Federal State Budgetary Educational Institution of Higher Education "Privolzhsky Research Medical University" Ministry of Health of the Russian Federation

BANK OF ASSESSMENT TOOLS FOR DISCIPLINE

PATHOLOGICAL PHYSIOLOGY, PATHOPHYSIOLOGY OF HEAD AND NECK

Training program (specialty): **31.05.03 DENTISTRY** *code, name* Department: **PATHOLOGICAL PHYSIOLOGY**

Mode of study: FULL-TIME

Nizhniy Novgorod 2021

1. Bank of assessment tools for the current monitoring of academic performance, midterm assessment of students in the discipline / practice

This Bank of Assessment Tools (BAT) for the discipline "**Pathological physiology**, **pathophysiology of head and neck** " is an integral appendix to the working program of the discipline "**Pathological physiology**, **pathophysiology of head and neck**". All the details of the approval submitted in the WPD for this discipline apply to this BAT.

(Banks of assessment tools allow us to evaluate the achievement of the planned results stated in the educational program.

Assessment tools are a bank of control tasks, as well as a description of forms and procedures designed to determine the quality of mastering study material by students.)

2. List of assessment tools

The following assessment tools are used to determine the quality of mastering the academic material by students in the discipline/ practice:

No.	Assessment tool	Brief description of the assessment tool	Presentation of the assessment tool in the BAT
1	Tests	A system of standardized tasks that allows you to automate the procedure of measuring the level of knowledge and skills of a student	Bank of test tasks

3. A list of competencies indicating the stages of their formation in the process of mastering the educational program and the types of evaluation tools

Code and formulation of competence*	Stage of competence formation	Controlled sections of the discipline	Assessment tools
UC-1. Able to carry out critical analysis of problem situations based on a systematic approach, develop an action strategy	Current	Subject and tasks of pathophysiology. Basic concepts of nosology. Pathogenic effects of external environmental factors. Modeling of major dental diseases. Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage. Disorders of peripheral blood circulation and microcirculation. Microcirculation disorders in the development of pathological processes in maxillofacial region. Disorders of barrier functions of the body. Blood-salivary barrier. Acute inflammation. Etiology and pathogenesis of maxillofacial inflammatory processes. Sialoses and sialoadenitis: their pathogenesis, principles of modeling and diagnostics of salivary gland s diseases. Wound healing. Pathology of the wound healing in the tissues of the dento-maxillofacial region. Fever. Overheating. Overcooling. Pathophysiology of water-salt	Tests

		metabolism. Edema. Pathophysiology of phosphorus-calcium metabolism, osteoporosis,	
		osteomalacia. Pathophysiology of the acid-base balance (ABB). The role of ABB in the development of caries and inflammatory diseases, periodontal diseases and pathology of the oral mucosa.	
		Pathophysiology of protein, lipids and carbohydrates' metabolism. The role of metabolic disorders in the development of pathology of the dento-maxillofacial region.	
		Tumor growth. The most important etiological factors in the development of head and neck tumors.	
		Hypoxia. The role of hypoxia in the development of dental diseases.	
		Pathophysiology of red blood cells. Mechanisms of disorders in the oral cavity tissues in different types of anemia.	
		Pathophysiology of white blood cells. Changes in the oral in disorders of the white blood cells. Hemoblastosis: their pathogenesis and dental manifestations.	
		Pathophysiology of hemostasis. Significance of hemostatic disorders in the development of dental diseases.	
		Pathophysiology of external respiration. The role of respiratory disorders in the formation of the dental-maxillary system. Changes in external respiration with deformities of the jaws and diseases of the dento-maxillofacial region.	
		Pathophysiology of the cardiovascular system. Heart failure. Coronary insufficiency. Cardiac arrhythmias. Disorders of vascular tone. Arterial hyper-and hypotension. Peculiarities of major dental diseases in patients with arterial hypertension.	
		Pathophysiology of the gastrointestinal system. Peptic ulcer disease. Relationship of gastrointestinal pathology with the state of the oral cavity.	
		Pathophysiology of the liver. The role of liver pathology in the development of dental diseases.	
		Pathophysiology of the kidneys. The role of kidney pathology in the development of diseases of dento- maxillofacial region. Pathophysiology of the nervous system. Pain.	
		Pathophysiology of the endocrine system. Manifestations of endocrine pathology in stomatology.	
		Subject and tasks of pathophysiology.	
GPC-1. Able to implement moral		Basic concepts of nosology.	
and legal norms, ethical and		Pathogenic effects of external environmental factors.	
deontological	Current	Modeling of major dental diseases.	Tests
principles in professional activities		Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage.	
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Disorders of peripheral blood circulation and microcirculation. Microcirculation disorders in the development of pathological processes in maxillofacial region.	
Disorders of barrier functions of the body. Blood-salivary barrier.	
Acute inflammation. Etiology and pathogenesis of maxillofacial inflammatory processes. Sialoses and sialoadenitis: their pathogenesis, principles of modeling and diagnostics of salivary gland s diseases.	
Wound healing. Pathology of the wound healing in the tissues of the dento-maxillofacial region.	
Fever. Overheating. Overcooling.	
Pathophysiology of water-salt metabolism. Edema. Pathophysiology of phosphorus-calcium metabolism, osteoporosis, osteomalacia.	
Pathophysiology of the acid-base balance (ABB). The role of ABB in the development of caries and inflammatory diseases, periodontal diseases and pathology of the oral mucosa.	
Pathophysiology of protein, lipids and carbohydrates' metabolism. The role of metabolic disorders in the development of pathology of the dento-maxillofacial region.	
Tumor growth. The most important etiological factors in the development of head and neck tumors.	
Hypoxia. The role of hypoxia in the development of dental diseases.	
Pathophysiology of red blood cells. Mechanisms of disorders in the oral cavity tissues in different types of anemia.	
Pathophysiology of white blood cells. Changes in the oral in disorders of the white blood cells. Hemoblastosis: their pathogenesis and dental manifestations.	
Pathophysiology of hemostasis. Significance of hemostatic disorders in the development of dental diseases.	
Pathophysiology of external respiration. The role of respiratory disorders in the formation of the dental-maxillary system. Changes in external respiration with deformities of the jaws and diseases of the dento-maxillofacial region.	
Pathophysiology of the cardiovascular system. Heart failure. Coronary	
insufficiency. Cardiac arrhythmias. Disorders of vascular tone. Arterial hyper-and hypotension. Peculiarities of major dental diseases in patients with arterial hypertension.	
Pathophysiology of the gastrointestinal system. Peptic ulcer disease. Relationship of gastrointestinal pathology with the state of the oral	
cavity. Pathophysiology of the liver. The role of liver	
pathology in the development of dental diseases. Pathophysiology of the kidneys. The role of kidney pathology in the development of diseases of dento-	
panology in the development of diseases of denito-	

