**FacultY OF MEDICINE**

**STUDY PROGRAM 0912.1 MEDICINE**

**CHAIR OF PATHOPHYSIOLOGY AND CLINICAL PATHOPHYSIOLOGY**

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| APPROVEDat the meeting of the Commission for Quality Assurance and Evaluation of the Curriculum faculty Medicine 2Minutes No.\_7 of \_\_\_\_06.03.2018Chairman, M.D., associate professorSuman Serghei\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ | APPROVEDat the Council meeting of the Faculty Medicine 2Minutes No.\_4\_\_ of \_\_\_\_\_\_20.03.2018Dean of Faculty Ph.D., associate professorBetiu Mircea \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ |
| APPROVEDapproved at the meeting of the chairMinutes No.9. of 25.10.2017Head of chair M.D., professorLutan Vasile\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ |

**SYLLABUS**

DISCIPLINE PATHOPHYSIOLOGY

**Integrated studies**

Type of course: **Compulsory**

Chisinau, 2017

1. **INTRODUCTION**

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

Pathophysiology is a fundamental medical science and preclinical discipline studied during the physician's instruction. Pathophysiology studies the general laws of the disease origin, disease evolution, the ending of the disease and disease structure. The general object of study of pathophysiology is the sick organism. Primordial object of study of pathophysiology is the laboratory animal. Studies done in laboratory animals gave essential information about the pathological processes and experimental disorders, which being extrapolated and adjusted to the human organism constitutes the theoretical basis of experimental pathology and therapy.

● Mission of the curriculum (aim) in professional training:

1. formation of biological and medical concepts of the essence of pathological processes and diseases;
2. aquisition of skills for pathophysiological experiment and interpretation of the obtained information in the experiment;
3. general laws of origin, occurence, evolution and end of typical pathological processes and nosology recursion;
4. knowledges of functional disorders and morphological changes at the molecular, cellular, tissue, organ, system and systemic organism levels in typical pathological processes and diseases;
5. pathogenetic principles for correction of disorders and pathogenetic treatment of pathological processes and diseases;
6. clinical interpretation of laboratory data and laboratory investigations of the organism systems;
* Language of the course: romanian, english
* Beneficiaries: students of the III rd, Medicine faculty no. 2
1. **MANAGEMENT OF THE DISCIPLINE**

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| --- | --- |
| Course code | **F.05.O.045/F.06.O.054** |
| Name of course | **Pathophysiology**  |
| Responsible (s) of discipline | **V. Lutan, M.D., professor****V. Cobet, M.D., professor****V. Rotaru, Ph.D., associate professor** |
| Year | **III** | Semesters | **V, VI** |
| Total hours including: | **300 hours** |
| Course | **68** | Practical classes  | **50** |
| Seminars | **52** | Individual work  | **130** |
| Form of assessment | **Examination****test** | Numbers of credits provided for the course | **6** |

1. **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:*

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

# *at the application level:*

1. to be able to plan, organize and conduct a pathophysiological experiment;
2. to be able to interpret information obtained in the experiment;
3. to record physiological paramerters of nervous activity, heart rate, external respiration, digestive system and kidney activity;
4. to realize laboratory investigations used in pathological experiment (determine the number or erythrocytes, leukocytes, leukogram, level of hemoglobin and color index);

# *at the integration level:*

1. to interpret clinical hemograme, urograme, electrogram, analysis of gastric and duodenal, analysis of exudation and trassudation;
2. to analyze and interpret clinic data of cases that include pathological processes and syndomes of organs systems of the body;
3. to be able to differentiate different pathological processes with similar clinical morphological changes;
4. to be able to formulate the principles of aetiothropic and pathogenetic therapy in different pathological processes;
5. **provisional terms and conditions**

***Student of the third year requires the following:***

● knowledge of the language of instruction;

● confirmed competences in lyceum sciences (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.

