**FacultY OF MEDICINE**

**STUDY PROGRAM 0912.1 MEDICINE**

**CHAIR OF PATHOLOGY**

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| --- | --- | --- | --- |
| APPROVED at the meeting of the Commission for Quality Assurance and Evaluation of the Curriculum  faculty Medicine  Minutes No. \_\_\_\_ of \_\_\_\_\_\_\_\_\_\_\_\_\_  Chairman, PhD, university professor  Padure Andrei\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ | | APPROVED at the Council meeting of the faculty  Medicine 1  Minutes No.\_\_\_\_ of \_\_\_\_\_\_\_\_\_\_\_\_\_  Dean of Faculty Ph.D., university professor  Placinta Gheorghe \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ | |
| APPROVED at the meeting of the chair  Minutes No.6 of 01.03.2024  Head of chair  PhD, university professor  Melnic Eugen\_\_\_\_\_\_\_\_\_\_\_ | |

**CURRICULUM**

DISCIPLINE: **PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM**

**Integrated studies/ Cycle I, License**

Course kind: **Optional discipline**

Curriculum elaborated by:

**Ciobanu Nicolae, PhD, research professor**

Chisinau, 2024

1. **PRELIMINARIES**

**General presentation of the discipline: the place and role of the discipline in the formation of specific competencies of the vocational training program / specialty**

• Cardiovascular Pathophysiology as an optional subject within the Study Program 0912.1 Medicine is an important landmark in the university training of students, primarily in the subject of Cardiology from the spectrum of Internal Medicine.

• Within this optional subject, students are promoted to acquire detailed pathophysiological knowledge of the basic entities of cardiovascular pathology (e.g., essential and pulmonary hypertension, heart failure, cardiorenal syndrome), regarding the pathogenetic mechanisms of triggering and evolution, the pathophysiological significance of functional and biochemical diagnostic markers, of prognosis predictors, as well as the conceptually argued landmarks of pathogenetic treatment.

• The objectives of the discipline are: knowledge, deepening and correct use of the pathophysiological entities of circulatory homeostasis disorder, comprehensive acquisition of the molecular and cellular mechanisms underlying vascular endothelium impairment, the evolution of essential and pulmonary hypertension, diastolic heart failure and cardio-renal syndrome, consolidation of the algorithm of functional and biochemical markers that have diagnostic and prognostic value, as well as justification of pathogenetic treatment guidelines.

**Curriculum mission (purpose) in professional training**

• The optional discipline "Pathophysiology of the Cardiovascular System" has the role of providing students with theoretical knowledge, contemporary conceptual elements and practical skills regarding the comprehensive understanding of the pathogenetic mechanisms of triggering cardiovascular diseases, as well as their complications, in order to use the knowledge received in the formation of the algorithm of feasible markers and predictors in diagnostic and prognostic estimation, as well as in the justification of pathogenetic treatment.

• The discipline aims to provide students with additional easy premises in the complex training within the Bachelor of Medicine program, in the perspective of the employment required after graduation in residency programs.

• The discipline uses and promotes the principle of "training through research", in which it is important to adjust the conceptual material treated within the theoretical course and practical works to the evidence obtained within the scientific research at the molecular and cellular level in the "Molecular Medicine" center.

* Language(s) of teaching of the subject: Romanian, English, Russian.
* Beneficiaries: 4th year students, Faculty of Medicine 1 and 2, Medicine specialty.

1. **ADMINISTRATION OF DISCIPLINE**

|  |  |  |  |
| --- | --- | --- | --- |
| Code of discipline | | **CD 8.5.1.** | |
| Name of discipline | | **Pathophysiology of the cardiovascular system** | |
| Executors of discipline | | **Cobet Valeriu, Tacu Lilia** | |
| Year | **IV** | Semester | **8** |
| Number of hours, inclusive: | | |  |
| Cours | **10** | Practical/laboratory works | **10** |
| Seminars |  | Individual work | **20** |
| Evaluation form | **E** | Number of credits | **1** |

# III. TRAINING OBJECTIVES WITHIN THE DISCIPLINE

# *At the end of studying the discipline, the student will be able to possess:*

# *● at the level of knowledge and understanding:*

# Ꝩ Knowledge of the pathogenetic mechanisms of endangering circulatory homeostasis in the context of the vessel-cord-vessel continuum.

