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### FACULTY OF MEDICINE

#### STUDY PROGRAM 0912.1 MEDICINE

### CHAIR OF PATHOPHYSIOLOGY AND CLINICAL PATHOPHYSIOLOGY

#### APPROVED

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at the Council meeting of the Faculty Medicine II

at the meeting of the Commission for Quality Assurance and Evaluation of the Curriculum in Medicine Protocol nr. 16,09,21

Chairman, PhD, university professor

Suman Serghei

Dean of Faculty Ph.D., associate professor Bețiu Mircea

Protocol nr. 1 from 21,

#### APPROVED

approved at the meeting of the Chair of physiopathology and clinical physiopathology Protocol nr.1 from 11.09.2021 Head of the Chair PhD, university professor Cobet Valeriu U.Cobet

## SYLLABUS

#### DISCIPLINE PATHOPHYSIOLOGY

#### **Integrated studies**

Type of course: Compulsory

Syllabus elaborated by authors:

Cobet Valeriu, PhD, university professor

Lutan Vasile, PhD, university professor

Hangan Cornel, PhD, associate professor

Chișinău, 2021



## I. INTRODUCTION

General presentation of the discipline: place and role of the discipline in the formation of the specific competences of the professional trening program.

Pathologic physiopathology (pathophysiology) is a fundamental medical science and preclinical discipline which at the level of univesity training allows: a) formation of biological and medical concepts of the essence of pathological processes and diseases; b) aquisition of skills for pathophysiological experiment and interpretation of the obtained information in the experiment; c) studing the general laws of origin, occurence, evolution and resolution of typical pathological processes and nosology entities; d) studing of the functional disorders and morphological changes at the molecular, cellular, tissue, organ, system and systemic organism levels in typical pathological processes and diseases; e) to know pathogenetic principles for correction of disorders and pathogenetic treatment of pathological processes and diseases; f) to know clinical interpretation of laboratory data and laboratory investigations of the organism systems.

Physiopathology involves general physiopathology and special physiopathology (teaching program for faculty of General Medicine, Stomatology, Farmacy, Public Health, III year of study) and clinical physiopathology (studied at the Faculty of General Medicine, IV year of study and in residency).

## Mission of syllabus (goal) in profesional development

Studing the functional and biochemical changes at molecular, cellular, tissular, organ and systemic levels during pathological processes and diseases; studing general laws of origin, onset, evolution and resolution of pathologic tipical processes and nosologic entities.

Language/languages of the course: romanian, english, russian.

• Beneficiaries: students of the III <sup>rd</sup>year, Faculaty of Medicine no. 2

## II. MANAGEMENT OF THE DISCIPLINE

(autumn semester)			
Code of the course		F.05.O.043	
Name of the course		PHYSIOPATHOLOGY	
Responsible for course		Cobeț Valeriu, HanganCorneliu	
Year	III	Semester/Semesters	5
Total number of hours, inclusive:120		120	
Course	30	Practical work/ laboratory	25
Seminars	20	Individual work	45
Form of evaluation	E	Number of credits	4



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		(spring semester)	
Code of the course		F.06.O.051	
Name of the course		PHYSIOPATHOLOGY	
Responsable forthecourse		CobețValeriu, HanganCorneliu	
Year	III	Semester/Semesters	5
Total number of hours, inclusive:			120
Course	30	Practical work/ laboratory	25
Seminars	20	Individual work	45
Form of evaluation	E	Number of credits	4

## I. TRAINING AIMS WITHIN THE DISCIPLINE At the end of the course the student will be able to:

# • at the level of knowledge and understanding:

- know the laws of origin, occurence, development and end of typical pathological processes localized in different organs and systems;
- know the structural changes, biochemical imbalances and functional disorders at molecular, cellular, tissue, organ and organs system level in typical pathological processes and diseases;
- know the pathogenetic therapy principles of pathological processes and diseases;
- know behavior rules to deal with pathophysiological experiment methodology and interpretation of information obtained in the experiment;
- define the theoretical basis of general, special and clinical pathophysiology;
- know general definition accepted in pathology.

# • at the application level:

- to be able to interpret information obtained during pathophysiological experiments and to be able to export these data in clinical settings;
- to be able to interpret clinical, morphologic, biochemical and functional changes in the clinical cases studied during laboratory classes;
- to be able to answer and to argue the correct answers (and incorrect answers) to questions in tests at pathophysiology;
- to be able to generate general conclusions and to differentiate nosologies on the basis of complex investigations:
  - 1. general analyses of transudate and inflammatory exudate;
  - 2. biochemical investigation of the blood protein level, glucose level, lipid spectrum



- in systemic metabolic changes;
- 3. immune and allergic status in allergic disorders and immune deficiencies;
- 4. hydric and electrolytic balance in water dysbalance and electrolytic dysbalance;
- 5. acid-base balance in acidosis and alalosis of different origin;
- 6. oxigen balance in the body in hypoxia of diferent origin;
- at integrative level
- to be able to use knowledge obtained at other previous courses (anatomy, histology, physiology, biochemistry) during study of pathophysiology;
- to be able to integrate knowledge obtained at concomitant courses (physiopathology, morphopathology) in one single nosological entity;
- to be able to integrate knowledge obtained at pathophysiology with nosological entities studied at clinical courses;
- to be able to integrate knowledge obtained at pathophysiology with information from pharmacology in the context of pharmacologic pathogenic correction of pathologic processes;
- to be able to integrate knowledge obtained at pathophysiology with current problems of theoretic and practical medicine.

# II. PROVISIONAL TERMS AND CONDITIONS

- Student of the third year requires the following:
- knowledge of the language of instruction;
- confirmed competences in high school level (biology, chemistry, physics);
- confirmed competences in science at the level of academic II year (anatomy, biology
- molecular, histology, physiology, biochemistry);

• digital skills (internet usage, processing of document, tables, electronic presentations and the use of graphics programs);

- ability to communicate and team work;
- personal qualities tolerance, compassion, autonomy.

