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DEPARTMENT: PRECLINICAL SCIENCES

DISCIPLINE: PHYSIOPATHOLOGY

Course responsible teacher: Lecturer Marian Ghiță, DVM PhD

THE SUBJECT AND BIBLIOGRAPHY FOR LICENCING EXAMINATION, 2020

1. Shock

General knowledge. Classification of shock. The pathophysiology of shock. Hypovolemic shock. Cardiogenic shock. Dysvolemic (distributive) shock. Obstructive shock. Metabolic disorders secondary to shock. Stages of shock. Implications of the shock's metabolic disturbances at the level of organs. Pages 46-64.

Total: 18 pages (text figures and tables).

2. Inflammatory reaction

Definition, aims and etiology of inflammation. Clinical manifestation of inflammation. Naming of inflammation. Classification of inflammation. Stages of inflammatory reaction. The vascular stage. The cellular stage. The tissular repair stage. Evolution of inflammation, Systemic effects of inflammation. Pages 65-84.

Total: 19 pages (text, figures and tables).

TOTAL 37 pages (text, figures and tables).

BIBLIOGRAPHY

1.M.Ghiță, G. Cotor (2019) – General Pathophysiology, Ed. Printech, București, 2019, 194 pag., ISBN 978-606-23-1029-5

110 questions 5 appropriate answer variants. (Of these five variants, only one is correct)	
1	**Which of the following pathological manifestations is an inflammation?** a) pulmonary fibrosis; b) kidney congestion; c) encephalosis; d) hepatitis; e) muscular dystrophy.
2	**Local manifestations of the inflammatory process are:** a) lesions secondary to the action of an inflammatory agent and body's defensive reactions;

	b) tissue degenerations induced by the phlogogenic factor and protective reactions of the body; c) tissue inflammation induced by the phlogogenic factor and protective reaction of the body; d) tissue alterations and compensatory reactions of the body; e) tissue alterations and adaptive reactions of the body;
3	**The protective reactions of the body that take place during the inflammatory response are grouped into the following categories of processes:** a) vasculo-proliferative processes and alterative processes; b) vasculo-alterative processes and degenerative processes; c) exudative-vascular processes and proliferative processes; d) vasculo-degenerative processes and alterative processes; e) vasculo-alterative processes and exudative processes;
4	**Rubor is a cardinal sign of inflammation and involves:** a) the swelling of the inflamed tissue b) pain sensitivity of the inflamed tissue c) increasing the temperature of the inflamed tissue d) redness of the inflamed tissue e) all the answers are wrong
5	**Calor is a cardinal sign of inflammation and involves:** a) the swelling of the inflamed tissue; b) pain sensitivity of the inflamed tissue; c) increasing the temperature of the inflamed tissue; d) coloring of the inflamed tissue; e) decreasing the temperature of the inflamed tissue;
6	**Dolor is a cardinal sign of inflammation and involves:** a) dilation of the inflamed tissue; b) pain sensitivity of the inflamed tissue; c) increasing the temperature of the inflamed tissue; d) redness of the inflamed tissue; e) all the answers are wrong;
7	**Tumor is a cardinal sign of inflammation and involves:** a) the swelling of the inflamed tissue; b) tumor evolution of inflamed tissue; c) increasing the temperature of the inflamed tissue; d) redness of the inflamed tissue; e) pain sensitivity of the inflamed tissue;
8	**The cardinal signs' intensity of the inflammatory reactions is higher in:** a) acute and generalized forms; b) acute and well localized inflammation; c) chronic and localized forms; d) chronic and generalized forms; e) all types of inflammation;
9	**Which of the following pathologic manifestations is not an inflammation?** a) dermatitis; b) perinephritis; c) hepatitis; d)myocardosis; e) pneumonia.
10	**Which of the following pathologic manifestations is the inflammation of the hepatic capsule:** a) hepatic serositis;