# Ꝩ Knowledge of the basic mechanisms of endothelial injury and dysfunction.

# Ꝩ Understanding the mechanisms and molecular and cellular events leading to vascular and myocardial remodeling.

# Ꝩ Knowledge of the mechanisms of elevation of blood pressure and damage to target organs in essential hypertension.

# Ꝩ Knowledge of the mechanisms of elevation of pulmonary blood pressure and the pathophysiology of *cor pulmonale* development.

# Ꝩ Knowledge of the mechanisms of impairment of the lusitropic function of the myocardium and elevation of diastolic heart failure.

# Ꝩ Knowledge of the pathophysiological patterns and pathogenesis of cardiorenal syndrome.

# Ꝩ Knowledge and understanding of the pathophysiological significance of functional and biochemical markers of endothelial dysfunction, vascular remodeling and heart failure.

# Ꝩ Knowledge and understanding of pathophysiological landmarks based on circulating and functional biomarkers useful in pathogenetic and personalized treatment of cardiovascular diseases.

# *at the level of application:*

# Ꝩ Familiarizing students with the pathophysiological landmarks applied in the estimation and diagnosis of endothelial dysfunction and vascular remodeling based on the analysis of the algorithm of functional and biochemical markers.

# Ꝩ Using the algorithm for estimating the severity and prognosis of essential and pulmonary arterial hypertension, including the connotation of target organ damage.

# Ꝩ Using the algorithm for diagnosing and estimating the origin of diastolic heart failure.

# Ꝩ Using the algorithm for diagnosing and estimating the pathophysiological patterns of cardio-renal syndrome.

# Ꝩ Applying the evidence of assisted scientific research in the pathogenetic scheme of the explored cardiovascular diseases.

# *at the level of integration:*

Ꝩ Familiarization with the main research directions of the pathophysiology of cardiovascular diseases and the application of the conceptual elements learned in their diagnostic and prognostic algorithm and in the consolidation of pathogenetic and personalized treatment.

ꝨContinuous formation and consolidation of the complex and integral algorithm of the acquired knowledge and skills.

ꝨPracticing the capacity for synthesis, exegesis and selective bibliographic documentation.

**IV. CONDITIONS AND PREREQUISITES**

For the acquisition of the optional subject "Pathophysiology of the Cardiovascular System":

Ꝩ knowledge of the language of instruction.

Ꝩ digital skills (using the Internet, processing documents, spreadsheets and presentations);

Ꝩ communication and teamwork skills.

Ꝩ qualities - tolerance, avidity, competitiveness, autonomy.

Ꝩ deep knowledge in fundamental and clinical sciences: anatomy, human physiology, molecular biology, medical genetics, biochemistry, microbiology, pathophysiology, morphopathology, medical semiology, radiology, etc.

1. **TOPICS AND INDICATIVE DISTRIBUTION OF HOURS**

***Courses (lectures), practical/ laboratory works/seminars and individual work***

| Nr.  d/o | ТЕМА | Number of hours | | |
| --- | --- | --- | --- | --- |
| Courses | Practical works/seminars | Individual work |
|  | Pathophysiology of endothelial dysfunction and vascular remodeling | 2 | 2 | 4 |
|  | Pathophysiology of essential hypertension | 2 | 2 | 4 |
|  | Pathophysiology of pulmonary arterial hypertension | 2 | 2 | 4 |
|  | Pathophysiology of diastolic heart failure | 2 | 2 | 4 |
|  | Pathophysiology of cardiorenal syndrome | 2 | 2 | 4 |
| **Total** | | **10** | **10** | **20** |
| **Total** | | **40** | | |

**VI. PRACTICAL SKILLS ACQUIRED AT THE END OF THE DISCIPLINE:**

• Algorithm of functional tests for detecting circulatory dyshomeostasis.

• Algorithm of functional and biochemical markers for estimating endothelial dysfunction, vascular remodeling and myocardium.

• Algorithm for assessing the pathophysiological patterns of cardiorenal syndrome.

