APPROVED BY
The decision of the Academic Council of the Institute of Medicine, Ecology and Physical Culture of UlSU
of «18» May 2022, Record № 9/239
Chairman _____ Midlenko V.I.
(signature, signature clarification)
«18» May 2022

THE EVALUATION FUNDS (EFS) OF THE DISCIPLINE

Discipline	Pathophysiology of extreme states
Faculty	Medical named of T.Z.Biktimirov
Department	Physiology and pathophysiology
Course	3

Direction (speciality)_____31.05.01. «General medicine» _____
(Course code (speciality), full name)
Orientation (profile/specialty) not provided_____

The form of traning_____full-time_____

Date of introduction into the academic process at Ulyanovsk State University «1» September 2022.

Revised at the Department meeting, Record No. 13 of 27 « 06 » 2023.
Revised at the Department meeting, Record No. 10 of 16 « 05 » 2024.
Revised at the Department meeting, Record No._____ of_____ «_____» 20___.
Revised at the Department meeting, Record No._____ of_____ «_____» 20___.

Information about developers:

Initials	Department	Degree, scientific rank
Antoneeva I.I.	Physiology and pathophysiology	Professor, Doctor of medical science
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AGREED	AGREED
Head of department of physiology and pathophysiology developing discipline _____/Tatyana P. Gening/ *Signature* *Full name* « 18 » April 2022	Head of the graduation department of hospital therapy _____/Marina A.Vize-Khripunova/ *Signature* *Full name* « 11 » May 2022

THE LIST OF COMPETENCIES ON THE SUBJECTS (MODULES) FOR STUDENTS IN THE DIRECTION OF TRAINING (SPECIALTY) INDICATING STAGE OF THEIR FORMATION IN THE PROCESS OF MASTERING THE MAIN PROFESSIONAL EDUCATIONAL PROGRAM

No se-mester	Name of discipline (module) or practices	Index of competence	
		UC-1	PC-1
1	Bioethics	+	
3	Philosophy	+	
2,3	Biochemistry	+	
9	Occupational diseases	+	
1	Physics mathematics	+	
1	Chemistry	+	
7	Modern aspects of neurology	+	
B	Topical issues of hospital surgery	+	
C	Surgical gastroenterology and endoscopy	+	+
C	Polliative medicine	+	
9	Clinical pathological anatomy	+	
A	Diabetology and emergency endocrinology	+	+
6	Pathophysiology of extreme states	+	+
5	Nanotechnology in medicine	+	
C	Current issues of HIV infection	+	
C	Clinical electrocardiography	+	
B	Emergency Medicine		+
B	Anesthesiology, resuscitation and intensive care		+
5	Extreme medicine		+
2	Surgical care		+
4	Introductory practice		+
6	Nurse Assistant		+
6	Practice for obtaining professional skills and experience of professional activity in the positions of paramedical person-nel		+
8	Hospital Physician Assistant		+
C	Preparation for passing and passing the state exam	+	+

2. Requirements for the results of practical training

№	Competence index	Content of a competence (or a part of it)	The proposed results of the course students are:		
			to know	to be able to	to have skills in
1.	UC-1	Able to carry out a critical analysis of problem situations based on a systematic approach, develop an action strategy	- basic concepts of general nosology; - the role of the causes, conditions, reactivity of the organism in the occurrence, development and completion (outcome) of extreme conditions; - about the unity of damage and protective-adaptive reactions, as well as their variability at different stages of extreme conditions; - pathological changes of different levels - from micro- to macroscopic in extreme conditions; - functional reserves of the patient, the level of compensation for dysfunctions in extreme conditions	- to carry out a pathophysiological analysis of clinical, laboratory, experimental, and other data and formulate a conclusion based on them; - substantiate the principles of pathogenetic therapy of extreme conditions;	- skills of presentation of an independent point of view, analysis and logical thinking; - skills in analyzing the patterns of functioning of individual organs and systems in normal and pathological conditions; - skills of analysis and interpretation of the results of modern diagnostic technologies;
2.	PC-1	Willingness to participate in the provision of emergency medical care in conditions requiring urgent medical intervention	Willingness to participate in the provision of emergency medical care in conditions requiring urgent medical intervention	Be able to make a diagnosis based on pathophysiological analysis of specific data on pathological processes, condi-	Possess skills for early diagnosis of diseases

				tions, reactions and diseases;	

3. The passport Fund of assessment tools in practice

№	Controlled sections (stages)	Index of controlled competence (or its parts)	Evaluation tools evaluation		Technology (method of control)
			name	№№ task	
1.	**Pathophysiology of extreme conditions**	UC-1	Issues of current control during practical work (to know)	1-72	the interview
			Questions for the credit (to know)	1-39	the interview
			Questions for the exam (to know)	1-56	the interview
			Test tasks (to know)	1-19	testing
2 .	**Pathophysiology of syndromes developing in extreme conditions**	PC-1	Issues of current control during practical work (to know)	73-109	the interview
			Questions for the credit (to know)	40-54	the interview
			Questions for the exam (to know)	20-37	the interview
			Test tasks (to know)	57-83	testing

4. Evaluation tools for interim certification

4.1. Tests (test tasks) for the current control and monitoring of independent work of students

Index of competency	№ test	Test
Section 1. Pathophysiology of extreme conditions.		
UC-1	1.	Pathological physiology studies 1) structural features 2) functions of the main systems and organs 3) manifestations of diseases 4) general patterns of occurrence, development, course and outcomes of diseases.

UC-1	2.	Nosology is 1) the doctrine of the causes of the disease 2) the general doctrine of the disease 3) the doctrine of the mechanisms of occurrence, development and outcomes of the disease 4) the doctrine of the mechanisms of recovery
UC-1	3.	The latent period of infectious diseases is called 1) incubation period 2) predisease 3) prodromal period 4) peak period
UC-1	4.	The appearance of nonspecific signs of the disease is typical for: 1) latent period 2) prodromal period 3) incubation period 4) the height of the disease 5) the outcome of the disease.
UC-1	5.	Indicate the correct sequence of the main stages of dying 1) pre-agony, terminal pause, agony, clinical death, biological death 2) terminal pause, preagony, agony, clinical death, biological death 3) preagony, agony, terminal pause, clinical death, biological death 4) pre-agony, terminal pause, agony, biological death, clinical death 5) pre-agony, agony, clinical death, terminal pause, biological death.
UC-1	6.	The doctrine of the causes and conditions of the onset of disease is called 1) nosology 2) pathogenesis 3) etiology 4) pathology
UC-1	7.	The cause of the disease is 1) contributing to the onset of the disease; 2) which causes the disease and gives it specific features; 3) determining the nonspecificity of the disease 4) affecting the frequency of occurrence of the disease; 5) affecting the severity and duration of the disease
UC-1	8.	Heredity plays a role in etiology 1) only the causes of the disease 2) only disease conditions 3) causes and conditions of the disease 4) only contributing to the onset of the disease 5) only preventing the onset of the disease
UC-1	9.	Specify non-enzymatic factors of antioxidant protection of cells: 1) divalent iron ions

		2) SOD 3) vitamin D 4) vitamin C
UC-1	10.	In the pathogenesis of neurosis, there are: 1) neurophysiological, neurochemical, psychophysiological aspects 2) psychological, neurochemical, psychophysiological, neurophysiological aspects 3) neurochemical, psychophysiological, psychological, biological aspects 4) biological, psychological, social aspects 5) neurophysiological, psychological, neurochemical, social aspects
UC-1	11.	The most common manifestations of disorders of the cardiovascular system in neurosis are 1) low cardiac output syndrome 2) cardialgic syndrome, vascular dystonia syndrome, shortness of breath 3) cardialgic syndrome and vascular dystonia syndrome 4) cardialgic and arrhythmic syndromes 5) arrhythmic syndrome, vascular dystonia syndrome, cardialgic syndrome
UC-1	12.	The state of adaptation to a substance, accompanied by the development of tolerance and the appearance of a withdrawal or withdrawal syndrome, is called 1) abuse 2) addiction 3) mental addiction 4) physical dependence 5) tolerance
UC-1	13.	The main mechanism of the pathogenic action of ethanol is 1) hepatotropic 2) psychotropic 3) cardiotoxic 4) enteropathogenic
UC-1	14.	Negative nitrogen balance in the body occurs 1) during pregnancy 2) during the growth of the body 3) with burn disease 4) with an excess of anabolic hormones
UC-1	15.	Ketone bodies are formed 1) in the intestine; 2) in the liver; 3) in the lungs; 4) in the kidneys; 5) in the muscles.

