FACULTY MEDICINE NO. 2

STUDY PROGRAM 0912.1 MEDICINE

CHAIR OF PATHOLOGY

APPROVED
at the meeting of the Commission for Quality Assurance and Evaluation of the Curriculum in Medicine
Minutes No. 5 of 17.02.25
Chairman Ph.D., associate professor
Andrei Pădure _____
(signature)

APPROVED
at the Council meeting of the Faculty Medicine II
Minutes No. 5 of 25.02.25
Dean of Faculty Ph.D., associate professor
Mircea Bețiu _____
(signature)

APPROVED
approved at the meeting of the chair of Pathology
Minutes No. 6 of 01.03.2024
Head of chair PhD, associate professor
Eugen Melnic _____
(signature)

SYLLABUS

DISCIPLINE **PATHOPHYSIOLOGY**

Integrated studies / Cycle I, License

Tipe of course: **Compulsory**

Syllabus elaborated by authors:
Lutan Vasile, PhD, associate professor
Cobeț Valeriu, PhD associate professor

Chișinău, 2024

I. INTRODUCTION

- General presentation of the discipline: place and role of the discipline in the formation of the specific competences of the professional / specialty training program

  Pathologic physiopathology (pathophysiology) is a fundamental medical science and preclinical discipline which at the level of university training allows: a) formation of biological and medical concepts of the essence of pathological processes and diseases; b) acquisition of skills for pathophysiological experiment and interpretation of the obtained information in the experiment; c) studding the general laws of origin, occurrence, evolution and resolution of typical pathological processes and nosology entities; d) studding 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 the curriculum (aim) in professional training

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

- Language (s) of the discipline: romanian, english, russian, french;
- Beneficiaries: students of the IIIrd year, faculty of General Medicine.

II. MANAGEMENT OF THE DISCIPLINE

(autumn semester)

Code of discipline	**F.05.O.043**
Name of the discipline	**PATHOPHYSIOLOGY**
Person(s) in charge of the	**Melnic Eugen, associate professor**

discipline		**Tacu Lilia, assistant professor**	
Year	**III**	Semester/Semesters	**5**
Total number of hours, including:			**120**
Lectures	**30**	Practical/laboratory hours	**25**
Seminars	**20**	Self-training	**45**
Form of assessment	**E**	Number of credits	**4**

(spring semester)

Code of discipline		**F.05.O.043**	
Name of the discipline		**PATHOPHYSIOLOGY**	
Person(s) in charge of the discipline		**Melnic Eugen, associate professor** **Feghiu Iuliana, assistant professor**	
Year	**III**	Semester/Semesters	**6**
Total number of hours, including:			**120**
Lectures	**30**	Practical/laboratory hours	**25**
Seminars	**20**	Self-training	**45**
Form of assessment	**E**	Number of credits	**4**

III. TRAINING AIMS WITHIN THE DISCIPLINE

At the end of the discipline study the student will be able to:

- **at the level of knowledge and understanding:**
  - know the laws of origin, occurrence, 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: general analyses of transudate and inflammatory exudate; biochemical investigation of the blood – protein level, glucose level, lipid spectrum in systemic metabolic changes; immune and allergic status in allergic disorders and immune deficiencies; hydric and electrolytic balance in water dysbalance and electrolytic dysbalance; acid-base balance in acidosis and alkalosis of different origin; oxygen balance in the body in hypoxia of different origin;

- **at the integration 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.

IV.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.