• Algorithm for estimating the pathophysiological patterns of pulmonary arterial hypertension, as well as *cor-pulmonale*.

1. **REFERENCE OBJECTIVES AND CONTENT UNITS**

| **Objectives** | **Content units** |
| --- | --- |
| **Topic (chapter) 1. Pathophysiology of endothelial dysfunction and vascular remodeling** | |
| * To define | The entities and functional support of the vascular endothelium in the context of the control of basal vascular tone, proliferation, hypertrophy, migration and cellular apoptosis. The basic expressions and manifestations of endothelial dysfunction. The morpho-functional entity of vascular remodeling and its impact on circulatory dyshomeostasis. |
| * To know | Causal factors and main cardiovascular risk factors (modifiable and non-modifiable) leading to endothelial damage and dysfunction. Mechanisms of nitric oxide (NO), prostacyclin (PGI2) and hydrogen sulfide deficiency and the impact of these endothelium-derived factors on vascular physiology. Pathogenetic contribution of renin-angiotensin-aldosterone system activation in endothelial dysfunction.  Mechanisms of the pathogenetic interface formed by oxygen free radicals and pro-inflammatory cytokines in endothelial injury and dysfunction. Mechanisms of vascular remodeling and its repercussions. Functional and biochemical markers reflecting compromised endothelium-dependent vascular reactivity and their pathophysiological significance in the context of strengthening pathogenetic and personalized treatment. |
| * To demonstrate | Knowledge about the functional complexity of the vascular endothelium, the consequences deriving from endothelial dysfunction and their pathophysiological role in circulatory dyshomeostasis.  Knowledge about the phenomenon of vascular remodeling and its impact on the pathophysiology of cardiovascular diseases.  Skills to understand the role of NO deficiency in jeopardizing the control of basal vascular tone and the pathophysiology of vascular remodeling.  Skills to understand the pathogenetic contribution of hyperhomocysteinemia, hypercholesterolemia, hyperglycemia, inflammation, oxidative stress, excess of asymmetric dimethyl-arginine, L-arginine and tetrahydrobiopterin deficiency in the quantitative decline of NO.  Skills to understand the role of endothelial dysfunction in triggering the vascular remodeling process, including through the enhancement of the mitogenic, growth, proliferative and fibrotic effects of neuroendocrine factors, mediators of inflammation against the background of NO deficiency. |
| * To apply | Knowledge regarding the pathophysiological significance of markers of endothelial dysfunction and vascular remodeling in diagnostic and prognostic terms, as well as in establishing personalized pathogenetic treatment. |
| * To integrate | Knowledge regarding the etiology and pathogenesis of endothelial dysfunction and vascular remodeling in understanding and mastering the material covered in clinical disciplines. |
| **Topic (chapter) 2. Pathophysiology of essential hypertension** | |
| * To define | Essential or primary arterial hypertension (HTA) as a pathophysiological pattern of circulatory dyshomeostasis, as a clinical entity of cardiovascular diseases, as well as as an independent cardiovascular risk factor. Diagnostic landmarks of HTA in accordance with the European Society of Cardiology Guidelines. |
| * To know | Causal factors and main cardiovascular risk factors (modifiable and non-modifiable) that lead to sustainable elevation of blood pressure and the evolution of HTA. Mechanisms of elevation of blood pressure as a result of the incompetence of the endothelial system to control basal vascular tone determined preferentially by NO and PGI2 deficiency. Mechanisms of HTA triggered by activation of the sympatho-adrenergic system, the renin-angiotensin-aldosterone system and excess endothelin 1 (ET-1).  Pathogenetic contribution of the imbalance of Ang 1-7/Ang II in vasoconstriction and elevation of blood pressure.  Pathogenetic contribution of genetic polymorphism of HTA in the name of the basic pathophysiological components. Pathophysiological connection between vascular remodeling, HTA and the impact on target organs: brain, eyes, myocardium and kidneys.  Functional and biochemical markers for estimating pathophysiological patterns of endothelial dysfunction and vascular remodeling for predicting the evolution of HTA and strengthening personalized pathogenetic treatment. |