# **III. THEMES AND ESTIMATE DISTRIBUTION OF HOURS**

### (autumn semester)

Lectures, practical hours/ laboratory hours/seminars and self-training

		Nun	nber of ho	ours
No	TUEME		Practical	Individ
	THEME	Lectures	work/Se	ual
			minars	work
	Theoretic and general nosology. Object, aim and goals of			
1.	pathophysiology, methods of research. General etiology.	2	3	4
	General pathogeny. General sanogenesis.			
2.	2. Cellular typical processes. Cell injury.		3	4
3.	3. Cellular tipical processes. Dystrophy. Apoptosis. Necrosis.		3	4
4.	Typical tissular pathologic processes. Dedifferentiation.	2	3	4



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30

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45

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4

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4

4

5

5

3

45

	No THEME		Number of hours		
No			Practical	Individ	
			work/Se	ual	
			minars	work	
	Physiologic and pathologic regeneration. Hyperplasia and				
	metaplasia. Physiologic and pathologic hypertrophy.				
	Physiologic and pathologic atrophy.				
5.	Microcirculatory disorders. Arterial hyperaemia. Embolism.	2	3	3	
6.	Venous hyperaemia. Edema. Thrombosis				
7.	Inflammation	4	6	4	
	Hypersensitivity disorders. Allergy. Allergic reaction type I, II,				
8.	III, IV. Autoimmune reactions. Non-specific hypersensitivity.	4	6	4	
	Humoral, cellular, and combined immunodeficiency.				
0	Pathophysiology of carbohydrates, lipid and protein metabolic		(	4	
9.	changes		0	4	
10.	. Pathophysiology of water and electrolytic imbalances		3	4	
11.	Pathophysiology of acid-basic imbalances		3	4	
10	Pathophysiology of thermic dyshomeostasis. Hypotermia		2	2	
12.	Hyperthermia. Fever.		3	3	
	Total (120 hours, 4 credits)		45	45	
	spring semester				
		Nun	nber of ho	ours	
Nb.	THEME		Practical	Individ	
		Lectures	work/Se	ual	
			minars	work	
1.	Pathophysiology of endocrine system	4	6	5	

# IV. PRACTICAL SKILLS ACHIEVED AT THE END OF THE COURSE

Practical skills are (autumn semester):

Pathophysiology of red blood cells

Pathophysiology of white blood cells

Pathophysiology of respiratory system

Pathophysiology of digestive system

Pathophysiology of the liver

Pathophysiology of kidneys

Pathophysiology of cardiovascular system

Pathophysiology of central nervous system

Total (120 hours, 4 credits)

2.

3.

4.

5.

6.

7.

8.

9.

• appreciate erythrocyte sedimentation rate in inflammatory processes;



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- Appreciate acute phase proteins in the blood;
- Appreciate pro- and ant-inflammatory cytokines;
- Appreciate pH, HCO3 in the blood;
- Appreciate humoral immune status, immunoglobulin spectrum;
- Appreciate lymphocytes population;
- Appreciate hematocrit in different form of hydric imbalance;
- Appreciation of lipid spectrum in the blood;
- Appreciate glycated hemoglobin.

Practical skills (spring semester):

- Appreciate peripheral blood analysis (hemogram, leucogram);
- To interpret ECG;
- Appreciate hormonal profile.

# V. OBJECTIVES AND CONTENT UNITS

	autumn semester
Objectives	Content
Theme (chapter 1) N	losology. Etiology. Pathogeny.
to define	the main definition of nosology: pathology, pathological physiology, pathophysiological experiment, nosology, disease, pathological process, etiology, cause, condition, pathogen, lesion, reactivity, adaptive reaction, compensatory, protective, reparative, pathogenetic factor, pathogenetic chain, main pathogenetic link, vicious circle, sanogenesis.
to know	Classification and characteristics of causes and conditions, classification and characteristics of physiological reactions. Mechanisms of generalization and localization of pathological processes.
to demonstrate	The role of experiment in studying of pathological processes.
to apply	the notions of nosology in the interpretation of pathophysiological experiments and in medical practice
to integrate	Observations from the researched experiments; (hypervolemia, algic shock, hyperadrenalineemia, hypoxia) in the form of a pathogenetic chain of pathological processes with the interpretation of observed phenomena.
Theme (chapter) 2. T	ypical cellular pathologic processes. Cell injury.
to define	cellular lesion, cellular dysmetabolism



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to know	classification, mechanism of action and primary effects of mechanical, physical, chemical, biological, osmotic, oxidative, enzymatic, immunopathological factors, hypoxia, hydrogen ions, energy depletion. To know the subsequent effects of cellular lesions until resolution of the process.	
to demonstrate	the complete pathogenetic chain of the cell death at the action of mechanical, physical, chemical, biological, osmotic, oxidative, enzymatic, immunopathic factors, and hypoxia, hydrogen ions, energy depletion. To demonstrate the pathogenesis of cellular dysmetabolism in discirculatory disorders and general dysmetabolism: hyperglycemia, hypoglycaemia, starvation, alimentary, transport and retention hyperlipidemia. To integrate local dysmetabolic processes at the cellular level with general disorders in the body in both directions: the impact of cellular dysmetabolism on the organism and the impact of general metabolic disorders on the cell.	
to apply	knowledge of the pathogenesis of cellular metabolic changes in the explanation of metabolic diseases: liver lipid dystrophy, obesity, atheromatosis.	
to integrate	Relation between local pathologic phenomena in necrosis and apoptosis with general changes in the body. Relation between necrosis and start of inflammatory reaction and other systemic changes (enzymemia, hyperkalemia, acute-phase response syndrome, fever, stress).	
Theme (chapter) 3.	Typical cellular pathologic processes. Apoptosis. Necrosis.	
to define	the concepts of apoptosis, intrinsic and extrinsic, positive and negative apoptogenic factors, degenerative and proliferative diseases. The notions of necrosis, necrobiosis, physiological and pathological death, thanatogenic factors	
to know	the intrinsic and extrinsic, positive and negative apoptogenic factors, the mechanism of initiation, execution and resolution of apoptosis, the biochemical processes in apoptosis, the structural manifestations of apoptosis. Periods of necrosis: cellular disease, cellular agonism, cell death, post-mortem period. Biochemical, functional and structural changes in the cell during dying.	
to demonstrate	the complete pathogenetic chain of apoptosis to the action of extrinsic factors (TNF alpha) and intrinsic (cytochrome C). The pathogenetic chain of cell death at the action of various pathogens factors. To apply the informations about necrobiosis pathogenesis in the amplification of sanogenetic processes and cellular resuscitation.	
to apply	Information of apoptosis in explaining the pathogenesis of proliferative (tumor) and degenerative diseases (Parkinson disease). Local processes in apoptosis and necrosis with general disorders in the organism.	
to integrate	Cell death with local (inflammation) and general processes in the body (enzymemia, hyperkalemia, acute phase reaction, fever, stress).	
Theme (chapter) 4. Tissular pathologic processes.		
to define	Definitions of cellular dedifferentiation, to tipotential, multipotential, pluripotential, unipotential cells, differentiation and cloning. Physiological and pathological regeneration. Homeostatic, adaptive, reparative, protective, compensatory regeneration. Pathological atrophy. Labile, stable, progressive sclerosis. Collagenogenesis. Collagenolysis. Functional, adaptive, reparative, protective, compensatory hypertrophy. Hypofunctional, involutive, senile, endocrine, post-hypertrophic physiological atrophy.	