UC-1	16.	What is the maximum life expectancy of an adult during starvation without water under normal temperature conditions? 1) 2-4 days 2) 6-8 days 3) 10-12 days 4) 55-60 days
UC-1	17.	Hypotonic dehydration may be due to: 1) indomitable vomiting 2) a decrease in the production of vasopressin (ADH) 3) osmotic diuresis 4) an increase in the production of vasopressin (ADH)
UC-1	18.	Water intoxication develops when 1) excess water intake in the body 2) insufficient removal of water from the body 3) excessive intake of mineral salts 4) excessive intake of water into the body against the background of its insufficient excretion 5) forced use of sea water
UC-1	19.	Negative water balance is observed when 1) cirrhosis of the liver 2) heart failure 3) diabetes insipidus 4) nephrotic syndrome 5) acute diffuse glomerulonephritis
UC-1	20.	The cause of hyperosmolal dehydration may be 1) diarrhea 2) repeated vomiting 3) intestinal fistulas 4) diabetes 5) aldosterone deficiency.
UC-1	21.	Hyperosmolal dehydration is characterized by 1) an increase in the total water content in the body 2) a decrease in osmotic pressure in the extracellular fluid 3) an increase in the water content in the cells 4) the movement of water from cells into the extracellular space 5) lack of feeling of thirst
UC-1	22.	Hypoosmolar dehydration occurs when 1) diarrhea 2) diabetes 3) diabetes insipidus 4) esophageal atresia 5) water starvation.
UC-1	23.	When dehydrated, there is:

		1 - thickening of the blood; 2 - violation of microcirculation; 3 - increase in central venous pressure; 4 - lowering blood pressure; 5 - decrease in the volume of circulating blood 1) 1, 3 2) 1, 2, 4, 5 3) 1, 2, 3 4) 1, 5 5) 4, 5
UC-1	24.	Hypoosmolar dehydration occurs when 1) prolonged vomiting 2) diabetes 3) diabetes insipidus 4) esophageal atresia 5) water starvation.
UC-1	25.	Hypoosmolar dehydration is characterized 1) an increase in the total water content in the body 2) a decrease in osmotic pressure in the extracellular fluid 3) a decrease in the water content in the cells 4) the movement of water from cells into the extracellular space 5) a painful feeling of thirst
UC-1	26.	Clinical sign of portal hypertension 1) cyanosis 2) ascites 3) jaundice 4) hypertension of the systemic circulation 5) acholia
UC-1	27.	Clinical sign of portal hypertension 1) cyanosis 2) acholia 3) expansion of the saphenous veins of the abdominal wall ("jellyfish head") 4) jaundice 5) hypertension of the systemic circulation
UC-1	28.	Hepatic coma 1) this is a condition characterized by impaired liver function and usually manifested by jaundice, hemorrhagic syndrome and neuro-psychiatric disorders 2) this is a state of deep depression of the function of the central nervous system, characterized by a loss of consciousness, loss of response to external stimuli and a disorder in the regulation of vital body functions, due to an extreme degree of liver failure
UC-1	29.	The development of encephalopathy in acute liver failure leads to 1) hyperglycemia

		2) hypocholia 3) acholia 4) paraproteinemia 5) endotoxemia
UC-1	30.	In the liver, in its insufficiency, 1) decrease in the synthesis of triglycerides 2) increased synthesis of triglycerides 3) increased cholesterol synthesis 4) decrease in the formation of ketone bodies 5) increase in the synthesis of phospholipids
UC-1	31.	With liver failure 1) increases the formation and deposition of glycogen 2) the formation and deposition of glycogen decreases 3) the conversion of galactose and fructose into glucose is enhanced 4) gluconeogenesis is activated 5) the formation of glucuronic acid is activated
UC-1	32.	Under the influence of stressors develops: 1. thymus involution; 2. adrenal hypertrophy; 3. hypoplasia of lymphoid tissue; 4. ulcerative lesions of the stomach and duodenum; 5. hyperplasia of lymphoid tissue 1) 1, 2, 3, 4 2) 1, 2, 4, 5 3) 1,2,3,4,5 4) 1,3,5
UC-1	33.	The correct sequence of stages of development of the general adaptation syndrome according to G. Selye: 1) anxiety stage, exhaustion stage, resistance stage 2) anxiety stage, resistance stage, exhaustion stage 3) exhaustion stage, anxiety stage, resistance stage 4) exhaustion stage, resistance stage, anxiety stage 5) resistance stage, anxiety stage, exhaustion stage
UC-1	34.	The resistance stage of the general adaptation syndrome is characterized by: 1) hyperplasia of the adrenal cortex. 2) atrophy of the anterior pituitary gland. 3) a persistent decrease in the level of corticotropin in the blood. 4) low level of glucocorticoids in the blood. 5) decreased activity of the sympathetic nervous system.
UC-1	35.	The main importance in the adaptation of the body under stress belongs to: 1) glucocorticoids 2) insulin 3) thyroxine

		4) aldosterone 5) growth hormone
UC-1	36.	The mechanisms for the implementation of the general adaptation syndrome are associated with hyperproduction: 1) antidiuretic hormone 2) oxytocin 3) androgens 4) glucocorticoids
UC-1	37.	The stress-limiting system includes: 1) Sympathoadrenal system. 2) Pituitary. 3) Adrenals. 4) GABAergic system
UC-1	38.	The extreme conditions are 1) preagony 2) agony 3) clinical death 4) biological death 5) coma
UC-1	39.	The extreme conditions are 1) preagony 2) agony 3) clinical death 4) biological death 5) traumatic shock
UC-1	40.	The most common cause of cardiogenic shock is 1) myocardial infarction 2) arterial hypotension 3) pericarditis 4) myocardiopathy 5) damage to the tricuspid valve
UC-1	41.	Changes in hemodynamics in the erectile stage of shock: 1) increase in the volume of circulating blood 2) increase in cardiac output 3) increase in blood flow velocity 4) drop in blood pressure 5) increased blood pressure 1) 1, 2, 3, 5 2) 1, 2, 3, 4 3) 1, 2, 3, 4, 5 4) 1, 4
UC-1	42.	Changes in hemodynamics in the torpid stage of shock: 1) increase in the volume of circulating blood

		2) increase in cardiac output 3) increase in blood flow velocity 4) drop in blood pressure 5) increased blood pressure 1) 1, 2, 3, 5 2) 1, 2, 3, 4 3) 1, 2, 3, 4, 5 4) 1, 4 5) 4
UC-1	43.	Pathological deposition of blood during shock is observed mainly in 1) heart 2) vessels of the abdominal organs 3) vessels of the lower extremities 4) lungs
UC-1	44.	Stress-limiting systems do not include: 1) complement system 2) opioid peptide system 3) serotonergic system 4) antioxidant systems
UC-1	45.	Maladaptive diseases do not include: 1) hypertension 2) glomerulonephritis 3) ischemic heart disease 4) gastric ulcer
UC-1	46.	What is the sequence of inclusion of pathogenetic links in the formation of stress? Make a pathogenetic chain: 1) increased secretion of ACTH by the anterior pituitary gland, the effect of a stressor on the body, activation of the production of corticoliberin in the hypothalamus, the release of glucocorticoids into the blood, the effect of glucocorticoids on target cells 2) the effect of a stressor on the body, increased secretion of ACTH by the anterior pituitary gland, activation of the production of corticoliberin in the hypothalamus, release of glucocorticoids into the blood, the effect of glucocorticoids on target cells 3) the effect of a stressor on the body, activation of the production of corticoliberin in the hypothalamus, increased secretion of ACTH by the anterior pituitary gland, release of glucocorticoids into the blood, the effect of glucocorticoids on target cells 4) the effect of a stressor on the body, the release of glucocorticoids into the blood, the effect of glucocorticoids on target cells, activation of the production of corticoliberin in the hypothalamus, increased secretion of ACTH by the anterior pituitary gland 5) increased secretion of ACTH by the anterior pituitary gland, activation of the production of corticoliberin in the hypothalamus, re-

		lease of glucocorticoids into the blood, the effect of glucocorticoids on target cells, the effect of a stressor on the body
UC-1	47.	At what stage of OSA (general adaptation syndrome) does hypertrophy of the adrenal cortex develop? 1) into the alarm stage 2) to the stage of resistance 3) in the stage of exhaustion
UC-1	48.	The content of which hormones does not increase in the blood during a stress reaction? 1) ACTH 2) glucocorticoids 3) adrenaline 4) androgens
UC-1	49.	Select manifestations that characterize the erectile phase of shock: 1) activation of the sympathetic-adrenal system 2) motor and speech stimulation 3) hyperventilation of the lungs 4) all answers are correct
UC-1	50.	Select manifestations that are not typical for the torpid phase of shock: 1) weakening the effects of the sympathetic-adrenal system 2) motor and speech stimulation 3) decrease in cardiac output 4) blood deposition
UC-1	51.	In the pathogenesis of coma matters: 1) hypoxia and energy deficiency of brain neurons 2) intoxication 3) violation of the acid-base state 4) violation of electrolyte homeostasis 5) all answers are correct
UC-1	52.	Indicate the correct sequence of development of disorders of the central nervous system in coma: 1) mental anxiety, confusion, co-por, deep loss of consciousness 2) deep loss of consciousness, stupor, confusion, mental anxiety 3) confusion, deep loss of consciousness, stupor, mental anxiety 4) mental anxiety, stupor, confusion, deep loss of consciousness 5) deep loss of consciousness, confusion, stupor, mental anxiety
UC-1	53.	Choose the correct statement: 1) coma always develops gradually, with distinct stages of impaired consciousness 2) coma can develop "with lightning speed", without a distinct staging of impaired consciousness

UC-1	54.	As a result of the reduction of blood circulation in shock, microcirculatory disorders are most pronounced in: 1) heart 2) stomach 3) brain 4) liver
UC-1	55.	An increase in nonspecific resistance under stress is due to: 1) mobilization and redistribution of energy resources 2) increasing the power and stability of the ion pumps 3) stabilization of cell membranes 4) all answers are correct
Section 2. Pathophysiology of syndromes developing in extreme conditions		
PC-1	56.	Meaning of pain: 1) pain warns and protects the body from harmful external influences 2) pain is one of the most common clinical symptoms 3) pain is a clinical symptom, a signal of danger, as well as a cause or component of the pathogenesis of various diseases and disease states 4) pain is a danger signal
PC-1	57.	Pain mediators include: 1) fibrinogen 2) bradykinin 3) erythropoietin 4) tryptophan
PC-1	58.	Excitation threshold of true nociceptors during inflammation and hypoxia: 1) goes up 2) is decreasing 3) does not change
PC-1	59.	Has no sensitivity to pain 1) mesentery 2) parietal peritoneum 3) lung tissue 4) parietal pleura 5) pericardium
PC-1	60.	Has a high sensitivity to pain 1) heart muscle 2) visceral pleura 3) lung tissue 4) pericardium
PC-1	61.	Epicritic pain is characterized by: 1) takes a long time

