FACULTY OF STOMATOLOGY

STUDY PROGRAM 0911.1 STOMATOLOGY

CHAIR OF PATHOPHYSIOLOGY AND CLINICAL PATHOPHYSIOLOGY

APPROVED
at the meeting of the Committee for Quality Assurance and Evaluation of the Curriculum faculty of Stomatology
Minutes No. 1 of 13.09.2021
Committee Prezident, Ph.D.,DMS, Associate professor, Stepco Elena _____

APPROVED
at the Council meeting of the Faculty of Stomatology
Minutes No. 2 of 19.10.2021
Dean of Faculty of Stomatology, PhD, associate professor
Solomon Oleg _____

APPROVED
at the meeting of the chair
Pathophysiology and Clinical Pathophysiology
Minutes No.15. of 18.09.2021
Head of chair M.D., DHMS, Professor
Valeriu Cobeț _____

CURRICULUM

DISCIPLINE PATHOPHYSIOLOGY

Integrated studies

Type of course: **Compulsory**

Responsible (s) of discipline V. Cobeț, M.D., DHMS, professor
V. Rotaru, Ph.D.,DMS, associate professor
C.Hangan, Ph.D.,DMS, associate professor

Chisinau, 2021

I. INTRODUCTION

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

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:

Pathological physiology (pathophysiology) is a preclinical medical discipline, the study of which at the university stage pursues the following aims:

a) cquisition of the methodology of the physiopathological experiment and interpreting the information achieved in the experiment;
b) knowledge of general laws of origin, appearance, evolution and end of typical pathological processes and nozological entities, which underlie dental and orofacial pathology;
c) knowledge of functional disorders and biochemical imbalances at molecular, cellular, tissue, organ, system and body levels in typical pathological processes and diseases;
d) general knowledge of pathological processes with localization in other systems of the body, which have repercussions in the organs of the oral cavity.
e) knowledge of pathogenetic principles of correction of functional disorders and pathogenetic treatment of pathological processes and diseases;
f) clinical interpretation of laboratory and paraclinical data of body systems.

- Language of the course: romanian, english, russian

- Beneficiaries: students of the III rd, Dentistry no. 2

- **MANAGEMENT OF THE DISCIPLINE**

Course code	**F.05.O.060**
Name of course	**Pathophysiology**
Responsible (s) of discipline	**V. Cobeț, M.D., DHMS, professor** **V. Rotaru, Ph.D.,DMS, associate professor** **C.Hangan, Ph.D.,DMS, associate professor**

Year	**III**	Semester	**V**
Total hours including:			**90 hours**
Course	**17**	Practical classes	**17**
Seminars	**34**	Individual work	**22**
Form of assessment	**Examination test**	Numbers of credits provided for the course	**3**

I. 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 the rules of behavior with the laboratory animals, the methodology of the pathophysiological experiment and the interpretation of the information obtained in the experiment;
2. To define the theoretical bases of general, special and clinical pathophysiology;
3. To know the laws of origin, appearance, evolution and end of typical pathological processes;
4. To know the laws of origin, appearance, evolution and end of typical pathological processes located in different organs and systems;
5. To know the structural changes, biochemical imbalances and functional disorders at the molecular, cellular, tissue, organ, system and body levels in typical pathological processes and diseases;
6. To know the principles of pathogenetic therapy of pathological processes and diseases.

- ***at the application level:***

1. To be able to plan, organize and perform a physiopathological experiment;
2. To be able to interpret the information obtained in the experiment;
3. To be able to record physiological parameters of nervous, cardiac, external respiration, digestive system, kidney systems;
4. To be able to perform the laboratory investigations used in the pathophysiological experiments (determination of erythrocyte count, leucocytes, leukogram, amount of hemoglobin, chromaticity index).

- ***at the integration level:***

1. To interpret clinically hemograms, urograms, electrocardiograms, spiromograms, gastric and duodenal analysis, exudate and transudate analysis;
2. To be able to analyze and interpret clinically the complex situation issues, including pathological processes and syndromes located in the organism systems;

3. To be able to differentiate different pathological processes with similar clinical-morphological manifestations;
4. To be able to formulate the principles of the etiotropic and pathogenetic therapy of various pathological processes.

II. PROVISIONAL TERMS AND CONDITIONS

Student of the third year requires the following:

Studying and acquiring the discipline of pathophysiology requires the knowledge of the teaching language, confirmed skills in lyceum (biology, chemistry, physics), ability to communicate and team work, parallel study of preclinical disciplines general such as patomorphology, internal and surgical disease semiology, general pharmacology, ability to select and integrate achieved knowledge, applying of clinical thinking skills, pathogenetic analysis of diseases and principles of pathogenetic therapy. Good knowledge of the subject matter requires good knowledge of normal physiology, biochemistry, histology.

III. THEMES AND ESTIMATE DISTRIBUTION OF HOURS

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

THEME	Hours		
	Lectures	Practical classes/ seminars	Individual work
Object, tasks and methods of pathophysiology. Lesions c cytoplasmic membrane, mitochondria, lysosomes. Cause Mechanisms. Consequences.	**2**	**3**	
Cellular lessions. Necrosis. Apoptosis. Dystrophies		**3**	**2**
Disorders of regeneration. Atrophy. Hypertrophy. Sclerosis.		**3**	
Disorders of local microcirculation. Arterial hyperaemia. Venous hyperemia. Stasis. Thrombosis. Embolism. Ischemia.		**3**	**2**
Pathophysiology of the fluid-coagulating system. Hypo- and hypercoagulation.		**3**	**2**
Inflammation. Etiology. Pathogenesis. Biological significance. Fever. Etiology. Pathogenesis. Stages.	**2**	**3**	
Inflammatory process in the oral cavity organs		**3**	
Allergy. Immunodeficiency states in the oral cavity	**2**	**3**	**2**