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	Pathological atrophy. Scierosis, collagenogenesis, collagenolysis.
to know	causes, pathogenesis, and role in pathology of cellular dedifferentiation. Pathogenesis of
	physiological regeneration: homeostatic, adaptive, reparative, protective, compensatory.
	Mechanisms of pathological regeneration. Pathogenesis of functional, adaptive,
	reparative, protective, compensatory hypertrophy. Pathogenesis of physiological atrophy:
	hypofunctional, involutive, senile, endocrine, posthythrotrophic. Pathogenesis of
	pathological atrophy. Causes, pathogenesis, consequences of sclerosis. Principles of
	pathogenic correction of the sclerosant process.
to demonstrate	pathogenesis of cancer due to cellular dedifferentiation. The pathogenetic chain of
	homeostatic physiological regeneration (e.g. regeneration of the intestinal
	epithelium)adaptive (e.g. erythroblastic series regeneration in altitude hypoxia in healthy
	persons), compensatory (e.g. regeneration of the erythroblastic series in circulatory
	hypoxia in the patient with cardiac defect), reparative (e.g. regeneration of the epidermis
	to mechanical injuries), protective (e.g., proliferation of mesenchymal elements at tissue
	inoculation of the infect). The pathogenetic chain of functional hypertrophy (hypertrophy
	of skeletal muscle at exercises), adaptive (hypertrophy of the heart at altitude),
	compensatory (hypertrophy of the heart in hypertension). The pathogenetic chain of
	hypotunctional physiological atrophy, involutive, senile, endocrine, posthypertrophic.
	The pathogenetic chain of pathological atrophy in cellular lesions. The pathogenetic
(	chain of scierosis in cellular lesions.
to apply	aws of tissue pathological processes in the explanation of disease pathogens, tumoral,
	insufficiency multiple atrophy of organs in senility. To differentiate physiological and
	pathological regeneration, physiological and pathological hypertrophy, physiological and
	pathological atrophy
to integrate	processes of regeneration hypertrophy and atrophy based on common cellular processes
to integrate	To integrate the cellular pathological processes into the structure of the diseases.
Theme (chapter) 5	. Microcirculatory disorders. Arterial hyperemia. Ischemia. Embolism. Venous
hyperemie Edeme	
nyperenna. Euema.	I nrombosis.
to define	Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional
to define	Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia.
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to define to know to know to demonstrate to apply to integrate	<ul> <li>Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, wth amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus.</li> <li>etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia.Obstructive, obliterant, compressive venous hyperemia. Ischemia, red and white infarction; etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema; of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, and consequences of trombogenesis in arteries and veins.</li> <li>the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema.</li> <li>the theoretical information in the pathogenic correction of microcirculatory disturbances.</li> <li>the theoretical information about local microcirculatory disturbances in pathogenesis of</li> </ul>
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to demonstrate to apply to integrate	Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, wth amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus. etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia.Obstructive, obliterant, compressive venous hyperemia. Ischemia, red and white infarction;etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema; of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of trombogenesis in arteries and veins. the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema. the theoretical information in the pathogenic correction of microcirculatory disturbances. the theoretical information about local microcirculatory disturbances in pathogenesis of the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension.
to demonstrate to apply to integrate <b>Theme (chapter) 6. I</b>	Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, wth amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus. etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia.Obstructive, obliterant, compressive venous hyperemia. Ischemia, red and white infarction; etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema; of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of trombogenesis in arteries and veins. the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema. the theoretical information in the pathogenic correction of microcirculatory disturbances. the theoretical information about local microcirculatory disturbances in pathogenesis of the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension. <b>nflammation.</b>
to demonstrate to demonstrate <u>to apply</u> to integrate <b>Theme (chapter) 6. I</b>	Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, wth amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus. etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia.Obstructive, obliterant, compressive venous hyperemia. Ischemia, red and white infarction; etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema; of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of trombogenesis in arteries and veins. the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema. the theoretical information in the pathogenic correction of microcirculatory disturbances. the theoretical information about local microcirculatory disturbances in pathogenesis of the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension.
to define to know to know to demonstrate to apply to integrate <b>Theme (chapter) 6. I</b> to define	Infomosis.Definitions: neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterant, compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, wth amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus.etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia.Obstructive, obliterant, compressive venous hyperemia. Ischemia, red and white infarction; etiology, pathogenesis, manifestations and consequences of congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema; of air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus; etiology, pathogenesis, manifestations and consequences of trombogenesis in arteries and veins.the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema.the theoretical information in the pathogenic correction of microcirculatory disturbances. the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension.nflammation.Definitionsinflammation, alteration, pattern of lesional and pathogenic molecules, cell-and plasma-derived mediators. inflammatory arterial and venous hyperemia