		2) has a diffuse character 3) occurs immediately after injury 4) occurs after a short period of time after injury 5) is clearly detected when internal organs are damaged
PC-1	62.	Protopathic pain is characterized by: 1) clearly localized 2) takes a long time 3) occurs immediately after injury 4) is accompanied by activation of the sympathetic nervous system 5) clearly expressed under the action of the algogenic factor on the skin, mucous membranes
PC-1	63.	The purpose of acupressure and acupuncture is usually: 1) activation of the nociceptive system 2) oppression of the nociceptive system 3) activation of the antinociceptive system 4) inhibition of the antinociceptive system
PC-1	64.	Pain resulting from damage to the somatosensory (peripheral or central) nervous system is called 1) chronic 2) nociceptive 3) thalamic 4) neuropathic 5) reflected
PC-1	65.	Acute, clearly localized and well described by the patient pain, which quickly regresses with the appointment of painkillers, is called 1) nociceptive 2) reflected 3) chronic 4) neuropathic 5) thalamic
PC-1	66.	Causalgia is characterized by: 1) episodes of severe, difficult to bear, debilitating polytopic pain, combined with autonomic, motor and psycho-emotional disorders 2) the appearance of pain in the missing part of the body 3) the appearance of nociceptive sensations caused by irritation of the internal organs, but localized not in it (or not only in it) itself, but also in remote superficial areas of the body 4) paroxysmal intensifying burning pain in the area of damaged nerve trunks
PC-1	67.	The starting point in pathogenesis is 1) secondary damage 2) exacerbation of the disease 3) transition to a chronic form 4) primary damage 5) the formation of a vicious circle
PC-1	68.	The vicious circle in the pathogenesis of diseases is: 1) the transition of the primary acute phase to the chronic form with periods of exacerbation and remission 2) the cyclic course of the disease, in which each new cycle differs from the previous one by a progressive increase in the severity of

		disorders 3) the emergence of a positive feedback between the individual links of pathogenesis, contributing to the progression of the disease
PC-1	69.	Specify the mechanisms of cell damage: 1) increased conjugation of oxidative phosphorylation 2) increased activity of enzymes of the DNA repair system 3) reduction of free radical lipid oxidation 4) the release of lysosomal enzymes into the hyaloplasm
PC-1	70.	Name the main reasons for the activation of phospholipases during cell damage: 1) increase in the intracellular content of sodium ions 2) increase in the intracellular content of calcium ions 3) decrease in the intracellular content of potassium ions 4) increase in intracellular water content
PC-1	71.	The consequence of lipid peroxidation in membranes is an increase 1) membrane permeability 2) surface tension 3) electrical strength of membranes 4) potassium in cells
PC-1	72.	Damage to lysosome membranes leads to 1) activation of tissue respiration 2) increase in hydrolysis processes in the cell 3) activation of protein synthesis 4) increase in membrane potential 5) apoptosis
PC-1	73.	The adaptive mechanisms of the cell include 1) rupture of mitochondrial cristae 2) decreased activity of respiratory enzymes 3) increased activity of lysosomal enzymes 4) hypertrophy and hyperplasia of intracellular structures
PC-1	74.	Thalamic pain is characterized by: 1) the appearance of nociceptive sensations localized by the patient in the missing part of the body 2) episodes of severe, difficult to bear, debilitating polytopic pain, combined with autonomic, motor and psycho-emotional disorders 3) paroxysmal intensifying burning pain in the area of damaged nerve trunks 4) the appearance of nociceptive sensations caused by irritation of the internal organs, but localized not in it (or not only in it) itself, but also in remote superficial areas of the body
PC-1	75.	A nociceptive sensation caused by irritation of the internal organs, but localized not in it (or not only in it) itself, but also in remote superficial parts of the body, is called - 1) nociceptive pain 2) pathological pain of central origin 3) neuropathic pain 4) projected pain 5) referred pain

PC-1	76.	The antinociceptive system is 1) generator of pathologically enhanced excitation 2) pain relief system 3) pain intensifying system 4) a system that ensures the formation of epicritical pain 5) a system that provides the formation of protopathic pain
PC-1	77.	The response of the acute phase is characterized by a decrease 1) albumin 2) fibrinogen 3) C-reactive protein 4) gamma globulins 5) serum amyloid A
PC-1	78.	The most important mediator of the acute phase response is 1) histamine; 2) platelet activating factor; 3) lymph node permeability factor; 4) interleukin-1
PC-1	79.	The correct statement is 1) acute phase response - predominantly local reaction of the body to damage 2) acute phase response - the general reaction of the body to damage 3) all manifestations of the acute phase response always have an exceptionally positive value for the body 4) the response of the acute phase is always accompanied by a decrease in the body's resistance 5) the acute phase response develops only when the body is damaged by mechanical factors
PC-1	80.	Causalgia is: (1)) pain projected to the areas of the amputated limb 2) excruciating pain that occurs after damage to a large somatic nerve 3) pain that occurs in certain areas on the surface of the skin with the development of pathology in the internal organs
PC-1	81.	Phantom Pain:(1) 1) is projected onto the areas of the amputated limb 2) painful and occurs after damage to a large nerve 3) occurs in certain areas of the skin surface during development
PC-1	82.	Illness is 1) an unusual reaction of the body to any effect 2) a combination of damage phenomena and protective-adaptive reactions in damaged tissues, organs or organism 3) persistent deviation from the norm, which has no adaptive value for the body 4) a qualitatively new state of the body, unlike health, that occurs when it is damaged by environmental factors and is characterized by a limited ability to adapt to environmental conditions and a decrease in working capacity

PC-1	83.	The syndrome is 1) the same signs of different diseases 2) a set of symptoms of a disease with a single pathogenesis 3) relapse of the disease 4) a set of symptoms of a disease of various pathogenesis 5)the most important symptoms of the disease

Evaluation criteria and scales:
- evaluation criteria – correct answers to questions;
- score – percentage of correct answers to questions;
- scale of assessment(assessment) – 4 levels of assessment of competences are allocated:
high - more than 80% of correct answers;
sufficient – from 79 to 70 % of correct answers;
satisfactory– from 69 to 60% of correct answers;
critical – less than 59% of correct answers.

Ключ к тестовым заданиям

Раздел, тема	№ практического (практических), семинарского (семинарских) занятия (занятий)	№ правильного ответа
Pathophysiology of extreme conditions	1.	Answer: 4
Pathophysiology of extreme conditions	2.	Answer:2
Pathophysiology of extreme conditions	3.	Answer:1
Pathophysiology of extreme conditions	4.	Answer:2
Pathophysiology of extreme conditions	5.	Answer:1
Pathophysiology of extreme conditions	6.	Answer:3
Pathophysiology of extreme conditions	7.	Answer:2
Pathophysiology of extreme conditions	8.	Answer:3
Pathophysiology of extreme conditions	9.	Answer:4
Pathophysiology of extreme conditions	10.	Answer:2
Pathophysiology of extreme conditions	11.	Answer:5
Pathophysiology of extreme conditions	12.	Answer:4
Pathophysiology of extreme conditions	13.	Answer:1
Pathophysiology of extreme conditions	14.	Answer:3
Pathophysiology of extreme conditions	15.	Answer:2
Pathophysiology of extreme conditions	16.	Answer:2
Pathophysiology of extreme conditions	17.	Answer:2
Pathophysiology of extreme conditions	18.	Answer:4
Pathophysiology of extreme conditions	19.	Answer:3
Pathophysiology of extreme conditions	20.	Answer:4
Pathophysiology of extreme conditions	21.	Answer:4
Pathophysiology of extreme conditions	22.	Answer:1
Pathophysiology of extreme conditions	23.	Answer:3
Pathophysiology of extreme conditions	24.	Answer:1
Pathophysiology of extreme conditions	25.	Answer:2
Pathophysiology of extreme conditions	26.	Answer:2

Pathophysiology of extreme conditions	27.	Answer:3
Pathophysiology of extreme conditions	28.	Answer:2
Pathophysiology of extreme conditions	29.	Answer:5
Pathophysiology of extreme conditions	30.	Answer:1
Pathophysiology of extreme conditions	31.	Answer:2
Pathophysiology of extreme conditions	32.	Answer:1
Pathophysiology of extreme conditions	33.	Answer:2
Pathophysiology of extreme conditions	34.	Answer:1
Pathophysiology of extreme conditions	35.	Answer:1
Pathophysiology of extreme conditions	36.	Answer:4
Pathophysiology of extreme conditions	37.	Answer:4
Pathophysiology of extreme conditions	38.	Answer:5
Pathophysiology of extreme conditions	39.	Answer:5
Pathophysiology of extreme conditions	40.	Answer:1
Pathophysiology of extreme conditions	41.	Answer:1
Pathophysiology of extreme conditions	42.	Answer:5
Pathophysiology of extreme conditions	43.	Answer:2
Pathophysiology of extreme conditions	44.	Answer:1
Pathophysiology of extreme conditions	45.	Answer:2
Pathophysiology of extreme conditions	46.	Answer:3
Pathophysiology of extreme conditions	47.	Answer:2
Pathophysiology of extreme conditions	48.	Answer:4
Pathophysiology of extreme conditions	49.	Answer:4
Pathophysiology of extreme conditions	50.	Answer:1
Pathophysiology of extreme conditions	51.	Answer:5
Pathophysiology of extreme conditions	52.	Answer:1
Pathophysiology of extreme conditions	53.	Answer:2
Pathophysiology of extreme conditions	54.	Answer:4
Pathophysiology of extreme conditions	55.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	56.	Answer:3
Pathophysiology of syndromes developing in extreme conditions	57.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	58.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	59.	Answer:3
Pathophysiology of syndromes developing in extreme conditions	60.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	61.	Ответ:3
Pathophysiology of syndromes developing in extreme conditions	62.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	63.	Answer:3
Pathophysiology of syndromes developing in extreme conditions	64.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	65.	Answer:1

Pathophysiology of syndromes developing in extreme conditions	66.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	67.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	68.	Answer:3
Pathophysiology of syndromes developing in extreme conditions	69.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	70.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	71.	Answer:1
Pathophysiology of syndromes developing in extreme conditions	72.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	73.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	74.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	75.	Answer:5
Pathophysiology of syndromes developing in extreme conditions	76.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	77.	Answer:1
Pathophysiology of syndromes developing in extreme conditions	78.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	79.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	80.	Answer:2
Pathophysiology of syndromes developing in extreme conditions	81.	Answer:1
Pathophysiology of syndromes developing in extreme conditions	82.	Answer:4
Pathophysiology of syndromes developing in extreme conditions	83.	Answer:2

