**PATHOLOGICAL PHYSIOLOGY TESTS**

**for training and knowledge control**

1) Nosology is …

A) the study about causes of desease origin

B) the study of the conditions of disease origin

C) the general study of the disease +

D) the study of the mechanisms of occurrence, development and outcomes of the disease

E) the study of recovery mechanisms

2. The “disease” is characterized by

 1. state of complete physical, mental and social well-being

 2. reduced work capacity +

 3. standard state

 4. “the third” state

 5. pre-illness development

3. Which of the following statements is incorrect?

 A) the pathological process is the basis of any disease

 B) the same pathological process can be caused by various factors

 C) the concepts of the pathological process and the disease are identical +

 D) the same pathological process may be a component of various diseases

4. The pathological condition is:

1. a condition intermediate between health and illness

2. a new qualitative state of the body

3. slowly developing pathological process+

4. the simplest form of the pathological process

5. lowering the body work capacity

5. The pathological condition is:

 1. the response of the body that occurs under the influence of damaging factors

 2. persistent deviation from the norm, not having adaptive value for the body+

 3. an unusual reaction of the body to environmental factors

 4. damage to organs and tissues by environmental factors

 5. a combination of damage phenomena and protective-adaptive reactions in damaged tissues, organs or the body +

1. Pathologicalreaction is:

 1. a short-term elementary unusual reaction of the body to the stimulus+

 2. a steady, slowly developing process or its consequences

 3. apainful change in function and structure

 4. a symptom of the disease

 5. a complication of the disease

7. Indicate examples of pathological conditions:

A) hypoglycemia due to the introduction of large doses of insulin

B) [cicatrical tissue changes](https://translate.academic.ru/cicatrical%20skin%20changes/ru/en/)+

C) atrophy of the alveolar processes in connection with tooth extractions+

D) spasm of cardiac arterioles with pulmonary thrombosis

E) acquired heart valve disease defect+

8.The appearance of nonspecific symptoms of the disease is characteristic of

* 1. the latent period
	2. prodromal period+
	3. incubation period
	4. the height of the disease
	5. the outcome of the disease.

10. For the occurrence of the disease

 1. the action of one reason is enough

2. the action of a set of conditions is necessary, which does not always include a reason

 3. genetic predisposition is necessary

 4. a reason, and conditions conducive to disease development is necessary

 5. the action of a set of conditions is necessary

11. The following statement is correct

 1. conditions of the disease determine the specific features of the disease

 2. specific symptoms of a disease are due to the interaction of the cause of the disease with the body+

 3. etiology is the study of causation

 4. the specificity of the disease is determined mainly by the changed body reactivity

 5. conditions of the disease are factors without which the disease does not occur

12. The cause of iatrogenic disease is

 1. infection

 2. incorrect actions of the doctor+

 3. improper patient behavior

 4. decreased body reactivity

 5. action of extremely strong pathogenic factors

13. The vicious circle in the pathogenesis of diseases is:

 A) transition of the initial acute phase into a chronic form with

periods of exacerbation and remission

 B) cyclical course of the disease, in which each new the cycle differs from the previous one in a progressive increase of severity of disorders

 C) positive feedback between individual links of pathogenesis contributing to the progression of the disease +

14. The leading or main link in pathogenesis is

1. a change that determines the development of the remaining stages of the disease +

2. a change that occurs under the influence of a pathogenic factor

3. disorder of oxygen transport and utilization systems

4. hemodynamic disorder

5. "vicious circle" in pathogenesis blood flow velocity.

15. An example of damage on a cellular level is:

A) The replacement of glutamic acid with Valine in the beta chain of globin in sickle cell anemia.

C) Immune thrombocytopenia. +

C) Violation of the biosynthesis of vasopressin in diabetes insipidus.

D) Inherited deficiency of coagulation factor VIII.

E) Stenosis of the atrioventricular orifice.

16. The main factor in the development of [hypobaropathy](https://translate.academic.ru/hypobaropathy/ru/en/)is

1. increased barometric pressure

2. reduced partial pressure of oxygen in the air +

3. ultraviolet radiation

4. infrared rays

5. low temperature

16. What changes in the body are not typical for the stage of compensation for [hypobaropathy](https://translate.academic.ru/hypobaropathy/ru/en/) ?

A) increase in heart rate

B) pulmonary hyperventilation

C) increase in the number of erythrocytes in the blood

D) increased production of erythropoietins by the kidneys

E) lowering blood pressure +

17. Only etiotropic disease prevention can be classified by

1. isolation of the patient +

2. immunization of the patient

3. hardening

4. healthy lifestyle

5. physiotherapy exercises

18. Body reactivity is

 A) the response of the body to the stimulus

 B) the property of the organism as a whole to respond with life changes to environmental influences +

 C) a protective body reaction to the action of a pathogenic irritant

 D) resistance to disease

 E) non-specific body resistance

19. Dysergia is

1. increased body response to stimulus

2. reduced body response to stimulus

3. lack of response to the stimulus

4. a perverse response to the stimulus +

5. an adequate response of the body to the stimulus

20. Chernorutsky’s classification of body types corresponds

1. normosthenic +

2. sanguine

3. athletic type

4. muscle type

5. Strong, balanced, mobile type.

21. I.P. Pavlov’ s classifications of body types corresponds

1. choleric

2. phlegmatic

3. melancholy

4. strong, balanced, mobile +

5. asthenic.

22. Hypersthenics are prone to

1. lowering blood pressure

2. anemia

3. lower blood glucose

4. increase blood cholesterol +

5. lower intestinal absorption capacity

23. Asthenic type predisposes to development

1. hypertension

2. coronary heart disease

3. cholelithiasis

4. peptic ulcer of the stomach and duodenum +

5. diabetes.

24. What statements are true?

A) reactivity depends on the body type +

B) reactivity depends on the state of the nervous and endocrine systems +

C) reactivity is independent of environmental factors

D) the reactivity and resistance of the body are not dependent on the state

metabolism

E) the reactivity of the body depends on gender and age +

25. Select signs characteristic of asthenic type of the body type:

A) obtuse epigastric angle

B) high level of basal metabolism +

C) low basal metabolic rate

D) tendency to obesity

E) tendency to increase blood pressure

26. The development of hereditary diseases due to mutations in

1. hepatocytes

2. gametes +

3. macrophages

4. fibrocytes

5. myocytes.

27. An example of a monogenic disease is

1. diabetes

2. hypertension

3. atherosclerosis

4. hemochromatosis

5. glycogenosis. +

28. A set of sex chromosomes with Klinefelter syndrome is

1. ХХУ +
2. ХО
3. ХХХ
4. УО
5. XY

29. Coronary heart disease belongs to the group of

1. hereditary diseases

2. multifactorial diseases (polygenic) +

3. monogenic

4. chromosome

5. diseases in the occurrence of which an exceptional role is played by environmental factors.

30. Puckering of the cell nucleus is called

1. pycnosis; +

2. karyorexis;

3. karyolysis;

4. autolysis;

5. necrobiosis.

31. Ionizing radiation has the greatest impact

1. on the ribosomes;

2. on the sarcoplasmic reticulum;

3. on the cell nucleus in mitosis; +

4. to the Golgi complex;

5. on the mitochondria.

32. A specific manifestation of cell damage in cyanide poisoning is

1. denaturation of protein molecules

2. enhanced lipid peroxidation

3. blockade of cytochrome oxidase +

4. acidosis

5. uncoupling of oxidation and phosphorylation processes

33. Suppression of cytochrome oxidase activity is a specific manifestation in

A) exposure to radiation

B) cyanide poisoning +

C) high temperature

D) mechanical injury

E) the effects of antioxidants

34. The statement is correct

1. intracellular acidosis and cell hyperhydria are a strictly specific manifestation of cell damage

2. increased permeability of cell membranes is a strictly specific manifestation of cell damage

3. an increase in the permeability of cell membranes accompanies any damage to the cell +

4. the ability to stain damaged cells is reduced

5. the electrical conductivity of damaged cells is usually reduced

35. The development of cell edema with damage is facilitated by:

A) an increase in the concentration of intracellular sodium +

B) increased intracellular potassium concentration

C) increasing the permeability of the cytoplasmic membrane for

ions +

D) inhibition of anaerobic glycolysis

E) decreased hydrophilicity of the cytoplasm

36. What are the mechanisms of cell damage:

A) increased conjugation of oxidative phosphorylation

B) increased activity of enzymes of the DNA repair system

C) enhancement of free radical oxidation of lipids +

D) the output of lysosomal enzymes in the hyaloplasm +

E) acidosis +

37. Cell damage may occur due to a change in its genetic program during:

A) expression of pathological genes +

B) repression of normal genes +

C) gene translocation +

D) a change in the structure of genes +

E) gene expression of the main histocompatibility complex

38. Indicate the causes of cell overhydration in case of damage:

A) a decrease in the activity of Na + / K + - ATPase +

B) increase of intracellular osmotic pressure +

Lipid peroxidation intensification) +

D) an increase in the hydrophilicity of cytosolic proteins +

E) a decrease in the activity of Ca ++ - ATPase +

39. Which of the following indicators indicate damage of transmembrane ion pumps?

A) an increase in intracellular calcium +

B) decrease in intracellular sodium

C) increased intracellular potassium

D) decrease in intracellular potassium +

E) increase in intracellular sodium +

40. Excessive activation of free radical and peroxide reactions causes:

A) conformational changes in lipoprotein complexes cell membranes +

B) inactivation of sulfhydryl groups of proteins +

C) suppression of oxidative phosphorylation +

D) a decrease in the activity of sodium-calcium transmembrane exchange mechanism +

E) activation of membrane-bound receptor function

41. An indicator of cell damage is

1. increase the pH of the cytoplasm

2. increase in cell membrane potential

3. increased intracellular potassium concentration

4. increase in intracellular calcium concentration +

5. decrease in intracellular sodium concentration

42. What are the main reasons for the activation of phospholipases in cell damage:

A) an increase in the intracellular content of sodium ions

B) an increase in the intracellular content of calcium ions +

C) a decrease in the intracellular content of potassium ions

D) increased intracellular water content

E) decrease in intracellular pH +

43. Which of the following substances weaken the damaging effect of free radicals on the cell?

A) glutathione peroxidase +

B) superoxide dismutase +

C) Vitamin E +

D) unsaturated fatty acids

E) ionol +

44. The consequence of lipid peroxidation in membranes is an increase of

1. membrane permeability +

2. surface tension

3. dielectric strength membranes

4. potassium in the cells

5. macroergs in cells

45. Violation of interstitial carbohydrate metabolism leads to increased formation

1. a-ketoglutaric acid

2. arachidonic acid

3. pyruvic acid +

4. b-hydroxybutyric acid

5. glutamic acid

46. In the pathogenesis of hyperlactatacidemia is important

1. increased glycogenesis

2. increased glycogenolysis

3. activation of lipolysis

4. activation of anaerobic glycolysis +

5. enhanced oxidation in the Krebs cycle.

47. The cause of acute hypoglycemia is

1. insulinoma

2. insulin overdose +

3. glycogenosis

4. chronic adrenal insufficiency

5. fasting

48. What is the main pathogenetic link of hypoglycemic coma?

A) carbohydrate and energy “starvation” of brain neurons +

B) carbohydrate "starvation" of the myocardium

C) blood hyperosmia

D) uncompensated ketoacidosis

49. Hormonal hyperglycemia develops with

1. insulin deficiency +

2. glucocorticoid deficiency

3. excess parathyroid hormone

4. excess vasopressin

5. deficiency of growth hormone.

50. The leading pathogenesis of emotional hyperglycemia is

1. increased renal glucose reabsorption

2. increased glycogenesis

3. inhibition of gluconeogenesis

4. impaired glucose utilization by the cell

5. increased glycogenolysis. +

51. In the pathogenesis of extrapancreatic insulin deficiency is important:

a - the formation of antibodies to beta cells of islets of Langerhans; b - genetic defect

insulin synthesis; c - increased activity of liver insulinase; d - blockade of insulin receptors; e - violation of the blood supply to the pancreas

A) a, b, c

B) c, d +

C) c, e

D) a, d, e

E) e, a, b

52. Glucosuria causes diabetes

1. ketonemia

2. hyperglycemia +

3. hyperlipidemia

4. polyuria

5. hyperlactatacidemia

53. Glucosuria in the early stages of diabetes is a consequence of:

A) ketonemia

B) Hyperglycemia. +

C) Hyperlipidemia.

D) Polyuria.

E) Hyperlactatacidemia.

54. The cause of polyuria in the early stages of diabetes mellitus is

1. kidney microangiopathy

2. glucosuria +

3. ketonuria

4. hypostenuria

5. thirst and polydipsia

55. Ketosis in diabetes mellitus is caused by

a) activation of lipolysis

b) decrease in the excretion of ketone bodies by kidneys

c) increased formation of ketone bodies

d) insufficient utilization of ketone bodies

e) violation of the oxidation of ketone bodies in the Krebs cycle.

1. a, b, c

2. b, c

3. b, c, d

4. a, c, d, e +

5. b, c, d, e

56. For a violation of carbohydrate metabolism in diabetes mellitus is characteristic:

a - decrease in glycogenogenesis in the liver;

b – increase in gluconeogenesis;

c - violation of glucose utilization by cells;

d - a decrease in the concentration in the blood of lactate and pyruvate;

e - a decrease in gluconeogenesis;

A) a, b, d

B) a, c, d

C) a, b, c +

D) a, c, e

E) c, d, e

57. The development of angiopathies in diabetes is associated with

1. ketosis

2. hyperglycemia +

3. hypoproteinemia

4. lactic acidosis

5. hypoosmolarity

58. A positive nitrogen balance in the body develops during

A) excess insulin +

B) excess glucocorticoids

C) protein fasting

D) tumor cachexia

E) diabetes

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60. Hypoproteinemia is

1. appearance of unusual (pathological proteins) in the blood

2. increase in protein in the blood

3. a decrease in the total amount of protein, mainly due to albumin +

4. change in the ratio of blood proteins

5. decrease in gamma globulins level in the blood

61. Hypoproteinemia is accompanied with

a - lowering of oncotic blood pressure; b - violation of the transport function of plasma proteins; c - hypocoagulation; d - the development of edema

e - increase of hormones level of free fraction in the blood

1. c, d

2. a, b, d, e +

3. b, e

4. a, c, d

5. b, c, e

62. The pathogenetic factor of edema is:

A) Increase of oncotic blood pressure.

B) Lowering of the hydrostatic pressure of the blood.

C) Reducing the permeability of the pared vascular

D) Increase of osmotic and oncotic pressure in tissues. +

E) Lowering of aldosterone secretion.

