МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ

УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ «ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Кафедра патологической физиологии

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ТЕСТОВЫЕ ЗАДАНИЯ ПО ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ

Учебно-методическое пособие для иностранных студентов, обучающихся на английском языке

TEST TASKS ON PATHOLOGICAL PHYSIOLOGY

The educational-methodical work for foreign students educates on English language

> Гомель ГГМУ 2009

УДК 616-092 (042.3) = 20 ББК 52.52 Б 24

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Б 24 Тестовые задания по патологической физиологии: учеб.-метод. пособие для иностранных студентов, обучающихся на английском языке / Я. А. Баранчук, О. А. Ермакова; пер. А. А. Шиханцова, О. А. Ермакова; под. ред. Т. С. Угольник = Test tasks on pathologic physiology: The educational-methodical work for foreign students educates on English language / Y. A. Baranchuk, O. A. Ermakova; tr. A. A. Shihantsova, O. A. Ermakova; edited by T. S. Ugolnik. — Гомель: учреждение образования «Гомельский государственный медицинский университет», 2009. — 116 с.

ISBN 978-985-506-241-8

Составлены в соответствии с типовой «Программой по патологической физиологии для студентов лечебно-профилактического факультета высших медицинских учебных заведений», утвержденной Министерством здравоохранения Республики Беларусь 3 сентября 1997 г., № 08 14/5927. Решение этих заданий позволит углубить и закрепить знания студентов при изучении патологической физиологии.

Утверждено и рекомендовано к изданию Центральным учебным научно-методическим советом учреждения образования «Гомельский государственный медицинский университет» 2 апреля 2008 г., протокол № 4.

УДК 616-092(042.3) ББК 52.5

ISBN 978-985-506-241-8

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INTRODUCTION. GENERAL DOCTRINE ABOUT DESEASE. HEREDITY AND PATHOLOGY

Specify a correct variant of the answer

1. Nosology includes the following sections:

a) doctrine about typical forms of pathology of organs and tissues;

b) doctrine about typical changes of structure of organs and tissues in conditions of pathology;

c) general doctrine about disease;

d) doctrine about typical pathological processes.

2. The etiologic factor of disease is:

a) factor influencing on severity and duration of disease;

b) factor which is necessary for development of disease;

c) factor raising frequency of occurrence of disease;

3. It does not concern to typical pathological processes:

- a) anemia;
- b) inflammation;
- c) fever;
- d) hypoxia;
- e) allergy.

4. Choose the incorrect statement:

a) pathological process doesn't always lead to the development of disease;

b) disease cannot arise without pathological process;

c) concepts «pathological process» and «disease» are identical;

d) the same pathological process can be a component of various diseases.

5. Choose the most exact characteristic of pathological process:

a) the process arising in the organism on a constant action of pathogenic factor;

- b) qualitatively original combination of processes of damage and adaptation;
- c) set of protectively-adaptive reactions, whose intensity exceeds the norm.

6. It refers to pathological reactions:

- a) hypoxia;
- b) trauma;
- c) burn;
- d) tumour;
- e) pathological reflex.

7. Pathological reaction:

a) develops on the action of only extreme stimulus;

b) it is a biologically inadequate answer of the organism;

c) it is an original form of the adaptation of the organism to conditions of existence.

8. Choose the true statement:

a) concepts «pathological process» and «disease» are absolutely equivalent;

b) concepts «pathological process» and «disease» essentially differ;

c) on some cases we name a disease a pathological process, and pathological process is named as a disease.

9. Choose the most exact statement. Disease is a result of:

a) actions on of pathogenic factor on the organism;

b) interaction of etiologic factor and the organism;

c) decrease in adaptive opportunities of the organism;

d) acute change of conditions of existence of the organism.

10. Name the form of pathology which is not reffered to complications of the basic disease:

a) hypostasis of lungs in left heart failure;

b) syndrome of disseminated intravascular blood clotting in plural traumas of soft tissues;

c) pneumonia in the condition of immunodeficiency;

d) chronic glomerulonephritis after suffered quinsy;

e) insult in atherosclerosis.

11. Choose the correct statement:

a) disease is a qualitatively new stage of development of pathological process;

b) disease is a qualitatively new condition of organism in which the new reactions which are absent in a healthy organism are formed;

c) disease does not create anything qualitatively new in the organism, it arises on the background of preservation of existing functional interrelations in the organism.

12. Specific features of disease depend on:

a) reasons of disease;

b) conditions promoting the development of disease;

c) reactance of the organism.

13. The vicious circle in pathogenesis of diseases is:

a) transition of primarily arisen acute phase in chronic form with periods of aggravation and remission;

b) cyclic current of disease in case of which every new cycle differs from the previous one by progressing rising of expressiveness of frustration;

c) occurrence of positive feedback between separate parts of pathogenesis, promoting the progression of disease.

14. The basic etiologic factor of acute mountain disease is:

- a) decrease in barometric pressure;
- b) decrease in partial pressure of O_2 in the air;
- c) ultra-violet radiation;
- d) low temperature.

15. Deenergizing of thermotax reactions in deep hypothermy is observed owing to inhibition:

- a) cortex of the brain;
- b) thalamus;
- c) limbic structures;
- d) extrapyramidal centers;
- e) hypothalamus.

16. Specify category of pathology to which congenital dislocation of hip is referred:

a) diseases;

- b) pathological process;
- c) pathological condition;
- d) pathological reaction.

17. Specify category of pathology to which congenital lubfoot is reffered:

- a) diseases;
- b) pathological process;
- c) pathological condition;
- d) pathological reaction.

18. Reactance of the organism is a:

a) property of the organism to perceive the action of factors of the environment;

b) property of the organism to resist to action of factors of external and internal environment;

c) property of the organism definitely to react to influence of factors of external and internal environment.

19. Specify the types of body constitution corresponding to M. V. Chernorutski classification:

a) asthenic, fibrous, lypomatous, pastose;

b) choleric persons, sanguine persons, phlegmatic persons, melancholiacs;

c) respiratory, digestive, muscular, brain;

d) asthenic, hypersthenic, normsthenic;

e) impetuous, fast, inert, weak.

20. Specify functional features corresponding to hypersthenic type of body constitution on M. V. Chernorutski:

a) blood pressure is lower;

b) vital capacity of lungs is lower;

c) hypochlorydria;

d) stomach hypermotility;

e) hyperfunction of the thyroid gland and the hypophysis;

f) hypofunction of sex glands and adrenal glands.

21. Specify the position most fully describing the concept of «pathogenesis»:

a) doctrine about mechanisms of occurrence, current and outcome of diseases;

b) doctrine about the reasons and conditions of occurrence of diseases;

c) concrete mechanisms of development of pathological processes;

d) doctrine about typical pathological processes;

e) doctrine about typical forms of pathology of organs.

22. Specify the name of poorly changing impairments of structure and function of the organ (tissue) refers to:

a) pathological reaction;

b) pathological process;

c) pathological condition;

d) disease.

23. It testify about hereditary character of disease:

a) high concordance of diseases in the hetero-ovular twins living in identical conditions;

b) high concordance of illnesses in the mono-ovular twins living in different, sharply contrasting conditions;

c) low concordance of disease in the mono-ovular twins living in different conditions.

24. Specify a set of sex chromosomes, characteristic of Turner syndrome:

a) YO;
b) XY;
c) XXX;
d) XX;
e) XO;
f) XXY;
g) XXXY.

25. Choose karyotype, characteristic of Kleinfelter's syndrome:

- a) 22 pairs of autosomes + XO;
- b) 22 pairs of autosomes + XX;
- c) 22 pairs of autosomes + XXY;
- d) 22 pairs of autosomes + XXX;
- e) 23 pairs of autosomes + YO.

26. Specify conformity of karyotype to Turner's syndrome:

- a) XO;
- b) XXУ;
- c) trisomy on 21 autosomes;
- d) XXX.

27. Specify conformity of karyotype to Down disease:

- a) XO;
- b) XXУ;
- c) trisomy on 21 autosomes;
- d) XXX.

28. Choose the correct statement:

- a) in case of Turner's syndrome there is one Barr's corpuscle;
- b) in case of Kleinfelter's syndrome there are two Barr's corpuscles;
- c) in case of trisomy X syndrome there are two Barr's corpuscles;
- d) in case of Down syndrome there are three Barr's corpuscles.

29. Specify the uncharacteristic attribute of Down syndrome:

- a) dementia;
- b) mongoloid type of the face;
- c) decrease in immunity;
- d) reduction of the brain size;
- e) «a simian crease» on the palm;
- f) high frequency of occurrence of leukosis.

30. Specify the form of pathology which is inherited on an autosomedominant type:

- a) phenylketonuria;
- b) myopia;
- c) daltonism.

31. Specify the form of pathology which is inherited on a dominant type:

- a) polydactylia;
- b) albinism;
- c) alcaptonuria;
- d) daltonism.

32. Specify the form of pathology which is inherited on the recessive type of X-linked chromosome:

a) phenylpiruvic oligophrenia;

b) Marfan's syndrome;

c) hemophilia A;

d) hemophilia C.

33. Specify an attribute of congenital diseases with the changed genetic program:

a) it is shown in a family tree not less than during two generations;

b) there are anomalies in the genetic program of the patient;

c) there are no anomalies in the genetic program, but the mechanism of transmission of hereditary information is broken;

d) only as result of anomalies of sex chromosomes;

e) only in case of anomalies of autosomes.

34. Specify the disease, in which its hereditary predisposition plays an important role in occurrence and development:

a) hemophilia A;

- b) hemophilia C;
- c) insulin independent diabetes;

d) albinism.

35. Specify disease which is referred to sex linked hereditary:

a) alkaptonuria;

b) polydactylia;

c) albinism;

d) Down syndrome;

e) hemophilia A;

f) phenylketonuria.

36. Specify the agent, capable to cause mutations of genes:

a) hypertonic solution of NaCl;

b) oncoprotein;

c) denaturated protein;

d) formaldehyde;

e) urea.

37. Point the syndrome developing in case of abnormalities of divergence of sex chromosomes:

a) Down syndrome;

b) Kleinfelter's syndrome;

c) Marfan's syndrome;

d) hemophilia A;

e) hemophilia B.

38. Penetrance of pathological gene is:

a) severity of its clinical manifestation;

b) plurality of manifestation of mutation of the same gene;

c) probability of phenotypical manifestations of a gene.

39. Specify the disease with a polygenic type of inheritance:

a) hemophilia;

b) alkaptonuria;

c) phenylketonuria;

d) stomach ulcer;

e) Down syndrome.

40. Choose the correct statement:

a) the gene, defining dominant pathology, can be contained in the genotype of phenotypicallyly healthy persons;

b) the gene, defining recessive pathology, can be contained in the genotype of phenotypically healthy persons;

c) recessive pathology is always shown in one generation;

d) dominant pathology can miss a number of generations.

41. In stage of decompensation (the period of excitation) of exogenous overheatings, heat production changes into the side of:

a) increasing;

b) decreasing;

c) initial decreasing with the subsequent increasing;

d) does not change.

42. Etiology is a:

a) doctrine about the reasons and conditions of occurrence of disease;

- b) doctrine about mechanisms of the development of disease;
- c) doctrine about a set of the conditions causing the development of diseases.

43. The basic part of pathogenesis of diseases is a:

a) primary damage of the organism;

- b) the damage causing the occurrence of vicious circles;
- c) the damage causing the majority of disease manifestations;
- d) reasons and conditions of occurrence of disease;
- e) damages, which are irreversible.

44. On an autosome-dominant type of transmission of hereditary diseases the parents can have phenotypic healthy children:

a) when both of them are homozygotes by abnormal attribute;

b) when both of them are heterozygotes by abnormal attribute;

c) when one of them is a homozygote by abnormal attribute, and another one is heterozygote;

d) when one of them is a homozygote by abnormal attribute, and another one is healthy.

45.The vicious circle in pathogenesis of disease means:

a) exhaustion of compensatory mechanisms leading to deterioration of condition;

b) occurrence of any pathological reaction;

c) gradual change of stages of disease;

d) aggravation of any part of pathogenesis as a result of arising reactions of the organism;

e) the sequence of terminal conditions.

46. Pressure of blood in the arch of the aorta of the longitudinal overload:

a) increases;

b) decreases;

c) does not change.

47. Mutagen is named:

a) the substance, capable to cause condition of the raised sensitivity in man;

b) the factor, capable to cause changes in genic structure of biological object which are hereditary transmitted.

48. The primary part of pathogenesis of diseases is:

a) conditions of action of damaging factor on the organism;

- b) the initial damage leading to further pathological changes in the organism;
- c) part of pathogenesis with which the vicious circle begins;

d) a primary stage of terminal conditions;

e) a primary reaction of the organism to a damage.

49. Specify the basic part of pathogenesis on diabetes:

a) stress;

- b) absolute or relative insufficiency of insulin;
- c) hyperglycemia on sugar reception;
- d) obstruction of biliary ducts with a stone;
- e) polyuria.

ETIOLOGY AND PATHOGENESIS OF DAMAGE OF A CELL

Specify a correct variant of the answer

1. Specify parameters of damage of a cell:

- a) increase in extracellular concentration of ions of potassium;
- b) increase in endocellular concentration of ions of potassium;
- c) reduction of endocellular concentration of ions of calcium;
- d) reduction of endocellular concentration of ions of sodium.

2. Specify «specific»manifestation of damage of a cell:

- a) denaturation of molecules of protein;
- b) strengthening of peroxidations of lipids;
- c) acidosis;
- d) labilization of membranes of lysosomes;
- e) dissociation of processes of oxidation and phosphorylation;
- f) deletion of chromosomes;
- g) swelling of a cell.

3. Specify an attribute, characteristic of apoptosis of cells:

- a) chaotic breaks of DNA;
- b) splitting of DNA in strictly certain sites;
- c) perhydration of cells.

4. Specify an attribute, uncharacteristic of apoptosis of cells:

- a) chromatin condensation;
- b) swelling of cells;
- c) shrinkage of cells;
- d) formation of the cellular fragments containing chromatin.

5. Name the consequence of apoptosis of cells:

- a) phagocytosis of fragments of the cells limited by a membrane;
- b) formation of a zone from a set of the amount of dead and damaged cells;
- c) development of inflammatory reaction;
- d) autolysis of the dead cells.

6. Specify an attribute, not characteristic of necrosis of cells:

- a) karyolysis;
- b) condensation of chromatin;
- c) swelling of cells;
- d) damage of membranes.

7. Specify a nonenzymatic factor of antioxidate protection of cells:

- a) bivalent ions of iron;
- b) glucuronidase;
- c) vitamin D;
- d) vitamin E.

8. Specifiy manifestation of a cell injury with radiactive factors:

- a) activation of lysosomal enzymes;
- b) radiolysis of water;
- c) impairment of distribution of electrolits;
- d) acidosis.

9. Specify the manifestation of disbalance of ions and water in a cell in ischemic damage:

a) accumulation of K^+ ;

- b) decrease in maintenance of Cl;
- c) accumulation of Na⁺;
- d) decrease in maintenance of H⁺;
- e) accumulation of HCO₃;
- f) accumulation of OH .

10. Specify the substance not protecting a cell from the action of free radicals:

- a) tocopherols;
- b) superoxidedismutase;
- c) peroxidase;
- d) vitamin A;
- e) sulphatase.

11. Specify the substance rendering a detergentive action on cellular membranes:

- a) ketonic bodies;
- b) lactate;
- c) noneterificative fat acids;
- d) aminoacids;
- e) glycogen.

12. Excessive activation of free radical and peroxide reactions does not cause:

- a) conformational changes of lipoproteid complexes of membranes of a cell;
- b) inactivation of sulfhydrative groups of proteins;
- c) activation of phospholipase;
- d) suppression of processes of oxidizing phosphorilation;

e) reduction of activity of Na⁺ Ca²⁺ transmembrane exchange mechanism; f) activation of function of membrane coupled receptors.

13. Specify enzyme of antimutational system of cell:

a) restrictase;

b) histaminase;

c) hyaluronidase;

d) creatine phosphokinase.

14. Name ions with which phospholipase and protease of lysosomes are mainly activated in a damaged cell:

a) H⁺; b) Mg²⁺; c) Na⁺; d) K⁺; e) Ca²⁺.

15. Name ions with which membrane coupled phospholipases are mainly activated in a damaged cell:

a) H⁺;
b) Mg²⁺;
c) Na⁺;
d) K⁺;
e) Ca²⁺.

16. A parameter of oxidizing phosphorilation in a mechanical damage of a cell:

a) decreases;

b) does not change;

c) increases.

17. Specify the organells protecting cell from excessive accumulation of ionized calcium in it:

a) lysosomes;

b) ribosomes;

c) nuclei;

d) mitochondria;

e) complex Goldgi.

18. Specify the cellular organells which, as a rule, first of all and in the greatest measure react to damaging influences:

a) ER (endoplasmic reticulum);

b) ribosomes;

c) lysosomes;

d) complex of Golgi.

19. Specify the cells capable of intensive proliferation on reparation of damaged tissues:

- a) cardiomyocytes;
- b) cells of friable fibrous connecting tissue;
- c) skeletal muscular fibres;
- d) neurons.

20. Specify the enzyme providing an antioxidative protection of cells:

- a) hyaluronidase;
- b) phenylalanine decarboxylase;
- c) glutationperoxidase II;
- d) phospholipase A₂;
- e) adeninnucleotide transferase.

21. Specify the reason of hyperhydration of a cell:

- a) increase in activity of Na⁺, K⁺-ATPase;
- b) increase in the endocellular maintenance of lipids;
- c) suppression of oxidizing phosphorilation;
- d) activation of glycolysis;
- e) increase in a current of K^+ inside of a cell.

22. Specify the factor promoting ischemic damage of a cell:

a) decrease in functional activity of a cell;

- b) high dependence of power maintenance of a cell on phosphorilation;
- c) high dependence of power maintenance of a cell on glycolyis;
- d) stimulation of the facilitated diffusion of glucose with insulin;

e) decrease in the temperature of a cell.

23. Specify the mechanism of damage of cellular membranes:

- a) output of lysosomal hydrolase in cytosol;
- b) activation of membrane transferase;
- c) activation of transport of glucose in a cell;
- d) adsorption of proteins on cytolemm;
- e) endocellular acidosis.

24. Specify the action which amphiphylic compounds in high concentration render on membranes of cells:

a) activate glycolysis;

- b) aggregate in micelles and invade into membranes of cells;
- c) build into hydrophilic layer of membranes in the form of monomers;
- d) increase orderliness of the structure of a membrane.

25. Increase of maintenance of Ca²⁺ in cytosol of a cell in ischemia is caused by:

a) increase in activity of Na⁺, K⁺-ATPase;

- b) activation of glyolysis;
- c) decrease in activity of Ca^{2+} , Mg^{2+} ATPases of ER;

d) increase in intensity of endocellular transport of glucose.

26. Excess accumulation of calcium in cells does not:

- a) separate oxidation and phosphorilation;
- b) activate phospholipases;
- c) reduce permeability of membranes of cells;
- d) raise the formation of macroergs
- e) promote the formation of actyn-myosin ;complex;
- f) promote the hyperhydration of cells.

ABNORMALITIES OF MICROCIRCULATION

Specify a correct variant of the answer

1. Specify the vessels which are not referred to microcirculatoric:

- a) precapillary;
- b) arteriovenular anastomoses;
- c) lymphatic venules;
- d) arteriovenules shunts;
- e) arteriols;
- f) capillaries.

2. Specify the changes referred to extravascular impairments of microcirculation:

a) aggregation of uniform elements, phenomenon of «sludge»;

b) damage of endothelium, damage of basic membranes;

c) increase in volume of interstitium liquids, delay of its outflow, change of the function of corpulent cells, stromal dystrophies.

3. Specify the changes referred to intravascular impairments of microcirculation:

a) aggregation of uniform elements, phenomenon of «sludge»;

b) damage of endothelium, damage of basic membranes, increase of permeability of microvessels walls;

c) increase in volume of interstitium liquids, delay of its outflow, change of the function of mast cells, stromal dystrophies.

4. Specify the changes referred to vascular abnormalities of microcirculation:

a) aggregation of uniform elements, phenomenon of «sludge»;

b) damage of endothelium, damage of basic membranes;

c) increase in volume of interstitium liquids, delay of its outflow, change of function of mast cells, stromal dystrophies.

5 .The juxtacapillary blood flow performs:

a) deposition of blood in a zone of microcirculation;

b) regulation of permeability of microvessels;

c) acceleration of venous blood flow;

d) regulation of capillary blood flow and transcapillary exchange;

e) mobilization of deposited blood;

f) participation in thermoregulation.

