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Перечень итоговых тестовых заданий для студентов 3 курса факультета иностранных студентов по учебной дисциплине «Патофизиология, клиническая патофизиология».

General nosology

1. What questions is considering the general nosology?
a) general laws dysfunction of various systems; diagnosis of human diseases , the pathophysilogy of organs and systems;
+b) understanding of the nature of the disease at different stages of development of medicine, disease classification, forms the origin, development, course and outcome of disease.

2. What is etiology?
+a) teaching about the causes and conditions of the disease;
b) teaching about the causes of diseases;
c) teaching about the set of conditions that cause the development of diseases.

3. Which of the following is the correct definition of the pathogenesis? Pathogenesis - is:
a) a set of mechanisms that occur in the body under the influence of harmful factors in violation of reflex reactions, leading to a change in the biochemical and enzymatic composition of the blood, causing occurrence , course and outcome of the disease;
+b) a set of mechanisms, including in the organism under the influence of action of harmful factors and it manifested in a dynamic and also the time stereotypical deployment of number of physiological, biochemical and morphological responses that determine the occurrence , course and outcome of disease.

4. Specify the most adequate (of these) definition of "disease"?
a) the disease is the amount of abnormal vital functions that take place in the affected one way or another, organs and tissues;
+b) disease - peculiar life process associated with the action on the body of harmful factors , manifested in a complex structural and functional changes that violate the adaptive capabilities and reducing its ability to work and socially useful activities ;
c) the disease - is a complex reaction to the action of the pathogenic agent , a new process of life, characterized by restriction or violation of the regulation of functions and adaptability.

5. Specify periods of illness:
+a) latent, prodromal, the height, the outcome; b) an acute, subacute, chronic.

6. What is the duration of acute illness?
+a) up to 2 weeks; b) up to 4 weeks;
c) up to 6 weeks; d) up to 8 weeks.

7. What is the duration of the course of chronic diseases?
a) more than 2 weeks; +b) more than 4 weeks;
c) more than 6 weeks; d) more than 8 weeks.

8. What do you call a temporary weakening or disappearance of the clinical manifestations of chronic disease?
a) complications; +b) remission;
c) recurrence; d) aggravation.

9. What is the beginning of a new cycle of the same disease after a period of disappearance or reduction of its clinical manifestations?
a) complications; b) remission;
c) recurrence; +d) aggravation.

10. Specify the possible outcomes of the disease:
+a) recovery, death; b) recurrence;
c) remission; d) complication; e) incubation.

11. What is causal factor?

+ a) it is factor (object or phenomenon), which is directly acting on the body, under certain conditions, causing illness and gives her specific features;
b) it is factor (object or phenomenon), which itself does not cause disease, but it affects to occurrence and during of disease.

12. What is condition?

a) it is factor (object or phenomenon), which is directly acting on the body, under certain conditions, causing illness and gives her specific features;
+b) it is factor (object or phenomenon), which itself does not cause disease, but it affects to occurrence and during of disease.

13. Specify the basic properties of the causal factor in the occurrence of the disease:

+ a) the need for the occurrence of the disease; + b) the indispensability;
c) broad interchangeability; d) direct effect on the body;
e) the indirect action; + f) the definition of the specific features of the disease.

14. Is it right to consider the pathological process as inadequate response to the stimulus?

a) yes; +b) no.

15. Is it right to consider the pathological reaction as inadequate response to the stimulus?

+a) yes; b) no.

16. Is it right to consider an inadequate response to the stimulus as pathological state?

a) yes; +b) no.

17. Specify examples of pathological processes:

a) acquired valvular heart defect; + b) fever in typhoid fever;
c) furunculosis; d) myocardial infarction.

18. Under what conditions is a pathogenic effect of electric current on the body will be more significant?

+ a) when passing an electrical current through the moist skin;
b) when passing electrical current through the skin dry.

19. How changes the body's sensitivity to the damaging effects of electric current in thyrotoxicosis, hyperthermia, blood loss?

+ a) increases; b) decreases.

20. In which case, the pathogenic effect of electric current on the body will be more?

+ a) in the waking state; b) during narcotic sleep.

21. In which case, the effect of electric current on the body will be more significant?

+ a) in case the lesion occurs unexpectedly;
b) in case the is expected effect of the electric current.

22. Which of the following factors is the main link in the pathogenesis of traumatic shock?

a) injury; b) shock; + c) pain.

23. Select manifestations that characterize the erectile phase of shock:

a) the weakening effects of sympathetic-adrenal and pituitary-adrenal systems;
b) hypotension;
+ c) motor and verbal excitation;
+ d) hyperventilation;
+ e) hyperreflexia.

24. Select manifestations that characterize the torpid phase of shock:

+ a) the weakening effects of sympathetic-adrenal and pituitary-adrenal systems;
b) tachycardia, hypertension;
c) motor and verbal excitation;
+ d) decrease in cardiac output;
+ e) depositing blood;
+ f) arterial hypoxemia;
+ g) oliguria.

25. The first 6 hours of acute radiation sickness in adults after a single total irradiation at a dose of 2-6 Gy, is characterized by:

+ a) nausea, vomiting; + b) lymphopenia;
+ c) neutrophilic leukocytosis; d) bleeding;
e) erythema; f) anemia.

26. Sensitivity of the organ (tissue) to ionizing radiation increases:

a) in hypoxia; + b) in deficit of vitamin E;
+ c) in the process of tissue regeneration; d) in the presence of cytotoxic drugs;
+ e) in hyperthermia ; + f) in excess of oxygen.

27. The main target in the cell under the influence of ionizing radiation is:

a) the cytoplasmic membrane; + b) DNA;
c) endoplasmic reticulum; d) ribosomes;
e) mitochondria; f) lysosomes;
g) Golgi complex.

28. Radiation damage to the cells is facilitated by:

+ a) increase of the oxygen content in the blood;
b) decrease of oxygen content in the blood;
+c)deficit of vitamin E;
+ d) high mitotic activity;
e) low mitotic activity.

29. What cells are affected more by the action of ionizing radiation?

a) mature; + b) embryonic; + c) undifferentiated.

30. What is characteristic of the second period of acute radiation sickness?

+ a) beginning of the inhibition of hematopoiesis;
b) severe inhibition of hematopoiesis;
c) bleeding into internal organs;
+d) lack of visible clinical manifestations of the disease;
e) expressed clinical manifestations of the disease.

31. What is characteristic of the third period of acute radiation sickness?

+ a) severe inhibition of hematopoiesis;
b) insignificant inhibition of hematopoiesis;
+ c) bleeding into internal organs;
+d) increased permeability of cell membranes;
e) reduced permeability of cell membranes;
+f) immunosuppression;
g) the immunity does not suffer.

32. Specify the main changes in peripheral blood of the first period of acute radiation sickness:

+a) leukocytosis; + b) lymphopenia;
c) leucopenia; d) lymphocytosis.

33. Specify the main changes in peripheral blood of the second period of acute radiation sickness:

a) leukocytosis; + b) lymphopenia;
+c) leucopenia; d) lymphocytosis.

34. Specify the main changes in peripheral blood of the third period of acute radiation sickness:

a) leukocytosis; + b) leucopenia;
c) polycythemia; + d) anemia;
e) thrombocytosis; +f) thrombocytopenia .

35. Specify the main clinical manifestations of the third period of acute radiation sickness:

+ a) infectious complications; b) radiation shock;
+ c) haemorrhage in the skin; d) excitation of the CNS;
+ e) internal bleeding; f) "X- hangover".

36. Pathogenesis is teaching about

a) the causes and conditions of the development of the disease;
b) the disease;
+ c) the mechanisms of the development of the disease;
d) the reactivity of the organism;
e) heredity of the organism.

37. The initial link of the pathogenesis is

a) secondary damage; b) the aggravation of the disease;
c) the transition to the chronic form; + d) primary damage;
e) the formation of a vicious cycle.

38. The main link of the pathogenesis of the disease is:

a) damage, which leads to appearance a vicious circle;
+ b) damage, underlying most of the manifestations of the disease;
c) the reasons and conditions for the occurrence of the disease;
d) damage that is not reversible.

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

a) transition of the arising primary acute phase to chronic phase with periods of exacerbation and remission;
b) cyclical course of the disease, in which each new cycle differs from the previous by progressive increase in the severity of disorders;

+c) occurrence of a positive feedback between the individual links of pathogenesis, which promote to progress of the disease.

40. Vicious circle in the pathogenesis of the disease means:
a) depletion of compensatory mechanisms, leading to a deterioration;
b) occurrence of any pathological reaction;
c) gradual change of stages of the disease;
+d) worsening of any link of pathogenesis as a result of reaction of the body;
e) sequence of terminal states.