	b) perihepatitis; c) perihepatosis; d) hepatitis; e) hepatosis.
11	**The vascular stage of the inflammatory reaction entails the successive unfolding of the following phases:** a) initial vasodilatation, arterio-capillary vasoconstriction and increased vascular permeability; b) initial vasoconstriction and arterio-capillary vasodilatation; c) arterio-capillary vasodilation and increased vascular permeability; d) initial vasoconstriction, arterio-capillary vasodilation and increased vascular permeability; e) vasoconstriction and increased vascular permeability.
12	**Based on the nature of the pathogenic factor involved in producing the inflammatory process, inflammation is classified as:** a) physiological and pathological inflammation; b) non-immunological and immunological inflammation; c) acute and chronic inflammation; d) septic and aseptic inflammation; e) normoergic, hyperergic and hypoergic inflammation.
13	**Chemotaxis is:** a) the feature of the pro-inflammatory cells to secrete proteolytic enzymes; b) the ability of the pro-inflammatory cells to move through the vascular bed; c) the feature of the pro-inflammatory cells to emit pseudopodia; d) the ability of pro-inflammatory cells to move towards the inflammatory center; e) the feature of the pro-inflammatory cells to perform phagocytosis.
14	**The inflammatory reaction goes through the following stages:** a) vascular, cellular and tissue repair; b) vascular, tissular and tissue repair; c) cellular, vascular and tissue repair; d) cellular, tissular and tissue repair; e) tissular, cellular and vascular.
15	**The second phase of the vascular stage in the inflammatory reaction is characterized by:** a) arteriocapillary vasodilation and venous vasodilation (postcapillary) followed by venous vasoconstriction; b) arteriocapillary vasodilation and venous vasodilation (postcapillary) followed by arteriocapillary vasoconstriction; c) arteriocapillary vasodilation accompanied by venous vasoconstriction followed by arteriocapillary vasodilation and venous vasodilation; d) arteriocapillary vasodilation and venous vasoconstriction (postcapillary) followed by arteriocapillary and venous vasoconstriction; e) arteriocapillary vasodilation followed by arteriocapillary and venous vasoconstriction.
16	**The soluble mediators of inflammation that induce the second phase of the vascular stage, which is characterized by arteriocapillary vasodilation and venous vasoconstriction (postcapillary), are:** a) histamine and bradykinin; b) histamine and PAF; c) histamine and I and E prostaglandin; d) histamine and nitric oxide; e) TNF.
17	**The manifestations of the second phase of the vascular stage in the inflammatory reaction, which are characterized by arteriocapillary vasodilation and venous vasodilation (postcapillary), are due to:**

	a) the development of an antidromic reflex; b) the catecholamines; c) the development of a parasympathetic reflex; d) some soluble mediators of inflammation; e) acetylcholine.
18	**The second phase of the vascular stage in the inflammatory reaction lasts about:** a) 2 hours; b) 6 hours; c) 12 hours; d) 24 hours; e) 36 hours.
19	**The activation of the complement system takes place during the:** a) cellular stage of the inflammatory reaction; b) tissular stage of the inflammatory reaction; c) vascular stage of the inflammatory reaction; d) tissue reconstruction stage of the inflammatory reaction; e) vasculo-tissue stage.
20	**The third phase of the vascular stage of the inflammatory reaction is characterized by:** a) increasing the vascular permeability; b) hemorrhage; c) arteriocapillary and venous (postcapillary) vasoconstriction, followed by arteriocapillary vasodilation; d) diapedesis; e) pooling.
21	**The third phase of the vascular stage of the inflammatory reaction is induced, among others, by:** a) nitric oxide and cytokines; b) catecholamines and leukotrienes; c) histamine and bradykinin; d) PAF and cytokines; e) nitric oxide and histamine.
22	**Plasma leakage which follows the third phase of the vascular stage in the inflammation reactions, induces:** a) formation of the inflammatory transudate and edema; b) intratissular accumulation of inflammatory exudate; c) formation of the inflammatory transudate and stasis; d) formation of inflammatory exudate and ischemia; e) formation of the inflammatory transudate.
23	**Vascular stasis (venous congestion) is characterized by a decreased blood flow and the stagnation thereof in the affected area, an occurrence called:** a) sludge; b) Disseminated Intravascular Coagulation (DIC); c) passive hyperemia; d) pooling; e) congestion.
24	**Which of the following modifications are not specific to the septic inflammation:** a) increasing the body temperature; b) increasing level of immunoglobulin; c) increasing the number of red blood cells; d) increasing erythrocyte sedimentation rate (ESR); e) leukocytosis.