6. Specify the factor not promoting stasis of blood:

a) increase in the filtration of albumins from microvessels into surrounding tissues;

b) direct influence of high or low temperature on a tissue;

c) dilation of arteriols;

d) damage of tissues by acids or alkalis;

e) constriction of arteriols.

7. Erythrocyte aggregation does not assist:

a) increase in the maintenance of blood globulins;

b) increase in electrostatic charge of erythrocytes;

c) microaneurysms of fine vessels;

d) falling of the systemic blood pressure;

e) extracellular dehydration.

8. «Sludge» syndrome is a:

a) a formation of a thromboleukocytic agregate on a microvessel wall;

b) aggregation of uniform elements of blood in a microvessel lumen;

c) condensation of blood owing to loss of its liquid part;

d) coagulation of blood proteins.

9. Specify, whether the separation of cellular elements and blood plasma is characteristic of «sludge syndrome»:

a) yes;

b) no.

10. «Sludge syndrome» does not develop in case of:

a) sepsis;

b) general inflammation;

c) introduction into a vascular channel of great volume of protein containg blood substitutes into a vascular channel;

d) hyperhydrations of the organism;

e) hemoconcentration.

11. Specify the way of passage of blood cells through walls of capillaries and venules:

- a) chemotaxis;
- b) diapedesis;
- c) filtration;
- d) phagocytosis.

12. Specify the factor not raising permeability of vascular membranes:

- a) increase of speed of blood flow in capillaries;
- b) acidosis;
- c) products of degranulation of mast cells;
- d) rounding off endothelial cells.

13. Specify pathogenic factor not promoting a thrombus formation:

- a) damage of vascular wall;
- b) delay of blood-flow;
- c) increase in activity of the factors of the coagulative system of blood;
- d) increase in viscosity of blood;
- e) activation of factors of the anticoagulative system of blood;
- f) hyperadrenalinemia.

14. Thrombolysis occurs under the influence of:

- a) streptokinase;
- b) $PgF_{2\alpha}$;
- c) histaminase;
- d) glutathione peroxidase.

15. Name the mechanism of increase of permeability of vessels walls under influence of histamine:

- a) rounding off endotheliocytes;
- b) spasm of arteriols;
- c) spasm of venules.

LOCAL DISORDERS OF BLOOD CIRCULATION

Specify a correct variant of the answer

1. Specify the conditions which are not referred to typical abnormalities of the peripheral blood circulation:

a) arterial hyperemia;

b) venous hyperemia;

c) ischemia;

d) coarctation of aorta.

2. Name the basic type of venous hyperemia on its reason:

a) cardiogenic (in heart failure);

b) cardiogenic (on increase in cardiac output);

c) neuroparalitic;

d) myoparalitic.

3. Name basic types of arterial hyperemia on its origin:

a) neurotonic;

b) obstructive;

c) compressive.

4. Arterial hyperemia arises on neurotonic mechanism due to:

a) strengthenings of tonic influences of smooth muscular cells of arteriol walls on β -adrenore ceptors;

b) strengthenings of tonic influences of smoothmuscular cells of arteriol walls on α -adrenoreceptors;

c) spontaneous myogenic tone of arteriols;

d) decrease in parasympathetic influences on arteries.

5. Arterial hyperemia is characterized by:

a) narrowing of lumen of arterial vessels;

b) impairment of outflow of blood by veins and lymphatic vessels;

c) decrease in volumetric speed of blood flow;

d) increase of lymphformation;

e) decrease of lymphformation.

6. Specify a set of changes of the regional blood circulations and microcirculation, corresponding to arterial hyperemia:

a) expansion of arteriols, acceleration of blood flow, increase in the amount of functioning capillaries, increase of blood pressure in capillaries;

b) narrowing of arteriols, acceleration of blood flow, increase in the amount of functioning capillaries, reduction of blood pressure in capillaries; c) expansion of arteriols, slowing down of blood flow, reduction of the amount of functioning capillaries, increase in blood pressure in capillaries.

7. Specify a set of changes of the regional blood circulations and microcirculation, corresponding to venous hyperemia:

a) acceleration of blood flow, increase in the diameter of capillaries, postcapillaries, venules, reduction of blood pressure in capillaries, postcapillaries, venules;

b) delay of blood flow, reduction of the diameter of capillaries, postcapillaries, venules, increase in blood pressure in capillaries, postcapillaries, venules;

c) delay of blood flow, increase in the diameter of capillaries, postcapillaries, venules, increase in blood pressure in capillaries, postcapillaries, venules.

8. Specify a set of changes of the regional blood circulations and microcirculation, corresponding to ischemia:

a) reduction of the diameter of arteriols and precapillaries, decrease in the amount of functioning capillaries, delay of blood current;

b) increase in the diameter of arteriols and precapillaries, decrease in the amount of functioning capillaries, acceleration of blood flow;

c) reduction of the diameter of arteriols and precapillaries, increase in the amount of functioning capillaries, acceleration of blood current.

9. Specify the factor causing scarlet painting of the organ in arterial hyperemia:

a) increase in maintenance of oxihemoglobin in blood;

- b) reduction of the maintenance of oxihemoglobin in blood;
- c) increase in arterio-venous difference on oxygen;
- d) reduction in volumetric speed of blood flow;

e) increase in the maintenance of the restored hemoglobin in blood.

10. The following signs are not characteristic of venous hyperemia:

a) increase of blood filling of the organ or the tissue;

b) reduction of the amount of blood flow through organ or tissue;

c) difficulty of outflow of blood along veins;

d) increase in resorption of liquids in venules;

e) cyanosis of tissues.

11. Specify the incorrect statement:

a) ischemia can be result of increase in arterial blood flow;

b) acute ischemia can lead to ischemic necroses.

12. Specify the change which is not arising in zone of ischemia:

a) necrosis;

b) acidosis;

c) lowering of function;

d) accumulation of Ca^{2+} in cytosol;

e) increase in concentration of K⁺ in cell;

f) increase in concentration of Na⁺ in cell.

13. Specify a possible consequence of thrombosis of deep veins of the lower limbs:

a) embolism of brain arteries;

b) thromboembolism of pulmonary arteries;

c) portal hypertensia;

d) embolism of kidney vessels;

e) embolism of intestinal vessels.

14. Specify possible consequences of arterial hyperemia:

a) micro- and macrohaemorrhages in surrounding tissues;

b) thrombosis of vessels of hyperemic site;

c) stasis in capillaries of hyperemic site;

d) decrease of oxigenation of tissues;

e) inhibition of exchange processes in hyperemic site.

15. Specify the consequence which is not characteristic of venous hyperemia:

a) growth of connective tissue;

b) strengthening of organ function;

c) dystrophy of tissues;

d) haemorrhage.

16. The term «retrograde embolism» is applied in case of:

a) transition of embolus from one circle of blood circulation to another, passing a capillary channel;

b) movement of embolus against flow of blood;

c) movement of embolus through arterio-venous shunts;

d) movement of embolus through an open atrial septum;

e) movement of embolus through an open Botallo's duct.

17. Specify embolus of endogenic origins:

a) congestion of parasites;

b) tumoral cells;

c) lots of microorganisms;

d) vesicles of air.

18. Specify the department of the cardiovascular system from which embolus can be brought in the systemic blood circulation:

a) venous system of the systemic blood circulation;

b) arterial system of the pulmonary blood circulation;

- c) venous system of the pulmonary blood circulation;
- d) the right heart.

19. Specify the incorrect statement:

a) emboluses can be particles of blood clot, foreighn bodies, cells of tissues, fat, vesicles of air, parasites;

b) arterial emboluses usually remain in the pulmonary vascular network, venous emboluses can obturate vessels of any ogran;

c) embolism of the main pulmonary artery and its branches, coronary and cerebral arteries is life threating;

d) arterial thromboembolism can arise in case of destruction of blood clots in the left ventricle of heart in myocardial infarction and some arrhythmias.

20. Specify the factor which is not promoting activation of collateral blood circulation in the zone of ischemia and around it:

a) increase in concentration of adenosine in ischemic tissues;

b) increase in gradient of pressure of blood above and below occlusion of arteries;

c) acidosis in the zone of ischemia;

d) K⁺ hypertension in the zone of ischemia;

e) tachycardia.

21. Specify the possible reasons of gas embolism:

a) fast increase in barometric pressure;

b) injury of large veins of neck;

c) a fast shift of barometric pressure from normal to low;

d) inhalation of air with a high concentration of inert gases.

22. A possible source of thrombembolism of the pulmonary artery is:

a) cusps of aortal valve;

b) venous blood clot of lower limbs;

c) the aorta.

ACUTE AND CHRONIC INFLAMMATION. ACUTE PHASE RESPONSE

Specify a correct variant of answer

1. The acute inflammatory response is characterized by:

a) formation of inflammatory granulomas;

b) accumulation of huge multinuclear cells in the inflammatory center;

c) neutrophils accumulation in the inflammatory center.

2. Note the process strengthening the inflammation:

- a) angiospasm;
- b) increase in permeability of a vascular wall;
- c) decrease in permeability of a vascular wall;
- d) lowering of emigration of leukocytes.

3. Specify the usual sequence of emigration of leukocytes in an acute inflammation site:

- a) monocytes, lymphocytes, neutrophils;
- b) monocytes, neutrophils, lymphocytes;
- c) neutrophils, monocytes, lymphocytes.

4. Specify the sequence of changes of blood flow, as a rule, observable in an inflammation site:

- 1. Arterial hyperemia;
- 2. A short-term spasm;
- 3. Venous hyperemia;
- 4. Stasis;
- 5. Pendulous movement in microvessels:
 - a) 1, 2, 3, 4, 5;
 - b) 2, 3, 1, 4, 5;
 - c) 2, 1, 3, 5, 4.

5. Specify the factor which does not influence the occurrence of pain in case of inflammation:

a) Pg of E group;

- b) histamine;
- c) H⁺-hyperionation;
- d) K⁺-hyperionation;
- e) kinins;
- f) rise in temperature of tissue;
- g) mechanical irritation of the nerve endings.

6. Specify the substances which are not referred to mediators of inflammation:

- a) kinins;
- b) Pg;
- c) biogenic amins;
- d) lymphokins;
- e) nucleinic acids;
- f) leukotriens.

7. Specify mediators of inflammations which have a cellular origin:

- a) serotonin, lymphokine, histamine, lysosomal enzymes;
- b) Hagemen's factor, complement system, kallikrein-kinin system.

8. Name newly synthesized mediators of mast cells:

a) neutrophil chemotaxic factor, histamine, basophilic kallikrein;

b) Pg, thromoxane, leukotriens.

9. Activation of the kallikrein-kinin system begins with the activation of:

a) high-molecular kininogene;

b) Hagemen's factor;

c) prekallikrein;

d) bradikinin.

10. Name molecules of adhesion providing an initial attachment of leukocytes to endothelium of microcirculatoric vessels in inflammation:

a) selectins;

b) integrins;

c) molecules of immunoglobuline family.

11. Name the statement describing the factor of thrombocytes activation:

- a) it is formed on splitting of plasma protein;
- b) it is stored in a preformative form of granules of mast cells;

c) it is a derivation of arachidonic acid;

d) it causes positive neutrophil chemotaxis.

12. Adhesion of leukocytes to the endothelium of microcirculatoric vessels is found out first of all in:

a) arteriols;

b) metarteriols;

c) capillaries;

d) postcapillary venule.

13. Name the obligatory condition for leukocytes sticking to endothelium of microcirculatoric vessels in inflammation:

a) slowing down of a blood flow;

b) stasis;

- c) formation of blood clots in vessels;
- d) occurrence on a membrane of molecules of adhesion for leukocytes.

14. Specify the factor causing an exit of plasma proteins from microcirculatoric vessels in the site of inflammation:

a) reduction of endothelial cells;

- b) increase in hydrostatic pressure of blood in capillaries;
- c) slowing down of blood flow;
- d) decrease of oncotic pressure of interstitial liquids.

15. Name the cells which are carrying out phagocytosis in the site of inflammation:

- a) reticulocytes;
- b) mast cells;
- c) neutrophils;
- d) B-lymphocytes;
- e) thrombocytes.

16. Specify the physical and chemical change which is not observable in the center of acute aseptic inflammation:

a) acidosis;

- b) alkalosis;
- c) hyperosmium;
- d) hyperoncium;
- e) ionic disbalance.

17. Specify the factor rendering stimulating influence on process of cell proliferation in the site of inflammation:

- a) antibodies;
- b) cAMP;
- c) cGMP;
- d) glucocorticoids.

18. Specify the substance which does not possess the properties of chemoattractance for neutrophils:

- a) lipopolysaccharide of bacteria;
- b) leukotrien B₄;
- c) IL-8;

d) IL-2;

- e) component C₅b of complement system;
- f) the factor of activation of thrombocytes.

19. Acute inflammation is characterized by:

a) the formation of inflammatory granulems;

- b) increase in permeability of microcirculatoric vessels walls;
- c) accumulation of huge multinuclear cells in the center of inflammation;
- d) infiltration with mononuclear leukocytes in the site of inflammation.

20. We refer to «cells of acute inflammation»:

- a) macrophages;
- b) lymphocytes;
- c) neutrophils;
- d) epithelial cells.

21. Specify the mechanism determining abnormalities of protective functions of the organism in Chediac Higasi syndrome:

a) the impairment of cellular immunity;

b) the impairment of humoral immunity;

c) the combined impairment of humoral and cellular immunity;

d) impairment of phagocytosis;

e) combined impairment of humoral immunity, cellular immunity, phagocytosis.

IMPAIRMENTS OF THERMOREGULATION

Specify a correct variant of the answer

1. Choose the correct statement:

a) rise in temperature of body of a person is always an evidence of the development of feverish reaction;

b) in fever there are no signs of intoxication of the organism;

c) fever is a reaction of warm-blooded animals to an action of pyrogenic factors.

2. Specify the minimal value of body temperature of a person, at which high-grade restoration of ability to live is possible:

a) 20 °C;

- b) 21 °C;
- c) 23 °C;
- d) 24 °C;
- e) 25 °C;
- f) 27 °C;
- g) 28 °C;
- h) 30 °C.

3. It assit to the inhibition of heat emission in the first stage of fever:

a) amplification of heat emission;

b) spasm of skin vessels.

4. Specify the chemical nature of endogenous pyrogenes, produced by microorganisms:

a) lipopolysaccharids;

b) phospholipids;

c) mucopolysacchaids.

5. Specify the group of primary pyrogenes, possessing to the most expressed pyrogenic activity:

a) mucopolysaccharids;

- b) strange proteins;
- c) lipopolysaccharids;
- d) phospholipids;
- e) lipoprotein.

6. It does not possess of properties of endogenous pyrogenes:

- a) IL-1;
- b) IL-2;
- c) IL-6;.
- d) TNF

7. Choose the incorrect statement:

a) not only pathogenic microoganisms, but also nonpathogenic ones possess a pyrogenic activity;

b) pyrogenic properties of pathogenic microorganisms do not always correlate with its virulence;

c) only endotoxins possess a pyrogeneic activity;

d) components of membranes of bacterial cells can possess a pyrogenic activity.

8. Specify the change caused by secondary pyrogenes in neuron of the hypothalamic thermotaxic centers:

a) increase in formation of IL-1;

- b) accumulation of lipopolysaccharide;
- c) strengthening of formation of group of PgE;
- d) easing of formation of group of PgE;
- e) easing of formation of cAMP;
- f) increase of excitability of «thermal» neurons.

9. Specify characteristic manifestations of the first stage of fever:

a) fever, skin pallor, skin dryness, increase of diuresis;

b) sensation of heat, skin hyperemia, reduction of diuresis.

10. Specify characteristic manifestations of the second stage of fever:

- a) sensation of heat, skin hyperemia, reduction of diuresis;
- b) fever, skin pallor, dryness of skin, increase of diureis.

11. Fast rise in temperature of the body in pyretic fever, as a rule, is accompanied by:

- a) reddening of the skin and fever;
- b) pallor of the skin and fever;
- c) reddening of the skin and sensation of heat;
- d) strengthening of the secretory function of kidneys;
- e) strengthening of sweating.

12. Specify the mechanism which does not participate in the rise of body temperature in fever:

a) increase in interlinking of oxidation and phosphorilation;

b) peripheral vasoconstriction;

c) strengthening of contractile («muscular») thermogenesis;

d) reduction of sweating;

e) activation of biological oxidation.

13. Choose the correct statement:

a) the organism loses the ability to support constant body temperature at changes of external temperature in fever;

b) there are essentially the same changes in the system of thermotax in exogenous hyperthemia, as in fever;

c) the thermotax of the organism remains in fever.

14. Specify changes of absolute value of heat prodution and heat emission at the first stage of development of feverish reaction:

a) heat production increases, heat emission decreases;

b) heat production and heat emission change equivalently;

c) heat production decreases, heat emission does not change.

15. The character of a temperature curve in fever does not depend on:

a) the etiologic factor;

b) features of pathgenesis of the underlying disease;

c) the functional condition of the endocrine systems;

d) ambient temperatures;

e) medical actions;

f) the functional condition of the immune system.

16. Negative influence of fever can be caused by:

a) hyperfunction of heart in long high fever;

b) fast decrease in body temperature from pyretic up to normal or subnormal levels;

c) hectic dynamics of body temperature;

d) the metabolic abnormalities caused by heat;

e) the all above listed.

17. Compensatory reaction in man on substantial increase of ambient temperature is:

a) narrowing of peripheral vessels;

b) expansion of peripheral vessels;

c) expansion of vessels of internal organs;

d) increase of muscular tone;

e) muscular shiver.

18. Specify the change in the organism on the stage of compensation (adaptation) of hypothermia:

a) expansion of peripheral vessels;

b) narrowing of vessels of internal organs;

- c) the maximal voltage of mechanisms of thermotax;
- d) strengthening of sweating;

e) bradycardia.

19. Specify change in organism on stage of decompensation (deadaptation) of hypothermia:

a) increase in a level of basic exchange;

- b) narrowing in peripheral vessels;
- c) arterial hypertensive reaction;
- d) the maximal voltage of mechanisms of thermotax;
- e) suppression of activity of brain cortex;
- f) strengthening of muscular shiver;

g) tachycardia;

h) hyperglycemia.

20. Compensatory reaction in man in general overcooling is:

a) expansion of peripheral vessels;

b) narrowing of vessels of internal organs;

- c) muscular relaxation;
- d) decrease in gas exchange;
- e) narrowing of peripheral vessels;

f) hypoglycemia.

21. Choose the correct statement:

a) febrifugal therapy is necessary for treatment of subfebrile fever;

b) febrifugal therapy is necessary for treatment of long pyretic fever.

TYPICAL FORMS OF IMPAIRMENTS OF TISSUE GROWTH. TUMOURS

Specify a correct variant of the answer

1. Specify the sign, not characteristic of benign tumours:

- a) slow formation of tumoral node;
- b) expansive growth;

c) relapse;

- d) rather high degree of cellular and functional differentiation;
- e) low expressiveness of tumoral progression.

2. Oncoproteins are:

- a) the proteins blocking of cellular respiration;
- b) the proteins suppressing glycolisis;
- c) the proteins causing tumoral transformation of a normal cell.

3. Specify metabolic feature, characteristic of tumoral tissue:

- a) tissue breath amplifies;
- b) the concentration of lactic acid raises;
- c) there is a shift of pH to the alkaline side.

4. Specify the factor promoting growth of tumoral cells:

- a) ill-defined antigenic properties of tumoral cells;
- b) production of the tumour necrosis factor of the organism;
- c) strengthening of processes of final differentiation of tumoral cells;
- d) activation of natural killers (NK-cells).

5. Carcinogenesis is a result of:

- a) excessive expression of normal genes supervising cell division;
- b) structural modifications of DNA under the action of carcinogens;
- c) inactivation of regulatoric cytoplasmic proteins by carcinogens;
- d) the all above listed.

6. The first stage of chemical carcinogenesis is called:

- a) promotion;
- b) cocarcinogenesis;
- c) progression;
- d) initiation;
- e) procarcinogenesis.

7. Specify the structure of the cell, which is a target for chemical carcinogens:

- a) a cytoplasmic membrane;
- b) sarcoplasmic reticulum;
- c) molecules of endocellular matrix;
- d) nuclear DNA;
- e) lysosomes;
- f) mitochondria.

8. The pathological hypertrophy of tissue is an:

a) increase in weight and volume of structural elements after the end of formation of organs and tissues;

b) increase in weight and volume of structural elements of organs and tissues after extreme physical load; c) increase in weight and volume of structural elements of tissues and organs, inadequate to its function.