1. **themes and ESTIMATE distribution of hours**

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

| Nb. | THEME | Hours |
| --- | --- | --- |
| Lectures | Practical classes/seminars | Individual work |
|  | **General nosology.** General aetiology. General pathogenesis. General sanogenesis. | 2 | 3/3 |  |
|  | **Cellular tipical processes.** Injury of nucleum, cellular membrane, mitochondria, lisosomes. Causes. Mechanisms. Consequence. | 2 | 3/3 | 4 |
|  | **Cellular tipical processes.** Dystrophy. Apoptosis. Necrosis. | 2 | 3/3 | 4 |
|  | **Cellular adaptative processes**. Disorders of differention, proliferation, regeneration. Atrophy. Hypertrophy. Sclerosis | 2 | 3/3 | 4 |
| 5. | **I Atestation** |  | 3/3 |  |
| 6. | **Microcirculatory disorders.** Arterial hyperemia, venous hyperemia, ischemia, stasis, thrombosis.Edemas. | 4 | 6/6 | 8 |
| 7. | **Inflammation.** General etiology and pathogenesis. Alteration. Inflammatory mediators. Vascular reactions in inflammatory focus. Exudation. Leukocytes migration. Proliferation and regeneration. | 4 | 6/6 | 8 |
| 8. | **Hypersensitivity disorders.** General characteristic. Classification. General aetiology. General pathogenesis. **Autoimmune reactions. Immunodeficiency.** | 4 | 6/6 | 8 |
| 9. | **II Atestation** |  | 3/3 |  |
| 10. | **Pathophysiology of metabolic disorders.** General aetiology and pathogenesis of carbohydrates, lipids and proteic dysmetabolisms. | 2 | 3/3 | 8 |
| 11. | **Pathophysiology of water and electrolytic imbalances.** | 2 | 3/3 | 8 |
| 12. | **Pathophysiology of acid-basic imbalances.** | 2 | 1.5/2 | 4 |
| 13. | **III Atestation** |  | 3/3 |  |
| 14. | **Pathophysiology of dysoxia.** Classification. Compensatory reactions. | 2 | 1.5/2 | 4 |
| 15. | **Pathophysiology of termic dyshomeostasis.**Hypotermia,Hypertermia. **Fever.** | 2 | 2/3 | 2 |
| 16. | **Terminal states. Principles of resuscitation.** | 2 |  | 3 |
| 17. | **Reactivity of the organism.** | 2 |  |  |
|  | **V semester** | 17 | 25/26 | 65 |
|  | **Total** | **34** | **50/52** | **65** |
|  |  |  |  |  |
|  | **Pathophysiology of the CNS.** | 2 | 3/3 | 4 |
| 2. | **Pathophysiology of Endocrine system** | 4 | 6/6 | 8 |
| 3. | **IV Atestation** |  | 3/3 |  |
| 4. | **Pathophysiology of Red Blood cell. Polycythemia. Anemia** | 2 | 3/3 | 4 |
| 5. | **Pathophysiology of White blood cells and Lymphoid Tissues** | 2 | 3/3 | 4 |
| 6.  | **Pathophysiology of cardiovascular function Circulatory and cardiac insufficiency** | 2 | 3/3 | 4 |
| 7. | **Pathophysiology of cardiovascular function Vascular insufficiency** | 2 | 3/3 | 4 |
| 8. | **Pathophysiology of respiratory function** | 4 | 3/3 | 4 |
| 9. | **V Atestation** |  | 3/3 | 4 |
| 10. | **Pathophysiology of digestive system** | 4 | 6/6 | 8 |
| 11. | **Pathophysiology of the liver** | 4 | 6/6 | 8 |
| 12. | **Pathophysiology of kidneys** | 4 | 6/6 | 8 |
| 13. | **VI Atestation** |  | 2/2 |  |
| 15. | **Pathophysiology of the pain** | 2 | 0/2 | 3 |
| 16. | **Pathophysiology of the shock** | 2 |  | 2 |
| **VI semester** | **34** | **50/52** | **65** |
| **Total**  | **68** | **102** | **130** |