63. The development of edema contributes to

1. high blood albumin

2. increased production of aldosterone and ADH +

3. enhanced drainage of interstitial lymph vessels

4. reduced production of antidiuretic hormone

5. reduced permeability of the pared vascular

64. Neuro-endocrine edema factor is

1. activation of the sympathoadrenal system

2. insufficient formation of antidiuretic hormone

3. secondary aldosterone deficiency

4. hyperinsulinism

5. a secondary increase in the formation of aldosterone and ADH. +

65. The tissue factor of edema is

1. lowering of osmotic pressure of tissue

2. lowering of plasma osmotic pressure

3. lowering of oncotic blood pressure

4. increase of hydrostatic pressure of the tissue

5. hyperosmia and hyperoncia of tissues. +

66. The leading role in the pathogenesis of allergic edema plays

1. vascular factor +

2. tissue factor

3. hemodynamic factor

4. neuro-endocrine factor

5. oncotic factor

67. The oncotic factor plays a major role in development of

1. cardiac edema

2. cachectic edema +

3. allergic edema

4. Toxic edema

5. jade edema

68. The factor playing the main role in the development of congestive edema is

1. neuro-endocrine

2. oncotic

3. tissue

4. hemodynamic +

5. vascular.

69. The starting pathogenetic factor in development of cardiac edema is

 1. increase of permeability of the pared vascular

 2. increase of colloid osmotic pressure of tissues

 3. increase of venous blood pressure +

 4. lowering of oncotic blood pressure

 5. decrease in lymphatic outflow

70. The initial link in the cardiac edema pathogenesis is:

A) Stimulation of the secretion of aldosterone.

B) Irritation of the volumo- and baroreceptors.

C) Reduced cardiac output. +

D) Increased sodium reabsorption in the kidneys.

E) Increased production of antidiuretic hormone and water reabsorption in kidneys;

71. Pathogenetic therapy of hungry (cachectic) edema includes

1. the introduction of antihistamines

2. appointment of diuretics

3. administration of glucocorticoids

4. salt-free diet

5. intravenous administration of albumin. +

72. Edema of the abdominal cavity is indicated by the term

1. hydrothorax

2. hydropericardium

3. ascites +

4. peritonitis

5. hydronephrosis

73. Water intoxication develops during

1. excess water intake

2. insufficient elimination of water from the body

3. excess intake of mineral salts

4. excessive intake of water in the body against the background of its insufficient elimination +

5. forced consumption of sea water

74. Hyperosmolar hyperhydration occurs

1. during transfusing a large amount of saline;

2. during transfusing hypoosmolal solutions;

3. during intravenous injection of 5% glucose;

4. during excess consumption of fresh water;

5. during forced excess consumption of sea water +

75. Indicate compensatory reactions during dehydration;

A) increased aldosterone production +

B) centralization of blood circulation +

C) increased vasopressin excretion +

D) decreased renin excretion

E) decrease in daily urine output +

76. Indicate the signs of the syndrome of general hyperhydration;

A) edema, dropsy +

B) weight gain +

C) increased hematocrit

D) increase in circulating blood volume +

E) increase in blood pressure +

77. A negative water balance is observed during

1. liver cirrhosis

2. heart failure

3. diabetes insipidus +

4. nephrotic syndrome

5. acute diffuse glomerulonephritis

78. The cause of hyperosmolar dehydration may be

1. diarrhea

2. repeated vomiting

3. intestinal fistulas

4. diabetes +

5. Aldosterone deficiency.

79. Hyperosmolar dehydration is characterized by

1. an increase in the total water content in the body

2. lowering the osmotic pressure in the extracellular fluid

3. increase in water content in cells

4. the movement of water from cells into the extracellular space +

5. lack of thirst

80. Hypoosmolar dehydration occurs during

1. diarrhea +

2. diabetes

3. diabetes insipidus

4. atresia of the esophagus

5. water starvation.

80. Гипоосмолярнаядегидратацияхарактеризуется

1. увеличением общего содержания воды в организме
2. понижением осмотического давления во внеклеточной жидкости+
3. снижением содержания воды в клетках
4. перемещением воды из клеток во внеклеточное пространство
5. мучительным чувством жажды

81. During dehydration we can observe:

a - blood thickening; b - disorder of microcirculation; c– increase of

central venous pressure; d - lowering of blood pressure

e - a decrease in the volume of circulating blood

A) a, c

B) a, b, d, e +

C) a, b, c

D) a, e

E) d, e

82. Hypernatremia develops during

1. increased aldosterone secretion +

2. increased vasopressin secretion

3. increased parathyroid hormonesecretion

4. decreased angiotensin secretion

5. decreased parathyroid hormone secretion

83. Hyperkalemia occurs

1. during enhanced tissue decay +

2. duringexcessive production of aldosterone

3. duringexcessive production of 11-deoxycorticosterone

4. duringindomitable vomiting

5. duringprolonged diarrhea

84. Hypercalcemia occurs during

1. hypersecretion of aldosterone

2. hypersecretion of vasopressin

3. hypersecretion of parathyroid hormone +

4. hypersecretion of calcitonin

5. alkalose

85. The pathogenesis of nutritional hyperlipidemia is due to

1. increased mobilization of fat from the depot

2. increased intake of fat with food +

3. delayed transition of fat from blood to tissue

4. hypoalbuminemia

5. low activity of lipoprotein lipase in the blood.

86. Ketone bodies are formed

1. in the intestine;

2. in the liver; +

3. in the lungs;

4. in the kidneys;

5. in the muscles.

87. \_\_\_\_\_\_\_\_\_\_have antiatherogenic properties

1. low density lipoproteins;

2. very low density lipoproteins;

3. intermediate density lipoproteins;

4. high density lipoproteins; +

5. chylomicrons.

88. The accumulation of lipids in the intima of blood vessels and monocytes are due to the ability to

1. activate lysosomal enzymes that break down esterified cholesterol

2. capture atherogenic lipoproteins by nonspecific endocytosis +

3. inhibit the activity of lecithin-cholesterol-acyl transferase

4. incorporate esterified cholesterol into the phospholipid membrane layer

5. activate lipoprotein lipase

89. The correct sequence of phenomena in the process of atherogenesis is

a) migration of smooth muscle cells to the focus of lipid accumulation;

b) capture of lipoproteins by macrophages, their transformation into "foam cells"

c) damage to the endothelium and the accumulation of lipoproteins in the intima of the arteries

d) the formation of a fibrous capsule around the focus of lipid accumulation

1. b, c, d, a

2. c, b, a, d +

3. a, c, d, b

4. c, d, b, a

5. a, b, c, d

90. The signs of hypovitaminosis C include

1. xerophthalmia

2. paresis and paralysis

3. hemorrhagic diathesis +

4. hemeralopia

5. polyneuritis

91. Vitamin B12 deficiency is characterized by development of

1. iron deficiency anemia

2. megaloblastic anemia +

3. normoblastic anemia

4. aplastic anemia

5. microspherocytic anemia

92. pH that corresponds to compensated acidosis and alkalosis is:

1. 7,35-7,45 +
2. 7,0 – 8,0
3. 7,2-7,8
4. 7,45-7,50
5. 7,0-7,4.

93. The most capacious buffer of blood plasma is

1. protein

2. bicarbonate +

3. hemoglobin

4. phosphate

5. acetate

94. The basis of gas acidosis is

1. excess non-volatile acids

2. mineral acid poisoning

3. enhanced release of CO2 from the body

4. the accumulation of carbon dioxide in the body +

5. excess bases in the blood

95. \_\_\_\_\_\_\_\_\_ leads to the development of gas alkalosis

1. increase in carbon dioxide in the atmosphere

2. lung hypoventilation

3. circulatory failure

4. hypercapnia

5. hyperventilation of the lungs. +

96. The cause of non-gas acidosis is

1. prolonged vomiting

2. shortness of breath during encephalitis

3. hypersecretion of steroid hormones of the adrenal glands

4. profuse diarrhea +

5. sodium bicarbonate poisoning

97. The most important in compensating for non-gas acidosis is

1. hydrocarbonate buffer +

2. protein buffer

3. sulfate buffer

4. hemoglobin buffer

5. phosphate buffer

98. At what shift of the acid-base balance is the compensation of the disturbed state due to hyperventilation of the lungs?

A) metabolic acidosis +

B) metabolic alkalosis

C) gas acidosis

D) gas alkalosis

99. Loss of large amounts of gastric juice with indomitable vomiting can lead to

1. exogenous acidosis

2. excretory acidosis

3. gas alkalosis

4. excretory (non-gas) alkalosis +

5. exogenous alkalosis

100. Metabolic alkalosis is characterized by:

A) blood pH> 7.45 +

B) an increase in the concentration of bicarbonates in plasma +

C) a compensatory decrease in the voltage of CO2 in the blood plasma

D) weakening of ammoniogenesis in the kidneys +

E) lower titratable acidity of urine +

101. What are the mechanisms involved in body temperature rise at fever?

A) increased conjugation of oxidation and phosphorylation

B) peripheral vasoconstriction +

C) enhancing contractile muscle thermogenesis +

D) decrease in perspiration +

E) activation of oxidative processes +

102. Endogenous pyrogens are formed in

1. red blood cells

2. platelets

3. white blood cells (leucocytes) +

4. hepatocytes

5. parenchymal cells

103. The producers of endogenous pyrogens are:

a - macrophages, b - mast cells, c - plasma cells, d - neutrophils,

e - red blood cells

A) a, d +

B) b, c

C) d, e

D) c, e

E) a, e

104. Endogenous pyrogens include

a) Interleukin 1; b) interleukin 4; c) interleukin 8; d) interleukin 6;

e) tumor necrosis factor; f) endotoxins of microorganisms

1. b, c

2. f

3. a, b, c, d

4. a, b, c

5. a, c, d, e +

105. Leukocyte pyrogensinfluence at:

1. heat-sensitive peripheral receptors;

2. spinal cord motor neurons;

3. neurons of the preoptic area of the hypothalamus; +

4. neuromuscularpathways;

5. back-corticalpathways

106. The increase in temperature during fever is due to the influence of endopyrogens on the thermoregulation center, which is located in

1. cerebral cortex

2. reticular formation

3. hypothalamus +

4. medulla oblongata

5. limbic system

107. The statement is correct

1. pyrogenic properties of bacterial cells depend on their virulence

2. endotoxins of gram-negative bacteria belong to endogenous pyrogens

3. exogenous pyrogens are lipopolysaccharides +

4. the degree of temperature increase during fever depends on the ambient temperature

5. during fever, thermoregulation mechanisms are violated

108. \_\_\_\_\_\_ contributes to reducing heat transfer

1. vasodilation of the skin

2. increased perspiration

3. increased tone of the parasympathetic nervous system

4. rapid breathing

5. increased tone of the sympathetic nervous system. +

109. A rapid temperature rise in the first stage of a fever is accompanied by

1. muscle tremors and chills +

2. tachypnea

3. skin redness

4. fall in blood pressure

5. enhanced perspiration.

110. The degree of temperature rise during fever depends on:

A) the concentration of exogenous pyrogens in tissues +

B) the intensity of the formation of cytokines by producer cells +

C) the sensitivity of the thermoregulation center to the action of pyrogens +

D) the age of the patient +

E) insulating properties of clothing

111. Choose the correct statement

1. in the first stage of fever, skin temperature is lower than blood temperature +

2. muscle trembling reduces heat production

3. blood pressure in the first stage of fever is lowered, sometimes very sharply

4. in the first stage of fever, diuresis decreases

5. heat balance in the second stage of fever is characterized by the predominance of heat production over heat transfer

112. In the first stage of fever \_\_\_\_\_\_\_\_is observed

1. increased heat production without changing heat transfer

2. increased heat production and heat transfer

3. reduction of heat transfer and increased heat production +

4. reduction of heat production and heat transfer

5. Decrease in heat production and increase in heat transfer.

113. The first stage of fever is characterized by:

a - increase in blood pressure; b - tachycardia; c - chills, muscle tremors; d - decrease in urine output; e - increased sweating

A) a, d, e

B) a, b, c +

C) c, d, e

D) b, d, e

E) a, b, e

114. The second stage of fever is characterized by:

a - bradycardia; b - redness of the skin; c - increased motility of the gastrointestinal tract; d - increase in urine output; e - tachycardia

A) a, b

B) b, c, d

C) b, e +

D) d, e

E) d

115. The increase in heat transfer in the third stage of fever is associated with

1. vasoconstriction;

2. suppression of sweating processes

3. increased metabolism;

4. increased sweating (perspiration); +

5. high blood pressure.

116. "Critical"decrease of temperature during a fever may lead to:

1. the development of collapse +

2. increased gastrointestinal motility

3. the development of hyperhydration

4. increased heart rate

5. increaseinbloodpressure.

117. A febrile temperature curve characterized by diurnal temperature variations not exceeding 1° C (one) is called

1. laxative

2. intermittent

3. constant +

4. atypical

5. debilitating

118. Tachycardia during fever occurs as a result of the direct influence of warm blood

1. at the set point;

2. atthe sinus node; +

3. atthe thermostat;

4. atthe spinal center of thermoregulation;

5. atperipheral heat-sensitive receptors.

119. Fever is characterized by:

1. the predominance of lipogenesis over lipolysis

2. the prevalence of glycogenogenesis over glycogenolysis

3. activation of glycogenolysis and lipolysis +

4. inhibition of ketogenesis

5. inhibition of proteolysis

120. Characteristic of a change in water-salt metabolism in the second stage of fever is

1. hyperosmolar dehydration

2. the elimination of sodium and water from the body

3. isoosmolar dehydration

4. hypoosmolar dehydration

5. retention of sodium and chloride in the body. +

121. The statement is correct

1. chills are a characteristic sign of the second stage of fever

2. the feeling of thermal comfort is characteristic of the first stage of fever.

3. pallor and cooling of the skin are characteristic of the first stage of fever +

4. sweating is most shown in the first stage of fever

5. diuresis decreases in the third stage of fever.

122. The positive role of fever is

1. a decrease in the antitoxic function of the liver;

2. suppression of antibody synthesis;

3. increased phagocytosis; +

4. enhancing the reproduction of fibroblasts;

5. enhancing catabolic processes.

123. The leading link in the pathogenesis of arterial hyperemia is

A) obstruction of blood outflow

B) dilation of arterioles and increased blood flow +

C) reduced blood flow

D) increase in linear blood flow velocity

E) increase in the number of functioning capillaries

124. Which of the following factors can lead to the development of arterial hyperemia?

A) transection of the sympathetic peripheral nerves +

B) mechanical irritation of the tissue or organ +

C) removal of an elastic tourniquet from extremities +

D) closure of the lumen of an artery with a thrombus

E) the effect of mustard on the skin +

125. Characteristic for arterial hyperemiais

1. reduction of the cross-sectional area of blood vessels

2. decrease in volumetric blood flow velocity

3. venous blood arterialization +

4. proliferation of connective tissue

5. reducing the number of functioning capillaries.