Pathophysiology of the nervous system. Pain. Pathophysiology of the endocrine system. Manifestations of endocrine pathology in stomatology. Subject and tasks of pathophysiology. Basic concepts of nosology. Pathogenic effects of external environmental factors. Modeling of major dental diseases. Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage. Disorders of parishered blood simulation
system. Manifestations of endocrine pathology in stomatology. Subject and tasks of pathophysiology. Basic concepts of nosology. Pathogenic effects of external environmental factors. Modeling of major dental diseases. Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage.
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Subject and tasks of pathophysiology. Basic concepts of nosology. Pathogenic effects of external environmental factors. Modeling of major dental diseases. Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage.
GPC-8 Able to use basic physical and chemical, mathematical and professional problemsDisorders of peripheral blood circulation and microcirculation. Microcirculation disorders in the development of pathological processes in maxillofacial region.GPC-8 Able to use basic physical and chemical, mathematical and natural science concepts and methods in solvingDisorders of peripheral blood circulation maxillofacial region.GPC-8 concept same maxillofacial circulationAcute inflammatory processes. Sialoses and sialoadenitis: their pathogenesis, principles of modeling and diagnostics of salivary gland s diseases. Wound healing. Pathology of the wound healing in the tissues of the dento-maxillofacial region.GPC-8 Able to use basic physical and chemical,

		and diseases of the dento-maxillofacial region.	
		Pathophysiology of the cardiovascular system. Heart failure. Coronary	
		insufficiency. Cardiac arrhythmias. Disorders of	
		vascular tone. Arterial hyper-and hypotension. Peculiarities of major dental diseases in patients	
		with arterial hypertension.	
		Pathophysiology of the gastrointestinal	
		system. Peptic ulcer disease. Relationship of gastrointestinal pathology with the state of the oral	
		cavity.	
		Pathophysiology of the liver. The role of liver pathology in the development of dental diseases.	
		Pathophysiology of the kidneys. The role of kidney	
		pathology in the development of diseases of dento- maxillofacial region.	
		Pathophysiology of the nervous system. Pain.	
		Pathophysiologyof the endocrine	
		system. Manifestations of endocrine pathology in stomatology.	
		Subject and tasks of pathophysiology.	
		Basic concepts of nosology.	
		Pathogenic effects of external environmental	
		factors. Modeling of major dental diseases	
		Modeling of major dental diseases. Acute non-specific cell injury. Features of the	
		reaction of pulp cells, mucous membranes	
		and of bone tissue for acute and chronic	
		damage. Disorders of peripheral blood circulation	
		and microcirculation. Microcirculation disorders in	
		the development of pathological processes in maxillofacial region.	
		Disorders of barrier functions of the	
GPC-9 Able to		body. Blood-salivary barrier.	
assess		Acute inflammation. Etiology and pathogenesis of maxillofacial inflammatory	
morphofunctional		processes. Sialoses and sialoadenitis: their	
states and pathological	Current	pathogenesis, principles of modeling and diagnostics of salivary gland s diseases.	Tests
processes in the		Wound healing. Pathology of the wound healing	10000
human body to solve professional		in the tissues of the dento-maxillofacial region.	
problems		Fever. Overheating. Overcooling.	
		Pathophysiology of water-salt metabolism. Edema. Pathophysiology of	
		phosphorus-calcium metabolism, osteoporosis,	
		osteomalacia. Pathophysiology of the acid-base balance	
		(ABB). The role of ABB in the development of	
		caries and inflammatory diseases, periodontal diseases and pathology of the oral mucosa.	
		Pathophysiology of protein, lipids and	
		carbohydrates' metabolism. The role of metabolic	
		disorders in the development of pathology of the dento-maxillofacial region.	
		Tumor growth. The most important etiological	
		factors in the development of head and neck tumors.	
		Hypoxia. The role of hypoxia in the	
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		development of dental diseases.	
		Pathophysiology of red blood cells. Mechanisms	
		of disorders in the oral cavity tissues in	
		different types of anemia.	
		Pathophysiology of white blood cells. Changes in	
		the oral in disorders of the white blood cells.	
		Hemoblastosis: their pathogenesis and dental manifestations.	
		Pathophysiology of hemostasis. Significance of hemostatic disorders in the development of dental	
		diseases.	
		Pathophysiology of external respiration. The role	
		of respiratory disorders in the formation of the	
		dental-maxillary system. Changes in external	
		respiration with deformities of the jaws	
		and diseases of the dento-maxillofacial region.	
		Pathophysiology of the cardiovascular	
		system. Heart failure. Coronary	
		insufficiency. Cardiac arrhythmias. Disorders of vascular tone. Arterial hyper-and hypotension.	
		Peculiarities of major dental diseases in patients	
		with arterial hypertension.	
		Pathophysiology of the gastrointestinal	
		system. Peptic ulcer disease. Relationship of	
		gastrointestinal pathology with the state of the oral	
		cavity.	
		Pathophysiology of the liver. The role of liver	
		pathology in the development of dental diseases.	
		Pathophysiology of the kidneys. The role of kidney	
		pathology in the development of diseases of dento-	
		maxillofacial region.	
		Pathophysiology of the nervous system. Pain.	
		Pathophysiologyof the endocrine system. Manifestations of endocrine pathology in	
		stomatology.	
DC 1 Able and		stoniatology.	
PC- 1 Able and ready to		Subject and tasks of pathophysiology.	
implement a set		Basic concepts of nosology.	
of measures		Pathogenic effects of external environmental	
aimed at		factors.	
preserving and		Modeling of major dental diseases.	
strengthening		Acute non-specific cell injury. Features of the	
health and		reaction of pulp cells, mucous membranes	
including the formation of a		and of bone tissue for acute and chronic	
healthy lifestyle		damage.	
of the patient		Disorders of peripheral blood circulation and microcirculation. Microcirculation disorders in	
(their relatives/	Current	the development of pathological processes in	TT (
legal		maxillofacial region.	Tests
representatives).		Disorders of barrier functions of the	
Prevention of the		body. Blood-salivary barrier.	
occurrence and /		Acute inflammation. Etiology and pathogenesis	
or spread of		of maxillofacial inflammatory	
dental diseases, their early		processes. Sialoses and sialoadenitis: their	
diagnosis,		pathogenesis, principles of modeling and	
identification of		diagnostics of salivary gland s diseases.	
the causes and		Wound healing. Pathology of the wound healing in the tissues of the dento-maxillofacial region.	
conditions of			
occurrence and		Fever. Overheating. Overcooling.	
development, as		Pathophysiology of water-salt	

well as prevention.		metabolism. Edema. Pathophysiology of phosphorus-calcium metabolism, osteoporosis,	
		osteomalacia. Pathophysiology of the acid-base balance (ABB). The role of ABB in the development of caries and inflammatory diseases, periodontal diseases and pathology of the oral mucosa.	
		Pathophysiology of protein, lipids and carbohydrates' metabolism. The role of metabolic disorders in the development of pathology of the dento-maxillofacial region.	
		Tumor growth. The most important etiological factors in the development of head and neck tumors.	
		Hypoxia. The role of hypoxia in the development of dental diseases.	
		Pathophysiology of red blood cells. Mechanisms of disorders in the oral cavity tissues in different types of anemia.	
		Pathophysiology of white blood cells. Changes in the oral in disorders of the white blood cells. Hemoblastosis: their pathogenesis and dental manifestations.	
		Pathophysiology of hemostasis. Significance of hemostatic disorders in the development of dental diseases.	
		Pathophysiology of external respiration. The role of respiratory disorders in the formation of the dental-maxillary system. Changes in external	
		respiration with deformities of the jaws and diseases of the dento-maxillofacial region.	
		Pathophysiology of the cardiovascular system. Heart failure. Coronary insufficiency. Cardiac arrhythmias. Disorders of vascular tone. Arterial hyper-and hypotension. Peculiarities of major dental diseases in patients with arterial hypertension.	
		Pathophysiology of the gastrointestinal system. Peptic ulcer disease. Relationship of gastrointestinal pathology with the state of the oral cavity.	
		Pathophysiology of the liver. The role of liver pathology in the development of dental diseases.	
		Pathophysiology of the kidneys. The role of kidney pathology in the development of diseases of dento- maxillofacial region.	
		Pathophysiology of the nervous system. Pain.	
		Pathophysiologyof the endocrine system. Manifestations of endocrine pathology in stomatology.	
PC-6 Ready to collect and		Subject and tasks of pathophysiology.	
analyze		Basic concepts of nosology. Pathogenic effects of external environmental	
complaints and other information	Current	factors.	Tests
from the patient		Modeling of major dental diseases.	Tests
(relatives/ legal representatives), his / her medical history,		Acute non-specific cell injury. Features of the reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage.	