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	exudation-serous, fibrinous, purulent, hemorrhagic, putrid; leukocyte emigration,
	phagocytosis, inflammatory proliferation; acute phase reaction, fever and leukocytosis.
to know	causes of the inflammation, pathogenesis of the alteration caused by the different flogogenic factors, the sources of the cell-and plasma derived mediators, the effects of mediators, pathogenesis of vascular reactions in the inflammatory focus, pathogenesis of
	the exudation and the composition of various forms of exudate, the mechanisms of leukocytes migration and the role of leukocytes in the inflammatory focus: sources
	mechanisms and role of proliferation in the inflammatory site; mechanisms and variants
	of post-inflammatory regeneration. Systemic disorders in the body during local
	inflammation: acute phase reaction, fever, leukocytosis. To know the pathogenesis, manifestations and consequences of the systemic inflammatory reaction syndrome.
to demonstrate	the pathogenetic chain of different forms of inflammation: alterative, exudative, proliferative. To demonstrate the pathogenetic chain of the systemic inflammatory reaction.
to apply	information about the composition of the exudate for differentiation of the inflammation
	variants. To interpret general disorders in the body for the diagnosis and monitoring of the information of the information of the rather and constrained of information to
	modulate the inflammatory process and use anti-inflammatory preparations
to integrate	information about the etiology, pathogenesis and manifestations of inflammation in the
	pathogenesis and evolution of inflammatory diseases.
Theme (chapter) 7.	Hypersensitivity disorders. Allergy. Allergic reactions type I, II, III, IV. Autoimmune
disorders. Humoral,	, cellular and combined immunodeficiency.
to define	Definition of hypersensitivity disorders, immediate type allergic reactions: immediate hypersensitivity, antibody mediated, immune complex mediated, T-cell mediated; active and passive sensitisation; immunological pathochemical and pathochemical phases
	of allergic reactions: anaphylactic shock hypersensitivity unspecific hypersensitivity
	autoimmunity, autoantigens, autoantibody, humoral, cellular and mixed type of immunodeficiency.
to know	the etiology of hypersensitivity disorders and the classification of antigens, pathogenesis of the immunological phase with antibody synthesis or sensitization of lymphocytes, pathogenesis of pathochemical phase, sources of cell- and plasma-derived mediators, primary mediators and their biological effects; pathogenesis of vascular reactions, smooth muscles cells, mesenchymal structures, CNS and endocrine glands, pathogenesis of hyposensitization. To know the etiology, pathogenesis, manifestations and consequences of humoral, cellular and mixed immunodeficiencies. To know alterating mechanisms in hypersensitivity disorders. To know the pathogenesis of stimulant allergic reactions type. To know the pathogenesis of pseudoallergic reactions by non-specific degranulation of mast cells, complement defects, disorders of cyclooxygenase and lipoxygenase pathways. To know the pathogenesis of autoimmunity - transforming self antigens.
to demonstrate	the complete pathogenetic chain from inoculating the allergen to structural damage in all types of allergic reactions.
to apply	the theoretical information about pathogenesis of allergic reactions to formulate the principles of pathogenetic therapy. To apply the theoretical information about pathogenesis of allergic reactions for diagnosis in vitro and in vivo. To apply the theoretical knowledge for diagnosing and formulating the principles of pathogenetic correction of immunodeficiencies.
to integrate	the theoretical information about pathogenesis of allergic reactions for involvement in the pathogenesis of allergic, autoallergic and pseudo-allergic diseases.



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Theme (chapter) 8. General carbohydrate, lipid and protein metabolic changes.		
to define	Definitions: metabolic abnormalities of carbohydrates, lipids, proteins. Hypo- and hyperglycemic factors. Alimentary, transport hyperglycemia in hypoinsulinism. Hypoglycaemia in starvation, in hyperinsulinism. Ketonemia. Hyperglycemic hyperosmolar coma. Ketoacidotic coma. Hypoglycemic coma. Galactosemia. Congenital and acquired dyslipidemia. Alimentary, transport, retention hyperlipidemia. Hyperlipoproteinaemia. Hyperlipidemia. Hypercholesterolemia. Atheroma. Hyperproteinemia. Dysproteinemia.	
to know	causes, pathogenesis, manifestations and consequences of hyperglycemia, hypoglycemia, ketonemia, galactosemia. Causes, pathogenesis, manifestations and consequences of transport, retention, alimentary dyslipidemias. Causes, pathogenesis, manifestations and consequences of hypercholesterolemia. Pathogenesis of atheromatosis. Causes, pathogenesis, manifestations and consequences of hyperproteinemia, of dysproteinemia.	
to demonstrate	the pathogenetic chain of hyperglycemia (alimentary, transport, hypoinsulinemia, hypercorticism, hyperkalaemia, hyperthyroidism). The pathogenetic chain of hyperlipidemias (congenital, alimentary, transport, retention). The pathogenetic chain of hypoproteinemias (in starvation, in diabetes, hyperacortism, hyperthyroidism).	
to apply	theoretical information in the interpretation of clinical and laboratory manifestations in diseases: type I diabetes mellitus, insulin resistance, metabolic syndrome, hyperosmolar hyperglycaemic coma, ketoacidotic coma, hypoglycaemic coma.	
to integrate	biochemical, nervous, endocrine and functional disturbances in diseases: type I diabetes, insulin resistance, metabolic syndrome, hyperosmolar hyperglycaemic coma, ketoacidosis coma, hypoglycaemic coma.	
Theme (chapter) 9. W Theme (chapter) 10. A	/ater and electrolytic imbalances. Acid-base imbalance.	
Theme (chapter) 11.T	'hermic dyshomeostasis.	
to define	Definitions: iso-, hypo- and hyperosmolar overhydration. Iso-hypo- and hyperosmolar dehydration. Hyper- and hyponatremia. Hyper- and hypokalaemia. Hyper- and hypocalcemia. Hyper- and hypochloremia. Hyper- and hypophosphatemia. Acidosis (respiratory, metabolic, excretory, exogenous). Alkalosis (respiratory, metabolic, excretory, exogenous). Alkalosis (respiratory, metabolic, excretory, exogenous). Respiratory, circulatory, hemic, tissue hypoxia. Hyperoxia. Hypothermia. Hyperthermia. Fever. Primary and secondary pyrogens, exogenous and endogenous factors. Stages of fever: increase, maintenance and decrease of temperature. Subfebrile, febrile, high and hyperpiretic fever. Patterns of the fever: continuous, intermittent, remitting, recurrent, hectic, atypical. Crisis. Lysis.	
to know	causes, pathogenesis, manifestations and consequences of fluid dyshomeostasis. Iso-, hypo- and hyperosmolar overhydration, iso-hypo- and hyperosmolar dehydration). Causes, pathogenesis, manifestations and consequences. Causes, pathogenesis, manifestations and consequences of dymineralosis. Hyper- and hyponatremia. Hyper- and hypokalaemia. Hyper- and hypocalcemia. Hyper- and hypochloraemia. Hyper- and hypochloraemia. Hyper- and hypophosphataemia. Causes, pathogenesis, manifestations and consequences of acid-base imbalance. Respiratory, metabolic excretory, exogenous acidosis; respiratory, metabolic, exogenous, exogenous alkalosis). Causes, pathogenesis, manifestations and consequences of respiratory, circulatory, hemic, tissue hypoxia. Causes, pathogenesis, manifestations and consequences of hyperoxia. Causes, pathogenesis, compensatory reactions, manifestations and consequences of hyperthermia. Etiology and pathogenesis of fever. Pathogenesis and stages of fever evolution: increase, maintance, decrease of temperature. Metabolic and functional disorders. Biological rmportance. Pathogenetic correction.	