THEME	Hours		
	Lectures	Practical classes/ seminars	Individual work
Pathophysiology of the metabolism		**3**	
Disorders of the sensitivity. Pain	**2**	**3**	**2**
Pathophysiology of the RBC, WBC	**3**	**3**	**2**
Pathophysiology of the cardiovascular system	**2**	**3**	**1**
Pathophysiology of the digestive system.	**2**	**3**	**2**
Pathophysiology of the liver. Hepatic failure		**3**	**2**
Pathophysiology of endocrine glands		**3**	**2**
Pathophysiology of the respiratory system		**3**	**2**
Pathophysiology of the kidneys. Renal failure	**2**	**3**	**1**

THEME	Lectures	Practical classes/ seminars	Individual work
Total	**17**	**51**	**22**

VI. OBJECTIVES AND CONTENT UNITS

Objectives	Content units
Theme (chapter) 1. General Nosology	
• To define the notions of reaction, process, pathological condition, disease, dental focal disease • To know the structure of the disease and its general characteristic • To define the notions of etiology, general pathogenesis • To know the differences between the cause of the disease and the condition of the disease • To comment the role of the lesion in the mechanism of the disease, the cause-effect correlations, the vicious circle in the evolution of pathology or disease • To demonstrate the role of the main pathogenetic link in various diseases • To comment the ways of generalizing of the local process • To comment the mechanisms which lead to the localization of the pathological process or disease • To demonstrate the role of primary and secondary sanogenic mechanisms in the disease progression. • To apply knowledge about specific and non-specific resistance of organs in the oral cavity in the evolution of oro-buccal pathologies • To comment the significance of morbid process end, general tanatogenesis • To apply knowledge to other disciplines • To make conclusions • To develop own opinions on the main pathogenetic link, the vicious circle, the mechanisms of sanogenesis	General Nosology - general etiology, general pathogenesis, general nozology, general sanogenesis, general tanatogenesis. Etiology - studies the causes and conditions of disease emergence. Patogeny - studies the mechanisms of the appearance, development and resolution of the disease. Knowing the cause of the disease allows the correct application of etiotrope therapy or its prevention. Knowing the main pathogenetic link from the vicious circle allows the using of treatment to be removed - pathogenetic treatment. General sanogenesis studies general health laws. Primary sanogenic mechanisms include adaptive, protective and compensatory responses, which are included until the lesions occur and are directed towards maintaining the homeostasis of the body confronted with the pathogen. Secondary sanogenic mechanisms include protective, compensatory and terminal mechanisms, aimed to restoring of homeostasis that are already unbalanced (during the onset of illness).
• **Theme (chapter) 2. Typical cellular and tissue pathological processes**	

Objectives	Content units
• To define the notions of cellular lesions, dystrophy, apoptosis, necrosis, pathological regeneration, hyperplasia, hypertrophy, cancer, atrophy, sclerosis. • To know the mechanisms of cellular membrane lesions and their impact on cellular function • To know the mechanisms of cellular lesions and their impact on organ and body functioning. • To demonstrate the role of the main pathogenetic link in the evolution of the pathological process in functional disorders of mitochondria, lysosomes, nucleus, endoplasmatic reticulum. • To comment the role of cellular lesions in the mechanisms of dystrophy, necrosis, apoptosis, and sclerosis. • To apply the consequences of cellular lesions for the estimation of pathological processes located in organs. • To apply the particularities of the regenerative process in the oral cavity organs in the evolution of the dental pulp affection, the periodontal • To demonstrate the differences between apoptosis and necrosis • To demonstrate the differences between hyperplasia and organ hypertrophy. • To demonstrate pathogenetic variants of the sclerosis development • To apply knowledge for the reasoning of pathogenetic treatment • To apply knowledge to other disciplines • To formulate conclusions • To apply and integrate knowledge for the study of typical pathological processes in organs and whole organs	Cell lesion is the persistent dyshomeostasis of biochemical imbalance, structural change and functional disorder at the action of the damaging factor. Primary lesions of the cytoplasmic membrane bears the specific fingerprint of the etiological factor Consequences of irreparable and irreversible cellular lesions are: cellular dystrophies, apoptosis, necrosis, necrosis, inflammation, atrophy, sclerosis, acute phase of lesions, hyperkalemia, enzyme, fever. Dystrophy - a typical cellular pathological process caused by general or cellular metabolic disorders and manifested by functional disorders and structural changes of the cell Apoptosis is an intrinsic tanatogenic program of cell suicide, aimed at preserving the quantitative and qualitative homeostasis of the cell population. Necrosis - irreversible shut down of cell activity in the living organism with subsequent disintegration of the structure. Hyperplasia - the process of amplifying cellular multiplication, which leads to an increase in the number of cells in the cell population or organ. Organ hypertrophy is the increase in volume and mass of cell-mediated cell hyperplasia and / or cell volume and mass (cell hypertrophy). Atrophy - a overvital process of reducing cellular organelles, cells, tissues and organs associated with the reduction or cessation of their functions. Sclerosis is the process of pathological regeneration linked to cellular necrosis, which consists in the substitution of specialized parenchymal structures or specialized connective tissue with identical structures.
Theme (chapter) 3. Typical pathological processes in organs	
• To define the notions of arterial hyperemia, venous hyperemia, ischemia, blood stasis,	Arterial hyperaemia is the excessive filling of an organ or portions of tissue with