9. The second stage of chemical carcinogenesis is named as:

- a) promotion;
- b) cocarcinogenesis;
- c) progression;
- d) initiation;
- e) procarcinogenesis.

10. Specify features of protein exchange in cells of malignant tumours:

a) suppression of synthesis of protein;

- b) prevalence of protein catabolism;
- c) opportunity of formation of embryonal protein.

11. Term «tumoral progression» indicates:

a) increase in weight of tumour;

- b) constant allocation of more malignant clones of cells;
- c) eluding of tumours from immune supervision;
- d) the beginning of synthesis of oncoproteins;
- e) metastasis of tumoral cells.

12. Specify the sequence of basic stages of hematogenic and lymphogenic ways of metastasis of tumoral cells:

1. invasion of tumour cells from a vessel into the normal tissue;

- 2. destruction of normal tissue surrounding the tumour;
- 3. invasion of tumour cells in a gleam of a vessel;

4. carrying of tumour cells in a current of blood, lymph and the formation of thromboembolus:

a) 4, 3, 1, 2; b) 3, 4, 1; c) 2, 3, 4, 1.

13. Specify the possible reason of tumours relaps:

a) suppression of factors of local immunity;

b) low activity of anticellular mechanisms of antineoplastic protection of the organism;

c) penetration of a fragment of DNA of the tumoral cell containing active oncogene, in the genome of a normal cell;

d) penetration of a fragment of «tumoral» RNA into a normal cell.

14. Among methods of therapy of malignant tumours there is:

a) elimination of carcinogens from the environment;

b) prevention of contact of carcinogens with the organism;

c) increase of the activity of mechanisms of antineoplastic protection;

d) revealing and treatment of benign tumours.

15. Specify the factor, inhibiting cell division:

a) factors of growth;

b) decrease in superficial tension of cells;

c) cGMP;

d) keylones.

16. Specify the factor protecting tumoral cells from the action of immune mechanisms of the organism:

a) allogenic inhibition;

b) internalization of antigenic structures of a tumoral cell;

c) T-killers;

d) T-helpes;

e) phagocytes.

17. Choose the correct statement:

a) carcinogen is an agent causing a tumour;

b) carcinogen is a substance, secreted by tumoral cells and promoting their duplication.

18. Choose the correct statement:

a) cellular oncogene is an oncogenic gene incorporated into a cellular genome;

b) cellular oncogene is a gene supervising the division of a cell and incorporated into a normal cell from a tumoral one;

c) cellular oncogene is a gene of a cell supervising its division, turned into an oncogenic gene under the influence of carcinogen.

19. Specify the cells, which are the basic producers of the tumour necrosis factor:

a) neutrophils;

b) eosinophils;

c) monocytes;

d) thrombocytes;

e) erythrocytes.

20. Specify the typical form of pathology of tissue growth:

a) necrosis of tissue;

b) pathological hypertrophy;

c) hyperplasia of mitochondrias;

d) sarcomas;

e) carcinoma.

21. Displasia can be a result of:

a) abnormalities of the genetic program of cells;

- b) acute hyperglycemia;
- c) extracellular acidosis;
- d) respiratory alkalosis.

PATHOLOGICAL PHYSIOLOGY OF THE ERYTHROCYTES SYSTEM

Specify a correct variant of the answer

1. In deficiency of the internal factor there is:

a) iron-defcient anemia;

- b) enzyme-deficient anemia;
- c) B₁₂-deficienct anemia;
- d) protein-deficienct anemia.

2. Specify the leading mechanism of abnormalities of functions of the organism in anemias:

- a) polycytemic hypovolemia;
- b) hemic hypoxia;
- c) circulatoric hypoxia;
- d) oligocytemic hypervolemia.

3. Specify the correct sequence of stages of maturing of RB cells (red blood cells):

- 1. polychromatophilous normoblast;
- 2. basophilic normoblast;
- 3. oxiphilic normoblast;
- 4. erythrocyte;
- 5. reticulocyte;
- 6. pronormoblast;
- 7. erythroblast:
 - a) 7, 6, 2, 1, 3, 5, 4;
 - b) 2, 1, 3, 6, 5, 7, 4;
 - c) 7, 6, 5, 2, 1, 3, 5.

4. Reticulocytes of a healthy adult are:

a)reticulocytes, in norm contained in the bone marrow (0,1-1,6%), in circulating blood are absent;

b) predecessors of mature erythrocytes, in norm contained in peripheral blood (0,2-1,2%) and in the bone marrow.

5. Characterize the condition of erythroid growth in the bone marrow in anemia proceeding with the maintenance of reticulocytes in peripheral blood, equal to 3 %:

- a) regeneratoric;
- b) hyporegeneratoric;
- c) hypoplastic.

6. Specify the condition accompanied, as a rule, by the development of absolute erythrocytosis:

- a) megaloblastic anemia;
- b) chronic hypoxia;
- c) lymphoma;
- d) hemodilution;
- e) hemoconcentration.

7. Specify the condition, accompanied by the development of relative erythrocytosis:

- a) megaloblastic anemia;
- b) hemodilution;
- c) Vaquez's disease;
- d) ischemia of kidneys;
- e) stress-reaction.

8. Specify a hematological parameter, characteristic of acute hemolitic anemia:

- a) expressed hypochromia of erythrocytes;
- b) increase in iron associated ability of serum;
- c) expressed reticulocytosis.

9. Specify the parameter of exchange of iron, characteristic of aplastic anemia:

- a) reduction of latent iron associated ability of serum;
- b) reduction of factor of transferin saturation;
- c) increase in latent iron associated ability of serum;
- d) increase in the general iron associated ability of serum.

10. Specify anemia referred to hemoglobinopathias:

- a) hereditary microspherocytosis;
- b) sickle-cell anemia;
- c) paroxysmal nocturnal hemoglobinuria;
- d) Biermer's anemia.

11. Specify anemia characterized by the displacement of Price-Johns curve to the right:

a) Biermer's anemia;

b) iron-deficient anemia;

c) hereditary microspherocytosis;

d) acute posthemorrhagic anemia.

12. Specify anemia, characterized by the displacement of Price-Johns curve to the left:

a) Biermer's anemia;

b) hereditary microspherocytosis;

c) anemia in dyphylobothriasis.

13. Specify the change of blood volume, observable right after acute blood loss:

a) oligocythemic hypovolemia;

b) simple hypovolemia;

c) oligocythemic normovolemia;

d) simple normovolemia.

14. Specify the time of restoration of volume of circulating blood (at loss up to 1 liter) due to activation of erythropoesis:

a) within 1–2 days;

b) within 2–3 days;

- c) within 1 hour;
- d) after 4-5 days;
- e) after 40 days.

15. Specify the change of blood volume, observable in 5–6 hours after acute blood loss of medium severity:

a) oligocythemic hypovolemia;

b) simple hypovolemia;

c) oligocythemic normovolemia;

d) simple normovolemia.

16. Specify anemia with the greatest increase of concentration of erythropoetins in blood:

a) acute hemolitic anemia of medium severity;

b) acute posthemorrhagic anemia of medium severity;

c) chronic posthemorrhagic anemia.

17. Specify anemia which is not characterized by hyperbilirubinemia:

a) hereditary microspherocytosis;

b) Biermer's anemia;

- c) folic acid deficienct anemia;
- d) chronic posthemorrhagic anemia.

18. Specify the condition at which simple hypovolemia is observed:

a) in 30–40 min after acute blood loss;

b) in 6-8 hours after acute blood loss of medium severity;

c) in case of a burn shock;

d) in case of overheating of the organism.

19. Specify the time of restoration of volume of circulating blood (in loss of blood up to 11) due to tissue liquid receipt into vessels:

- a) within 1–2 days;
- b) within 2–3 days;
- c) within 1 hour;
- d) after 4-5 days;
- e) after 40 days.

20. Specify the parameter of hematocrite which is possible to consider an increased one:

- a) 0,55 l/l;
- b) 0,45 l/l;
- c) 0,35 l/l.

21. The raised viscosity of blood is not observed in case of:

- a) relative erythrocytosis;
- b) absolute erythrocytosis;
- c) erythremia (Vaquez's desease);
- d) Biermer's anemia.

22.Specify oedemas accompained by oligocythemic hypervolemia:

- a) cardial;
- b) nephritic;
- c) hepatic;
- d) nephritic;
- e) allergic;
- f) cachexic.

23.Hemolytic anemia is characterized by:

- a) oligocythemic hypovolemia;
- b) oligocythemic hypervolemia;
- c) polycythemic hypovolemia;

d) oligocythemic normovolemia;

e) polycythemic normovolemia.

24. Specify the earliest terms of restoration of volume of circulating blood after acute blood loss of medium severity:

a) in 7–8 hours;

b) in 24-48 hours;

c) in 4-5 days.

25. Specify the basic part of pathogenesis of the first stage of acute posthemorhagic anemia:

a) damage of a vessel;

b) reduction of volume of circulating blood;

c) hypoxia of hemic type;

d) deficiency of iron;

e) decrease in the maintenance of erythrocytes in blood.

26. Specify a condition in which reduction of hematocrite is observed:

a) in case of decrease in the maintenance of 2,3-biphosphoglicerate in erythrocytes;

b) within the first hour after massive blood loss;

c) in 4–5 days after acute blood loss of medium severity;

d) in unrestrained vomiting.

27. Specify the change of volume of blood observable in 4–5 days after acute blood loss of medium severity:

a) oligocythemic hypovolemia;

b) simple hypovolemia;

c) oligocythemic normovolemia;

d) simple normovolemia.

28. Specify the parameter of hematocrite, corresponding to the condition after acute blood loss of medium severity in 20–30 min after blood loss:

a) 0,26–0,32 l/l; b) 0,36–0,48 l/l;

c) 0,52–0,58 l/l.

29. It arises in first minutes after acute blood loss of medium severity:

a) oligocythemic normovolemia;

b) normocythemic hypovolemia;

c) oligocythemic hypovolemia;

d) polycythemic hypovolemia.

30. Specify the process having an adaptive value for the organism in the nearest minutes and hours after acute blood loss:

a) reduction of venous return of blood;

b) peripheral vasoconstriction;

c) tissue hyperperfusion;

d) polyuria;

e) hypoventilation.

31.The abnormality observed by the end of the first-second day after acute blood loss of moderate degree is:

a) polycythemic hypovolemia;

- b) normocythemic hypovolemia;
- c) oligocythemic normovolemia;
- d) oligocythemic hypovolemia;
- e) oligocythemic hypervolemia.

32. Specify the attribute, not characteristic of β-thalassemia:

- a) hereditary character of occurrence;
- b) development of anemia;
- c) decrease in synthesis of β -globin;
- d) increase in maintenance of HbF in blood;
- e) decrease in maintenance of HbA₁ in blood;
- f) hereditary erythrocytosis.

PATHOLOGICAL PHYSIOLOGY OF THE SYSTEM OF LEUKOCYTES

Specify a correct variant of the answer

1. Specify an attribute of neutrophils:

a) they get through endothelial layer;

- b) they are predecessors of antibodyproducents;
- c) they are producers of albumins of plasma of blood;
- d) they generate superoxide anion-radical.

2. Specify an attribute of B-lymphocytes:

- a) they are producers of albumins of plasma of blood;
- b) they are predecessors of cells-antibodyproducents;
- c) microphages;
- d) macrophages;
- e) they are the cells providing the realization of cellular part of immunity.

3. Specify an attribute of T-lymphocytes:

- a) they are predecessors of cells-antibodyproducents;
- b) they are producers of albumins of plasma of blood;
- c) microphages;
- d) macrophages;
- e) they are the cells providing the realization of a cellular part of immunity.

4. The variant of pathological leukocytosis is:

- a) miogenic;
- b) inflammatory;
- c) digestive;
- d) newborns.

5. The reason of absolute lymphocytosis is:

- a) blood loss;
- b) plasma loss;
- c) reduction of formation of neutrophils;
- d) increase in formation of lymphocytes;
- e) dehydration of organism.

6. Specify the mechanism of occurrence of true leukocytosis:

a) activation of leukopoesis;

b) mobilization of bone marrow reserve of leukocytes without activation of leukopoesis;

- c) the slowed down destruction of leukocytes;
- d) the raised output of leukocytes from a vascular channel in tissues.

7. Specify the disease which is not accompanied by eosinophilia:

- a) pollinosis;
- b) echinococcosis of liver;
- c) chronic lympholeukosis;
- d) allergic rhinitis;
- e) chronic myeloleukosis.

8. Specify diseases, accompanied by absolute lymphocytosis:

- a) virus infections;
- b) tuberculosis;
- c) infectious mononucleosis;
- d) the all above listed.

9. Specify changes of hematologic parameters, characteristic of leukemoid reaction of myeloid type:

a) occurrence of individual myeloblasts in blood;

b) increase in maintenance of leukocytes up to tens billions in 1 litre of blood;

- c) occurence of myelocytes and metamyelocytes in blood;
- d) increase in quality of nonsegmental neutrophils;

e) the all above listed.

10. Specify changes in hemogram, characteristic of neutrophilic leukocytosis with regeneratoric nuclear shift to the left:

a) increase in percentage of nonsegmental neutrophils on the background of neutrophilia;

b) occurrence of neutrophilic metamyelocytes in blood;

c) reduction of the relative maintenance of lymphocytes in blood;

d) the all above listed.

11. Specify the change in peripheral blood reflecting neutropholic nuclear shift to the right:

- a) increase in maintenance of nonsegmental neutrophils;
- b) polysegmentation of nuclei of neutrophils;
- c) occurrence of myelocytes;
- d) leukocytosis.

12. Anti-infectious stability of the organism in agranulocytosis:

- a) is raised;
- b) is lowered;
- c) is not changed.

13. Specify the change of peripheral blood which is not observable in case of agranulocytosis:

- a) significant reduction of neutrophils in blood;
- b) eosinipenia;
- c) absolute lymphocytosis;
- d) relative lymphocytosis.

PATHOLOGICAL PHYSIOLOGY OF LEUKEMIAS

Specify a correct variant of the answer

1. Specify the etiological factor of leukemia:

- a) heavy current infections;
- b) psychological abnormalities;
- c) endocrine frustration;
- d) posthemorrhagic anemias;
- e) chemical cancerogenic substances.

2. Specify, whether it is always possible to find out blasts cells in peripheral blood in leukemias:

a) yes;

b) no.

3. It takes place in bone marrow in case of acute leukemia:

a) hyperplasia of white blood elements;

- b) decrease in quantity of red blood cells family;
- c) reduction of number of megacariocytes;
- d) the all above listed.

4. Specify the change of hemogram, characteristic of chronic lympholeukosis:

a) occurrence of myelocytes in blood;

- b) increase in percentage of neutrophils;
- c) occurrence of metamyelocytes in blood;
- d) occurrence of lymphocytes shadows in blood smear;
- e) relative lymphocytosis.

5. Specify the most typical variant for typical current of chronic lymoholeukosis:

a) leukopenia with relative lymphocytosis;

- b) normal quantity of leukocytes with absolute lymphocytois;
- c) substantial growth of quantity of leukocytes with lymphocytosis up to 40 %;
- d) substantial growth of quantity of leukocytes with lymphocytosis up to 80 %.

6. Specify changes in peripheral blood, characteristic of acute lymphoblasts leukemia:

a) occurrence of blast cells with negative reaction on lipids;

- b) occurrence of blast cells with positive Shick reaction;
- c) anemia and thrombocytopenia;

d) the all above listed.

7. Acute myeloleukosis in peripheral blood is characterized by the presence of:

a) myeloblasts, promyelocytes, myelocytes, metamyelocytes, nonsegmental neutrophils, segmental neutrophils;

b) myeloblasts, promyelocytes, nonsegmental neutrophils, segmental neutrophils;

c) myelocytes, metamyelocytes, nonsegmental neutrophils, segmental neutrophils, eosinophils.

8. In case of chronic myeloleukosis the peripheral blood smear is characterized by:

a) myeloblasts, promyelocytes, myelocytes, metamyelocytes, nonsegmental neutrophils, segmental neutrophils, basophils;

b) myeloblasts, promyelocytes, nonsegmental neutrophils, segmental neutrophils, eosinophils, basophils;

c) myeloblasts, metamyelocytes, nonsegmental neutrophils, segmental neutrophils, eosinophils, basophils.

9. Specify the attribute essentially distinguishing acute myeloblastic leukosis from chronic myeloleukosis:

a) blast cells in peripheral blood;

- b) vitamin B₁₂-deficient anemia;
- c) «hiatus leukemicus»;
- d) presence of extramedullary centers of bloodformation.

10. Specify the type of leukemia in which positive cytochemical reactions on peoxidase, phosphotase and lipids is manifested:

a) acute lympholeukosis;

- b) nondifferentiated leukosis;
- c) acute myeloleukosis.

11. In childhood these diseases occur most often:

- a) chronic myeloleukosis;
- b) chronic lympholeukosis;
- c) acute lymphoblasts leukosis.

12. Pancytosis (is an increase in maintenance of erythrocytes, leukocytes and thrombocytes in blood) is characteristic of:

a) chronic myeloleukosis;

- b) chronic lympholeukosis;
- c) erythremia (Vaqueze's illness).

13. Name the most frequent reasons of death of leukemia:

- a) bleeding;
- b) connection of secondary infection;
- c) haemorrhages in the vital organs;
- d) the all above listed.

14. Specify, whether neutropenia can be combined with leukemoid reaction:

a) yes;

b) no.

TYPICAL FORMS OF IMPAIRMENTS OF THE SYSTEM OF HEMOSTASIS

Specify a correct variant of the answer

1. Adhesion of thrombocytes to vascular wall amplifies under the action of the following factors:

a) formations of active thrombin;

b) liberation of fibrinogen from thrombocytes;

c) exposures of subendothelial layer collagen;

d) liberation of prostacyclin from endothelial cells;

e) liberation of thromboxane A₂ from thrombocytes.

2. The impairments of the secondary (coagulative) hemostasis is characteristic of the following diseases and forms of pathology:

a) thrombocytopathias;

b) hemophilias;

c) hemorrhagic vasculitis;

d) thrombotic thrombocytopenic purples;

e) Willebrand disease;

3. Activity of anticoagulative systems will be lowered in case of deficiency of anticoagulants:

a) antithrombin III;

b) prothrombinase;

c) thromboxane A₂.

4. Specify the mechanism of development of hemophilia A:

a) absence of Willebrand factor (gp Ib) in the membrane of thrombocytes receptors;

b) impairment of synthesis of factor VIII;

c) absence of fibrinogen (gp IIb/IIIa) in the membrane of thrombocytes receptors.

5. Specify the mechanism of development of Bernard-Soulier thrombocytopathia:

a) absence of Willebrand factor (gp Ib) in the membrane of thrombocytes receptors;

b) impairment of synthesis of factor VIII;

c) absence of fibrinogen (gp IIb/IIIa) in the membrane of thrombocytes receptors.

6. Lysis of blood clot is carried out by:

a) plasmin;

- b) antithrombin III;
- c) heparin.

7. Specify the mechanism of Glanzmann's disease development:

a) absence of Willebrand factor (gp Ib) in the membrane of thrombocytes receptors;b) impairment of synthesis of factor VIII;

c) absence of fibrinogen (gp IIb/IIIa) in the membrane of thrombocytes receptors.

8. Specify the mechanisms making up the basis of hemophilia:

a) propensity to bleedings owing to the impairment of vascular part of hemostasis;

b) propensity to bleeding owing to the impairment of coagulative hemostasis;

c) propensity to bleeding owing to functional inferiority of thrombocytes;

d) propensity to thrombus formation owing to activation of mechanisms of hemostasis.

9. Absolute deficiency of vitamin K in the organism will lead:

a) to the impairment of adhesion of thrombocytes;

b) to dysbacteriosis of intestines;

c) hypercoagulations;

d) to the impairment of aggregation of thrombocytes;

e) to deficiency of factors of coagulation in plasma of blood.

10. This parameter will change in case of hemophilia A:

a) duration of bleeding;

b) spontaneous aggregation of thrombcytes;

c) time of coagulation of blood;

d) thrombin time;

e) size of retraction of a clot.

11. In thrombocytosis abnormalities of hemocoagulation develop mainly:

a) in arteries and veins of a large diameter;

b) in arteries and veins of an average diameter;

c) in microvessels.

12. Specify pathological conditions and diseases which are accompanied by hypocoagulation:

a) chronic mechanical jaundice;

b) acute hemolitic anemia;

c) hypertension;

d) hyperlipidemia;

e) atherosclerosis.