25	**Which of the following substances do not belong to the second line of soluble mediators of inflammation:** a) PAF; b) PgE and PgI; c) leukotrienes; d) histamine; e) $TbA_{2.}$
26	**Which of the following belong to the proinflammatory cells:** a) neutrophilic granulocytes; b) blood platelets; c) basophilic granulocytes; d) mast cells; e) lymphocytes.
27	**The endothelial cells functionally support the proinflammatory cells by:** a) releasing PAF and prostaglandins; b) releasing coagulation factors; c) releasing histamine; d) releasing heparin; e) releasing TNF.
28	**Proinflammatory cells from the tissue compartment act, among others, through:** a) chemotaxis and chemokinesis; b) leukocyte margination; c) reversible adherence on the vascular endothelium level; d) irreversible adherence on the vascular endothelium level; e) platelet adherence.
29	**The first chemotactic wave is characterized by:** a) duration of approximately 2-4 hours sustained by macrophages; b) duration of approximately 2-4 hours sustained by neutrophils; c) duration of approximately 36 hours sustained by macrophages; d) duration of approximately 36 hours sustained by neutrophils; e) duration of approximately 36 hours sustained by lymphocytes.
30	**The tissue repair stage of the inflammatory reaction is characterized by:** a) maintaining of the vasculo-exudative processes; b) intensification of proliferative phenomena; c) intensification of the macrophages' action; d) intensification of the vasculo-exudative processes; e) intensification of the lymphocytes' action.
31	**One of the two essential shock inducing elements is:** a) decreasing the cardiac frequency; b) decreasing the amplitude of cardiac contractions; c) decreasing the tissue perfusion; d) decreasing hematosis; e) increasing hematosis.
32	**Hypovolemic shock can be induced by:** a) cardiac arrhythmia; b) pulmonary embolism; c) severe plasmorrhagia; d) valvular insufficiency; e) valvular stenosis.
33	**Which of the following mechanisms are not activated during the shock with the tendency to restore the circulating blood volume?**

	a) mobilization of blood stored in venous deposits; b) secondary hyperaldosteronism; c) increase water ingestion; d) hyper-secretion of ADH; e) intravasation of interstitial water.
34	**Disvolemic (distributive) shock can be induced by:** a) acute intoxication with depressants; b) pulmonary embolism; c) pneumothorax; d) cardiomyopathies; e) plasmorrhagia.
35	**Obstructive shock can be induced by:** a) depressor drugs intoxications; b) massive pulmonary embolism; c) allergies; d) cardiomyopathies; e) massive plasmorrhagia.
36	**In hypovolemic compensated shock it is noticed:** a) vasoconstriction induced by the closing of pre- and post-capillary sphincters and opening of arteriolo-venular shunts; b) pre and post capillary vasoconstriction and the closing of arterio-venous shunts; c) pre and post capillary vasodilatation and the opening of arterio-venous shunts; d) pre and post capillary vasodilatation and the closing of arterio-venous shunts; e) venous vasoconstriction and opening of capillary shunts.
37	**In decompensated hypovolemic shock it is noticed:** a) acidosis and closing of the pre and post capillary sphincters; b) acidosis and opening of precapillary sphincters; c) alkalosis and closing of pre and post capillary sphincters; d) alkalosis and opening of pre and post capillary sphincters; e) alkalosis and closing of venous sphincters.
38	**Consecutively to the increasing of the vascular permeability, in the hypovolemic decompensated shock, it is noticed:** a) plasma leakage and haemodilution; b) ischemia and hemodilution c) plasma leakage and increased blood viscosity; d) ischemia and haemoconcentration; e) pulmonary embolism.
39	**In the decompensated stage of hypovolemic shock, posthypoxic lesions and increased blood viscosity cause:** a) embolism; b) increasing the tissue perfusion; c) increasing the arterial tension; d) decreasing the cardiac frequency; e) Disseminated Intravascular Coagulation (DIC).
40	**In the decompensated hypovolemic shock it is noticed:** a) coronary hyperperfusion and amplification of the cardiac debit; b) diminution of cardiac output and general hypoperfusion; c) coronary hyperperfusion and diminution of the cardiac debit; d) coronary hypoperfusion and amplification of the cardiac debit; e) pulmonary hyperperfusion and amplification of the cardiac debit.
41	**Disorders of carbohydrate metabolism secondary to shock consist in:**