interpretation of	Disorders of peripheral blood circulation
the results of	and microcirculation. Microcirculation disorders in
examination,	the development of pathological processes in
laboratory,	maxillofacial region.
instrumental, and other studies in	Disorders of barrier functions of the body. Blood-salivary barrier.
order to recognize	Acute inflammation. Etiology and pathogenesis
the condition or	of maxillofacial inflammatory
establish the fact	processes. Sialoses and sialoadenitis: their
of the presence or	pathogenesis, principles of modeling and
absence of dental	diagnostics of salivary gland s diseases.
diseases,	Wound healing. Pathology of the wound healing
symptoms,	in the tissues of the dento-maxillofacial region.
syndromes of dental diseases	Fever. Overheating. Overcooling.
dental diseases	Pathophysiology of water-salt
	metabolism. Edema. Pathophysiology of
	phosphorus-calcium metabolism, osteoporosis,
	osteomalacia.
	Pathophysiology of the acid-base balance
	(ABB). The role of ABB in the development of caries and inflammatory diseases, periodontal
	diseases and pathology of the oral mucosa.
	Pathophysiology of protein, lipids and
	carbohydrates' metabolism. The role of metabolic
	disorders in the development of pathology of the
	dento-maxillofacial region.
	Tumor growth. The most important etiological
	factors in the development of head and neck
	tumors.
	Hypoxia. The role of hypoxia in the
	development of dental diseases.
	Pathophysiology of red blood cells. Mechanisms
	of disorders in the oral cavity tissues in
	different types of anemia.
	Pathophysiology of white blood cells. Changes in
	the oral in disorders of the white blood cells. Hemoblastosis: their pathogenesis and dental
	manifestations.
	Pathophysiology of hemostasis. Significance of
	hemostatic disorders in the development of dental
	diseases.
	Pathophysiology of external respiration. The role
	of respiratory disorders in the formation of the
	dental-maxillary system. Changes in external
	respiration with deformities of the jaws
	and diseases of the dento-maxillofacial region.
	Pathophysiology of the cardiovascular system. Heart failure. Coronary
	insufficiency. Cardiac arrhythmias. Disorders of
	vascular tone. Arterial hyper-and hypotension.
	Peculiarities of major dental diseases in patients
	with arterial hypertension.
	Pathophysiology of the gastrointestinal
	system. Peptic ulcer disease. Relationship of
	gastrointestinal pathology with the state of the oral
	cavity.
	Pathophysiology of the liver. The role of liver pathology in the development of dental diseases.
	Pathophysiology of the kidneys. The role of kidney
	pathology in the development of diseases of dento-

		 maxillofacial region. Pathophysiology of the nervous system. Pain. Pathophysiologyof the endocrine system. Manifestations of endocrine pathology in stomatology. Subject and tasks of pathophysiology. Basic concepts of nosology. Pathogenic effects of external environmental factors. Modeling of major dental diseases. Acute non-specific cell injury. Features of the 	
PC-12 Able to participate in scientific research, analysis and public presentation of medical information based on evidence- based medicine and to participate in the introduction of new methods and techniques aimed at protecting public health and reducing dental morbidity.	lysis of pased ne pate of and med and	reaction of pulp cells, mucous membranes and of bone tissue for acute and chronic damage. Disorders of peripheral blood circulation and microcirculation. Microcirculation disorders in the development of pathological processes in maxillofacial region. Disorders of barrier functions of the body. Blood-salivary barrier. Acute inflammation. Etiology and pathogenesis of maxillofacial inflammatory processes. Sialoses and sialoadenitis: their pathogenesis, principles of modeling and	
		diagnostics of salivary gland s diseases. Wound healing. Pathology of the wound healing in the tissues of the dento-maxillofacial region. Fever. Overheating. Overcooling. Pathophysiology of water-salt metabolism. Edema. Pathophysiology of phosphorus-calcium metabolism, osteoporosis, osteomalacia. Pathophysiology of the acid-base balance	Tests
		 (ABB). The role of ABB in the development of caries and inflammatory diseases, periodontal diseases and pathology of the oral mucosa. Pathophysiology of protein, lipids and carbohydrates' metabolism. The role of metabolic disorders in the development of pathology of the dento-maxillofacial region. Tumor growth. The most important etiological factors in the development of head and neck tumors. Hypoxia. The role of hypoxia in the 	
		development of dental diseases. Pathophysiology of red blood cells. Mechanisms of disorders in the oral cavity tissues in different types of anemia. Pathophysiology of white blood cells. Changes in the oral in disorders of the white blood cells. Hemoblastosis: their pathogenesis and dental manifestations. Pathophysiology of hemostasis. Significance of hemostatic disorders in the development of dental	
		diseases. Pathophysiology of external respiration. The role of respiratory disorders in the formation of the dental-maxillary system. Changes in external respiration with deformities of the jaws	

and diseases of the dento-maxillofacial region. Pathophysiology of the cardiovascular system. Heart failure. Coronary insufficiency. Cardiac arrhythmias. Disorders of vascular tone. Arterial hyper-and hypotension. Peculiarities of major dental diseases in patients	
with arterial hypertension.	
Pathophysiology of the gastrointestinal system. Peptic ulcer disease. Relationship of gastrointestinal pathology with the state of the oral cavity.	
Pathophysiology of the liver. The role of liver pathology in the development of dental diseases.	
Pathophysiology of the kidneys. The role of kidney pathology in the development of diseases of dento- maxillofacial region.	
Pathophysiology of the nervous system. Pain.	
Pathophysiologyof the endocrine system. Manifestations of endocrine pathology in stomatology.	

4. The content of the assessment tools of entry, current control Current control is carried out by the discipline teacher when conducting classes in the form of: assessment tool 1, assessment tool 2.

Assessment tools for current control.

Assessment tool 1

N⁰	Test	Answers	Developing competence code (according to the WPD)
1.	The main etiological factor of acute altitude sickness is: A) Decrease in atmospheric pressure B) Decrease in partial pressure of O ₂ in the air C) Ultraviolet radiation	В	UC 1 GPC 1,8,9 PC 1,6,12
	D) Low temperature E) High temperature		
2.	The conditions those promote overheating of the organism: A) High humidity and environment temperature B) Increase in perspiration	A, C, D	UC 1 GPC 1,8,9
	C) Decrease in perspirationD) Uncoupling oxidation and phosphorylationE) Dilatation of peripheral blood vessels		PC 1,6,12
3.	What cells, organs and tissues are the most radiosensitive: A) Brain B) Bone marrow	B, D, E	UC 1 GPC 1,8,9
	C) Erythrocytes D) Gastro-intestinal epithelium E) Gonads		PC 1,6,12
4.	Factor promoting radiation-damage of cells are: A) Vitamin E deficiency B) High mitotic activity C) Low mitotic activity	A, B	UC 1 GPC 1,8,9 PC 1,6,12
5.	Mark the signs of arterial hyperemia: A) Cyanosis of the organ B) Reddening of the organ or tissue C) Marked edema of the organ	B, D, E	UC 1 GPC 1,8,9 PC 1,6,12
	D) Increased tissue turgorE) Increased temperature in the organs localized superficially		
6.	Choose the basic types of arterial hyperemia according to its origin: A) Neurotonic	A, C, D	UC 1 GPC 1,8,9