13. Specify the correct sequence of events on the development of DIC-syndrome:

1. Activation of factors of cagulative system of blood and thrombocytic hemostasis;

2. Relative insufficiency of the anticoagulative system;

3. Disseminated thrombus formation with the development of hypoxa, dystrophy of tissues and organs;

4. Coagulopathia of consumption (coagulants and thrombocytes) with an exhaustion of anticoagulatie factors;

5. Hemorrhages:

- a) 1, 2, 3, 4, 5; b) 2, 1, 3, 4, 5;
- c) 3, 2, 1, 4, 5.

14. Specify the correct sequence of stages of DIC-syndrome:

1. Expressed hypocoagulation of proteins of blood;

2. Hypercoagulation of proteins of blood + initial signs of hypocoagulation;

3. Generalized hypercoagulation of proteins of blood and aggregation of thrombocytes:

a) 3, 2, 1; b) 1, 2, 3; c) 2, 1, 3.

15. Basically the first stage of DIC-syndrome is connected with:

a) activation of fibrinolysis;

b) activation of hemostasis;

c) exhaustion of factors of blood clotting;

d) suppression of fibrinolysis;

e) activation of primary anticoagulants.

16.Basically the second stage of DIC-syndrome is connected with:

a) increase in quantity of thrombocytes;

b) activation of hemostasis;

c) exhaustion of factors of blood clotting;

d) oppression of fibrinolysis;

e) activation of primary anticoagulants.

17. Specify the mechanism making up the basis of thrombophilia:

a) propensity to bleedings owing to abnormalities of vascular part of hemostasis;

b) propensity to bleeding owing to abnormalities of coagulative hemostasis;

c) propensity to bleeding owing to functional inferiority of thrombocytes;

d) propensity to thrombus formation owing to activation of mechanisms of hemostasis.

18. Specify the mechanism making up the basis of thrombocytopathia:

a) propensity to bleedings owing to abnormalities of vascular part of hemostasis;

b) propensity to bleeding owing to abnormalities of coagulative hemostasis;

c) propensity to bleeding owing to functional inferiority of thrombocytes;

d) propensity to thrombus formation owing to activation of mechanisms of hemostasis.

19. Thrombocytopenia is a decrease in quantity of thrombocytes in blood below:

a) 500×10⁹/l; b) 250×10⁹/l; c) 150×10⁹/l.

20. It is referred to hemorrhagies caused by thrombocytopathias:

- a) hemophilia C;
- b) Willebrand disease;

c) hemophilia B.

HYPOXIA

Specify a correct variant of the answer

1. Specify the most correct interpretation of the concept «hypoxia»:

a) pathological process which develops in connection with reduction of the maintenance of oxygen in arterial blood or tissues;

b) the pathological process, characterized by insufficiency of processes of biological oxidation;

c) the pathological process arising in abnormalities of blood supply of separate tissues or organs.

2. Hypoxia of a substrate type arises in cells in case of:

a) starvation;

b) acute respiratory insufficiency;

- c) miocardial infarction;
- d) poisoning with carbon monoxide.

3. Hypoxia of a reloading type arises in case of:

- a) chronic anemia;
- b) diabetes;
- c) emphysema of lungs;
- d) attack of stenocardia;

e) erectile stage of shock.

4. Specify the possible reason of hypoxia of a primarily-tissue type:

a) intravascular hemolysis;

b) acute blood loss;

c) increase in formation of pg E;

d) decrease in activity of enzymes of tissue respiration;

e) increase of activity of enzymes of tissue respiration.

5. Specify the condition in which affinity of Hb to oxygen decreases:

a) acidosis;

b) alkalosis;

c) hypocapnia.

6. Specify changes PaO₂ and pH in case of respiratory hypoxia:

- a) p_aO₂ and pH increase;
- b) p_aO₂ decreases, pH decreases;

c) p_aO₂ decreases, pH does not change;

d) p_aO₂ remains unchanged, pH increases;

e) p_aO₂ remains unchanged, pH decreases.

7. Specify the type of hypoxia, which develops in the organism during the first minutes after mass acute blood loss:

a) hemic;

- b) circulatoric;
- c) tissue;
- d) respiratory.

8. Specify the type of hypoxia which develops in the organism in 2-3 days after acute blood loss of average severity with a successful result of the carried out therapy:

a) mixed (tissue and circulatoric);

b) tissue;

c) hemic;

d) circulatoric.

TYPICAL FORMS OF EXTREME CONDITIONS

Specify a correct variant of the answer

1. The long stress can play the essential role in pathogenesis of the following diseases:

a) hypertension;

- b) ischemic disease of heart;
- c) stomach ulcer;
- d) neurosises;
- e) the all above listed.

2. Specify the stage of the general adaptable syndrome in which the hypertrophy of the cortex of adrenal glands develops:

a) in the stage of alarm;

b) in the stage of resistency;

c) in the stage of exhaustion.

3. The leading part of pathogenesis of cardiogenic shock is:

a) easing of the pumping function of the heart;

b) reduction of volume of blood;

c) falling of vascular tone.

4. Specify changes of nervous and endocrine systems, characteristic of the erectile stage of shock:

a) decrease in activity of the sympathoadrenal system, decrease in activity of the hypothalamic-pituitary system, block of the patient;

b) activation of the sympathoadrenal system, impellent and speech excitation, hyperventilation of lungs, hyperreflection.

5. Choose the manifestations describing the torpid phase of shock:

a) easing of effects of the sympathoadrenal system, reduction of cardiac output, deposition of blood, oligouria;

b) activation of the sympathoadrenal system, impellent and speech excitation, hyperventilation of lungs, hyperreflection.

6. The leading part of pathogenesis of septic shock is:

a) easing of the pumping function of the heart;

b) reduction of volume of blood;

c) falling of vascular tone.

7. Choose the correct statement:

a) coma always develops gradually, consistently passing some stages of consciousness disturbances;

b) coma can develop immediately, without expressed vicissitude.

8. It joins stress reaction firstly:

a) hypothalamic-pituitary-adrenal system;

b) sympathetic nervous system;

c) opioid system.

9. Specify the correct sequence of stages and reactions of the general adaptable syndrome:

a) resistency \rightarrow exhaustion \rightarrow alarm;

- b) alarm \rightarrow resistency \rightarrow exhaustion;
- d) alarm \rightarrow exhaustion \rightarrow resistency.

10. The most frequent complication of traumatic shock in patients with plural traumas is:

- a) fatty embolism;
- b) traumatic rhabdomyolysis (crush-syndrome);
- c) infections.

11. The leading part of pathogenesis of posthemorrhagic shock is:

- a) easing of the pumping function of heart;
- b) reduction of volume of blood;
- c) falling of vascular tone.

12. In case of development of general adaptable syndrome there is a primary hypertrophy of:

- a) glomerular zone of the cortex of adrenal glands;
- b) fascicular zone of the cortex of adrenal glands;
- c) reticular zone of cortex of adrenal glands;
- d) the medulla of adrenal glands;
- e) the thyroid gland.
- f) the posterior hypophysis.

13. The main stress-limiting systems are:

- a) the system of opioid proteins;
- b) the serotonergic system;
- c) the GABA-ergic system;
- d) the all above listed.

PATHOPHYSIOLOGY OF IMMUNE SYSTEM. ALLERGY. AUTOIMMUNE PROCESSES

Specify a correct variant of the answer

1. Allergy is:

- a) a specific raised immune secondary reaction to an allergen;
- b) a raised reaction to HLA-antigenes;
- c) a reaction strengthened by adjuvants;
- d) an immune reaction to the influence of several antigenes and allergens.

2. Immediate allergic reactions develop:

- a) in 30 min after the hit of allergen into the organism;
- b) in 1 day after the hit of allergen into the organism;
- c) in a day after a sting of a bee;
- d) in 2 days after an injection of drugs.

3. Allergic reactions of I type (IgE-dependent) develop on interaction of allergen with:

a) IgE-antibodies connected by basophils;

b) IgE-antibodies circulating in blood;

c) complexes of IgE-antibodies and complement;

d) Fc-fragments of IgE-antibodies.

4. In allergic IgE-dependent reactions we allocate:

- a) antibodies;
- b) histomine;
- c) IL-2;
- d) CD₄.

5. Anaphylaxis reactions is an interaction of:

- a) T-lymphocytes with antigenes;
- b) macrophages with bacteria;
- c) the IgE-antibodies connected with basophils and allergen;
- d) IgM-antibodies and antigenes.

6. IgE-antibodies to allergen have specific:

- a) Fc-fragments;
- b) Fab-fragments;
- c) joined sites;
- d) C-domains.

7. IgE-antibodies contact with basophils:

- a) Fab-fragments;
- b) Fce-fragments;
- c) Fcγ-fragments;
- d) Fcµ-fragments.

8. Cytotoxic allergic reactions arise on interaction:

a) IgE-antibodies and antigene on cells;

- b) IgG antibodies with cellular connected antigene and complement;
- c) IgG antibodies and soluble antigene;
- d) IgG antibodies and toxins;
- e) IgM antibodies with complement.

9. Immune complex reactions are characterized by:

- a) formation of complex of IgE-antibodies and antigene;
- b) formation of complex of IgG-antibody + antigene + complement;
- c) formation of B-lymphocyte + antigene complex;
- d) formation of macrophage + antigene complex.

10. Hypersenitivity of a delayed type develops:

- a) in 15 mines;
- b) in 2 hours;
- c) in 6 hours;
- d) in 48 hours.

11. Specify secondary cells-targets in allergic reactions developing on I type of the immune response:

a) neutrophils;

b) basophils;

c) fibroblasts;

d) mast cells.

12. Name the reaction which is not developing on I (reagin) type of immune damage:

a) urticaria;

b) «dust» bronchial asthma;

- c) anaphylactic shock;
- d) serum disease;

e) pollinosis.

13. Specify optimum terms for reproduction of anaphylatic shock in porpoises after carrying out of active sensitization:

a) 15-20 min;
b) 6-8 hours;
c) 24-48 hours;
d) 6-8 days;

e) 14–15 days.

14. Specify the time of the maximal manifestation of skin reactions after repeated influence of allergen in allergic reactions developing on IV type of immune damage:

- a) 15–30 min;
- b) 6–8 hours;
- c) 24-48 hour;
- d) 10-14 days.

15. Specify, what process plays the basic role in pathogenesis of diseases developing on I type of immune damage:

a) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is on the surface of cells-targets at participation of complement, phagocytes and NK-cells; b) interaction of circulating antibodies (IgG, IgM-classes) with antigen which is in excess supply and with formation of immune complexes at complement participation;

c) interaction of sensibilized lymphocytes with antigene;

d) interaction of the antibodies fixed on cells-targets (IgE, IgG_4) with antigene without complement participation.

16. Specify, what process plays the basic role in pathgenesis of diseases developing on II type of immune damage:

a) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is on the surface of cells-targets at complement participation , phagocytes and NK-cells;

b) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is in excess supply and with formation of immune complexes at complement participation;

c) interaction of sensibilized lymphocytes with antigene;

d) interaction of antibodies fixed on cells-targets (IgE, IgG_4) with an antigene without complement participation.

17. Specify, what process plays the basic role in pathogenesis of diseases developing on III type of immune damage:

a) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is on the surface of cells-targets at complement participation, phagocytes and NK-cells;

b) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is in excess supply and with formation of immune complexes at complement participation;

c) interaction of sensibilized lymphocytes with antigene.

d) interaction of antibodies fixed on cells-targets (IgE, IgG_4) with antigene without complement participation.

18. Specify, what process plays the basic role in pathogenesis of diseases developing on IV type of immune damage:

a) interaction of circulating antibodies (IgG, IgM-classes) with antigene which is being on a surface of cells-targets at complement participation, phagocytes and NK-cells;

b) interaction of circulating antibodies (IgG, IgM-classes) with antigene in excess supply and with formation of immune complexes at complement participation;

c) interaction of sensibilized lymphocytes with antigene;

d) interaction of antibodies fixed on cells-targets (IgE, IgG_4) with antigene without complement participation.

19. Reaction of immune damage on III type is observed in:

- a) contact dermatitis;
- b) allergic vasculitis;
- c) autoimmune hemolitic anemias;
- d) graft versus host reaction;
- e) hay fever.

20. Specify classes of antibodies to which antibodies are referred in diseases developing on II and III types of immune damage:

- a) Ig G₁;
- b) Ig G₃;
- c) Ig M;
- d) the all above listed.

21. Specify, which disease from the listed below can develop on III type of immune damage:

- a) serum disease;
- b) local reactions as Arthus phenomenon;
- c) allergic vasculitis;
- d) allergic alveolitis;
- e) the all above listed.

22. An active sensitization of the organism can be caused by:

- a) introductions of specific antibodies;
- b) introductions of antigenes;
- c) introductions of sensibilized lymhocytes effectors;
- d) introductions of immune stimulators;
- e) introductions of immunosupressants.

23. We don't refer to «back from barrier» the following organ's and tissues:

- a) tissue of crystalline lens of the eye;
- b) testicules tissue;
- c) tissue of the kidney;
- d) colloid of the thyroid gland;
- e) myelin.

24. Specify the diseases referred to autoimmune ones:

- a) Hashimoto's thyroiditis;
- b) rheumatoid arthritis;
- c) myasthenia gravis;
- d) systemic lupus erythematosus;
- e) the all above listed.

25. Specify diseases which are not referred to autoimmune ones:

a) Hashimoto's thyroiditis;

- b) Arthus phenomenon;
- c) rheumatoid arthritis;
- d) myasthenia gravis;
- e) systemic lupus erythematosus.

26. Specify autoimmune disease caused by formation of organspecific autoantibodies:

a) Hashimoto's thyroiditis;

- b) postvaccinal encephalomyelitis;
- c) systemic lupus erythematous;
- d) postinfarction myocarditis.

27. Specify, whether participation of T-lymphocytes — effectors is possible in pathogenesis of autoimmune diseases:

a) yes;

b) no.

PATHOPHYSIOLOGY OF THE IMMUNE SYSTEM. IMMUNODEFICIENT CONDITIONS

Specify a correct variant of the answer

1. The central organs of the immune system are:

- a) lymph nodes;
- b) spleen;
- c) tonsils;
- d) red bone marrow;
- e) Peyer's patches.

2. Specific immunity:

- a) arises after suffered infectious disease;
- b) is formed after vaccination;
- c) is formed after introduction of antibodies;
- d) is formed during embryogeneis;
- e) is caused by features of metabolism of a given kind.

3. The organ of differentiation of T-lymphocytes is:

- a) lymph nodes;
- b) spleen;
- c) liver;

- d) thymus;
- e) tonsils.

4. Specify the factor of interaction of cells of the immune system:

- a) antibodies;
- b) thrombocytes;
- c) thymosins;
- d) interleukins;
- e) lectins.

5. Specify cells participating in phagocytosis:

- a) erythrocytes;
- b) T-lymphocytes;
- c) neutrophils;
- d) B-lymphocytes;
- e) basophils.

6. Specify antibodies, characteristic of the primary immune response:

- a) IgE;
- b) IgD;
- c) IgA;
- d) IgM;
- e) IgG.

7. Specify the antibodies prevailing at the secondary immune response:

- a) IgE;
- b) IgD;
- c) IgA;
- d) IgM;
- e) IgG.

8. Specify antibodies, characteristic of allergic reactions of an immediate type:

- a) IgA;
- b) IgM;
- c) IgG;
- d) IgE;
- e) IgD.

9. Specify the marker of T-helpers:

- a) CD₁;
- b) CD₂;

- c) CD₄;
- d) CD₈;
- e) CD₅.

10. Specify the marker of T-cytotoxic lymphocytes:

- a) CD₁;
- b) CD₂;
- c) CD₄;
- d) CD₈;
- e) CD₅.

11. Specify the marker of B-lymphocytes:

- a) CD₁;
- b) CD₂;
- c) CD₄;
- d) CD_8 ;
- e) CD_{5;}
- f) CD₇₂.

12. Specify a noninfectious type of immunity:

- a) antineoplastic;
- b) antibacterial;
- c) anti-virus;
- d) antifungoid;
- e) antiparasitic.

13. Specify an anti-infectious type of immunity:

- a) autoimmunity;
- b) antibacterial;
- c) transplantation;
- d) antineoplastic;
- e) reproductive.

14. Specify the inductor of allergic reactions of a delayed type:

- a) B-lymphocytes;
- b) immunoglobulins E;
- c) complement;
- d) T-lymphocytes;
- e) immune complexes.

15. Specify the property of haptens:

- a) high molecular weight;
- b) immunogenicity;

c) low molecular weight;

- d) toxicity;
- e) albuminous nature.

16. A high -grade antigene is:

- a) a high-molecular protein;
- b) carbohydrates;
- c) low-molecular particles;
- d) mineral salts;
- e) lipids.

17. Specify the type of immunity developed after an introduction of anatoxin:

- a) natural active;
- b) natural passive;
- c) unsterile;
- d) antitoxic;
- e) artificial passive.

18. Specify the preparation for creation of artificial active immunity:

- a) γ-globulin;
- b) interferon;
- c) vaccine;
- d) interleukin;
- e) antitoxic serum.

19. Specify the purpose of application of antitoxic serums:

- a) stimulation of T-cellular immunity;
- b) creation of natural passive immunity;
- c) creation of natural active immunity;
- d) creation of artificial active immunity;
- e) creation of artificial passive immunity.

20. Specify the purpose of application of vaccines:

- a) creation of natural passive immunity;
- b) immunoprophylaxis, creation of artificial active immunity;
- c) creation of natural active immunity;
- d) creation of artificial passive immunity;
- e) diagnostics of infectious diseases.

21. The classical way of complement activation is started by:

- a) c3-c9;
- b) polysaccharide;
- c) complex of antigene-antibody;

d) toxin;

e) DNA.

22. Reaction for the definition of microbe type is called:

- a) blasttransfomation;
- b) reaction of neutralization;
- c) reaction of agglutination;
- d) reaction of precipitation;
- e) reaction of hemolisis.

23. Name the reaction in which complement participates:

- a) reaction of direct hemagglutination;
- b) Reiters complement fixation test, R(CFT);
- c) reaction of immune agglutination;
- d) immune-enzyme test;
- e) radioimmune sorbent test.

24. Specify the reaction in which erythrocyte diagnosticum is applied:

- a) reaction of precipitation;
- b) reaction of direct hemagglutination;
- c) immune-enzyme test;
- d) flocculation test;
- e) R(CFT).

25. Specify a component for immune-enzyme test:

- a) diagnosticum;
- b) complement;
- c) antiglobulin serum, labelled by fluorochromium;
- d) antiglobulin serum, labelled by isotope;
- e) antiglobulin serum, labelled by enzyme.

26. Specify the specific system of immunity:

- a) B-system;
- b) the system of mononuclear phagocytes;
- c) the system of granulocytes;
- d) the system of thrombocytes.

27. Specify the way of acquisition of natural active immunity:

- a) through milk of mother;
- b) after suffered infectious disease;
- c) through placenta from mother to fetus;
- d) after vaccination;
- e) after introduction of antibodies.

28. Specify the way of acquisition of natural passive immunity:

- a) after suffered infectious disease;
- b) after vaccination;
- c) after introduction of antibodies;
- d) through placenta from mother to fetus;
- e) after introduction of interferon.

29. Specify the way of acquisition of artificial active immunity:

- a) after suffered infectious disease;
- b) after vaccination;
- c) after introduction of antibodies;
- d) through placenta from mother to fetus;
- e) after blood transfusion.

30. Specify the way of acquisition of artificial passive immunity:

- a) after suffered infectious disease;
- b) after vaccination;
- c) after introduction of antibodies (antiserums);
- d) through placenta from mother to fetus;
- e) after introduction of anatoxins.

31. Specify a preparation which is used for treatment of tetanus:

a) BCG;

- b) tetanospasmin;
- c) tetanolysin;
- d) antitoxic serum;
- e) monoclonal antibodies.

32. Secondary immunodeficiencies can arise in:

- a) X-ray irradiation, corticosteroid therapy, thymectomia;
- b) leukemias;
- c) virus, bacterial, fungoid and protozoan infections;
- d) septic conditions;
- e) the all above listed.

33. Specify the mechanism defining impairments of protective functions of the organism in a Swiss type of immunodeficiency:

- a) impairments of cellular immunity;
- b) impairments of humoral immunity;
- c) the combined impairments of humoral and cellular immunity;
- d) impairments of phagocytosis.