	a) early post-aggressive hypoglycemia and, during the late stages of shock, hyperglycemia; b) hypoglycemia all the time; c) early post-aggressive hyperglycemia and, during the late stages of shock, hypoglycemia; d) the level of glycemia is not modified during the shock evolution; e) hyperglycemia all the time.
42	**The refractory shock (irreversible) is characterized by:** a) grave tissue acidosis; b) hypoglycemia; c) multiple organ failure; d) DIC; e) all the answers are correct.
43	**Which of the following modifications isn't specific for disorders of protein metabolism secondary to shock:** a) intensification of protein catabolism; b) decreased plasma protein levels; c) increased serum levels of certain compounds of protein catabolism; d) increased plasma protein levels; e) occurrence of harmful peptide compounds.
44	**In the renal failure induced by the decompensated shock it is noticed:** a) haematuria; b) anuria; c) haemoglobinuria; d) polyuria; e) ketonuria.
45	**During the compensatory stage of hypovolemic shock, it is noticed:** a) bradycardia and diminution the force of heart's contraction; b) bradycardia and increasing the force of heart's contraction; c) an intensification of cardiac activity and an amplification of cardiac output; d) tachycardia and diminution the force of heart's contraction; e) cardiac tamponade.
46	**In the decompensated shock it is noticed:** a) hyperglycemia and increasing the hepatic metabolism of lipids; b) hypoglycemia and decreasing the hepatic metabolism of lipids; c) hyperglycemia and accumulation of the lipids in the liver (fat overload); d) hypoglycemia and fat overload of the liver (hepatocellular failure); e) hyperglycemia and accumulation of lipids in the kidney (fat overload).
47	**In decompensated shock, regarding the hydro-mineral metabolism it is noticed:** a) water and Na^+ retaining at the vascular level; b) water is transferred inside cells due to intracellular accumulation of Na^+; c) water transfer from the interstitial space to the vascular space; d) the level of Na^+ inside the cells remain constantly; e) Na^+ transfer in the vascular space.
48	**In the compensated shock it is noticed:** a) extracellular transfer of K^+ and intracellular transfer of Ca^{++}; b) intracellular transfer of K^+ and Ca^{++}; c) intracellular transfer of K^+ and extracellular transfer of Ca^{++}; d) extracellular transfer of K^+ and Ca^{++}; e) all the answers are wrong.
49	**Which of the following modifications are not characteristic for hepatic failure that is instituted in the decompensated phase of shock.** a) stoppage of gluconeogenesis;