41. Necrosis is
a) the total change in the cytoplasm of damaged cells;
b) the transformation of cells into malignant;
c) genetically programmed cell death;
+d) irreversible damage to the cell;
e) trophic disturbances cells.

42. Specific manifestation of cell damage when cyanide poisoning is
a) denaturation of the protein molecules; b) increased lipid peroxidation;
+c) the blockade of cytochrome oxidase; d) acidosis;
e) uncoupling of oxidation and phosphorylation.

43. Inactivation of cytochrome oxidase is a specific manifestation at
a) the effect of radiation; +b) cyanide poisoning;
c) the effect of high temperature; d) mechanical trauma;
e) the action of antioxidants.

44. The main target in the cell under the influence of ionizing radiation:
a) cytoplasmic membrane; +b) DNA;
c) the sarcoplasmic reticulum; d) the ribosome;
e) mitochondria.

45. Factors which contribute to the radiation damage of the cells:
+a) increase of the oxygen content in the blood;
b) reduction of the oxygen content in the blood;
+c) lack of vitamin E;
+d) high mitotic activity;
e) low mitotic activity.

46. The mechanisms of cell damage:
a) increasing the conjugation of oxidative phosphorylation;
b) increase in the activity of enzymes of the DNA repair;
+c) increase the free radical oxidation of lipids;
+d) resulting action of lysosomal enzymes in hyaloplasm;
+e) oncogene expression.

47. Enzymes of antimutational system of the cell:
+a) restriction enzyme; b) histaminase;
c) hyaluronidase; +d) DNA polymerase;
e) creatine phosphokinase; +f) lipase.

48. The increase in the content of free ionized calcium in the cell is accompanied by:
+a) activation of phospholipase A2;
b) inactivation of phospholipase C;
+c) activation of lipid peroxidation;
d) hyperpolarization of the cytoplasmic membrane;
+e) increase in the K + out of the cell;
+f) overhydration of the cells.

49. The consequences of severe acidosis in ischemic damage of cardiomyocytes:
+a) ↓ Ca ++ - transporting function of the sarcoplasmic reticulum;
b) activation of the Na + / K + - ATPase;
c) inactivation of lysosomal proteases and phospholipases;
+d) activation of lipid peroxidation;
+e) reduction of contractile function of the myofibrils;
f) activation of enzymes of the creatine kinase system.

50. Signs that are typical for apoptosis:
a) chaotic DNA breaks;
+b) DNA cleavage in strictly defined areas;

c) release and activation of lysosomal enzymes;
+d) formation of vacuoles containing the fragments of the nucleus and organelles;
e) hyperhydration of the cells.

51. The consequences of apoptosis:
+a) phagocytosis of fragments of the cells bounded by a membrane;
b) formation of a zone of the plurality of dead and damaged cells;
+c) destruction and removal of a single cell;
d) development of the inflammatory response;
e) autolysis of dead cells.

52. Non-specific manifestations of cell damage are:
+a) damage to the genome; +b) acidosis;
c) alkalosis; +d) sodium accumulation in the cell;
+e) activation of lysosomal enzymes.

53. Reactivity – is
a) the response of the body to a stimulus;
+b) property of the organism as a whole to respond by changes of the vital activity on the impact of the environment;
c) a protective reaction of the organism to the action of pathogenic stimulus;
d) the body's resistance to pathogenic factors;
e) non-specific resistance of the organism.

54. Specific reactivity is a property
+a) of the body to respond to antigenic stimulus;
b) of an organism of this species to respond to environmental influence;
c) of the group of individuals of this species to respond to environmental influence;
d) of the body respond by certain way on the impact of physical factors;
e) of specific organism to respond to environmental influences.

55. An example of a non-specific pathological reactivity is reactivity for
a) allergy; b) immunodeficiency;
c) immunosuppressive conditions; d) immuno-proliferative diseases;
+e) state of shock.

56. An example of a specific physiological reactivity is
a) allergy; b) immunodeficiency states;
c) immunosuppressive condition; d) autoimmune processes;
+e) immunity.

57. Dysergy is called
a) increased the response of an organism to a stimulus;
b) reduced response of an organism to a stimulus;
c) lack of response of an organism to a stimulus;
+d) perverse response to the stimulus;
e) adequacy of the response of an organism to a stimulus.

58. The following assertion is true:
a) the reactivity is not depends from the constitution of the body;
+b) the reactivity depends from the state of nervous and endocrine systems;
c) the reactivity is not depends from the environmental factors;
d) resistance and reactance of an organism do not depend from the state of metabolism;
e) reaction of the organism is not depends from the age and sex.

59. The correct is the statement:
+a) the high reactivity of the organism is not always accompanied by a high resistance;
b) reactivity and resistance occur independently;
c) low reactivity of the organism is always accompanied by a high resistance;
d) low reactivity always promotes resistance to infection.

60. The uniqueness of each individual is determined by:
a) species reactivity; b) group reactivity;
+c) the individual reactivity; d) gender;
e) constitutional peculiarities.

61. Hibernation of animals is an example of
+a) species reactivity; b) group reactivity;
c) the individual reactivity; d) specific reactivity;
e) pathological reactivity.

62. Frogs are more resistant to hypoxia than rats, guinea pigs. This is an example of:
a) group reactivity; +b) species reactivity;
c) the individual reactivity; d) age-reactivity;
e) specific reactivity.

Typical pathological processes

1. Hyperglycemia can be caused by excess:
+a) epinephrine; +b) thyroid hormones;
+c) glucocorticoids; +d) growth hormone;
e) insulin.

2. The main pathogenetic link of hypoglycemic coma:
+a) carbohydrate and energy "starvation" of brain neurons;
b) carbohydrate "starvation" of the myocardium;
c) hypoosmia of the blood;
d) uncompensated ketoacidosis.

3. Complications of long-term diabetes mellitus:
a) immunodeficiency states;
b) acceleration of development of the atherosclerosis;
c) reduction of resistance to infections;
d) reduction of antitumor sustainability;
e) microangiopathy;
f) macroangiopathy;
+g) all answers are correct.

4. The basis of glycogen storage disease type II (Pompe disease) is a genetically determined defect of:
+a) 1,4-glucosidase; b) phosphorylase.

5. Synthesis of glycogen at hepatic failure, pulmonary failure, cardiovascular failure:
a) enhanced; +b) is weakened.

6. What are the changes in metabolism occur in diabetes mellitus?
a) increase in the synthesis of proteins in the body;
+b) inhibition of protein synthesis in the body;
c) increase of the accumulation of glycogen in the liver;
+d) inhibition of the accumulation of glycogen in the liver;
e) amplification of glycolysis, the pentose cycle;
+f) inhibition of glycolysis, the pentose cycle;
+g) the stimulation of lipolysis.

7. Factors causing hypoglycemia:
a) predominance of inhibitory processes in the CNS;
b) restriction of carbohydrate intake with food;
c) reduction of the activity of the sympathetic nervous system;
d) decreased secretion of thyroxine;
e) decrease in the secretion of glucocorticoids;
f) reduction of production of ACTH;
g) increase in insulin production;
+h) all answers are correct.

8. Factors causing hyperglycemia:
a) predominance of excitation in the central nervous system;
b) high intake of carbohydrates with food;
c) increasing the activity of the sympathetic nervous system;
d) increase in the secretion of thyroxine;
e) increase in the secretion of glucocorticoids;
f) increase in ACTH production;
g) decrease in the production of insulin;
+h) all answers are correct.

9. In which of these cases, hypoglycemia develops?
+a) hyperinsulinism; +b) Addison's disease;
+c) Girke's disease; d) thyrotoxicosis;
e) Cushing's disease; +f) renal diabetes;
+g) hypothyroidism.

10. Edema is

a) accumulation of fluid in the serous cavities;
+b) accumulation of fluid in the tissues and interstitial space;
c) increase in the formation of lymph;
d) increasing intravascular fluid;
e) an increase in intracellular fluid.

11. Edema is

a) pathological reaction; +b) pathological process;
c) pathological state; d) nosological form of the disease;
e) preillness.

12. Local pathogenetic factor of the edema is

a) increased oncotic pressure of the blood;
+b) increase of hydrostatic pressure of the blood;
c) decrease of permeability of the vessel wall;
d) lowering hydrostatic pressure of the blood;
e) increased tissue pressure.

13. Pathogenetic factor of the edema is:

a) increased oncotic pressure of the blood;
b) lowering hydrostatic pressure of the blood;
c) decrease of permeability of the vessel wall;
+d) increased osmotic and oncotic pressure in the tissue;
e) decrease in aldosterone secretion.