Objectives	Content units
embolism, thrombosis, inflammation, allergy, autoallergy, pseudoallergy, anaphylactic shock. • To know the pathogenetic mechanisms of arterial hypertension, venous ischaemia, blood stasis, embolism, thrombosis, inflammation, allergy, autoallergy, pseudoallergy, anaphylactic shock and their impact on organ function, including those in the oral cavity. • To argue the features of arterial and venous hyperaemia, ischemia, blood stasis, embolism, thrombosis, inflammation, allergy, autoallergy, pseudoallergy in the organs of the oral cavity. • To apply the effects of biologically active substances in estimating disorders in the oral cavity organs. • To demonstrate the principles of pathogenetic treatment for arterial and venous hyperemia, ischemia, blood stasis, embolism, thrombosis, inflammation, allergy, anaphylactic shock, autoelegia, pseudoallergy. • • To integrate pathogenetic mechanisms of arterial and venous hyperemia, ischaemia, blood stasis, embolism, thrombosis, inflammation, allergy, autoallergy, pseudo-allergy, anaphylactic shock with the functional, structural features of the organs in the oral cavity. • To explain the consequences of inflammation, allergy, autoallergy, pseudoallergy in the organs of the oral cavity on the whole body. • To apply the morphological and functional particularities of the oral cavity organs in the evolution of the dental pulp affection, the paradonitis within the inflammation, the drug allergy, the autoallergy • To apply the knowledge to other specialized disciplines. • To formulate conclusions	arterial blood as a result of increased blood inflow through dilated arterioles concurrently with increased perfusion Venous hyperemia is the excessive filling of a organ portion with venous blood or resulting in difficulty through the vein while reducing the perfusion. Ischemia is the disorder of peripheral blood circulation as a result of diminishing or interrupting the influx of blood concomitantly with organ hypoperfusion. Stasis is the slowing down or cessation of blood circulation at the microcirculatory level in an organ or tissue portion. Embolsm is the presence and circulation into the blood vessels of endogenous or exogenous foreign particles that obstruct the vascular lumen and disrupt blood circulation. Inflammation - typical pathological process, response to cell lesion of any etiology, directed to reduced action and elimination of the pathogen factor from the body, delimitation of the lesion, liquidation of injured structures and their replacement with viable structures. Allergy (hypersensitivity) is the exaggerated and qualitatively modified sensitivity and reactivity of the body to antigenic and haptenic substances, which are based on immune-related (humoral and / or cellular) reactions associated with cellular lesions, inflammation, necrosis. Autoimmunity (autoallergy) is a humoral or cellular immune reaction triggered against the body's own antigens.

Objectives	Content units
Theme (chapter) 4. Systemic dishomeostasis of metabolic processes. Hypoxia. Thermic dyshomeostasis	
• To define the notions of metabolic imbalances, hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-, hyponatremia, hyper-, hypocalcaemia, hyper-, hypokalaemia, hypophosphataemia, fluorosis, dehydration, acidosis, alkalosis, hypoxia, hyperthermia, fever. • To know the types of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia hyper-, hyponatremia, hyper-, hypocalcemia, hyper-, hypokalaemia, hypophosphataemia, fluorosis, dehydration, acidosis, alkalosis, hypoxia, hyperthermia, fever. •To know the pathogenetic mechanisms of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-, hyponatremia, hyper-, hypocalcaemia, hyper-, hypokalaemia, hypophosphataemia, fluorosis, dehydration, acidosis, alkalozei, hypoxia, hyperthermia, fever.	Metabolic dyshomeostasis are typical pathogenic processes occurring at the action of pathogenic factors and is characterized by deviations from the constant concentration of substances in the internal environment of the body, which leads to the disruption of metabolic processes at the cellular, organ and whole level. Hypoglycaemia is a decrease in blood glucose concentration below 0.08% (4.4, mmol / L) as a consequence of insufficient intake or intensive carbohydrate catabolism. Hyperglycaemia is an increase in blood glucose concentration above 6.6 Mmol / L or 0.12% as a consequence of the imbalance between glucose intake and metabolism. Hyperlipidemia is the increase of lipid content in the blood - chylomicrons, alpha-lipoproteins, pre-beta-lipoproteins, beta-lipoproteins and albumin
• **To know** compensatory reactions in hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper- and hyponatremia, hyper- and hypocalcemia, hyper- and hypokalemia, hypophosphatemia, fluorosis, dehydration, acidosis, alkalosis, hypoxia, hyperthermia, fever • **To demonstrate** the impact of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever on organs of the mouth. • **To argue** the correlation between protein, carbohydrate, lipid, hydroelectrolytic, acid-base disturbances, hypoxia and the functional and structural status of mouth structures	***Hiperlipidemia*** reprezintă sporirea conţinutului de substanţe lipidice în sânge - chilomicroni, alfa-lipoproteine, pre-beta-lipoproteine, beta-lipoproteine şi albumine *Hipoproteinemia* represents reduced level of proteins in the blood below 70 g/L. Protein defficiency leads to reduced local specific and non-specific protection in the mouth, development of local immunodefficiency – reduced immunoglobulin production, and reduced synthesis of protective factors at the level of mouth mucosal layer. ***Hipernatremia*** represents ehnanced level of sodium in the blood above 152 mEq/l. ***Hiponatremia*** represents reduced level of sodium in the blood bellow 135 mEq/l.

Objectives	Content units
• **To apply** the pathogeny of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever in evaluation of the changes in the organs of the mouth on organs of the mouth. • **To demonstrate** the principles of pathogenetic treatment in hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever on organs of the mouth. • **To integrate** the pathogenetic mechanisms of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever with functional and structural features of organs of the mouth. • To demonstrate the differences between fever and hyperthermia. • To demonstrate the metabolic role of fluoride in the teeth tissue. • To demonstrate the pathogenetic principles for treatment in hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever on organs of the mouth. • **To formulate** conclusions regarding the impact of hyperglycemia, hypoglycemia, hypoproteinemia, hyperlipidemia, hyper-and hyponatremia, hyper-and hypokalemia, hyper-and hypercalcemia, hyperphosphatemia, fluorosis, acidosis, alkalosis, hypoxia, hyperthermia, fever on organs of the mouth.	***Hiperkalemia*** represents enhanced level of potassium in the blood above 5,5 mEq/l. ***Hipokalemia*** represents reduced level of pottassium in the blood bellow 3,5 mEq/l. ***Hipercalcemia*** represents enhanced level of calcium in the blood above 5,3 mEq/l (or 2,6 mmol/l). ***Hipocalcemia*** represents reduced level of calcium in the blood bellow 4,5 mEq/l (or 2,1 mmol/l). ***Hypophosphatemia*** represents reduced level of phosphates in the blood bellow 0,8 mmol/l. *Fluoride* (F) in the human body is present in sma;ll amounts but is an compolsory element for normal development of the body. Fluoride is a remarcable osteotrop agent. Daily requirements in fluoride in a healthy adult is 0,2-0,6 mg. *Fluoride excess (fluorosis)* leads to disturbances in many system of organs but especially is associated with teeth and bone diseases. *Fever* - a tipical integral pathologic process that can develop in animals and humans in response to every cell injury and inflammatory reaction, characterised by restructuring of thermoreglatory centrer in the hypothalamus with change of set-point to a higher level. *Hypoxia* is a tipical pathological process characterized by reduced oxygen level in the cells, as the result of dysbalance between oxygen suply and oxygen requirements. *General hypoxia* is a tipical pathological process which lead to changes in all systems and organs of the body , and the degree of damage depend on the degree of resistence to