	b) annihilation of the liver's antitoxic function; c) annihilation of the function of metabolization of biliary pigments (increase bilirubinemia); d) amplification of protein synthesis; e) amplification of fibrinolysis.
50	**The cause for intracellular Ca^{++} accumulation, specific for decompensated shock, is:** a) functional blocking of the Ca^{++} pumps; b) inactivation of the Na^{+}/K^{+} pumps; c) activation of the Ca^{++} pumps; d) accumulation of water inside the cells; e) all the answers are wrong.
51	**In compensated shock it is noticed:** a) hyperglycemia and activation of lipolysis; b) hypoglycemia and intensification of the hepatic metabolism of lipids; c) hyperglycemia and accumulation of lipids in the liver (fat overload); d) hypoglycemia and accumulation of lipids in the liver (fat overload); e) hypoglycemia and accumulation of lipids in the kidney (fat overload).
52	**Plasma leakage, consecutively to the increasing of the vascular permeability in the hypovolemic shock, induces:** a) hypovolemia; b) haemorrhage; c) increasing of the arterial pressure; d) haemodilution; e) thrombosis.
53	**The tissue repair stage of the inflammatory reaction is composed of the next successive phases:** a) angiogenesis – specific tissue reconstruction – tissue remodeling – fibroplasia; b) angiogenesis – tissue remodeling – fibroplasia – specific tissue reconstruction; c) fibroplasia – angiogenesis - specific tissue reconstruction – tissue remodeling; d) fibroplasia – specific tissue reconstruction – angiogenesis – tissue remodeling; e) fibroplasia – tissue remodeling – angiogenesis – specific tissue reconstruction.
54	**The second chemotactic wave is characterized by:** a) duration of approximately 2-4 hours sustained by macrophages; b) duration of approximately 2-4 hours sustained by neutrophils; c) duration of approximately 36 hours sustained by macrophages; d) duration of approximately 36 hours sustained by neutrophils; e) duration of approximately 36 hours sustained by lymphocytes.
55	**The major effect of the third phase of the vascular stage in the inflammatory reaction is characterized by:** a) congestion; b) plasma leakage; c) hemodilution; d) erythema; e) acidosis.
56	**Proinflammatory cells of the circulant compartment act, among others, through:** a) chemotaxis and chemokinesis; b) irreversible adherence on the vascular endothelium level; c) maturation and multiplication; d) phagocytosis; e) pinocytosis.
57	**Acute inflammatory reactions are characterized by:** a) discreet manifestation of the cardinal signs of inflammation;

	b) predominance of the vasculo-exudative processes; c) long-term evolution; d) predominance of the proliferative processes; e) all the answers are wrong.
58	**The tissue repair stage of the inflammatory reaction is characterized by:** a) diminution of vasculo-exudative phenomena; b) diminution of the proliferative processes; c) intensification of the macrophages' action; d) intensification of the neutrophils' action; e) intensification of the vasculo-exudative processes.
59	**Proinflammatory cells of the tissue compartment act, among others, through:** a) maturation and multiplication; b) leukocyte margination; c) irreversible adherence on the vascular endothelium level; d) oxygen dependent cytotoxicity. e) diapedesis.
60	**Chronic inflammatory reactions are characterized by:** a) discreet manifestation of the cardinal signs of inflammation; b) predominance of the vasculo-exudative processes; c) short-term evolution; d) predominance of degenerative processes; e) all the answers are wrong.
61	**One of the two essential shock inducing elements is:** a) tissue hypoxia; b) blood stasis; c) tissue ischemia; d) tissue hyperemia; e) tissue hyperoxia.
62	**The activation of the coagulase system takes place during the:** a) cellular stage of the inflammatory reaction; b) hemorrhagic-tissue stage of the inflammatory reaction; c) vascular stage of the inflammatory reaction; d) tissue reconstruction stage of the inflammatory reaction; e) vasculo-tissue stage.
63	**In the decompensated shock of gastrointestinal tract (GIT) (organ failure) we can find:** a) vasoconstriction; b) amplification of motor and secretory functions of the GIT; c) lesions, overlapping infections and toxiemia; d) embolism; e) all the answers are wrong.
64	**The third phase of the vascular stage of the inflammatory reaction is induced, among others, by:** a) hypoxia and consecutive acidosis; b) catecholamines; c) nitric oxide; d) PAF; e) acetylcholine.
65	**The inflammatory reaction goes through the following stages:** a) vascular- cellular and tissue repair; b) vascular-tissular and tissue repair; c) cellular-vascular and tissue repair;