1. **OBJECTIVES AND CONTENT UNITS**

| **Objectives** | **Content units** |
| --- | --- |
| **Theme 1. General nosology** |
| 1. **1. To define:**
2. the main notions of nozology:pathology, pathological physiology, pathophysiological experiment,nozology, disease, pathological process, etiology, cause, condition, pathogen, lesion, reactivity, adaptive reaction,compensatory, protective, reparative, pathogenetic factor, pathogenetic chain, main pathogenetic link, vicious circle, sanogenesis.
3. **2. To know:**
4. classification and characteristics of causes and conditions, classification and characteristics of physiological reactions.Mechanisms of generalization and localization of pathological processes.
5. **3. To demonstrate:**
6. the role of experiment in studying of pathological processes.
7. **4. To apply:**
8. the notions of nosology in the interpretation of pathophysiological experiments and in medical practice
9. **5. 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. | General Pathology. Pathological physiology. Subject of studies. The tasks of pathophysiology.The pathophysiological experiment. Nosology. Disease. Latent, prodromal, complete devedlopment, resolution.Pathological process. General aetiology. Cause. Endogenous and exogenous condition.General pathogenesis. Lesion. Reactivity. Adaptive, compensatory, protective, reparative reactions.Pathogenic factor. Cause-effect relationship. Patogenetic chain. Main pathogenetic link. Vicious circle.Generalization and localization of pathological processes. General sanogenesis. Primary and secondary pathogenetic mechanisms. |
| **Theme IICellular typical pathological processes** |  |
| **Cellular lesions**.The general causes and the basic pathogenetic mechanisms of cellular injuries according to their origin and nature.Pathogenesis and consequences of lesions of cytoplasmic membrane, nucleus, mitochondria, endoplasmic reticulum ribosomes, lysosomes.Cellular physiological reactions to injuries. Pathogenesis of cell death in cell organ injury.Cellular Dismetabolisms. Etiology and pathogenesis of various forms of cellular dismetabolisms: lipids, carbohydrates, proteins.Pathogenesis of cellular dysmetabolisms in hypoxia, energy deficiency, intracellular accumulation of Ca, hydrogen ions, fatty acids, catecholamines overload, discirculatory disorders, general dismetabolisms.**1.To define:**cellular lesion, cellular dismetabolism**2.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.**3.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, hypoglycemia, starvation, alimentary, transport and retention hyperlipidemia.**4.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.**5. To apply:**knowledge of the pathogenesis of cellular dysmetabolisms in the explanation of metabolic diseases: liver lipid dystrophy, obesity, atheromatosis.**Apoptosis****1.To define:**the concepts of apoptosis, intrinsic and extrinsic, positive and negative apoptogenic factors, degenerative and proliferative diseases.**2.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.**3.To demonstrate:**the complete pathogenetic chain of apoptosis to the action of extrinsic factors (TNF alpha) and intrinsic (cytochrome C).**4.To apply:**information of apoptosis in explaining the pathogenesis of proliferative (tumor) and degenerative diseases (Parkinson disease).**5. To integrate:**local processes in apoptosis and necrosis with general disorders in the organism.**Necrosis:****1. To define:**the notions of necrosis, necrobiosis, physiological and pathological death, tanatogenic factors.**2. To know:**periods of necrosis: cellular disease, cellular agonism, cell death, post-mortem period.Biochemical, functional and structural changes in the cell during dying.**3.To demonstrate:**the pathogenetic chain of cell death at the action of various pathogens factors. To apply the informations about necrobiosis pathogenesis in the amplification of sanogenic processes and cellular resuscitation.**4. To integrate:**cell death with local (inflammation) and general processes in the body (enzymemia, hyperkalaemia, acute phase reaction, fever, stress). | Cellular lesion;lesion of cytoplasmic membrane, nucleus, mitochondria, endoplasmic reticulum, ribosomes, lysosomes. Cellular dysmetabolisms; lipidic, carbohydrates, proteic dysmetabolisms, hypoxia, energy deficiency, hypocalcemia, cellular acidosis, intracellular accumulation of fatty acids, catecholamines overload, dyscirculatory disorders, general dysmetabolism.Apoptosis. Intrinsic and extrinsic, positive and negative apoptogenic factors. Stages of apoptosis: initiation, execution, final. Degenerative and proliferative diseases. |
| **Theme III (chapter)Typical cellular adaptation processes.****Cellular differentiation. Regeneration. Hypertrophy. Atrophy. Sclerosis.** |  |
| **1.To define:**the notions of cellular dedifferentiation, totipotential, multipotential, pluripotential, unipotential cells, differentiation and cloning.Physiological and pathological regeneration. Homeostatic, adaptive, reparative, protective, compensatory regeneration.Pathological atrophy. Labile, stable, progressive sclerosis. Collagenogenogenesis. Collagenolysis.Functional, adaptive, reparative, protective, compensatory hypertrophy.Hypo functional, involutive, senile, endocrine, post-hypertrophic physiological atrophy.Pathological atrophy. Sclerosis, collagenogenesis, collagenolysis.**2.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.**3. 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 hypofunctional physiological atrophy,involutive, senile, endocrine, posthypertrophic. The pathogenetic chain of pathological atrophy in cellular lesions.The pathogenetic chain of sclerosis in cellular lesions.**4. To apply:**laws of tissue pathological processes in the explanation of disease pathogens: tumoral, 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.**5. 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. | Cellular differentiation.Physiological and pathological regeneration, homeostatic, adaptive, operative, protective, compensatory. Pathological regeneration, dysplasia,metaplasia, cancer, scleroderma.Functional, adaptive, reparative, protective, compensatory, endocrine hypertrophy.Hypo functional, involutive, senile, endocrine, post-hypertrophic physiological atrophy.Pathological atrophy. Labile, stable, progressive sclerosis. Collagenogenesis. Collagenolysis. |
| **Theme IVPathological processes in organs. Disorders of microcirculation.** |  |