Objectives	Content units
• To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	oxygen deficiency of the tissues. So, the bone tissue is one of the most resistent, and can preserve its viability without oxygen within several hours; skeletal muscles – aproximatelly 2 hours; myocardium – 20-40 min. The lesst resistent to hypoxia is the cerebral tissue. After 2-3 minutes of oxygen lak there can be attested the first signs of injury in the neuones, and within 6-8 minutes develops irreversible neuronal injuries.
Theme (chapter) 5. Physiopathology of central nervous system. The pain	
• To define the definitions of excitability, sensibility, pain, oro-facial pain. • To know the conductor pathways for different type o sensibility and pain. • To know the pathogenetic mechanisms of sensibility disturbances, pain and oro-facial pain. • To differentiate different types of sensibility and pain, inclusive the pathological types. • To differentiate the trigeminal pain, temporo-mandibular pain, mio-fascial pain. • To demonstrate the impact of pain on the body. • To demonstrate the role of oro-facial pain in the pathological processes in the mouth. • To argue the correlation between different sensibility disturbances and functional and structural features of the organs of the mouth. • To apply the pathogeny of trigeminal pain, temporo-mandibular pain, mio-fascial pain in understanding the changes in the organs of the mouth. • To demonstrate the principles of pathogenetic treatment for trigeminal, temporo-mandibular and mio-fascial pain. • To formulate conclusions regarding the impact of trigeminal, temporo-mandibular and mio-fascial pain on organs of the mouth and the whole body.	*Sensibility* represents a fundamental characteristic of biological material which allows the active equilibration with environmental factors and adequate adaptation to new existing conditions. The morpho-functional elements of analizer are: 1) receptors 2) the first sensitive neuron localized outside of the central nervous system (usuallly in the spinal ganglia); 3) the second neuron localized in the spinal chord, medulla oblongata or midbrain 4) the third neuron localized in the thalamus or geniculate bodies; 5)the fourth neuron localized at the level cerebral cortex. *Types of sensibility disturbances* - *hyperesthesia* - enhanced sensibility; develop in case of causalgia and several thalamic syndromes, when all sensations are registered as painful sensation; - *hypoesthezia* – represents reduced cutaneous sensibility to some peripheral stimuli (pressure, light touch, hot or cold); - *anesthezia* – total absence of cutaneous sensibility to the same stimul; - *hypoalgesia* – loss of painful perception; - *hyperalgesia* – represents an exagerate reaction to a painful stimului; - *paresthesia* – abnormal sensations, which take the form of pins, bites, numbness. etc.;

Objectives	Content units
• To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	*Pain* – specific, subjective sensation which is the body reaction with involvement of emotional component, vegetative reactions, changes in the functions of internal organs, unconditioned motor reflexes and voluntary movements oriented to remove the painful stimulus.
Theme (chapter) 6. Physiopathology of endocrine system	
• To define the notions of hyperfunction and hypofunction of endocrine glands • To knows the principles of organisation of endocrine system (trans-pituitary and para-pituitary pathways) • To knows the mechanisms of endocrine system auto regulation. • To differentiate primary, secondary and tertiary endocrine disturbances. • To estimate hormonal changes in the blood in primary, secondary and tertiary endocrine disturbances of adrenal glands, thyroid gland, sexual glands. • To differentiate different types of hyperaldosteronism, hypo- and hypercorticosolism, hypo- and-hyperthyroidism, insulin deficiency. • To know the effects of tropic hormones, vasopressin, ACTH, cortisol, thyroid hormones and insulin on the body tissues and on the organs of the mouth. • To demonstrate the role of somatotropin in development of diseases of the facial bones and teeth. • To demonstrate the role of insulin deficiency in development of body disturbances and disease of the periodontal structures. • To apply the pathogeny insulin deficiency in estimation of changes at the level of the mouth (epithelial hyporegeneration, local immunodeficiency, dystrophy of paradont, local acidosis). • To demonstrate the principles of pathogenetic treatment in endocrine disorders.	*Hypersecretion of growth hormone* manifests clinically by gigantism in the children and acromegaly in adults. The most frequent cause of somatotropin hypersecretion is the pituitary adenoma which develop from somatotroph pituitary cells. The cortical layer in adrenal glands consists from three layers: glomerular, fascicular and reticular. These three layers secret distinct three hormones – mineralocorticoids, glucocorticoids and sexual androgenes. *Hyperaldosteronism* represents hypersecretion of aldosteron and can be primary and secondary. The primary hyperaldosteronism is developing in aldosteron –secreting tumor in the cortex of adrenal glands. Secondary hyperaldosteronism develops in the result of hypovolemia or kidneys ischemia which is associated with activation of renin-angiotensin-aldosteron system. In 70-90% patients with diabetes mellitus is found the disease of periodontal structures. In the pathogeny of periodontal disorders in insulin deficiency can be implicated several pathogenetic factors: diabetic acidosis, microangiopathy, disturbances in protein synthesis, enhanced proteolysis, enhanced breakdown of proteoglycans and collagen fibers, diminished fibroblasts