126. What is the cause of the scarlet color of the organ during arterial hyperemia?

A) increase in the content of oxyhemoglobin in the blood +

B) decrease in the content of oxyhemoglobin in the blood

C) increase in arteriovenous oxygen difference

D) decrease in the volumetric blood flow velocity

E) increase in the content of reduced hemoglobin in the blood

127. Increase in the temperature of an organ or tissue in the area of arterial hyperemia is due to:

A) increased arterial blood flow +

B) enhancing of oxidative processes +

C) increased lymph formation

D) increase in the number of functioning capillaries +

E) decrease in the number of functioning capillaries

128. The consequences of arterial hyperemia are

a - tissue necrosis; b - an increase in the functional activity of the organ;

c - rupture of a vessel with hemorrhage and erythrocyte diapedesis

d - proliferation of connective tissue; e - intensive removal of products

metabolism and generalization of the pathological process

A) a, b, c

B) c, d, e

C) b, c, e +

D) a, d, e

E) a, b, e

129. Indicate the possible consequences of arterial hyperemia:

A) proliferation of connective tissue

B) enhancing of organ function +

C) tissue dystrophy

D) hemorrhage +

E) generalization of infection +

130. Venous hyperemia is:

A) increase of blood supply to the tissue due to increased blood flow.

B) increase ofblood supply to the tissue due to obstruction of the outflow of blood. +

C) decrease of blood supply to the tissue due to reduced blood flow.

D) local stoppage of blood flow in the vessels of the microvasculature.

E) circulation in the blood of particles that are not normally found.

131. The cause of venous hyperemia may be

1. angiospasm

2. clogging of the lumen of the leading artery with a thrombus

3. compression of veins by a tumor +

4. compression of the leading artery

5. tissue enhancement

132. What changes in microcirculation are characteristic of venous hyperemia?

A) slowing of blood flow in the small arteries, capillaries and veins +

B) pendulum-like blood flow +

C) increased lymph flow from the tissue

D) decreased functioning veins and capillaries

E) increase in arteriovenous pressure difference

133. Indicate the possible consequences of venous hyperemia:

A) proliferation of connective tissue +

B) organ function enhancement

C) tissue dystrophy +

D) hemorrhage

E) generalization of infection

134. The cause of the development of ischemia may be

1. vein obstruction thrombus

2. angiospasm +

3. tissue enhancement

4. damage to the vasoconstrictor nerves

5. compression of a vein by a tumor

135. What biologically active substances can cause ischemia?

A) histamine

B) catecholamines +

C) acetylcholine

D) bradykinin

E) thromboxane A2 +

136. A sign of ischemia is

1. increased tissue temperature

2. blanching tissue +

3. increased blood flow

4. cyanotic tissue

5. pulsation of small vessels

137. Absolutely sufficient number of collaterals is available in

1. skeletal muscle +

2. heart muscle

3. kidneys

4. spleen

5. the brain.

138. What changes can occur in the area of ischemia?

A) necrosis +

B) acidosis +

C) weakening function +

D) function enhancement

E) accumulation of Ca++in the cell hyaloplasm +

139. In the pathogenesis of true capillary stasis \_\_\_\_\_\_\_\_\_\_\_are significant:

a - lowering blood viscosity; b - increase in blood viscosity;

c- lowering the electric charge of blood cells

d - acceleration of blood flow; e - increased permeability of the vascular wall

A) a, b, d

B) c, d, e

C) b, c, e +

D) a, d

E) a, e;

140. One of the mechanisms of sludge is

1. decrease of blood viscosity

2. decrease of permeability of the vascular wall

3. increase of blood viscosity +

4. increased of blood flow

5. increase of electric charge of blood cells

141. Factors contributing to thrombosis: a: Slowing blood flow; b: Damage to the vascular wall

c: Increased blood coagulation ability; d: Thrombocytopenia, e. Activation of the anticoagulation system

A) c, d, e

B) a, b, c +

C) b, d, e

D) c, d

E) b, e

142. The statement is correct

1. gas embolism may develop during increased barometric pressure

2. gas embolism develops during damage to large arteries

3. gas embolism develops during damage to the subclavian vein

4. air embolism develops during damage to the subclavian vein+

5. air embolism develops during a rapid decrease in barometric pressure

143. The statement is correct

1. during arterial hyperemia, the arteriovenous oxygen difference increases

2. during venous hyperemia, the volumetric and linear blood flow velocity is reduced +

3. blanching of a tissue site is a symptom of arterial hyperemia

4. the number of functioning capillaries increases during ischemia

5. cyanosis with venous hyperemia is due to the accumulation of carboxyhemoglobin in the blood

144. The statement is correct

1. the number of functioning capillaries decreases sharply with arterial hyperemia

2. tissue edema usually develops during venous hyperemia +

3. the application of mustard plasters on the body leads to the development of ischemia

4. massage leads to the development of intravascular platelet aggregation

5. heart attack is usually a consequence of arterial hyperemia

**I**

145. The first stage of inflammation is

1. alteration; +

2. exudation;

3. emigration of white blood cells;

4. phagocytosis;

5. proliferation.

146. Primary alteration in inflammation results from

1. actions of inflammatory mediators

2. physico-chemical changes in the focus of inflammation

3. the damaging effect of the phlogogenic factor +

4. microcirculation disorders

5. metabolic disorders in the focus of inflammation.

147. The increase of decomposition of substances in the focus of inflammation is associated with

1. activation of lysosomal enzymes +

2. activation of mitochondrial enzymes

3. activation of adenylate cyclase

4. inhibition of enzymes of the anaerobic stage of glycolysis

5. inhibition of lipid peroxidation enzymes.

148. Characteristic for changes in carbohydrate metabolism in the focus of inflammation is:

1. activation of anaerobic glycolysis +

2. increased glycogen synthesis

3. increased synthesis of ketone bodies

4. increase in the content of nucleotides, nucleosides

5. activation of lipolysis.

149. The pathogenetic factor of local acidosis in inflammation is

1. arterial hyperemia

2. violation of vascular permeability

3. the accumulation of under-oxidized metabolic products +

4. white blood cell emigration

5. extravasation

150. The most short-term stage of circulatory disorders

with inflammation is

1. arterial hyperemia

2. spasm of arterioles (ischemia) +

3. local stoppage of blood flow

4. venous hyperemia

5. stasis.

151. Exudation is called

1. the exit of blood from the vascular bed into the inflamed tissue

2. the exit of the protein-containing liquid part of the blood into the inflamed tissue +

3. the release of leukocytes from blood vessels into tissue

4. accumulation of fluid in cavities

5. accumulation of fluid in the tissues.

152. In the pathogenesis of exudation is important

1. lowering of permeability of the vascular wall

2. lowering o blood pressure

3. increased of osmotic and oncotic pressure of tissues +

4. increased of oncotic blood pressure

5. increasing the concentration of coarse blood proteins.

153. \_\_\_\_\_\_ contributes to the exudation process

A) Lowering osmotic pressure in the focus of inflammation

B) Increased oncotic blood pressure

C) Reduced capillary permeability

D) Hyperosmia in the focus of inflammation +

E) Decrease in hydrodynamic pressure in capillaries

154. The main source of hydrolytic enzymes in the focus of inflammation are

A) microbial waste products

B) destroyed white blood cells +

C) mast cells

D) microbial cells

E) platelets

155. During inflammation caused by pyogenic microorganisms, \_\_\_\_\_\_\_\_\_ predominates in the composition of exudate:

1. neutrophils; +

2. eosinophils;

3. basophils;

4. monocytes;

5. lymphocytes,

156. \_\_\_\_\_\_\_\_\_contributes to the emigration of white blood cells

1. positive chemotaxis +

2. decrease in oncotic blood pressure

3. acceleration of blood flow

4. increased oncotic blood pressure

5. decrease in vascular wall penetrability.

157. The Latin name “redness” as a local sign of inflammation is

1. calor

2. dolor

3. rubor +

4. tumor

5. funciolaesa

158. The pathogenetic factor of local temperature increase during inflammation is

1. venous hyperemia

2. ischemia

3. arterial hyperemia +

4. stasis

5. embolism

159. Common signs of inflammation are:

a) pain, redness, organ dysfunction

b) symptoms of intoxication, c) acceleration of ESR, d) fever, leukocytosis

e) swelling due to edema.

A) b, d, e

B) b, c, e,

C) a, b, e

D) c, d, e

E) b, c, d +

**Allergy**

160. Acquired (secondary) autoallergens include

1. the brain;

2. testes

3. the lens;

4. colloid of the thyroid gland;

5. tissue + microbe. +

161. The pathochemical stage of allergic reactions is characterized by

1. disturbance of microcirculation

2. spasm of smooth muscle elements

3. increased vascular wall permeability

4. release of allergy mediators +

5. the formation of immune complexes.

162. The sensitization of the body develops:

1. at repeated administration of anaphylactogen;

2. at the initial intake of the allergen; +

3. after anaphylactic shock;

4. after immunotherapy with allergens;

5. after the administration of a permissible dose of allergen.

163. Passive sensitization develops at:

1. intramuscular injection of a foreign protein

2. intravenous injection of protein preparations

3. administration of antigen by inhalation

4. the receipt of antigen through the skin

5. the administration of specific antibodies or sensitized lymphocytes +

164. In the development of allergic reactions of the reagin type, immunoglobulins of the class \_\_\_\_\_\_take part:

1. А
2. М
3. Е +
4. G
5. D

165. The reagin type of allergic reactions plays a major role in the pathogenesis of:

A) tuberculosis.

B) Graft rejection reactions.

C) Autoimmune hemolytic anemia.

D) Atopic forms of bronchial asthma +.

E) Serum sickness.

166. Give examples of reactions developing according to I (reagin)

type of immune damage:

A) urticaria +

B) myasthenia gravis

C) anaphylactic shock +

D) Quincke's edema +

E) serum sickness

167. The immunological stage of allergic reactions of the reagin type is characterized by

1. the damaging effect of lysosomal enzymes on cells

2. the formation of immunoglobulins E and their fixation on the surface of mast cells +

3. mast cell degranulation

4. release of mediators

5. clinical manifestations.

168. Immunoglobulins E are fixed on the surface of mast cells

1. during the cytotoxic type of allergic reactions;

2. during a reagin type of allergic reaction; +

3. duringimmunocomplex type of allergic reactions;

4. during tuberculin type of hypersensitivity;

5. during a stimulating type of allergic reaction.

169. The missinglink in the pathogenesisof allergic reactionsof reaginictypeisreceiptofallergen into the body →synthesis of immunoglobulins E →?→re-entry of allergen→immune complex formation allergen+ antibody

1)fixation ofantibodies onthe surface of macrophages;

2)fixingof antibodies on the surface of hepatocytes;

3)fixingof antibodieson thesurface of mast cells and basophils +

4) fixingof antibodies onthe surface of T-lymphocytes;

5) fixingof antibodieson the surface ofhistiocytes;

170. The mediators of allergic reactions of the reagin type are: a. Lymphokines; b. prostaglandins; c. Histamine; d. Leukotrienes; e. Factor inhibiting macrophage migration

A) a, b, c

B) d, e

C) b, c, d +

D) a, b, c, d

E) a, e

171. The allergic reactions of the reagin type include: a. Bronchial asthma; b. Pollinosis; c. Transplant rejection; d. Serum sickness;e. Tuberculin test

A) a, b, e

B) b, e

C) a, b +

D) c, d, e

E) d, e

172. Severe bronchospasm during atopic bronchial asthma is caused by:

1. leukotrienes; +

2. catecholamines;

3. prostaglandins of group E;

4. cytokines;

5. kinins.

173. The reagin type of allergic reaction plays a major role in the pathogenesis of:

1. tuberculosis

2. transplant rejection reactions

3. autoimmune hemolytic anemia

4. atopic forms of bronchial asthma +

5. serum sickness.

174. For allergic reactions of the reagin type, it is characteristic:

A) the leading role in the pathogenesis of immunoglobulin E +

B) the reaction manifests itself 15-20 minutes after repeated contact with the allergen +

C) the reaction manifests itself in 24-48 hours after repeated contact with

allergen

175. Allergic reactions of the cytotoxic type include:

1. transplant rejection reaction

2. tuberculin test

3. urticaria

4. hay fever

5. agranulocytosis. +

176. Diseases that develop mainly in type III of immune damage are:

A) serum sickness +

B) immune agranulocytosis

C) exogenous allergic alveolitis +

D) reactions according to the type of Arthus phenomenon +

E) autoimmune hemolytic anemia

177. T-lymphocytes play a major role in the pathogenesis of allergic reactions of:

1. reagin type

2. anaphylactic type

3. immunocomplex type

4. cell-mediated type +

5. cytotoxic type.

178. The mediators of delayed-type allergic reactions are

1. histamine and serotonin

2. lymphokines +

3. acetylcholine and bradykinin

4. prostaglandins

5. heparin

179. \_\_\_\_\_\_refer to mediators of allergic reactions of the cell-mediated type:

1. histamine

2. factor of chemotaxis of eosinophils

3. thromboxane A2

4. leukotrienes

5. a factor inhibiting the migration of macrophages. +

180. Specific hypersensitivity is carried out by:

1. fractional administration of a specific allergen +

2. prescription of antihistamines

3. psychotherapy

4. physiotherapy.

181. Indicate the reactions and diseases developing according to the IY type of immune damage:

A) contact dermatitis +

B) Arthus phenomenon

C) bacterial allergy +

D) food allergies

D) transplant rejection +

182. Exogenous normobaric hypoxia occurs during

1. climbing mountains

2. lift in aircraft

3. caisson works

4. decrease in РСО2 in air

5. being in a non-ventilated room. +

183. The hypobaric form of exogenous hypoxia is characteristic

1. hypocapnia; +

2. hyperoxemia;

3. high arteriovenous oxygen difference;

4. arterialization of venous blood;

5. decrease in blood oxygen capacity.

184. Hypoxia associated with the development of disorders in the blood system is called

1. exogenous

2. circulatory

3. tissue

4. ischemic

5. hemic +

185. Carbon monoxide poisoning leads to the development of\_\_\_\_\_\_\_\_\_\_\_ hypoxia:

1. tissue

2. hemic +

3. circulatory

4. respiratory

5. exogenous.

186. Nitrate poisoning leads to the formation of

1. deoxyhemoglobin

2. methemoglobin +

3. carbhemoglobin

4. carboxyhemoglobin

5. oxyhemoglobin.