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to demonstrate	the pathogenetic chain of different forms of dehydration, iso-, hypo- and hyperosmolar of over- and dehydration); the pathogenetic chain of various forms of electrolyte imbalances (Na, K, Ca, Cl, PO <sub>4</sub> ); the pathogenetic chain of hyperthermia and hypothermia; the pathogenetic chain of various forms of hypoxia (respiratory, circulatory, hemic, tissue); the pathogenetic chain of hyperoxia; the pathogenetic chain of hypo- and hyperthermia; pathogenic chain of fever: increase, maintenance and decrease of temperature stages).
to apply	theoretical knowledge in the interpretation of haematological, biochemical parameters, clinical manifestations in the dehydration, electrolyte imbalances, hypoxia, acid-base imbalance, dystermia.
to integrate	theoretical information in the diseases (dehydration, overhydration, hyperkalaemia in massive hemolysis, hypoxia in blood, cardiovascular and respiratory apparatus diseases, cetodiabetic acidosis, asphyxia, diarrhea, alveolar hypophenelation, vomiting), dehydration in hyperthermia, the febrile component in the pathogenesis of infectious diseases.

# Spring semester

Objectives	Content
Theme (chapter) 1. Pa	athophysiology of endocrine system
To define	Hyper- and hyposecretion of GH-releasing hormone-somatotropin-somatomedins, corticotropin-releasing hormone-corticotrophin, thyroid-stimulating hormone-thyrotropin, gonadotropin-releasing hormone-gonadotropins-luteinizing hormone and follicle-stimulating hormone, prolactin-lactotropin. Hyper- and hypocortisolism. Hyper- and hypothyroidism. Male and female hyper- and hypogonadism. Hypoinsulinism. Type I diabetes mellitus. Hyperosmolar hyperglycemic coma, ketoacidotic coma. Microvascular complication. Macrovascular complication. Hypoglycemic coma. Insulin resistance. Type II diabetes mellitus.
To know	organization and functional principles of hypothalamus-pituitary peripheral gland axis. Etiology, pathogenesis and manifestations of hypothalamic neurosecretory disorders. Etiology, pathogenesis and manifestations of disorders of pituitary secretion: TSH, ACTH, GH, FSH, LH, prolactin. Etiology, pathogenesis and manifestations of peripheral glands disorders: adrenocortical, thyroid gland, gonads, endocrine pancreas. The organo- genetic and metabolic effects of the growth hormone and somatomedines, glucocorticosteroids, mineralocorticosteroids, thyroid hormones, sexual hormones, insulin and glucagon. Manifestations of insufficiency and hypersecretion of growth hormone and somatomedins, glucocorticosteroids, mineralocorticosteroids, thyroid gland hormones, sexual hormones, insulin and glucagon.
To demonstrate	the pathogenetic chain of primary endocrine disorders, secondary and tertiary for adrenal glands cortex, thyroid gland, gonads.
To apply	the theoretical knowledge to explain biochemical and clinical disorders in clinical forms of failure and hypersecretion of growth hormones, glucocorticosteroids, mineralocorticosteroids, thyroid hormones, sexual hormones, insulin and glucagon.



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	theoretical knowledge in the pathogenesis and manifestations of nosological entities:			
To integrate	gigantism and dwarfism, acromegaly, primary and secondary hypercortisolism			
	(Cushing's disease and syndrome), hypocortisolism (Addison's disease), hyperthyroidism (Graves's disease), hypothyroidism (endemic goiter, mixedem), primary hypersecretion			
	(Graves's disease), hypothyroidism (endemic goiter, mixedem), primary hypersecretion			
	of mineralocorticoids (Conn's disease), type I and type II diabetes mellitus, insulin resistance. Hyper-and hyposecretion of GH-releasing hormone-somatotropin-			
	resistance. Hyper-and hyposecretion of GH-releasing hormone-somatotropin-			
	somatomedine, corticotropin-releasing hormone- corticotrophin.			
Theme (chapter) 2. Pa	athophysiology of red blood system			
To define	the concepts of primary and secondary, absolute and relative polycythemia;			
	hyporegenerative, acute and chronic blood loss anemias, iron-deficiency and			
	megaloblastic anemias, congenital and acquired haemolytic anemias.			
To know	etiology, pathogenesis, manifestations and peripheral blood smear of primary and			
	secondary, absolute and relative erythrocytosis; hyporegenerative, acute and chronic			
	blood loss anemias; iron-deficiency and megaloblastic anemias, congenital and acquired			
	haemolytic anemias. Normal and pathological haematopoiesis. To know the mechanisms			
	of physiological and intracellular and intravascular pathological hemolysis; t			
	biochemistry of normal bilirubin metabolism and in haemolytic anemias.			
To demonstrate	peripheral blood smear of primary and secondary, absolute and relative polycythemia;			
	hyporegenerative, acute and chronic blood loss anemias; iron-deficiency and			
	megaloblastic anemias, congenital and acquired haemolytic anemias; absolute and			
	relative leukocytosis, neutrophilia, eosinophilia, lymphocytosis and monocytosis; proliferative disorders in the hematopoietic organs; hemoblastosis acute and chronic			
	proliferative disorders in the hematopoietic organs: hemoblastosis, acute and chronic			
	leukaemia, lymphomas.			
To integrate	theoretical knowledge in the pathogenesis of haematological diseases:: acute and chronic			
	naemorrhage, iron-deficiency, B12 deficiency and folate deficiency anaemia,			
autoimmune naemolytic anemia, intoxication with naemolytic toxins;				
Theme (chapter) 5. F	athophysiology of white blood system			
To define	The notions about absolute and relative neutrophilia, eosinophilia, lymphocytosis			
	and monocytosis. Definitions of absolute and relative leukocytopenia, neutropenia,			
	eusinopenia, agranulocytosis, lymphocytopenia. Definitions of hemoblastosis, acute			
	and chronic leukaemia, lymphocytopenia. Definitions of hemobilastosis, acute			
	smears.			
To know	Etiology, pathogenesis, manifestations and peripheral blood smear of absolute and			
	relative leukocytosis, neutrophilia, eosinophilia, lymphocytosis and monocytosis.			
	Etiology, pathogenesis, manifestations and peripheral blood smear of absolute and			
	relative leucopoenia, neutropenia, eusinopenia, agranulocytosis, lymphocytopenia.			
	Etiology, pathogenesis, manifestations and peripheral blood smear of proliferative			
	disorders in hematopoietic organs: hemoblastosis, acute and chronic leukemias,			
	lymphomas.			
To James 4 4	I Hamaganan of abachita and milating independently in material 2015 and 2015 1 - 1 - 4			
10 demonstrate	Hemogram of absolute and relative leukocytosis, neutrophilia, eosinophilia, lymphocytes			
	and monocytosis; of absolute and relative leukopenia, neutropenia, eosinopenia,			
To omnity	agranulocytosis, lymphocytopenia; of acute and chronic leukemias, lymphomas.			
	The theoretical knowledge in the interpretation of the peripheral blood smear and clinical manifestations in the pathology of white blood			
To integrate	The theoretical knowledge in the nethogeneois of inflammatory and neurositic discovery			
10 milegrate	The uncorrectal knowledge in the pathogenesis of inflammatory and parasitic diseases,			
	acute and chronic lymphocytic leukemia			