	1	1	
	B) Obstructive		PC 1,6,12
	C) Neuroparalytic		, ,
	D) Myoparalytic		
	E) Compressive		
	Mark the signs of venous hyperemia:		UC 1
	A) Increased tissue turgor	A, B, C	
	B) Edema of an organ	11, 2, 0	GPC 1,8,9
	C) Cyanosis of an organ or tissue		PC 1,6,12
	D) Redness of an organ or tissue		
	E) Decrease in temperature in internal organ		
8.	Mark the symptoms of ischemia:		UC 1
	A) Cyanosis of an organ	B, C, D	GPC 1,8,9
	B) Paleness of an organ or tissue		
	C) Pain		PC 1,6,12
	D) Decrease in tissue turgor		
	E) Reddening of the organ or tissue		
	Which bioactive substances are responsible for ischemia?		
	A) Histamine	B, D	UC 1
		В, D	GPC 1,8,9
	B) Catecholamines		PC 1,6,12
	C) Bradykinin		1 C 1,0,12
	D) Thromboxane A_2		
	E) Acetylcholine		
10.	Causes of aseptic inflammation may be the following:		UC 1
	A) Hemorrhage into tissues	A, B, C	
	B) The surgical operation that was done in aseptic conditions		GPC 1,8,9
	C) Parenteral injection of sterile foreign protein		PC 1,6,12
	D) Enteral administration of non-sterile foreign protein		
	E) Transient hyperoxia of tissues		
11.	Inflammation is regarded as an adaptive reaction of the organism because it:		UC 1
	A) Inactivates phlogogenic agent	A, C, D,	GPC 1,8,9
	B) Prevents allergization of the organism	E	
	C) Mobilizes defensive factors of the organism		PC 1,6,12
	D) Promotes the restoration or replacement of injured tissues		
	E) Restricts the site of injury (especially in venous hyperemia)		
	The sings that can show the presence of inflammatory process in the organism are:		UC 1
	A) Leukocytosis	A, C, D	001
	B) Erythrocytosis	п, с, р	GPC 1,8,9
	B) Erythrocytosis	Л, С, D	
	C) Fever	И, С, Б	GPC 1,8,9 PC 1,6,12
	C) Fever D) Increase in ESR	м, С, D	
	C) Fever D) Increase in ESR E) Thrombosis	M, C, D	PC 1,6,12
	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: 		
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis 	A, B, C	PC 1,6,12 UC 1
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: 		PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis 		PC 1,6,12 UC 1
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia 		PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia 		PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia 		PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: 		PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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13.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? 	A, B, C B, C, E	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
13. 14. 15.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? A) Leading role of IgE in pathogenesis 	A, B, C	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
13. 14. 15.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? A) Leading role of IgE in pathogenesis B) A response reveals itself in 15-20 minutes after the repeated contact with the 	A, B, C B, C, E	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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13. 14. 15.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? A) Leading role of IgE in pathogenesis B) A response reveals itself in 15-20 minutes after the repeated contact with the allergen C) A response reveals itself in 24-48 hours after the repeated contact with the allergen 	A, B, C B, C, E	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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13. 14.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? A) Leading role of IgE in pathogenesis B) A response reveals itself in 15-20 minutes after the repeated contact with the allergen C) A response reveals itself in 24-48 hours after the repeated contact with the allergen D) Histamine, bradykinine, leukotryens play the main role in the mechanism of allergic reaction 	A, B, C B, C, E	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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13. 14. 15.	 C) Fever D) Increase in ESR E) Thrombosis In an acute inflammation site there are such chemical and physical changes as: A) Acidosis B) Hyperosmia C) Hyperoncia D) Hyposmia E) Hyponcia Mediators of inflammation that cause an increase in vascular permeability in inflammation are: A) Heparin B) Histamine C) Bradykinine D) Interferon E) Leukotrienes What is common to the first type of an allergic response? A) Leading role of IgE in pathogenesis B) A response reveals itself in 15-20 minutes after the repeated contact with the allergen C) A response reveals itself in 24-48 hours after the repeated contact with the allergen D) Histamine, bradykinine, leukotryens play the main role in the mechanism of allergic reaction E) In the mechanism of allergy the main role belongs to lymphokines 	A, B, C B, C, E A, B, D	PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
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	D) The mechanism of development depends on lymphokines		
	E) The mechanism of development depends on histamine and bradykinin		
	Autoimmune diseases that develop according to 2 ^d type of allergy are:		UC 1
	A) Myasthenia gravis	A, C, E	GPC 1,8,9
	B) Serum disease		
	C) Immune agranulocytosis		PC 1,6,12
	D) Acute glomerulonephritis		
	E) Autoimmune hemolytic anemia		
18	Autoimmune diseases that develop according to the 3 rd type of allergy are:		UC 1
	A) Myasthenia gravis	B, D	UC 1
		D, D	GPC 1,8,9
	B) Serum disease		PC 1,6,12
	C) Immune agranulocytosis		rc 1,0,12
	D) Acute glomerulonephritis		
	E) Autoimmune hemolytic anemia		
19.	What changes in the organism are typical of acute-phase reaction?		UC 1
	A) Activation of immune system	A, B, D	
	B) Increase of ACTH production in hypophysis	,,	GPC 1,8,9
	C) Increase of albumin production in liver		PC 1,6,12
	D) Activation of phagocytosis		
	E) Increase in protein synthesis in muscles		
	Noninfectious fever arises in the following pathological processes:		UC 1
	A) Necrosis of tissues	A, C, D	
	B) Hyperproduction of thyroid hormones		GPC 1,8,9
	C) Malignant tumor		PC 1,6,12
	D) Intravascular hemolysis of erythrocytes		
	E) Exogenic overheating		
21.	What symptoms are typical of acute-phase reaction?		UC 1
	A) Fever	A, D, E	GPC 1,8,9
	B) Neutropenia		
	C) Positive nitrogen balance		PC 1,6,12
	D) Increase in cortisol production by adrenal glands		
	E) Negative nitrogen balance		
	Name mechanisms that take part in raising the temperature of the body in fever:		
	A) Peripheral vasoconstriction	A, B, C,	UC 1
			GPC 1,8,9
	B) Increase in contractile thermogenesis	D	PC 1,6,12
	C) Decrease in perspiration		, ,
	D) Activation of biological oxidation		
	E) Increase in perspiration		
23.	Mark the manifestations of malignant tumors growth:		UC 1
	A) Metastasis	A, B,C, E	
	B) Recurrence	7 7 - 7	GPC 1,8,9
	C) Invasive growth		PC 1,6,12
	D) Expansive growth		
		1	
	E) Weakening of contact inhibition of cells		
24.	Which factors are responsible for the destruction of tumor cells in the organism?		UC 1
24.	Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis	A, C, D	
24.	Which factors are responsible for the destruction of tumor cells in the organism?	A, C, D	GPC 1,8,9
24.	Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis	A, C, D	
24.	Which factors are responsible for the destruction of tumor cells in the organism?A) Macrophage phagocytosisB) T-lymphocyte suppressorsC) T-lymphocyte killers	A, C, D	GPC 1,8,9
24.	Which factors are responsible for the destruction of tumor cells in the organism?A) Macrophage phagocytosisB) T-lymphocyte suppressorsC) T-lymphocyte killersD) NK "Natural killers"	A, C, D	GPC 1,8,9
24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells 	A, C, D	GPC 1,8,9 PC 1,6,12
24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? 		GPC 1,8,9
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24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture 		GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis D) Production of the factor which intensifies angiogenesis 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
24.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis D) Production of the factor which intensifies angiogenesis E) Weakening of cellular differentiation 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
24. 25. 26.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis D) Production of the factor which intensifies angiogenesis E) Weakening of cellular differentiation Compensatory mechanisms of metabolic acidosis are: 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
24. 25. 26.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis D) Production of the factor which intensifies angiogenesis E) Weakening of cellular differentiation Compensatory mechanisms of metabolic acidosis are: A) Binding of hydrogen ions by proteins and bicarbonate buffer 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
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24. 25. 26.	 Which factors are responsible for the destruction of tumor cells in the organism? A) Macrophage phagocytosis B) T-lymphocyte suppressors C) T-lymphocyte killers D) NK "Natural killers" E) Fibrinous pellicle covering tumor cells What characterizes malignant growth? A) Weakening of contact inhibition of cells in tissue culture B) Availability of solid surface for grow of the cells in tissue culture C) Intensification of anaerobic glycolysis D) Production of the factor which intensifies angiogenesis E) Weakening of cellular differentiation Compensatory mechanisms of metabolic acidosis are: A) Binding of hydrogen ions by proteins and bicarbonate buffer B) Hyperventilation C) Intensified urine excretion of ammonia salt D) Intensified urine excretion of bicarbonate by kidneys 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
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	B) Increase in HCO ₃ reabsorbtion in kidney canaliculi		GPC 1,8,9
	C) Decrease in HCO ₃ reabsorbtion in kidney canaliculi		PC 1,6,12
	D) Binding of surplus of protons by reduced hemoglobin		1 C 1,0,12
	E) Hypokalemia		
28.	Which factors are the causes of respiratory acidosis?		UC 1
	A)Hypoventilation of lungs	A, B, D,	
	B)Accumulation of exudates in pleural cavity	Ē	GPC 1,8,9
	C) Hyperventilation of lungs	_	PC 1,6,12
	D) Decreased excitability of respiratory center		
	E) Inhalation of gaseous mixture with high content of CO_2		
20			
29.	Which hormones excess can give rise of hyperglycemia?		UC 1
	A) Adrenalin	A, B, D	GPC 1,8,9
	B) Glucocorticoids		
	C) Insulin		PC 1,6,12
	D) Glucagon		
	E) ADH		
30.	What is the main link in pathogenesis of diabetic coma in patients with diabetes		UC 1
	mellitus 1 type?	В	
	A) Hyperglycemia	D	GPC 1,8,9
	B) Hyperketonemia		PC 1,6,12
	C) Hyperpotassiumemia		,0,12
	D) Hypersodiumemia		
	E) Alkalosis		
31.	What is the cause of polyuria in an early stage of diabetes mellitus?		UC 1
	A) Microangiopathy of kidneys	В	GPC 1,8,9
	B) Hyperglycemia		
	C) Ketonemia		PC 1,6,12
	D) Hypercholesterolemia		
	E) Hyperpotassemia		
32.	What are the complications of long-term diabetes mellitus?		
32.			UC 1
	A) Fast development of atherosclerosis	A, B, C,	GPC 1,8,9
	B) Microangiopathy	E, F	PC 1,6,12
	C) Macroangiopathy		PC 1,0,12
	D) Polyuria		
	E) Nephropathy		
	F) Neuropathy		
33.	Choose the possible causes of right ventricle failure:		UC 1
	A) Arterial hypertension of the systemic circulation	B, C, D	
	B) Arterial hypertension of the pulmonary circulation	_, _, _	GPC 1,8,9
	C) Defect of interventricular septum		PC 1,6,12
			- , - ,
	D) Emphysema of lungs		
~ ·	E) Coarctation of aorta		
34.	Choose the possible causes of the left ventricle failure:		UC 1
	A) Aortic stenosis	A, B, D	GPC 1,8,9
	B) Infarction of the left ventricle		
	C) Arterial hypertension of the pulmonary circulation		PC 1,6,12
	D) Hypertonic disease		
	E) Emphysema of lungs		
35	Heart failure due to the overload by an increased blood volume develops in the		110.1
55.	following cases:	A, C, E	UC 1
		А, С, Е	GPC 1,8,9
	A) Inherited defects of heart septum		PC
	B) Hypertension of systemic circulation		IC
	C) Insufficiency of heart valves		
	D) Aortic stenosis		
	E) Aortic regurgitation		
36.	An overload of the left ventricle by an increased blood pressure develops in the		UC 1
-	following cases:	A, B, D	
	A) Coarctation of aorta	., _, _, _	GPC 1,8,9
	B) Essential hypertension		PC 1,6,12
			,0,12
	C) Mitral insufficiency		
	D) Symptomatic hypertension		
	E) Aortic regurgitation		
37.	Endogenous hypertensive agents promoting elevation of arterial pressure by rising		UC 1
37.		B, C, D	UC 1 GPC 1,8,9