4.2. Set of check lists for the current control and monitoring of independent work of students

Index of competency	№	Check list
UC-1	**1.**	Patient T., 48 years old, was taken to the emergency department with complaints of severe pain behind the sternum, radiating to both shoulder blades, not relieved by nitroglycerin. Over the past 10 days, he has noted intermittent chest pain of lesser intensity and duration. On examination: the condition is severe, the skin is pale, covered with sweat. Pulse - 120 beats / min., weak tension, arrhythmic. BP - 85/40 mm Hg. Art. Heart sounds are muffled. Respiratory rate - 28 per minute. Vesicu-

		lar breathing in the lungs. The liver is not enlarged, there are no edema. When talking with a doctor, he suddenly lost consciousness, while blood pressure dropped to 70/30 mm Hg. Art. After the urgent administration of cardiotonic drugs, the patient regained consciousness. ECG: in I, II, aVL, V2–V6 leads, the ST segment is displaced arcuately upwards, a negative T wave is recorded in the same leads. Questions: 1. What is your preliminary diagnosis? How to characterize the severity of the patient at the time of examination? 2. List the types (severity) of cardiogenic shock.
UC-1	**2.**	Medical college student K., 16 years old, attending a surgical operation for the first time, suddenly experienced a feeling of "lightheadedness", which was accompanied by tinnitus, dizziness, nausea and led to loss of consciousness. Objectively: the skin is very pale, the extremities are cold to the touch. The pupils are constricted. Heart sounds are muffled. Pulse - 40 per minute, weak filling. BP - 70/30 mm Hg. Art. Breathing is rare. Spraying her face with cold water and inhaling ammonia vapor quickly brought the patient to consciousness. Questions: 1. What pathology do these symptoms indicate? 2. What are the mechanisms of development of this condition? 3. What are the main causes of this pathology?
UC-1	**3.**	Patient Zh., aged 52, was admitted to the pulmonology department with bilateral pneumonia. Got sick 5 days ago. Objectively: the patient's condition is moderate. Body temperature - 40.2 ° C. The boundaries of the heart are expanded, the tones are muffled. A systolic murmur is heard at the apex. BP - 105/70 mm Hg. Art. Pulse - 105 beats / min, weak filling. Above the lower lobes of the right and left lungs, the percussion sound is dull, fine bubbling rales, crepitus are heard. Antibacterial therapy was prescribed. The patient sweated profusely at night. Body temperature dropped to normal in the morning. The condition worsened sharply, dizziness and nausea appeared. The pulse became thready, blood pressure dropped. The patient lost consciousness. Emergency drug therapy allowed to bring the patient out of this state. Questions: 1. What pathology do these symptoms indicate? 2. What is its pathogenesis? 3. List the types and main factors of the pathogenesis of this pathology?
UC-1	**4.**	Patient R., aged 52, was taken to the hospital in a state of uremia. The patient is adynamic, drowsy. The face is puffy, the skin is dry, flabby with traces of multiple scratching. There is shortness of breath with an increase in the phase of inhalation and exhalation, an increase in the rhythm. On the 4th day of hospital stay, despite the measures taken, deterioration occurred: a coma developed, the reaction of the pupils to light was sluggish, the patient was unconscious. There was a kind of noisy rapid breathing, in which deep breaths are evenly replaced by large exhalations. Questions:

		1. What form of respiratory disorder appeared in the patient? 2. Will dyspnea persist in a comatose patient? Justify the answer.
UC-1	**5.**	Patient K., 31 years old, was taken to the clinic by ambulance. On admission: passive, lethargic, apathetic, not always immediately and adequately answers questions. Coated tongue. Temperature - 36.5 ° C. The skin and mucous membranes are icteric, there are telangiectasias on the skin of the upper part of the body, erythema of the palms is noted. The abdomen is enlarged due to ascitic fluid, which makes it difficult to palpate the liver. Edema of the lower extremities is noted. The border of the left ventricle of the heart is somewhat enlarged. BP - 160/95 mm Hg. Art., heart rate - 90 per minute, rhythmic pulse. The results of a biochemical blood test: hyperbilirubinemia, hypoglycemia, hypoproteinemia, hypocholesterolemia, the urea content is reduced, the prothrombin index is lowered. The activity of ALT and AST in the blood is increased. The general blood test was within the normal range. Questions: 1. What are the mechanisms of development of telangiectasias and persistent erythema of the palms in a patient? What other symptoms are caused by the same effect? 2. What are the main causes of portal hypertension and ascites development? What is the role of ascites in secondary disorders of body functions? 3. Are there laboratory signs of liver failure? If yes, what is the mechanism of their development? 4. How can you characterize the state of consciousness in this patient?
UC-1	**6.**	Patient R., 39 years old, was admitted to the renal center in a severe precomatous state: marked weakness, apathy, pain in muscles and joints, skin itching, ammonia smell from the mouth were noted. It was found out that he had been suffering from kidney diseases since the age of 26. Objectively determined: edema on the legs, face, congestive enlarged liver. BP - 190/120 mm Hg. Art. Residual blood nitrogen - 148 mmol / l. Glomerular filtration by endogenous creatinine — 12.0 ml/min. Zimnitsky's test: with a daily diuresis of 360 ml, fluctuations in relative density of 1.003-1.007. Questions: 1. What type of renal failure and what stage does the patient have? 2. Are there signs of uremia in the patient? 3. Due to what substances did residual blood nitrogen increase?
UC-1	**7.**	In patient Z., 26 years old, shortly after the flu, edema, oliguria, proteinuria increased, and hematuria appeared. From the anamnesis, it was possible to establish that the patient had edema, proteinuria, and headache for several previous years. Clinical and laboratory examination reveals: residual blood nitrogen - 57 mmol / l, urea - 16.6 mmol / l, plasma creatinine - 200 μmol / l. Glomerular filtration by endogenous creatinine -28 ml/min. Zimnitsky's test: fluctuations in the relative density of urine 1.003–1.008 with a daily diuresis of 350 ml. Questions: 1. What type and what stage of renal failure does the patient have?

		2. How to explain the decrease in glomerular filtration in this type of insufficiency?
UC-1	**8.**	Patient D., 68 years old, suffering from chronic hepatitis and cirrhosis of the liver, underwent a puncture of the abdominal cavity to remove ascitic fluid. At the 15th minute of the procedure, after removing 5 liters of fluid, the patient complained of weakness, dizziness and nausea, but the procedure was continued. After removing another 1.5 liters of fluid, the patient lost consciousness. A few minutes after the emergency care, consciousness was restored, but the patient still complains of severe weakness, dizziness, and nausea. Questions 1. What was the doctor's mistake during the procedure for this patient? 2. What are the causes and mechanisms of syncope development in case of rapid removal of ascitic fluid? 3. What are the possible mechanisms for compensating for circulatory disorders in the brain in such a situation? 4. Why were the compensatory mechanisms of the circulatory system in this patient ineffective?
UC-1	**9.**	Patient Ch., 20 years old, a graduate of the institute, upon admission to the clinic, presented various complaints: poor sleep, irritability, tearfulness, lack of appetite, unstable mood, headaches. Somatic status without deviations from the norm. When studying the history of the development of the disease: the phenomena indicated at admission developed in Ch. during the last 10 months. During this period, the patient had a very difficult situation: an unsuccessful marriage and the need to leave for distribution (which she really did not want to do because of her lack of confidence in her abilities, and also because of the fear of losing contact with her husband). During her stay in the department, the patient constantly complained to the staff and demanded special attention. After each meal, vomiting occurred (usually in the presence of patients and staff). Questions 1. What is the origin of the symptom complex that develops in the patient? 2. In what type of higher nervous activity do such disorders develop more often?
UC-1	**10.**	Patient S., aged 42, grew up in a family where the main task in life was to achieve personal success and a position in society, although study was given to him with great difficulty. Wanting to satisfy the ambitions of his parents and his own, he strove to surpass his comrades in studies, but he spent great efforts on this. After school (at the request of his parents) he entered the institute. Studying at the institute required even more effort. He worked hard, often at night. After graduating from the institute, he went to work at the plant as a shift foreman. As soon as the position of the head of the shop was vacated, he began to actively seek it, despite the fact that the profile of the shop did not correspond to the specialty he received at the institute. In addition, he did not have sufficient organizational experience. When he became

		the foreman, he faced great difficulties. The workshop led by him ceased to fulfill production tasks, which caused complaints and criticism from the administration and the staff of the workshop. It was during this period that he developed headaches, pain in the region of the heart, insomnia, irritability, fatigue, decreased efficiency. On examination: BP 170/90 mm Hg, pulse 90. Focal neurological symptoms were not established. Questions 1. What caused pain in the heart area, tachycardia and arterial hypertension? 2. What form of pathology of the nervous system has developed in S.?
UC-1	**11.**	Patient K., 28 years old, went to the doctor with complaints of frequently recurring stomatitis, tonsillitis, tracheitis, otitis media, periodically pneumonia, including in summer. Attempts to increase the activity of the "non-specific resistance" of the body by hardening the effect did not give. Laboratory data: the reaction of lymphocytes to phytohemagglutinin and tuberculin is positive; activity of complement factors and levels of IgM, IgG, IgA in blood serum are normal; in the blood, the content of erythrocytes and Hb is within the normal range, the number of leukocytes is reduced due to monocytes. The number of granulocytes is normal. The phagocytic activity of macrophages is reduced by 45%. Questions and task: 1. In what part of the system of immunobiological surveillance of the patient's body is there a defect: in the immune system or in the system of non-specific protection factors? Justify your answer. 2. If there is a defect in the immune system, then due to the defeat of which of its cells: macrophages, B- or T-lymphocytes? How then to explain the origin of the symptoms present in K.? 3. If this is a defect in the system of factors of nonspecific defense of the organism, then which (or which) of them exactly? How would you explain the mechanisms of symptoms in K? Justify your opinion. 4. Formulate a general conclusion about the form of pathology that the patient has.
UC-1	**12.**	Patient D., 35 years old, was admitted to the clinic with complaints of severe paroxysmal pain (burning sensation) in the epigastric region, occurring 2–3 hours after eating; Recently, pain has been accompanied by nausea and sometimes vomiting. Vomiting brings relief to the patient. Pain also appears at night, in connection with which the patient wakes up and takes food "on an empty stomach." In this case, the pain disappears quite quickly. The patient is emotional, irritable; smokes a lot and abuses alcohol. On the basis of complaints, as well as the results of the examination of the patient, a diagnosis of duodenal ulcer was made and appropriate treatment was prescribed, which significantly alleviated his condition. Questions 1. Based on the results of what studies did the doctor make a diagnosis and prescribe an effective treatment? 2. What factors could cause this disease and what mechanisms underlie their action? Justify your answer based on your history.