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Theme (chapter) 4. Pathophysiology of the cardiovascular system				
To define	The coronary and non-coronary, metabolic, hematogenic cardiogenic circulatory failure. Vasogenic circulatory insufficiency. Primary and secondary arterial hypertension. Chronic hypotension. Acute arterial hypotension: collapse, shock. Tachycardia, bradycardia, sinusal arrhythmia. Extrasystole, atrial and ventricular flutter, atrial and ventricular fibrillation. The incomplete and complete atrioventricular block.			
10 know	The etiology, pathogenesis, compensatory reactions and manifestations of coronary and non-coronary, metabolic, hematogenic heart circulatory insufficiency. The pathogenesis of emergent and delayed compensatory reactions, pathogenesis of myocardial hypertrophy. The aetiology, pathogenesis, compensatory reactions and manifestations, compensatory reactions, the consequences of vasogenic circulatory insufficiency. Arterial hypertension. Chronic arterial hypotension. Acute arterial hypotension: collapse, shock. To know the etiology, pathogenesis, manifestations, compensatory reactions, consequences, electrocardiographic picture of heart arrhythmias: tachycardia, bradycardia, sinusal arrhythmia, extrasystole, atrial and ventricular flutter, atrial and ventricular fibrillation, incomplete and complete atrioventricular block.			
To demonstrate	The pathogenetic chain of compensatory reactions and hemo circulatory disorders in myocardial, endocardial, pericardial diseases. To demonstrate the pathogenetic chain of compensatory reactions and hemocirculatory disorders in vascular disorders -primary and secondary hypertension. To demonstrate the pathogenetic chain of compensatory reactions and hemo circulatory disorders in cardiac arrhythmia: tachycardia, bradycardia, sinusal arrhythmia, extrasystole, atrial and ventricular flutter, atrial and ventricular fibrillation, incomplete and complete atrioventricular block.			
To apply	The theoretical knowledge in the interpretation of clinical manifestations and ECG in cardiovascular pathology.			
To integrate	The theoretical knowledge in the nosological entities, as: myocarditis, valvulopathies, pericarditis, coronary insufficiency, atrial fibrillation, atrioventricular block.			
Theme (chapter) 5. P	Theme (chapter) 5. Physiopathology of external breathing			
To define	The notions of the external breathing pathophysiology. Restrictive ventilation disorders. Pulmonary edema. Pneumosclerosis. Pulmonary emphysema. Acute respiratory distress in adults and newborns. Obstructive ventilatory disorders. Obstruction of the upper respiratory airways. Asphyxia. Asthmatic syndrome. Disorders of alveolo-capillary gas diffusion. Disorders of the pulmonary perfusion. Disruptions of gas transport: oxygen and carbon dioxide.			
To know	The etiology, pathogenesis, manifestations and consequences of external breathing disorders in extrapulmonary restrictive processes: in diseases of the respiratory center and the respiratory reflex arc, chest skeleton, respiratory muscles, pleura. The etiology, pathogenesis, manifestations and consequences of external breathing disorders in intrapulmonary restrictive processes: pulmonary emphysema, pulmonary edema, pneumosclerosis, atherosclerosis, respiratory distress in newborns and adults. The etiology, pathogenesis, manifestations and consequences of external breathing disorders in obstructive processes: upper airways stenosis, asthmatic syndrome. The etiology, pathogenesis, manifestations and consequences of alveolo-capillary diffusion disturbances. The etiology, pathogenesis, manifestations and consequences of pulmonary perfusion disorders: pre- and post-capillary pulmonary hypertension, disorder of the ventilation-perfusion rate. The etiology, pathogenesis, manifestations and consequences of oxygen and carbon dioxide transport disorders: hypoxia and hypercapnia.			
To demonstrate	The pathogenetic chain of restrictive and obstructive external respiratory disturbances, disturbances of gas diffusion and transport.			
To apply	The theoretical knowledge in interpretation of clinical manifestations and functional disorders in various forms of external breathing disorders.			