V. THEMES AND ESTIMATE ALLOCATION OF HOURS

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

No. d/o	THEME	Number of hours		
		Lectures	Practical hours	Self-training
1.	Theoretic and general nosology. Object, aim and goals of pathophysiology, methods of research. General etiology. General pathogeny. General sanogenesis.	2	3	3
2.	Cellular typical processes. Cell injury. Apoptosis. Necrosis.	2	3	3
3.	Cellular typical processes. Dedifferentiation. Physiological and pathological regeneration. Physiological and pathological hyperplasia and hypertrophy. Physiological and pathological atrophy. Sclerosis.	2	3	3
4.	Cancerogenesis. Etiology. Pathogenic mechanisms. Cancerogenesis associated with chronic inflammation.	2	3	3
5.	Microcirculatory disorders. Arterial hyperaemia. Embolism. Venous hyperaemia. Transcapillary exchange disorders. Edema. Thrombosis	4	6	6
6.	Inflammation. Etiology. Alteration in the inflammatory foci. DAMP, PAMP and pattern recognition receptors. Inflammatory mediators. Systemic inflammatory response syndrome.	4	6	6
7.	Hypersensitivity disorders. Allergy. Allergic reaction type I, II, III, IV. Autoimmune reactions. Non-specific hypersensitivity. Humoral, cellular, and combined immunodeficiency.	4	6	6
8.	Pathophysiology of carbohydrates, lipid and protein metabolic changes	4	6	6
9.	Pathophysiology of water and electrolytic imbalances	2	3	3
10.	Pathophysiology of acid-basic imbalances	2	3	3
11.	Hypoxia. Hyperoxia. Classification. Etiology. Pathogenesis. Compensatory reactions. Dysthermias. Hyperthermia. Hypothermia. Fever.	2	3	3
Total		**30**	**45**	**45**

Spring semester

No. d/o	THEME	Number of hours		
		Lectures	Practical hours	Self-training
1.	Pathophysiology of red blood cells	2	3	3
2.	Pathophysiology of white blood cells	2	3	3
3.	Pathophysiology of cardiovascular system	4	6	6
4.	Pathophysiology of respiratory system	4	6	6
5.	Pathophysiology of digestive system	4	6	6
6.	Pathophysiology of the liver	4	6	6
7.	Pathophysiology of kidneys	4	6	6
8.	Pathophysiology of endocrine system	4	6	6
9.	Pathophysiology of central nervous system	2	3	3
Total		**30**	**45**	**45**

VI. PRACTICAL TOOLS PURCHASED AT THE END OF THE COURSE

Mandatory essential practical tools (autumn semester) are:

- To be able to interpret changes of erythrocyte sedimentation rate in inflammatory processes;
- To be able to interpret changes of acute phase proteins in the blood;
- To be able to interpret changes of pro- and ant-inflammatory cytokines;
- To be able to interpret changes of pH, HCO3 in the blood;
- To be able to interpret changes of humoral immune status, immunoglobulin spectrum;
- To be able to interpret changes of lymphocytes population;
- To be able to interpret changes of hematocrit in different form of hydric imbalance;
- To be able to interpret changes of lipid spectrum in the blood;
- To be able to interpret changes of glycated hemoglobin.

Practical skills (spring semester):

- To be able to interpret changes of peripheral blood analysis (hemogram, leucogram);
- To be able to interpret changes of ECG;
- To be able to interpret changes of hormonal profile.

VII. OBJECTIVES AND CONTENT UNITS

Objective	Content units
Theme (chapter 1) Theoretic and general nosology. Object, aim and goals of pathophysiology, methods of research. General etiology. General pathogeny. General sanogenesis.	
• **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.	1. Nosology. Object of study. Tasks of pathophysiology. The pathophysiological experiment. 2. General etiology. Cause. Endogenous and exogenous condition. 3. General pathogenesis. Lesion. Pathogenetic factor. Cause-effect relationship. Pathogenic chain. Main pathogenetic link. Vicious circle. 4. Sanogenesis. Reactivity. Adaptive, compensatory, protective, reparative reaction
Theme (chapter) 2. Cellular typical processes. Cell injury. Apoptosis. Necrosis.	
• **To define:** cellular lesion, cellular dysmetabolism, 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, tanatogenic factors. • **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	1. Cell injury 2. Cellular dystrophy

effects of cellular lesions until resolution of the process. 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 pathogenesis of cellular dysmetabolism in dyscirculatory disorders and general dysmetabolism: hyperglycemia, hypoglycaemia, starvation, alimentary, transport and retention hyperlipidemia. 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 information about necrobiosis pathogenesis in the amplification of sanogenetic processes and cellular resuscitation. To demonstrate the complete pathogenetic chain of the cell death at the action of mechanical, physical, chemical, biological, osmotic, oxidative, enzymatic, immunopathologic factors, and hypoxia, hydrogen ions, energy depletion. • **To apply:** knowledge of the pathogenesis of cellular metabolic changes in the explanation of metabolic diseases: liver lipid dystrophy, obesity, atheromatosis. Information of apoptosis in explaining the pathogenesis of proliferative (tumor) and degenerative diseases. Local processes in apoptosis and necrosis with general disorders in the organism. • **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).	3. Apoptosis. Stages of apoptosis: initiation, execution, final. Intrinsic and extrinsic apoptosis 4. Necrosis, necrobiosis, tanatogenic factors
Theme (chapter) 3. Tissular pathologic processes. Dedifferentiation. Physiological and pathological regeneration. Physiological and pathological hyperplasia and hypertrophy. Physiological and pathological atrophy. Sclerosis.	
• **To define:** Definitions of cellular dedifferentiation,	1. Physiologic and pathologic regeneration.