| Demonstration of arterial hypertension. Classification. Causes. Pathogenetic forms.Hemodynamic and metabolic disorders. Consequences.Demonstration of venous hyperemia.Classification. Causes. Pathogenetic forms.Hemodynamic and metabolic disorders. Consequences.Disorders of transcapilar exchange.Edema: classification. Causes. Pathogenesis. Manifestations and consequences. Pathogenetic correction.Demonstration of ischemia. Classification. Causes. Pathogenetic forms. Hemodynamic and metabolic disorders. Consequences. The importance of collateral hemocirculation. Heart.Demonstration of lipid embolism. Classification of emboli. Causes. Hemodynamic and metabolic disorders. Consequences.Demonstration of thrombogenesis with white thrombus and red thrombus formation. Causes and pathogenesis of thrombogenesis. Hemodynamic and metabolic disorders. Consequences.**1.To define:**neurotonic, neuroparalytic, neuromioparalytic, 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.**2. To know:**etiology, pathogenesis, manifestations and consequences of neurotonic neuroparalytic, neuromioparalythic, 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.**3. To demonstrate:**the pathogenetic chain of various forms of arterial hyperaemia, venous hyperaemia, ischemia, embolism. To demonstrate the patogenetic effect of different forms of edema.**4. To apply**: the theoretical information in the pathogenic correction of microcirculatory disturbances.**5. To integrate**:the theoretical information about local microcirculatory disturbances in pathogenesis of the following diseases: circulatory insufficiency, external breathing disorders, pulmonary hypertension, portal hypertension. | Neurotonic, neuroparalytic, neuromioparalytic, humoral, reactive functional arterial hyperaemia.Obstructive, obliterant, compressive local venous hyperemia.Ischemia, red and white infarction.Congestive, hypooncotic, hyperosmotic, membranogenic, lymphogenic edema.Air, gaseous, lipidic, thrombotic, amniotic fluid, and atheromatous masses embolus.White, red, mixed thrombus. |
| **Theme (chapter) 5**. **Inflammation** |  |
| **1.To define:**notions-inflammation, alteration,pattern of lesionaland pathogenic molecules, cell-and plasma-derived mediators, inflammatory arterial and venous hyperemia, exudation-serous, fibrinous, purulent, hemorrhagic, putrid; leukocyte emigration, phagocytosis, inflammatory proliferation; acute phase reaction, fever and leukocytosis.**2. 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 exsudation 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.**3. To demonstrate:**the pathogenetic chain of different forms of inflammation: alterative, exudative, proliferative.To demonstrate the pathogenetic chain of the systemic inflammatory reaction.**4. 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.**5. To integrate:** information about the etiology, pathogenesis and manifestations of inflammation in the pathogenesis and evolution of inflammatory diseases. | Inflammation, alteration,pattern lesionaland pathogenic molecules, cell-and plasma-derived mediators, inflammatory arterial and venous hyperemia; serous, fibrinous, purulent, hemorrhagic, putrid exudation; leukocyte emigration, phagocytosis, inflammatory proliferation; acute phase reaction, fever and leukocytosis. |
| **Theme (chapter) 6. Hypersensitivity disorders. Autoimmune reactions. Immunodeficiency.** |  |
| **1.To define:**the notions of 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, autoantigen, autoantibody, humoral, cellular and mixed type of immunodeficiency.**2. To define:**the etiology of hypersensitivity disorders and the classification of antigens,pathogenesis of the immunological phase with antibody synthesis or sensitization of lymphocytes,pathogenesisof 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 into non-self antigens.**3. To demonstrate:**the complete pathogenetic chain from inoculating the allergen to structural damage in all types of allergic reactions.**4. 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.**5. To integrate:**the theoretical information about pathogenesis of allergic reactions for involvement in the pathogenesis of allergic, autoallergic and pseudo-allergic diseases. | Allergy. Immediatete hypersensitivity, antibody mediated, immune complexes mediated , T-cell mediated. Active and passive sensitisation.Immunological, pathochemical and pathophysiological phases of allergic reactions.Anaphylactic shock.Hypo-sensitization.Nonspecific hypersensitivity.Autoimmunity.Autoantigen.Autoantibody.Immuno deficiencies of humoral, cellular and mixed types. |
| **Theme (chapter) 7. Integratypical pathological processes****l. Dysmetabolism of nutrients** |  |
| **1.To define:notions:** metabolic abnormalities of carbohydrates, lipids, proteins.Hypo- and hyperglycemic factors.Alimentary, transporthyperglycemia in hypoinsulinism. Hypoglycemia 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.**2. To know:**causes, pathogenesis, manifestations and consequences of hyperglycemia, hypoglycemia, ketoneemia, galactosaemia.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.**3. 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).**4. 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.**5. To integrate:**biochemical, nervous, endocrine and functional disturbances in diseases: type I diabetes, insulin resistance, metabolic syndrome, hyperosmolar hyperglycaemic coma, ketoacidosis coma, hypoglycaemic coma. | Metabolic abnormalities of carbohydrates, lipids, proteins. Hypo- and hyperglycemic factors. Alimentary, transporthyperglycemia in hypoinsulinism.Hyperglycemic hyperosmolar coma. Ketoacidotic coma.Hypoglycemic coma.Hypoglycemia in starvation, in hyperinsulinism.Ketonemia.Galactosemia.Congenital and acquired dyslipidemia.Alimentary, transport, retention hyperlipidemia. Hyperlipoproteinaemia. Hypercholesterolemia. Atheroma. Hyperproteinemia. Dysproteinemia. |
| **Theme (chapter) 8. Disorders of fluid and electrolyte balance, acid-base balance, hypoxia and dystermias** |  |
| **1.To define:**notions: 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).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.**2. 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 hypothermia.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 Importance. Pathogenetic correction.**3.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 electrolite imbalances (Na, K, Ca, Cl, PO4);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).**4. To apply:**theoretical knowledge in the interpretation of haematological, biochemical parameters, clinical manifestations in the dehydration, electrolyte imbalances, hypoxia, acid-base imbalance, dystermia.**5. 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. | Iso-, hypo- and hyperosmolar overhydration. Iso-hypo- and hyperosmolar dehydration. Hyper- and hyponatremia.Hyper- and hypokalaemia.Hyper- and hypocalcemia.Hyper- and hypochloraemia.Hyper- and hypochloraemia.Hyper- and hypophosphataemia). Respiratory, metabolic excretory, exogenous acidosis; respiratory, metabolic, exogenous, exogenous alkalosis).Respiratory, circulatory, hemic, tissue hypoxia.Hyperoxia.Hypothermia, hyperthermia.Fever. Primary and secondary pyrogens, exogenous and endogenous factors. The stages of the fever: increase, maintance, decrease of temperature. Subfebrile, febrile, high and hyperpiretic fever. Patterns of the fever:continuous, intermittent, remitting, recurrent, hectic, atypical. Crisis. Lysis. |
| **Theme (chapter) 9. Pathophysiology of CNS**  |  |