187. Indicate the causes of hemic type hypoxia:

A) CO + poisoning

B) pulmonary emphysema

C) nitrate poisoning +

D) chronic blood loss +

E) hypovitaminosisВ12+

F) a decrease in the excitability of the respiratory center

188. Indicate the signs characteristic of circulatory type hypoxia:

A) decrease in blood flow velocity +

B) reduction of arteriovenous oxygen difference

C) decrease in oxygen content in arterial blood

D) an increase in arteriovenous oxygen difference +

E) acidosis +

189. Indicate the causes of respiratory-type hypoxia:

A) CO poisoning

B) pulmonary emphysema +

C) nitrate poisoning

D) chronic blood loss

E) hypovitaminosisВ12

F) a decrease in the excitability of the respiratory center +

190. Hypoxia, developing as a result of violations in the oxygen utilization system, is called

1. exogenous normobaric

2. tissue +

3. exogenous hypobaric

4. circulatory

5. respiratory

191. Deficiency of vitamins B1, B2, PP leads to the development of \_\_\_\_\_\_\_\_hypoxia:

1. tissue +

2. hemic

3. circulatory

4. respiratory.

5. exogenous

192. In the pathogenesis of tissue hypoxia, it matters:

a. Inactivation of respiratory enzymes;

b. Violation of the synthesis of respiratory enzymes;

c. Dissociation of oxidation and phosphorylation;

d. Damage to mitochondria;

e. Reduced tissue oxygen demand

A) a, b, c

B) a, b, c, d +

C) a, b, e

D) c, d, e

E) a, b, d, e

193. In the pathogenesis of hypoxic cell damage, it matters:

a) an increase in the sodium cell; b) activation of phospholipases;

c) the release of lysosomal enzymes;

d) a decrease in the intensity of lipid peroxidation;

e) accumulation of calcium in mitochondria;

f) malfunctioning of diaphragm pumping systems

A) a, b, c, d, e, f

B) a, b, c, d, e

C) a, f

D) a, b, c, e, f +

E) e

194. Urgent compensatory reactions during hypoxia are

a) release of deposited blood;b) tachycardia; c) hyperpnea;

d) hypertrophy of the respiratory muscles; e) activation of erythropoiesis

A) a, c, d

B) a, b, c +

C) d, e

D) c, e

E) b, d

195. Long-term adaptation to hypoxia is provided by:

a) release of deposited blood; b) tachycardia; c) hyperpnea

d) hypertrophy of the respiratory muscles; e) activation of erythropoiesis

A) a, c, d

B) a, b, c

C) d, e +

D) c, e

E) b, d

196. Compensation mechanisms for acute hypoxia do not include:

A) blood redistribution

B) increased lung ventilation

C) tachycardia

D) the release of red blood cells from the depot

E) decrease in blood flow velocity +

197. What are the mechanisms leading to an increase in oxygen capacity of blood at moderate repeated hypoxia:

A) increased cardiac output

B) an increase in the formation of erythropoietin +

C) the acceleration of the release of red blood cells from the bone marrow into the blood +

D) an increase in the number of red blood cells in the blood +

E) increase in alveolar ventilation

198. What changes in the cell can be considered compensatory for hypoxia?

A) a decrease in the activity of Na/К+ and Са2+ + ATPases

B) activation of glycolysis +

C) activation of phospholipase A2

D) glycogen mobilization +

E) LPO activation

199. What changes are most likely to occur in a person living for a long time high in the mountains?

A) increase of hematocrit +

B) heart hypertrophy +

C) lung hypoventilation

D) inhibition of the synthesis of nucleic acids and proteins

E) hypertrophy of the respiratory muscles +

**Tumors**

200. The primary, fundamental, sign of malignant tumors is

1. cachexia

2. the systemic effect of the tumor on the body

3. invasive growth +

4. recurrence

5. Pasteur's negative effect

201. Biological characteristics characteristic of malignant tumors

a) Unregulated cell growth; b) The loss of the "limit" of Hayflick division;

c) Expansive growth; d) Loss of contact braking; e) Invasive growth

A) a, b, c

B) a, c, d

C) a, b, d, e +

D) c

E) a, b, c, d

202. A feature of a tumor cell is

1. reduced ability to re- and de- amination of amino acids; +

2. decrease in the uptake of amino acids from the blood;

3. decrease in the uptake of glucose from the blood;

4. decrease in the uptake of cholesterol from blood;

5. decrease in the synthesis of nucleic acids.

203. Antigenic atypia of tumors includes:

a) synthesis of embryospecific proteins; b) the phenomenon of substrate traps

c) synthesis of proteins characteristic of other tissues; d) Pasteur negative effect

e) an increase in the synthesis of proteins of the main histocompatibility complex

1. a, c +

2. a, e

3. b, d

4. a, b

5. c, e

204. Indicate the second stage of tumor metastasis.

1. separation of one or a group of tumor cells from the primary tumor site

2. transportation of cells through the lymphatic and blood vessels +

3. the formation of thromboembolism and its fixation on the vessel wall

4. the exit of cells outside the vessel, their implantation

5. tumor progression.

205. Indicate the correct sequence of stages of carcinogenesis.

1. initiation, promotion, progression +

2. promotion, initiation, progression

3. progression, initiation, promotion

4. initiation, progression, promotion

5. promotion, progression, initiation

206. \_\_\_\_\_\_\_leadto the tumor transformation of the cell:

a) activation of oncogenes; b) inhibition of anti-oncogenes;

c) activation of apoptosis genes; d) the formation of oncoproteins;

e) activation of DNA repair systems;

A) a, b, c, d

B) a, b, d +

C) a, b, c

D) c

E) c, e

207. The following leads to tumor transformation of a cell:

A) Conversion of proto-oncogen to oncogene +.

B) Activation of anti-oncogenes.

C) Inactivation of antiapoptosis genes.

D) Activation of apoptosis genes.

E) Activation of proto-oncogenes.

208. Oncogen activation occurs due to

1. inflammation

2. hypoglycemia

3. mutations +

4. necrosis

5. hypoxia

209. Choose properties that characterize tumor cells

A) lack of contact inhibition during growth in culture +

B) increased adhesion between cells

C) a decrease in adhesive molecules on the cell membrane +

D) increase in intracellular Ca++

E) a decrease in the content in the cytoplasm of Ca+++

210. What are the properties of oncoproteins?

A) growth factors +

B) growth factor receptors +

C) membrane G-proteins +

D) ceylon

E) transmit growth signals to DNA +

210. Indicate the state of increased cancer risk:

A) acute inflammatory processes

B) chronic inflammatory processes +

C) irradiation of the body +

D) immunodeficiency states +

E) BCG vaccination

211. What factors contribute to the metastasis of tumor cells?

A) high level of contact braking

B) the production of type 4 collagenase by tumor cells +

C) increased adhesion between tumor cells

D) a decrease in the content of adhesive molecules in the cytoplasmic membrane +

E) enhancing the expression of molecules of the HLA complex

213. ESR (erythrocyte sedimentation rate) increases with increase in blood

A) coarse proteins +

B) low molecular weight proteins

C) lecithin

D) carbon dioxide partial voltage

E) red blood cell diameter

214. Oligocytemicnormovolemiais observed during

1. dehydration

2. hypoxia

3. renal failure

4. erythremia

5. massive hemolysis of red blood cells. +

215. Polycythemic hypervolemia is observed during

1. erythremia +

2. renal failure

3. dehydration

4. anemia

5. blood loss.

216. Under what conditions is simple hypovolemia observed?

A) 30-40 minutes after acute blood loss +

B) 24 hours after acute moderate blood loss

C) in case of burn shock

D) overheatingofthebody

217. Under what conditions is polycythemic hypovolemia observed?

A) extensive burns +

B) overheating of the body +

C) water intoxication

D) Wakez disease (erythremia)

218. What edema is accompanied by oligocytemic hypervolemia?

A) cardiac

B) hepatic

C) nephritic (renal) +

D) allergic

E) cachectic

219. Indicate the processes that have adaptive significance for the body in the coming minutes and hours after acute blood loss:

A) decrease in venous return of blood

B) peripheral vasoconstriction +

C) centralization of blood circulation +

D) tissue hypoperfusion

E) hyperventilation +

220. In what cases does normocytemic hypervolemia occur?

A) when transfusing a large amount of blood +

B) patients with kidney disease

C) patients with erythremia

D) during intravenousinjection of saline

E) during intravenousinjection of blood substitutes

221. Relative erythrocytosis is observed during:

1. anemia

2. indomitable vomiting +

3. kidney disease

4. chronic hypoxia

5. erythremia.

222. Poikilocytosis of red blood cells is

1. a change in the average diameter of red blood cells

2. a change in the shape of red blood cells +

3. red blood cells with basophilic puncture

4. red blood cells with Jolly bodies

5. red blood cells with Cape rings.

223. An erythrocyte anisocytosis is

1. red blood cells with pathological inclusions

2. target red blood cells

3. red blood cell hyperchromia

4. oval red blood cells

5. the presence in the blood of red blood cells of various sizes. +

224. The blood picture in acute posthemorrhagic anemia at 4-5 days is characterized by:

a) an increase in polychromatophiles; b) an increase in reticulocytes

c) the appearance of megaloblasts; d) the development of neutrophilic

leukocytosis with a nuclear shift to the left; e) the appearance of microspherocytes

1. a, c, e

2. a, b, d +

3. c, d

4. d, e

 5. a, b, c, d, e

225. Hereditary hemolytic anemia by the type of membranopathy include:

1. microspherocytosis +

2. anemia due to deficiency of glucose-6-phosphate dehydrogenase

3. a-thalassemia

4. sickle cell anemia

5. b- thalassemia

226. The development of sickle cell anemia is associated with

1. a genetic defect in the protein-lipid structure of the red blood cell membrane

2. destroy in nucleotide metabolism

3. violation of the synthesis rate of globin beta-chains

4. blocking the oxidation reaction of glucose-6-phosphate in the pentose-phosphate cycle

5. substitution of glutamic acid in the beta chain of globin with valine. +

227. For hemolytic anemia is characteristic

A) megaloblastic type of hematopoiesis

B) fatty regeneration of red bone marrow

C) shortening the life expectancy of red blood cells +

D) iron deficiency in the body

E) increased osmotic resistance of red blood cells

228. Chronic blood loss leads to the development of:

A) Iron deficiency anemia +

B) Vitamin B12-deficient anemia.

C) hemolytic anemia

D) Fanconi Anemia

E) Microspherocytic anemia of Minkowski-Shoffar

229. Iron deficiency anemia is characterized by

1. reticulocytosis

2. hypochromia +

3. megaloblastic type of hematopoiesis

4. hemosiderosis

5. hyperbilirubinemia

230. During iron deficiency anemia in the peripheral blood we can observe:

A) Reticulocytosis.

B) Hyperchromia of red blood cells.

C) Microcytes. +

D) Neutrophilic leukocytosis with a shift to the left.

E) Sickle-shaped red blood cells.

231. The megaloblastic type of hematopoiesis is characteristic of

1. iron deficiency anemia

2. aplastic anemia

3. Vitamin B-12 deficiency anemia +

4. posthemorrhagic anemia

5. hemolytic anemia

232. Pantocytopenia is

1. decrease in the numberof red blood cells in peripheral blood

2. decrease in the number of white blood cells in peripheral blood

3. decrease in the number of platelet count in peripheral blood

4. decrease in the number of red blood cells, white blood cells and platelets in the peripheral blood +

5. an increase in blood of all uniform elements.

233. The patient has helminthic invasion with a wide ribbon. In the analysis: hemoglobin - 95 gm / dl, red blood cells - 2.8 x 1012 / l, reticulocytes - 0.5%, megalocytes and megaloblasts were detected. Determine the nature of anemia

1. iron deficiency anemia

2. hemolytic anemia

3. Vitamin B-12 - deficiency anemia +

4. sidereal anemia

5. hypoplastic anemia

234. What anemia is characterized by red blood cell microcytosis?

A) iron deficiency anemia +

B) hemolytic anemia of Minkowski-Shoffar +

C) acute posthemorrhagic anemia

D) chronic posthemorrhagic anemia +

E) hypoplastic anemia

235. What anemia is characterized by severe red blood cell hypochromia?

A) chronic posthemorrhagic anemia +

B) hypoplastic anemia

C) acute posthemorrhagic anemia

D) diphyllobotriasis anemia

E) thalassemia +

236. In what anemia is a megaloblastic type of hematopoiesis observed?

A) α-thalassemia

B) folic acid deficiency anemia +

C) anemia associated with gastrectomy

D) aplastic anemia

E) diphyllobotriasis-related anemia +

237. Indicate what hematological parameters are characteristic for acute hemolytic anemia:

A) marked red blood cell hypochromia

B) the appearance of megaloblasts

C) severe reticulocytosis +

D) anisocytosis and poikilocytosis +

E) marked neutrophilic leukocytosis with a nuclear shift to the left +

238. What hematological parameters are most characteristic for hypoplastic anemia?

A) red blood cell hypochromia

B) neutrophilia

C) red blood cell normochromia +

D) relative lymphocytosis +

E) neutropenia +

239. What is not typical for folic acid deficiency anemia:

A) red blood cell hyperchromia

B) leukopenia and thrombocytopenia

C) atrophic glossitis

D) a high percentage of ineffective erythropoiesis

E) symptoms of funicular myelosis +

240. What causes can cause the development of B12-deficient anemia?

A) radiation sickness

B) subtotal gastrectomy +

C) ileum resection +

D) jejunum resection +

E) defillobotriosis +

241. Intravascular hemolysis of red blood cells most often occurs with:

A) erythrocytopathy

B) hemoglobinosis

C) incompatible blood transfusion +

D) the action of hemolytic poisons +

242. Indicate anemia, which is characterized by megaloblastic type of hematopoiesis:

A) posthemorrhagic

B) hereditary hemolytic

C) Addison-Birmer anemia +

D) anemia due to defillobotriosis +

E) folic acid deficiency anemia +

243. Which of the following anemia is attributed to hemoglobinopathies?

A) hereditary microspherocytic

B) sickle cell +

C) paroxysmal nocturnal hemoglobinuria

D) Addison-Birmer anemia

E) thalassemia +

244. To thethe factors most often contributing to the development of iron deficiency anemia we canadd:

A) acute massive blood loss

B) chronic blood loss +

C) achlorhydric states +

D) radiation

E) protracted enteritis +

245. Intravascular hemolysis is characteristic of:

A) ABO system conflict +

B) sickle cell anemia

C) sepsis +

D) poisoning with acetic acid +

E) hereditary microspherocytic anemia

245. The degenerative changes in white blood cells include:

a) toxogenic granularity; b) anisocytosis; c) Heinz bodies;

d) hypersegmentation of nuclei; e) Jolly bodies and Cape rings.

1. a, b, c

2. a, c, d

3. a, b, d +

4. c, d, e

5. a, b, e

246. Lymphocytosis is observed during:

a) purulent inflammation; b) acute posthemorrhagic anemia

c) tuberculosis; d) helminthic invasion; e) brucellosis.