to totipotential, multipotential, pluripotential, unipotently 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. Pathological atrophy. Sclerosis, 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:** 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 hypofunctional physiological atrophy, involutive, senile, endocrine, posthypertrophic. The pathogenetic chain of pathological atrophy in cellular lesions. The pathogenetic chain of sclerosis in cellular lesions. to demonstrate pathogenesis of cancer due to cellular dedifferentiation. • **To apply**: laws of tissue pathological processes in the explanation of disease pathogens: tumoral,	2. Physiologic and pathologic hyperplasia and hypertrophy. 3. Physiologic and pathologic atrophy. 4. Pathologic regeneration. Metaplasia and dysplasia 5. Sclerosis. Collagenogenesis. Collagenolysis

organ compensatory hypertrophy, multiple sclerosis of organs in circulatory 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 the cellular pathological processes into the structure of the diseases.	
Theme (chapter) 4. Cancerogenesis. Etiology. Pathogenetic mechanisms. Cancerogenesis associated with chronic inflammation.	
• **To define:** The concept of carcinogenicity Cancer aetiology. • **To know:** General pathogenesis of neoplastic transformation. Molecular basis of the differentiation process disorder. Genetic injuries (role of mutations in carcinogenesis) Role of genome instability in carcinogenesis. Micro-RNA molecules and their role in carcinogenesis. Phenotypic attributes of carcinogenesis • **To demonstrate:** Pathogenetic mechanisms leading to the formation of the malignant phenotype. Describing the mechanisms leading to tumor cell self-sufficiency for growth factors and the mechanisms leading to their insensitivity to growth inhibitory signals. Mechanisms to prevent apoptosis. Mechanisms of immune evasion of tumor cells. Mechanisms of angiogenesis and metastasis in carcinogenesis. Mechanism of carcinogenesis against a background of chronic inflammation. • **To apply:** Knowledge to integrate the pathogenic links of carcinogenesis. To apply • knowledge in understanding the methods of diagnosis and pathogenetic treatment of cancer. • **To integrate:** Theoretical information about the pathological processes of carcinogenesis in the pathogenesis of various neoplastic diseases.	1. Cancer etiology. Pathogenetic mechanisms 2. Mechanisms of tumor cell immune evasion. 3. Mechanisms of angiogenesis and metastasis in carcinogenesis. 4. Mechanism of carcinogenesis against a background of chronic inflammation
Theme (chapter) 5. Microcirculatory disorders. Arterial hyperaemia. Embolism. Venous hyperaemia. Transcapillary exchange disorders. Edema. Thrombosis	
• **To define:** neurotonic, neuroparalytic, neuromyoparalytic, humoral, reactive functional arterial hyperaemia. Obstructive, obliterate,	1. Arterial hyperemia neurotonic, neuroparalytic, neuroparalytic,