	B) Catecholamines		PC 1,6,12
	C) Angiotensin II		
	D) Vasopressin		
	E) Nitric oxide		
	Endogenous antihypertensive agents promoting arterial pressure fall by decreasing of		UC 1
	peripheral vascular resistance are:	B, C, D	
	A) Catecholamines		GPC 1,8,9
	B) Bradykinin		PC 1,6,12
	C) Prostaglandin E		
	D) NO (nitric oxide)		
	E) Angiotensin		
	Factors that are responsible for pathogenesis of edemas in decompensated heart		
	failure are:		UC 1
		A, B, D, E	GPC 1,8,9
	A) An increase in hydrostatic pressure in the venous part of capillaries	E	PC 1,6,12
	B) An increase in aldosterone and vasopressin content in the blood		1 C 1,0,12
	C) A decrease in aldosterone and vasopressin content in the blood		
	D) Dynamic lymphatic failure		
	E) A decrease in oncotic pressure of blood		
40.	Compensatory mechanisms in acute hypoxia are:		UC 1
	A) Blood redistribution	A, B, C, E	
	B) Increase in lung ventilation	, _, _, _, _	GPC 1,8,9
	C) Tachycardia		PC 1,6,12
	D) Decrease in cardiac output		
	E) Release of erythrocytes from blood storages		
	Inspiratory dyspnea can be revealed in the following pathological conditions:		UC 1
	A) Pulmonary emphysema	B, D, E	GPC 1,8,9
	B) Larynx edema		
	C) Bronchial asthma attacks		PC 1,6,12
	D) Stenosis of trachea		
	E) I asphyxia stage		
	Expiratory dyspnea can be revealed in the following pathological conditions:		UC 1
	A) Pulmonary emphysema	A, C	
	B) Larynx edema	А, С	GPC 1,8,9
			PC 1,6,12
	C) Bronchial asthma attacks		1 C 1,0,12
	D) Stenosis of trachea		
	E) I asphyxia stage		
43.	Respiratory insufficiency may be characterized by the following changes in gas		LIC 1
			UC 1
	composition and acid-base balance of arterial blood:	A, C, D	
	composition and acid-base balance of arterial blood: A) Hypoxemia	A, C, D	GPC 1,8,9
	A) Hypoxemia	A, C, D	
	A) Hypoxemia B) Hyperoxemia	A, C, D	GPC 1,8,9
	A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis	A, C, D	GPC 1,8,9
	A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia	A, C, D	GPC 1,8,9
	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia 		GPC 1,8,9 PC 1,6,12
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: 	A, C, D,	GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea 		GPC 1,8,9 PC 1,6,12 UC 1
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: 	A, C, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hypoxia Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9
44.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: 	A, C, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection B) Overproduction of glycocorticoids 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection B) Overproduction of glycocorticoids C) Increased mucus excretion 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection B) Overproduction of glycocorticoids C) Increased mucus excretion D) Duodeno-gastric reflux 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection B) Overproduction of glycocorticoids C) Increased mucus excretion D) Duodeno-gastric reflux E) Increased evacuation of food from the stomach 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12
44. 45. 46.	 A) Hypoxemia B) Hyperoxemia C) Respiratory acidosis D) Hypercapnia E) Hypocapnia Respiratory insufficiency is characterized by: A) Dyspnea B) Anemia C) Tachycardia D) Cyanosis E) Hypoxia Hyperacidity and hypersecretion of gastric glands are characterized by the following symptoms: A) Predisposition to constipation B) Elevation of pepsin activity C) Spasm of pylorus D) Low pepsin activity The following factors can contribute to the development of gastric and duodenal ulcers: A) Infection B) Overproduction of glycocorticoids C) Increased mucus excretion D) Duodeno-gastric reflux 	A, C, D, E A, B, C	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6 UC 1 GPC 1,8,9 PC 1,6,12,12 UC 1 GPC 1,8,9 PC 1,6,12

