**Tests Questions of Final Exam 2023 (Students of 3rd year, Medicine 2)**

.1. For what cardiac defect there is characteristic heterometric hyperfunction?

a. mitral stenosis

b. aortic stenosis

\*c. insufficiency of bicuspid valves

d. stenosis of right atrioventricular orifice

e. stenosis of pulmonary artery orifice

2. For what cardiac defect there is characteristic heterometric hyperfunction?

a. mitral stenosis

b. aortic stenosis

\*c. insufficiency of bicuspid valves

\*d. insufficiency of semilunar valves

e. stenosis of pulmonary artery orifice

3. How is performed predominant heterometric hyperfunction of the myocardium?

a. by tachycardia

\*b. by Frank – Starling law

\*c. by increased systolic pressure

\*d. by increased contractility performance of the myocardium

e. by bradycardia

4. In what disorders can be attested leucocyturia?

\*a. urinary infection

\*b. inflammation of the pelvicalyceal system

\*c. cystitis

d. glomerulonephritis

e. acute tubular necrosis

5. In what disorders can be attested lipiduria?

\*a. nephrotic syndrome

b. nephritic syndrome

\*c. lipodystrophy of tubular epithelium

d. liver failure

e. hyperlipidemia

6. What disorder is manifested by tubular proteinuria?

\*a. disturbance of renal lymphatic circulation

b. glomerulonephritis

c. urolithiasis

d. hydronephrosis

e. nephrosclerosis

7. What disorders are manifested by tubular proteinuria?

\*a. inflammatory tubulopathy

\*b. disturbances of lymphatic flow in the kidney

c. glomerulonephritis

d. urolithiasis

e. hydronephrosis

8. How are modified intra-thoracic pressure and venous return in shallow breathing?

a. intra-thoracic pressure increases, venous return increase

b. intra-thoracic pressure decreases, venous return decrease

c. intra-thoracic pressure increases, venous return does not change

d. intra-thoracic pressure decreases, venous return is difficult

\*e. intra-thoracic pressure increases, venous return decreases

9. What conditions decrease alveolar capillary diffusion?

\*a. pneumoconiosis

b. atherosclerosis of pulmonary circuit arteries

c. lungs hypoperfusion

d. atmospheric hypobaria

e. airways obstruction

10. What is the cause of relative hypoxia in hypertrophied myocardium?

\*a. myocardial hyperfunction

b. formation into the vessels of atheromatous plaques

c. disturbance of oxygen using

d. disturbance of energogenesis

e. relative insufficiency of myocardial vasculature

11. What are the changes of blood cells counts in hyperproliferation of red bone marrow?

a. increasing the number of erythroblasts

d. increasing the number of stem cells

\*c. increasing number of young red blood cells

d. intensification of the ineffective erythropoiesis

e. erythrocytosis with hemodilution

12. Which is one of the signs of hypochromic erythrocytes?

a. anulocyte with the hemoglobin content of 29 pg

b. anulocyte with the hemoglobin content of 40 pg

\*c. anulocytes with hemoglobin content less than 27 pg

d. eliptocyte with hemoglobin content 29 pg

e. eliptocyte and mean hemoglobin concentration of 33%

13. Absorbtion of what substances is disturbed in dysfunction of small intestine mucosa?

a. proteins

\*b. monosaccharides

\*c. aminoacids

d. disaccharides

e. water

14. Absorbtion of what substances is disturbed in large intestine disorders?

a. proteins

b. aminoacids

\*c. mineral salts

d. monosaccharides

\*e. water

15. By what is manifested disturbance of cardiac excitability?

a. bradycardia

b. complete transversal bloc

c. longitudinal block

d. tachycardia

\*e. extrasystole

By what is manifested disturbance of intracardiac conductibility?