compressive local venous hyperemia. Ischemia. Red and white infarction. Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema. Gaseous, lipidic, air, thrombotic, with amniotic fluid and atheromatous masses embolism. White, red, mixed thrombus. • **To know:** etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromyoparalytic, humoral, functional, reactive, arterial hyperaemia. Obstructive, obliterate, 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 thrombogenesis in arteries and veins. • **To demonstrate:** the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the pathogenetic effect of different forms of edema • **To apply**: the theoretical information in the pathogenic correction of microcirculatory disturbances. • **To integrate:** the theoretical information about local microcirculatory disturbances in pathogenesis of the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension.	neuromyoparalytic, humoral, functional reactive. 2. Ischemia. Embolism, types. 3. Obstructive, obliterating, compressional local venous hyperemia. Prestasis and stasis. 4. Mechanism of thrombogenesis. Formation of white thrombus and red thrombus. 5. Edema. Hypooncotic, hyperosmotic, hydrostatic, membranogenic and lympho-static mechanisms of edema formation.
Theme (chapter) 6. Inflammation. Etiology. Alteration in the inflammatory foci. DAMP, PAMP and pattern recognition receptors. Inflammatory mediators. Systemic inflammatory response syndrome.	
• **To define:** inflammation, alteration, pattern of lesional and pathogenic molecules, cell-and plasma-derived mediators, inflammatory arterial and venous hyperemia, exudation-serous, fibrinous, purulent, haemorrhagic, 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	1. Inflammation. Alteration. Pattern recognition receptors. Cellular and humoral proinflammatory mediators. 2. Vascular reactions in the inflammatory focus. Arterial hyperemia, inflammatory venous hyperemia, ischemia, ischemia, prestasis, stasis,

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 inflammatory process. Apply information on the pathogenesis of inflammation 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.	thrombosis. 3. Exudation. Exudate serous, fibrinous, purulent, haemorrhagic, putrid. 4. Leukocyte emigration. Phagocytosis. Proliferation. Regeneration. Acute phase reaction. Fever. Leukocytosis
Theme (chapter) 7. Hypersensitivity disorders. Allergy. Allergic reaction type I, II, III, IV. Autoimmune reactions. Non-specific hypersensitivity. Humoral, cellular, and combined immunodeficiency	
• **To define:** hypersensitivity disorders, immediate type allergic reactions: immediate hypersensitivity, antibody mediated, immune complex mediated, T-cell mediated; active and passive sensitisation; immunological, pathochemical and pathophysiological 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	1. Allergy. Immediate-type allergic reactions: anaphylactic, cytolytic, immune complex. Anaphylactic shock. Hyposensitisation. 2. Delayed-type allergic reactions 3. Autoimmunity autoantigen, autoantibody. 4. Immunodeficiencies 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 hyposensitisation. To know the etiology, pathogenesis, manifestations and consequences of humoral, cellular and mixed immunodeficiencies. To know 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 into non-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, autoallergy and pseudo-allergic diseases.	humoral, cellular and mixed type. 5. Non-specific hypersensitivity.
Theme (chapter) 8. Pathophysiology of carbohydrates, lipid and protein metabolic changes	
• **To define:** metabolic abnormalities of carbohydrates, lipids, proteins. Hypo- and hyperglycemic factors. Alimentary, . Hypoglycaemia in starvation, in hyperinsulinism. Ketonemia transport hyperglycemia in hipoinsulinism. Hyperglycemic hyperosmolar coma. Ketoacidotic coma. Hypoglycemic coma. Galactosemia. Congenital and acquired dyslipidemia. Alimentary, transport, retention hyperlipidemia. Hyperlipoproteinemia. Hyperlipidemia. Hypercholesterolemia. Atheroma.	1. Carbohydrate dysmetabolism, hyperglycemia and hypoglycemia, ketonemia, hyperosmolar and ketoacidotic hypoglycemic coma 2. congenital and acquired lipid dysmetabolism.

Hyperproteinaemia. Dysproteinaemia. • **To know** causes, pathogenesis, manifestations and consequences of hyperglycemia, hypoglycemia, ketonemia, galactosemia. Causes, pathogenesis, manifestations and consequences of transport, retention, alimentary dyslipidemia. Causes, pathogenesis, manifestations and consequences of hypercholesterolemia. Pathogenesis of atheromatosis. Causes, pathogenesis, manifestations and consequences of hyperproteinaemia, of dysproteinaemia. • **To demonstrate** the pathogenetic chain of hyperglycemia (alimentary, transport, hyperinsulinemia, hipercorticism, hyperkalaemia, hyperthyroidism). The pathogenetic chain of hiperlipidemia (congenital, alimentary, transport, retention). The pathogenetic chain of hypoproteinemia (in starvation, in diabetes, hipercorticism, 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.	Transport, retention, dietary hyperlipidemia. Hyperlipoproteinemia. Hyperchylomicronaemia. Hypercholesterolemia. Atheromatosis. 3. Protein dysmetabolism. Hypoproteinemia. Hyperproteinaemia
Theme (chapter) 9. Pathophysiology of water and electrolytic imbalances	
• **To define:** Definitions: iso-, hypo- and hyperosmolar overhydration. Iso-hypo- and hyperosmolar dehydration. Hyper- and hyponatremia. Hyper- and hypokalaemia. Hyper- and hypocalcemia. Hyper- and hypochloraemia. Hyper- and hypophosphatemia. • **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.	1. hyperhydrate iso-, hypo-, hypo- and hyperosmolar, 2. dehydration iso-, hypo-, hypo- and hyperosmolar. 3. Hyper- and hiponatriemia. 4. Hyper- and hipokaliemia.