| **1.To define:**Hypoexcitability. Hyperexcitability. Causes. Inhibition of depolarization and hyperpolarization. ynthesis, transaxonal transport,storage, release, recapture and degradation of mediators.Sympathicotonia and parasympathicotonia.**2. To know**: mechanisms and disorders of neuron excitation and inhibition(precursors and enzymes for the synthesis of acetylcholine, noradrenaline, dopamine, serotonin, GABA);mechanisms and disturbances of transaxonal 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 parasympathicotonia.**3. To demonstrate:**the chain of neurophysiological 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 parasympathericvegetative reflex arcand the pathogenetic chain of segmental parasympathetic vegetative disorders; the bulbar parasympathetic vegetative reflex arcand 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;**4.To apply:**the theoretical knowledge for explaining clinical manifestations in neural function disordersand transnaptic transmission; within the disorders of the autonomic nervous system-withinthe paralysis of sympathetic and parasympathetic.**5. To integrate:**theoretical knowledge within the nosological entities:b.Parkinson, neurotrophic intoxication. | Hyperexcitability of the neuron.Neuronal hypoexcitability. Inhibition of depolarization and hyperpolarization. Synapse.Mediator.MAO. COMT.Acetylcholinesterase. Sympathetic and parasympathetic vegetative nervous system. Segmental and suprasegmental vegetative disorders.Increased tonus of Sympathetic nervous system. Increased tonus of parasympathetic nervous system. |
| **Theme (chapter) 10. Pathophysiology of endocrine system**  |  |
| **1.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.Insulinresistance.Type II diabetes mellitus.**2. 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:adrenal glands cortex, 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.**3. To demonstrate:**the pathogenetic chain of primary endocrine disorders,secondary and tertiary for adrenal glands cortex, thyroid gland, gonads.**4. To apply:**the theoretical knowledge to explain biochemicaland clinical disorders in clinical forms of failureand hypersecretion of growth hormones, glucocorticosteroids, mineralocorticosteroids, thyroid hormones, sexual hormones, insulin and glucagon.**5. 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 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-somatomedine,corticotropin-releasing hormone- corticotrophin. | Hyper-and hyposecretion of GH-releasing hormone-somatotropin-somatomedins,corticotropin-releasing hormone- corticotrophinthyroid-stimulating hormone-thyrotropin,gonadotropin-releasing hormone-gonadotropins-luteinizing hormone and follicle-stimulating hormone, prolactin-lactotropin. Hyper-and hypocortisolism. Causes. Hyper-and hypothyroidism. Androgens excess and deficiency. Excess and deficiency of female sex hormones. Hypoinsulinism. Type I and type II diabetes mellitus. Hyperosmolar Hyperglycemic Coma. Ketoacidotic coma. Micro-and macrovascular complications.Hypoglycemic coma. |
| **Theme (chapter) 11. Pathophysiology of the blood system** |  |
| **1.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 aquired hemolytic anemias,the notions of absolute and relative leukocytosis,neutrophilia, eosinophilia, lymphocytosis and monocytosis.The notions of absolute and relative leucopenia, neutropenia, eosinopenia,agranulocytosis, lymphocytopenia.• The notions of hemoblastosis, acute and chronic leukemias. Etiology. Pathogenesis. Peripheral blood smear. **2.To know:**etiology, pathogenesis, manifestations and 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 aquired hemolytic anemias. Normal and pathological hematopoiesis.To know the mechanisms of physiological and intracellular and intravascular pathological hemolysis;the biochemistry of normal bilirubin metabolism and in hemolytic anemias.• **To know** the etiology, pathogenesis, manifestations and peripheral blood smear of absolute and relative leukocytosis, neutrophilia, eosinophilia, lymphocytosis and monocytosis.• **To know** the etiology, pathogenesis, manifestations and peripheral blood smear of absolute and relative leucopenia,neutropenia, eosinopenia, agranulocytosis, lymphocytopenia.• **To know** the etiology, pathogenesis, manifestations and peripheral blood smearof the proliferative disorders in the hematopoietic organs:hemoblastosis, acute and chronic leukemia, lymphomas.**3.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 aquired hemolytic anemias; absolute and relative leukocytosis, neutrophilia, eosinophilia, lymphocytosis and monocytosis; proliferative disorders in the hematopoietic organs:hemoblastosis, acute and chronic leukemia, lymphomas.**4. To integrate**: theoretical knowledge in the pathogenesis of haematological diseases:: acute and chronic haemorrhage, iron-deficiency and megaloblastic anemias, autoimmune hemolytic anemia, intoxication with hemolytic toxins;inflammatory and parasitic diseases,immunodeficiencies, autoimmune leukocytopenia,acute and chronic myeloid leukemia,acute and chronic lymphoid leukemia. | Primary and secondary, absolute and relative polycythemia; hyporegenerative, acute and chronic blood loss anemias, iron-deficiency and megaloblastic anemias, congenital and aquired hemolytic anemias. Bilirubin metabolism. Absolute and relative leukocytosis, neutrophilia, eosinophilia, lymphocytosis and monocytosis.Neutropenia, eosinopenia, agranulocytosis, lymphocytopenia.Hemoblastosis, acute and chronic leukemias, lymphomas. |
| **Theme (chapter) 12. Pathophysiology of the cardiovascular system** |  |
| **1.To define**: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.Extrasistolia, atrial and ventricular flutter, atrial and ventricular fibrillation.The incomplete and complete atrioventricular block.**2.To know:**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.•To know 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 etiology, pathogenesis, manifestations, compensatory reactions, consequences,electrocardiographic picture of heart arrhythmias:tachycardia, bradycardia, sinusal arrhythmia, extrasistolia, atrial and ventricular flutter, atrial and ventricular fibrillation, incomplete and complete atrioventricular block.**3.To demonstrate:** the pathogenetic chain of compensatory reactions and hemo circulatory disorders inmyocardial, endocardial, pericardial diseases.To demonstrate the pathogenetic chain of compensatory reactions and hemo circulatory disorders in vascular disorders -primary and secondary hypertension.To demonstratethe pathogenetic chain of compensatory reactions and hemo circulatory disorders in cardiac arrhythmia:tachycardia, bradycardia, sinusal arrhythmia, extrasistolia, atrial and ventricular flutter, atrial and ventricular fibrillation, incomplete and complete atrioventricular block.**4. To apply:**the theoretical knowledge in the interpretation of clinical manifestations and ECG in cardiovascular pathology.**5. To integrate:**the theoretical knowledge within the entitiesnosological:myocarditis, valvulopathies, pericarditis, coronary insufficiency, atrial fibrillation, atrioventricular block. | Coronary and non-coronary, metabolic, hematogenic cardiogenic circulatory failure. Vasogenic circulatory insufficiency. Primary and secondary arterial hypertension. Acute and chronic arterial hypertension. Tachycardia, bradycardia, sinusal arrhythmia. Extrasistolia, atrial and ventricular flutter, atrial and ventricular fibrillation.The incomplete and complete atrioventricular block. |