34. Specify the mechanism defining impairments of protective functions of the organism at syndrome of Dee-Georgeses:

a) impairments of cellular immunity;

- b) impairments of humoral immunity;
- c) the combined impairments of humoral and cellular immunity;
- d) impairments of phagocytosis;

e) the combined impairments of humoral immunity, cellular immunity, phagocytosis.

35. Specify the mechanism defining impairments of protective functions of the organism in Bruttons syndrome:

- a) impairments of cellular immunity;
- b) impairments of humoral immunity;
- c) the combined impairments of humoral and cellular immunity;
- d) impairments of phagocytosis;
- e) the combined impairments humoral immunity, cellular immunity, phagocytosis.

36. Specify the way of transmission of HIV-infection:

- a) alimentary;
- b) sexual;
- c) fecal-oral;
- d) air-drop;
- e) inoculable.

37. Specify cells which are mainly affected by HIV:

- a) T-helpers;
- b) granulocytes (CD₁₄, CD₁₈);
- c) B-lymphocytes;
- d) erythrocytes.

38. Specify the method which is used for diagnostic of AIDS:

- a) bacteriological;
- b) serological;
- c) light microscopy;
- d) skin-allergic test.

39. Specify natural killers:

- a) cells of memory;
- b) T-lymphocytes;
- c) natural cytotoxic lymphocytes;
- d) activated T-lymphocytes;
- e) phagocytic monocytes.

40. T-helpers of 1 type form:

- a) IL-4;
- b) IL-2;
- c) IL-5;
- d) IgD;
- e) IgA.

41. Macrophages allocate:

- a) IL-1;
- b) IL-2;
- c) IgM;
- d) IgE;
- e) agglutinin.

42. T-helpers 2 allocate:

- a) lysozyme;
- b) IgA₁;
- c) IL-4;
- d) CRP;
- e) γ-interferon.

43. Allogenic antigenes (or isoantigenic) are:

a) antigenes of mycobacteria;

- b) antigenes of the cells different in individuals of the given type;
- c) antigenes different in cells of different types;
- d) antigenes of synthetic substances and subjects;
- e) the molecules defining organic specificity.

44. They are simultaneously available only on erythrocytes of man:

- a) antibodies and A antigens;
- b) antibodies and B antigenes;
- c) antibodies and AB antigenes;
- d) antibodies and A antigenes;
- e) AB antigenes.

45. Endocellular bacteria destroy:

- a) activated macrophages;
- b) neutrophils;
- c) natural killers;
- d) antibodies.

46. Interferons:

a) lysis viruses;

- b) induce enzymes destroying virus in cells;
- c) inhibit division of bacteria;
- d) strengthen phagocytosis of viruses;
- e) strengthen formation of peroxide.

47. It is characteristic of T-cellular deficiencies:

- a) absence of antibodies;
- b) absence of complement;
- c) presence of virus infections;
- d) oppression of phagocytosis.

48 It is observed. in case of B-cellular deficiencies:

- a) decrease in a level of antibodies;
- b) cytotoxic lymphocytes;
- c) activation of phagocytosis;
- d) increase in a level of all IL;
- e) absence of HLA-antigenes.

49. The syndrome of Dee-Georgeses is accompanied by:

- a) aplasia of thymus;
- b) hypoplasia of spleen;
- c) immaturity of extremites;
- d) absence of macrophages;
- e) hyperplasia of tonsils.

50. It is characteristic of Bruton's agammaglobulinemia:

- a) viral infections in girls;
- b) bacterial infections in boys;
- c) absence of T-lymphocytes;
- d) hypocomplementemia.

51. It is observed in deficiencies of phagocytes:

- a) suppression of digestion of bacteria;
- b) suppression of digestion of viruses;
- c) absence of IL-1;
- d) absence of T-helpers.

52. Immunological tolerance is:

- a) unresponsiveness of the immune system to antigens;
- b) suppression of phagocytosis of bacteria;
- c) suppression of synthesis of IgA-antibodies;
- d) presence of high activity of natural killers.

53. Graft immunity is a:

a) reaction to AB antigenes;

b) immune reaction to HLA-antigenes;

c) immunity to autoplast;

d) a high level of antibodies to HLA-antigenes.

54. «Processing» of antigene is:

a) a processing of peptides of a certain size in antigenrepresenting cells;

b) a carry from T- to B-lymphocytes;

c) a connection to CD_4 and CD_8 molecules;

d) splitting up to amino acids.

55. Antibodies are synthesized and secreted by:

a) T-lymphocytes;

b) neutrophils;

c) plasmatic cells;

d) macrophages.

56. Complement is capable to attach:

a) IgM and IgG;

b) IgA;

c) IgD;

d) IgE.

57. It is capable to pass through the placentary barrier:

- a) IgM;
- b) IgG;

c) IgA;

d) IgD.

58. Secretory IgA:

a) protects the skin;

b) protects mucous membranes;

- c) connects complement;
- d) neutralizes parasites.

59. IgE participates in:

- a) neutralizations of bacteria;
- b) linkage of complement;
- c) allergic reactions;
- d) a primary immune response.

60. The human immunodeficiency virus affects:

- a) neutrophils;
- b) thrombocytes;
- c) T-helpers;
- d) erythrocytes.

61. The child of the first weeks of life is protected by the following antibodies:

- a) IgG;
- b) IgM;
- c) IgA;
- d) IgD.

62. Secretory IgA is synthesized by plasmatic cells of:

- a) lymph nodes;
- b) spleen;
- c) mucous membranes;
- d) bone marrow.

63. Plasmatic cells are formed from:

- a) B-lymphocytes;
- b) T-lymphocytes;
- c) macrophages;
- d) fibroblasts.

64. Deficiency of antibodies is observed in case of:

- a) agammaglobulinemia;
- b) deficiency of T-lymphocytes;
- c) insufficiency of phagocytes.

65. Increase of IgG in blood is characteristic of:

- a) agammaglobulinemia;
- b) stimulation of phagocytosis;
- c) a primary immune response;
- d) a secondary immune response.

66. Increase of IgM in blood is marked in case of:

- a) a primary immune response;
- b) activation of macrophages;
- c) syndrome of Dee-Georgeses;
- d) activation of complement.

67. The group of blood on a standard serum cannot be defined:

a) for an adult man;

- b) for a young man;
- c) for a teenager;
- d) for a newborn;
- e) for a pregnant woman.

68. Killer cells are:

- a) NK-cells;
- b) corpulent cells;
- c) erythrocytes;
- d) thrombocytes.

69. Specify the central organs of T-part of immunity:

- a) thymus;
- b) tonsils;
- c) spleen;
- d) lymph nodes;
- e) appendix.

70. Specify the central organ of B-part of immunity:

- a) thymus;
- b) bone marrow;
- c) spleen;
- d) lymph nodes;
- e) appendix.

71. It refers to the system of mononuclear phagocytes:

- a) macrophages;
- b) neutrophils;
- c) erythrocytes;
- d) thrombocytes;
- e) lymphocytes.

72. The HLA-system includes molecules of:

a) IgM, IgG;
b) HLA-A, B, C;
c) Ig and Ig;
d) CD₃-CD₈;
e) tumor necrosis factor.

73. HLA-molecules of II class are:

- a) HLA-B;
- b) HLA-DR, DP, DQ;
- c) HLA-M;
- d) HLA-C.

74. A tumour differs from a normal tissue on:

- a) group of antigenes;
- b) HLA-antigenes;
- c) tumour specific antigenes;
- d) virus antigenes.

75. Name the most widespread evidence of HIV-infection:

- a) revealing of antibodies to antigenes of HIV-virus with immne-enzyme analysis;
- b) reaction of neutralization of virus;
- c) method of animal infection.

76. Specify immunodeficiency, characteristic of virus infection:

- a) B-system;
- b) complement;
- c) the system of granulocyte macrophages monocytes;
- d) T-system.

77. Specify immunodeficiency from which only boys suffer (disease is X-linked):

- a) Louis-Bar syndrome;
- b) agammaglobulinemia;
- c) Nezelof type of thymic dysplasia;
- d) Wiskott-Aldrich syndrome.

78. Specify the congenital immunodeficiency disease which is combined with secondary albinism:

a) agammaglobulinemia;

- b) chronic granulematous disease;
- c) Wiskott-Aldrich syndrome;
- d) Chediak-Higashi syndrome;
- e) Loui-Bar synrome.

79. Immunodiagnosis on definition of antibodies to cellular receptors is carried out for revealing of:

a) thyreotoxicosis;

- b) Sjogren syndrome;
- c) rheumatiod arthritis;
- d) Goodpasture's syndrome.

80. Specify the vitamin possessing an immunostimulative effect on phagocytosis:

- a) vitamin C;
- b) vitamin B₁;
- c) vitamin B₁₂.

81. Specify, what forms of chronic lympholeukosis occurs more often:

- a) T-cellular;
- b) thrombocytic;
- c) granulocytic;
- d) monocytic;
- e) B-cellular.

IMPAIRMENTS OF ACID-BASE BALANCE

Specify a correct variant of the answer

1. Specify the reason of gas alkalosis:

- a) superfluous receipt of alkalis into the organism;
- b) alveolar hyperventilation;
- c) significant loss of gastric juice;
- d) hyperproduction of mineralocorticoids;
- e) insufficient excretion of bases by kidneys.

2. pH of capillary blood in case of compensated impairments of acidbase balance of the organism can be displaced within the limits of:

- a) 7,30–7,50;
- b) 7,35–7,45;
- c) 7,30-7,35.

3. Occurrence of respiratory alkalosis is possible in case of:

- a) unrestrained vomiting;
- b) pneumosclerosis;
- c) profuse diarrheas;
- d) increase of concentration of CO₂ in air;
- e) staying in conditions of high mountains.

4. Acidosis in case of chronic renal insufficiency mainly develops owing to:

a) expressed decrease of proximal reabsorption of bicarbonate;

b) excessive return diffusion of ions of hydrogen from a lumen of renal tubules in intersticium;

c) significant decrease of excretion of NH4⁺;

d) acute decrease in allocation of titrable acids (uni-and bi-replaced salts of phosphoric acid).

5. Acidosis in the center of inflammation is caused by:

- a) increase in maintenance of polypeptides;
- b) accumulation of ions of sodium;

c) accumulation of ions of potassium;

d) accumulation of lactic acid.

6. Impairments of physiological processes arise in case of shift of pH of arterial blood:

a) on 0,1 and more;

- b) not less than on 0,2;
- c) not less than on 0,3;
- d) not less than on 0,4.

7. Specify the reasons of gas acidosis:

a) superfluous introduction of mineral acids;

- b) alveolar hyperventilation;
- c) superfluous formation of sour products of exchange;

d) loss of intestinal juice in a great amount;

e) insufficient allocation of sour metabolits by kidneys.

8. Compensated gas alkalosis is characterized by:

a) reduction of P_aCO₂ and standard bicarbonate of blood;

b) reduction of P_aCO₂ and increase of standard bicarbonate of blood;

c) increase of P_aCO_2 and standard bicarbonate of blood.

9. Specify the type of shift of acid-base balance in which indemnification of the broken condition is carried out due to hyperventilation of lungs:

a) metabolic acidosis;

b) metabolic alkalosis;

c) gas acidosis;

d) gas alkalosis.

10. The parameter of titratable acidities of urine in case of metabolic acidosis:

a) will raise;

b) will go down;

c) will not change.

11.Compensatory reactions of the organism in case of metabolic acidosis are:

a) linkage of ions of hydrogen by proteins;

b) hyperventilation of lungs;

c) exchange of ions of hydrogen for ions of potassium and calcium of cells;

d) the all above listed.

12. In case of compensated gas acidosis we observe:

a) increase of P_aCO_2 and reduction of standard bicarbonate of blood

b) reduction of P_aCO_2 and standard bicarbonate of blood

c) increase P_aCO₂ and standard bicarbonate of blood

13. Nervous-muscular excitability in case of noncompensated gas alkalosis:

- a) will go down;
- b) will raise;
- c) will not change.

14. Specify the type of impairments of acid-base balance of the organism in which pH of capillary blood is 7,49:

- a) compensated alkalosis;
- b) compensated acidosis;
- c) noncompensated alkalosis;
- d) noncompensated acidosis.

15. Alveolar hyperventilation can result:

- a) to gas alkalosis;
- b) to nongas alkalosis;
- c) to gas acidosis.

16. Alveolar hyperventilation can result:

- a) to nongas alkalosis;
- b) to gas acidosis;
- c) to gas alkalosis.

17. Specify the type of impairments of acid-base balance of the organism in case of which pH of capillary blood is 7,25:

a) compensated alkalosis;

- b) about noncompensated alkalosis;
- c) about compensated acidosis;
- d) about noncompensated acidosis.

18. The following factor participates in pathogenesis of hypostases in case of heart failure:

- a) increase of hydrostatic pressure in venous part of capillaries;
- b) reduction of the maintenance of aldosterone and vasopressin in blood;
- c) reduction of sodium and water reabsorption in renal tubules.

19. Development of tetany is characteristic of:

- a) metabolic acidosis;
- b) metabolic alkalosis;
- c) gas acidosis.

PATHOPHYSIOLOGY OF TYPICAL IMPAIRMENTS OF EXCHANGE OF PROTEINS

Specify a correct variant of the answer

1. Specify the condition, accompanied by positive nitrogenous balance:

- a) starvation;
- b) superfluous secretion or administration of catabolic hormones;
- c) infectious diseases;
- d) intensive regeneration.

2. Specify conditions, accompanied by negative nitrogenous balance:

- a) starvation;
- b) thermal burns;
- c) superfluous secretion or administration of catabolic hormones;
- d) infectious diseases;
- e) the all above listed.

3. Specify the pathological condition accompanied by hyperproteinemia:

- a) strengthening of synthesis of antibodies;
- b) diseases of liver;
- c) impairment of protein aspiration;
- d) proteinuria.

4. Specify the form of pathology the basis of which is made with hereditary defect in the metabolism of amino acid phenylalanine:

- a) alkaptonuria;
- b) tyrosinosis;
- c) phenylketonuria;
- d) albinism.

5. Specify, what change accompanies the deficiency of vitamin E:

- a) oppression of oxidation-reduction reactions;
- b) impairments of mineralization of bone tissue;
- c) impairments of synthesis of factors of blood coagulation;
- d) development of sterility;
- e) increase of excitability of nervous tissue.

6. Specify, what change accompanies the deficiency of vitamin K:

- a) suppression of oxidation-reduction reactions;
- b) impairments of mineralization of bone tissue;
- c) impairments of synthesis of factors of blood coagulation;

- d) development of sterility;
- e) increase of excitability of nervous tissue.

7. Absolute deficiency of vitamin K in the organism will lead:

- a) to impairments of adhesion of thrombocytes;
- b) to dysbacteriosis of intestines;
- c) hypercoagulations;
- d) to impairments of aggregation of thrombocytes;
- e) to deficiency of coagulative factors in plasma of blood.

8. Specify, what change accompanies with the deficiency of vitamin D:

- a) suppression of oxidation-reduction reactions;
- b) impairments of mineralization of bone tissue;
- c) impairments of synthesis of factors of blood coagulation;
- d) development of sterility;
- e) increase of excitability of nervous tissue.

9. Specify, what change accompanies the deficiency of vitamin C:

- a) suppression of oxidation-reduction reactions;
- b) impairments of mineralization of bone tissue;
- c) impairments of synthesis of factors of blood coagulation;
- d) development of sterility;
- e) increase of excitability of nervous tissue.

10. Specify, what change accompanies the deficiency of vitamin B₆:

- a) suppression of oxidation-reduction reactions;
- b) impairments of mineralization of bone tissue;
- c) abnormalities of synthesis of factors of blood coagulation;
- d) development of sterility;
- e) increase of excitability of nervous tissue.

PATHOPHYSIOLOGY OF IMPAIRMENTS OF LIPID EXCHANGE. ATHEROSCLEROSIS

Specify a correct variant of the answer

1. «Foamy» cells are formed in case of accumulation of lipids in:

- a) macrophages;
- b) lymphocytes;
- c) neutrophils;
- d) endothelial cells.

2. Specify the most probable sequence of phenomena during atherosclerosis:

- 1. Migration of smooth muscle cells into the center of accumulation of lipids;
- 2. Capture of lipoproteins by macrophages, transformation into «foamy» cells;
- 3. Allocation of growth and chemotaxic factors for smooth muscle cells;
- 4. Damage of endothelium and accumulation of lipoproteins in intim of arteries;
- 5. Activation of synthesis of collagen and elastin by smooth muscle cells;
- 6. Formation of fibrous capsule around the center of lipids accumulation:
 - a) 4, 3, 1, 2, 5, 6;
 - b) 4, 2, 3, 1, 5, 6;
 - c) 2, 4, 5, 1, 3, 6.

3. Specify risk factors of development of atherosclerosis:

- a) hypoinsulinism;
- b) hyperlipidemia;
- c) adiposity;
- d) arterial hypertensia;
- e) chronic damage of a vascular wall;
- f) smoking;
- g) the all above listed.

4. The development of atherosclerosis is promoted by:

- a) hereditary or acquired hypercholesterinemia;
- b) hereditary hyperlipidemia;
- c) arterial hypertensia;
- d) diabetes;
- e) the all above listed.

5. Specify possible consequences of atherosclerotic lesions of arteriol walls:

- a) occlusion of a lumen of vessels;
- b) stenosis of a lumen of vessels;
- c) parietal thrombosis;
- d) microaneurysms;
- e) necrosis;
- f) ulceration;
- g) the all above listed.

6. Specify the factors promoting the development of atherosclerosis in case of diabetes:

- a) superfluous accumulation of sorbite in walls of vessels;
- b) excessive glycosylation of proteins of tissue of vascular wall;
- c) dislipoproteidemia;
- d) hyperlipoproteidemia;
- e) the all above listed.

7. Specify the factor which is not promoting the development of atherosclerosis in case of diabetes:

a) superfluous accumulation of sorbite in walls of vessels;

b) excessive glycosylation of proteins of tissue of vascular wall;

c) dislipoproteidemia;

d) hyperlipoproteidemia;

e) the all above listed.

8. Specify the factor promoting the development of atherosclerosis in case of adiposity:

a) polyuria;

b) hypercholesterinemia;

c) polydipsia.

9. Primary atherosclerotic changes of arteries (lipid strips) for the first time appear at the age of:

a) till 10 years;

b) 20-25 years;

c) 30–35 years;

d) 40-45 years;

e) 50 years and older.

10. Choose the fullest answer. Skewendger-receptors are stipulated for contact with:

a) glycosylated lipoproteids;

b) lipoproteins of low density of all modification;

c) desialic lipoproteinds;

d) lipoproteids as a result of peroxidation.

11. The development of adiposity is pathogenetically atypical for:

a) diabetes of II type;

b) diabetes of I type.

12. Specify, whether hyperlipidemia is possible without expressed hypercholesterinemia:

a) yes;

b) no.

13. Specify, whether adiposity without change of number of adipocytes is possible:

a) yes; b) no.

72

14. Specify the type of adiposity in case of which the number of adipocytes increases in organism:

a) hypertrophic;

b) hyperplastic.

15. Choose the correct statement:

a) android adiposity is a more significant risk factor of atherosclerosis, than gynoid;

b) gynoid adiposity is a more significant risk factor of atherosclerosis, than android;

c) subcutaneous adiposity is a more significant risk factor of atherosclerosis, than visceral.

16. The cholesteric factor of atherogenicy becomes a significant risk factor of atherosclerosis when it exceeds:

a) 1;

- b) 2;
- c) 3;
- d) 4.

PATHOPHYSIOLOGY OF IMPAIRMENTS OF CARBOHYDRATE EXCHANGE. DIABETES

Specify a correct variant of the answer

1. Specify the correct statement concerning diabetes:

a) polyuria secondary causes polydipsia;

b) polydipsia secondary causes polyuria.

2. Choose the most typical manifestation of impairments of lipid exchange in case of diabetes of 1 type:

a) strengthening of lipolysis;

b) fatty infiltration of liver;

c) suppression of lipogenesis;

d) strengthening of ketogenesis;

e) the all above listed.

3. Specify the impairment which plays role of the basic part in pathogenesis in diabetic coma in patients with diabetes of 1^{st} type:

a) hypernatremia;

b) hyperglycemia;

- c) hyperketonemia;
- d) hyperpotassemia.

4. Specify the basic pathogenetic factor of occurrence of diabetes of the $2^{\underline{d}}$ type:

- a) the block of transformation of proinsulin in insulin;
- b) deficiency and/or low affinity to insulin of receptors of cells-targets;
- c) hyperglycemia;
- d) hyperketonemia.