Causes, pathogenesis, manifestations and consequences of dymineralosis. Hyper- and hyponatremia. Hyper- and hypokalaemia. Hyper- and hypocalcemia. Hyper- and hypochloraemia. Hyper- and hypochloraemia. Hyper- and hypophosphatemia. • **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_4); • **To apply:** theoretical knowledge in the interpretation of haematological, biochemical parameters, clinical manifestations in the dehydration, electrolyte imbalances • **To integrate:** theoretical information in the diseases (dehydration, overhydration, hyperkalaemia in massive hemolysis	5. Hyper- and hipocalciemia. 6. Hyper- and hypophosphatemia Hyper- and hypochloraemia.
Theme (chapter) 10. Pathophysiology of acid-basic imbalances	
• **To define:** Acidosis (respiratory, metabolic, excretory, exogenous). Alkalosis (respiratory, metabolic, excretory, exogenous). • **To know**: Causes, pathogenesis, manifestations and consequences of acid-base imbalance. Respiratory, metabolic excretory, exogenous acidosis; respiratory, metabolic, exogenous, exogenous alkalosis). • **To demonstrate:** The pathogenetic chain of metabolic acidosis. respiratory, the compensatory reactions that are included and the respective clinical manifestations. The pathogenetic chain of metabolic alkalosis. respiratory, the compensatory reactions included and the respective clinical manifestations. • **To apply:** Theoretical knowledge in the interpretation of hematological, biochemical parameters, clinical manifestations recorded in different types of acid-base imbalance • **To integrate:** Theoretical information to integrate within the diseases ketodiabetic acidosis, in asphyxia, alkalosis in alveolar hyperventilation, in vomiting).	1. Acidosis (respiratory, metabolic, excretory, exogenous) 2. Alkalosis (respiratory, metabolic, excretory, exogenous).
Theme (chapter) 11. Thermic dyshomeostasis. Hyperthermia. Hypothermia. Fever. Hypoxia. Hyperoxia. Classification. Etiology. Pathogenesis. Compensatory reactions.	

• **To define:** 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 respiratory, circulatory, hemic, tissue hypoxia. Causes, pathogenesis, manifestations and consequences of hyperoxia. Causes, pathogenesis, compensatory reactions, manifestations and consequences of hypothermia. Causes, pathogenesis, compensatory reactions, manifestations and consequences of hyperthermia. Etiology and pathogenesis of fever. Pathogenesis and stages of fever evolution: increase, maintains, decrease of temperature. Metabolic and functional disorders. Biological importance. Pathogenetic correction. • **To demonstrate:** 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 recorded in hypoxia, dyspthermia. • **To integrate:** Theoretical information in diseases (hypoxia in diseases of the blood, cardiovascular and respiratory system, febrile component in pathogenesis of infectious diseases.	1. Respiratory, circulatory, hemic, tissue hypoxia. 2. Hyperoxia. Etiology. Pathogenesis. Manifestations. Consequences 3. Hypothermia. Hyperthermia. Fever. Stages of fever. Types of fever. Thermal curve

Spring semester

Objective	Content units
Theme (chapter) 1. Pathophysiology of red blood system	

• **To define:** the concepts of primary and secondary, absolute and relative polycythaemia; 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; the biochemistry of normal bilirubin metabolism and in haemolytic anemias. • **To demonstrate:** peripheral blood smear of primary and secondary, absolute and relative polycythaemia; 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 leukaemia, lymphomas. • **To apply**: theoretical knowledge in interpretation of hematologic picture and clinical manifestations in red blood pathology. • **To integrate:** theoretical knowledge in the pathogenesis of haematological diseases: acute and chronic haemorrhage, iron-deficiency, B12 deficiency and folate deficiency anaemia, autoimmune haemolytic anemia, intoxication with haemolytic toxins;	1. Primary and secondary, absolute and relative erythrocytosis. 2. Iron deficiency anemia 3. B12 deficient and folic acid deficient anemia. 4. Acute and chronic posthemorrhagic anemias. 5. Congenital, acquired hemolytic anemias
Theme (chapter) 2. Pathophysiology of white blood system	