| **Theme (chapter) 13. Physiopathology of external breathing** |  |
| **1.To define::** the notions -the pathophysiology of external breathing. Restrictive ventilation disorders.Pulmonary edema.Pneumosclerosis.Pulmonary emphysema.Acute respiratory distress in adults and newborns.Obstructive ventilation 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.**2. To know:**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.•To know: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.• To know:etiology, pathogenesis, manifestations and consequences of external breathing disorders in obstructive processes:upper airways stenosis, asthmatic syndrome.•To know:etiology, pathogenesis, manifestations and consequences of alveolo-capillary diffusion disturbances.•To know: etiology, pathogenesis, manifestations and consequences of pulmonary perfusion disorders:pre- and post-capillary pulmonary hypertension, disorder of the ventilation-perfusion rate.•To know: etiology, pathogenesis, manifestations and consequences of oxygen and carbon dioxide transport disorders: hypoxia and hypercapnia.**3.To demonstrate:**pathogenetic chain of restrictive and obstructive external respiratory disturbances, disturbances of gas diffusion and transport.**4. To apply:**theoretical knowledge in interpreting of clinical manifestations and functional disorders in various forms of external breathing disorders.**5. To integrate:**theoretical knowledge in the pathogenesis of nozological 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. | Pathophysiology of external breathing. Restrictive ventilation disorders. Pulmonary edema. Pneumosclerosis. Pulmonary emphysema. Acute respiratory distress in adults and newborns. Obstructive ventilation 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. |
| **Theme (chapter) 14. Pathophysiology of digestion** |  |
| **1.To define:**notions: 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.**2.To know:**etiology, pathogenesis, manifestations and consequences of salivation disorders: hypo-and hypersalivation.•To know: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.•To know: etiology, pathogenesis, manifestations and consequences of gastric and duodenal ulcerogenesis.•To know: etiology, pathogenesis, manifestations and consequences of pancreatic secretion disorders. Pancreatic Maldigestion.•To know: etiology, pathogenesis, manifestations and consequences of bile secretion disorders: acholia.•To know: etiology, pathogenesis, manifestations and consequences of intestinal digestive disorders: maldigestion, malabsorption, malnutrition.•To know: etiology, pathogenesis, manifestations and consequences of bowel disorders: constipation, diarrhea, gastrointestinal intoxication.**3.To demonstrate:**pathogenetic chain of maldigestion of carbohydrates, lipids and proteins throughout the digestive convex: the oral cavity, the stomach, the small intestine.•To demonstrate:the pathogenetic chain of malabsorption and malnutrition of carbohydrates, lipids and proteins.•To demonstate:the pathogenetic chain of pancreatic maldigestion and in the absence of the bile.**4.To apply:**the theoretical knowledge in interpreting clinical manifestations and laboratory investigations (gastric juice, duodenal juice, the coprologic exam) in digestive diseases.**5.To integrate:**theoretical knowledge in the digestive system diseases: hypertrophic and atrophic gastritis with hyperacidity and hypoacidity, stomach and duodenum ulcer, chronic pancreatitis, acholya, enteritis, diarrhea of different pathogenesis, constipation. | Hypo-and hypersalivation, gastric hypoacidity and hyperacidity.Gastric atonia.Vomiting. Chimostasis in the stomach. Gastric maldigestion. Ulcerogenesis. Pancreatic Maldigestion. Acholia. Intestinal malabsorption. Atonia of bowel. Constipation. Diarrhea. Gastrointestinal poisoning. |
| **Theme (chapter) 15. Liver physiopathology** |  |
| **1.To define:**Liver pathophysiology. Hepatic failure. Causes. Pathogenesis. Events. Consequences. Metabolic disorders in hepatic failure.Jaundice: suprahepatic, parenchymatous, posthepatic.Etiology, pathogenesis, manifestations, consequences.Hyperbilirubinemia.Cholemia. Cholalemia. Acholya.Hepatic cirrhosis: etiology, pathogenesis, manifestations, consequences.**2. To know:**etiology, pathogenesis, manifestations and consequences of liver failure.• To know:disorders of protein, carbohydrate, lipid metabolism, metabolism of bilirubin in hepatic failure.•To know the aetiology, pathogenesis, manifestations and consequences of hepatic coma.•To know the manifestations and consequences of digestive disturbances in hepatic insufficiency.• To know the etiology, pathogenesis, manifestations and consequences of liver cirrhosis.• To know the etiology, pathogenesis, manifestations and consequences of prehapatic, parenchymatous (pre-microsomal, microsomal and submicrosomal) and subhepatic jaundice.**3.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.**4.To apply:**theoretical knowledge in the interpretation of clinical manifestations and laboratory investigations in hepatic disorders.**5.To integrate:**theoretical knowledge in hepatic nosological entities: hepatitis, hepatitis, steatosis, jaundice, hepatic failure. | Hepatic failure. Suprahepatic, parenchymatous, posthepatic jaundice. Hyperbilirubinemia.Cholemia. Cholalemia. Acholya. Hepatic cirrhosis. |
| **Theme (chapter) 16. Pathophysiology of the kidneys** |  |
| **1.To define:**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.**2.To know:**etiology, pathogenesis, manifestations and consequences of pre-renal, intrarenal and subrenal disorders of glomerular filtration.•To know: etiology, pathogenesis, manifestations and consequences of canalicular reabsorption disorders of water, electrilites, proteins, amino acids.•To know: etiology, pathogenesis, manifestations and consequences of urinary evacuation disorders during nephron and urinary tract.•To know: etiology, pathogenesis, manifestations and consequences of acute and chronic renal insufficiency.•To know the etiology, pathogenesis, manifestations and consequences of nephritic and nephrotic syndrome.**3.To demonstrate:** the pathogenetic chain of hydroelectrolytic, acido-basic disorders in renal failure.**4. To apply:**the theoretical knowledge in interpreting clinical manifestations and laboratory investigations in kidney disorders.**5. To integrate:** theoretical knowledge into the pathogenesis of nosologic entities: nephritis, nephrotic syndrome, renal failure, nephrolithiasis. | 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.Quantitative and qualitative disorders of urinary output.Pathological constituents of urine:oliguria, polyuria, nicturia, anuria pollakiuria, proteinuria, glucosuria, bilirubinuria, cilindria.Hypostenuria, hyperstenuria and isostenuria. |