| * To demonstrate | Knowledge of the contemporary concept of the etiology and pathogenesis of HTA, as well as its risk on target organs in particular and on homeostasis in general. Pathogenetic scheme of the contribution of HTA in the development of hypertensive heart, myocardial hypertrophy and diastolic heart failure with preserved ejection fraction.  Knowledge of the role of HTA in the installation of the phenomenon of vascular remodeling and remodeling of the extracellular matrix of the heart.  Skills to understand the connection between endothelium-dependent peripheral vascular reactivity, pathological vascular remodeling and the evolution of HTA.  Skills to understand the pathogenetic contribution of dyslipidemia and diabetes mellitus in the onset and exacerbation of the evolution of HTA.  Skills to understand the pathogenetic role of HTA in the evolution of heart failure, ischemic cardiopathy and vascular accidents. |
| * To apply | Knowledge regarding the pathophysiological significance of functional and biochemical markers of disruption of homeostasis of basal vascular tone control in estimating the risk of HTN evolution, the prognosis of target organ damage, as well as the foundation of personalized pathogenetic treatment. |
| * To integrate | Knowledge regarding the etiology and pathogenesis of HTN in understanding and mastering the theoretical and practical aspects of circulatory dyshomeostasis, as well as the benchmarks of the subject matter covered in clinical disciplines. |
| **Topic (chapter) 3. Pathophysiology of pulmonary arterial hypertension** | |
| * To define | Pulmonary arterial hypertension (PAH) according to the latest conceptual consensus presented at the 6th World Symposium on Theoretical and Practical Issues of Pulmonary Hypertension regarding the value of mean pulmonary pressure (>20 mm Hg) and Wood index (>3 units). Principles of classification of pathophysiological patterns of PAH. Pathophysiological entity of cor pulmonale syndrome and right ventricular remodeling as repercussions of the aggravated evolution of PAH. |
| * To know | The main causal factors leading to the elevation of pulmonary arterial pressure. Mechanisms of PAH in the context of pathophysiological patterns of PAH: hereditary, idiopathic, related to left ventricular failure, related to per se damage to the pulmonary system (eg, pneumosclerosis, chronic obstructive pulmonary disease), determined by embolism and obstruction of the pulmonary arteries, mixed.  The pathophysiological contribution of NO and PGI2 deficiency, excess of endothelin 1 (ET-1) and thromboxane A2 (TxA2), hypoxia and pathological remodeling of precapillary arterioles and metarterioles.  Pathogenetic mechanisms of hypoxia-induced pulmonary arterial vasoconstriction.  Pathogenetic mechanisms of the evolution of *cor pulmonale* and the imminent consequences for the left ventricle.  Functional and biochemical markers for estimating pathophysiological patterns of PAH for the prediction of the risk of cor pulmonale and the consolidation of personalized pathogenetic treatment. |
| * To demonstrate | Knowledge of the contemporary concept of the etiology and pathogenesis of the pathophysiological patterns of PAH, as well as the main repercussions: the evolution of cor pulmonale, left ventricular remodeling and failure. Knowledge of the pathogenetic contribution of pulmonary endotheliocytes, smooth myocytes and pulmonary artery fibroblasts in the context of the impact of hypoxia, inflammation and oxidative stress.  Knowledge of the role of transforming growth factor (TGF-β) and marrow morphogenetic receptor protein 2 (BMPR2) in the evolution of the hereditary pattern of PAH.  Skills to understand the pathogenetic connection of NO and PGI2 deficiency on the one hand and ET-1 and TxA2 excess on the other hand in PAH evolution.  Skills to understand the important pathogenetic interface of PAH reinforced by the high pro-apoptotic activity of endothelial cells in contiguity with increased proliferative activity, which results in the obliteration of pulmonary arterioles. |
| * To apply | Knowledge regarding the pathophysiological significance of functional and biochemical markers of pulmonary artery and left ventricular remodeling in the diagnosis of pathophysiological patterns of PAH, prediction of *cor pulmonale* evolution and establishment of pathogenetic treatment based on PGI2 analogues and IP receptor agonists, ET-1 ETA receptor blockers, phosphodiesterase-5 inhibitors, cGMP stimulators. |
| * To integrate | Knowledge regarding the etiology and pathogenesis of PAH in understanding and mastering the theoretical and practical aspects of circulatory dyshomeostasis, as well as the benchmarks of the subject matter covered in clinical disciplines. |
| **Topic (chapter) 4. Pathophysiology of diastolic failure** | |
| * To define | The pathophysiological entity of diastolic heart failure (DHF).  The classical algorithm of factors and preconditions leading to impaired cardiac lusitropic function.  The DHF paradigm with preserved ejection fraction. |