• **To define:** The notions about absolute and relative neutrophilia, eosinophilia, lymphocytosis and monocytosis. Definitions of absolute and relative leukocytopenia, neutropenia, eosinopenia, agranulocytosis, lymphocytopenia. Definitions of hemoblastosis, acute and chronic leukaemia, lymphomas. Etiology, pathogenesis. Peripheral blood 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, eosinopenia, agranulocytosis, lymphocytopenia. Etiology, pathogenesis, manifestations and peripheral blood smear of proliferative disorders in hematopoietic organs: hemoblastosis, acute and chronic leukemias, lymphomas. • **To demonstrate:** Hemogram of absolute and relative leukocytosis, neutrophilia, eosinophilia, lymphocytes and monocytosis; of absolute and relative leukopenia, neutropenia, eosinopenia, agranulocytosis, lymphocytopenia; of acute and chronic leukemias, lymphomas. • **To apply:** 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 pathogenesis of inflammatory and parasitic diseases, immunodeficiencies, autoimmune leukocytopenia, acute and chronic myeloid leukemia, acute and chronic lymphocytic leukemia.	1. Absolute and relative leukocytosis. Neutrophil leukocytosis. Eosinophilic leukocytosis. Basophilic leukocytosis. 2. Lymphocytosis and monocytosis. 3. Absolute and relative leukopenia, neutropenia, eosinopenia, agranulocytosis, lymphocytopenia. 4. Hemoblastosis. Acute and chronic leukemias, lymphomas. Etiology. Pathogenesis. Hematologic picture.
Theme (chapter) 3. 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. • **To 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. 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 hemocirculatory 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 hemocirculatory 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.	1. Cardiogenic circulatory insufficiency, non-coronary cardiogenic, coronary, metabolic, hematogenous. Vasogenic circulatory insufficiency. 2. Primary and secondary arterial hypertension. 3. Chronic and acute arterial hypotension: collapse, shock. 4. Cardiac arrhythmias: Sinus tachycardia and bradycardia. Extrasystoles, atrial and ventricular flutter, atrial and ventricular fibrillation. Incomplete and complete atrioventricular block
Theme (chapter) 4. Pathophysiology of respiratory system	

• **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. • **To integrate:** The theoretical knowledge in the pathogenesis of nosological entities: respiratory paralysis, diaphragm paralysis, myasthenia gravis, pleurisy, pneumothorax, cardiac asthma,	1. Pathophysiology of external respiration. Restrictive ventilatory disorders. 2. Obstructive ventilatory disorders. Upper airway obstruction. 3. Alveolo-capillary gas diffusion disorders. Lung perfusion disorders. 4. Gas transportation disorders: oxygen and carbon dioxide.

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.	
Theme (chapter) 5. Pathophysiology of digestive system	
• **To define:** The notions as: hypo-and hypersalivation, gastric hypoacidity and hyperacidity, chymostasis 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	1. Salivation disorders. Hypo- hypersalivation. 2. Disorders of secretion, motility and evacuation of the food bolus from the stomach. Gastric and duodenal ulcerogenesis. 3. 4. Disorders of pancreatic secretion. Acute and chronic pancreatic insufficiency. 5. Bile secretory disorders. Acholia.