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To integrate Theme (chapter) 6. Pa	The theoretical knowledge in the pathogenesis of nosological entities: respiratory paralysis, diaphragm paralysis, myasthenia gravis, pleurisy, pneumothorax, cardiac asthma, noncardiogenic pulmonary edema, alpha-antitrypsin insufficiency, pneumosclerosis, chronic obstructive pulmonary disease, bronchial asthma, pulmonary shock, pulmonary hypertension. Disruptions of gas transport: oxygen and carbon dioxide.	
To define	The notions as: hypo-and hypersalivation, gastric hypoacidity and hyperacidity, chemostasis in the stomach, dumping syndrome, ulcerogenesis: the aggressive and protective factors of the stomach. Pancreatic insufficiency. Pancreatic Maldigestion. Acholia. Intestinal Maldigestion. Intestinal malabsorption. Constipation. Diarrhea. Gastrointestinal poisoning	
To know	The etiology, pathogenesis, manifestations and consequences of salivation disorders: hypo-and hypersalivation. The etiology, pathogenesis, manifestations and consequences of disorders of secretion, motility and evacuation of the alimentary bolus in the stomach: gastric hyperacidity and hypoacidity. Chymostasis. Dumping syndrome. The etiology, pathogenesis, manifestations and consequences of gastric and duodenal ulcerogenesis. The etiology, pathogenesis, manifestations and consequences of pancreatic secretion disorders. Pancreatic Maldigestion. The etiology, pathogenesis, manifestations and consequences of bile secretion disorders: acholia. The etiology, pathogenesis, manifestations and consequences of intestinal digestive disorders: maldigestion, malabsorption, malnutrition. The etiology, pathogenesis, manifestations and consequences of bowel disorders: constipation, diarrhea, gastrointestinal intoxication.	
To demonstrate	The pathogenetic chain of maldigestion of carbohydrates, lipids and proteins throughout the digestive tract: the oral cavity, the stomach, the small intestine. The pathogenetic chain of malabsorption and malnutrition of carbohydrates, lipids and proteins. The pathogenetic chain of pancreatic maldigestion and in the absence of the bile.	
To apply	The theoretical knowledge in interpretation of clinical manifestations and laboratory investigations (gastric juice, duodenal juice, the coprology exam) in digestive diseases.	
To integrate	The theoretical knowledge in the digestive system diseases: hypertrophic and atrophic gastritis with hyperacidity and hypoacidity, stomach and duodenum ulcer, chronic pancreatitis, acholia, enteritis, diarrhea of different pathogenesis, constipation.	
Theme (chapter) 7. Li	iver physiopathology	
To define	Liver pathophysiology. Hepatic failure. Causes. Pathogenesis. Events. Consequences. Metabolic disorders in hepatic failure. Jaundice: prerahepatic, parenchymatous, posthepatic. Etiology, pathogenesis, manifestations, consequences. Hyperbilirubinemia. Cholemia. Cholalemia. Acholia. Hepatic cirrhosis: etiology, pathogenesis, manifestations, consequences.	
To know	The etiology, pathogenesis, manifestations and consequences of liver failure. The disorders of protein, carbohydrate, lipid metabolism, and of bilirubin metabolism in hepatic failure. The aetiology, pathogenesis, manifestations and consequences of hepatic coma. The manifestations and consequences of digestive disturbances in liver failure. The etiology, pathogenesis, manifestations and consequences of liver cirrhosis. The etiology, pathogenesis, manifestations and consequences of prehapatic, parenchymatous and posthepatic jaundice.	
To demonstrate	The pathogenetic chain of metabolic disorders in hepatic failure. To demonstrate the pathogenetic chain of bilirubin metabolism disorders in various forms of jaundice.	



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To apply	The theoretical knowledge in interpretation of clinical manifestations and laboratory			
To integrate	The theoretical knowledge in henatic nosological entities: henatitis henatitis steatosis			
10 megiate	jaundice, hepatic failure			
Theme (chapter) 8. I	Pathophysiology of the kidneys			
To define	The disorders of glomerular filtration, reabsorption and tubular secretion. Pre-renal, renal			
	and postrenal renal insufficiency. Acute and chronic renal failure: etiology, pathogenesis,			
	manifestations, consequences. Nephritic and nephrotic syndrome. Oliguria, polyuria,			
	anuria, proteinuria, glucosuria, bilirubinuria, cilindria. Hypostenuria, hyperstenuria and			
	isostenuria.			
To know	The etiology, pathogenesis, manifestations and consequences of pre-renal, intrarenal and			
	subrenal disorders of glomerular filtration. The etiology, pathogenesis, manifestations			
	and consequences of canalicular reabsorption disorders of water, electrolytes, proteins,			
	amino acids. The etiology, pathogenesis, manifestations and consequences of uri evacuation disorders during nephron and urinary tract. The etiology, pathogen			
	evacuation disorders during nephron and urinary tract. The etiology, pathogene			
	manifestations and consequences of acute and chronic renal failure. The etiological			
	pathogenesis, manifestations and consequences of nephritic and nephrotic syndrome.			
To demonstrate	The pathogenetic chain of hydroelectrolytic acid-base disorders in renal failure			
To apply	The theoretical knowledge in interpretation of clinical manifestations and laboratory			
	investigations in kidney disorders.			
To integrate	The theoretical knowledge into the pathogenesis of nosological entities: nephritis.			
0	nephrotic syndrome, renal failure, nephrolithiasis.			
Theme (chapter) 9. Pathophysiology of CNS				
To define Hypoexcitability. Hyperexcitability. Causes. Inhibition of depolarity				
	hyperpolarization. synthesis, transaxonal transport, storage, release, recapture and			
	degradation of mediators. Sympathicotonia and parasympathicotonia			
T. I	The mechanisms and disorders of neuron excitation and inhibition (anonymous and			
10 Know	anzymes for the synthesis of acetylcholine, noradrenaline, donamine, serotonin, GARA);			
	mechanisms and disturbances of transavonal transport of mediators mechanisms and			
	disturbances of the storage and release of mediators, mechanisms and disorders of			
	recapture and degradation of mediators in the synaptic cleft postinaptic disorders			
	Pathophysiology of central nervous system. Disruption of neuron functions.			
	Hyperexcitability. Causes. Mechanisms. Manifestations. Consequences.			
	Hypoexcitability. Causes. Mechanisms. Manifestations. Consequences. Inhibition of			
	depolarization. Disorders of transsinaptic transmission. Disruptions in the synthesis,			
	transaxonal transport, storage, release, recapture and degradation of mediators.			
	Pathophysiology of the vegetative nervous system. Causes. The pathogenesis of			
	segmental and suprasegmental disorders. Manifestations of sympathicotonia and			
To Jan 4 4	parasympathicotonia.			
10 demonstrate	The chain of neurophysiologic processes in the excitation and inhibition of excitable			
	vegetative disturbances: the pathogenetic chain of supressegmental vegetative			
	vegetative disturbances; the pathogenetic chain of suprasegmental vegetative sympathetic disorders; spinal parasympathetic vegetative reflex are and the pathogenetic			
	sympatience disorders; spinal parasympathetic vegetative disorders; the hulbar parasympathetic			
	vegetative reflex arc and the pathogenetic chain of segmental parasympathetic vegetative			
	disorders; the pathogenetic chain of suprasegmental parasympathetic vegetative			
	disorders; the biochemical chain of synaptic transmission(synthesis, transport, storage.			
	release, post-synapse mechanisms, re-uptake and degradation of mediators; the			
	pathogenetic chain of trans-synaptic transmission disorders;			



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To apply	The theoretical knowledge to explain clinical manifestations of neural function disorders and trans -synaptic transmission; of the autonomic nervous system disorders, as: sympathicotonia, parasympathicotonia, sympathoplegia and parasympathoplegia.	
To integrate	The theoretical knowledge within the nosological entities, as: Parkinson, intoxication with neurotropic substances.	