		1 1	
	B) Acholia		PC 1,6,12
	C) Achilia		
	D) Hypochilia		
	E) Hypocholia		
48.	Mark the factors which play an important role in ascites pathogenesis in portal		UC 1
	hypertension:	A, C, D,	
	A) Elevation of hydrostatic pressure in a portal vein system	Е	GPC 1,8,9
	B) Lowering of lymph-formation		PC 1,6,12
	C) Elevation of lymph-formation		
	D) Lowering of oncotic pressure of blood		
	E) Activation of RAAS		
	Mark the manifestations of malabsorption syndrome:		
49.	A) Diarrhea	A, C, D	UC 1
		A, C, D	GPC 1,8,9
	B) Constipation		PC 1,6,12
	C) Weight loss		, ,
	D) Hypoproteinemia		
	E) Hyperproteinemia		
	Which pigment stains urine in dark color in posthepatic jaundice?		UC 1
	A) Conjugated bilirubin	Α	
	B) Unconjugated bilirubin		GPC 1,8,9
	C) Urobilin		PC 1,6,12
	D) Stercobilin		
	E) Hemoglobin		
31.	Which pigments stain urine in a dark color in prehepatic jaundice?		UC 1
	A) Conjugated bilirubin	C, D	GPC 1,8,9
	B) Unconjugated bilirubin		
	C) Urobilin		PC 1,6,12
	D) Stercobilin		
	E) Hemoglobin		
52.	The symptoms characteristics of cholemia are:		UC 1
	A) Bradycardia	A, B, D	
	B) Skin itch	, ,	GPC 1,8,9
	C) Tachycardia		PC 1,6,12
	D) Decrease in arterial pressure		
	E) Rising of arterial pressure.		
	Which vitamins absorption will became worse in acholia?	1	
55.			UC 1
	A) Vitamin A	A, C, D,	GPC 1,8,9
	B) Vitamin B1	E	
	C) Vitamin D		PC 1,6,12
	D) Vitamin E		
	E) Vitamin K		
54.	Which of the following indexes characterize a tubular function disorder of kidneys?		UC 1
		1	
	A) Aminoaciduria	A. C. D	
		A, C, D	GPC 1,8,9
	B) Hematuria	A, C, D	
	B) Hematuria C) Isosthenuria	A, C, D	GPC 1,8,9
	B) Hematuria C) Isosthenuria D) An unselective proteinuria	A, C, D	GPC 1,8,9
	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance 	A, C, D	GPC 1,8,9 PC 1,6,12
	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: 		GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage 		GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin 	A, B, D,	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12
55. 56.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1
55. 56.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55. 56.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55. 56.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin 	A, B, D, E A, C, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin Parameters describing reduction in glomerular filtration rate are: A) Leukocyturia 	A, B, D, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 UC 1
55.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin 	A, B, D, E A, C, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9
55. 56.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin Parameters describing reduction in glomerular filtration rate are: A) Leukocyturia 	A, B, D, E A, C, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 UC 1
55. 56. 57.	 B) Hematuria C) Isosthenuria D) An unselective proteinuria E) A lowering of creatinin clearance Mark the main mechanisms of the glomerular filtration rate lowering: A) Decrease in systemic arterial pressure B) Primary urine outflow damage C) Falling of oncotic pressure of blood D) Elevation of oncotic pressure of blood E) Lowering of a functional nephrons number Polyuria can be caused by the lack of: A) Vasopressin B) Adrenaline C) Aldosterone D) Oxytocin E) Insulin Parameters describing reduction in glomerular filtration rate are: A) Leukocyturia B) Azotemia 	A, B, D, E A, C, E	GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9 PC 1,6,12 UC 1 GPC 1,8,9

58.	Choose the diseases that are typical of the development of secondary diabetes mellitus: A) Acromegaly B) Insulinoma C) Cushing's syndrome D) Myxedema E) Addison's disease	A, C	UC 1 GPC 1,8,9 PC 1,6,12
59.	 Which hormones insufficiency may develop in the organism after a sudden cessation of the prolonged corticosteroid therapy? A) Cortisol B) Adrenalin C) ACTH D) ADH E) Insulin 	A, C	UC 1 GPC 1,8,9 PC 1,6,12

Assessment tool 2

<u>№</u> 1.	Test Match the following pathologic states and their consequences of 1) Acholia and 2) Pancreatic achilia A) Bile absence in the duodenum	Answers 1 A, C, D 2 B, E	Developing competence code (according to the WPD) UC 1 GPC 1,8,9 PC 1,6 12
	B) Lipase absenceC) Low lipase activityD) Emulgence of the lipids disorderE) Lipids splitting disorder		PC 1,6,12
2.	Match the diseases and the disturbances of the hormone production: 1) STH 2) ACTH 3)TSH 4) GTH 5) ADH A) Diabetes insipidus B) Cushing's disease C) Hyperthyroidism D) Premature sexual maturation E) Pituitary dwarfism	1 – E 2 – B 3 – C 4 – D 5 - A	UC 1 GPC 1,8,9 PC 1,6,12
3.	 Match the classification variant of endocrine disorders and their examples. 1) Disturbance of the central regulation of the endocrine glands 2) Pathological processes in the gland 3) Peripheral mechanisms of the hormones activity disorder A) Formation of antibodies to some hormones B) Genetic defect of the hormone synthesis C) Lack of substrates for the hormone synthesis D) Disturbance of the hormone connection with the protein carrier E) Damage of the hypothalamus F) Injury of the limbical structures of the brain G) Disturbance of the receptor expression to hormones in the target cells 	1) E, F 2) B, C 3) A, D, G, H	UC 1 GPC 1,8,9 PC 1,6,12
4.	 Put into the correct order the sequence of changes leading to hyperpigmentation of skin and mucous membranes in the Addison's disease A) Increase in a synthesis and a secretion of the proopiomelanocortin by the hypophysis (the precursor of ACTH) B) Insufficiency of a cortisol production by the adrenal gland cortex C) Increased production of ACTH and melanocortin by the hypophysis D) Hyperproduction of melanin by the melanocytes E) Increase in the pigment accumulation in the skin and mucous membranes 	B, A, C, D, E	UC 1 GPC 1,8,9 PC 1,6,12
5.	 Match the variant of dyspnea and the most possible causes 1) Tachypnea 2) Bradypnea A) Hypoxia B) Decrease in the respiratory center excitability C) Hyperoxia D) Elevation of the respiratory center excitability E) Compensative acidosis F) Increased arterial pressure 	1) A, D, E 2) B, C, F	UC 1 GPC 1,8,9 PC 1,6,12
6.	Match diuresis disorders and their definitions 1) polyuria	1-C, 2-D,	UC 1