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.	6. Digestive disorders of the small and large intestine
Theme (chapter) 6. Pathophysiology of the liver	
• **To define:** Liver pathophysiology. Hepatic failure. Causes. Pathogenesis. Events. Consequences. Metabolic disorders in hepatic failure. Jaundice: prehepatic, 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 prehepatic, 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. • **To apply:** The theoretical knowledge in interpretation of clinical manifestations and laboratory investigations in hepatic disorders. • **To integrate:** The theoretical knowledge in hepatic nosological entities: hepatitis, hepatitis, steatosis, jaundice, hepatic failure	1. Liver failure. Etiology, pathogenesis, manifestations and consequences. 2. Hepatic coma. Etiology, pathogenesis, manifestations and consequences 3. Liver cirrhosis. Etiology, pathogenesis, manifestations and consequences. 4. Jaundices. Prehepatic, parenchymal and posthepatic jaundice. Etiology, pathogenesis, manifestations and consequences.
Theme (chapter) 7. 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, cylindruria. Hyposthenuria, hypersthenuria and isosthenuria. • **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 urinary evacuation disorders during nephron and urinary tract. The etiology, pathogenesis, manifestations and consequences of acute and chronic renal failure. The etiology, 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, nephrotic syndrome, renal failure, nephrolithiasis.	1. Prerenal, intrarenal and subrenal glomerular filtration disorders. 2. Tubular reabsorption disorders. 3. Tubular secretory disorders. 4. Prerenal, intrarenal and subrenal renal failure. Acute and chronic renal failure. 5. Nephritic and nephrotic syndrome.
Theme (chapter) 8. Pathophysiology 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. Hipoinsulinism. Type I diabetes mellitus.	1. Hyper- and hyposecretion of somatoliberin-somatotropin-somatomedin.

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, manifestations of insufficiency and hypersecretion of the growth hormone and somatomedins, glucocorticosteroids, mineralocorticosteroids, thyroid 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. • **To integrate:** theoretical knowledge in the pathogenesis and manifestations of nosological entities: gigantism and dwarfism, acromegaly, primary and secondary hypercortisolism (Cushing's disease and syndrome), hypocortisolism (Addison's disease), hyperthyroidism (Graves's disease), hypothyroidism (endemic goitre, myxedema), primary hypersecretion of mineralocorticoids (Conn's disease), type I and type II diabetes mellitus, insulin resistance. Hyper-and hyposecretion of GH-releasing hormone-somatotropin-somatomedin, corticotropin-releasing hormone- corticotrophin.	2. Hyper- and hyposecretion of corticoliberin-corticotropin. Hyper- and hypocorticism. 3. Hyper- and hyposecretion of thyroliberin-thyrotrotropin. 4. Hyper- and hypothyroidism. 5. Hyper- and hyposecretion of gonadoliberin-gonadotropins. Male and female hyper- and hypogonadism
Theme (chapter) 9. Pathophysiology of CNS	

• **To define:** Hypoexcitability. Hyperexcitability. Causes. Inhibition of depolarization and hyperpolarization. synthesis, trans-axonal transport, storage, release, recapture and degradation of mediators. Sympathicotonia and parasympathicotonia • **To know:** The mechanisms and disorders of neuron excitation and inhibition (precursors and enzymes for the synthesis of acetylcholine, noradrenaline, dopamine, serotonin, GABA); mechanisms and disturbances of trans-axonal 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, postsinaptic 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 transsynaptic transmission. Disruptions in the synthesis, trans-axonal 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 parasympathicotonia. • **To demonstrate:** The chain of neurophysiologic processes in the excitation and inhibition of excitable cells; the segmental vegetative reflex arc and the pathogenetic chain of segmental vegetative disturbances; the pathogenetic chain of suprasegmental vegetative sympathetic disorders; spinal parasympathetic vegetative reflex arc and the pathogenetic chain of segmental parasympathetic vegetative disorders; the bulbar 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;	1. Neuronal excitability disorders. Hyperexcitability. Hypoexcitability. Depolarizing inhibition. 2. Trans-synaptic transmission disturbances (synthesis, transport, storage, release, postsynaptic mechanisms, reuptake and degradation of mediators). 3. Sympathetic and parasympathetic vegetative disorders The vegetative reflex arch.

• **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.	

VIII. PROFESSIONAL (SPECIFIC (SC)) AND TRANSVERSAL (TC) COMPETENCES AND STUDY FINALITIES

✓ **Professional (specific) (SC) competences**

- PC1. Responsible performance of professional tasks in accordance with the values and rules of professional ethics and the provisions of the legislation in force
- PC2 Adequate knowledge of the sciences about the structure of the organism, physiological functions and behavior of the human organism in various physiological and pathological states, as well as the relationships between the state of health, the physical and social environment.
- PC5. Interdisciplinary integration of the physician's team work with efficient use of all resources.
- PC6. Conducting scientific research in health and other branches of science.