14. Hypoosmolar dehydration occurs when

+a) diarrhea; b) diabetes mellitus;
c) diabetes insipidus; d) esophageal atresia;
e) water fasting.

15. Hypoosmolar dehydration occurs when

+a) prolonged vomiting; b) diabetes mellitus;
c) diabetes insipidus; d) esophageal atresia;
e) water fasting.

16. Hypoosmolar dehydration is characterized by

a) increase in total body water;
+b) decreased osmotic pressure of the extracellular fluid;
c) reduction of water content in the cells;
d) movement of water from the cells into the extracellular space;
e) agonizing thirst.

17. Dehydration is characterized by:

+a) hemoconcentration; +b) disturbance of microcirculation;
c) increase in central venous pressure; +d) decrease in blood pressure;
+e) reduction of blood volume.

18. Hypernatremia occurs when excessive secretion of

a) sexual hormones; b) thyroid hormones;
c) natriuretic hormone; d) antidiuretic hormone;
+e) aldosterone.

19. Which of the definitions of "arterial hyperemia" is correct?

+a) increasing the blood supply to part of the tissue or organ due to the enhanced inflow of blood to him by dilation of arterioles and capillaries;
b) an increase in blood volume and increased blood pressure.

20. Which of these features are characteristic of arterial hyperemia?

a) redness of organ with a cyanotic shade, blood pressure in the hyperemic area is not changed, decrease in volume of hyperemic area;
+b) tissue redness (bright red color), increased blood pressure in the vessels of hyperemic area, the increase in volume of hyperemic area, pulsation of small blood vessels, increasing the temperature of surface of the integument;
c) blood pressure in the hyperemic area is not changed, decrease in volume of hyperemic area;
d) pulsation of small vessels is terminated.

21. Which of the following types of arterial hyperemia are pathological?

+a) postischemic, inflammatory, neuroparalitical;
b) arterial, arising on the mechanism of the conditioned reflex (paint shame, anger), reflectory that occurs under the action of adequate doses of physical and chemical factors, working.

22. What can be the cause of arterial hyperemia?

a) increased action against the norms of physiological stimuli (sun rays, heat);
b) the action of heat;
c) the action of toxins of microorganisms;
d) the primary lesion of the neuromuscular apparatus of vessels;
e) increasing the sensitivity of vessels to physiological stimuli;
+f) all answers are correct.

23. What can cause venous hyperemia?

a) compression of the veins without damaging the arteries;
b) reduction of the suction effect of the chest;
c) the weakening of the heart;
d) reducing muscle tone of the limb;
e) defective valve apparatus of veins;
+f) all answers are correct.

24. What are the signs of venous hyperemia?

a) blanching of the organ, raise of temperature of the affected area, lowering of blood pressure in the veins of the hyperemic area, pulsation of small vessels;
+b) redness of the organ with a cyanotic shade, lowering the temperature of the affected area, increased blood pressure in the veins to the periphery of the obstacle, increase in volume of the hyperemic area.

25. What are the possible consequences of venous hyperemia?

a) a reactive proliferation of connective tissue;
b) the acceleration of wound healing;
c) disorders of nutrition and function of the organs;
+d) all answers are correct.

26. What can be consequences of ischemia?

+a) infarction, atrophy of parenchymal elements, weakening of metabolism;
b) hemorrhage;
c) enhancing the metabolism.

27. What are the signs typical for ischemia?

a) pulsation of small vessels, cyanosis organ or area of tissue, increasing the temperature of the affected area, increased blood pressure in the veins, lowering blood pressure above obstacle and increase below of obstacle;
+b) lowering the temperature of the affected area, blanching of tissue, volume reduction of ischemic area, increase in blood pressure above the obstacle and fall below obstacle, the feeling of pain and appearance of the paresthesias in the ischemic area.

28. Which of the definitions of ischemia is correct?

+a) ischemia is decrease in blood supply of an area of tissue due to the weakening or stopping of blood flow through the arteries to it;
b) ischemia is a decrease amount of circulating blood;
c) ischemia is reduction of the number of erythrocytes per unit volume of blood.

29. Indicate the main pathogenetic factors of thrombogenesis (by R.Virhov):

+a) the rough surface of the vessel wall, increased activity of the coagulation system and lowering activity of the anticoagulation system of blood, slowdown of the blood flow;
b) the smooth surface of the vessel wall, lowering the activity of coagulation system and increase of the anticoagulation system of blood, acceleration of blood flow.

30. What factors contribute to the activity of the blood coagulation system in thrombosis?

a) reduction of the thrombin concentration in the blood, lowering blood viscosity, reducing the concentration of Ca2 + ;
+b) increasing the concentration of thrombin in the blood, increase in blood viscosity, increased concentration of Ca2 + .

31. What is the adhesion of platelets?

a) the "sticking" of platelets to each other;
+b) settling of platelets on the injured surface of the inner wall of the vessel.

32. Specify that promotes platelet adhesion in the cell phase of thrombogenesis?

+ a) reduction of value of the negative charge of platelets, reducing content of ATP in them, increased content of ADP in them;
b) increase of value of the negative charge of platelets, increased content of ATP in them, decrease content of ADP in them.

33. Specify the initiators of platelet aggregation:

a) thrombin; b) thromboxane A2;
c) prostaglandin H2; d) prostaglandin D2;

+e) all true.

34. Choose adverse consequences of thrombosis:

+a) ischemia, venous hyperemia; b) uremia, erythremia.

35. What is embolism?

+a) circulation in the blood and lymph channels of unusual particles of exogenous and endogenous origin;
b) process of intravital formation of blood clots (embolus) on the inner wall surface of vessels.

36. What types of emboli depending on the final stop of embolus?

a) embolism of the systemic circulation; b) embolism of the pulmonary circulation;
c) embolism of the portal vein; +d) all of the above.

37. What determines the clinical manifestations of embolism of the systemic circulation?

a) the localization of the of embolus;
b) severity of the reflex spasm of surrounding vessels in the pool of vessel, which is occluded by embolus;
c) the type of collateral;
+d) all of the above.

38. Specify the classic clinical triad of portal vein embolism:

+a) ascites, enlargement of superficial veins of the abdominal wall, splenomegaly;
b) hydropericardium, acromegaly.

39. What reaction to the damaging factor is inflammation?

a) general reaction;
+b) local reaction.

40. The causes of aseptic inflammation may be:

a) thrombosis of the veins;
b) tissue necrosis;
c) hemorrhage in the tissue;
d) surgical intervention in aseptic conditions;
e) parenteral administration of sterile foreign protein;
+f) all of the above.

41. The local signs of inflammation are

+a) swelling, redness, dysfunction, pain, increase of local temperature in inflammatory focus;
b) arterial hyperemia, venous hyperemia, stasis;
c) acidosis, hyperospheresia, hyperoncia of inflammatory focus;
d) alteration, poor circulation with exudation, proliferation;
e) leukocytosis, increased erythrocyte sedimentation rate, increase in body temperature.

42. Damage of tissues in the inflammatory focus is called

+a) alteration; b) exudation.

43. The primary alteration in the inflammation occurs as a result of

a) action of inflammatory mediators;
b) physico-chemical changes in inflammatory focus;
+c) damaging effect of flogogenic factor;
d) microcirculatory disorders;
e) metabolic disorders in inflammatory focus.

44. Increase of disintegration of substances in the inflammatory focus associated with

+a) activation of lysosomal enzymes;
b) the activation of mitochondrial enzymes;
c) activation of adenylate cyclase;
d) inhibition of enzymes of anaerobic stage of glycolysis;
e) inhibition of enzymes of lipid peroxidation.

45. Mediators of inflammation of the humoral origin

a) histamine; b) serotonin;
c) prostaglandins; +d) bradykinin;
e) cytokines.

46. Inflammatory mediators of cellular origin is

+a) interleukin-1; b) bradykinin; c) fibrinopeptides.

47. Sequence of changes of blood circulation in inflammatory focus:

+a) short-term ischemia, arterial hyperemia, venous hyperemia, stasis;
b) arterial hyperemia, venous hyperemia, ischemia, stasis;
c) arterial hyperemia, stasis, ischemia, venous hyperemia;
d) ischemia, arterial hyperemia, stasis, venous hyperemia;
e) ischemia, venous hyperemia, arterial hyperemia, stasis.

48. The most short term stage of blood circulation disorders in inflammation is:
a) arterial hyperemia; +b) spasm of arterioles (ischemia);
c) local stop of blood flow; d) venous hyperemia;
e) stasis.

49. Contributes to emigration of leukocytes
+a) positive chemotaxis;
b) reducing oncotic pressure of the blood;
c) acceleration of blood flow;
d) increasing oncotic pressure of the blood;
e) reducing permeability of the vascular wall.