\*a. atrioventricular block

b. sinus bradycardia

c. sinus tachycardia

d. extrasystole

e. ventricular fibrillation

16. By what method was modeled hyperthyroidism in rats?

a. by administration of methyluracil

b. by administration of caffeine

c. by administration of chloralhydrate

\*d. by administration of L – thyroxin

c. by administration of NaCl

17. By what method was modeled sinus tachycardia in frog?

A\*. by the application of adrenaline on the heart

B . by the application of acetylcholine on the heart

C. by the application of AgNO3 on the heart

D. by the application of cold physiological solution on the heart

E. by the application of HCl solution on the heart

18. By what method was modeled sinus tachycardia in frog

A. by the application of AgNO3 on the heart

B. by the application of acetylcholine on the heart

C\*.by the application of warm physiological solution on the heart

D. by the application of cold physiological solution on the heart

E. by the application of HCl solution on the heart

19. By what method was modeled ventricular extrasystole in frog?

A. by the application of KCl solution on the heart

B. by the application of adrenaline on the heart

C. by the application of acetylcholine on the heart

D\*.by the mechanical excitation of the heart

E. by the electrical excitation of the heart

20. By what methods was modeled sinus bradycardia in frog?

A. by the application of adrenaline on the heart

B\*.by the application of acetylcholine on the heart

C. by the application of warm physiological solution on the heart

D\*.by the application of cold physiological solution on the denudated heart

E\*.by the application of KCl solution on the denudated heart

21. By what methods was modeled sinus bradycardia in frog?

A. by the application of adrenaline on the heart

B\*.by the application of acetylcholine on the heart

C. by the application of warm physiological solution on the heart

D\*.by the application of cold physiological solution on the denudated heart

E. by the application of HCl solution on the denudated heart

22. By which method the deep and accelerated respiration was reproduced in rabbit?

A. ammonia inhalation

B. oxygen inhalation

C\*.carbon dioxide inhalation

D. carbon monoxide inhalation

E. by closing one nostril

23. For what pathology is characteristic homeometric hyperfunction of the heart?

A. stenosis of left atrioventricular orifice

B\*.hypertension into the systemic circulation

C. insufficiency of aortic valves

D. insufficiency of mitral valves

E. insufficiency of tricuspid valves

24. How are modified intra-thoracic pressure and venous return in accelerated and deep respiration?

A. intra-thoracic pressure increases, venous return decreases

B\*.intra-thoracic pressure decreases, venous return increases

C. intra-thoracic pressure increases, venous return does not change

D. intra-thoracic pressure decreases, venous return is difficult

E. intra-thoracic pressure increases, venous return is facilitated

25. How are modified intra-thoracic pressure and venous return in shallow breathing?

A. intra-thoracic pressure increases, venous return increase

B. intra-thoracic pressure decreases, venous return decrease

C. intra-thoracic pressure increases, venous return does not change

D. intra-thoracic pressure decreases, venous return is difficult

E\*.intra-thoracic pressure increases, venous return decreases

26. How blood pressure (BP) and breathing rate (BR) change in painful stimulation?

A\*.BP increase

B. BP decrease

C. BR decrease

D\*.BR increase

E. BP decrease, BR unchanged

27. How do the arterial and venous pressure change in heart failure?

A. arterial and venous pressure increase

B\*.arterial hypotension and venous hypertension

C. arterial and venous pressure don’t change

D. arterial pressure increases but venous decreases

E. arteial and venous pressure change

28. How do the systolic volume and cardiac output change in heart failure?

A. cardiac output increases and systolic volume decreases

B. cardiac output decreases and systolic volume increases

C\*.cardiac output decreases and systolic volume decreases

D. cardiac output increases and systolic volume increases

E. cardiac output decreases systolic volume unchanged

29. How does acid-base balance change in patients with type I diabetes mellitus?

A. metabolic acidosis caused by increased level of CO2

B\*.metabolic acidosis caused by accumulation of acetylacetic acid

C. excretory acidosis with accumulation of acids

D. metabolic acidosis caused by accumulation of oxaloacetate

E. lactoacidosis

30. How does blood cells count change in hypo- and aplastic anemia?

a. lymphocytopenia

b. sickle cell disease

c. megalocytosis

d. thrombocytosis

\*e. erythrocytopenia

31. How does blood hormones concentration change in primary hypothyroidism?

a. low thyroid-releasing hormone, low thyrotropin, low thyroid hormones

b. low thyroid-releasing hormone, high thyrotropin, decreased thyroid hormones

\*c. increased thyroid-releasing hormone, increased thyrotropin, low thyroid hormones

d. low thyroid-feleasing hormone, low thyrotropin, raised thyroid hormones

e. increased thyroid-releasing hormone, low thyrotropin, low thyroid hormones

32. How does blood hormones concentration change in secondary hypothyroidism?

a. low thyroid-releasing hormone, low thyrotropin, low thyroid hormones

b. low thyroid-releasing hormone, high thyrotropin, decreased thyroid hormones

c. increased thyroid-releasing hormone , increased thyrotropin , low thyroid hormones