✓ **Transversal competences (TC)**

- TC1. Autonomy and responsibility

✓ **Study finalities**

Note. Discipline finatities (are deduced from the professional competences and the formative valences of the informational content of the discipline).

IX. STUDENT'S SELF-TRAINING

No.	Expected product	Implementation strategies	Assessment criteria	Implementation terms
1.	Working with textbooks	Studying the material from the recommended manuals. Summary the material in the	The ability to reproduce the main notions and the content of the material;	Year of study

		form of postulates. Exposing the material in the form of improvised schemes. Marking the questions that require special consultation	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;	
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
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 collection of tests in pathophy	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

	siology			
6.	Working with online materials	Studying materials on-line from the Department SITE; Working with encyclopaedic 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
7.	Preparation and support of papers, presentations	Selection of research topic, purpose, selection of materials, formulation of conclusions, bibliography.	Workload	Year of study

X. METHODOLOGICAL SUGGESTIONS FOR TEACHING-LEARNING-ASSESSMENT

XI. 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.

XII. **Applied** (specific to the discipline) **teaching strategies / technologies**

In the process of teaching pathophysiology are used: (1) The real and virtual pathophysiological experiment; (2) Logical solving of situational problems

XIII. **Methods of assessment** (including the method of final mark calculation)

Current: includes 2 concluding, in the form of computerized tests consisting of 25 questions each other (single choice and multiple choice) and the evaluation of individual work for each semester of studies (5 and 6 separately), which consists from presentation of homework copybook with resolved clinical cases with explanation of them.

Thus, the annual average mark is calculated from the marks obtained in the totals during the semester (2 marks in the SIMU tests) and 1 mark attributed to individual work.

Students who have at least one negative mark in concluding or who have not recovered absences from practical lessons and seminars will not be admitted to the final exam.

***Final**:* takes place in the computerized assessment room of the USMF. The computerized final assessment computerized test consists of 50 test papers each from all the Pathophysiology course topics and the practical work topics for each semester of study (5 and 6 separately).

The student has 50 minutes to answer the tests. The test is marked with marks from 0 to 10.

The final mark consists of 2 components: annual average mark X 0.5; SIMU computerized test X 0.5.

Method of mark rounding at different assessment stages

Intermediate marks scale (annual average, marks from the examination stages)	National Assessment System	ECTS Equivalent
1,00-3,00	**2**	**F**
3,01-4,99	**4**	**FX**
5,00	**5**	**E**
5,01-5,50	**5,5**	
5,51-6,0	**6**	
6,01-6,50	**6,5**	**D**
6,51-7,00	**7**	
7,01-7,50	**7,5**	**C**
7,51-8,00	**8**	
8,01-8,50	**8,5**	**B**
8,51-9,00	**9**	
9,01-9,50	**9,5**	**A**
9,51-10,0	**10**	

The average annual mark and the marks of all stages of final examination (computer assisted, test, oral) - are expressed in numbers according to the mark 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 in the failed exam.

XIV. RECOMMENDED LITERATURE:

A. Compulsory :

1. Theoretical support elaborated by the department's staff (electronic format)
2. Medical Physiopathology. Collection of situational problems (Under ed. Prof. V. Lutan). Chisinau, 2005 (for individual work).

B. Additional

1. Colour Atlas to Pathophysiology. Stefan Silbernagl, Florian Lang. Calisto Medical Publishing Ed., 2011
2. Robins & Cotran. Pathologic Basis of Diseases. Lippincott Williams & Wilkins, 9th Edition, 2018.
3. Essentials of Pathophysiology: Concepts of Altered States, Carol Mattson Porth, 4th edition, 2014.
4. Understanding pathophysiology, Sue E. Huether, Kathryn L. McCance; section editors, Valentin L. Brashers, Neal S. Rote. 6th edition. Elsevier, 2017.
5. Guyton and Hall textbook of medical physiology, Thirteenth edition 2016 by Elsevier, Inc. All rights reserved, 2016.
6. Cellular and Molecular Immunology, 9th Ed, by Abul K. Abbas, Andrew H.H. Lichtman and Shiv Pillai, 2018.