| * To know | Pathophysiological patterns of DHF.  Mechanisms of myocardial hypertrophy development and the contribution of its concentric pattern in the evolution of DHF and the decrease in minute volume against the background of normal ejection fraction.  Mechanisms of the increase in the interstitial fibrosis space of the myocardium (reactive fibrosis) and its impact on the dysregulation of diastolic relaxation and the evolution of DHF.  Mechanisms of the metabolic impact on diastolic relaxation. The pathogenetic contribution of energy deficiency, incompetence of the sarcoplasmic reticulum calcium pump (SERAC2a), dephosphorylation of phospholamban and activation of oxidative stress.  Mechanisms of the neuroendocrine impact on diastolic relaxation. The pathogenetic contribution of the activation of the renin-angiotensin-aldosterone system and the role of aldosterone in myocardial fibrosis.  Mechanisms of the ischemic impact on the dysregulation of diastolic relaxation. The pathogenetic contribution of substitutive fibrosis (post-infarction), stunned and hibernated myocardium in the evolution of DHF. |
| * To demonstrate | Knowledge of the contemporary concept of DHF pathophysiology, as well as the integration of compromised diastolic relaxation in the impairment of left ventricular pump function.  Knowledge of the pathogenetic contribution of concentric hypertrophy in reducing cardiac output associated with a normal value of ejection fraction.  Knowledge of the algorithm of functional indices of central and peripheral hemodynamics in DHF.  Skills to understand the pathogenetic interface based on 3 key components: extracellular matrix remodeling, functional feasibility of cardiomyocytes and impaired myocardial energy support in the evolution of DHF.  Skills to interpret the pathophysiological significance of biomarkers of myocardial fibrosis (eg, galectin 3, tenascin, extracellular matrix metalloproteinases and their tissue-specific inhibitors) as a pathogenetic factor of DHF. |
| * To apply | Knowledge of the pathophysiology of impaired diastolic relaxation and myocardial remodeling, as well as of imminent functional and biochemical markers in the diagnosis and prognosis of DHF evolution.  Knowledge of the evaluation of pathophysiological patterns of DHF in consolidating pathogenetic treatment guidelines. |
| * To integrate | Knowledge regarding the etiology and pathogenesis of DHF in understanding and mastering the theoretical and practical aspects of circulatory dyshomeostasis, as well as the benchmarks of the subject matter covered in clinical disciplines. |
| **Topic (chapter) 5. Pathophysiology of cardiorenal syndrome** | |
| * To define | The pathophysiological entity of cardiorenal syndrome (CRS).  Pathophysiological patterns of CRS depending on the cardiac or renal central element. |
| * To know | Mechanisms of the evolution of acute cardiorenal syndrome with the central cardiac landmark.  Mechanisms of the evolution of chronic cardiorenal syndrome with the central cardiac landmark.  Mechanisms of the evolution of acute cardiorenal syndrome with the central renal landmark.  Mechanisms of the evolution of chronic cardiorenal syndrome with the central renal landmark.  Mechanisms of the evolution of acute cardiorenal syndrome with the concomitant mixed cardiac and renal landmark.  The pathophysiological contribution of neuroendocrine activation (sympathoadrenergic system and renin-angiotensin-aldosterone system), pro-inflammatory cytokines, oxidative stress and renal congestion in the CRS. |
| * To demonstrate | Knowledge able to appreciate the pathophysiological significance of functional markers and biochemical markers in the context of the diagnosis of CRS.  Skills in understanding and interpreting the functional test to estimate the renal filtration rate in primary renal pathology or secondary to heart disease.  Skills in authentic interpretation of changes in serum creatinine and cystatin C levels imminent to vascular endothelial damage.  Skills in estimating the diagnostic and prognostic role of microalbuminuria in patients with CRS.  Skills in estimating the predictive role of renal dysfunction in chronic heart failure by determining in urine the tubular markers: neutrophil gelatinase-associated lipocalin (NGAL), N-acetyl-beta-glucosaminidase (NAG) and kidney injury molecule (KIM). |
| * To apply | Knowledge of the pathophysiology of acute or chronic renal damage induced by acute or chronic heart failure.  Knowledge of the pathophysiology of acute or chronic cardiac damage induced by acute or chronic kidney failure.  Knowledge of the significance of pathophysiology in strengthening the diagnostic and prognostic algorithm based on functional and biochemical markers inherent to cardiac and renal pathology, as well as pathogenetic treatment guidelines. |
| * To integrate | Knowledge regarding the etiology and pathogenesis of CRS in understanding and mastering the theoretical and practical aspects of circulatory dyshomeostasis, as well as the benchmarks of the subject matter covered in clinical disciplines. |