Objectives	Content units
• To formulate conclusions regarding the impact of insulin deficiency, hypercortisolism, excessive somatotropin on the organs of the mouth. • To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	proliferation. All these lead to weaken the ligamentary apparatus of the teeth. On the basis of metabolic disturbances there can be attested reduced chemotactic abilities of the neutrofils as well as reduced phagocytosis. All these favor the development of pathogenic microorganism in the oral cavity. *(Porphyromonas gingivaiis, Prevotella melaninogenica, Streptococcus intermedius, B. intermedius. B. gingivaiis, Candida albicans etc...).*
Theme (chapter) 7. Physiopathology of the blood	
• To define the notion of hypo- and hypervolemia, anemia, erythrocytopenia, erythremia, leucocytosis, leucopenia, leukaemia. • To know the types and characteristics of hypo- and hypervolemia and to differentiate them on blood analysis. • To know the pathogenetic classification of anemias. • To differentiate the notion of erythrocyte hypochromia and hyperchromia, microcytosis, macrocytosis, hyper – and hyporegeneration of erythroblast series. • To estimate the causes and consequences of iron deficiency, B_{12} vitamin deficiency and folate deficiency anemia for the body. • To estimate and differentiate the disorders in the organs of the mouth in iron deficiency anemia, B_{12} vitamin deficiency and folate deficiency anemia, haemolytic anemias, acute and chronic post-bleeding anemias. • To know how to differentiate iron deficiency anemia, B_{12} vitamin deficiency and folate deficiency anemia, haemolytic anemias, acute and chronic post-bleeding anemia in the blood analysis. • To demonstrate the pathogenetic chains within iron deficiency anemia, B_{12} vitamin deficiency and folate deficiency anemia, haemolytic anemias, acute and chronic post-bleeding anemias and their impact on whole body and organs of the mouth.	The blood represents a fluid tissue which consists frpm blood cells (erythrocytes, thrombocytes and leucocytes) and the liquid part – the blood plasma. Due to its components the blood has multiple functions: nutrition, respiration, protection, excretion. *Erythrocytosis* represents enhanced number or erythrocytes and hemoglobin in a unit of blood, usually 1L. Red blood count in females is 4,7x 10^{12}/L, in males 5,7x 10^{12}/l *Anemia* represents a syndrom characterized by reduced level of red blood cells and hemoglobin in a unit of blood. According to pathogenetic mechanism anemia can be classified in several types: I. anemia caused by disturbances in differentiation and proliferation in the bone marrow caused by hypoplasia. II. Anemia caused by disturbances in the process of erythrocytes maturation. III. Anemia caused by excessive hemolysis of red blood cells. IV. Anemia caused by excessive blood loss blood-loss anemia acute or chronic). Disturbances in the mouth that develop in iron deficiency anemia depends on several pathogenetic mechanisms: a) reduced tisular perfusion in the organs of the mouth, hypoxia and hyponutrition; b) hyposecretion of salivary glands; c) reduced immune reactivity and trophic reactivity in the tissues of the mouth;

Objectives	Content units
• To demonstrate the correlations between diseases of the mouth and diseases of the blood. • To demonstrate the role of leucocytes in maintenance of oral cavity homeostasis. • To know the causes and general mechanisms of leucocytosis, leukopenia and leucosis. • To differentiate different types of leucocytosis and leukaemia according to changes in the peripheral blood picture. • To apply the pathogenetic mechanisms of leucocytosis and leucosis for understanding the changes at the level of the organs of the mouth (local immunodeficiency, dystrophy of periodontal structures, inflammation, mucosal necrosis) • To demonstrate the pathogenetic principles of treatment in anemia, leucocytosis and leukosis. • To estimate the changes in blood analysis in the treatment of inflammatory disorders, leucosis. • To formulate conclusions regarding changes in the blood analysis and their impact on the functional and structural homeostasis of the organs of the mouth. • To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	d) reduced physiologic regeneration of cells and antioxidant systems, leading to reduced resistence to microorganisms, chemical and physical factors. The mucosal layer of the mouth is characerized by a gray-green discoloration, is dry and many inflamamtory reactions can be triggered. *Lack of* B_{12} vitamin and folic acid leads to development of anemias which in the organs of the mouth are associated with inhibition of the processes of physiological regeneration, reduced resistence of mucosa to action of traumatic factors. There can develop disturbances of cell metabolic reactions, epithelial desquamation, reduced local immune reactivity. There is caharacteristic the development of inflammatory-atrophic foci on the mouth and lingual mucosa with atrophy of papilla - "*Hunter glossitis*
Theme (chapter) 8. Physiopathology of fluido-coagulant balance	
• To define the notion of hemostasis, thrombosis, hypocoagulation, hemorrhagic syndrome. • To know the characteristics of primary and secondary hemostasis. • To know the causes and mechanisms of thrombosis, hypocoagulation, hemorrhagic syndromes and their consequences for whole body. • To know how to differentiate the hemorrhagic syndrome according to changes in the hemogram and/or biochemical blood analysis.	Fluidocoagulant balance is maintained by a diversity of physiological and biochemical reactions which involves the coagulation system, anti-clotting system and fibrinolytic system. The hypercogulability state is determined by several pathogenetic mechanisms:

Objectives	Content units
• To differentiate the notions of thrombocytopenia and thrombocytopathy. • To differentiate different types of coagulopathy. • To estimate the role of anti-coagulant and fibrinolytic syndrome in pathogeny of hemorrhagic syndromes. • To estimate the pathological changes in the organs of the mouth in hemorrhagic syndromes. • To demonstrate the correlations between diseases in the mouth and teeth and changes in the status of fluido-coagulant balance. • To apply the pathogeny of hypercoagulation and hypocoagulation for understanding the changes in the organs of the mouth (local immune protection, dystrophy of periodontal structures, local inflammation, mucosal necrosis). • To demonstrate the risks of hypo- and hypercoagulation syndromes with odontogen origin in development of whole body dyshomeostasis. • To demonstrate the principles of pathogenetic treatment in hypocoagulation and hypercoagulation syndromes. • To formulate conclusions regarding the changes in fluido-coagulant balance and the impact on organs of the mouth. • To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	*a) Enhanced level of pro-clotting substances in the blood;* *b) enhanced level of activator of clotting factors in the blood , this can develop in shock, septicemia, burns* etc.; *c) reduced level or inhibition of anti-coagulant substances in the blood* (anti-thrombin III deficiency in liver disorders, heparin deficiency in enhanced level of lipids in the blood); *d) reduced blood level or inhibition of fibrinolytic system in the blood.* In function of the main stages of hemostasis, the hemorrhagic syndromes can be grouped in 5 big categories: a) Hemorrhagic syndrome caused by disturbances in blood vessles; b) Hemorrhagic syndrome caused by disturbances in thrombocytes; c) Hemorrhagic syndrome caused by disturbances in the clotting system (coagulopathy); d) Hemorrhagic syndrome caused by excessive activation of anti-coagulant sytem; e) Hemorrhagic syndrome caused by excessive activation of fibrinolytic system. Bleeding represents a early sign in dieases and injury of periodontal structures. If the bleeding last more than 2-3 days than this is considered to be a complication of trauma. Surgical drainage of flegmons and abseces in the anatomical area of the mouth are assocaiated with considerable activation of clotting system caused by release of tissue factor as well as inhibition of fybrinolysis. Odontogen sepsis, abseces, flegmones, oro-facial ostemielitis can trigger dissiminated intravascular coagultion, characterized by intravascular hypercoagulation, depression of fibrinolysis, consumption of thrombocytes and fibrinogen in the proces of enhanced coagulation.
Theme (chapter) 8. Physiopathology of cardiovascular system	