		3. What are the reasons for the sensation of pain (burning) in the epigastric region? 4. What are your recommendations for the treatment of patient D.?
UC-1	**13.**	Patient B., 26 years old, was admitted to the intensive care unit of the hospital in serious condition with a deep stab wound to the forearm. He was injured 1.5 hours ago, since then he has lost about 1 liter of blood. There is pallor of the skin and visible mucous membranes, cold sweat. Pulse -96 beats / min, blood pressure - 75/40 mm Hg. Breathing is frequent and deep; rhythmic. 1. Determine the type of abnormal breathing in the examined patient. 2. What is the mechanism of violation of the function of external respiration?
UC-1	**14.**	Patient K., aged 27, was admitted to the clinic in a severe coma. There was a progressive difficulty in breathing, in connection with which he was transferred to artificial respiration. In the study of CBS, it was found: pH = 7.30, pCO2 = 60 mm Hg. BB = 52 mmol/l, SB = 28 mmol/l, BE = +6 mmol/l. 1. Explain whether there is a need for further artificial lung ventilation?
UC-1	**15.**	Patient T., aged 56, was admitted for examination with a diagnosis of diabetes mellitus. On the 6th day of hospital stay, the patient's condition deteriorated sharply; there was a smell of acetone in the exhaled air; tongue dry, furred. Pulse - 110 beats / min of weak filling, blood pressure - 80/50 mm Hg. In this case, noisy rapid breathing is observed, in which, after a deep breath, an increased exhalation follows with the active participation of the expiratory muscles. 1. Determine the type of respiratory disorders in the patient. 2. What are the causes and mechanisms of this type of respiratory disorder?
UC-1	**16.**	Kolya V..2 years; sent to the hospital in a serious septic condition on the background of purulent otitis media. Blood test upon admission: erythrocytes - 3.9 * 10^{12} / l, hemoglobin -121 g/ l, color index - 0.93, platelets - 250 * 10^{9} / l, leukocytes - 42 * 10^{9} / l. Leukocyte formula: promyelocytes-0.5%, myelocytes-3%, metamyelocytes-5.5%, bands-13%, segmented-54%, eosinophils-1%, basophils-0.5%, monocytes-4%, lymphocytes-18.5%. In the smear: normochromia, reticulocytes-0.4%, toxic granularity of neutrophils. Questions: 1). What do the shifts in the parameters of the neutrophilic germ of hematopoiesis indicate in a patient? 2). Is it possible, on the basis of the presented data, to suggest the causes of hematological changes?
UC-1	**17.**	Patient B., aged 36, was taken to the clinic in serious condition. Confusion of consciousness, lethargy are noted; body temperature -39.4°C. The liver and spleen are sharply enlarged. Blood test at admission: erythrocytes - 4.3 * 10^{12} / l, hemoglobin - 122 g / l,

		color index - 0.85, platelets - 150 * 10^9 / l, leukocytes - 45 * 10^9 / l. Leukocyte formula: metamyelocytes-0, bands-0, segmented-4%, eosinophils-0, basophils-0, monocytes-3%, lymphocytes-93%. In the smear: normochromia, reticulocytes-0.7%. Single lymphoblasts, Gumprecht-Botkin shadows. Questions: 1) For what pathology of the blood system is the presented hemogram typical? 2) What is the likely nature of the detected hematological changes?
UC-1	**18.**	Patient A., aged 49, was sent to the clinic for examination for pain in the bones and joints of unknown origin. A blood test at admission showed: erythrocytes - 3.4 * 10^{12}/ l, hemoglobin - 110 g / l, color index - 0.97, platelets - 250 * 10^9/ l, leukocytes - 18 * 10^9 / l. Leukocyte formula: myeloblasts-2%, promyelocytes-7%, myelocytes-12%, metamyelocytes-22%, bands-20%, segmented-14%, eosinophils-6%, basophils-2%, monocytes-4%, lymphocytes-11%. In the smear: normochromia, moderately pronounced aniso-, poikilocytosis, reticulocytes-0.6%. Questions: 1) What pathology does the presented blood test indicate?
UC-1	**19.**	Patient D., aged 48, was taken to the intensive care unit in an unconscious state. Pulse - 120 beats / min, weak filling, blood pressure - 90/50 mm Hg. Kussmaul breathing. Pungent odor of acetone in exhaled air. Tongue dry, furred. Blood test at admission: erythrocytes - 4.5 * 10^{12}/ l, hemoglobin - 138 g/l, color index - 0.92, platelets - 280 * 10^9/l, leukocytes - 12 * 10^9/l, ESR - 16 mm / h. Fibrinogen in blood plasma - 3.5 g / l, prothrombin time - 24 s, plasma recalcification time - 95 s, thrombotest - 4 points, plasma tolerance to heparin - 15 min. Blood glucose - 18.4 mmol / l. Questions: 1) Analyze the presented clinical and laboratory data. 2) Describe the state of hemostasis in the patient.
PC-1	**20.**	Patient A., aged 50, was admitted to the intensive care unit with complaints of pressing pain behind the sternum, weakness, shortness of breath, lasting 20 hours. On examination: a state of moderate severity, hyperemia of the face. On auscultation in the lungs, breathing is vesicular, there are no wheezing. Respiratory rate - 16 per minute, heart sounds are muffled, rhythmic. Heart rate - 80 beats / min. BP -130/85 mm Hg. Art. On the ECG: sinus rhythm, deepened Q wave and ST segment elevation in the first lead with a mirror image in lead III. The activity of AcAT, MB-CPK and LDH in the blood is sharply increased. Leukocytes - 12.3 x10^9/l. Platelets - 450.0 x10^9/l. Prothrombin index - 120% (norm up to 105%). Questions: 1. The development of what disease is evidenced by the changes described in the problem?

		2. In what part of the heart is the pathological process localized? 3. How would you explain the increase in blood ACT activity in this form of pathology? 4. What are the main syndromes characteristic of this disease that the patient has developed? 5. What is the possible cause of the development of the disease?
PC-1	**21.**	Patient V., 35 years old, was taken to the emergency room in a serious condition with complaints of sharp tearing pain behind the sternum, weakness. Accompanying the patient reported that he became ill in the tram, he suddenly turned pale, covered with a cold sweat. Tram passengers took him to the hospital. The doctor seated the patient, began to count the pulse and measure blood pressure. The patient is pale, the skin is moist, covered with droplets of sweat. Pulse - 100 beats / min, weak filling and tension. BP - 80/40 mm Hg. Art. The doctor had not yet finished the examination, as the patient lost consciousness. At the same time, the pulse and pressure were not determined. The pupils dilated, did not react to light, single respiratory movements were noted, cyanosis rapidly increased. The ECG recorded in lead I looked like a wavy line. The doctor and assistants began resuscitation measures: external cardiac massage, mouth-to-mouth breathing, intravenous administration of lidocaine. However, despite the ongoing treatment, the patient, without regaining consciousness, died. No pathology that could cause death was found in the section. Coronary arteries - no changes, no focal changes in the myocardium. Questions: 1. Based on the clinical presentation and autopsy findings, what do you think is the immediate cause of death? 2. Were mistakes made in helping the patient?
PC-1	**22.**	Patient T., 48 years old, was brought to the emergency department with complaints of severe pain behind the sternum, radiating to both shoulder blades, not relieved by nitroglycerin. Over the past 10 days, he has noted intermittent chest pain of lesser intensity and duration. On examination: the condition is severe, the skin is pale, covered with sweat. Pulse - 120 beats / min., weak tension, arrhythmic. BP - 85/40 mm Hg. Art. Heart sounds are muffled. Respiratory rate - 28 per minute. In the lungs, respiration is vesicular. The liver is not enlarged, there are no edema. When talking with a doctor, he suddenly lost consciousness, while blood pressure dropped to 70/30 mm Hg. Art. After the urgent administration of cardiotonic drugs, the patient regained consciousness. ECG: in I, II, aVL, V2–V6 leads, the ST segment is displaced arcuately upwards, a negative T wave is recorded in the same leads. Questions: 1. What is your preliminary diagnosis? How to characterize the degree of severity of the patient at the time of examination? 2. List the types (severity) of cardiogenic shock.
PC-1	**23.**	Patient V., aged 10, complains of general weakness, headaches, loss of appetite, thirst. A history of frequent sore throats. At clinical and laboratory