	2) olyguria	3-E, 4-B,	GPC 1,8,9
	3) anuria	5-A.	PC 1,6,12
	4) hyposthenuria		1 0 1,0,12
	5) isosthenuria		
	A) monotonous diuresis with the urine density of 1.010		
	B) diuresis with the urine density of 1.012-1.006		
	C) increased day urine amount		
	D) lowered day urine amount		
	E) urine cessation (no urine flow)		
7.	Match the variants of cholestasis and their causes.		UC 1
	1) Primary cholestasis	1) B, C, E, F	
	2) Secondary cholestasis	2) A, D	GPC 1,8,9
	A) Obturation of a common bile duct by a stone or by a tumor	2) M, D	PC 1,6,12
	B) Condensation of bile in dehydratation		
	C) Cholangitis		
	D) An edema of a Fatter's papilla due to the duodenum inflammation		
	E) Infectious hepatitis		
	F) Toxic hepatitis		
8.	Put into a correct order the sequences of changes leading to the development of		UC 1
0.	cardiac edemas.	3-2-1-4-7-6-	
	1. Stimulation of the aldosterone secretion.	5-8.	GPC 1,8,9
		5-0.	PC 1,6,12
	2. Irritation of the baroreceptors.		, ,
	3. Decrease in the cardiac output.		
	4. Increase in the sodium reabsorption by kidneys.		
	5. Increase in the water reabsorption by kidneys.		
	6. Increase in the ADH production.		
	7. Irritation of osmoreceptors.		
	8. Accumulation of water by tissues.		
0			
9.	What is the sequence of changes leading to the development of nephrotic		UC 1
	edemas?	6-5-3-4-1-2-	GPC 1,8,9
	1. Increase in the aldosterone and ADH production.	7	PC 1,6,12
	2. Increase in the sodium and water reabsorbtion by kidneys.		FC 1,0,12
	3. Increase in the water filtration from the vessels into the tissues.		
	4. Hypovolemia.		
	5. Hypoproteinemia.		
	6. Proteinuria.		
	7. Release of water from the vessels into the tissues and the development of		
	edemas.		
10.	Put into a correct order the sequences of changes leading to the normalization		UC 1
	of the sodium concentration in blood in electrolytes disorders.	2-5-6-1-3-7-	
	1. Increase in the ADH production.	4	GPC 1,8,9
		7	PC 1,6,12
	2. Hypersodiumemia.		
	3. Increase in the sodium and water reabsorbtion by kidneys.		
	4. Normalization of sodium concentration in blood.		
	5. Increase in the plasma osmolality.		
	6. Irritation of the osmoreceptors.		
	7. Increase in the circulation blood volume.		
11.	Match the type of body's resistance and their manifestations.		
11.	1) Active resistance		UC 1
	,	1) A, B, D,	GPC 1,8,9
	2) Passive resistance	Н	PC 1,6,12
	A) Emigration of leukocytes and phagocytes		1,0,12
	B) Neutralization and elimination	2) C, E, G	
	C) Hereditary immunity		
	D) Acute-phase reactions		
	E) Barrier functions of the skin and mucous membranes		
	G) HCI content in the gastric juice		
10	H) Tachypnea and tachycardia in hypoxia		
12.	Match the somatotypes and their signs		UC 1
	1) Hypersthenic type of the human constitution	1). B, D, E	
	2) Asthenic type of the human constitution		GPC 1,8,9
	A) Narrow chest	2) A, C, F	PC 1,6,12
	B) Horizontal position of the heart	-,, -, -, -	
	C) Acute epigastric angle		
		1	
	D) Dull epigastric angle E) Tendency to obesity		

	F) Tendency to hypoglycemia		
13.	 Match the somatotypes and their biochemical peculiarities 1) Hypersthenic type of the human constitution 2) Asthenic type of the human constitution A) basic metabolism is decreased B) content of sugar in blood is decreased C) content of cholesterol in blood is increased D) processes of dissimilation prevail 	1) A, C 2) B, D	UC 1 GPC 1,8,9 PC 1,6,12
14.	 Match the somatotypes and predisposition to the diseases. 1) Hypersthenic type of the human constitution 2) Asthenic type of the human constitution A) Gastric and duodenal disease B) Addison's disease C) Diabetes mellitus D) Hypertonic disease E) Abdominal hernia 	1) C, D 2) A, B, E	UC 1 GPC 1,8,9 PC 1,6,12
15.	Chose the sentences to complete the definition of "resistance" Resistance is 1.Stability of cells, tissues, organs and the organism as a whole to resist to the action 2.Ability of organism to resist to the action a) of certain factors of the environment b) of pathogenic factors of the environment	1, b)	UC 1 GPC 1,8,9 PC 1,6,12

5. The content of the assessment tools of mid-term assessment

Mid-term assessment is carried out in the form of an **exam**. *The content of the assessment tool (questions.)*

The bank of assessment tools for conducting current control and mid-term assessment of students in this discipline is presented on the Educational Portal of the PRM. A link to this electronic resource:

https://sdo.pimunn.net/course/view.php?id=2765

https://sdo.pimunn.net/course/view.php?id=2766

5.1 The list of control tasks and other materials necessary for the assessment of knowledge, skills and work experience (*the teacher indicates only those tasks and other materials that are used within the framework of this discipline*)

5.1.1. Questions for the discipline exam.

		Competence
	Question	code (according
		to the WPD)
1.	Health (norm) and disease. Characteristics of the diseases. Pathological process,	
	pathological reaction, pathological state, typical (common) pathological process.	
2.	Stages and outcomes of a disease.	
3.	Etiology. Causes of diseases.	
4.	Pathogenesis (definition). Cause and effect relations. Conception of vicious circle.	
5.	Reactivity of the body: definition, kinds, mechanisms. Resistance: definition,	
	kinds, mechanisms. Reactivity and resistance.	
6.	The role of heredity in pathology: hereditary and congenital diseases,	
	genetic predispositions. Causes and kinds of mutations. Types of genetic	
	diseases transduction. Molecular-genetic and chromosome diseases.	
7.	Stress-reaction (general adaptation syndrome). Adaptation diseases.	
8.	Shock. Definition, kinds, phases, pathogenesis.	
9.	Coma. Definition, classification, pathogenesis.	
10.	Etiology and pathogenesis of cell injury.	
11.	Mechanisms of cell injury compensation. Necrosis and apoptosis.	
12.	Arterial hyperemia. Causes, kinds, pathogenesis, external signs, consequences,	