Objectives	Content units
• To define notion of circulatory insufficiency, heart failure, coronary insufficiency, vascular insufficiency, arterial hypertension, sinus tachycardia and sinus bradycardia, extrasystole. • To know the causes and pathogenetic mechanisms, compensatory reactions involved in heart failure. • To differentiate the heterometric heart hyperfunction and homeometric heart hyperfunction. • To estimate the changes of hemodynamic parameters in cardiovascular failure. • To differentiate on ECG sinus heart arrhythmias, atrial and ventricular extrasystole, myocardial ischemia, myocardial necrosis. • To differentiate primary arterial hypertension from secondary arterial hypertension. • To know the pathogenetic mechanisms of acute vascular failure, collapse. • To know the principles of pathogenetic treatment in heart and vascular disorders. • To formulate conclusions regarding the impact of cardiovascular pathogenetic changes on structural and functional state of the organs of the mouth. • To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	**Cardiac failure** is determined by inability of the heart to supply an adequate cardiac output to satisfy the metabolic requirements of the tissues of the body. The main pathogenetic factors of cardiac failure are reduced force and velocity of myocardial contraction, indiferently of etiological factors. **Coronarian insufficiency** is a tipical form of heart failure characterized by a disbalance between coronarian blood flow to the myocardium and the current metabolic requirements of the heart. **Arterial hypertension** represents a persisten enhanced systolic pressure above 140 and dyastolic pressure above 90 mm Hg. Arterial hypertension can be classified in two groups: primary arterial hypertension or essential arterial hypertension ans secondary or symptomatic arterial hypertension. Symptomatic arterial hypertension develop in the result of disturbances in organs and system of organs, which are involved in maintanance of systemic arterial pressure. **Acute vascular failure** is characterized by sudden drop of systemic arterial pressure and venous pressure.
Theme (chapter) 9. Pathophysiology of respiratory system	

Objectives	Content units
• To define the notion of external respiratory failure. • To know the arterial blood gas pressure in norm and respiratory diseases. • To differentiate the notion of pulmonary restriction and pulmonary obstruction. • To know the changes of lung volumes and lung capacities in obstructive lung disease and restrictive lung disease. • To apply the knowledge for explanation of pathogenetic mechanism of pulmonary fibrosis, lung atelectasis, pulmonary edema, bronchial asthma. • To define the notion of dyspnea, asphyxia. • To know the types of dyspnea and asphyxia, the causes and the mechanisms of development, the changes on spirometry. • To demonstrate the role of oral pathology in pathogenesis of lung obstruction disorders. • To demonstrate the principles of pathogenetic treatment in pulmonary fibrosis, lung atelectasis, pulmonary edema, bronchial asthma. • To apply and integrate the knowledge in understanding the diseases of the mouth in the context of other clinical fields.	*Respiratory failure* is the inability of the breathing system to satisfy the body requirements in oxygen in resting as well as during physical activity, in condition of normal atmospheric pressure. *Restrictive respiratory failure* develop when there is lung restriction caused by disorders located at the level of lung parenchyma, thoracic cage, pleura and neuro-muscular structures. *Obstructive respiratory failure* is the result of obstruction at the level of superior or inferior airways. *Dyspnea* represents a presisten change in the rythm, amplitude and frequency of breathing, associated with enhanced effort of breathing and subjective feeling of lack of satisfaction from breathing. *Asfixia* represents acute respiratory failure characterized by disorders in oxigen supply (hypoxia) and removal of carbon dioxide (hypercapnia).
Theme (chapter) 10. Pathophysiology of digestive system and liver	