5. The main pathogenetic part of hypoglycemic coma is:

- a) carbohydrate and power «starvation» of brain neurons;
- b) carbohydrate «starvation» of myocardium;
- c) blood hyposmium;
- d) noncompensated ketoacidosis.

6. Specify the impairment which plays a role of the basic part in pathogenesis of hyperosmium diabetic coma:

- a) expressed hypernatremia;
- b) expressed hyperpotassemia;
- c) noncompensated ketoacidosis.

7. Specify the reason of polyuria in early stage of diabetes:

- a) microangiopathia of kidneys;
- b) hyperglycemia;
- c) ketonemia;
- d) cholesterolemia.

8. Glucosuria is observed in case of:

- a) diabetes insipidus;
- b) hyperosmium diabetic coma;
- d) hyperlipidemia;
- e) hyperlactacidemia.

9. Specify the basic manifestations of diabetes insipidus:

- a) polyuria;
- b) dehydration of the organism;
- c) low density of urine;
- d) hypotension;
- e) the all above listed.

10. Etiological factors of diabetes insipidus are not:

a) hereditary caused superfluous production of oxytocin;

b) damage of anterior nuclei of hypothalamus in case of traumatic, tumoral, infectious nature;

- c) hereditary inability to production of vasopressin;
- d) congenital or acquired resistency of kidneys to antidiuretic hormone.

11. Specify complications of long-lasting diabetes:

a) immunodeficiency conditions;

- b) decrease in resistency to infections;
- c) microangiopathy;
- d) macroangiopathy;
- e) the all above listed.

12. Hyperglycemia is caused with the excess of:

- a) adrenaline;
- b) thyroid hormones (T₃, T₄);
- c) glucocorticoids;
- d) somatotropic hormone;
- e) glucagon;
- f) the all above listed.

13. Specify the factors causing hyperglycemia:

- a) prevalence of processes of CNS braking;
- b) restriction of consumption of carbohydrates with food;
- c) decrease in activity of the sympathetic nervous system;
- d) the all above listed.

14. Specify the factor which is not promoting the development of diabetic angiopathias:

- a) excessive glycosylation of proteins;
- b) hyperlipoproteinemia;
- c) dislipoproteinemia;
- d) accumulation of sorbite in walls of vessels;
- e) strengthening of glycogenesis in cells of vessels walls.

15. Specify the possible reason of aglycogenosises:

- a) alimentary hypoglycemia in case of starvation;
- b) repression of genes coding the synthesis of glycogensynthetases;
- c) inhibition of glycogen synthesis from amino acids in the liver;

d) glucosuria;

- e) low sensitivity of receptors to «anti-insulin» hormones;
- f) high activity of glycigenolysis enzymes.

TYPICAL FORMS OF IMPAIRMENTS OF HEART FUNCTIONS

Specify a correct variant of the answer

1. Specify the reason of acute heart failure:

a) acute myocardial infarction;

b) acute myocarditis;

c) acute decompensation of hypertrophied myocardium;

d) attack of paroxismal tachycardia;

e) the all above listed.

2. Choose the true statement:

a) in case of heart failure loading on heart exceeds its ability to make work adequate to it and that is accompanied by decrease in cardiac output below the needed one and also by the development of circulatoric hypoxia;

b) in case of heart failure volumetric speed decreases and linear speed of blood-flow increases that is accompanied by decrease in stroke output and perfuse pressure in arteriols increases.

3. Specify, whether heart failure can develop in fast renewal of coronary blood-flow in earlier occlused arteries:

a) yes;

b) no.

4. Specify, whether the concepts «cardiac asthma» and «heart failure» are identical:

a) yes;

b) no.

5. Specify the type of heart failure which will not lead to the development of oedema of lungs:

a) right ventricular;

b) left ventricular;

c) total.

6. Choose the true statement:

a) hypertrophy with myogenic dilation of heart is usually characterized by increase in cardiac output;

b) hypertrophy with myogenic dilation of heart is characterized by increase in contractile ability of myocardium;

c) dilation hypertrophy develops as a result of increase in systolic volume of blood and increase in final diastolic pressure in cavities of heart.

7. Specify the major factor of risk of myocardial infarction:

- a) hypothyroidism;
- b) hypotonic illness;
- c) fatty exhaustion;
- d) atherosclerosis.

8. Specify noncoronarogenic necrosis (infarction) of myocardium:

- a) electrolite-steroid;
- b) in case of thrombosis of coronal vessels;
- c) in case of embolia of coronal vessels.

9. The following changes testify to insufficiency of the left heart with the greatest probability:

- a) systemic arterial pressure;
- b) central venous pressure;
- c) pressure in capillaries of the pulmonary artery;
- d) pulse pressure.

10. Suppression of contractile ability of the left ventricle as a result of ischemia or necrosis of myocardium is always accompanied by:

- a) increase in final diastolic volume of the left ventricle;
- b) reduction of final diastolic volume of the left ventricle;
- c) increase in frequency of heart contractions;
- d) increase in blood pressure.

11. Specify urgent mechanisms of compensation of hemodynamic abnormalities in case of heart failure:

- a) tachycardia;
- b) homeometric mechanism;
- c) homeometric mechanism of Franc-Starling;
- d) the all above listed.

12. Specify the substance most strongly dialating coronary vessels in ischemia of myocardium:

- a) lactic acid;
- b) histomine;
- c) bradykinin;
- d) adenosine.
- e) CO₂.

13. Tachycardia in case of heart failure occurs as a result of:

- a) a raised blood supply of lungs;
- b) a lowered blood supply of lungs;
- c) Bainbridge reflex.

14. Specify the type of heart failure on the mechanism of its occurrence:

- a) systolic;
- b) diastolic;
- c) in case of an overload with pressure;
- d) compensated.

TYPICAL FORMS OF IMPAIRMENTS OF FUNCTIONS OF VESSELS

Specify a correct variant of the answer

1.Concept «arterial reaction» means:

a) temporary rise of blood pressure above the norm;

b) proof rise of blood pressure: systolic above 160 mm hg, diastolic – 95 mm hg.

2. Specify sizes of blood pressure in mm hg, testifying hypertension in people of the age from 20 till 60:

- a) 125/75;
- b) 135/85;
- c) 120/90;
- d) 90/60;
- e) 160/95.

3. Primary pathogenesis of arterial hypertension presumably includes the following part:

a) exhaustion of function of cortex of adrenal glands;

b) generalized hereditary defect of membrane of ionic pumps: Calcium and sodium-potassium;

c) genetically caused hypoproduction of mineralocorticoids.

4. Specify types of «symptomatic» arterial hypertensia:

a) thyreoid;

b) cerebro-ischemical;

c) renal;

d) reflexogenic;

e) the all above listed.

5. Specify differences of primary arterial hypertensia from other types of «symptomatic» arterial hypertensia:

a) increase of arterial pressure arises in case of absence of significant organic defeats of internal organs; b) occurs in case of primary abnormalities of function of kidneys and endocrine glands;

c) occurs in case of abnormalities of function of adrenal glands;

d) develops owing to primary damage of receptors of an arch of the aorta and carotid sinus zones.

6. Authors of neurogenic theory of pathogenesis of primary arterial hypertension are:

a) G. F. Lang and A. L. Miasnikov;

b) E. Gellgorn;

c) J. V. Postnov and S. N. Orlov;

d) A. C. Guyton.

7. Authors of membane concept of pathogenesis of primary hypertensia are:

a) G. F. Lang and A. L. Miasnikov;

b) E. Gellgorn;

c) J. V. Postnov and S. N. Orlov;

d) A. C. Guyton.

8. It does not enter the structure of antihypertensive system:

a) angiotensin II;

b) prostacyclin;

c) bradykinin;

d) NO;

e) Na-urethic hormone.

9. Portal hypertensia can arise owing to:

a) heart failure of the left ventricle;

b) imposings of portocaval anastomosis;

c) cirrhosis of liver;

d) hypovolemia.

10. Specify the mechanism of development of renovasular arterial hypertensia:

a) activation of the renin-angiotensin-aldosterone system;

b) insufficiency of prostaglandin and kinin systems of kidneys;

c) insufficiency of the renin-angiotensin-aldosterone system;

d) activation of prostaglandin and kinin systems of kidneys.

11. Specify the substance possessing of a direct vasopressor action:

a) angiotensin II;

b) vasopressin;

c) adrenaline;d) noradrenaline;e) the all above listed.

12. Specify the substance possessing a vasodilating effect:

- a) glucocorticoids;
- b) vasopressin;
- c) aldosterone;
- d) prostacyclin.

13. Endocrine hypertensia arises in case of:

- a) total hypofunction of a cortex layer of adrenal glands;
- b) hypofunction of medulla of adrenal glands;
- c) hypofunction of glomerular zones of cortex of adrenal glands;
- d) hypofunction of the thyroid gland;
- e) hypophysical cachexy;
- f) thyreotoxicosis.

TYPICAL FORMS OF IMPAIRMENTS OF CARDIAC RHYTHM

Specify a correct variant of the answer

1. In case of insufficiency of aortal valves the coronary blood-flow decreases:

- a) yes;
- b) no.

2. Specify pathogenetic factors of development of cardiac arrhithmias:

a) endocellular acidosis in cardiomyocytes;

- b) loss of ions of potassium by cardiomyocytes;
- c) deficiency of ATP in cells of myocardium;
- d) accumulation of Ca^{2+} in sarcoplasm and mitichondria of cardiomyocytes;
- e) the all above listed.

3. Specify types of arrhythmias which can be caused by the mechanism of repeated input of wave of excitation («re-entry»):

- a) paroxismal tachycardia of ventricles;
- b) atrial fibrillation;
- c) ventricular premature beats;
- d) the all above listed.

4. During atrial fibrillation the rhythm of ventricle excitation:

- a) is correct;
- b) is defined by pacemaker cells of atrioventricular node;
- c) is defined by ventricle ectopic centers of excitation;
- d) is defined by the impulses acting from atria.

5. We refer to sinus bradycardia:

- a) frequency of heart contractions above 90/min;
- b) speed of diastolic depolarization is raised;
- c) heterotopic arrhythmia;
- d) P wave is deformed as a rule;
- e) automatism of sinus node is lowered.

6. Sinus tachycardia is characterized by:

- a) frequency of heart contractions reaches 90-180/min;
- b) arises on physical activity;
- c) arises on rise in temperature of the body;
- d) arises in heart failure;
- e) the all above listed.

7. Paroxismal tachycardia of ventricles corresponds:

- a) heart rate increases up to 140-250/min;
- b) complex QRS is deformed and widened;
- c) to a sudden onset;
- d) the all above listed.

8. We refer to atrioventricular blockades the blockades caused by the impairments of impulses transmission on:

- a) transeferring system of atria;
- b) atrioventricular unit;
- c) the basic trunk of His's bundle;
- d) to all branches of His's bundle.

9. Specify the factor which can cause broadening and deformations of a ventricle complex (QRS):

- a) hypertrophy of one of the ventricles;
- b) blockade of brunches of His'bundle;
- c) migration of supraventricular pacemaker.

10. Atrioventricular blockade of the first degree is characterized by:

- a) from complex to complex ellongation lengthening of PQ interval;
- b) stable lengthening of PQ interval more than 0,20 sec;

c) periodic loss of ventricle complexes (QRS);

d) full dissociation of atrial and ventricle complexes.

11. Ventricular extrasystoles are characterized by the following electrocardiograms-signs:

a) shortening of R-R interval before extrasystola;

b) absence of P wave before extrasystolic QRS complex;

c) deformation and broadening of extrasystolic QRS complex;

d) full compensatory pause;

e) the all above listed.

12. In the basis of compensatory pauses after ventricular extrasystolia there is:

a) decrease in excitability of cells of sinus-atrial node;

b) increase in excitability of cells of sinus-atrial node;

c) arrival of the next impulse of excitation at ventricular myocardium in the phase of absolute refracterity;

d) positive dromotropic effect of premature beat.

13. Intra-atrial blockade is characterized by the following electrocardiograms-signs:

a) lengthening of PQ interval more than 0,20 sec;

b) deformation of QRS complex;

c) occurrence (-) of P wave;

d) increase in duration P wave more than 0,11 sec.

14. We don't refer to nonotopic arrhythmias :

a) sinus tachycardia;

- b) sinus bradycardia;
- c) sinus arrhythmia;

d) atrial Flutter.

15. Fibrillation of ventricles is a:

a) group ventricular premature beat;

b) full dissosiation of reductions of atria and ventricles;

c) chaotic contractions of separate groups of cardiomyocytes;

d) tachycardia with a rhythm of 250–300 per minute.

16. Choose the incorrect statement:

a) peroxide oxidation of lipids in the zone of ischemia of myocardium and infarction amplifies;

b) substrata of peroxide oxidation of lipids are nonsaturated fat acids of bimolecular lipid layer of membranes;

c) activation of peroxide oxidation of lipids promotes output of enzymes from lysosomes into cytosole;

d) peroxides and radicals formed in case of peroxide oxidation of lipids increase interface of oxidation and phosphoilation of ATP that promotes formation of microerges.

TYPICAL FORMS OF FRUSTRATION OF SYSTEM OF EXTERNAL RESPIRATION

Specify a correct variant of the answer

1. Specify change of, in comparison with the norm, ventilation-perfusion parameter, if minimal alveolar ventilation = 3 l/mines, and cardiac output = 5 l/mines

a) decreases;

b) increases;

c) does not change.

2. Specify, whether the decrease of excitability of the respiratory center leads to occurrence of Cheyne-Stokes respiration:

a) yes;

b) no.

3. Specify, whether Kussmaul's respiration is characteristic of diabetic coma:

a) yes;

b) no.

4. Specify change of parameters, characteristic of obstructive type of impairments of ventilation:

a) FEV₁ (forced expiratory volume) is decreased, Tiffeneau index is decreased;

b) the maximal volumetric speed of exhalation is lowered, frequency of inhalation is increased;

c) the all above listed.

5. Amplitude of respiration in case of Biot's respiration:

a) accrueing, then decreasing;

b) constant;

c) decreasing;

d) accrueing.

6. Specify the reason of impairments of passableness of the lower respiratory ways:

a) the presence of liquids in lumen of bronchioles;

b) compression of walls of larynx and trachea from the outside;

c) laryngospasm.

7. Specify the reason of reduction of perfusion of lungs:

- a) cardiovascular insufficiency;
- b) heart diseases;
- c) embolim of the pulmonary artery;
- d) the all above listed.

8. In the development of emphysema of lungs the following pathogenetic factor plays the role:

- a) increase of excitability of cholinergic receptors;
- b) sensitization of organism;
- c) earlier expirative closing of respiratory ways.

9. Specify conditions in case of which inspiratory dyspnea is observed:

- a) narrowing of lumen of thrachea;
- b) laryngeal oedema;
- c) I stage of asphyxia;
- d) compression of trachea by increased thyroid gland;
- e) the all above listed.

10. Minute alveolar ventilation in case of frequent superficial respiration:

- a) decreases;
- b) increases;
- c) does not change.

11. Specify changes of parameters, characteristic of obstructive, and restrictive types of impairments of ventilation:

a) frequency of respiration is increased;

- b) FEV (forced expiratory volume)₁ is reduced;
- c) the all above listed.

12. Specify the type of coma in diabetes, accompanied by Kussmaul's respiration:

- a) hypoglycemic;
- b) hyperosmium;
- c) ketoacidotic.

13. Amplitude of Cheyne-Stokes respiration:

- a) accrueing, then decreasing;
- b) constant;
- c) decreasing;
- d) accrueing.

14. Specify the reasons of impairment of patency of the lower airways:

a) laryngospasm, thickening of larynx and trachea;

b) the presence of liquids in lumen of bronchial tubes, spasm of bronchiols, decrease of elastic properties of lungs.

15. Name the reasons of periodic respiration:

a) uraemia, inhibition of CNS during a sleep;

- b) hypoxia of brain, medicinal intoxications;
- c) the all above listed.

16. Specify the type of respiration corresponding to the periodic one:

a) Kussmaul's;

b) gasping-respiration;

c) Biot's.

17. Specify the type of respiration corresponding to the agonal one:

a) Cheyne-Stoke's;

b) Biot's;

c) gasping-respiration;

d) wavy.

18. Specify conditions in case of which expiratory dyspnea is observed:

a) narrowing of lumen of trachea, laryngeal oedema;

b) emphysema of lungs, attack of bronchial asthma;

c) compression of trachea by increased thyroid gland.

19. Impairments of ventilation of lung of restrictive type develop in case of:

a) emphysema, chronic bronchitis, bronchial asthma;

b) intercostal myositis, bilateral closed pneumothorax, pneumonias, dry pleurisy, atelectases of lungs.

20. Specify, whether hypocapnia will result in decrease of pH of blood:

a) yes;

b) no.

21. Minute alveolar ventilation in case of rare superficial respiration:

a) decreases;

b) increases;

c) does not change.

22. The following abnormality is not characteristic of respiratory insufficiency:

a) dyspnea;

b) tachycardia;

c) anemia;

d) cyanosis;

e) hypoxia.

23. Specify, to which type of respiration we refer frequent respiration:

a) bradypnea;

b) tachypnea;

c) gasping-respiration;

d) apnea.

24. Specify reasons of impairments of patency of the lower airways:

a) afflux of liquid in lumen of bronchioles;

b) thickening of mucous of bronchioles;

c) spasm of bronchioles;

d) decrease of elastic properties of lungs;

e) the all above listed.

25. Character of deficiency of surfactant in case of respiratory distresssyndrome of adults is:

a) primary;

b) secondary.

26. Specify, whether the phenomenon of expiratory compressions in norm develops in case of the forced exhalation:

a) yes;

b) no.

27. Minute alveolar ventilation in case of frequent deep respiration:

a) decreases;

b) increases;

c) does not change.

28. The following abnormality is characteristic of crupous pneumonia:

a) frequent deep respiration (hyperpnea);

b) deep rare respiration;

c) Biot's respiration;

d) frequent superficial respiration (polypnea);

e) Kussmaul's respiration.

29. Specify changes of parameters, characteristic of restrictive type of impairments of ventilation:

a) FEV (forced expiratory volume)₁ is reduced;

b) Tiffeneau index is not changed;

c) frequency of respiration is increased;

d) the all above listed.

30. The following abnormality is characteristic of respiratory insufficiency:

a) dyspnea;

- b) change of C₂ pressure and CO in blood;
- c) changes of parameters of ventilation of lungs;
- d) changes of acid-base condition;
- e) the all above listed.

31. Specify changes, in comparison with the norm, of ventilation-perfusion parameter, if MAV = 8 l/min, and cardiac output = 9 l/min:

a) increases;

b) decreases;

c) does not change.

32. Amplitude of respiration in case of gasping respiration is:

a) accrueing, then decreasing;

b) constant;

c) decreasing;

d) accrueing.

33. Specify the reasons of impairments of patency of the lower airways:

a) laryngospasm;

- b) compression of larynx and trachea from the outside;
- c) thickening of larynx walls and trachea;
- d) thickening of mucous of bronchiols.

34. Specify the factor defining adequacy of pulmonary loading to the level of alveolar ventilation:

- a) pulmonary vascular resistance;
- b) volume of circulating blood;
- c) intraalveolar pressure of air;
- d) effective work of left and right ventricles of the heart;
- e) the all above listed.

35. Specify the type of oedema of lungs in respiratory distress-syndrome of adults:

a) cardiogenic;

b) noncardiogenic.

36. Specify possible parameters of Tiffeneau index in healthy people:

- a) 40–50 %;
- b) 50–60 %;
- c) 80–90 %.

37. Specify diseases in which impairments of ventilation of lungs, as a rule, develop on obstructive type:

a) crupous pneumonia, pleurisy;

b) chronic obstructive bronchitis, bronchial asthma;

c) atelectases of lungs.

38. An initial and leading part in pathogenesis of respiratory disrtesssyndrome of adults is:

a) pulmonary arterial hypertensia;

b) hypostasis of lungs;

- c) impairments of diffusion of gases;
- d) reduction of quantity of surfactant;
- e) increase of permeability of vessels of lungs for protein.

39. Occurrence of Kussmaul's respiration in the sick with the greatest probability testifies about the development of:

- a) respiratory alkalosis;
- b) metabolic alkalosis;
- c) respiratory acidosis;

d) metabolic acidosis.

40. Specify what type of respiration rare respiration is referred to:

a) apnea;

b) bradypnea;

c) tachypnea.