10	significance.	
13.	Venous hyperemia. Causes, pathogenesis, manifestations, consequences,	
14	outcomes.	
14.	Ischemia. Causes, kinds, pathogenesis, signs, consequences, outcomes. Reperfusion.	
15	Thrombosis as a cause of peripheral disorders of blood circulation.	
	Embolism as a cause of peripheral disorders of blood circulation.	
	Inflammation. Etiology. Pathogenesis of local signs of acute inflammation.	
	Mediators of inflammation. Kinds, mechanisms of action.	
	Disorders of blood circulation and microcirculation in the focus of inflammation.	UC 1
	Mechanisms of exudation. Kinds of exudates and their qualities. Comparison of	GPC 1,8,9
20.	exudation and transudation.	PC 1,6,12
21.	Mechanism of leukocytes emigration. Phagocytosis. Kinds, stages,	
	significance.	
22.	Proliferation in inflammation. Effects of inflammation. Biological significance of	
	inflammation.	
23.	Acute phase response.	
	Fever, definition. Kinds of fever. Pyrogens, kinds, the mechanism of action. Fever	
	pathogenesis.	
25.	Allergy (hypersensitivity). Definition. Etiology. Kinds of allergens. Sensitization	
	mechanisms. Classification of allergic reactions.	
26.	Allergy reactions type I.	
	Allergy reactions type II.	
	Allergy reactions type III.	
	Allergy reactions type IV.	
30.	Tumor growth (neoplasia). Definition. Tumor growth and other hyperbiotic	
	processes. Benign and malignant tumors, comparative characteristics.	
31.	Etiology of neoplastic growth. Chemical, physical and biological carcinogens	
	effects.	
32.	Mechanism of carcinogenesis (transformation, promotion, progression). Modern	
22	conceptions about mechanisms of transformation.	
	Absolute and relative insulin deficiency. Diabetes mellitus. Disorders of acid-base balance. Acidosis. Alkalosis.	
	Causes, kinds, pathogenesis and results of hypo- and hyperhydration of the body.	
	Edema. Definition, kinds, causes, pathogenesis, significance.	
	Pathogenesis of cardiac edema.	
	Pathogenesis of renal edema.	
	Pathogenesis of edema in liver failure.	
	Hypoxia. Definition. Kinds of hypoxia. Gas content of the blood in different kinds	
	of hypoxia. Compensation mechanisms, pathological changes in the body.	
41.	Changes of blood volume. Causes, kinds, pathogenesis. Acute and chronic blood	
	loss (causes, pathogenesis, results).	
42.	Anemia. Definition. Principles of classification. Qualitative changes of	
	erythrocytes in anemia.	
	Causes, pathogenesis, blood test changes in hemolytic anemia.	
44.	Causes, pathogenesis, blood test changes in anemia, caused by erythropoesis	
	abnormalities.	
45.	Leukocytosis and leukopenia. Definitions, causes, kinds, mechanisms of	
	development. Qualitative changes of leukocytes in peripheral blood.	
46.	Leukemia. Definition. Etiology. Kinds. Peripheral blood and changes in	
47	hemopoetic organs in leukemia.	
	Hemorrhagic syndrome. Causes, pathogenesis and results.	
48.	Blood circulation failure. Definition, kinds. Hemodynamic characteristics in	
40	vascular and cardiac failure.	
	Causes, kinds and pathogenesis of heart failure.	
	Ischemic heart disease, etiology, pathogenesis, manifestations. Myocardial hypertrophy. Definition. Stages.	
	Compensation mechanisms in heart failure.	
	Hemodynamic and clinical manifestations of cardiac failure.	
55.	remoty nume and enheur mannestations of cardiae failure.	

54.	Modem conceptions about causes, kinds, and pathogenesis of symptomatic	
	hypertension and hypertensive diseases.	
55.	Insufficiency of external respiration. Kinds. Gas composition of blood in external	
	respiration insufficiency.	
	Influence of external respiration insufficiency on the organism.	
57.	Dyspnea: causes, kinds and pathogenesis.	
58.	Asphyxia and pneumothorax as causes of insufficiency of external respiration.	
59.	Causes of maldigestion. Compensation reactions of the digestive system. Modern	
	conception about causes and pathogenesis of gastric and duodenal ulcers.	
60.	Causes and results of maldigestion in the small intestine.	
61.	Causes of hepatic failure. Changes in the body in liver pathology. Hepatic coma.	
	Kinds, pathogenesis.	
62.	Disorders of bile formation and bile excretion. Jaundice.	
63.	Disorders of diuresis and urine compound in kidneys diseases.	
64.	Disorders of glomerular filtration and the function of renal tubules.	
	Acute renal failure: causes, pathogenesis, stages and outcomes.	
	Chronic renal failure: causes, pathogenesis, stages. Uremia.	
67.	General etiology and pathogenesis of endocrine disorders. Disorders of the central	
	mechanisms of endocrine glands regulation.	
68.	Causes and pathogenesis of endocrine disorders connected with the abnormalities	
	of endocrine glands proper. Disorders of feedback mechanism.	
69.	Peripheral mechanisms of the endocrine pathologies.	
	Disorders of the pituitary gland.	
	Disorders of the thyroid gland.	
	Disorders of the adrenal glands.	
	Etiology and pathogenesis of the nervous system disorders.	
	Pathology of the nervous system. Motor disorders.	
	Pathology of the nervous system. Sensor disorders.	
	Modern conceptions of pain mechanism. Kinds of pain. Effect of pain on the	
	body.	

6. Criteria for evaluating learning outcomes

For the credit (example)

Looming outcomes	Evaluation criteria			
Learning outcomes	Not passed	Passed		
Completeness of knowledge	The level of knowledge is below the minimum requirements. There were bad mistakes.	The level of knowledge in the volume corresponding to the training program. Minor mistakes may be made		
Availability of skills	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes.	Basic skills are demonstrated. Typical tasks have been solved, all tasks have been completed. Minor mistakes may be made.		
Availability of skills (possession of experience)	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes.	8		
Motivation (personal attitude)	Educational activity and motivation are poorly expressed, there is no willingness to solve the tasks qualitatively	Educational activity and motivation are manifested, readiness to perform assigned tasks is demonstrated.		

Characteristics of competence formation*	The competence is not fully formed. The available knowledge and skills are not enough to solve practical (professional) tasks. Repeated training is required	The competence developed meets the requirements. The available knowledge, skills and motivation are generally sufficient to solve practical (professional) tasks.
The level of competence formation*	Low	Medium/High

* - not provided for postgraduate programs

For the exam (example)

Learning	Assessment of competence developed				
outcomes					
	unsatisfactory	satisfactory	good	excellent	
Completeness of knowledge	The level of knowledge is below the minimum requirements. There were bad mistakes	The minimum acceptable level of knowledge. A lot of light mistakes were made	The level of knowledge in the volume corresponding to the training program. A few light mistakes were made	The level of knowledge in the volume corresponding to the training program, without errors	
Availability of skills	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes	Basic skills are demonstrated. Typical problems with light mistakes have been solved. All tasks have been completed, but not in full.	All basic skills are demonstrated. All the main tasks have been solved with light mistakes. All tasks have been completed, in full, but some of them with shortcomings	All the basic skills were demonstrated, all the main tasks were solved with some minor shortcomings, all the tasks were completed in full	
Availability of skills (possession of experience)	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes	There is a minimal set of skills for solving standard tasks with some shortcomings	Basic skills in solving standard tasks with some shortcomings are demonstrated	Skills in solving non-standard tasks without mistakes and shortcomings are demonstrated	
Characteristics of competence formation*	The competence is not fully formed. The available knowledge and skills are not enough to solve professional tasks. Repeated training is required	The formation of competence meets the minimum requirements. The available knowledge and abilities are generally sufficient to solve professional tasks, but additional	The formation of competence generally meets the requirements, but there are shortcomings. The available knowledge, skills and motivation are generally sufficient to	The formation of competence fully meets the requirements. The available knowledge, skills and motivation are fully sufficient to solve complex professional tasks	

Learning outcomes	Assessment of competence developed			
	unsatisfactory	satisfactory	good	excellent
		practice is required for most practical tasks	solve professional tasks, but additional practice is required for some professional tasks	
The level of	Low	Below	Intermediate	High
competence formation*		average		

For testing:

Mark "5" (Excellent) - points (100-90%) Mark "4" (Good) - points (89-80%) Mark "3" (Satisfactory) - points (79-70%)

Less than 70% – Unsatisfactory – Mark "2"

Developer(s): Full name, position, academic degree, academic title