Objectives	Content units
• To define the notions of dental caries, paradontosis, hypo- and hypersalivation, xerostomia, gastric hyposecretion, stomachal hypoacidity, ulcerogenesis, malabsorbtion, maldigestion, liver failure, jaundice. • To know the causes and the mechanisms of pathological processes from the oral cavity: dental caries, paradontosis, hypo- and hypersalivation, disturbance of mastication and deglutition. • To estimate the role of macroorganisms, of local immune system, of saliva, of masticatory muscles in the pathogenesis of dental caries, paradontosis, pulpitis and dysphagia (difficulty swelling). • To explain the disturbances of gastric and duodenal digestion in dental disorders. • To estimate the role of masticatory muscles in the pathogenesis of gastric and duodenal disorders. • To explain the consequences of oral cavity's disorders on the organs of gastrointestinal tract. • To estimate the role of pancreas in digestion processes. • To estimate the disturbance of duodenal digestion. • To explain the pathogenesis of meteorism and of gastro-intestinal autointoxication. To estimate the correlation between pathology of gastrointestinal tract and state of oral cavity. • To estimate the role of liver in digestive processes. • To be able to explain the disturbances of metabolic processes and hemostasis in liver failure. • To be able to differentiate the liver specific functions and role for the organs of oral cavity. • To explain the role of hemorrhagic syndrome in oral cavity in case of liver failure.	The disturbance of digestion can interest one or more compartments of the gastro-intestinal tract: oral cavity, stomach and intestines. *Dental caries* – it is a pathological process, characterized by the progressive destruction of tough dental tissues (enamel, dentine) with formation of defect like a cavity. A big importance is attributed to the strecococic microflora group A of the oral cavity. *Streptococcus mutans* and *Lactobacillus* are the most common in formation of bacterial plaque. Disturbances of salivary secretion (hyposalivation, xerostomia etc.) contribute to the formation of the dental tartar (calculus), composed by the adhesion polyglycans that are formed under microbial splitting of glucose. Mastication acts like as a reflex with voluntary control. Disturbances of mastication are determined by the alimentary deficiency, by alteration of the proper mastication, by the alteration of time mastication and by the disturbance of the regulatory mechanisms of mastication, by the oral cavity malformation ("cleft lip" - palatoschisis), by the abnormal position of the teeth and maxillary, by the dental affection (severe periodontal diseases, alveolar periostitis, alveolar pyorrhea, pulpitis) which weaken the fixation of the teeth. Ulcer disease represents the autodigestion of the gastric mucosa by the own pepsin secretion in the presence of hydrochloride acid as a consequence of imbalance between factors that damage the gastric mucosa and protective factors. Intraluminal digestion is preponderant assured by the pancreatic enzymes, biliary components and specific intraluminal factors. *Intestinal maldigestion* represents disturbance of intraluminal digestion and/or parietal digestion, so being considered a component of malabsorbtion syndrome.

Objectives	Content units
• To estimate different types of jaundice (prehepatic, hepatic, posthepatic) – causes and pathogenetical mechanisms. • To explain the biochemical changes of the blood in gastrointestinal disorders. • To be able to differentiate different types of jaundice from the biochemical blood analyzes and of urinary test. • To be able to define the mechanisms and manifestations of cholemic syndrome and acholia. • To be able to make correlation between abnormal function of the liver and periodontal disorders. • To demonstrate the principles of pathogenetic treatment in paradontosis, dental caries, stomach hyperacidity, ulcerogenesis, pancreatitis, jaundice, liver failure. • To be able to make conclusion regarding to changes of gastrointestinal tract and its impact on the sate of oral cavity. • To apply and integrate the knowledges for studying the pathophysiology of diseases from oral cavity.	The disturbance of pancreatic secretion could be determined by the reduction of secretory parenchyma mass in acinar canalicullar atrophy and sclerosis. Between the organs of oral cavity and the gastrointestinal tract there are tight correlation. So, disorders of the teeth, tong, pharynx lead to the digestive disturbances, but pathology of the internal organs frequently are followed by the pathological manifestations in the oral cavity. Liver failure represents incapacity of the liver to performs one or more its functions, appeared as a result of hepatocytes injury. In liver failure is decreased synthesis of specific proteins (prothrombin, proconvertine, fibrinogen etc.), which with the *hypovitaminosis K* lead to *bleeding syndrome*. *Jaundice* represents one complex syndrome, characterized by the yellowish of sclera, of the mucosa and the skin, appeared under increased bilirubin level in the blood (hyperbilirubinemia).
Theme (chapter) 11. Pathophysiology of kidney	
• To define the notion of glomerulonephritis, nephritic syndrome, nephrolytiasis, pielonephritis, uretritis and cystitis. • To know the causes and pathogenetical mechanisms of filtration disturbances, of abnormal reabsorbtion and secretion, concentration and dilution of the urine, and disturbance of urinary elimination. • To be able to estimate phospho– calcium metabolic disturbance at the level of the kidney. • To be able to estimate the role of rennin-angiotensin system, of erythropoietin and of prostaglandin in homeostasis of the body. • To be able to explain the mechanisms of hematuria, proteinuria, glucosuria, leucocyturia, hypo – and hypersthenuria.	The main function of the kidney is to maintain the internal environment homeostasis (hydric homeostasis, electrolytic homeostasis, acid-base balance, volemic and osmotic homeostasis *Effective filtration pressure (EFP)* is equal with 30-40 mmHg. Summary volume of filtrate of two kidneys is 125 - 130 milliliter/minute, which corresponds with quantity of 170- 180 liters of glomerular filtrate during 24 h. *Glomerular proteinuria* is determined by the increased permeability of the renal filter as consequences of inflammatory glomerular processes. *Hematuria* represents presence of the erythrocytes in the final urine and can be caused by the different renal diseases (acute

Objectives	Content units
• To explain the causes and mechanisms of oliguria, anuria and poliuria. • To be able to estimate the general analyzes of urine (quantitative changes of the urine). • To be able to make correlation between abnormal renal function and affection of periodontal disease. • To demonstrate the principles of pathogenetic treatment in glomerunephritis, pielonephritis, uretritis and cystitis. • To apply and integrate the knowledges for studying the pathophysiology of diseases from oral cavity.	and chronic nephritis, nephrolytiasis, nephrocalcinosis, tuberculosis, urinary pathway infection). *Glomerular leukocyturia* is a sign of renal disorder and of urinary tract, being frequent found in infection of urinary pathways. *Hyposthenuria (watery diuresis)* is realising of urine with lower osmolarity than of plasma. *Hypersthenuria* is characterized by the realising of urine with high osmolarity with density over 1035 and can be found in dehydration states. In case of chronic pathological processes of the kidney, also develop microcirculatory disorders, general intoxication, which act unfavorable on the barrier function of the oral cavity mucosa, decreasing the resistance to the environmental factors.