		examination, there is a lag in physical development, the skin is pale, dry, flaky. BP - 130/90 mm Hg. Art. Blood tests show mild anemia. Blood urea - 8.9 mmol/l. The daily amount of urine is 6-8 times a day, night urination takes place. Straw-yellow urine, transparent, acid reaction, fluctuations in relative density 1.009-1.017, protein - 0.2 g/l. In the sediment: a small amount of epithelium, leukocytes - 0-2 in the field of view, erythrocytes, hyaline casts - single in the preparation. The glomerular filtration rate for insulin is 50 ml/min. Questions: 1. Are there signs of renal failure in the patient? 2. Does the patient have nocturia? 3. Have you received evidence of pollakiuria?
PC-1	**24.**	Patient V., 30 years old, was mistakenly transfused with 150 ml of blood of another group. A typical picture of hemotransfusion shock, severe anuria, developed. Vigorous anti-shock therapy was immediately started: exchange transfusion, hemodialysis. Gradually the patient's condition improved. For the 8th day from the moment of shock, the patient excreted 4.5 liters of light urine, the relative density of which was 1.008–1.012. Urine contains a large amount of protein, erythrocytes, leukocytes, epithelial cells. Residual blood nitrogen - 34 mmol / l, urea - 12 mmol / l. Questions: 1. What type and what stage of renal failure does the patient have? 2. What is the mechanism of polyuria in this case?
PC-1	**25.**	Patient I., aged 26, was taken to the clinic in an unconscious state. According to her husband, after the flu, the patient developed thirst, weight loss, poor appetite, abdominal pain, weakness, and headache. The day before, there was pain in the abdomen, repeated vomiting, confusion. On examination: there is no consciousness, Kussmaul's breathing, the smell of acetone from the mouth, signs of dehydration - the skin is dry, pale, cold, the tongue is dry, covered with a brown coating. Pulse - 120 beats / min; small filling and tension, blood pressure - 95/60 mm Hg. Art. The abdomen is soft, painless; blood sugar level - 21 mmol/l, hyperketonemia, blood pH - 7.0. Questions: 1. What disease can we talk about? 2. Describe the patient's condition at the time of admission. 3. What is the pathogenesis of hyperketonemia in this pathology? 4. List the main links in the pathogenesis of coma in this pathology.
PC-1	**26.**	Patient V., 36 years old, was taken to the clinic in an unconscious state. The patient at home developed mental and motor excitement, after which she lost consciousness. According to the neighbors, the patient has been suffering from diabetes for many years, is treated with insulin, eats irregularly due to constant business trips. On examination: there is no consciousness, the skin is moist, there is a twitching of the muscles of the face, the pupils are dilated; respiratory rate - 32 per minute, pulse - 70 per minute, rhythmic, blood pressure - 130/80 mm Hg. Art.; the level of glucose in the blood - 2.45 mmol / l.

		Question: What is the name of the condition that developed in the patient?
PC-1	**27.**	What symptoms are typical for diabetic ketoacidosis (A) and hypoglycemic state (B): 1) abdominal pain; 2) nausea; 3) vomiting; 4) feeling of hunger; 5) lack of appetite; 6) disorientation; 7) anxiety state; 8) jitter; 9) apathy; 10) indifference; 11) dry skin; 12) skin moisture; 13) normal breathing; 14) deep breathing; 15) skin, muscles are flabby; 16) pupils are narrowed; 17) pupils are dilated; 18) tachycardia; 19) arterial hypotension; 20) hypo-, areflexia; 21) hyperketonemia; 22) hyperglycemia; 23) acetonuria; 24) hypoglycemia; 25) alkaline blood reserve is normal; 26) the alkaline reserve of the blood is reduced.
PC-1	**28.**	A 46-year-old patient was admitted to the clinic due to extensive trauma (multiple bone fractures, soft tissue injuries), accompanied by massive blood loss. Upon admission, consciousness is inhibited, the skin is pale, cold, covered with sweat. BP - 95/60 mm Hg. Art. Pulse - 120 beats / min. Severe shortness of breath, thirst. Oliguria. In the study of CBS, the following data were obtained: pHart. blood = 7.26; paCO2 = 28 mm Hg. Art.; HCO3- = 14.5 mmol/l; BE = -12 mmol/l; SB = 14 mmol/l; lactate = 6.8 mmol/l. Make a conclusion about the nature of the violations of the acid-base state.
PC-1	**29.**	Patient E., 27 years old, was admitted to the clinic due to chills, fever up to 40 ° C, complaints of bloody discharge from the uterus, bruising at the injection site, and profuse nasal and gingival bleeding. Doctors suspected a criminal abortion, which was later confirmed by a gynecological examination, and, as a result, sepsis.

		Blood tests revealed: anemia, leukocytosis, hyperregenerative shift of the leukocyte formula to the left, toxic granularity of leukocytes, platelets - 21x109/l, ESR - 45 mm per hour, hyperbilirubinemia. From the blood and the uterine cavity during bacteriological examination, St. aureus. By the end of the first day of hospital stay, the patient developed acute renal failure (oligo-anuria, azotemia, edema), profuse bloody vomiting was noted. Data of laboratory diagnostics: APTT - 115 s, the content of FV, FVIII, fibrinogen, ATIII (antithrombin III) is reduced, the amount of fibrin degradation products is increased. Questions: 1. What type of hemostasis disturbances developed in patient E. against the background of a criminal abortion? 2. What link of pathogenesis is leading in the development of this pathology? 3. What syndromes are revealed in patient E.? 4. Give a detailed explanation of the changes in laboratory values that are taking place. 5. Name the principles of treatment of this disorder of hemostasis.
PC-1	**30.**	Patient I., 3 years old, had a fever up to 39 o C, bloody diarrhea appeared. A day later, oliguria with proteinuria, azotemia, and petechial intradermal hemorrhages developed. There was bloody vomiting, nosebleeds. Examination of feces revealed the presence of Shigella dysenteriae. In the blood: erythrocytes - 2 x 1012 / l, Hb - 30 g / l, ESR - 15 mm per hour, reticulocytes - 2%, L - 1.8 x 109 / l, platelets - 23 x 109 / l. VC -13 min, APTT - 38 s, PT - 14 s. Questions: 1. Name the type of hemostasis disorders. 2. What mechanism of hemostasis is impaired? Justify your answer using history and laboratory data. 3. Describe the causes of the pathology, the links of its pathogenesis. 4. What therapeutic measures will be effective in this violation of hemostasis?
PC-1	**31.**	An ambulance doctor at the scene of the accident, to relieve pain shock in a victim with a combined closed chest injury and an open fracture of the lower limb, after applying a tourniquet to it and stopping severe bleeding, injected a large dose of anesthetic (morphine) s / c, as well as cardiac stimulants activities. Despite the introduction of morphine, the victim groans loudly from pain, complains of a feeling of lack of air. During the examination: BP 70/35 mm Hg, pulse 126, determined only on large vascular trunks, breathing in the left half of the chest is weakened, not heard on the right. In front of the doctor's eyes, the victim's condition continues to deteriorate: breathing becomes shallow, he catches air in his mouth, cannot take a breath. To activate respiration, the doctor introduced a s/c stimulator of the respiratory center - cytiton. However, even after that, the victim's condition did not improve. Questions 1. Is it possible to assume that the ineffectiveness of the anesthetic is associ-

		ated with an increased tolerance of the victim to morphine? If so, was the introduction of the drug justified at all? 2. Based on the ideas about the pathophysiological reactions of the body in the conditions of the development of posthemorrhagic shock, which of them could cause the ineffectiveness of drug therapy in this situation? 3. The ineffectiveness of the doctor's therapeutic measures to restore breathing is due only to insufficient absorption of cytiton when it is administered s/c Do you think that the ineffectiveness of the doctor's therapeutic actions to restore breathing is due only to insufficient absorption of cytiton when it is administered s/c?
PC-1	**32.**	60-year-old patient M. was delivered to the emergency department of the hospital in an unconscious state. On examination: the skin is dry, the skin turgor and the tone of the eyeballs are lowered, the breathing is shallow, the pulse is 96, the blood pressure is 70/50 mm Hg, the tongue is dry; convulsions of the limbs and mimic muscles of the face are periodically observed. Express blood test: hyperglycemia - 600 mg%, hyperazotemia, hypernatremia, pH 7.32. From a survey of a relative who accompanied the patient, it turned out that he M. had diabetes. In this regard, he took small doses of oral hypoglycemic agents. In the last month he suffered from an exacerbation of chronic cholecystitis and colitis; often there were vomiting, diarrhea; complained of constant thirst and excretion of large amounts of urine. Questions: 1. What is the name of the condition in which the patient is brought to the hospital? Justify the answer. 2. What caused this condition? Name and characterize the main links of its pathogenesis. 3. Why is consciousness lost during the development of such pathological conditions? 4. What methods are used to bring patients out of such conditions?
PC-1	**33.**	Patient A., aged 50, was admitted to the intensive care unit with complaints of pressing pain behind the sternum, lasting about 15 hours. On examination: a state of moderate severity. There is hyperemia of the face. Auscultation in the lungs revealed vesicular breathing, no wheezing. The frequency of respiratory movements is 16 per minute, the heart sounds are muffled, rhythmic. HR 80. BP 180/100 mm Hg. ECG: sinus rhythm, deep Q wave and ST segment elevation in lead 1 with its mirror image in lead III. The activity of AST in the blood is significantly increased. Leukocytes $12.0x10^9$/l. Platelets $450.0x10^9$/l. Prothrombin index 120% (norm up to 105%). Questions 1. The development of what form of pathology is evidenced by the changes described in the task? 2. In what region of the heart is the pathological process localized? 3. How would you explain the increased activity of AST in the blood in this form of pathology? 4. What is the main mechanism of development of this form of pathology?