1. a, b

2. b, c

3. d, e

4. c, e +

5. a, d

247. The increase in blood levels of stab and the appearance of metamyelocytes are called

1. nuclear shift to the right

2. degenerative nuclear shift to the left

3. hyperregenerative nuclear shift to the left

4. regenerative nuclear shift to the left +

5. agranulocytosis

248. The classic clinical manifestation of agranulocytosis is

1. peptic ulcer

2. ulcerative necrotic tonsillitis +

3. trophic skin ulcers

4. duodenal ulcer

5. ulcerative conjunctivitis.

249. The consequences of pronounced prolonged leukopenia include:

a) increased phagocytosis; b) violation of immunity;

c) decrease in anti-blastoma resistance

d) generalization of infections; e) Strengthening regeneration processes

A) a, b, e

B) d, e

C) b, c, d +

D) a, e

E) c, d, e

250. What hemogram changes are characteristic for chronic lymphocytic leukemia?

A) the appearance of myelocytes in the blood

B) the appearance of pro-lymphocytes in the blood +

C) the appearance of Botkin-Gumprecht shadows in the blood smear +

D) relative lymphocytosis

E) anemia +

251. Under what conditions is neutrophilic leukocytosis with regenerative nuclear shift to the left observed?

A) myogenic leukocytosis

B) digestive leukocytosis

C) croupous pneumonia +

D) appendicitis

A) acute myocardial infarction +

252. Pathological neutrophilic leukocytosis is observed during:

A) furunculosis +

B) pregnancy

C) otitis media +

D) feeling of fear

E) myocardial infarction +

253. What changes in peripheral blood are observed during agranulocytosis?

A) a significant decrease of neutrophils in blood +

B) neutrophilia

C) aneosinophilia +

D) absolute lymphocytosis

E) relativelymphocytosis +

254. Leukemia is characterized by: a) hyperplasia of the erythroid sprout of the red bone marrow; b) bleeding from the gums, nose, intestines; c) infectious complications; d) the appearance of leukemic infiltrates in the tissues; e) reduction of blast cells in the bone marrow.

1. a, b, e

2. b, c, d +

3. c, d, e

4. a, c, d

5. a, b, c

255. During leucosis, the manifestations of tumor progression include transformation

1. leucosisinto a leukemoid reaction

2. leucosisfrom monoclonal to polyclonal +

3. normoblastic type of hematopoiesis into megaloblastic type

4. hematopoietic cells into fat cells

5. hematopoietic cells into hormone-producing cells

256. Leukemic "; gaping"; (hiatus leukaemikus) is

1. the absence of basophils and eosinophils in the leukocyte formula

2. the appearance of a significant number of myeloblasts in the leukocyte formula

3. the absence of intermediate forms in the presence of blast and mature forms of leukocytes +

4. increase in mature forms of white blood cells

5. the absence of blast forms of white blood cells.

257. A large number of blast cells in the blood is characteristic of

A) acute leucosis+

B) chronic leucosis

C) leukocytosis

D) leukopenia

E) leukemoid reaction

258. Up to 90% of mature lymphocytes and single lymphoblasts in the leukocyte formula are observed with

1. acute myeloid leukemia

2. chronic myeloid leukemia

3. acute lymphocytic leukemia

4. chronic lymphocytic leukemia +

5. monocytic leukemia.

259. The presence of all forms of maturation of neutrophilic granulocytes in the blood (from myeloblasts to mature neutrophils) is characteristic of:

A) Acute myeloid leukemia.

B) Acute lymphoblastic leukemia.

C) Monocyticdicosis.

D) Chronic lymphocytic leukemia.

E) Chronic myeloid leukemia. +

260. Hemorrhagic syndrome with leukemia due to

1. leukopenia

2. erythrocytopenia

3. hemolysis of red blood cells

4. thrombocytopenia +

5. lack of antithrombin.

261. For leukemoid reactions of a neutrophilic type characteristic

1. basophilic-eosinophilic association

2. hyperregenerative shift of the leukocyte formula to the left +

3. the appearance in the blood of plasma cells

4. cell atypism of granulocytes

5. pancytopenia

262. The total number of leukocytes is 70 x 10x109 / L; myeloblasts - 1%;

promyelocytes - 3%; myelocytes - 8%; metamyelocytes - 8%;

stab neutrophils - 15%; segmented neutrophils -36%; basophils - 2%; eosinophils -8%; monocytes - 3%; lymphocytes -16%. The leukogram indicates -

1. acute lymphoblastic leukemia;

2. acute myeloid leukemia;

3. chronic myelocytic leukemia; +

4. chronic monocytic leukemia;

5. chronic lymphocytic leukemia.

263. The total number of leukocytes is 80 x 109 / L; myeloblasts - 58%; promyelocytes - 0%; myelocytes - 0%; metamyelocytes - 0%; stab neutrophils - 3%; segmented neutrophils - 30%; basophils - 0%; eosinophils –0.5%; monocytes - 0.5%; lymphocytes - 8%. Such leukogram is typical for

A) chronic monocytic leukemia

B) chronic lymphocytic leukemia

C) chronic myelocytic leukemia

D) acute lymphoblastic leukemia

E) acute myeloid leukemia +

264. What type of leukemia is more common in childhood?

A) chronic myelogenous leukemia

B) chronic lymphocytic leukemia

C) acute lymphoblastic leukemia +

265. Indicate what changes in hematological parameters are characteristic of chronic myeloid leukemia?

A) blood lymphoblasts

B) blood myeloblasts +

C) in the blood myelocytes and metamyelocytes +

D) absolute neutrophilia +

E) relative lymphopenia +

266. Hemorrhagic syndrome in the form of hematomas occurs with

1. thrombocytopenia

2. thrombocytopathy

3. hemophilia +

4. vasopathies

5. thrombophilia.

267. Punctate hemorrhages and bleeding from the mucous membranes

characteristic for:

a) hemophilia A; b) thrombocytopenia; c) hypofibrinogenemia

d) hypovitaminosis K; e) thrombocytopathy.

1. a, c

2.b, e +

3. c, d

4. a, b

5. d, e

268. For hemophilia A is characteristic:

a) sex-related inheritance; b) autosomal recessive inheritance type;

c) deficiency of YIII factor; d) deficiency IX; e) Petechiah; f) hemarthrosis; g) violation of the formation of active prothrombinase

A) b, d, e

B) a, c, f, g +

C) a, b, d, e

D) a, c, e, f

E) b, d

269. Hemophilia B is caused by a deficiency of:

A) Platelets.

B) Antithrombin-Sh.

C) Calcium

D) VIII plasma coagulation factor.

E) IX plasma coagulation factor. +

270. Coagulopathies with a violation of the third phase of blood coagulation are observed with:

A) Excess antithrombin III.

B) Hypofibrinogenemia. +

C) Deficiency of coagulation factorVIII.

D) Deficiency of coagulation factor IX.

E) Deficiency of coagulation factor XI.

271. Increased bleeding due to qualitative inferiority and platelet dysfunction is observed with

A) thrombocytosis

B) thrombocytopenia

C) thrombocytopathy +

D) thrombosis

E) prethrombotic conditions

272. Thrombotic syndrome occurs when:

A) Reducing the activity of procoagulants.

B) Hyperprothrombinemia +.

C) Thrombocytopenia

D) Thrombocytopathy.

E) Deficiency of von Willebrand factor.

273. Deficiency of antithrombin-III leads to the development of:

A) Thrombotic syndrome +.

B) Hemorrhagic diathesis.

C) Hemophilia.

E) Teleangiectasia.

E) Thrombocytopathy.

274. Thrombotic syndrome occurs during:

a) increased activity of procoagulants; b) damage to the vascular wall;

c) thrombocytosis; d) thrombocytopenia; e) antithrombin III deficiency

A) b, c

B) a, b, c, d, e

C) a, b, c, e +

D) c, d

E) d, e

275. Hypercoagulationduring massive tissue damage is associated

1. with a decrease in the activity of physiological anticoagulants

2. with the appearance of pathological anticoagulants

3. entry into the blood of activated X11 coagulation factor and tissue thromboplastin +

4. with a decrease in the adhesive-aggregation function of platelets

5. with activation of the fibrinolysis system

276. Hypercoagulationduring DIC syndrome is due to:

a) activation of "external" or "internal" blood coagulation mechanism

b) the entry into the blood of a large amount of tissue thromboplastin

c) activation of fibrinolysis and anticoagulants

d) coagulopathy of consumption;

e) thrombocytopenia.

1. a, b +

2. a, c

3. a, d

4. a, e

5. b, c

277. Thromboresistance of a vascular wall is caused by:

A) extraction of tissue thromboplastin

B) synthesis of tissue plasminogen activator +

C) activation of the anticoagulant system +

D) synthesis of prostacyclin (PGI2) +

E) synthesis of von Willebrand factor

278. Activation and adhesion of platelets contribute to:

A) increased synthesis of von Willebrand factor +

B) a decrease in the concentration of calcium ions in the cytoplasm of platelets

C) exposure of collagen fibers in case of vascular damage +

D) expression on the platelet membrane of von Willebrand factor receptors +

279. Vascular-platelet hemostasis may be impaired due to:

A) decrease in platelet count +

B) platelet dysfunction +

C) hereditary vasopathy +

D) deficiency of factor YIII

E) deficiency of von Willebrand factor +

280. The thrombus lysis is carried out by:

A) plasmin +

B) antithrombin III

C) heparin

281. The development of hemorrhagic syndrome may be a consequence of:

A) deficiency of procoagulants +

B) increasing the activity of the fibrinolysis system +

C) increasing the concentration of fibrinolysis inhibitors

D) decrease in platelet count +

E) impaired functional properties of platelets +

282. Indicate the changes characteristic of von Willebrand disease:

A) an increase in the duration of capillary bleeding +

B) prolongation of coagulation time +

C) von Willebrand factor deficiency +

D) a violation of the synthesis of factor YIII

E) a decrease in the procoagulant activity of factor YIII +

283. The following reasons may lead to the development of thrombotic syndrome:

A) deficiency of antithrombin III +

B) deficiency of tissue plasminogen activator +

C) increased synthesis of fibrinolysis inhibitors +

D) increased prostacyclin synthesis

E) deficiency of protein C +

284. Disorder of coagulation hemostasis is characteristic of the following diseases:

A) cirrhosis of the liver +

B) thrombocytopathy

C) hemophilia +

D) hemorrhagic vasculitis

285. Enhanced bleeding in DIC syndrome is due to:

A) activation of the plasminogen system +

C) increased consumption of procoagulants +

D) thrombocytopenia of intake +

E) increased secretion of tissue thromboplastin

286. The following factors play a significant role in the pathogenesis of DIC syndrome:

A) disseminated microthrombosis +

B) massive damage to body tissues +

C) decreased tissue thromboplastin production

D) activation of platelet aggregation +

E) decreased activity of the fibrinolytic system

287. The pathogenetic therapy of thrombosis includes the following principles:

A) normalization of hemodynamics +

B) the appointment of antiplatelet agents +

C) a decrease in the activity of the plasminogen system

D) the appointment of anticoagulants +

E) normalization of the rheological properties of blood +

288. Indicate pathological conditions and diseases accompanied by hypocoagulation:

A) chronic obstructive jaundice +

B) acute hemolytic anemia

C) hypertension

D) hyperlipidemia

E) atherosclerosis

289. Indicate the factors that cause platelet adhesion and aggregation:

A) endothelial damage +

B) excess prostacyclin

C) an increase in blood concentration of ADP +

D) platelet degranulation +

E) exposure of the subendothelial collagen layer +

300. Indicate the causes of DIC:

A) Crush syndrome +

B) acute leukemia +

C) deficiency of vitamin K

D) sepsis +

E) shock +

301. During hemophilia A, the following will change:

A) duration of bleeding

B) spontaneous platelet aggregation

C) coagulation time +

D) thrombin time

E) the amount of retraction of the clot

302. Hemorrhagic manifestations in DIC syndrome are mainly due to:

A) depletion of fibrinogen reserves +

B) depletion of antithrombin III

C) thrombocytopenia +

D) inhibition of fibrinolysis and proteolysis

E) activation of fibrinolysis +

303. Compensatory-adaptive reactions in acute blood loss include:

a) spasm of peripheral vessels; b) bradycardia; c) centralization of blood circulation

d) the exit of blood from the depot; e) red blood cell aggregation and extravasation of the liquid part of the blood in the tissue.

1. a, b

2. a, c, d +

3. b, c, e

4. a, b, c

5. a, d, e

304. The cause of acute right ventricular failure may be

1. aortic insufficiency

2. mitral valve insufficiency

3. aortic stenosis

4. pulmonary stenosis +

5. mitral stenosis

305. The cause of right ventricular failure may be:

A) Aortic valve insufficiency.

B) Mitral valve insufficiency.

C) Coarctation of the aorta.

D) Arterial hypertension of the pulmonary circulation. +

E) Arterial hypertension of the pulmonary circulation

306. One of the signs of right ventricular failure is

1. pallor

2. attacks of suffocation

3. hemoptysis

4. pulmonary edema

5. cyanosis of the skin and visible mucous membranes, ascites +

307. An overload form of heart failure develops during:

a) decrease in blood volume; b) myocardial ischemia; c) myocarditis;

d) heart defects; e) hypervolemia

A) a, b, c

B) c, d, e

C) d, e +

D) b, c

E) a, d

308.Long-term adaptation of heart function is provided by:

A) tachycardia

B) myocardial hypertrophy +

C) heterometric reduction mechanism

D) homeometric reduction mechanism

E) myogenicdilatation

309. The stage of compensation for heart failure is characterized by:

a) tonogenic dilatation; b) tachycardia; c) myocardial hypertrophy;

d) myogenic dilatation; e) increase in residual blood in the cavities of the heart

A) b, d

B) a, b, c +

C) a, b, e

D) c, d

E) d, e

310. The emergency stage of heart hyperfunction according to F.Z. Meersonis characterized by:

1. hyperfunction of non-hypertrophic myocardium +

2. hyperfunction of hypertrophic myocardium

3. normalization of energy production per unit mass of mikard

4. proliferation of connective tissue

5. a decrease in protein synthesis per unit of muscle mass

311. The cause of relative coronary insufficiency is

1. infectious myocarditis

2. metabolic disturbances in the myocardium

3. coronary arteriosclerosis

4. coronary artery thromboembolism

5. hyperproduction of adrenaline. +

312. The characteristic disorders of metabolic processes in cardiomyocytes with ischemia include

1. decrease in the formation of ATP +

2. increase in ATP

3. potassium accumulation

4. metabolic alkalosis

5. hypohydration

313. Ionic imbalance in damaged cardiomyocytes is manifested by:

a) increased intracellular sodium concentration; b) an increase in intracellular calcium concentration; c) increase in potassium concentration; d) a decrease in the concentration of sodium; e) a decrease in intracellular potassium concentration

A) a, b, e +

B) b, c, d

C) a, c

D) b, c

E) e

314. Re-entry mechanism with arrhythmias is

1. in the re-entry of the pulse into the area of the conduction system and contractile myocardium with its subsequent reduction; +

2. in slowing down the process of spontaneous diastolic depolarization of the cells of the sinus-atrial node;

3. in fluctuations in the speed of slow spontaneous spontaneous diastolic depolarization of pacemaker cells;

4. in the migration of a pacemaker of the first order;

5. in hyperpolarization in diastole.