1. **SPECIFIC PROFESSIONAL COMPETENCES (CS) AND TRANSVERSAL (TC) AND STUDY finalIties**

**Professional competences (specific) (PC)**

• PC1. Application of the values ​​and norms of professional ethics, as well as the provisions of the legislation in force in the field of responsible approach and execution of professional objectives and tasks.

• PC2. Thorough knowledge of natural medical disciplines regarding the morpho-functional peculiarities and adaptive-compensatory reactivity inherent in the homeostasis of the human body, its dysfunction, including the circulatory system and cardiovascular pathologies, as well as the interface of the relationship between the state of health and the physical and social environment.

• PC3. Consolidation of the conceptual and technical-methodological algorithm in order to solve various problems and clinical situations in the connotation of the diagnosis of cardiovascular diseases and consolidation of the principles and benchmarks of pathogenic treatment, as well as emergency medicine maneuvers.

• PC4. Knowledge and application of benchmarks for promoting a healthy lifestyle, as well as prevention and self-care measures.

• PC5. The ability to integrate the specialist into interdisciplinary activity, effectively using the conceptual and technical-methodological potential.

• PC6. Understandable approach and implementation of current scientific research in the field of physiology of circulatory homeostasis and cardiovascular pathophysiology.

1. **Transversal competences (tC)**

* TC1. Training of professional skills regarding autonomy and responsibility of activity in the field of solving the approaches and imminent demands of the optional discipline Pathophysiology of the Cardiovascular System.

1. **Finalities of study**

• To know the physiological entity of circulatory homeostasis and the physiopathological landmarks of the main cardiovascular diseases.

• To be able to justifiably indicate the performance of current functional and biochemical investigations.

• To correctly interpret the results of functional and biochemical investigations.

• To know the particularities of pathogenetic mechanisms and the methodology of diagnosing

pathologies of the cardiovascular system.

• To know the role of clinical, laboratory and instrumental investigations in the diagnosis of

cardiovascular system diseases.

• To know the indications, contraindications and limits of certain functional explorations.

• To be able to deduce the interrelationships between fundamental and clinical cardiology and other

medical disciplines (nephrology, endocrinology, hepatology) performing multidisciplinary integration.

• To be able to daily master new achievements in cardiovascular pathophysiology.

• Be able to extrapolate the arrangements learned in molecular and cellular research of the cardiovascular

system to the knowledge obtained in the teaching process, and vice versa, in order to consolidate a

conclusive theoretical-practical approach in the field of pathophysiology and cardiology.

**Note. Finalities of discipline** (are deduced from the professional competencies and formative values ​​of the informational content of the discipline).

