.
4. successfully pass sub-module 1 and 2

Students have an opportunity to pass sub-module control for the second time during two weeks. If sub-module control was not successfully passed in two weeks student will not be allowed to attend the lectures and practical classes and rating mark for sub-module 3 control will be "zero".

SUB-MODULE 3 PRACTICAL SKILLS

1. Students should be able to analyze:

- the role of causes and conditions in typical metabolism disturbances development;
- typical metabolism disturbances due to classification principles, local and general manifestation;
- the role of typical metabolism disturbances in different diseases development;
- the stages of in typical metabolism disturbances pathogenesis;
- etiology, pathogenesis clinical manifestations and complications of diabetes mellitus and differentiate type 1 and type 2 diabetes mellitus.

2. To obtain practical skills:

- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in the case of:
carbohydrate metabolism disturbances, diabetes mellitus, ABB imbalances, misbalances of water and salt metabolism, edema, disturbances of energy metabolism, disturbances of lipid metabolism and obesity, disturbances of protein metabolism and gout, disturbances of vitamins metabolism, starvation.
- to describe mechanisms of pathogenesis of the given states, to reveal the leading event in the pathogenesis, to make a prognosis of development, to explain the main principles of diseases prophylaxis and treatment on the basis of etiology, pathogenesis and clinical manifestation of the disease analysis.

UNIT 19
FINAL MODULE CONTROL. MODULE 1.

General nosology. Typical pathological processes. Typical metabolism disturbances.

The final module control consists of estimating student's rating grade. To be allowed to pass final module control the student should:

1. get no less than 51 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. successfully pass sub-module 1, 2, 3

Final module control consists of computer testing. In order to pass final module control successfully, student should obtain such theoretical and practical skills:

- to analyze basic definitions of general nosology, etiologic factors influence on the human organism;
- to analyze the role of reasons and conditions, causality-effective relations in pathogenesis;
- to define and classify typical pathological processes and typical metabolism disturbances;
- to define the role of typical pathological processes and typical metabolism disturbances in the pathogenesis of different diseases;
- to reveal protective and pathogenic features of typical pathological processes, short-term and long term adaptive and compensatory reactions in the pathogenesis of typical pathological processes and typical metabolism disturbances;
- to analyze the reasons, mechanisms of development, consequences of cell injury and to define their role in typical pathological processes and diseases pathogenesis;
- to define and analyze the disturbances in immune system function (immunodeficiency and allergy) and tissues injury mechanism during allergic reaction development;
- to define and analyze etiology, pathogenesis clinical manifestations and complications of diabetes mellitus;
- be able to solve situational problems – to define etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment of such conditions:
electrical current affection, burns and frostbites, excessive influence of ultraviolet and infrared rays (sunstroke and heat stroke), radiation sickness, decompression and compression sickness, intoxication, congenital and inborn diseases, immune deficiency, allergy, autoimmune diseases, pseudoallergic reactions;
arterial and venous hyperemia, ischemia, thrombosis, embolism, stasis, sludge syndrome, inflammation, fever, tumor, hypoxia.
carbohydrate metabolism disturbances, diabetes mellitus, ABB imbalances, misbalances of water and salt metabolism, edema, disturbances of energy metabolism, disturbances of lipid metabolism and obesity, disturbances of protein metabolism and gout, disturbances of vitamins metabolism, starvation.
- to explain principles of typical pathological processes and typical metabolism disturbances experimental modeling;
- to analyze laboratory findings and be able to use them in diagnosing different pathological processes and diseases

UNIT 20
Summing of final module control results.

1. Analysis of final module control results.
2. Students reports on the topic on independent studying
3. Estimation of the student's marks for the autumn semester.