315. How does intracardiac hemodynamics change with myogenic dilatation of the ventricles of the heart?
A) the rate of systolic expulsion of blood from the ventriclesincreases
B) diastolic blood volume in the cavity of the ventriclesincreases +
C) the final systolic blood volume in the cavity of the ventriclesincreases +
D) blood pressure in the right atrium and the mouth of the vena cava decreases
E) stroke output of the heart decreases +

316. Indicate the signs characteristic of right ventricular failure:

A) the tendency to develop ascites +

B) swelling of the jugular veins +

C) swelling of the lower extremities +

D) hepatomegaly +

E) pulmonaryedema

317. What type of heart failure can cause pulmonary edema?

A) right ventricular

B) left ventricular +

C) total +

318. Coronaryinsufficiencymayresultfrom:

A) stenotic coronary sclerosis +

A) accumulation of adenosine in the myocardium

C) paroxysmal tachycardia +

D) coronary artery spasm +

E) activation of β-adrenergic receptors of coronary vessels

319. Indicate the consequences of a prolonged attack of paroxysmal ventricular tachycardia:

A) increased cardiac output

C) decrease in cardiac output +

C) decrease in coronary blood flow +

D) increased systolic blood pressure

E) reduction of impact emission +

320. Violation of which of these functions of the heart can lead to cardiac arrhythmias?

A) automatism +

B) excitability +

C) conductivity +

D) contractility

321. Which of the following characterize sinus tachycardia?

A) heart rate reaches 90-180 / min +

B) heart rate exceeds 200 / min

C) occurs during physical exertion +

D) occurs with heart failure +

E) characterized by pronounced changes in the shape of P wave

322. Which of the following items correspond to sinus bradycardia?

A) the automatism of the sinus node is reduced +

B) heart rate less than 40 / min

C) the rate of depolarization of the CA-node cell membrane is reduced +

D) the interval PQ is reduced

E) P wave is usually deformed

323. Atrial extrasystole is characterized by the following ECG signs:

A) the presence of the P wave in front of an extraordinary ventricular complex +

B) deformation of the P wave extrasystoles +

C) reduction and deformation of the ventricular complex

D) incomplete compensatory pause +

324. Atrioventricular block I degree is characterized by:

A) increasing from complex to complex by lengthening the PQ interval

B) stable elongation of the interval PQ more than 0.20 s +

C) Periodic prolapse of the ventricular complexes (QRS)

D) complete dissociation of the atrial and ventricular complex

325. The re-entry wave re-entry mechanism may cause the following arrhythmias.

A) atrial fibrillation +

B) paroxysmal tachycardia +

C) extrasystole +

D) atrio-ventricular block

326. Indicate the changes in biochemical blood parameters characteristic of acute myocardial infarction:

A) a decrease in the activity of lactate dehydrogenase (LDH)

B) increased activity of creatine phosphokinase (CPK) +

C) increase in troponin +

D) a decrease in the content of lactic acid

E) increased activity of lactate dehydrogenase (LDH) +

327. Heart failure is characterized by:

A) a decrease in myocardial contractility +

B) as a rule, decrease in stroke volume +

C) usually a decrease in cardiac output +

D) a decrease in residual systolic blood volume

E) dilatation of heart cavities +

328. Overload of cardiomyocytes Ca2 + in heart failure leads to:
A) uncoupling of oxidation and phosphorylation in mitochondria +
B) activation of Ca2 + -dependent phospholipases and damage to sarcolemma +
C) intensification of lipid peroxidation +
D) impaired relaxation of myofibrils +
E) increase the strength and speed of myocardial contraction

329. In case of myocardial ischemia, the following is not observed:

A) oxidative phosphorylation activity decreases

B) glycolysis is intensified

C) lactic acid accumulates

D) ATP reserves are rapidly depleted

E) increases the concentration of creatine phosphate +

330. Indicate urgent cardiac mechanisms for compensating hemodynamic disturbances during heart failure:

A) bradycardia

B) tachycardia +

C) homeometric mechanism +

D) the Frank-Starling heterometric mechanism +

E) myocardialhypertrophy

331. The substances withdepressor activity include

A) adrenaline

B) angiotensin-II

C) aldosterone

D) prostaglandins of group E and A +

E) leukotrienesC4andD4

332. The substances withdepressor activity include

A) nitric oxide +

B) Angiotensin II.

C) Aldosterone

D) Endothelin.

E) Adrenaline.

333. \_\_\_\_\_\_\_\_ has pressor activity

A) kallikrein

B) endothelin- I +

C) Prostaglandin E

D) nitric oxide

E) atrial natriuretic hormone

334. Secondary arterial hypertension is a symptom of:

A) Chronic adrenal insufficiency.

B) gastric ulcer.

C) Primary aldosteronism. +

D) Hypocorticism.

E) Intestinal autointoxication.

335. Secondary arterial hypertension is a symptom of:

A) Itsenko-Cushing's Disease +

B) gastric ulcer.

C) Chronic adrenal insufficiency.

D) Hypocorticism.

E) Intestinal autointoxication.

336. Secondary arterial hypertension is a symptom of:

a) chronic adrenal insufficiency; b) peptic ulcer of the stomach;

c) primary aldosteronism; d) Itsenko-Cushing's disease (hypercortisolism);

e) intestinal auto-toxicity

A) a, b

B) b, c

C) c, d +

D) d, e

E) a, b, c, d, e

337. In the pathogenesis of primary arterial hypertension \_\_\_\_\_\_\_\_\_\_\_\_\_are involved:

a) persistent increased excitability and hyperergy of higher sympathetic

nerve centers; b) prolonged re-excitation of the emotional centers;

c) a decrease in the inhibitory effect of the cerebral cortex on the pressor

centers; d) hereditary deficiency of smooth muscle membrane ion pumps

vascular cells; e) insufficiency of the adrenal cortex

A) a

B) a, b

C) a, e

D) a, b, c, d +

E) a, b, c, e

338. A genetic defect in cell membranes in hypertension leads:

1. to increase the calcium content in the cytoplasm of cells; +

2. to increase the electrical potential of the cell membrane;

3. to increase the rate of reuptake of mediators by nerve endings;

4. to suppress the ATPase activity of myosin;

5. to reduce the time of action of mediators on the vascular wall.

339. For the period of stabilization of primary hypertension is characteristic:

A) Decreased endothelin production.

B) Increased renin secretion by the kidneys. +

C) Activation of the kallikrein-kinin system.

D) Increased production of natriuretic hormone.

E) Increased kidney production of prostaglandins E1 and E2.

340. For the period of stabilization of hypertension is characteristic

1. reduced production of endothelin;

2. reduced production of nitric oxide; +

3. activation of the kallikrein-kinin system;

4. increased production of natriuretic hormone;

5. increased kidney production of prostaglandins E1 and E2 ..

341. Orthostatic collapse occurs during:

A) massive blood loss

B) pancreatic crush

C) rapid decrease in oxygen in the inhaled air

D) intestinal infections

E) a sharp transition from horizontal to vertical +

342. Symptomatic hypotension is observed during:

1. anemia +

2. pheochromocytoma

3. hyperaldosteronism

4. acute diffuse glomerulonephritis

5. Itsenko-Cushing's disease

343. In the pathogenesis of sinus bradycardia, the following matters:

1. retardation of the spontaneous depolarization of the membrane of cardiomyocytes +

2. acceleration of spontaneous depolarization of the membrane of cardiomyocytes

3. the appearance of damage currents in the myocardium

4. lengthening the period of refractoriness of cardiomyocytes

5. re-entrymechanism.

344. In the pathogenesis of sinus (respiratory) arrhythmia, the following matters:

1. the formation of an ectopic focus of impulse

2. fluctuations in vagus tone +

3. destroy of the excitation from the atria to the ventricles

4. re-entrymechanism.

5. destroy of the processes of excitation.

345. Transverse blockade of the heart is

1. destroy of the conduct of excitation on the right leg of the bundle of Hiss

2. destroy of the excitation on the left leg of the bundle of Hiss

3. destroy of the impulse from the atria to the ventricles through the atrioventricular node +

4. destroy of the conduct of excitation in the atria

5. destroy of the excitation of Purkinje fibers.

346. The number of endogenous substances that contribute to lowering blood pressure by lowering peripheral vascular resistance include:

A) catecholamines

B) + bradykinin

C) angiotensin II

D) + adenosine

E) + NO

347. Experimentalmethodsformodelinghypertensioninclude:

A) + bilateral transection of the depressor nerves

B) ischemia of both adrenal glands

C) + removal of one kidney and compression of the renal artery of another

D) electrical irritation of the depressor nerves

E) + reproductionofneurosis

348. The etiological factors of primary hypertension can be:

A) + frequent psycho-emotional overstrain

B) + hereditary defects of Na+and Ca++- membrane ion pumps

C) stenotic atherosclerosis of the renal arteries

D) adrenal hyperplasia

349. An insufficiency of external respiration is accompanied by

A) an increase in the partial pressure of oxygen (рО2) and

carbon dioxide (рО2) in the blood

B) an increase in рО2and a decrease in рО2in the blood

C) a decrease in рО2and рО2in the blood

D) a decrease in рО2and an increase in рО2in the blood +

E) an increase in рО2and normal рО2in the blood

350. An obstructive type of hypoventilation develops during:

a) a decrease in the total lumen of the bronchi; b) restriction of lung expansion during breathing; c) reduction of the pulmonary surface; d) violation of patency of the airways; e) inhibition of the function of the respiratory center

1. a, b, c

2. a, d +

3. b, c

4. d, e

5. a, d, e

351. An obstructive type of pulmonary hypoventilation is observed during:

a) bronchial and bronchial spasm; b) thickening of the bronchial mucosa; c) impaired respiratory muscle function; d) gland laryngeal edema; e) a decrease in the respiratory surface of the lungs

A) a, b, e

B) a, b, c

C) a, b, d +

D) d, e

E) c, e

352. A restrictive type of hypoventilation develops during:

a) a decrease in the total lumen of the bronchi; b) restriction of lung expansion during breathing; c) reduction of the pulmonary surface; d) violation of patency of the airways; e) bronchial spasm

1. a, b, c

2. a, d

3. b, c +

4. d, e

5. a, d, e

353. A restrictive type of pulmonary hypoventilation occurs during:

a) swelling of the larynx; b) hypersecretion of the bronchial mucosa; c) pulmonary edema;

d) pneumonia; e) pleurisy

A) a, c

B) b, d

C) c, d, e +

D) a, e

E) a

354. A perfusion form of respiratory failure develops during:

a) shocked; b) embolism of the branches of the pulmonary artery; c) weakening of the contractile function of the heart; d) dehydration

A) a, b, c, d +

B) c, d

C) a, b, c

D) d

E) a, d

355. Shortness of breath is

1. frequent deep breathing

2. frequent shallow breathing

3. rare deep breathing

4. rare shallow breathing

5. feeling of lack of air +

356. Bradypnea is observed during:

A. a sharp increase in blood pressure;

B. depression of the respiratory center; c) pleurisy; d) hypoxemia; e) pneumonia

A) a, c

B) a, b +

C) c, d

D) d, e

E) e

357. Tachypnea is observed during:

a) drug poisoning; b) pneumonia; c) swelling of the larynx;

d) fever; e) increase in blood pressure;

A) a

B) d, e

C) b, c

D) a, b

E) b, d +

358. \_\_\_\_\_ breathing is intermittent.

1. tissue

2. dissociated

3. Kussmaul

4. Gasping

5. Chain-Stokes +

359.\_\_\_\_\_\_\_\_\_\_\_\_ is called periodic breathing

1. breathing with a changed ratio between inhalation and exhalation

2. alternation of periods of respiration with periods of apnea +

3. rapid breathing

4. breath with varying amplitude

5. respiratoryarrest.

360. In the pathogenesis of periodic respiration, it matters:

1. decreased sensitivity of the respiratory center to CO2 +

2. increased sensitivity of the respiratory center to CO2

3. excitation of the respiratory center

4. continuous stimulation of inspiratory neurons of the respiratory center

5. reduction of the respiratory surface of the lungs.

361. Biot'srespiration is
1. alternation of apnea with respiratory movements that increase in depth, then decrease
2. alternation of apnea with respiratory movements of the same frequency and depth +
3. deep, rare breathing movements
4. deep, frequent breathing movements
5. gradually fading respiratory movements

362. The Cheyne–Stokes respiration is

1. alternation of apnea with respiratory movements that increase in depth, then decrease +

2. alternation of apnea with respiratory movements of the same frequency and depth

3. deep, rare breathing movements

4. deep, frequent breathing movements

5. gradually fading respiratory movements

363. The terminalrespiration includes

1. Biot’srespiration

2. Cheyne–Stokes respiration

3. externalrespiration

4. dissociatedrespiration

5. Gasping +

364. Asphyxia is

1. respiratory arrest

2. difficulty and lengthening the breath

3. difficulty and prolongation of exhalation

4. lung hyperventilation

5. acute respiratory failure. +

365. The first stage of asphyxia is characterized by: a) increased blood pressure; b) tachycardia;

c) inspiratory dyspnea; d) lowering the tone of the sympathetic nervous system;

e) breathing reduction.

1. b, c, d

2. a, b, c +

3. c, d, e

4. a, c, e

5. b, d, e

366. The second stage of asphyxia is characterized by:

a) increased blood pressure; b) bradycardia; c) expiratory dyspnea; d) increased tone of the parasympathetic nervous system; e) excitation of the respiratory center.