50. Serous fluid:
+a) is characterized by moderate protein content;
+b) is characterized by a low relative density;
c) is characterized by an increased content of cell elements;
+d) typical for inflammation of the serous membranes (chest, heart, joints, etc.);
+e) the most frequently observed during the burn, immune inflammation.

51. Type of exudate during inflammation caused by staphylococcus and streptococcus
a) hemorrhagic; +b) purulent;
c) fibrinous; d) serous;
e) mixed.

52. At an inflammation caused by pyogenic microorganisms is dominated in the composition of exudates
+a) neutrophils; b) eosinophils;
c) basophils, d) monocytes;
e) lymphocytes.

53. The sequence of emigration of leukocytes in acute inflammation:
a) neutrophils, eosinophils, monocytes; +b) neutrophils, monocytes, lymphocytes;
c) monocytes, lymphocytes, neutrophils; d) lymphocytes, monocytes, neutrophils;
e) macrophages, neutrophils, monocytes.

54. Macrophages are
+a) monocytes; b) lymphocytes;
c) neutrophils; d) mast cells;
e) fibroblasts.

55. The local manifestations of inflammation are:
a) fever, leukocytosis, accelerated erythrocyte sedimentation rate;
+b) pain, redness, fever, organ dysfunction;
c) headache, sleep disturbance, loss of appetite;
d) myalgia, ossalgiya;
e) hypoalbuminemia, hypergammaglobulinemia.

56. Indicates to presence of inflammatory process in the body
+a) ESR acceleration; b) leukopenia; c) anemia.

57. What causes to acidosis in inflammatory focus?
+a) release and accumulation of large amounts of acids;
b) accumulation of chloride ions;
c) increase in the content of the polypeptide;
d) accumulation of sodium ions;
e) accumulation of potassium ions.

58. What hormones induce amplification of the inflammatory response?
a) glucocorticoids; +b) mineralocorticoids;
c) thyroxine; d) STH;
e) insulin.

59. Fever is
a) a pathological reaction; +b) a typical pathological process;
c) a pathological state; d) a disease;

60. Mechanisms of physical thermoregulation at a fever is
+a) reduction of heat loss; b) increase in heat production;
c) heat production does not change.

61. Endogenous pyrogens are formed in
a) red blood cells; b) platelets;

+c) leukocytes; d) hepatocytes;
e) parenchymal cells.

62. Producers of endogenous pyrogens are:
+a) macrophages; b) mast cells;
c) plasma cells; +d) neutrophils;
e) erythrocytes.

63. Endogenous pyrogens include
+a) interleukin-1; b) interleukin-4;
+c) interleukin-8; +d) interleukin 6;
+e) tumor necrosis factor; f) endotoxins of microorganisms.

64. Leukocytic pyrogens act on
a) the heat-sensitive peripheral receptors;
b) otoneurons of the spinal cord;
+c) neurons of the preoptic area of the hypothalamus;
d) neuro-conductive path;
e) the spin-cortical path.

65. The missing link of the pathogenesis of increase of body temperature in fever is
Exogenous pyrogens→ phagocytes→ ?→ the thermoregulation center
a) microbial endotoxins; +b) endogenous pyrogens;
c) prostaglandins; d) cyclic nucleotides;
e) lipoproteins.

66. In the first stage of fever is observed
a) increase of the heat production without changing of heat loss;
b) increase of the heat production and heat loss;
+c) reduction of the heat loss and enhancement of the heat production;
d) reduction of the heat production and heat loss;
e) reduction of the heat production and increase of the heat loss.

67. Acute-phase response is characterized by a decrease in
+a) albumin; b) fibrinogen;
c) C-reactive protein; d) gamma-globulin;
e) serum amyloid A.

68. The most important mediator of the acute phase response is
a) histamine; b) leukotriene C4;
c) platelet activating factor; d) factor of permeability of lymph nodes;
+e) IL-1.

69. The following statement is correct
a) the acute phase response - mainly local reaction of the organism to injury;
+b) the acute phase response - the overall reaction of the organism to injury;
c) the all manifestations of the acute phase response always have a exceptionally positive value for the body;
d) acute phase response is always accompanied by a decrease in resistance of the organism;
e) the acute phase response develops only when the body is damaged by mechanical factors.

70. Endogenous chemical carcinogens are
a) polycyclic aromatic hydrocarbons; +b) oxygen free radicals and nitrogen oxide;
c) aminoazocompounds; d) nitrosamines;
e) simple chemical compounds.

71. An agent that enhances the action of carcinogens, but did not cause the development of tumors:
a) procarcinogen; b) oncogene;
c) protooncogene; d) anti-oncogene;
+e) co- carcinogen.

72. The primary, fundamental sign of malignant tumors is
a) cachexia; b) the systemic effects of the tumor on the body;
+c) the invasive growth; d) recurrence;
e) negative effect of Pasteur.

73. Malignant tumors are characterized by
+a) suppression of the mechanisms of apoptosis;
b) strengthening of the mechanisms of apoptosis.

74. Biological features that are typical of malignant tumors
+a) uncontrolled proliferation of cells; +b) loss of "limit" of the division of the Heyflik;
c) expansive growth; +d) loss of contact inhibition;

+e) invasive growth.

75. Simplification of structural and chemical organization, reducing the differentiation of tumor tissue is called

a) dysplasia; b) the increasing complexity;
c) convergence; +d) anaplasia; e) hypertrophy.

76. Specify the correct sequence of stages of the carcinogenesis

+a) initiation, promotion, progression; b) promotion, initiation, progression;
c) progression, initiation, promotion; d) initiation, progression, promotions;
e) promotion, progression, initiation.

77. The initiation stage of carcinogenesis is

a) the qualitative changes of the properties of the tumor cells aside malignization;
b) the appearance of the more malignant clone of the cells;
+c) transformation of the normal cell into the tumor cell;
d) the ability of tumor cells to metastasis;
e) activation mechanisms of antineoplastic resistance of the organism.

78. Leads to malignant transformation of cells:

+a) activation of oncogenes; +b) inhibition of anti-oncogenes;
c) activation of apoptotic genes; +d) formation of oncoproteins;
e) activation of DNA repair systems.

79. Leads to malignant transformation of cells:

+a) conversion of the protooncogene to oncogene; b) activation of anti-oncogenes;
c) inactivation of the genes of anti-apoptosis; d) activation of apoptotic genes;
e) activation of protooncogenes.

80. What features characterize the tumor progression?

+a) increasing anaplasia of the cells;
+b) invasiveness;
+c) infiltrative growth;
d) strengthening of processes of the final differentiation of cells;
e) strengthening of antigenic stimulation of the body by tumor cells.

81. At the shock blood supply is maintained due to the centralization of the blood circulation, primarily in the:

+a) brain and heart; b) intestines and lungs;
c) liver and kidney; d) pancreas and stomach;
e) muscles and bones.

82. Complication, which is not typical for hemorrhagic shock

a) failure of the liver and kidneys; b) adrenal insufficiency;
+c) leukemoid reaction; d) hypoxic coma;
e) disseminated intravascular coagulation.

83. Is it true that collapse, as the shock, is characterized by centralization of circulation?

a) yes; +b) no.

84. The shock occurs because of:

a) massive irritation of interoceptors;
b) massive irritation of exteroreceptors;
c) massive irritation of interoreceptors and exteroreceptors simultaneously;
+d) all right.

85. Select manifestations that characterize the erectile phase of shock:

a) the weakening effects of the sympathetic-adrenal and pituitary-adrenal systems;
b) arterial hypotension;
+c) motor and verbal excitation;
+d) hyperventilation of lungs;
e) decrease in cardiac output;
+f) hyperreflexia.

86. Select the manifestations that characterize the torpid phase of shock:

+a) the weakening effects of the sympathetic-adrenal and pituitary-adrenal systems;
b) tachycardia, arterial hypertension;
c) motor and verbal excitation;
+d) reduction of cardiac output;
+e) depositing blood;
+f) arterial hypoxemia.