	d) cellular-tissular and tissue repair; e) cellular-vascular-tissular.
66	**The first phase of the vascular stage in the inflammatory reaction is characterized by:** a) arteriocapillary vasodilation; b) arteriocapillary vasoconstriction; c) venous vasoconstriction (postcapillary); d) venous vasodilation; e) all the answers are wrong.
67	**In the decompensated stage of hypovolemic shock it is noticed:** a) blood stasis, that induces relative hypervolemia; b) embolism, that induces blood stasis; c) blood stasis, that induces relative hypovolemia; d) blood stasis, that induces increase of arterial tension; e) thrombosis and embolism.
68	**In the decompensated stage of hypovolemic shock, it is noticed:** a) haemodilution; b) ischemia; c) hyperpermeabilization of the vascular endothelium; d) pulmonary embolism; e) all the answers are wrong.
69	**Which of the following clinical signs are not specific to the inflammations:** a) dolor; b) tumor; c) necrosis; d) calor; e) rubor.
70	**The manifestations of the first phase of the vascular stage in the inflammatory reaction is due to:** a) thromboxane $A_{2,}$ serotonin and catecholamines; b) PAF; c) histamin; d) bradykinin; e) citokines.
71	**Proinflammatory cells of the tissue compartment act, among others, through:** a) maturation and multiplication; b) leukocyte margination; c) phagocytosis; d) irreversible adherence on the vascular endothelium level; e) viscous metamorphosis.
72	**Which of the following also belong to the proinflammatory cells:** a) endothelial cells; b) monocytes - macrophages; c) basophilic granulocytes; d) mast cells; e) lymphocytes.
73	**What kind of inflammation are those caused by biotic phlogogenic factors:** a) biogenic; b) septic; c) hyperergic; d) immunologic; e) acute.

74	**The aims of the inflammatory reaction are:** a) healing lesions and eliminating the pathogenic agent and the negative effects it has produced; b) stimulating of hematopoiesis; c) increase volemia; d) increasing the blood coagulability; e) to induce chronic diseases.
75	**A morphofunctional structure is formed at the periphery of the inflammatory outbreak; its role is to limit the diffusion, it is called:** a) fibrinous barrier; b) immune-leukocyte barrier; c) fibrin-leukocyte barrier; d) fibrin-immuno-leukocyte barrier; e) hemato-fibrinous barrier.
76	**Early or initial shock (reversible) corresponds to the immediate imbalance phase of the SRA and is characterized by:** a) low arterial blood pressure; b) metabolic alkalosis; c) tissue hyperperfusion; d) mitochondrial hyperfunction; e) all the answers are wrong.
77	**Which of the following are cells that functionally support the proinflammatory cells:** a) eosinophil granulocytes; b) endothelial cells; c) macrophages; d)neutrophil granulocytes; e) lymphocytes.
78	**Which of the following are soluble mediators of inflammation that are involved in inducing the second phase of the vascular stage, characterized by arteriocapillary and venous (postcapillary) vasodilation:** a) histamine, catecholamines and bradykinin; b) histamine, cytokines and PAF; c) bradykinin, nitric oxide and cytokines; d) histamine, bradykinin and I and E prostaglandin; e) TNF and nitric oxide.
79	**Which of the following substances do not belong to the second line of soluble mediators of inflammation:** a) PAF; b) Pg E and Pg I; c) Tb A_2; d) leucotrienes; e) nitric oxide.
80	**Which of the following cells is specialized in phagocytosis of antigen-antibody complexes?** a) macrophages; b) monocytes; c) neutrophils; d) eosinophils; e) lymphocytes.
81	**The manifestations of the second phase of the vascular stage in the inflammatory reaction, which are characterized by arteriocapillary vasodilation and venous vasoconstriction (postcapillary), are due to:**