1. **individual work of student**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Nr. | Expected product | Achievement strategies | Evaluation criteria | Completion date |
| 1. | Working with informational sources | Reading the material presented in the course and in the textbook on the respective topic.  Highlighting the topics in the topic that require reflection.  Getting acquainted with the list of additional information sources on the respective topic.  Formulating generalizations and conclusions regarding the importance of the addressed topic. | The ability to extract the essential.  Skills in interpreting the main pathogenetic landmarks.  The volume of material acquired. | During the course |
| 2. | Working with sources *on-line* | Studying online materials from database sites and specialized literature. | The quality and conceptual depth of the presentation of information in practical work. | During the course |
| 3. | Applying of different learning techniques |  | Efficiency of the concentric and eccentric model of research and learning of the material.  Feasibility of transposing the evidence of scientific research assisted within the Center for "Molecular Medicine" to the entity of the studied subject.  Establishment of the diagnostic algorithm of cardiovascular diseases and individualized pathogenetic treatment. | During the course |
| 4. | Preparing and giving presentations on various topics of the pathophysiology of the cardiovascular system. | Selection of presentation topics and deadlines.  Strengthening the research algorithm and designating its phasing. | The degree of mastery of the essence of the subject addressed.  The depth of the research, its competitiveness and the level of scientific argumentation of the consolidated postulates. The creativity of solving the objectives and the formation of the personal concept. The iconography of the presentation. | During the course |

**XII. METHODOLOGICAL SUGGESTIONS FOR TEACHING-LEARNING-ASSESSMENT**

***• Used teaching and learning methods***

The optional discipline *Pathophysiology of the Cardiovascular System* is taught in a classical manner: with lectures and seminars/practical works. The theoretical course is taught by the course instructors. Along with traditional methods, modern methods are used: lecture-conference, lecture-debate and with exegesis of a situation or clinical case problem, which are oriented towards the efficient acquisition and achievement of the set objectives. In the practical works, forms of individual, frontal, group activity, virtual solving of the clinical case are used. In the lessons and extra-curricular activities, information communication technologies are used - Power-Point presentations, video, etc. The use of theoretical and practical skills obtained in the research at the molecular and cellular level of circulatory homeostasis and cardiovascular pathologies in the center "Molecular Medicine" under the umbrella of the principle of Training through Research, for the deepening and thorough acquisition of the subject material.

***• Applied teaching strategies/technologies (subject-specific)***

"Problem-based learning"; "Research-based learning"; "Brainstorming"; "Multi-voting"; "Round table"; "Group interview"; "Case study"; "Creative controversy"; "Focus group technique", "Portfolio"; "Role playing".

***• Evaluation methods*** *(including indication of the method of calculating the final grade)*

**Current**:

• (a) administering tests,

• (b) solving problems,

• (c) analyzing case studies,

• (e) control evaluations.

**Final**: exam in SIMU.

**How to round off grades at evaluation stages**

|  |  |  |
| --- | --- | --- |
| Intermediate grade grid (annual average, grades from exam stages) | National grading system | Equivalent  of 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 grade and the grades of all stages of the final examination (computer-assisted, testing, oral response) - all will be expressed in numbers according to the grading scale (according to the table), and the final grade obtained will be expressed in a number with two decimal places, which will be entered in the gradebook.

Failure to appear for the exam without good reason is recorded as “absent” and is equivalent to a grade of 0 (zero). The student has the right to 2 repeated sittings of the exam without passing.

**recommanded Bibliografy:**

*A. Mandatory:*

1. Peter Libby, Robert O. Bonow, Douglas L. Mann, Gordon F Tomaselli, Deepak Bhatt, Scott D Solomon, Eugene Braunwald. Braunwald’s Heart Disease: A Textbook of Cardiovascular Medicine.  [Elsevier](https://medbookvn.com/chu-de/elsevier). 2022. Edition 12. 1227 p.
2. Carmen Ginghină, Dragoş Vinereanu, Bogdan A. Popescu. Manual de cardiologie al Societății Române de cardiologie. 2020. București, Editură medical, 1028 p.
3. Чазова И.Е., Терещенко А.С., Меркулов Е.В. Кардиология, учебник для ВУЗов. ГЭОТАР-Медиа, 2024. 920 стр.

*B. Supplementary*

1. Cobet V., Todiras M., Rotaru V. Pathophysiology of essential arterial hypertension: mechanisms and predictors. University course*.* 2020. 56 p.

2. Cobeț V., Rotaru V. Pathophysiology of left heart failure. Methodical support. Chisinau, 2017, 40 p.

3. Bivol E., Grib L., Sasu B. Sindromul cardiorenal. Arta medica, 2016, 1(58), p.20-25.