87. Causes of coma can be:
a) autointoxication; b) lack of necessary metabolic substrates;
c) exogenous intoxication; d) hypoxia;
e) severe endocrinopathies; f) head injury;
+g) all of the above.
88. In an allergic reaction, in contrast to the immune response, is observed
a) the production of antibodies;
b) the plasmatization of B-lymphocytes;
c) the destruction of the antigen;
+d) damage of the organism's own tissues;
e) increasing the phagocytic activity of macrophages.
89. Which of the following allergens is the most common cause of allergic reactions of type I?
+a) house dust; b) bacterial toxins;
+c) bed micromites; +d) epidermal allergens;
+e) pollen of plants.
90. What organs and tissues can be attributed to the "behind the barrier"?
a) red blood cells; +b) tissue of the eye lens;
+c) testicular tissue; d) kidney tissue;
+e) colloid of the thyroid gland.
91. Haptens acquire antigenic properties only after
a) influence on the immunocompetent cells;
+b) connection with proteins of the organism;
c) connection with bile acids;
d) forming of the paired compounds with sulfuric acid;
e) prior interaction with macrophages.
92. The basis of immunological stage of allergic reactions is
a) formation of the mediators of allergy;
b) degranulation of mast cells;
c) cell response to the action of the mediators of allergy;
+d) the production of antibodies, sensitized T-lymphocytes;
e) reduction of the antibody titer.
93. Pathochemical stage of the allergic reactions is characterized by
a) violation of the microcirculation;
b) spasm of smooth muscle elements;
c) an increase in the permeability of the vessel walls;
+d) release of mediators of allergy;
e) formation of immune complexes.
94. Pathophysiological stage of the allergic reactions is characterized by
a) formation of immune complexes;
b) activation of the biologically active substances;
+c) structural and functional abnormalities in organs and tissues;
d) synthesis of antibodies;
e) forming of the sensitized lymphocytes.
95. The main mediator of allergic reactions of cytotoxic type is
a) histamine; b) bradykinin;
+c) activated complement components; d) lymphotoxins; e) kinins.
96. Damage by immune complexes underlies of development
a) atopic bronchial asthma; +b) glomerulonephritis;
c) graft rejection; d) anaphylactic shock; e) hay fever.
97. T-lymphocytes play a major role in the pathogenesis of allergic reactions of:
a) reaginic type; b) anaphylactic type;
c) immunocomplex type; +d) cell-mediated type;
e) cytotoxic type.
98.Hypoxia is:
+ a) typical pathological process; b) pathological reaction;
c) pathological state; d) the disease;
e) symptom complex.
99. Hypoxia, a developing when lower partial pressure of oxygen in the inspired air, is called:
+ a) exogenous; b) hemic;

c) the circulatory; d) tissue; e) endogenous.

100. Specify the characteristic change in the composition of the blood when exogenous hypobaric hypoxia:

a) hyperglycemia; + b) hypocapnia;
c) hypercapnia; d) reduction of blood lactate; e) hyperproteinemia.

101. For hypobaric form of exogenous hypoxia is characteristic:

+ a) hypocapnia; b) hyperoxemia;
c) high arteriovenous oxygen difference; d) arterialization of the venous blood;
e) reducing the oxygen capacity of the blood.

102. Carbon monoxide poisoning leads to the development of hypoxia:

a) tissue; +b) hemic;
c) circulatory; d) respiratory; e) exogenous.

103. Carbon monoxide poisoning leads to formation of:

a) desoxyhemoglobin; b) methemoglobin;
c) carbohemoglobin; +d) carboxyhemoglobin; e) sulfohemoglobin.

104. The main mechanism of hemic hypoxia is:

a) reduction of arterio-venous oxygen difference;
b) an increase in arterial oxygen saturation;
+c) reduction in blood oxygen capacity;
d) an increase in pCO2 blood;
e) violation of blood flow velocity.

105. Specify the features that are characteristic for the circulatory type of hypoxia:

+a) reducing the flow velocity;
b) reduction of arterio-venous oxygen difference;
c) reduction of the oxygen content in the arterial blood;
+d) an increase in arterio-venous oxygen difference;
+e) acidosis.

106. Specify the reasons of the hypoxia of respiratory type:

a) CO poisoning; +b) emphysema of lungs;
c) nitrate poisoning; d) chronic blood loss; e) hypovitaminosis B12;
+f) decrease in excitability of the respiratory center.

107. Urgent compensatory reactions during hypoxia are:

+ a) the release of deposited blood; + b) tachycardia;
+ c) hyperpnea; d) respiratory muscle hypertrophy;
e) activation of erythropoiesis.

Typical pathological processes of organs and systems

1. Does not refer to primary immunodeficiencies:

+ a) HIV infection;
b) DiGeorge syndrome;
c) Bruton agammaglobulinemia;
d) agammaglobulinemia of Swiss type.

2. Hereditary and congenital immunodeficiencies may be:

+ a) combined: with the defeat of the cell (T) and humoral (B) components of immunity;
+ b) with preferred defects of cellular immunity;
+ c) with preferred defects of antibody production by B-lymphocytes;
d) with defects of phagocytosis by microphages (neutro-, baso-, eosinophils);
+ e) with defects of the system of mononuclear phagocytes;
f) with defects of the production of chemotactic factors;
g) with insufficiency of the humoral factors of nonspecific protection.

3. Secondary immunodeficiencies may occur when:

a) extensive burns;
b) X-ray irradiation, corticosteroid therapy, thymectomy;
c) leukemia;
d) viral, bacterial, fungal, protozoal infections and helminthoses;
e) malignant tumors;
+ f) all of the above.

4. What cells of the immune system cells are the main target for the AIDS virus:

a) B-lymphocytes; b) T-killers;
+ c) T-helper cells; d) T-suppressors.

5. What organs and tissues can be attributed to "behind a barrier"?
a) red blood cells; + b) lens of the eye;
+ c) colloid of the thyroid gland; d) kidney;
+ e) gray matter of the brain; + f) testes.
6. Specify the primary immunodeficiencies:
+ a) thymic hypoplasia (DiGeorge syndrome);
b) acquired immunodeficiency syndrome in childhood;
+ c) Bruton's agammaglobulinemia;
d) Klinefelter's syndrome.
7. Specify the cells, tissues and organs containing autoantigens:
+ a) the thyroid gland; + b) lens of the eye;
c) cells of the periosteum; + d) nerve cells.
8. What is one of the important differences between antigen-presenting cells of the immune system from other cells having phagocytic activity?
a) is not capable to completeness of phagocytosis;
b) have a more high phagocytic activity;
c) have the phagocytic activity only in cooperation with the T- and B-lymphocytes;
+ d) are able to transmit information about alien Ag to T- and B-lymphocytes.
9. Immunodeficiency states with predominant lesion of cellular immunity include:
+ a) DiGeorge syndrome; b) Higashi syndrome;
+ c) Wiskott-Aldrich syndrome; d) Louis-Bar syndrome.
10. What is the main danger of immunodeficient states?
+ a) in an increased tendency to infection, its chronization and generalization;
+ b) an increased tendency to neoplastic processes;
+ c) possibility of development of autoimmune diseases on the basis of immunodeficiency;
d) reduced propensity to neoplastic processes;
e) low propensity to infection, its chronization and generalization.
11. Insufficiency of external breathing is accompanied by:
a) increasing the partial pressure of oxygen (pO2) and carbon dioxide (pCO2) in the blood;
b) an increase in the pO2 and decrease pCO2 in blood;
c) a decrease in pO2 and pCO2 in blood;
+ d) a decrease in pO2 and increase pCO2 in blood;
e) an increase in pO2 and normal pCO2 in blood.
12. Alveolar hypoventilation is characterized by:
+ a) hypoxemia, hypercapnia, acidosis;
b) hyperosmia, hypocapnia, alkalosis.
13. Obstructive type of hypoventilation develops at:
+ a) reducing of the total lumen of bronchi;
b) the restriction of unfolding of the lungs during breathing;
c) decrease in pulmonary surface;
+ d) impaired patency of the airway;
e) inhibition of the function of the respiratory center.
14. Restrictive type of hypoventilation occurs when:
a) laryngeal edema; b) hypersecretion of the bronchial mucosa;
c) spasm of bronchioles; d) strangulation; + e) pleuritis.
15. Perfusion form of respiratory failure develops at:
a) shock;
b) embolism of branches of the pulmonary artery;
c) the weakening of the contractile function of the heart;
+ d) all of the above.
16. The distance for diffusion of gases can be increased:
a) during hyperventilation;
b) in violation of the mechanics of breathing;
c) by increasing the number of functioning alveoli;
+ d) wnen fibrotic changes in the lungs;
e) the depression of the respiratory center.
17. Shortness of breath - is:
a) hyperpnea; b) tachypnea;
c) bradypnea; d) gasping breath; + e) dyspnea.