	a) the development of an antidromic reflex and some soluble mediators of inflammation; b) the catecholamines and some soluble mediators of inflammation; c) some soluble mediators of inflammation; d) the development of a vascular reflex and some soluble mediators of inflammation; e) the acetylcholine and some soluble mediators of inflammation.
82	**Which of the following substances do not belong to the third line of soluble mediators of inflammation:** a) bradykinin; b) TNF; c) interferon; d) interleukins; e) nitric oxide.
83	**Acute inflammatory reactions are characterized by:** a) less manifestation of the cardinal signs of inflammation; b) predominance of the proliferative processes; c) long-term evolution; d) short-term evolution; d) all the answers are wrong.
84	**Mast cells and basophiles functionally sustain the proinflammatory cells through:** a) release of PAF and prostaglandins; b) release of coagulation factors; c) release of histamine; d) release of complement factors; e) release of TNF.
85	**Which of the following features does not belong to the soluble mediators of inflammation :** a) attracting pro-inflammatory cells; b) activating pro-inflammatory cells; c) inducing vasodilation; d) increasing vascular permeability; e) inactivating the adhesion receptors found on the membranes of pro-inflammatory and endothelial cells.
86	**In the liver of decompensated shock (organ failure), it is noticed:** a) hypercoagulable status; b) amplification of the antitoxic function; c) gluconeogenesis amplification; d) diminution of protein synthesis; e) all the answers are wrong.
87	**Disorders of lipid metabolism secondary to shock consist in the activation of lipolysis via certain catabolic hormones such as:** a) noradrenaline, glucocorticoids, glucagon and iodine thyroid hormones; b) noradrenaline, glucocorticoids and growth hormone; c) iodine thyroid hormones, insulin and adrenaline; d) noradrenaline, insulin and iodine thyroid hormones; e) glucocorticoids, insulin and iodine thyroid hormones.
88	**In decompensated shock, the intracellular accumulation of Ca^{++} determines:** a) cellular edema; b) cardiac arrhythmias; c) the activation of endonuclease involved in programmed cell death (apoptosis); d) hypovolemia; e) calcinosis.

89	**In decompensated shock, the extracellular K^+ accumulation determines:** a) cellular edema; b) cardiac arrhythmias; c) activation of cellular proteases; d) tahipneea; e) cellular dehydration.
90	**In the decompensated shock, the intracellular accumulation of Na^+ determines:** a) cellular edema; b) cardiac arrhythmias; c) cellular proteases activation; d) cellular dehydration ; e) generalized edema.
91	**The cause of cellular edema, specific for decompensated shock is:** a) the inactivation of the calcium pumps; b) functional impairment of Na^+/K^+ pumps; c) the activation of the calcium pumps; d) microlessions at the plasmalema level; e) hyperactivity of the Na^+/K^+ pumps.
92	**In the lung of decompensated shock (organ failure), it can be found:** a) pulmonary ischemia; b) blockage of the pulmonary microcirculation with formation of thrombi (DIC) and stasis in the pulmonary microcirculation; c) pulmonary emphysema; d) pneumonia; e) pulmonary bleeding.
93	**In the lung of decompensated shock (organ failure), it can't be found:** a) pulmonary edema; b) blockage of the pulmonary microcirculation with formation of thrombi (DIC); c) stasis in the pulmonary microcirculation; d) pulmonary obstruction due to the desquamation of cells (cellular destruction); e) pulmonary bleeding.
94	**In the liver of decompensated shock (organ failure), it can be found:** a) diminution of the synthesis of coagulation factors; b) amplification of the liver's antitoxic function; c) amplification of gluconeogenesis; d) diminution of fibrinolysis; e) amplification of protein synthesis.
95	**Cardiogenic shock cannot be induced by:** a) severe arrhythmias, b) pulmonary embolism; c) extensive myocardial infarction; d) valvular insufficiency; e) cardiomyopathy.
96	**Dysvolemic (distributive) shock can be induced by:** a) bacterial endotoxins; b) pulmonary embolism; c) plasmorrhagia; d) kidney faillure; e) cardiac failure.
97	**Obstructive shock can be induced by:** a) myocardial infarction;