1. **PROFESSIONAL (specific (Sc)) and TRANSVERSAL (Tc) COMPETENCES AND STUDY OUTCOMES**
* **Professional (specific) (Sc) competences:**

• CP1. Knowledge, understanding and deciphering of the notions of theoretical, general and special pathophysiology;

• CP2. Knowing of the hierarchical structure of the organism and the characteristic of the pathological processes at molecular, subcellular, cellular, tissue, organ, system and integral levels.

• CP3. The ability to reveal the content of pathological phenomena according to etiology,

pathogens, manifestations and consequences.

• CP4. The ability of dialectical vision of pathological phenomena to highlightpure pathological phenomena and physiological reactions of the body.

• CP5. The ability to see the ambiguous nature of one and the same pathological phenomenon and to prescribe the physician's tactic in relation to this phenomenon.

• CP6. Ability to formulate the principles of pathogenic correction of disturbances during the disease.

• CP7. Knowing and ability to highlight the basics of the disease structure:symptom, syndrome, pathological process, disease.

• CP8. Ability to highlight disease pathogenicity, the primary and subsequent effects of the harmful factor action.

• CP9. Ability to evidence the pathogenetic chain of pathological, diseases processes and to explainthe pathogenetic factors, the main link of pathogenesis, the vicious circle.

• CP10. Ability to classify pathological processes according to aetiology, succession and the appearance, pathogenesis, clinical form of development.

• CP11. The ability to assemble elementary processes (lesions, pathological process) into the integral structure of the disease.