41. Specify the reason of impairment of patency of upper airways:

- a) afflux of liquid in gleam of bronchioles;
- b) thickening of mucous of bronchioles;

c) laryngospasm;

d) decrease of elastic properties of lungs.

42. Specify the factor causing discrepancy between ventilation and perfusion of lungs in physiological conditions:

a) anatomic and biophysical heterogeneity of pulmonary units;

b) local distinctions of transpulmonal pressure, tone of bronchial tubes and vessels;

- c) gravitation;
- d) all above listed.

43. Specify possible parameters of Tiffeneau index in patients with emphysema of lungs:

a) 40–60 %;

- b) 80–90 %;
- c) 90–100 %.

44. Specify disease of lungs in which impairments of ventilation, as a rule, develop on obstructively-restrictive (mixed) type:

a) pleurisy;

b) atelectases of lungs;

c) bronchial asthma;

d) emphysema of lungs.

45. Specify changes of parameters, characteristic of obstructive type of impairment of ventilation:

a) decrease in MPV, streaming parameters;

b) decrease in static volumes.

46. Early expiratory closing of respiratory ways arises in case when during the moment of an exhalation... (to continue):

a) resistance to an air stream increases;

b) axial pressure of an air stream in bronchiole increases;

c) transpulmonal pressure increases;

d) radial pressure of air stream in bronchiole decreases;

e) all above listed.

47. We refer to terminal types of respiration:

a) oligopnea, bradypnea, polypnea;

b) apnoic respiration, Kussmaul's respiration, gasping-respiration.

48. Specify the disease accompanied by inspiratory dyspnea:

- a) bronchial asthma;
- b) emphysema of lungs;

c) asphyxia(I stage).

49. The parameter of ventilation-perfusion attitudes in norm is equal:

- a) 0,5–0,7;
- b) 0,8–1,0;
- c) 1,3–1,5.

50. Specify blood cells for which there are special traps (catch trap) in lungs:

a) reticulocytes;

b) thrombocytes;

- c) polymorph-nuclear leukocytes;
- d) erythrocytes.

51. Specify possible parameters of Tiffeneau index in patients with atelectase of lungs:

- a) 40–50 %;
- b) 50-60 %;
- c) 80–90 %;

52.In stenosis of throat the following abnormality develops:

a) frequent superficial respiration(polypnea);

b) frequent deep respiration (hyperpnea);

c) rare deep inhalation with the complicated exhalation;

d) rare deep respiration with the complicated breath;

e) Biot's respiration.

53. Specify changes of parameters, characteristic of obstructive type of impairment of ventilation:

a) MPV is reduced;

b) MVI is increased;

c) the reserve of respiration is lowered;

d) the all above listed.

54. Specify possible consequences of early expiratoric close of airways:

a) increase in alveolar dead space;

b) reduction of MAV;

c) lymphostasis;

d) hypoxia;

e) the all above listed.

55. In downturn of excitability of the respiratory center the following abnormalities can develop:

a) Cheyne-Stoke's respiration, oligopnea, Biot's respiration;

b) Kussmaul's respiration, polypnea, hyperpnea;

c) polypnea, hyperpnea.

56. Specify the reasons of abnormalities of patency of the top airways:

a) laryngospasm, foreign bodies, compression of larynx and trachea from the outside;

b) afflux of liquids in lumen of bronchial tubes, thickening of mucous of bronchioles;

c) spasm of bronchioles, decrease of elastic properties of lungs.

57. Name types of dyspnea:

a) expiratory, inspiratory;

b) hyperpnea, pneumotonic;

c) terminal respiration, periodic respiration.

58. The basic function of bronchial arteries is:

a) trophic;

b) gas exchange.

59. Specify, whether reduction of Tiffeneau index is characteristic of the respiratory insufficiency developing on restrictive type:

a) yes;

b) no.

60. Specify change of, in comparison with norm, ventilation-perfusion parameter, if MAV = 8 l/min, and cardiac output = 3 l/min:

a) will increase;

b) will decrease;

c) will not change.

61. Specify the factor, which is an initial and leading part in pathogenesis of respiratory distress-syndrome of newborns:

a) pulmonary arterial hypertensia;

b) oedema of lungs;

c) reduction of quantity of surfactant;

d) increase of permeability of vessels of lungs for protein;

e) impairments of diffusion of gases.

62. Specify type of respiration which develops in premature newborn in impairments of synergism in work of respiratory muscles:

a) Cheyne-Stoke's respiration;

b) gasping-respiration;

c) apneac respiration;

d) dissociated respiration;

e) wavy respiration.

63. Specify receptors with which irritation of which the occurence of dyspnea can be connected:

a) central and peripheral chemo-and baroreceptor;

b) receptors of fall of alveoli;

c) juxstacapillary receptors;

d) irrhitant receptors of the top airways;

e) receptors of muscular spindles of respiratory muscles;

f) the all above listed.

64. Name types of insufficiency of respiration depending on mechanisms of impairments of ventilation:

a) ventilating, diffuse;

b) obstructive, regulatoric;

c) obstructive, restrictive;

d) diffuse, perfuse.

65. Specify the most probable reasons of bradypnea:

- a) downturn of excitability of the respiratory center;
- b) hyperoxia;
- c) increase of arterial pressure;
- d) all above listed.

66. Specify the type of respiration which will develop in animal after bilateral vagotomy at a level of the neck:

- a) frequent superficial;
- b) rare deep;
- c) frequent deep;
- d) rare superficial.

67. Specify the type of pathology in case of which impairments of lungs perfusion plays a role in respiratory insufficiency:

- a) left heart failure;
- b) blood loss;
- c) thrombembolism of the pulmonary artery;
- d) the all above listed.

68. Name pathogenetic forms of impairments of ventilation of lungs:

a) ventilating, diffuse, perfuse, mixed;

- b) obstructive, restrictive, regulatoric, mixed;
- c) obstructive, perfuse, mixed.

69. Name the reason of periodic respiratrion:

- a) hypoxia of brain;
- b) uraemia;
- c) medicinal intoxications;
- d) all above listed.

70. MAV in case of polypnea:

- a) increases;
- b) decreases;
- c) does not change.

71. Inspiratory dyspnea is observed in the following pathological conditions:

- a) I stage of asphyxia;
- b) oedema of larynx;
- c) stenosis of trachea;
- d) all above listed.

72. Specify the possible reasons of development of respiratory insufficiency of a restrictive type mainly:

- a) diffuse fibrousing alveolitis;
- b) extensive pneumonia;
- c) atelectase of lung;
- d) pneumofibrosis;
- e) the all above listed.

73. In case of inspiratory dyspnea:

- a) the exhalation is complicated and extended;
- b) the inhalation and exhalation is complicated;
- c) amplitude is constant;
- d) the inhalation is complicated and extended.

74. Specify the type of pathology which can be accompanied by the development of alveolar hyperventilation:

- a) exudative pleurisy;
- b) silicosis;
- c) bronchial asthma;
- d) tumour of a lung;
- e) overheating.

75. Specify the pathogenetic factors corresponding to Kussmaul's respiration:

- a) deenergizing of the pneumotoxic center;
- b) oppression of the center of inhalation;
- c) oppression of the centers of inhalation and exhalation;
- d) prevalence of the center of exhalation;
- e) excitation of the apneatic center.

76. Specify the most probable reasons of bradypnea:

- a) decrease in excitability of the respiratory center;
- b) compensated alkalosis;
- c) increase of arterial pressure;
- d) hyperoxia;
- e) the all above listed.

77. MAV in case of hyperpnea:

- a) increases;
- b) decreases;
- c) does not change.

78. Expiratory dyspnea is observed in the following pathological conditions:

a) emphysema of lungs;

b) attack of bronchial asthma;

c) the all above listed.

79. Impairment of diffuse properties of alvelocapillary membranes plays the basic role in the development of respiratory insufficiency in case of:

- a) impairment of synthesis of surfactant;
- b) bronchial asthma, laryngeal oedema;
- c) interstitial oedema of lung, silicosis.

80. The character of respiration in case of expiratory dyspnea is characterized by:

a) difficulty and lengthening of inhalation;

- b) difficulty and lengthening of exhalation;
- c) difficulty of inhalation and exhalation.

81. We refer to metabolic functions of lungs:

- a) transformation of angiotensin I into angiotensin II;
- b) inactivation of prostaglandins E and F;
- c) synthesis of alkaloid peptides;
- d) inactivation of bradykinins;
- e) inactivation of noradrenaline;

f) the all above listed.

82. Specify types of pathology which can be accompanied by the development of alveolar hypertensia:

- a) overheating, hysteria, blood loss;
- b) tumour of a lung, silicosis;
- c) exudative pleurisy, bronchial asthma.

83. MAV in case of oligopnea:

- a) increases;
- b) decreases;
- c) does not change.

84. It develops in base of inhibitory influence on vagus inspirative neurons and pneumotoxic center:

a) apneatic breath;

- b) Cheyne-Stoke's respiration;
- c) gasping respiration;
- d) Biot's respiration;
- e) Kussmaul's respiration;
- f) alternating breath.

85. In the basis of alveolar hyperventilation, arising on frequent and superficial respiration there is:

a) increase in resistance of air-conducting ways;

b) impairments of diffuse properties of alveolocapillary membranes;

c) increase in functional dead space.

86. Specify the pathogenetic factor, corresponding to apneatic respiration:

a) prevalence of the center of exhalation, excitation of gasping-center;

b) deenergizing of the centers of inhalation and exhalation;

c) oppression of the center of inhalation;

d) deenergizing of the pneumotoxic center;

e) excitation of the apneatic center.

87. Specify the most probable reasons of tachypnea:

a) decrease of excitability of the respiratory center, hyperoxia, alkalosis;

b) hypoxia, increase of excitability of the respiratory center, hyperoxia, compensated acidosis;

c) increase of arterial pressure, compensated alkalosis.

88. In pathogenesis of stenotic respiration the leaging role is playsed by:

a) decrease of excitability of the respiratory center;

b) increase of excitability of the respiratory center;

c) acceleration of Hering-Breuer reflex;

d) delay of Hering-Breuer.

89. Tiffeneau index in case of emphysema of lungs:

- a) increases;
- b) decreases;

c) does not change.

90. Alveolar hyperventilation can cause:

a) oedema of oblong brain;

b) obstructive defeats of airways;

c) restrictive defeats of lungs;

d) impairments of respiratory innervations of muscles;

e) the all above listed.

91. Specify the substances causing increase of vascular resistance in lungs:

a) serotonin, acetylcholin;

b) noradrenaline, prostocyclin.

92. Impairments of lungs perfusion plays the basic role in respiratory insufficiency in case of:

a) bronchial asthma;

b) myasthenia;

c) tuberculosis of lung;

d) left heart failure.

93. Specify the type of respiration corresponding to periodic:

- a) gasping respiration;
- b) Kussmaul's respiration;
- c) apneatic respiration;
- d) wavy respiration.

TYPICAL FORMS OF PATHOLOGY OF GASTROINTESTINAL TRACT

Specify a correct variant of the answer

1. In case of simultaneous increase of secretion and acidity of gastric juice the evacuation of food mass from the stomach:

- a) will be slowed down;
- b) will be accelerated.

2. Iatrogenic «steroid» ulcers of gastrointestinal tract are caused by:

- a) insulin;
- b) adrenaline;
- c) mineralocorticoids;
- d) glucocorticoids;
- e) sex hormones.

3. Specify factors of pathogenesis of «aspirin» stomach ulcers:

- a) reduction of synthesis of E group prostaglandins;
- b) reduction of formation of mucous;
- c) increase in return diffusion of H + in the mucous of the stomach;
- d) the all above listed.

4.It has the greater value, as a rule, in pathogenesis of duodenum:

- a) acid-peptic aggression;
- b) decrease in protective properties of the mucous membrane of the intestine.

5. Absence of enzymes and hydrochloric acid in gastric juice is called as:

- a) achlorhydria;
- b) acholia;
- c) achylia.

6. Specify, what combinations of types of secretion and types of acidity of gastric juice occur more often:

a) hyposecretion with the lowered acidity;

b) hyposecretion with the raised acidity;

c) hypersecretion with the lowered acidity.

7. Occurrence of steatorrhea can cause:

a) insufficiency of digestion and intestinal absorption of carbohydrates;

b) insufficiency of synthesis of pancreatic and intestinal lipases;

c) insufficiency of synthesis of tripsinogen in pancreas.

8. Specify, how activity of pepsin varies in case of hypoacid condition:

a) decreases;

b) increases;

c) does not change.

9. In case of hyperproduction of glucocorticoids:

a) secretion of pepsin increases, secretion of hydrochloric acid and mucous is opressed;

b) secretion of pepsin decreases , secretion of hydrochloric acid and mucous increases;

c) secretion of pepsin and hydrochloric acid decreases, production of mucous increases;

d) secretion of pepsin and hydrochloric acid increases and production of mucous is also opressed.

10. Specify the possible reasons of decrease of secretoric activity of pancreas:

a) strengthening of parasympathetic stimulation of the gland;

b) easing of parasympathetic stimulation of the gland;

c) increase of development and allocation of cholycystokinin;

d) increase of development and allocation of secretin.

11. Specify consequences of the discontinuance or a sharp reduction of receipt of bile into the intestines:

a) easing of motility of the intestines;

b) reduction of absorption of vitamins A, D, E, K;

c) strengthening of rotting of proteins in the intestines;

d) the all above listed.

12. Development of pancreatic collapse is connected with:

a) exessive production of pancreatic enzymes;

b) insufficient production of pancreatic enzymes;

c) activation of the kallikrein-kinin systems;

d) regurgitation of pancreatic enzymes in the stomach in case of duode-nogastric reflux.

13. Alcohol strengthens gastric secretion owing to:

- a) local irritating action;
- b) activation of sympathetic nervous system;
- c) braking of mucous carbanhydrase.

PATHOLOGICAL PHYSIOLOGY OF HEPATOBILIARY SYSTEM

Specify a correct variant of the answer

1. One of the way of prevention of coma development in hepatic insufficiency is restriction in diet of:

- a) carbohydrates;
- b) fats;
- c) proteins;
- d) liquids;
- e) salts.

2. Name the processes describing the increase of ketone bodies in blood:

- a) starvation;
- b) diabetes insipidus;
- c) insufficiency of the liver.

3. Specify the factor which gives dark color to urine in case of under renal jaundice:

- a) conjugated bilirubin;
- b) nonconjugated bilirubin;
- c) urobilin;
- d) stercobilin.

4. Specify the factor which gives dark color to urine in case of hemolytic jaundice:

- a) conjugated bilirubin;
- b) nonconjugated bilirubin;
- c) urobilin.

5. Urobilinuria can arise in case of:

- a) hepatocellular jaundice (I stage);
- b) mechanical jaundice;
- c) not in one case of the listed.

6. Indirect bilirubin in urine can appear in case of:

- a) hemolytic jaundice;
- b) hepatocellular jaundice;
- c) mechanical jaundice;
- d) not in one case of the listed.

7. Occurrence of hepatic transaminases in blood is characteristic of:

- a) hepatocellularcellular jaundice;
- b) hemolytic jaundices;
- c) enzyme jaundices;
- d) for any type.

8. Occurrence of sreatorrhea and plenty of muscular proteins in fecal mass after reception of meat and fat food can testify:

- a) about pancreatic achylia;
- b) about hypersecretion of gastric juice;
- c) about absence of gastric juice.

9. In clinically expressed stage of hepatocellular jaundice urobilinogen disappears in blood and urine , because:

- a) capture and destruction of urobilinogen by hepatocytes is normalized;
- b) allocation of bilirubin in intestines is broken;
- c) urobilinogen absorption in intestines worsens.

TYPICAL FORMS OF IMPAIRMENTS OF FUNCTIONS OF KIDNEYS

Specify a correct variant of the answer

1. Impairments of ultrafiltration in kidneys can testify about:

- a) glucosuria;
- b) aminoaciduria;
- c) proteinuria;
- d) urobiliuria.

2. The reason promoting decrease of glomerular filtration, is:

- a) decrease in systemic arterial pressure;
- b) spasm of afferent glomerular arteriole.

3. Specify change of facultative water reabsorption in renal tubules, arising right after acute massive blood loss:

- a) increase;
- b) reduction;
- c) normal level.

4. Polyuria can cause lack of:

- a) somatotropin;
- b) adrenaline;
- c) oxytocuin;
- d) anti-diuretic hormone.

5. Specify impairments of diuresis, characteristic of pollakiuria:

- a) monotonous diuresis with density of urine 1,010;
- b) monotonous diuresis with density of urine 1,012–1,006;
- c) increase in daily quantity of urine;
- d) speeded up (over 6 times a day) urination;
- e) reduction of daily quantity of urine;
- f) the termination of urination (< 50 ml/day).

6. To the group of metabolic nephropathias we refer:

- a) polycystic degeneration of a kidney;
- b) pyelonephritis;
- c) urolithic disease;
- d) nephropathia in pregnant women;
- e) tuberculosis of a kidney.

7. To the group of immune nephropathias we refer:

- a) glomerulonephritis;
- b) polycystic degeneration of a kidney;
- c) urolithic disease;
- d) pyelonephritises.

8. Choose typical complications of glomerulonephritis, menacing a patient's life:

- a) massive proteinuria;
- b) acute hepatic failure.

9. Specify impairments of diuresis, characteristic of poliguria:

- a) monotonous diuresis with density of urine 1,010;
- b) monotonous diuresis with density of urine 1,012–1,006;
- c) increase in daily quantity of urine;
- d) speeded up (over 6 times day)urination;

e) reduction of daily quantity of urine;

f) the termination of urination (< 50 ml/day).

10. Specify impairments of diuresis, characteristic of anuria:

- a) monotonous diuresis with density of urine 1,010;
- b) monotonous diuresis with density of urine 1,012–1,006;
- c) increase in daily quantity of urine;
- d) speeded up (over 6 times day)urination;
- e) reduction of daily quantity of urine;
- f) the termination of urination (< 50 ml/day).

11. Specify impairments of diuresis, characteristic of hypoproteinura:

a) the termination of urination (< 50 ml/day);

b)monotonous diuresis with density of urine 1,012–1,006;

c) increase in daily quantity of urine;

- d) speeded up (over 6 times day)urination;
- e) reduction of daily quantity of urine.

12. Specify impairments of diuresis, characteristic of polyuria:

- a) monotonous diuresis with density of urine 1,010;
- b) monotonous diuresis with density of urine 1,012–1,006;
- c) increase in daily quantity of urine;
- d) speeded up (over 6 times day)urination;
- e) reduction of daily quantity of urine;
- f) the termination of urination (< 50 ml/day).

13. Specify impairments of diuresis, characteristic of isosthenuria:

- a) monotonous diuresis with density of urine 1,010;
- b) the termination of urination (< 50 ml/day);
- c) increase in daily quantity of urine;
- d) speeded up (over 6 times day)urination;
- e) reduction of daily quantity of urine.

14. Anuria can be caused by:

- a) heavy mental trauma;
- b) significant painful irritations;
- c) excess or compression ureters;
- d) reduction of systemic arterial pressure up to 50 mm hg;
- e) all above listed.

15. Osmolarity of plasma of blood is considered to be increased if it exceeds:

- a) 100 mOsm/kg;
- b) 285 mOsm/kg;
- c) 80 mOsm/kg;
- d) 50 mOsm/kg.

PATHOLOGICAL PHYSIOLOGY OF ENDOCRINE PATHOLOGY

Specify a correct variant of the answer

1. Specify the most probable change of sensitivity of cells-targets to hormones in case of long increase of their level in blood:

- a) increase;
- b) decrease;
- c) absence of changes.

2. In case of acromegalia we observe:

- a) increase of tolerance to carbohydrates;
- b) increase of sensitivity to insulin;
- c) all above listed.

3. Specify, how generation of hormones changes in case of acromegalia:

- a) synthesis of somatotropin is increased;
- b) synthesis of somatotropin is lowered;
- c) synthesis of thyrotroic is lowered.

4. Specify, how generation of hormones changes in case of giantism:

- a) synthesis of somatotropin is increased;
- b) synthesis of somatotropin is lowered;
- c) synthesis of thyrotroic is lowered.

5. Manifestations of panhypopituitarism are not:

- a) hypothyroidism;
- b) hypogonadism;
- c) hypercorticism;
- d) Cachexia.

6. The reason of primary aldosteronism (Conn's syndrome) is:

- a) tumour of the medulla of adrenal glands;
- b) tumour of reticular zone of the cortex of adrenal glands;
- c) increase of secretion of aldosterone under influence of angiotensin;
- d) tumour of zona fasciculata of the cortex of adrenal glands;
- e) tumour of glomerular zone of the cortex of adrenal glands.