1. a, b, c

2. a, c, d

3. b, c, d +

4. c, d, e

5. a, d, e

367. In the pathogenesis of disorders in the body during asphyxia, the following matters:

a) hypercapnia b) hypoxemia; c) acidosis; d) dypocapnia e) alkalosis

A) a, b, e

B) c, d

C) a, b, c +

D) d, e

E) e

368. The main pathogenetic factor in the development of pulmonary edema is

1. lower vascular permeability

2. lower lymphatic drainage

3. increased colloidal osmotic pressure of plasma

4. increased aldosterone production

5. increase in hydrostatic pressure in the pulmonary capillaries +

369. What type of breathing is characteristic of croupous pneumonia?

A) frequent deep breathing (hyperpnea)

B) deep rare breathing

C) Biot'srespiration

D) + frequent shallow breathing (polypnoea)

E) Kussmaulbreathing

370. In what diseases do lung ventilation disorders in most cases develop in a restrictive manner?

A) + intercostal myositis

B) + pneumonia

C) + bilateral bilateral pneumothorax

D) + dry pleurisy

E) + lung atelectasis

371. Terminal types of respiration include:

A) + Kussmaul breathing

B) + apneystic breathing

C) polypnoea

D) bradypnea

E) + gas-breathing

372. In the pathogenesis of stenotic respiration, the main role is played by:

A) a decrease in the excitability of the respiratory center

B) increased irritability of the respiratory center

C) acceleration of the Hering–Breuer reflex

D) + delay of the Hering–Breuer reflex

373. Respiratory dyspnea is observed under following pathological conditions:

A) + I stage of asphyxiation

B) pulmonary emphysema

C) + laryngeal edema

D) asthma attacks

E) + tracheal stenosis

374. Experimental dyspnea is observed under following pathological conditions:

A) stage I asphyxia

B) + emphysema

C) laryngeal edema

D) + asthma attacks

E) trachealstenosis

375. What complex changes in the gas composition of the blood occurs during alveolar hypoventilation?

A) hypoxemia, hypocapnia, acidosis

B) hypoxemia, hypocapnia, alkalosis

C) + hypoxemia, hypercapnia, acidosis

D) hypoxemia, hypercapnia, alkalosis

376. Give a characteristic of breathing during expiratory dyspnea:

A) constant amplitude

B) shallow breathing

C) difficulty and elongation of inhale

D) + difficult and prolonged exhalation

E) difficulty in inhaling and exhaling

377. In what cases does the respiratory surface of the lungs decrease?

A) with increased muscle work

B) + with pneumothorax

C) + for croupous pneumonia

D) with significant blood loss

E) + with lung atelectasis

378. The signs of liver failure include: a) an increase in the content of ammonia in the blood; b) hypoproteinemia; c) a decrease in the activity of ALT and AST in the blood

d) bleeding; e) dehydration.

1. a, b, d +

2. b, c, d

3. a, b, c, d, e

4. d, e

5. c, d, e

379. Destroy of protein metabolism in liver failure is characterized by:

a) hyperproteinemia; b) hypoproteinemia; c) hyperazotemia; d) hypoprothrombinemia; e)hyperaminacidemia

A) a, c

B) b, c, d, e +

C) a, d, e

D) a, b, c, d, e

380. Hypocoagulation in case of liver failure is caused by: a) impaired absorption of vitamin K; b) a violation of the synthesis of fibrinogen; c) a violation of the synthesis of prothrombin; d) a violation of the synthesis of antithrombin III; e) a violation of the synthesis of protein C and S

1. a, d, e

2. b, c, d

3. a, b, c +

4. a, b, c, d

5. d, e

381. Ekka's direct fistula is used to study

1. metabolic function of the liver

2. urea-educational function of the liver

3. barrier, detoxification function of the liver +

4. biliary function

5. biliary function of the liver.

382. In the pathogenesis of hepatic coma, it matters: a) insufficiency of the neutralizing function of the liver; b) metabolic acidosis; c) insufficiency of the urea-forming function of the liver; d) increase in blood direct bilirubin; e) hyperglycemia

A) a, b, c +

B) a, d

C) d, e

D) b, e

E) a, d, e

383. Hyperbilirubinemia corresponds to the level of total bilirubin in the blood:

1. 1-3 umol/l (μm / L)

2.4-5 umol/l (μm / L)

3.5-6 umol/l (μm / L)

4.8-20 umol/l (μm / L)

5.30-40 umol/l (μm / L) +

384. The causes of suprahepatic jaundice include: a) the effect of hemolytic poisons;

b) rhesus incompatibility between the mother and fetus; c) transfusion of incompatible blood; d) posthemorrhagic anemia; у) dyskinesia of the gallbladder.

1. d, e

2. b, c, d

3. a, b, c +

4. b, c, d

5. c, d, e

385. The leading link in the pathogenesis of suprahepatic jaundice is:

1. body dehydration

2. heart failure

3. insulin deficiency

4. violation of the outflow of bile

5. enhanced hemolysis of red blood cells. +

386. For hemolytic jaundice is characteristic

1. increase in free bilirubin in the blood +

2. increased bound bilirubin in the blood

3. discoloration of feces

4. digestion in the intestines.

5. allocation of free bilirubin with urine.

387. Hemolytic jaundice is characterized by an increase of\_\_\_\_\_\_\_ in blood

1. indirect bilirubin +

2. direct bilirubin

3. urobilin

4. sterkobilina.

5. Bile acids.

388. The leading link in the pathogenesis of subhepatic jaundice is

1. damage to hepatocytes

2. sialolithiasis

3. urolithiasis

4. enhanced red blood cell hemolysis

5. violation of the outflow of bile +.

389. During obstructive jaundice we can observe: a) hypotension; b) bilirubinuria; c) Acholia;

d) skin itching; e) tachycardia

A) a, b, c, d +

B) a, b, c, d, e

C) a, c, d

D) b, e

E) a, e

400. For obstructive jaundice is characteristic: a) hyperbilirubinemia; b) increased activity of ALT and AST; c) cholemia; d) bilirubinuria; e) light urine.

1. a, b

2. a, b, c

3. a, b, c, d

4. a, c, d +

5. c, d, e

401. Cholemia is characterized by: a) a decrease in blood pressure; b) the appearance of skin itching; c) bradycardia; d) tachycardia; e) hypertension.

1. a, b, c +

2. b, c, e

3. a, d

4. d, e

5. b, d

402. The presence in the blood of bile salts causes

1. increase in blood pressure

2. bradycardia +

3. tachycardia

4. fever

5. shortness of breath

403. \_\_\_\_\_\_\_ is typical for acholia:

1. activation of emulsification of fat in the intestine

2. hypervitaminosis of fat-soluble vitamins

3. increased fat breakdown and absorption

4. steatorrhea +

5. darkstoolcolor.

404. Стеаторея при механической желтухе связана с

А) нарушением всасывания жиров в кишечнике+

B) активацией панкреатической липазы

C) наследственной ферментопатией

D) активацией липолиза

E) развитием первичной мальабсорбции

405. Bleeding with prolonged subhepatic jaundice occurs due to

1. destroy in heparin synthesis

2. disturbances in the synthesis of prothrombin due to a decrease in the absorption of vitamin K +

3. destroy in synthesis of fibrinolysis inhibitors

4. destroy in the synthesis of antithrombin

5. destroy in synthesis of kallikrein

406. Parenchymal jaundice is characterized by: a) urobilinogenemia; b) a decrease in the activity of AST and ALT in the blood; c) cholemia; d) hyperglycemia; e) hypocholia.

1. a, c, d, e

2. b, c, d

3. a, c, e +

4. a, b, c

5. c, d, e

407. Hyperbilirubinemia, accompanied by an increase in the level of both free and bound bilirubin in the blood, is noted at:

1. cholecystitis

2. sickle cell anemia

3. Botkin's disease +

4. cholangitis

5. insulinoma

408. Digestive insufficiency is accompanied by: a) positive nitrogen balance; b) negative nitrogen balance; c) hypovitaminosis; d) depletion of the body; e) a decrease in nonspecific resistance

A) b, c, d, e +

B) a

C) e

D) a, c, d, e

E) a, d, e

409. Dysphagia is

1. lack of appetite

2. feeling full

3. excessive appetite

4. increased food intake

5. violation of swallowing. +

410. The consequences of hypersalivation include

1. plaque in the language

2. hypokalemia +

3. multiple dental caries

4. the development of inflammatory processes in the oral cavity.

5. xerostomia

411. Hyposalivation leads to

1. neutralization of gastric juice

2. maceration of the skin around the mouth

3. hypokalemia

4. ptyalism (salivation)

5. xerostomia. +

412. Indomitable vomiting contributes

1. development of metabolic acidosis

2. the development of non-gas alkalosis +

3. increase blood pressure

4. hyperglycemia

5. hyperchlorhydria

413. Hyperchlorhydria of gastric juice is accompanied by: a) pyloric spasm; b) stagnation in the stomach of food contents; c) diarrhea; d) belching sour, sometimes vomiting; e) gaping gatekeeper.

A) a, b, c, d, e

B) a, b, d +

C) c, d, e

D) e

E) d, e

414. Achlorhydria is accompanied by: a) a decrease in the bactericidal effect of hydrochloric acid; b) the occurrence of fermentation in the stomach; c) difficulty in evacuating food masses from the stomach; d) spasm of the pylorus; e) rapid neutralization of food masses from the stomach by duodenal contents.

A) a, b, c

B) c, d

C) a, b, e +

D) d, e

E) c

415. Hyposecretion of gastric juice is accompanied by:

1. heartburn

2. constipation

3. diarrhea +

4. hypervolemia

5. flatulence

416. The following contribute to the development of ulcerative lesions of the gastric mucosa:

a) hyperchlorhydria of gastric juice; b) the presence of Helicobacter pylori; c) the presence of a mucous barrier; d) an increase in pepsin activity; e) a decrease in the synthesis of DNA, RNA, protein

A) b, c

B) a, b, d, e +

C) b, e

D) a, b, c, d, e

E) a, c, d, e

417. Choose the correct statement:

1. Low acidity of gastric juice contributes to the occurrence of gastric ulcer

2. Rapid regeneration of the gastric mucosa contributes to the occurrence of gastric ulcer

3. In the experiment, a stomach ulcer can be simulated with chronic irritation of the vagus +

4. Nncrease in pepsin activity and acidity of gastric juice contributes to the occurrence of gastric ulcer +

5. Peptic ulcer always develops in the presence of Helicobacter pylori

418. Malabsorption is called

A) a syndrome due to increased intake of bile into the intestines

41 intestines +

C) syndrome due to increased intestinal motility

D) fasting syndrome

E) a syndrome due to a violation of the endocrine function of the pancreas

419. Malabsorption syndrome is characterized by malabsorption of nutrients.

1. in the stomach;

2. in the duodenum 12;

3. in the small intestine; +

4. in the colon;

5. in the rectum.

420. Steatorrhea develops with

1. hypersecretion of gastric juice

2. acholias +

3. high activity of intestinal lipases

4. difficulty in intestinal motility

5. excessive intake of protein.

421. Mechanical intestinal obstruction occurs

1. with spasm or paralysis of the intestinal muscles;

2. with thrombosis of the vessels of the intestinal wall;

3. with paresis of the intestinal muscles;

4. with tumors and helminthiases of the intestine; +

5. with paralysis of the vessels of the intestinal wall.

422. The pathogenesis of intestinal auto-toxicity is due to toxic effects of:

1. products of rotting proteins in the intestines and biogenic amines (cadaverin, putrescine) +

2. indirect bilirubin

3. ketone bodies

4. bile acids

5. direct bilirubin

423. Indicate the factors involved in the pathogenesis of heartburn:

A) + gaping cardia

B) + gastroesophageal reflux

C) + spasm and anti-motility of the esophagus

D) lowering the acidity of gastric juice

E) + increase in the content of organic acids in the stomach

424. Indicate the possible causes of the development of gastric hypersecretion:

A) + excessive parasympathetic stimulation of the stomach

B) excessive sympathetic stimulation of the stomach

C) + increased production and excretion of gastrin

D) deficiency of gastrin production

E) + increased formation and release of histamine in the wall of the stomach

425. For hyperchlorhydria and increased secretory function of the gastric glands, it is characteristic:

A) + tendency to constipation

B) + increased pepsin activity

C) gaping gatekeeper

D) + pyloric spasm

E) decreased gastric motility

426. Indicate the possible causes of the development of gastric hyposecretion:

A) excessive parasympathetic stimulation of the stomach

B) + excessive sympathetic stimulation of the stomach

C) + decreased production and excretion of gastrin

D) increased production and release of histamine

E) decreased secretin production and secretion

427. The following factors may participate in the development of gastric ulcer and duodenal ulcer:

A) + infection

B) + excess glucocorticoid production

C) + increased tone of parasympathetic nerves

D) increased mucus formation

E) + increased tone of the sympathetic nerves

428. Iatrogenic "steroid" gastrointestinal ulcers are caused by:

A) insulin

B) adrenaline

C) mineralocorticoids

D) + glucocorticoids

E) sex hormones

429. What vitamins absorption will significantly worsen with acholia?

A) + Vitamin A

B) Vitamin B1

C) + Vitamin D

D) + Vitamin E

E) + Vitamin K

430. Indicate the reasons for the disorder of membrane digestion:

A) + diseases of the liver, pancreas, leading to disturbance of digestive digestion

B) + violation of the structure and ultrastructure of the wall of the small intestine

C) + violation of the enzyme layer on the surface of the intestinal wall

D) + violation of the intestinal microflora

E) + impaired motor and excretory functions of the small intestine

431. Indicate the main causes of the development of malabsorption syndrome:

A) + atrophy of the small intestine microvilli

B) + extensive small bowel resection

C) hyperacid gastritis

D) + chronic enteritis

E) + acholia

432. Indicate the manifestations of malabsorption syndrome:

A) + flatulence

B) + diarrhea

C) constipation

D) + weight loss

E) + hypoproteinemia

433. Indicate the factors leading to increased intestinal motility:

A) + Achilles

B) a decrease in the excitability of the center of the vagus nerve

C) + increased excitability of the intestinal wall receptors

D) + inflammation in the intestines (acute enteritis)

E) the continued consumption of fiber-poor foods

434. The pathogenesis of renal glucosuria is associated with

1. an increase in the permeability of the filter membrane of the Bowman-Shumlyansky capsule

2. disorder of the reabsorption of water in the distal renal tubules

3. a decrease in the activity of hexokinase in the epithelium of the renal tubules +

4. hyperglycemia exceeding "renal threshold";

5. disorder of the secretion processes in the renal tubules.

435. Massive proteinuria / up to 40 g/day / is noted during:

1. pyelonephritis

2. acute glomerulonephritis

3. chronic glomerulonephritis

4. nephrotic syndrome +

5. nephrolithiasis

436. The relative density of the final urine duringisostenuria is:

1. 1025-1035
2. 1020-1030
3. 1016-1020
4. 1006-1012
5. 1010-1011.+

437. Prerenal oligo-anuria develops during

1. reducing the production of antidiuretic hormone

2. insulin hypoproduction

3. arterial hypertension

4. excessive fluid intake

5. shock. +

438. Prerenal polyuria develops during

1. prostate adenoma

2. reducing the production of antidiuretic hormone +

3. body dehydration

4. acute arterial hypotension

5. urinary tract stricture.