18. Periodic breathing is:
a) tissue; b) dissociated; c) Kussmaul;
d) gasping; + e) Cheyne-Stokes
19. Inspiratory dyspnea is observed the following pathological conditions:
+ a) I stage of asphyxia; b) emphysema of lungs;
+ c) laryngeal edema; d) bronchial asthma; + e) tracheal stenosis.
20. Expiratory dyspnea is observed the following pathological conditions:
a) I stage of asphyxia; + b) emphysema of lungs;
c) laryngeal edema; + d) bronchial asthma; e) tracheal stenosis.
21. Specify the stages of acute renal failure:
a) latent, prodromal, urogenic sepsis, uremic;
+ b) oligo-anuric, initial, recovery of diuresis and polyuria, reconvalescence.
22. What is the stage of acute renal failure syndrome is characterized by development of uremia?
a) initial; b) reconvalescence;
+ c) oligo-anuric; d) prodromal;
e) recovery of diuresis and polyuria.
23. What factors reduce the filtration in the renal glomerulus?
+ a) reducing the hydrostatic pressure in the glomerular capillaries, increasing the oncotic pressure of the blood, increasing intrarenal pressure, reduction of the filtering surface;
b) increase in hydrostatic pressure in the glomerular capillaries, decreased oncotic pressure of the blood, reducing the intrarenal pressure, increased filtration surface.
24. How changes the hydrostatic pressure in the renal glomeruli when constriction of the renal artery and afferent arterioles?
a) increases;
+ b) decreases.
25. Diffuse glomerulonephritis is accompanied by:
+ a) increased permeability of glomerular basement membrane;
b) decrease in the permeability of the glomerular basement membrane.
26. What changes reduce the hydrostatic pressure in the renal glomeruli?
a) increased blood pressure, increased cardiac output, increased blood volume, increased plasma volume;
+ b) decreased blood pressure, decreased cardiac output, decrease in blood volume, decreased plasma volume.
27. The combination of hypostenuria with polyuria indicates on the primary lesion of the nephron structures, such as:
a) glomerulus; + b) tubules.
28. Hyponatremia in acute renal failure leads to:
a) intracellular dehydration; + b) intracellular hyperhydration.
29. Uremia is characterized by:
+ a) anemia, leukocytosis, thrombocytopenia, hypocalcemia;
b) polycythemia, leukopenia, increase of platelets per unit volume of blood, hypercalcemia.
30. How changes filtration when selective increase in tonus efferent arterioles of renal glomeruli?
+ a) is incremented; b) decreases.
31. Degenerative forms of red blood cells include:
a) Reticulocytes +b) The red blood cells of different sizes
c) Polychromatic erythrocytes d) Normocytes
e) Red blood cells with the remnants of a nuclear substance
32. Poikilocytosis erythrocytes is:
a) Change of average diameter of erythrocytes
+b) Changing the shape of red blood cells
c) Red blood cells with punctate basophilia
d) Red blood cells with Jolly's body
e) Red blood cells with Cabot's ring
33. Anisocytosis erythrocytes is:
a) With pathological inclusions in RBC
b) Target RBC
c) Hyperchromia erythrocytes
d) The oval shape of red blood cells
+ e) The presence of red blood cells in the blood of various sizes
34. Reticulocytosis with anemia indicates:
a) The emergence of hyper- or hypo-chromic RBC

+ b) increase the functional activity of bone marrow
c) Changes in the shape of red blood cells
d) Changes in the diameter of RBC
e) Type of megaloblastic hematopoiesis

35. What of these statements are correct?

a) Anemia is always characterized by a decrease in the number of red blood cells per unit volume of blood
+ b) In most cases of anemia observed decrease in the number of erythrocytes per unit volume of blood
+ c) Anemia is always characterized by a decrease in the total amount of hemoglobin
d) Anemia is not necessarily accompanied by a decrease in the total amount of hemoglobin

36. Show basic pathogenetic factors that contribute to the development of anemia:

+ a) insufficient production of red blood cells
+ b) Increased destruction of red blood cells
c) increased production of red blood cells
d) Decreased of destruction of red blood cells
+ e) Disorders of the output of erythrocytes from bone marrow

37. Anemia divided into posthemorrhagic, hemolytic and diserythropoietic depending on:

a) Cause b) Type of erythropoiesis
c) Color index + d) Pathogenesis

38. Anemia divided into: norma-, hyper-, hypo-, dis- or aregeneratic depending on:

a) Essence b) Cause
c) Mechanism d) Nature
+ e) Of the functional state of bone marrow

39. Anemia divided into normoblastic and megaloblastic depending on:

a) Mechanism
b) Nature
c) The functional state of the bone marrow
+ d) Type of erythropoiesis
e) Color index

40. Anemia divided into: norma-, hyper-, hypochromic depending on:

a) the functional state of the bone marrow b) Type of erythropoiesis
+ c) color index d) The etiology
e) Pathogenesis

41. What states there is a simple hypovolemia?

+ a) 30-40 min after acute blood loss
b) 24 hours after the acute blood loss moderate
c) When the burn shock
d) When hyperthermia

42. What time after acute hemorrhage of moderate severity develops reticulocytosis?

a) After 5-6 hours + b) After 4-5 day
c) 24-48 hour d) Immediately after blood loss

43. Select processes that have adaptive significance for the organism in the next minutes and hours after acute blood loss:

a) Reduction of venous return + b) of peripheral vasoconstriction
+ c) Centralization of blood circulation d) of tissue hypoperfusion
+ e) Hyperventilation

44. By single massive blood loss occurs:

a) Iron deficiency anemia b) B-12 deficiency anemia
c) Acute hemolytic anemia d) Acute aplastic anemia
+ e) Acute posthemorrhagic anemia

45. Picture of blood in acute posthemorrhagic anemia is characterized by 4-5 day:

+ a) Increase polychromatophilia
+ b) increase of reticulocytes
c) The emergence of megaloblast
+ d) development of neutrophilic leukocytosis with nuclear shift to the left
e) the appearance of microspherocytes

46. Chronic blood loss to development:

+ a) Iron Deficiency Anemia b) Vitamin B12 deficiency anemia
c) Hemolytic anemia d) Fanconi Anemia
e) microspherocytic anemia Minkowsky-Shauffard

47. Iron Deficiency Anemia is characterized by:
a) reticulocytosis
+ b) hypochromia
c) the type of megaloblastic hematopoiesis
d) hemosiderosis
e) hyperbilirubinemia
48. What anemia characterized by red blood cells expressed hypochromia?
+ a) Chronic posthemorrhagic anemia b) Hypoplastic anemia
c) Acute posthemorrhagic anemia d) Helminthic B12-deficient anemia
+ e) Thalassaemia
49. What hereditary hemolytic anemia by type membranopathy include:
+ a) microspherocytosis
b) Anemia due to a deficiency of glucose-6-phosphate dehydrogenase
c) a-thalassemia
d) Sickle cell anemia
e) b-thalassemia
50. What cause cans development of B12-deficiency anemia?
a) Radiation sickness + b) Subtotal Gastrectomy
+ c) ileal resection + d) Resection of the jejunum
+ e) Helminthic B12-deficient anemia
51. What a leukocytosis?
a) Increase in the number of leukocytes in the bone marrow
+ b) Increasing the number of leucocytes per unit volume of blood (usually over 9.0 gig / liter)
c) Increasing the number of leukocytes in tissues
52. What mechanisms play a major role in the emergence of leukocytosis?
+ a) Stimulation leukopoiesis
+ b) Accelerated exit of leukocytes in peripheral blood
c) Increased capillary pressure
d) Decrease capillary pressure
53. Neutrophilic leukocytosis observed in:
a) Allergic reactions + b) pyoseptic processes
c) helminthic invasion + d) Traumatic brain injury + e) Myocardial Infarction
54. Physiological leukocytosis observed in:
a) Myocardial Infarction b) Pneumonia
c) Bone fractures + d) Neonatal e) Acute posthemorrhagic anemia
55. General mechanism is pathological leukocytosis
a) The redistribution of blood in the vessels + b) Stimulation leukopoiesis
c) The emigration of leukocytes d) diapedesis of leukocytes
e) phagocytosis of leukocytes
56. Eosinophilia characterized by:
a) acute purulent process + b) for asthma
c) Sepsis d) For infectious mononucleosis e) For Measles
57. Lymphocytosis characterized by:
+a) for tuberculosis b) For septic diseases
c) For measles d) For asthma e) For the myocardial infarction
58. Nuclear neutrophilic shift to the left is called the increase in the percentage of blood:
a) agranulocytes
b) Granulocytes
+ c) immature forms of neutrophils
d) of the mature form of neutrophils
e) Hypersegmentated forms of neutrophils
59. Neutrophilic leukocytosis with regenerative nuclear shift to the left is:
a) Leukocytosis with increasing band neutrophils
b) leukocytosis with a predominance of neutrophils hypersegmentated
+ c) Leukocytosis with increasing band neutrophils and the emergence of metamyelocytes
d) The appearance of myeloblasts hemogram
e) The appearance of myelocytes hemogram
60. Agranulotsitoz is:
a) Increase in the number of lymphocytes and monocytes in the blood