7. The reason of secondary aldosteronism is:

- a) tumour of the medulla of adrenal glands;
- b) tumour of reticular zone of the cortex of adrenal glands;
- c) increase of secretion of aldosterone under influence of angiotensin;

d) tumour of zona fasciculata of the cortex of adrenal glands;

e) tumour of glomerular zone of the cortex of adrenal glands.

8. Chronic adrenocortical insufficiency can be a result of:

- a) lowered production of adenocorticotropic hormone by adenohypophysis;
- b) autoimmune defeat of the cortex layer of adrenal glands;
- c) presence of antibodies to receptors of adrenocorticotropic hormone;
- d) long reception of glucocorticoid preparations;
- e) all above listed.

9. In case of defeat of the cortex of adrenal glands the following syndromes and diseases can develop:

a) Conn's syndrome;

- b) Addison's syndrome;
- c) Itsenko-Cushing syndrome;
- d) adrenogenital syndrome;
- e) all above listed.

10. Specify the condition in which production of adrenocorticotropic hormone by hypophysis is increased:

a) Itsenko-Cushing disease;

b) Itsenko-Cushing syndrome;

c) tumours of the cortex of adrenal glands.

11. Specify the basic manifestations of hypothyreosis:

a) rise in temperature of the body;

- b) strengthening of catabolism of proteins and fats;
- c) hyperglycemia;

d) all above listed.

12. The following abnormality is chaqracteristic of expressed hypothyreosis of adults:

a) adiposity;

- b) bradycardia;
- c) dryness of skin;
- d) all above listed.

13. Tetany can develop in case of:

- a) normocalcemia;
- b) hypercalcemia;
- c) hypocalcemia;
- d) decrease of pH of blood.

14. Exophtalmus is a characteristic sign of:

- a) hypothyreosis;
- b) hypogonadism;
- c) diabetes insipidus;
- d) hypercortisolism;
- e) hyperthyreosis.

15. Hypoparathyreosis arises in case of:

- a) mistakes in case of strumectomia;
- b) panhypopituitarism;
- c) chronic renal insufficiency;
- d) excessive secretion of calcitonin;
- e) Itsenko-Cushing syndrome (hypercortisolism).

16. In case of hyperparathyreosis the most typical abnormalitry is:

- a) decrease in the maintenance of potassium in plasma of blood;
- b) increase of the maintenance of calcium in plasma of blood;
- c) increase of the maintenance of sodium in plasma of blood.

17. Hypoparathyreosis is characterized by:

- a) osteoporosis;
- b) decrease in sensitivity of renal tubules to antidiuretic hormone;
- c) polyuria;
- d) development of peptic ulcers of duodenum;
- e) all above listed.

18. Hypofunction of the thyroid gland at children's age can be shown as:

- a) delay of intellectual development;
- b) easing of muscular tone;
- c) easing of immunity;
- d) backlog in growth;
- e) the all above listed.

19. Changes of the cardiovascular system in thyreotoxicosis are characterized by:

a) bradycardia;

- b) increase in systolic and decrease of diastolic pressure;
- c) decrease in systolic and increase in diastolic pressure.

20. The diabetes of the $2^{\underline{d}}$ type (insulin nondependent) is characterized by:

- a) occurrence of the disease at average and advanced age;
- b) the raised propensity to ketoacidosis;
- c) significant decrease in the level of insulin or its full absence in blood;
- d) presence of antibodies to β -cells of islets of Langerhans.

TYPICAL FORMS OF IMPAIRMENTS OF THE NERVOUS SYSTEM. PAIN

Specify a correct variant of the answer

1. The most frequent reason of hemiparesis in person is:

a) damage of the cortex of the brain;

- b) haemorrhage into internal capsule;
- c) damage of pyramidal tract on the level of medulla oblongata;
- d) damage of pyramidal tract on the level of the spinal cord.

2. Mediators of the antinociceptive system are:

- a) meth-enkephalin;
- b) leu-enkephalin;
- c) endorphine;
- d) dinorphine;
- e) all above listed.

3. A triad of symptoms: muscular tremor at rest, strengthening of muscular tone (rigidity), difficulties on performance of any movements – is characteristic of:

- a) Parkinson's disease;
- b) Alzheimer's disease;
- c) epilepsy;
- d) damages of cerebellum;
- e) damages of motor cortex of the brain.

4. Development of it can be pathogenetically directly connected with neurosis:

- a) Itsenko-Cushing disease;
- b) diffuse glomerulonephritis;
- c) hepatitis;
- d) stomach ulcer.

5. It is characteristic of denervative syndrome:

- a) presence of trophic impairments in the zone of innervation;
- b) change in synaptic apparatus of denervated structures;
- c) all above listed.

6. Expressed denervated syndrome develops as a result of:

a) dissociation of the central departments of vegetative nervous system and peripheral neurons;

b) partial decortication;

c) dissociation of the nervous system with organs and tissues;

d) dissociation of the cortex of neoencefalon with subcortial centers.

7. Specify, what clinical features correspond to spinal type of hyperkinesis:

- a) chorea;
- b) clonic convulsions;
- c) athetosis;
- d) tonic convulsions;
- e) tremor;
- f) fibrillation of muscles.

8. The positive effect of usage of L-DOPA in case of Parkinson's disease is caused by:

- a) restoration of nigrostriatal connections;
- b) restoration of nigrothalamic connections;
- c) restoration of striatal connections;
- d) restoration of thalamocortical connections.

9. Impairments of truncus cerebri are accompanied by:

- a) clonic convulsions;
- b) tonic convulsions;
- c) chorea;
- d) tremor;
- e) sensitive ataxy.

10. Impairments of fibres carrying deep sensitivity are accompanied by:

- a) clonic convulsions;
- b) tonic convulsions;
- c) chorea;
- d) tremor;
- e) sensitive ataxy.

11. Impairments of motor cortex of the brain are accompanied by:

- a) clonic convulsions;
- b) tonic convulsions;
- c) chorea;
- d) tremor;
- e) sensitive ataxy.

12. Specify the clinical features corresponding to extrapyramidal type of hyperkinesis:

- a) chorea;
- b) athetosis;
- c) tremor;
- d) all above listed.

13. The most frequent reason of monoparesises caused by destruction of motoneurons in the person is:

a) damage of the cortex of the brain;

b) haemorrhage into internal capsule;

- c) damage of pyramidal tract on the level of medulla oblongata;
- d) damage of pyramidal tract on the level of spinal cord.

14. Peripheral endings of nociceptive fibres are excited by:

a) strong mechanical stimuli;

b) heating of skin above 45 °C;

c) electric stimuli;

d) H^+ ions;

e) all above listed.

15. We refer to algesive agents:

a) ions of potassium;

b) serotonin;

c) bradykinin;

d) histamine;

e) all above listed.

16. Impairments of subcortial centers of motor analyzer are accompained by:

a) clonic convulsions;

- b) tonic convulsions;
- c) chorea;
- d) tremor;

e) sensitive ataxy.

17. Impairment of energy metabolim in nervous tissue can be connected with:

a) hypoxia of brain;

- b) overdose of insulin;
- c) deficiency of vitamin B₁;
- d) deficiency of nicotinic acid;
- e) all above listed.

18. The central type of paralysis is characterized by:

- a) loss of any movements;
- b) increase of muscular tone;
- c) increase of tendon reflexes;
- d) occurrence of pathological reflexes;
- e) all above listed.

19. What signs are characteristic of peripheral paralysis:

- a) hypothrophy of muscles;
- b) muscular hypotonia;
- c) hypo- and areflexy;
- d) all above listed.

20. Specify types of fibres of peripheral nerves which carry «painful impulsation»:

- a) fibres A-alpha;
- b) fibres A-beta;
- c) fibres A-delta.

21. We refer to hyperkinesia:

- a) clonic convulsions;
- b) paresis;
- c) triplegia;
- d) paralysis.

22. Specify properties of «physiological» pain:

- a) it is adequate to force and character of influence;
- b) provides mobilization of protective-adaptive reactions;
- c) stops in case of elimination of stimulus;
- d) all above listed.

23. Specify properties which correspond to pathological type of pain:

- a) it is inadequate to influence;
- b) disorganizes the organism;
- c) it is long;
- d) arises in case of absence of pathogenic stimulus;
- e) the all above listed.

24. «Physiological» as opposed to pathological pain is characterized by:

a) occurrence in case of damage, excessive irritation or destruction of nerves and/or receptors;

b) occurrence in case of damage or irritation of the thalamic zone of the nervous system;

c) decrease in resistency of the organism to pathogenic influences;

d) its continuous sensation;

e) the all above listed.

II STANDARDS OF ANSWERS TO TEST TASKS

INTRODUCTION. GENERAL DOCTRINE ABOUT DISEASE. HEREDITY AND PATHOLOGY

1-c	8-c	15-е	22-с	29-d	36-d	43-с
2 - b	9-b	16-c	23-ь	30-b	37-b	44-b
3-a	10-с	17 - c	24-е	31-a	38-с	45-d
4-c	11 - b	18-c	25-с	32-с	39-d	46-a
5-b	12 - a	19 - d	26-a	33-b	40-b	47-b
6-e	13-с	20-ь	27-с	34-c	41-a	48-b
7-b	14-b	21-а	28-с	35-е	42-a	49-b

ETIOLOGY AND PATHOGENESIS OF DAMAGE OF A CELL

1 - a	5-a	9-c	13 - a	17 - d	21-c	25-с
2-f	6-b	10-е	14 - a	18-c	33-b	26-d
3-b	7-d	11 - c	15-e	19 - b	23-а	
4-b	8-b	12-f	16 - a	20-с	24-b	

ABNORMALITIES OF MICROCIRCULATION

1-d	4-b	7-b	10 - d	13-е
2-c	5-d	8-b	11 - b	14-a
3-a	6-c	9-a	12 - a	15-a

LOCAL DISORDERS OF BLOOD CIRCULATION

1 - d	4-b	7-c	10 - d	13-b	16-b	19-b	22-b
2-a	5-d	8-a	11 - a	14 - a	17 - b	20-е	
3-a	6-a	9-a	12-е	15-b	18 - d	21-c	

ACUTE AND CHRONIC INFLAMMATION. ACUTE PHASE RESPONSE

1-c	4-c	7-a	10-a	13-d	16-b	19-b
2-b	5-f	8-b	11 - d	14 - a	17 - c	20-с
3-c	6-e	9-b	12-d	15-с	18 - d	21-d

IMPAIRMENTS OF THERMOREGULATION

1-c	4-a	7-c	10 - a	13-c	16-e	19-е
2-d	5-c	8-c	11 - b	14 - a	17 - b	20-е
3-b	6-b	9-a	12 - a	15-d	18-c	21-b

TYPICAL FORMS OF IMPAIRMENTS OF TISSUE GROWTH. TUMOURS

1-c	4-a	7-d	10 - c	13-c	16-b	19-с
2-c	5-d	8-c	11 - b	14-c	17 - a	20-ь
3-b	6-d	9-a	12 - b	15-d	18-c	21-а

PATHOLOGICAL PHYSIOLOGY OF THE ERYTHROCYTES SYSTEM

1-c	5-a	9-a	13-b	17-d	21-d	25-b	29-ь
2-b	6-b	10-b	14-e	18-a	22-d	26-с	30-ь
3-a	7-е	11 - a	15-a	19-a	23-d	27-с	31-с
4-b	8-c	12 - b	16 - a	20-а	24-b	28-b	32-f

PATHOLOGICAL PHYSIOLOGY OF THE SYSTEM OF LEUKOCYTES

1-d	4-b	7-c	10 - d	13-c
2-b	5-d	8-d	11 - b	
3-е	6-a	9-e	12-b	

PATHOLOGICAL PHYSIOLOGY OF LEUKEMIAS

1-e	4-d	7-b	10-c	13-d
2-b	5-d	8-a	11 - c	14-a
3-d	6-d	9-c	12-c	

TYPICAL FORMS OF IMPAIRMENTS OF THE SYSTEM OF HEMOSTASIS

1-c	5-a	9-e	13-a	17-d
2-b	6-a	10-с	14 - a	18-c
3-a	7-c	11 - c	15-b	19 - c
4-b	8-b	12 - a	16-c	20-ь

HYPOXIA

1-b	3-е	5-a	7-b
2-a	4-d	6-b	8-c

TYPICAL FORMS OF EXTREME CONDITIONS

1-e	4-b	7-b	10-c	13-d
2-b	5-a	8-b	11 - b	
3-a	6-c	9-b	12-b	

PATHOPHYSIOLOGY OF THE IMMUNE SYSTEM. ALLERGY. AUTOIMMUNE PROCESSES

1 - a	5-c	9-b	13-е	17 - b	21-е	25-b
2-a	6-b	10-d	14 - c	18-c	22-b	26-с
3-a	7-b	11 - a	15-d	19 - b	23-с	27-а
4-b	8-b	12-d	16 - a	20-d	24-е	

PATHOPHYSIOLOGY OF THE IMMUNE SYSTEM. IMMUNODEFICIENCT CONDITIONS

1 - d	12 - a	23-b	34-a	45-a	56-a	67-d	78-d
2-е	13 - b	24-b	35-b	46-b	57-b	68-a	79-а
3-d	14 - d	25-е	36-b	47-c	58-b	69-a	80-a
4-d	15-c	26-а	37-a	48-a	59-с	70-b	81-е
5-c	16 - a	27-b	38-b	49-a	60-c	71 - a	
6-d	17-d	28-d	39-с	50-b	61 - a	72 - b	
7-е	18-c	29-ь	40-b	51-a	62-с	73-b	
8-d	19-е	30-с	41 - a	52-a	63 - a	74-c	
9-c	20 - b	31-d	42-c	53-b	64-a	75-a	
10-d	21-с	32-е	43-b	54-a	65-d	76-d	
11 - f	22-с	33-с	44-е	55-с	66-a	77 - b	

IMPAIRMENTS OF ACID-BASE BALANCE

1 - b	5-d	9-a	13-b	17-d
2 - b	6-a	10 - a	14-c	18 - a
3-е	7-b	11 - d	15-a	19 - b
4-c	8-a	12-c	16-b	

PATHOPHYSIOLOGY OF TYPICAL IMPAIRMENTS OF EXCHANGE OF PROTEINS

1-d	3-a	5-d	7-е	9-a
2-е	4-c	6-c	8-b	10-е

PATHOPHYSIOLOGY OF IMPAIRMENTS OF LIPID EX-CHANGE. ATHEROSCLEROSIS

1 - a	5-g	9-a	13 - a
2-b	6-e	10 - b	14 - b
3-g	7-d	11 - b	15-a
4-e	8-b	12 - a	16-c

PATHOPHYSIOLOGY OF IMPAIRMENTS OF CARBOHYDRATE EXCHANGE. DIABETES

1 - a	4-b	7-b	10 - a	13-d
2-е	5-a	8-b	11-e	14-е
3-с	6-b	9-e	12 - f	15-b
		-		

IMPAIRMENTS pathogenetically

TYPICAL FORMS OF IMPAIRMENTS OF HEART FUNCTIONS

1-e	4-b	7-d	10 - a	13-с
2-a	5-a	8-a	11 - d	14-c
3-a	6-c	9-c	12 - d	

TYPICAL FORMS OF IMPAIRMENTS OF FUNCTIONS OF VESSELS

1 - a	4-е	7-c	10 - a	13-f
2-е	5-a	8-a	11-е	
3-b	6-a	9-c	12-d	

TYPICAL FORMS OF IMPAIRMENTS OF CARDIAC RHYTHM

1 - a	5-e	9-b	13-d
2-е	6-e	10 - b	14-d
3-d	7-d	11-е	15-c
4-d	8-b	12-c	16-d

TYPICAL FORMS OF FRUSTRATION OF SYSTEM OF EXTERNAL RESPIRATION

1 - a	13-a	25-b	37-b	49-b	61-c	73-d	85-c
2-a	14 - b	26-a	38-е	50-с	62-d	74-е	86-e
3-a	15-c	27-b	39-d	51-c	63-f	75-a	87-b
4-c	16-c	28-d	40-b	52-d	64-c	76-е	88-d
5-b	17 - c	29-d	41-c	53-d	65-d	77-a	89-b
6-a	18-b	30-е	42-d	54-e	66-b	78-с	90-е
7-d	19-b	31-c	43-a	55-a	67-d	79-с	91-a
8-c	20-b	32-с	44-d	56-a	68-b	80-b	92-d
9-e	21-a	33-d	45-a	57-a	69-d	81 - f	93-d
10 - a	22-с	34-е	46-e	58-a	70-b	82-a	
11 - c	23-ь	35-b	47-b	59-b	71-d	83-b	
12-c	24-е	36-с	48-c	60-a	72-е	84-a	

TYPICAL FORMS OF PATHOLOGY OF GASTROINTESTINAL TRACT

1 - a	4-a	7-b	10 - b	13-а
2-d	5-c	8-a	11 - d	
3-d	6-a	9-d	12-с	

PATHOLOGICAL PHYSIOLOGY OF HEPATOBILIARY SYSTEM

1-c	4-c	7-a
2-a	5-b	8-a
3-a	6-d	9-b

TYPICAL FORMS OF IMPAIRMENTS OF FUNCTIONS OF KIDNEYS

1-c	4-d	7-a	10-f	13-a
2-a	5-d	8-b	11 - b	14-е
3-a	6-c	9-e	12-c	15-b

PATHOLOGICAL PHYSIOLOGY OF ENDOCRINE PATHOLOGY

1 - b	5-c	9-е	13-с	17-е
2-a	6-e	10 - a	14-e	18-е
3-a	7-с	11 - d	15-a	19-b
4 - a	8-e	12-d	16-b	20-а

TYPICAL FORMS OF IMPAIRMENTS OF THE NERVOUS SYSTEM

1 - b	5-c	9-d	13-a	17-е	21-а
2-е	6-c	10-е	14-e	18-е	22-d
3-a	7-f	11 - a	15-e	19 - d	23-е
4-d	8-a	12-d	16-b	20-с	24-е

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CONTENTS

I EXAMPLES OF TEST TASKS	3
1. Introduction. General doctrine about disease. Heredity and pathology	3
2. Etiology and pathogenesis of damage of a cell	11
3. Abnormalities of microcirculation	15
4. Local disorders of blood circulation	18
5. Acute and chronic inflammation. Acute phase response	21
6. Impairments of thermoregulation	25
7. Typical forms of impairments of tissue growth. Tumours	28
8. Pathological physiology of the erythrocytes system	32
9. Pathological physiology of the system of leukocytes	37
10. Pathological physiology of leukemias	39
11. Typical forms of impairments of the system of hemostasis	42
12. Hypoxia	45
13. Typical forms of extreme conditions	46
14. Pathophysiology of the immune system. Allergy. Autoimmune processes	48
15. Pathophysiology of the immune system. Immunodeficient conditions	
16. Impairments of acid-base balance	66
17. Pathophysiology of typical impairments of exchange of proteins	69
18. Pathophysiology of impairments of lipid exchange. Atherosclerosis	70
19. Pathophysiology of impairments of carbohydrate exchange. Diabetes	73
20. Typical forms of impairments of functions of heart	76
21. Typical forms of impairments of functions of vessels	78
22. Typical forms of impairments of functions of cardiac rhythm	80
23. Typical forms of frustration of system of external respiration	83
24. Typical forms of pathology of gastrointestinal tract	96
25. Pathological physiology of hepatobiliary system	98
26. Typical forms of impairments of functions of kidneys	99
27. Pathological physiology of endocrine pathology	102
28. Typical forms of impairments of the nervous system. Pain	105
II STANDARDS of ANSWERS	109
LITERATURE	114

Учебное издание

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ТЕСТОВЫЕ ЗАДАНИЯ ПО ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ (на английском языке)

Учебно-методическое пособие для иностранных студентов, обучающихся на английском языке

Под редакцией Т. С. Угольник Перевод на англ. язык А. А. Шиханцова, О. А. Ермакова Редактор Т. Ф. Рулинская Компьютерная верстка А. М. Елисеева

Подписано в печать 18.05.2009 Формат 60×84¹/₁₆. Бумага офсетная 65 г/м². Гарнитура «Таймс» Усл. печ. л. 6,74. Уч.-изд. л. 7,37. Тираж 100 экз. Заказ № 129

Издатель и полиграфическое исполнение Учреждение образования «Гомельский государственный медицинский университет» 246000, г. Гомель, ул. Ланге, 5 ЛИ № 02330/0549419 от 08.04.2009