PC-1	**34.**	Describe the data below in medical terms. Based on the analysis of the data, formulate and justify the conclusion about the form of kidney pathology. TABLE 1. Diuresis 420 ml 2. Density 1.011 3. Protein 2 g/l 4. No glucose 5. Ketone bodies no 6. Sediment microscopy: leached erythrocytes 25-30 per field of view; hyaline, waxy and granular casts. 7. BP — 175/98 mm Hg. 8. Residual blood nitrogen 190 mg%
PC-1	**35.**	In patient M., 2 weeks after suffering a sore throat, edema began to appear under the eyes, especially after sleep. Urinalysis: daily diuresis 750 ml, beats. weight 1.028, protein - 0.1%, sugar and acetone are absent. Microscopy of urine sediment: erythrocytes, including leached ones, 10–26 per field of view; hyaline and erythrocyte cylinders in a small amount. BP 180/110 mm Hg Blood test: residual nitrogen - 60 mg%, total protein - 7.3%. Clearance of endogenous creatine - 50 ml / min. Questions 1. For what form of pathology are the signs of the disease found in patient M. characteristic? 2. What is the initial link in the pathogenesis of this form of pathology? 3. What are the mechanisms of development of azotemia and arterial hypertension in patient M.?
PC-1	**36.**	Boy N. 2 years ago was diagnosed with "Acute diffuse glomerulonephritis". Currently, weakness, headaches, dizziness, and mild widespread edema are noted. Urinalysis: daily diuresis 3100 ml, beats. weight 1.008, protein 0.2%, sugar and acetone are absent. Microscopy of urine sediment: single erythrocytes in the field of view, hyaline cylinders in a small amount. BP 180/100 mmHg Blood test: residual nitrogen 90 mg%, total protein 5.9 g%. Clearance of endogenous creatine 40 ml/min. Questions 1. Does the presence of polyuria contradict the diagnosis of "Acute diffuse glomerulonephritis" made 2 years ago? 2. What are the mechanisms of development of polyuria and hypostenuria in this situation. 3. What is the significance of the polyuria developed in the patient?
PC-1	**37.**	Patient A, 24 years old, was taken to the intensive care unit with an extensive stab wound of the thigh. BP - 80/40 mm Hg; pulse - 94 beats / min, rhythmic. Clinical and laboratory data. Blood: Er- 3.4 x 10^{12}/l; Hb - 98 g/l; Color index - 0.86; Leukocytes - 8.3 x 10^{9}/l; Platelets - 210 x 10^{9}/l; ESR - 15 mm / h, Urea - 13.5 mmol/l (norm: 2.5-8.3 mmol/l), creatinine - 185 μmol/l. Daily diuresis 249 ml. 1. Determine the type and stage of renal failure in the patient? 2. What is the mechanism for reducing daily diuresis.

Evaluation criteria and scales:
- evaluation criteria –performing a skill according to the checklist;
- score – percentage of correct stapes of the check-list;
- scale of assessment(assessment) – 4 levels of assessment of competences are allocated:
high - more than 80% of correct answers;
sufficient – from 79 to 70 % of correct answers;
satisfactory - from 69 to 60 % of correct answers
critical – less than 59% of correct answers.

4.3. Questions for the current control in the performance of workshops

Index of competency	№	Questions
Section 1. Pathophysiology of extreme conditions		
UC-1	1.	Stress as a non-specific reaction of the body to injury. Types of stress.
UC-1	2.	Causes and main ways of realization of stress reaction.
UC-1	3.	The role of the neuroendocrine system in the development of the stress response.
UC-1	4.	General adaptation syndrome, stages, mechanism of development.
UC-1	5.	Pathogenetic features of emotional stress.
UC-1	6.	The main stress-limiting systems of the body, their role in modulating the stress response.
UC-1	7.	Protective-adaptive and pathogenic significance of the stress reaction, implementation mechanisms.
UC-1	8.	The role of stress in the development of cardiovascular diseases (hypertension, coronary artery disease, myocardial infarction).
UC-1	9.	The role of stress in the pathogenesis of diseases of the gastrointestinal tract (gastric and duodenal ulcer, nonspecific ulcerative colitis).
UC-1	10.	The role of stress in the development of secondary immunodeficiency, mental, oncological and other diseases.
UC-1	11.	Starvation, definition of the concept, types.
UC-1	12.	Endogenous and exogenous causes of starvation.
UC-1	13.	Fasting periods, changes in metabolism and physical functions during different periods of fasting.
UC-1	14.	Absolute, complete, incomplete, partial starvation, their characteristics.
UC-1	15.	Protein starvation, causes, mechanism of development, manifestations, consequences.
UC-1	16.	Factors that determine the duration of fasting.
UC-1	17.	Clinical manifestations of starvation, mechanism of development.
UC-1	18.	The concept of therapeutic fasting.
UC-1	19.	Factors that determine the intensity of energy metabolism in the body.
UC-1	20.	Pathology of energy metabolism, causes and mechanism of development. Clinical manifestations.
UC-1	21.	Infectious process, definition of the concept.
UC-1	22.	Infectious process as one of the forms of interaction between macro- and microorganisms.

UC-1	23.	The etiology of the infectious process, the concept of exo- and endotoxins.
UC-1	24.	Factors that determine the virulence of microorganisms.
UC-1	25.	The role of external conditions and reactivity of the organism in the occurrence, development and outcome of the infectious process.
UC-1	26.	Mechanisms of anti-infective resistance of the organism.
UC-1	27.	Entrance gates of infection, ways of spreading an infectious agent in a macroorganism.
UC-1	28.	Stages of the course of the infectious process, their characteristics.
UC-1	29.	General patterns of development of infectious diseases (inflammation, fever, hypoxia, etc.)
UC-1	30.	Mediators of infectious-allergic reactions (the role of cytokines, lymphokines, monokines, complement components, etc.), their mechanism of action.
UC-1	31.	Violation of the function of organs in the infectious process.
UC-1	32.	Nonspecific and specific humoral and cellular mechanisms of anti-infective protection.
UC-1	33.	Principles of therapy of infectious process.
UC-1	34.	Septic shock, definition, etiology and mechanisms of development.
UC-1	35.	Mediators of septic shock, their mechanism of action.
UC-1	36.	Action of endotoxins and exotoxins on target cells.
UC-1	37.	Violation of the state of the hemostasis system in septic shock.
UC-1	38.	Principles of etiopathogenetic treatment of septic shock and emergency care.
UC-1	39.	Anaphylactic shock, definition, causes, mechanisms of development.
UC-1	40.	Mediators of anaphylactic shock, their mechanism of action.
UC-1	41.	Clinical variants of the course of anaphylactic shock, their characteristics.
UC-1	42.	Mechanisms of formation of pathological changes in anaphylactic shock, determination of the severity of the course.
UC-1	43.	Etiopathogenetic therapy and prevention of anaphylactic shock.
UC-1	44.	Collapse, definition, classification. Fainting.
UC-1	45.	Characteristics of the main types of collapse, development mechanism, clinical manifestations.
UC-1	46.	Etiological and pathogenetic principles of treatment.
UC-1	47.	Shock, definition, etiopathogenetic classification, mechanism of development of the main syndromes in shock.
UC-1	48.	Mechanisms of damage to organs and systems in shock, the formation of "shock lungs", "shock kidneys".
UC-1	49.	Traumatic shock, etiology, stages of development.
UC-1	50.	The role of neuroendocrine and humoral mechanisms in the development of traumatic shock.
UC-1	51.	Clinical manifestations of traumatic shock, assessment of its severity.
UC-1	52.	Syndrome of prolonged crushing, causes, mechanism of development.
UC-1	53.	Cardiogenic shock, etiology.
UC-1	54.	Mechanism of development of cardiogenic shock. The role of a decrease in myocardial contractility, vascular insufficiency, impaired microcirculation, pain syndrome, etc. in the development of cardiogenic shock.
UC-1	55.	Clinical manifestations of cardiogenic shock. Etiopathogenetic principles of treatment.

UC-1	56.	Coma, definition of the concept, types. Etiology of coma, main pathogenetic factors of development.
UC-1	57.	Metabolic coma, definition of the concept, classification.
UC-1	58.	Ketoacidotic coma, etiology, mechanism of development, clinical manifestations, pathogenetic principles of treatment.
UC-1	59.	Hyperosmolar coma, causes, mechanism of development, clinical manifestations, ways of prevention and pathogenetic therapy.
UC-1	60.	Lactacidic coma, causes, mechanism of development, clinical manifestations, pathogenetic therapy.
UC-1	61.	Hypoglycemic coma, causes, mechanism of development, clinical manifestations and pathogenetic therapy.
UC-1	62.	Chlorhydropenic coma, causes, mechanism of development, clinical manifestations and pathogenetic therapy.
UC-1	63.	The main mechanisms that provide the function of GNI.
UC-1	64.	General characteristics, causes and mechanisms of development of the pathology of higher nervous activity.
UC-1	65.	Violation of higher nervous activity. General characteristics of the causes and mechanisms of the pathology of higher nervous activity.
UC-1	66.	"Neurosis", definition, concept.
UC-1	67.	Methods of reproduction of experimental neuroses.
UC-1	68.	Classification of neuroses (classical neuroses).
UC-1	69.	Reasons for the development of neuroses and factors contributing to their development.
UC-1	70.	Anxiety-phobic states (obsessive-compulsive disorder), causes, formation of psychological conflict, clinical manifestations, stages of development. Types of phobias.
UC-1	71.	Hysterical neurosis, factors contributing to the development, the formation of psychological conflict, clinical manifestations.
UC-1	72.	Neurasthenia, causes, mechanism of development, formation of psychological conflict, clinical manifestations.
Section 2. Pathophysiology of syndromes developing in extreme conditions		
PC-1	73.	Pain as an integrative response of the body to injury.
PC-1	74.	The biological significance of pain as a signal of danger and damage.
PC-1	75.	Nociceptive stimuli and mechanisms of their perception.
PC-1	76.	Receptor, conduction and central links of the pain apparatus.
PC-1	77.	Humoral pain factors.
PC-1	78.	The pathogenesis of acute and chronic pain.
PC-1	79.	The concept of "physiological" and "pathological" pain.
PC-1	80.	Endogenous mechanisms of pain suppression.
PC-1	81.	Pathophysiological basis of anesthesia.
PC-1	82.	Visceral pain, definition of the concept, mechanism of development.
PC-1	83.	Reflected pain, definition of the concept, mechanism of development.
PC-1	84.	Phantom pain, definition of the concept, mechanism of development.
PC-1	85.	Vegetative compensation of pain reactions.
PC-1	86.	Acute renal failure, definition, concepts.
PC-1	87.	Prerenal, renal and postrenal factors in the development of acute renal failure.
PC-1	88.	Pathogenesis of acute renal failure, clinical manifestations, stages of devel-