d. low thyroid-releasing hormone, low thyrotropin, raised thyroid hormones

\*e. increased thyroid-releasing hormone, low thyrotropin, low thyroid hormones

33. How does blood hormones concentration change in tertiary hyperthyroidism?

a. increased thyroid-releasing hormone, increased thyrotropin , low thyroid hormones

b. increased thyroid-releasing hormone , low thyrotropin, raised thyroid hormones

c. low thyroid-releasing hormone, high thyrotropin, raised thyroid hormones

d. low thyroid-releasing hormone, low thyrotropin, raised thyroid hormones

\*e. increased thyroid-realising hormone, increased thyrotropin, raised thyroid hormones

34. How does blood hormones concentration change in tertiary hypothyroidism?

a. increased thyroid-releasing hormone, increased thyrotropin, low thyroid hormones

b. increased thyroid-releasing hormone, low thyrotropin, raised thyroid hormones

\*c. low thyroid-releasing hormone, low thyrotropin, low thyroid hormones

d. low thyroid-releasing hormone, low thyrotropin, raised thyroid hormones

e. increased thyroid-releasing hormone, increased thyrotropin, raised thyroid hormones

35. How does body weight change in diabetes mellitus type I?

a. increases due to reduced lypolysis in lack of insulin

b. increases due to sodium retention and water caused bu insulin deficiency

c. increase caused by enhanced lipogenesis in lack of insulin

d. decreases due to reduced appetite and reduced food intake

\*e. decreases due to enhanced lypolysis which is the effect of glucocorticoids

36. How does carbohydrate metabolism change in GH hypersecretion in children?

a. hyperglycemia caused by enhanced glycogenogenesis with insulin-resistence

\*b. hyperglycemia caused by enhanced glycogenolysis with insulin-resistence

c. hyperglycemia caused by enhanced gluconeogenesis from free fatty acids released in the result of enhanced lipolysis

d. hyperglycemia caused by enhanced gluconeogenesis from glycerol released in the result of enhanced lipolysis

e. hyperglycemia caused by enhanced gluconeogenesis from amonoacids released in the result of enhanced proteolysis

37. How does energy metabolism change in hypothyroidism?

\*a. reduced oxidation processes with conservation of heat due to vasoconstriction

\*b. decreased basal metabolism caused by decoupling of oxidative phosphorilation

c. decreased basal metabolism caused by increased oxidative phosphorilation

d. decreased basal metabolism caused by reduced oxidative phosphorilation

e. reduced oxidation processes with loss of energy in form of heat due to vasodilation