### VI. PROFESSIONAL (SPECIFIC (SC)) AND TRANSVERSAL (TC) COMPETENCES AND STUDY OUTCOMES

#### ✓ Professional (specific) (SC) competences:

• CP1. Responsible execution of professional tasks with the application of the values and norms of professional ethics, as well as in accordance with the current legislation;

• CP2. Knowing the sciences about body's structure, physiological functions and behaviour of the human body in various physiological and pathological states, as well as the existing relationships between health, physical and social environment.

• CP3. The interdisciplinary integration of the doctor's activity in the team with the efficient use of all resources.

• CP4. Conducting scientific research in health and other branches of science.

#### ✓ Transversal competences (TC)

• TC1. Autonomy and responsibility in the activity

**Note. Study findings** (are deduced from the professional competencies and formative valences of the informational content of the discipline).

No.	Expected product	Achievement strategies	Evaluation criteria	Deadline
1.	Working with textbooks	Studuing the material from the recommended manuals. Summary the material in the form of postulates. Exposing the material in the form of improvised schemes. Marking the questions that require special consultation	The ability to reproduce the main notions and the content of the material; the ability to give the essence of material; Ability to expose the material in logical schemes; Ability to explain the material; Ability to answer control questions;	Year of study
2.	Working with the materials of theoretical course	Studying the material of theoretical course; Studying the presentations of theoretical course; Summary of material in the form of postulates;	Ability to supplement the manual material with the information form theoretical course; Ability to reproduce textually and to interpret presentations of the theoretical course;	Year of study
3.	Working with the compendium of practical lessons	Studying the planned experiments for demonstration at the practical lesson: the experiment methodology, the obtained results and their interpretation.	Ability to integrate experiments into structure of the theoretical theme; The integration of experimental data in studied pathological processes; The illustration of the topic with real material; Explanation of experimental results with theoretical information; Translocation of the experiment into medical practice;	Year of study

### VII. STUDENT'S SELF-TRENING



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4.	Working with the situational problem recommended for the theme	Studying and solving of situational problems	The ability to answer correctly to the questions of the problems; The ability to interpret the pathogenetic summary of clinical, paraclinical, and laboratory information; The ability to make conclusions; Ability to make decisions about diagnosis, therapy and prognosis;	Year of study
5.	Working with the pathophysiological explanatory dictionary	Studying the dictionary of physiopathological terms	The ability to reproduce and interpret the essence of definition and notion	Year of study
6.	Working with the collection of tests in pathophysiology	Studying and solving control tests of the subject; Self-control of material acquisition using the control questions	Monitoring the cognitive process by autocontrol	Year of study
7.	Working with online materials	Studying materials on-line from the Department SITE; Working with encyclopedic materials, dictionaries, scientific activities; Selection of the research theme, purpose, selection of materials, formulation of conclusions, bibliography.	Supplementing informations with recent materials	Year of study
8.	Preparation and support of papers, presentations	Selection of research topic, purpose, selection of materials, formulation of conclusions, bibliography.	Workload	Year of study

### VIII.METHODOLOGICAL SUGGESTIONS FOR TEACHING-LEARNING-EVALUATION

## • Teaching and learning methods used

Different methods and didactic procedures are used to teach the pathophysiology, oriented towards efficient learning and achieving objectives of the teaching process. In the theoretical course along with traditional methods (course exposition, interactive course, synthesis course), PowerPoint presentations are used. Tests, situations problems, demonstration of the film are used in practical work with the modeling of pathological processes in laboratory animals. Teaching materials (tables, micrographs, transparencies) are used for deeper material acquisition.

- Applied teaching strategies / technologies (specific to the discipline)
- In the process of teaching pathophysiology are used: (1) The real and virtual pathophysiological experiment; (2) Logical solving of situational problems

### • *Methods of assessment* (inclusive including the method of final mark calculation)

*Current:* frontal oral interrogation, situational problem solving, test-control - 3 concludings in the SIMU system for each semester of studies (5 and 6 separately).

## Final: exam-test in SIMU system in the 5th and 6th semesters separately.

**The final mark:** it will consist of the average mark of three intermediate concludings for each semester separately (coefficient 0.5), and the final test exam for each semester (coefficient 0.5). Annual average mark and marks for all stages of the final examination (assisted by computer, testing) - all will be



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expressed in numbers according to the grading scale (according to the table), and the final mark obtained will be expressed in numbers by two decimals, which will be entered in the notebook.

Method of mark rounding at different assessment stages			
Intermediate marks scale (annual average, marks from the examination stages)	National Assessment System	ECTSE quivalent	
1,00-3,00	2	F	
3,01-4,99	4	FX	
5,00	5		
5,01-5,50	5,5	E	
5,51-6,0	6		
6,01-6,50	6,5	D	
6,51-7,00	7	D	
7,01-7,50	7,5	C	
7,51-8,00	8	C	
8,01-8,50	8,5	D	
8,51-8,00	9	D	
9,01-9,50	9,5		
9,51-10,0	10	A	

The average annual mark and the marks of all stages of final examination (computer assisted, test, oral answer) - are expressed in numbers according to the grade scale (according to the table), and the final mark obtained is expressed in number with two decimals, which is transferred to student's record-book.

Absence on examination without good reason is recorded as "absent" and is equivalent to 0 (zero). The student has the right to have two re-examinations.

## IX. RECOMMENDED LITERATURE:

### A. Compulsory:

- 1. Robins & Cotran. Pathologic Basis of Diseases. Lippincot Williams & Wilkins, VIII<sup>th</sup> edition. 2014
- 2. Carol Mattson Porth. Pathophysiology. Concepts of Altered Health State. Lippincot Williams & Wilkins, 2010.
- 3. Color Atlas of Pathophysiology .S Silbernagl et al, Thieme 2000.
- 4. ACC Atlas of Pathophysiology. Lippincot Williams & Wilkins, 2009.
- 5. Essentials of Pathophysiology. Lippincot Williams & Wilkins. 2003
- 6. Stephen S. Mc Phee and Gary D. Hammer. Pathophysiology of Diseases: An introduction to Clinical Medicine, 2010

### B. Extra

a. Colev Veronica. Pathophysiology. Iasi, 2001.