		opment, their characteristics and mechanisms of development.
PC-1	89.	The main syndromes in acute renal failure.
PC-1	90.	Etiopathogenetic principles of treatment of acute renal failure.
PC-1	91.	Chronic kidney disease (CKD), definition of the concept.
PC-1	92.	Principles and stages of CKD development, their characteristics.
PC-1	93.	Etiology and mechanism of development of CKD.
PC-1	94.	The role of hyperinsulinism, secondary hyperparathyroidism, changes in blood lipid profile in the pathogenesis of CRF.
PC-1	95.	The pathogenesis of uremic coma.
PC-1	96.	Mechanisms of progression of chronic kidney disease.
PC-1	97.	Principles of modern nephroprotective therapy.
PC-1	98.	Multiple organ failure, definition (MON). Etiology, classification.
PC-1	99.	Phases of development of multiple organ failure.
PC-1	100.	PON mediators, their mechanism of action.
PC-1	101.	The role of cytokines IL-1, IL-6, TNF in the development of PON as first-order mediators.
PC-1	102.	Formation of the systemic inflammatory response syndrome (SIRS).
PC-1	103.	Clinical manifestations of SIRS, stages of development, their characteristics.
PC-1	104.	The concept of primary and secondary multiple organ failure.
PC-1	105.	Markers of "survival" in multiple organ failure.
PC-1	106.	Prognostic signs of multiple organ failure. Assessment of the severity of the condition in multiple organ failure.
PC-1	107.	The general pattern of formation and the sequence of involvement of body systems in the syndrome of multiple organ failure.
PC-1	108.	Metabolic reactions to systemic damage (proteins, carbohydrates, lipids). Clinical and laboratory indicators of hypermetabolism in multiple organ failure.
PC-1	109.	Violation of the function of organs and systems in the syndrome of multiple organ failure (cardiovascular, respiratory system, liver, kidneys, gastrointestinal tract, etc.).

4.4.Abstract for the control of self-independent work of students – is not provided
4.5. Essay to control the self- independent work of students - is not provided
4.6. Exam questions - is not provided
4.7.Cases for the exam - is not provided

4.8. Questions and tasks (tasks) to offset

Index of competency	№	The task
Section 1. Pathophysiology of extreme conditions.		
UC-1	1.	Extreme states, definition of the concept, general characteristics, types. Mechanisms of development of extreme conditions.
UC-1	2.	Stress as a non-specific reaction of the body to injury. Types of stress. Causes of stress.
UC-1	3.	The role of neurohumoral factors in the formation of adaptive and compensatory reactions of the body to damage.

UC-1	4.	Protective-adaptive and pathogenic significance of stress.
UC-1	5.	Starvation, definition of the concept, types. Endogenous and exogenous causes of starvation. Fasting periods, changes in metabolism and physical functions during different periods of fasting.
UC-1	6.	Absolute, complete, incomplete, partial starvation, their characteristics. Factors that determine the duration of fasting.
UC-1	7.	Protein starvation, causes, mechanism of development, manifestations, consequences.
UC-1	8.	Clinical manifestations of starvation, mechanism of development. The concept of therapeutic fasting.
UC-1	9.	Factors that determine the intensity of energy metabolism in the body. Pathology of energy metabolism, causes and mechanism of development. Clinical manifestations.
UC-1	10.	Infectious process, definition of the concept.
UC-1	11.	The etiology of the infectious process, the concept of exo- and endotoxins.
UC-1	12.	Factors that determine the virulence of microorganisms. The role of external conditions and reactivity of the organism in the occurrence, development and outcome of the infectious process.
UC-1	13.	Mechanisms of anti-infective resistance of the organism. Entrance gates of infection, ways of spreading an infectious agent in a macroorganism.
UC-1	14.	Stages of the course of the infectious process, their characteristics.
UC-1	15.	General patterns of development of infectious diseases (inflammation, fever, hypoxia, etc.).
UC-1	16.	Mediators of infectious-allergic reactions (the role of cytokines, lymphokines, monokines, complement components, etc.), their mechanism of action.
UC-1	17.	Violation of the function of organs in the infectious process.
UC-1	18.	Nonspecific and specific humoral and cellular mechanisms of anti-infective protection. Principles of therapy of infectious process.
UC-1	19.	Hepatic encephalopathy, causes, clinical manifestations. Mechanism of development of PE (toxic theory, theory of false neurotransmitters, GABAergic processes in the pathogenesis of PE).
UC-1	20.	Stages of development of PE, their characteristics, clinical manifestations.
UC-1	21.	The main syndromes in the development of PE, the mechanism of development. Hepatic coma, types, mechanism of development, consequences. Pathogenetic therapy of PE.
UC-1	22.	Coma, definition of the concept, types. Etiology of coma, main pathogenetic factors of development. Metabolic coma, definition of the concept, classification.
UC-1	23.	Ketoacidotic coma, etiology, mechanism of development, clinical manifestations, pathogenetic principles of treatment. Hyperosmolar coma,

		causes, mechanism of development, clinical manifestations, ways of prevention and pathogenetic therapy.
UC-1	24.	Lactic acid coma, causes, mechanism of development, clinical manifestations, pathogenetic therapy.
UC-1	25.	Hypoglycemic coma, causes, mechanism of development, clinical manifestations and pathogenetic therapy.
UC-1	26.	Chlorhydropenic coma, causes, mechanism of development, clinical manifestations and pathogenetic therapy.
UC-1	27.	Collapse, definition, classification. Fainting. Characteristics of the main types of collapse, development mechanism, clinical manifestations. Etiological and pathogenetic principles of treatment.
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UC-1	31.	Syndrome of prolonged crushing, causes, mechanism of development.
UC-1	32.	Cardiogenic shock, etiology. Mechanism of development of cardiogenic shock. The role of a decrease in myocardial contractility, vascular insufficiency, impaired microcirculation, pain syndrome, etc. in the development of cardiogenic shock. Clinical manifestations of cardiogenic shock. Etiopathogenetic principles of treatment.
UC-1	33.	Septic shock, definition, etiology and mechanisms of development. Mediators of septic shock, their mechanism of action. Action of endotoxins and exotoxins on target cells. Violation of the state of the hemostasis system in septic shock.
UC-1	34.	Principles of etiopathogenetic treatment of septic shock and emergency care.
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UC-1	36.	Mechanisms of formation of pathological changes in anaphylactic shock, determination of the severity of the course. Etiopathogenetic therapy and prevention of anaphylactic shock.
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Section 2. Pathophysiology of syndromes developing in extreme conditions		

PC-1	40.	Pain as an integrative response of the body to injury. The biological significance of pain as a signal of danger and damage.
PC-1	41.	Nociceptive stimuli and mechanisms of their perception. Receptor, conduction and central links of the pain apparatus.
PC-1	42.	The pathogenesis of acute and chronic pain.
PC-1	43.	Visceral pain, definition of the concept, mechanism of development.
PC-1	44.	Pathological pain of central and peripheral origin (reflected, phantom, causalgia, thalamic pain). Definition of the concept, mechanism of development.
PC-1	45.	Multiple organ failure, definition (MOF). Etiology, classification. Phases of development of multiple organ failure.
PC-1	46.	MOF mediators, their mechanism of action. The role of cytokines IL-1, IL-6, TNF in the development of MOF as first-order mediators.
PC-1	47.	Formation of the systemic inflammatory response syndrome (SIRS). Clinical manifestations of SIRS, stages of development, their characteristics.
PC-1	48.	The concept of primary and secondary multiple organ failure. Markers of "survival" in MOF. Prognostic signs of MOF. Evaluation of the severity of the condition in MOF.
PC-1	49.	The general pattern of formation and sequence of involvement of body systems in the MOF. syndrome. Clinical and laboratory indicators of hypermetabolism in MOF. Violation of the function of organs and systems in MOF syndrome (cardiovascular, respiratory system, liver, kidneys, gastrointestinal tract, etc.).
PC-1	50.	Acute renal failure (ARF), definition, concepts. Prerenal, renal and postrenal factors in the development of acute renal failure. Pathogenesis of acute renal failure, clinical manifestations, stages of development, their characteristics and mechanisms of development.
PC-1	51.	The main syndromes in acute renal failure. Etiopathogenetic principles of treatment of acute renal failure.
PC-1	52.	Chronic kidney disease (CKD), definition of the concept. Principles and stages of CKD development, their characteristics.
PC-1	53.	Etiology and mechanism of development of CRF. The pathogenesis of uremic coma.
PC-1	54.	Mechanisms of progression of chronic kidney disease. Principles of modern nephroprotective therapy.

Evaluation of the results of training in the discipline is carried out on a 2-point scale of assessment

Evaluation criteria and scale:

- evaluation criteria – correct answers to the questions, the correct solution of tasks (tasks);
- evaluation index – the percentage of correct answers to questions, correctly solved tasks (com-

pleted tasks);
- scale of assessment (assessment) – 2 levels of assessment of competences are allocated:
sufficient level (counted) – 50% or more of correct answers and decisions (executions);
insufficient level (not counted) – less than 50 correct answers and decisions (executions).

Result of set-off the Level of development of competence

"credit" sufficient level of the Student showed knowledge of the basic provisions of the discipline, the ability to solve specific practical problems provided by the EP, navigate in the recommended reference books, the ability to correctly assess the results of calculations or experiments

"no credit" insufficient level In the response of the student revealed significant gaps in knowledge of the main provisions of the discipline, the inability of the teacher to get the right solution to a specific practical problem from among the provided EPs

4.9.Term paper/Course project – is not provided

Developer	Signature	Professor position	Antoneeva I.I. name
Developer	Signature	Associate Professor position	Kseiko D.A. name