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

Professional (specific) (SC) competences:

- ✓ Knowledge of the general lows of the etiology, pathogenesis, evolution and particularities of the typical pathological processes that underlie the dental and orofacial pathology
- ✓ Analysis of various cellular, subcellular processes that lead to various pathologies in the organs of the oral cavity
- ✓ Knowledge of the general consequences of pathological processes with localization in the oral cavity for the body
- ✓ Overview of pathological processes with localization in other body systems, which have repercussions in the oral cavity
- ✓ Estimation of disorders in haemogram, general urine analysis, general biochemical analysis of blood, coagulogram
- ✓ Solving the problems basic and formulating the conclusions

Transversal competences (TC)

- ✓ Improvement of self training capacity and decisional autonomy
- ✓ Improvement of the skills of clinical thinking
- ✓ Development of different learning techniques (modeling and estimation of didactic experiments, interpretation of hemograms, problem basic learning, clinical cases) experiments, interpretation of hemogram, learning based on clinical cases)

Nr.	Expected product	Implementation strategy	Assessment criteria	Implement ation terms
1.	Working with informational sources	Studying lectures and the didactic material according to the respective theme . Reading questions from the topic, which need a reflection on the subject To know the list of supplementary informational sources that referred to the respective topic. To select the supplementary informational sources. To read the all text of the topic very attentive and to write the essential content, to make schemes, tables. To formulate conclusions and importance of the studied topic.	To be able to understand essential from the studied topic and to be able to interrelated them.	During the whole semester
2.	Working with copybook of practical lesson	To understand didactic experiments and to be able to analyze information from the lectures, material, images, didactic videos of the respective topic. To resolve the consecutive tasks, to formulate conclusions at the end of each practical lesson. To check and to appreciate realizing of lessons.	The work volume, resolving the clinical cases, and abilities to formulate conclusions	During the whole semester
3.	***Applying different learning techniques***	Evaluation of knowledge by the discussing the topic in the group according to the set of questions to the respective topic. Learning by the utilization of clinical cases. Learning by estimation the results of pathophysiological experiments and formulation of conclusions.	The work volume, the degree of understanding the essence of different subjects, level of scientific argumentation, qualitative of conclusions, demonstrating of problem, formation of personal attitude.	During the whole semester
	Working with on-line material	Online autoevaluation, expressing the proper opinions by the forum and chart	The number and the time spending on the SITE, results of autoevaluation	During the whole semester
	Preparing and presentation the thesis/ portfolios:	Selection of the research theme, establishment the plan of thesis, establishment the term of realization. establishment of components of the project/ Power Point presentation, the goal, the results, conclusions, practical implications, bibliography. Reviews of the colleges and of the professors	The work volume, degree of understanding the topic of the project, level of scientifically argumentation, qualitative of conclusions creative elements, formation of personal attitude, concordance between exposure the material and	During the whole semester

			scientific rectitude, graphic presentation, model of presentation.	

Study findings

- ✓ To know particularities of the morbid processes evolution
- ✓ To know sanogenetic mechanisms in evolution of pathological processes or diseases
- ✓ To understand the mechanisms of localization and generalization of pathological processes or diseases
- ✓ To know general lows of etiology, pathogeny, evolution and particularities of typical pathological processes
- ✓ To understand repercussions between disorders of oral cavity and disorders of organs and organ's systems of the body
- ✓ To be able to identify the possible causes and conditions of disorders from oral cavity and their consequences on the cells, tissues and hole body
- ✓ To be able to differentiate notion of pathological process, pathological state and diseases
- ✓ To know particularities of pathological processes with localization in the organs of oral cavity
- ✓ To be able to use knowledge and methodology from pathophysiology in explanation the nature of some pathological destructive processes from oral cavity at the discipline of stomatology
- ✓ To be able to apply the accumulated knowledge in the researcher activity

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

IV. STUDENT SELF – TREINING

VIII. METHODOLOGICAL SUGGESTIONS FOR TEACHING – LEARNING - ASSESSMENT

Teaching and learning methods used

- The discipline pathophysiology and clinical pathophysiology is teaching by using different methods and didactic procedures, orientated toward efficient learning and to achievement of processes didactic objective's. During the theoretical course along with traditional methods (course exposition, interactive course, synthesis course), also are used clinical cases. During the practical lessons are used different types of individual evaluation, direct, in-group, laboratory works with estimation of didactic videos. For deeper understanding the material are used different semiotic systems (scientific language, graphic and computering language) and didactictic material (tables, schemes, microphotos).

- ***Methods of recommended learning***
- **Analysis** – imaginary dividing of the whole material into component parts. Highlighting the essential elements. Studying each element as a component part of the total material

- **Analysis of the scheme/ figure** - selecting the necessary information. Recognition the indicated structures in the scheme or figure, based on the knowledge and selected information.
- **Comparing** - analysis of one pathological process from one group and establishment essential features of it. Analysis the second pathological process and it's features. Comparing of the processes and highlighting their common characteristics. Comparing of the processes and highlighting their differences. Establishment differential criteria. Formulation of conclusions.
- **Classification** – identification of the processes, or disorders that should to be classified. Determination of criteria which need for classification. Distribution of the processes or disorders in the groups according to established criteria.
- **Realising of the schemes for the learning process** - selection of the elements, which should to figure in the schemes. Explanation of selected elements by the different symbols/ colors and indication of the relationship between them. Formulation of the adequate title and to use syllabus.
- **Experiment** – formulation of hypothesis, based on known facts, according to the studded processes or phenomena. Evaluation of experiment algorithm. Checking the hypothesis by realising of studded processes/phenomena in laboratory condition. Viewing of didactic videos. Formulation of conclusions, deduced from arguments or findings at the end of the video's viewing.

- ***Strategy/didactic technology that are applied*** *(specific for the discipline)*

- „Brainstorming", „Clinical case"; „Multi-voting"; „Round table"; „Creative controversy"; „Technical focus-group".

- ***Methods of assessment*** *(inclusive with indicating of calculation the final mark)*

✓ **Current***:* frontal control or/and individual by:
- Application of tests,
- Resolving problems/ exercises,
- Analysis of clinical case
- Test –control

✓ **Final:** exam

✓ **Final mark** will consist of middle average of three current attestations which were passed at the computer with positive mark (in SIMU) coefficient 0.5 and final test in computerized system with coefficient 0.5

Method of mark rounding at different assessment stages

Intermediate grill marks (annual average, marks from the examination stages)	National Assessment System	Equivalent ECTS
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.

V. *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:

A. 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

B. Extra

a. Colev Veronica. Pathophysiology. Iasi,2001.