	b) allergies; c) hemorrhages; d) pneumothorax; e) bacterial endotoxins.
98	**Obstructive shock cannot be induced by:** a) cardiac tamponade; b) massive pulmonary embolism; c) hemorrhages; d) pneumothorax; e) tumors of the lung.
99	**In hypovolemic compensated shock, it is noticed:** a) mobilization of blood stored in venous deposits; b) extravasation of interstitial water due to a reduction in the hydrostatic pressure in capillaries; c) amplification of the sanguine reserves of the hematopexic organs; d) secondary hypoaldosteronism; e) bradycardia and bradypnea.
100	**In the decompensated shock, it is noticed:** a) hyperkalemia; b) calcium extracellular accumulation; c) intracellular K^+ transfer and extracellular Ca^{++} transfer; d) extracellular transfer of K^+ and Ca^{++}; e) all the answers are wrong.
101	**Which of the following isn't specific to refractory shock (irreversible):** a) grave tissular acidosis; b) hyperglycemia; c) activation of hydrolase and onset of tissular necroses; d) multiple organ failure; e) DIC.
102	**Which of the following isn't specific to late compensated shock (reversible):** a) catecholamine release (a consequence of arterial hypotension); b) hyperventilation (resulting from metabolic acidosis); c) increased cardiac frequency and increased vasoconstriction; d) poliuria; e) diminution of venous return.
103	**Which of the following isn't specific to late decompensated shock (reversible):** a) vasodilation and stasis (pooling); b) low arterial blood pressure; c) plasma leakage; d) onset of organ insufficiencies; e) intensification of aerobic glycolysis.
104	**Based on the pathological implications of the inflammatory process, the inflammation is classified as:** a) physiological inflammation and pathological inflammation; b) non-immunological inflammation and immunological inflammation; c) septic inflammation and aseptic inflammation; d) acute inflammation and chronic inflammation; e) normoergic inflammation, hyperergic inflammation, and hypoergic inflammation.
105	**Based on the involvement of the mechanisms of specific immunity, the inflammation is classified as:** a) physiological inflammation and pathological inflammation;

	b) non-immunological inflammation and immunological inflammation; c) septic inflammation and aseptic inflammation; d) acute inflammation and chronic inflammation; e) normoergic inflammation, hyperergic inflammation, and hypoergic inflammation.
106	**Based on the nature of the pathogenic factor involved in producing the inflammatory process, inflammation is classified as:** a) physiological inflammation and pathological inflammation; b) non-immunological inflammation and immunological inflammation; c) septic inflammation and aseptic inflammation; d) acute inflammation and chronic inflammation; e) normoergic inflammation, hyperergic inflammation, and hypoergic inflammation.
107	**Based on the evolutive pattern, inflammation can be:** a) physiological inflammation and pathological inflammation; b) non-immunological inflammation and immunological inflammation; c) septic inflammation and aseptic inflammation; d) acute inflammation and chronic inflammation; e) normoergic inflammation, hyperergic inflammation, and hypoergic inflammation.
108	**Based on the body's reactivity, inflammation can be:** a) physiological inflammation and pathological inflammation; b) non-immunological inflammation and immunological inflammation; c) septic inflammation and aseptic inflammation; d) acute inflammation and chronic inflammation; e) normoergic inflammation, hyperergic inflammation, and hypoergic inflammation.
109	**Chemokinesis is the ability of pro-inflammatory cells:** a) to migrate from the vascular bed towards the inflammatory center; b) to increase vascular permeability; c) to adhere to vascular walls; d) to do diapedesis; e) to produce soluble mediators of inflammation.
110	**Which of these isn't a direct effect of PAF:** a) amplifies vascular permeability; b) chemotactic; c) inhibit PMN's activity; d) stimulates lymphocytes; e) stimulates thrombocytes.

10.01.2021 Șef lucr. dr. Marian Ghiță