• CP12. The ability to reveal the pathogenic essence of the clinical manifestations of the disease;

• CP13. Ability to interpret pathogenetically the clinical manifestations of the disease, biochemical, laboratory and functional investigations;

•CP14. The ability to solve pathogenetically the situation problems of the special pathophysiology;

• CP15. The ability to plan, perform and interpret the results of the pathophysiological experiments;

• CP16. The ability to integrate into the analysis and interpretation of pathological processes of previous knowledges (anatomy, histology, biochemistry, physiology, molecular biologyand genetic) and disciplines studied in parallel with pathophysiology(semiology, morphopathology, pharmacology);

• CP17. The ability to integrate clinical and paraclinical manifestations for formulation of conclusions about etiology, pathogenesis and evolution of the pathological process (disease).

* **Transversal competences (tc)**

 •TC1.Ability to create and decipher illustrative materials: tables, schemes, animations;

* TC2. Ability to play pathological processes in essays, presentations, animations;
* TC3. Ability to work with references;
* TC4. Ability to summarize briefly the essence of the bibliographic sources;
* TC5. Ability to make decisions based on available information;
* TC6.Ability to integrate multidisciplinary information;
* TC7. Ability to analyze critically the information with ambiguous dialectical or contradictory character;
* TC8. Formationof the personal position regarding the importance of the discipline studied for

future activity;

• TC9. The ability to evaluate and value the contribution of scholars in the historical aspect;

• TC10. The work with extracurricular information sources;

• TC11. Capacity of interdisciplinary integration.

* **Study findings**

• SF1. To know and apply the notions of theoretical physiopatology in later clinical studies: etiology, cause, condition, pathogenicity, pathogenetic factor, pathogenetic chain,vicious circle, sanogenesis, tanatogenesis;

• SP2. To know and to apply in the notions of pathological processes those of the cellular, tissue, organ, system and integral processes;

• SP3. To know and integrate the major pathological processes in clinical subsequent researches;

• SP4.To use the information about pathogenesis of pathological processes in the formulation

of pathogenetic therapy principles;

• SP5. To use the informations about pathogenesis of pathological processes in the interpretation of

clinical manifestations and paraclinical investigation results.

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

1. **STUDENT'S self-trening**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| No. | Expected product | Implementation strategies | Assessment criteria | Implementation terms |
| 1. | Working with textbooks | Studuing the material from the recommended manualsSummary the material in the form of postulatesPlaying the material in the form of improvised schemesMarking the questions that require special consultation | The ability to reproduce the main notionsand the content of the material; the ability togive the essence;Ability to express 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 material with informationof the theoretical course;Ability to reproduce textually and to interpret presentation the theoretical course; |  |
| 3. | Working with the compendium of practical lessons | Studying the expected experiments for demonstration at the practical lesson:the methodology of the experiment, the obtained results, their interpretation | Ability to integrate experiments intostructure of the theoretical theme;The integration of experimental datain studied pathological processes;The topic illustrationof real factice material;Explanation of experimental results withtheoretical information;Translocation of the experiment into medical practice; |  |
| 4. | Working with the situations problem recommended for the theme | Studying and solving of situation problems | The ability to answer correctly to the questions   of the problems;The ability to interpret the pathogenetic summary ofclinical, paraclinical, and laboratory information;The ability to make conclusions;Ability to make decisions aboutdiagnosis, therapy and prognosis; |  |
| 5. | Working with the pathophysiological explanatory dictionary | Studying the dictionary of physiopathological terms | The ability to reproduce and decipher the essenceof definition and notion |  |
| 6. | Working with the collection of tests in pathophysiology | Studying and solving control tests on the subject;Self-control of material acquisition using the control questions | Monitoring the cognitive process through  self control |  |
| 7. | Working with online materials | Studying materialson-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;Workload |  |

1. **METHODOLOGICAL SUGGESTIONS FOR TEACHING-LEARNING-assessment**
* ***Teaching and learning methods used***

Teaching of pathophysiology discipline uses different methods and teachingprocedures, oriented towards efficient learningand achievingobjectives of the teaching process.In the theoretical course along with traditional methods (courseexposition, interactive course, synthesis course), PowerPoint presentations are used.Tests, situations problems, demonstrationof the film are used in practical workwith the modeling of pathological processes in laboratory animals.Teaching materials (tables, micrographs, transparencies) are usedfor deeper material acquisition.

* ***Applied*** *(specific to the discipline)****teaching strategies / technologies***
* ***Methods of assessment****(including the method of final mark calculation)*

***Current****:*frontal oral evaluation, problem basic learning,test-control -6 attestations in the SIMU system

**Final**:exam-test in SIMU system

The final mark will consist of the middle averageof six intermediate totalizations(coefficient 0.5), and final test (coefficient 0.5).

The annual middle average and the marks of all the final stages of the examination (assisted by

computer, testing) -all will be expressed in numbers according to the rating scale(according to the table), and the final obtained mark will be expressed in number twodecimal places, which will be passed to the notes book.

**Method of mark rounding at different assessment stages**

|  |  |  |
| --- | --- | --- |
| Intermediate marksscale (annual average, marks from the examination stages)  | National Assessment System | ECTSEquivalent |
| **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** |
| **8,51-8,00** | **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.*

**RECOMMENDED literature:**

1. ***Compulsory:***
	1. Robins & Cotran. Pathologic Basis of Diseases. Lippincot Williams & Wilkins, VIIIth  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 Atlass 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
2. ***Extra***
	1. Colev Veronica. Pathiphysiology. Iasi,2001.