439. The prerenal factors causing acute renal failure include

1. acute glomerulonephritis

2. severe lead poisoning

3. mercuric poisoning

4. crush syndrome +

440. The structural basis for the development of acute renal failure is

1. amyloidosis;

2. lipoid nephrosis;

3. tubular necrosis; +

4. nephrosclerosis;

5. sarcoidosis

441. Proteinuria in acute glomerulonephritis is explained

1. glomerular hyperperfusion

2. decreased renal blood flow

3. a decrease in the electric charge of the capillary wall of the glomerulus

4. an increase in the permeability of capillaries of the glomerulus +

5. reduced glomerular capillary permeability

442. The pathogenesis of chronic renal failure is characterized by the following sequence: a) slowly progressive loss of renal function; b) the death of most of the nephrons; c) the development of nephrosclerosis; d) reduction in filtration; e) oligoanuria; f) uremia

1. a, b, c, d, e, f

2. b, c, a, d, e, f +

3. b, c, a, f, e, d

4. c, b, d, a, e, f

5. d, a, c, e, f, b

443. The biochemical composition of blood in uremia is characterized by:

a) hyperglycemia; b) hyperazotemia; c) Hypersulfatemia; d) Hyperphosphatemia;

e) hypercalcemia

A) b, c, d +

B) a, e

C) c, d, e

D) a, b, c, d, e

E) a, b

444. The pathogenetic factor of hypertension in acute glomerulonephritis is

1. the inclusion of renin-aldosterone-vasopressin mechanism +

2. increased production of prostaglandins

3. increase the production of kinins

4. hypervolemia +

5. lowering the sensitivity of vascular receptors to pressor influences

445. Which of the following indicators characterize renal tubular dysfunction?

A) + aminoaciduria

B) the presence in the urine of leached red blood cells

C) reduced creatinine clearance

D) + isostenuria

E) + decrease in the secretion of H+and ammonium ions

446. Which of the following factors does not play a significant role in the pathogenesis of oligoanuria in acute renal failure?

A) increased renin synthesis by the kidneys

B) tubular obstruction of the renal tubules

C) + increased glomerular filtration

D) decrease in effective filtration pressure

E) edema of the renal parenchyma

447. Select the mechanisms of glucosuria:

A) increased filtration pressure in the glomeruli of the kidneys

B) + blocking of phosphorylation enzymes in the epithelium

C) + structural damage to the proximal tubules

D) increased permeability of the glomerular capillaries of the kidneys

E) + excess blood glucose (more than 9 mmol /L)

448. Which of the following disorders of homeostasis are characteristic of the oligoanuric stage of acute renal failure?

A) + excretory acidosis

B) + increase in the concentration of urea in the blood

C) + an increase in the concentration of creatinine in the blood

D) hypovolemia

E) + hyperkalemia

449. For the azotemic stage of chronic renal failure is not characteristic:

A) hypostenuria

B) increased urea in the blood

C) + metabolic alkalosis

D) anemia

E) increased creatinine in the blood

450. Indicate the main mechanisms contributing to the formation of renal hypertension:

A) + activation of the renin-angiotensin system

B) activation of kallikrein-kinin system

C) + retention of sodium ions and leading in the body

D) decreased renin synthesis

E) + decreased synthesis of renal prostaglandins

451. Select the reasons that reduce glomerular filtration of the kidneys:

A) spasm of abducent glomerular arterioles

B) + spasm of glomerular arterioles

C) + decrease in filtration area

D) hypoproteinemia

E) + hyperproteinemia

452. The uremic stage of chronic renal failure is characterized by:

A) + azotemia

B) + acidosis

C) + decrease in creatinine clearance

D) + gastroenteritis phenomena

E) + development of pleurisy and pericarditis

453. The indicators characterizing the violation of glomerular filtration are:

A) leukocyturia

B) + azotemia

C) aminaciduria

D) + oligouria

E) + decrease in creatinine clearance

454. What disorders can be caused by hereditary defects of the enzymes of the tubular apparatus of the kidneys?

A) hemoglobinuria

B) + aminoaciduria

C) + hyperphosphaturia

D) + glucosuria

E) + Fanconi syndrome

455. Indicate the pathological components of urine of renal origin:

A) + leached red blood cells

B) indirect bilirubin

C) + protein in large quantities

D) bile acids

E) + cylinders

456. What can underlie renal acidosis?

A) increased ammoniogenesis

B) + decrease in tubular secretion of Н ions

C) excessive reabsorption of sodium ions

D) + decrease in ammonia secretion

E) + violation of the reabsorption of HCO3-

457. Lack of which hormones can cause polyuria?

A) chondrotropic hormone

B) + vasopressin

C) adrenaline

D) oxytocin

E) + insulin

458. Indicate diuresis disorders typical forpollakiuria:

A) monotonous diuresis with a density of urine of 1,010 – 1,012

B) an increase in the daily amount of urine

C) + rapid (more than 6 times a day) urination

D) a decrease in the daily amount of urine

E) cessation of urination (less than 50 ml / day)

459. Indicate diuresis disorders typical foranuria:

A) monotonous diuresis with a density of urine of 1,010 – 1,012

B) an increase in the daily amount of urine

C) frequent (over 6 times a day) urination

D) a decrease in the daily amount of urine

E) + cessation of urination (less than 50 ml / day)

460. Indicate diuresis disorders typical forisostenuria:

A) + monotonous diuresis with a density of urine of 1,010 – 1,012

B) an increase in the daily amount of urine

C) frequent (over 6 times a day) urination

D) a decrease in the daily amount of urine

E) cessation of urination (less than 50 ml / day)

461. Indicate the correct variant of transhypophysial regulation of endocrine glands:

A) The cerebral cortex -the peripheral gland.

B) The cerebral cortex - hypothalamus-pituitary gland - peripheral gland. +

C) The cerebral cortex - the hypothalamus - nerve conductors - peripheral glands.

D) The cerebral cortex - pituitary gland - hypothalamus - peripheral gland.

E) Subcortical centers - nerve conduits - peripheral gland.

462. Transhypophysial regulation is fundamental to: a) pancreas b) thyroid gland; c) gonads; d) parathyroid glands; e) adrenal cortex

A) a, b

B) d, e

C) b, c, e +

D) a, b, c, d, e

E) a, e

463. The basis for the violation of the feedback mechanism is

1. decreased sensitivity of the hypothalamic centers perceiving fluctuations in hormone concentrations in the blood +

2. decrease in liberin production

3. increased statin production

4. increase the production of hormones of the adenohypophysis.

5. decrease in statin production

464. The metabolism of hormones is being destroyed in diseasesof:

A) liver +

B) spleen

C) hearts

D) lung

E) nervous system

466. Hypoproteinemia is accompanied by:

1. an increase in the fraction of free hormones and an increase in their effects +

2. an increase in the fraction of free hormones and a decrease in their effects

3. a decrease in the fraction of free hormones and a decrease in their effects

4. a decrease in the fraction of free hormones and an increase in their effects

5. distortion of the effects of hormones.

467. The peripheral mechanisms of hormone disruption are:

a) violation of the binding of hormones to blood plasma proteins; b) hormone receptor blockade; c) destroy of the production of releasing hormones of the hypothalamus;

d) inactivation of circulating hormone; e) violation of the synthesis of the hormone;

A) a, b, d +

B) c, b, e

C) a, e

D) a, c

E) c, e

468. \_\_\_\_\_\_develops with eosinophilic pituitary adenoma during the growth period of the body

A) acromegaly;

B) gigantism; +

C) dysplasia;

D) pituitary dwarfism;

E) Itsenko-Cushing's disease.

469. Basophilic adenohypophysis adenoma leads to the development of

1. gigantism

2. acromegaly

3. hyperthyroidism

4. Itsenko-Cushing disease +

5. Simmonds disease

470. The development of diabetes insipidus is due to

1. hypersecretion of vasopressin

2. hyposecretion of vasopressin +

3. hypersecretion of aldosterone

4. hyposecretion of aldosterone

5. the presence of insulin antagonists in the blood

471. Damage of neurohypophysis is accompanied by parasecretion of:

A) thyroid stimulating hormone

B) growth hormone

C) adrenocorticotropic hormone

D) prolactin

E) vasopressin +

472. Deficiency of antidiuretic hormone is characterized by:

1. polyuria, hypostenuria, polydipsia +

2. polyuria, hyperstenuria, polydipsia

3. oliguria, edema

4. glucosuria, polyuria, polydipsia

5. oliguria, proteinuria, hematuria

473. The change in the secretion of oxytocin plays a role in the pathogenesis of:

A) birth disorders +

B) carbohydrate metabolism disorders in diabetes

C) circadian rhythm disturbances; "wake - sleep";

D) myxedema development

E) diffuse toxic goiter

474. Excitation of the central nervous system, increased blood pressure, hyperglycemia, osteoporosis, lymphocytolysis are observed with overproduction of:

A) melanostimulating hormones

B) parathyroid hormones

C) sex hormones

D) glucocorticosteroids +

E) catecholamines

475. Mostly the cause of Addison's disease is:

1. hypertrophy of the adrenal glands

2. adrenal atrophy +

3. pituitary tumor

4. autoimmune thyroiditis

5. pineal gland hyperplasia

476. Conn's syndrome (primary aldosteronism) is detected by:

1. sodium loss and potassium retention

2. sodium retention and potassium loss +

3. oliguria

4. hypotension

5. the accumulation of hydrogen ions

477. A sign of a hormonally active tumor of the adenohypophysis is:

A) + acromegaly

B) + gigantism

C) + hypercorticism

D) secondary aldosteronism

E) primary aldosteronism (Conn syndrome)

478. In what cases increases the secretion of aldosterone?

A) + decrease in circulating blood volume

B) an increase in circulating blood volume

C) + hyponatremia and hyperkalemia

D) hypernatremia and hypokalemia

E) + increased activity of the renin-angiotensin system

479. Hyperproduction of aldosterone leads to:

A) + increase in sodium in the blood

B) an increase in potassium in the blood

C) + decrease in potassium in the blood

D) a decrease in calcium in the blood

480. In case of insulin deficiency, hyperglycemia is caused by:

A) + decrease in glucose utilization by tissues

B) + increase in glucose production in the liver (gluconeogenesis)

C) increased lipogenesis

D) all of the listed factors

481. Select hormones of the anterior pituitary gland, excessive production of which can contribute to the development of diabetes mellitus:

A) prolactin

B) + chondrotropic hormone

C) + adrenocorticotropic hormone

D) follicle-stimulating hormone (FSH)

482. The resistance of the nervous system is determined by:

a) antisystems; b) the blood-brain barrier; c) a low degree of intracellular regeneration; d) safety braking; e) low activity of glial cells

1. a, b, d +

2. a, e

3. b, c, d

4. a, b, c, d

5. a, d, e

483. The antinociceptive system is

1. pathologically enhanced excitation generator

2. analgesic system +

3. pain enhancing system

4. a system for the formation of epicritical pain

5. a system for the formation of protopathic pain

484. The formation of pathologically enhanced excitation generators in the central nervous system is

1. etiological damage factor

2. the cellular mechanism of the pathological process

3. the mechanism of intercellular pathology +

4. еhe mechanism of pathology of intersystem relations

5. general pathological process

485. A pathologically enhanced excitation generator occurs when

1. amplification of the flow of efferent pulses

2. nerve cell parabiosis

3. amplification of the flow of afferent impulses +

4. spread excitement in the cerebral cortex

5. the development of neurogenic dystrophy

486. Neurogenic dystrophy is

1. autoimmune brain damage

2. violation of the synthesis of endorphins

3. metabolic disorders in tissues with a disorder of their innervation +

4. disruption of higher nervous activity

5. parabiosis of nerve cells

487. The loss of sensitivity is called

A) ataxia

B) alexia

C) atony

D) asthenia

E) anesthesia +

488. Paralysis of all limbs is called

A) paraplegia

B) tetraplegia +

C) hemiplegia

D) paresis

E) physical inactivity

489. The response of the acute phase is characterized by a decrease of:

1. albumin; +

2. fibrinogen;

3. C-reactive protein;

4. gammaglobulins;

5. serumamyloidA

490. The most important mediator of the acute phase response is

1. histamine;

2. leukotriene C4;

3. platelet activation factor;

4. lymph node permeability factor;

5. interleukin-1. +

491. Under the influence of stressors develops: a) thymus involution; b) adrenal hypertrophy; c) hypoplasia of lymphoid tissue; d) ulcerative lesions of the stomach and duodenum; e) hyperplasia of lymphoid tissue

A) a, b, c, d +

B) a, b, d, e

C) a, b, c, d, e

D) e

E) a, c, e

492. The mechanisms for implementing the general adaptation syndrome are associated with overproduction of:

1. antidiuretic hormone

2. oxytocin

3. androgen

4. glucocorticoids +

5. melatonin.

493. The stress-limiting system includes:

A) Sympathoadrenal system.

B) The hypothalamus.

C) The pituitary gland.

D) The adrenal glands.

E) GABAergic system. +

494. Extreme conditions include

1. preagony

2. agony

3. clinical death

4. biological death

5. coma +

496. Most often, cardiogenic shock develops along with:

A) myocardial infarction +

B) arterial hypotension

C) pericarditis

D) myocardiopathies

E) damage to the tricuspid valve

497. Hemodynamic changes in the erectile stage of shock:

a. increasing of circulating blood volume

b. increased cardiac output

c. increased blood flow

d. fall in blood pressure

e. increase in blood pressure

1. a, b, c, e +

2. a, b, c, d

3. a, b, c, d, e

4. a, d

 5. c, d

498. The stage of resistance of the general adaptation syndrome is characterized by:

A) + increased secretion of glucocorticoids

B) decreased secretion of glucocorticoids

C) + increased gluconeogenesis

D) attenuation of gluconeogenesis

E) + neutrophilic leukocytosis

499.Stress-limiting systems do not include:

A) + complement system

B) the system of opioid peptides

C) serotonergic system

D) GABAergic system

E) antioxidant systems

500. What the effects are connected with excess glucocorticoid production?

A) increasing of phagocytic activity of white blood cells

B) + inhibition of the phagocytic activity of leukocytes

C) increasing of the body's ability to produce antibodies

D) + decrease in the ability of the body to produce antibodies

E) + inhibition of cellular immune responses

501. At what stage of GAS (general adaptation syndrome) does adrenal cortical hypertrophy develop?

A) at the stage of anxiety

B) + at the stage of resistance

C) at the stage of exhaustion

502. Indicate the characteristic changes in the blood cells in stage I and II of GAS (general adaptation syndrome):

A) eosinophilia

B) + neutrophilia

C) + eosinopenia

D) + lymphopenia

E) neutropenia

503. What changes are characteristic for I stage of GAS (general adaptation syndrome)?

A) + reduction of the size of the thymus

B) depletion of adrenal cortex function

C) + activation of the adrenal cortex

D) lymph node size increasing

E) + lymph node size reduction

504. Indicate the correct sequence of development of dysfunctions of the central nervous system in coma:

A) + mental anxiety, confusion, stupor, profound loss of consciousness

B) profound loss of consciousness, stupor, confusion, mental anxiety

C) confusion, profound loss of consciousness, stupor, mental restlessness

D) mental anxiety, stupor, confusion, profound loss of consciousness

E) profound loss of consciousness, confusion, stupor, mental anxiety