+ b) The acute decrease in the number of neutrophils and eosinophils in the blood
c) Increase the number of agranulocytes
d) Increase in the number of blood neutrophils hypersegmentated
e) The acute decrease in the number of reticulocytes in the blood

61. What is leukemia?

+ a) systemic disease of tumor nature, causing from hematopoietic cells with primary bone marrow
b) Symptomatic conditions characterized by a significant increase in the number of leukocytes in the blood with the emergence of a large number of young forms

62. What a leukemoid reaction?

a) Systemic disease of tumor nature, causing from hematopoietic cells with primary bone marrow
+ b) Symptomatic conditions characterized by a significant increase in the number of leukocytes in the blood with the emergence of a large number of young forms

63. Name probable mechanism of leukemia:

+ a) Mutation of genes or epigenetic dysregulation division and differentiation of hematopoietic progenitor cells
b) Disturbance of the immunological status with the emergence of clone "forbidden" cells

64. Name acute forms of leukemia:

+ a) nondifferential b) myeloleukemia
+ c) myeloblastic d) lymphatic leukemia
+ e) promyelocytic + f) monoblastic

65. By acute leukemia in the bone marrow occur:

+ a) white blood elements hyperplasia
b) lack of white blood metaplasia elements
+ c) Decrease of the number of elements erythrocyte cell
d) Increase the number of elements erythrocyte cell
+ e) Reduction in the number of megakaryocytes
f) Increase in the number of megakaryocyte

66. What changes in the peripheral blood is accompanied by the development of most of leukemia?

+ a) Anemia b) Erythrocytosis
+ c) Thrombocytopenia d) Thrombocytosis

67. "hiatus leukemic" is:

a) Absence of young forms of leukocytes in peripheral blood
+ b) The absence of intermediate forms between blast maturing and mature forms of leukocytes
c) Lack of young and mature forms of leucocytes with a predominance of intermediate

68. Increase blast forms of granulocytes in blood and the absence of intermediate forms of maturation with a small number of mature cells (leukemic hiatus) is characterized by:

a) For chronic leukemia+ b) For acute leukemia
c) For leukemoid reaction d) To leukocytosis e) To leucopenia

69. To 90% of mature lymphocytes and lymphoblasts isolated in leucocyte count observed at:

a) acute myeloid leukemia b) chronic myeloid leukemia
c) acute lymphocytic leukemia + d) chronic lymphocytic leukemia
e) monocytic leukemia

70. For leukemoid reactions neutrophil type characteristic is:

a) basophilic-eosinophilic association
+ b) hyperregenerative leukocyte shift to the left
c) the appearance of plasma cells in the blood
d) granulocyte cell atypia
e) Pancytopenia

71. Cause acute right ventricular failure can be

a) Aortic insufficiency b) failure of the mitral valve
c) Aortic stenosis + d) pulmonary stenosis e) Mitral stenosis

72. One of the causes of left ventricular failure is

a) Lung disease b) stenosis of the pulmonary artery
c) the tricuspid valve d) right ventricular infarction + e) failure of the mitral valve

73. Overload form of heart failure results:

+ a) hypervolemia b) Myocardial ischemia
c) Myocarditis d) Extrasystole e) myocardial

74. Overload heart "resistance" develops in

a) eritremia + b) Hypertension
c) physical exertion d) hypervolemia

75. Long-term adaptation of cardiac function provides
a) Tachycardia + b) myocardial hypertrophia
c) Heterometric mechanism d) Homeometric mechanism e) Myogenic dilatation
76. For compensation stage heart failure is characterized by:
+ a) tonogenic dilatation of heart
+ b) Tachycardia
+ c) myocardial hypertrophy
d) Myogenic dilatation
e) Increase in residual blood in the cavities of the heart
77. Name extracardiac compensation mechanisms in heart diseases
+ a) Increased erythropoiesis
b) Tonogenic dilatation of heart
c) Hypertrophia myocardium
78. Emergency hyperfunction stage heart on Meerson characterized
+ a) Hyperfunction non hypertrophic infarction
b) Hyperfunction hypertrophic myocardium
c) normalization of energy production per unit mass of the myocardium
d) growth of connective tissue
e) reduction of protein synthesis per unit of muscle mass
79. Stage decompensation heart failure manifests itself:
+ a) cyanosis+
b) Edema
c) increase in systolic volume
+ d) increased residual volume of blood in the cavities of the heart
e) increase in blood pressure
80. Hemodinamic indicators in chronic heart failure characterized by
a) reduction of central venous pressure
+ b) reduction of cardiac output
c) increasing the flow velocity
d) increasing the capacity of the left ventricle contraction
e) increasing the total peripheral vascular resistance
81. Name likely causes of symptomatic arterial hypertension:
+ a) Hyperthyroidism
b) chronic psychoemotional overstrain
+ c) Chronic nephritis
d) Repeated prolonged negative emotions
+ e) of atherosclerotic vascular lesions
f) Genetic defects of the centers of the autonomic nervous system, regulating blood pressure
g) Genetic defects cation transport membrane systems that lead to calcium accumulation within the cytoplasm of smooth muscle cells of the vascular wall
82. Name diseases and conditions that are accompanied by increases in blood pressure:
+ a) Cushing's syndrome b) Klinefelter's syndrome
+ c) Cushing's disease d) hypo(adreno)corticism
e) Hypothyroidism + f) Hyperthyroidism
+ g) Cushing + h) Pheochromocytoma
83. Name hemodynamic form of hypotension:
+ a) heart failure
+ b) decreased the volume circulating of blood
+ c) When lowering the tone of resistance vessels
d) hypovolemia
84. Name substances that have a direct pressor effect:
+ a) Renin b) Angiotensin-2
+ c) ADH + d) Adrenaline
e) Histamine + f) Norepinephrine
85. Cause renoprive hypertension:
a) the two main stenosis of the renal arteries b) Polycystic Kidney Disease
c) nephrosis d) nephritis
+ e) All answers are correct
86. Complications of hypertension are:

+ a) Heart failure
+ b) Myocardial infarction
+ c) Stroke
+ d) Lack of intestinal blood flow
+ e) Disturbance of adrenal function

87. Pathogenesis of hypertension presumably includes the following links:

+ a) Stable increase of excitability and reactivity of the sympathetic nerve center of the back of the hypothalamus
+ b) Reducing the inhibitory effect of the cortex, it is normally exerted on the subcortical centers pressor
c) Depletion of adrenocortical function
+ d) Genetically caused a steady decline sodium, chlorine and water-excretory kidney function
+ e) Generalized hereditary defect of membrane ion pumps;
f) genetically determined hypoproduction mineralocorticoid

88. Increase renin secretion is caused by:

a) an increase in perfusion pressure in the arterioles of kidney cells
+ b) reduced perfusion pressure in the arterioles of kidney cells
c) hyponatremia and hyperkalemia
+ d) hypernatremia and hypokalemia
e) reduction of angiotensin II in the blood
+ f) Increased levels of angiotensin II in the blood

89. Name differences of hypertension from other arterial hypertension:

+ a) Increased blood pressure arises because of the lack of significant organic lesions of the internal organs involved in its regulation
b) result from primary renal dysfunction and endocrine glands
+ c) is important in its development has a genetic predisposition
d) resulting from a violation of the adrenal glands
e) develops as a result of damage to the primary receptor of the aortic arch and carotid sinus area
+ f) is essential to improve the reactive properties of neurons of the sympathetic center of the back of the hypothalamus

90. Pathogenesis of hypotension matter following mechanisms:

+ a) Increased activity of the parasympathetic nervous system while reducing the activity of the sympathetic
+ b) The genetic defect of ion transport into the cell with the accumulation of calcium in the cytoplasm smooth muscles cell vessel walls
+ c) Decrease in renal renin production
+ d) reducing the sensitivity of receptors smooth muscles cell vascular angiotensin II
e) disturbance of the conversion of dopamine to norepinephrine at nerve endings
f) Decrease production of glucocorticoids
g) Increased activity of the sympathetic division of the ANS

91. Name heterotopic rhythms:

+ a) Nodal rhythm
+ b) Migration supraventricular pacemaker
c) Sinus tachycardia
d) Sinus bradycardia
e) Sinus arrhythmia
+ f) AV-dissociation
+ g) idioventricular rhythm

92. For premature atrial contraction is characterized by:

+ a) The presence of P-wave
b) The absence of P wave
c) Severe deformity ventricular complex
+ d) Minor changes in ventricular complex
e) The full compensatory pause
+ f) Incomplete compensatory pause

93. For ventricular arrhythmia characterized by:

a) The presence of P-wave
+ b) The absence of P wave
+ c) Severe deformity ventricular complex
d) Minor changes in ventricular complex
+ e) Full compensatory pause
f) Incomplete compensatory pause

94. Name pathogenetic factors of ventricular fibrillation:

a) increase in the concentration of intracellular potassium
+ b) Decrease in the concentration of intracellular potassium
+ c) Reduction of the membrane potential of cardiomyocytesd) Increase in the membrane potential of cardiomyocytes

95. For flutter and fibrillation atrial is characterized by:

a) Lack of pulses AV-node
+ b) Partial functional unit of the excitation by AV-node
c) The normal conduction of impulses by AV-node

96. Name causes of ventricular fibrillation:

+ a) passage of electric current through the heart

+ b) Toxic doses of cardiac glycosides
c) Introduction of acetylcholine
+ d) Severe myocardial hypoxia
e) The action of botulinum toxin

97. Show pacemaker with ventricular flutter:
a) the atrioventricular node
b) the bundle of His
+ c) of ectopic foci located in the ventricles
d) Some of ectopic foci submitted by individual groups of muscle fibers of the ventricles

98. Show pacemaker with ventricular fibrillation:
a) the atrioventricular node
b) the bundle of His
c) of ectopic foci located in the ventricles
+ d) Several of ectopic foci submitted by individual groups of muscle fibers of the ventricles

99. Show source of impulses at a nodal rhythm:
a) the sinoatrial node + b) atrioventricular node
c) the bundle of His d) of ectopic foci located in the ventricles

100. Name conditions necessary for the formation of the mechanism of excitation of re-entry (re-entry):
a) Accelerated impulse conduction in a small portion of the conductive system (in the twig A Purkinje cells)
+ b) Delayed impulse conduction in a small section of wire system (a twig A Purkinje cells)
+ c) One-way flow of excitation in the twig B Purkinje cells
d) two-way flow of excitation in the twig B Purkinje cells

101. For acute intestinal auto-intoxication is characterized by:
a) a drop in blood pressure b) Reduction of pain sensitivity
c) The weakening of the heart rate d) Development of coma + e) All signs

102. Base pathogenesis of malabsorption syndrome are:
a) enhancing the hydrolysis of food components in the gut
b) accumulation in the intestinal lumen decay products of incomplete meals
c) the body hydropenia
d) Increased excretion of the vassel of water, protein electrolytes
+ e) All of the above features

103. Show main manifestations of insufficiency of digestion:
+ a) depletion of the body + b) hypo- and avitaminosis
c) Hypervitaminosis + d) negative nitrogen balance
e) A positive nitrogen balance f) Increasing resistance of the organism
g) Hyperproteinemia + h) Reduction of reactivity

104. Show protective and adaptive changes at the failure of the digestive system:
a) Reduction and dysorexia
b) Heartburn
c) Belching
d) Frequent and profuse vomiting
+ e) Increased production of lysozyme and hydrochloric acid
+ f) increased peristalsis
+ g) Increased intestinal barrier function

105. What changes digestion with hypo-secretion of gastric juice and achlorhydria?
a) slows gastric emptying
+ b) accelerates gastric emptying
+ c) Nutritional weight substantially do not undergo digestion in the stomach
d) digestion of food mass varies slightly

106. Show change the digestive process with achlorhydria:
a) Slowing food mass evacuation from the stomach into the intestine
+ b) Reducing the activity of enzymes peptic stomach
+ c) Reducing the formation of secretin in the duodenal mucosa
d) Decreased activity of enterokinase

107. Show hypersalivation consequences:
a) The difficulty of the act of chewing and swallowing
+ b) maceration and inflammatory changes in the skin around the lips
c) Occurrence of inflammatory processes in the oral mucosa
d) Decrease of gastric secretory function

+ e) neutralization of gastric acid salt

108. Show consequences hypoptyalism:

+ a) The difficulty of the act of chewing and swallowing
b) maceration and inflammatory skin changes in the lips
+ c) inflammatory processes in the oral mucosa
+ d) Decrease of gastric secretory function
e) neutralization of gastric acid salt

109. What disturbance occur when hypo- or acholia?

+ a) disturbance of digestion and absorption of fats
+ b) Deficiency of fat-soluble vitamins
c) Absorption of fat-soluble vitamins are not disturbance
+ d) Laxation weakens
e) enhanced Laxation
+ f) pH of the duodenal contents is shifted to the acid side
g) The pH of the duodenal contents is shifted to the alkaline side

110. Show substance, the lack of which can lead to hyposecretion of gastric juice:

+ a) Gastrin b) enteroanthelone + c) Cholecystokinin
d) Secretin + e) Glucocorticoids + f) Insulin g) Glucagon

111. Secondary liver failure develops in

a) the action of carbon tetrachloride + b) circulatory failure
c) phosphoric intoxication d) viral hepatitis
e) Chronic alcohol intoxication

112. Primary liver failure develops in

a) Heart Failure b) Shockt
c) Renal insufficiency + d) viral infection of the liver e) diabetes mellitus

113. Manifestations of liver failure include:

+ a) Increasing the concentration of ammonia in the blood
+ b) Hypoproteinemia
c) decrease in the activity of ALT and AST in the blood
+ d) Bleeding
e) Dehydration

114. Pathogenesis of hepatic coma significant dose:

+ a) Lack of neutralizing function of the liver
+ b) Metabolic acidosis
+ c) Lack ureaformation liver function
d) Increase in blood direct bilirubin
e) Hyperglycemia

115. Causes hemolytic jaundice include:

+ a) Action hemolitic poisons
+ b) Rhesus conflict between maternal and fetal body
+ c) incompatible blood transfusion
d) hemorrhagic anemia
e) gallbladder dyskinesia

116. For hemolytic jaundice characterized by an increase in blood

+ a) indirect bilirubin b) direct bilirubin c) urobilin
d) stercobilin e) bile acids

117. General pathogenesis obstructive jaundice is

a) Damage to hepatocytes
b) sialolithiasis
c) Urolithiasis
d) Reinforced hemolysis
+ e) Disturbance of the outflow of bile

118. Show causes of jaundice

+ a) Obturation hepatic and common bile duct
b) hemolysis

119. Obstructive jaundice is observed:

+ a) Hypotension + b) bilirubinuria + c) Acholia
+ d) Itching e) Tachycardia

120. For characterized by an increase in blood

a) biliverdin b) sterkobilinogen
c) urobilinogen + d) direct bilirubin e) indirect bilirubin

121. Peripheral breaking mechanism of activity of hormones are:

+ a) Disturbance of the hormone binding to plasma proteins
+ b) blockade of hormone receptors
c) Disturbance of generating releasing hormones of the hypothalamus
+ d) Inactivation of circulating hormone
e) Disturbance of hormone synthesis

122. Exceeding production adrenocorticotropic hormone to increased secretion:

a) Insulin b) PTH c) Thyroxine
+ d) Cortisol e) Adrenaline

123. Hyperproduction growth hormone can lead to

+ a) Gigantism b) pituitary dwarfism

124. Cause Addison's disease most often

a) adrenal hypertrophy + b) adrenal atrophy
c) pituitary tumor d) Autoimmune thyroiditis
e) Hyperplasia epiphysis

125. Increment concentrations of thyroid stimulating hormone in blood hypothyroidism indicates the localization of the pathological process in

a) by the pituitary gland + b) of the thyroid gland
c) of the parathyroid glands d) of the hypothalamus e) of the thymus

126. Show pathogenetic factors of disorders of the nervous system in meningitis and encephalitis:

+ a) gives products and outflow of cerebrospinal fluid
b) Products and outflow of cerebrospinal fluid did not change
+ c) Increased intracranial pressure
d) it reduces intracranial pressure
+ e) triggers autoimmune processes
f) activates the process of retrograde degeneration

127. What a generator of pathologically enhanced excitation?

+ a) A group of neurons that work with varying degrees of autonomy and produce excessive stimulation
b) A group of neurons that produce excessive stimulation due to sharp activation of the higher nerve centers

128. What characteristic of denervated tissues?

+ a) Increased sensitivity to mediators released upon stimulation of the transected nerve
b) Reduced sensitivity to mediators released during stimulation of the transected nerve

129. What is pain?

+ a) subjectively painful sensation reflecting psychophysiological state of a person, which arises as a result of the impact of super-strong or damaging stimuli
b) Increased sensitivity to stimuli of the senses, which arises as a result of the impact of super-strong or damaging stimuli

130. What characteristic epicritic pain?

+ a) arises immediately after injury b) arises after a short period of time after injury
+ c) clearly localized d) Has diffuse
+ e) Quickly subsides f) lasts a long time

131. What characteristic protopathic pain?

a) arises immediately after injury + b) arises after a short period of time after injury
c) clearly localized + d) has a diffuse character
e) Fast subsides + f) lasts a long time

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