38. How does lipid metabolism change in hypothyroidism?

\*a. reduced lypolisis with decreased level of free fatty acids in the blood

b. hypercholesterolemia with HDL without atheromatosis

c. hypercholesterolemia with fractions VLDL, LDL and HDL

d. reduced lypolisis with increased level of glycerol in the blood

\*e. hypercholesterolemia with reduced cholesterol metabolism

39. How does metabolism change in diabetes mellitus type I?

a. reduced catabolism due to insulin lack

b. increased lypogenesis due to excess of glucagon

c. reduced proteolysis caused by excess of glucocorticosteroids

\*d. excessive proteolysis caused by excessive glucagon

e. increased anabolism due to insulin deficiency

40. Which is one of the signs of erythrocytes hypochromia?

\*a. anulocyte with hemoglobin content below 27pg

b. anulocyte with the hemoglobin content of 40 p

c. 0.9-1.1 chromatic index

d. eliptocit with the hemoglobin content of 40 pg

e. spherocyte with mean hemoglobin concentration 33%

41. Which is one of the signs of erythrocytes macrocytosis?

\*a. diameter average of erythrocytes more than 8 μ

b. average volume of erythrocytes 90 fl

c. average thickness of erythrocytes 4 μ

d. ellipsoid shape of erythrocytes

e. mean concentration of hemoglobin into erythrocytes more than 33%

42. Which is one of the signs of hyperchromic erythrocytes?

a. the hemoglobin content of macrocytes more than 33 pg

b. the content of hemoglobin in an erythrocyte equal to 29 pg

\*c. macrocytes with chromatic index exceeding 1.1

d. macrocytes with chromatic index equal to 1

e. macrocytes with mean hemoglobin concentration below 33%

43. Which is one of the signs of hypochromic erythrocytes?

a. anulocyte with the hemoglobin content of 29 pg

b. anulocyte with the hemoglobin content of 40 pg

\*c. anulocytes with hemoglobin content less than 27 pg

d. eliptocyte with hemoglobin content 29 pg

e. eliptocyte and mean hemoglobin concentration of 33%

44. Which pathological processes deregulate digestion in the oral cavity?

\*a. oral mucosal hypoperfusion

b. hypersalivation

\*c. lack of salivary amylase

d. lack of lysozyme

\*e. lack of kallikrein

45. How does metabolism change in diabetes mellitus type I?

a. increased lypolysis with enhanced oxidation of glycerol

\*b. increased lypolysis with enhanced oxidation of FFA

c. reduced lypolysis caused by excess of glucocorticosteroids

d. increased lypolysis due to low level of glucagon

e. increased lypolysis due to low level of catecholamines

46. How does metabolism change in diabetes mellitus type I?

a. increased lypogenesis with body overweigh caused by insulin lack

\*b. increased lypolysis and proteolysis due to excess of glucocorticoids

c. reduced proteolysis with enhanced lypogenesis due to excess of glucocorticosteroids

d. reduced gluconeogenesis due to defficiency in glucocorticoids

e. reduced gluconeogenesis from glycerol caused by excess of catecholamines

47. How does metabolism change in diabetes mellitus type I?

a. reduced lypolysis and proteolysis caused by insulin lack

b. increased proteolysis and lypolisis caused by catecholamine deficiency

c. enhanced proteolysis and lypolysis caused by deficiency of glucocorticosteroids

d. increased anabolism due to excess of glucagon

\*e. increased lypolysis and proteolysis caused by excess of catecholamines

48. How does protein metabolism change in GH hypersecretion in children?

a. intensifies deamination of amino acids and increased their uptake from the blood with enhanced proteosynthesis

b. intensifies transamination of amino acids and increased their uptake from the blood with enhanced proteosynthesis

c. increase peripheral use of aminoacids leading to high level of ammonia in the blood

\*d. increased aminoacids uptake from the blood with enhanced proteosynthesis

e. increases peripheral use of aminoacids leading to high level of urea in the blood

49. How does protein metabolism change in patients with type I diabetes mellitus?

a. there is reduced protein breakdown with negative nitrogen balance caused by excessive catecholamine

b. there is enhanced protein breakdown with negative nitrogen balance caused by low level of glucagon

c. there is reduced protein breakdown with negative nitrogen balance caused by excessive glucocorticoids

\*d. there is enhanced protein breakdown with negative nitrogen balance caused by excessive glucocorticoids

e. there is reduced protein breakdown with positive nitrogen balance caused by insulin deficiency

50. How does the blood enzymatic spectrum change in the injuries of hepatocyte?

a. amylase activity increase into the blood

\*b. ASAT activity increase into the blood

c. cytochrome oxidize activity increase into the blood

d. trypsin activity increase into the blood

e. alkaline phosphatase activity increase into the blood

51. How does the blood enzymatic spectrum change in the injuries of pancreas?

\*a. amylase activity increase into the blood

b. ASAT activity increase into the blood

c. cytochrome oxidize activity increase into the blood

d. ALAT activity increase into the blood

e. alkaline phosphatase activity increase into the blood

52. How does the blood enzymatic spectrum change in the injuries of bile ducts epithelium?

a.amylase activity increase into the blood

b. ASAT activity increase into the blood

c. cytochrome oxidase activity increase into the blood

d. ALAT activity increase into the blood

\*e. alkaline phosphatase activity increase into the blood

53. How does the blood enzymatic spectrum change in the myocardiocyte injuries?

a. amylase activity increase into the blood

b. trypsin activity increase into the blood

c. cytochrome oxidize activity increase into the blood

\*d. ALAT activity increase into the blood

e. alkaline phosphatase activity increase into the blood

54. How does the cell excitability change with the decrease of resting potential?

a. excitability decreases caused by less negative threshold potential

\*b. excitability increases caused by less negative threshold potential

c. excitability decrese caused hyperpolarization

d. excitability increases caused by more negative threshold potential

\*e. excitability increases due to depolarization

55. How does the cell excitability change with the increase of resting potential?

a. excitability increases caused by depolarization

b. excitability decreases caused by less negative threshold potential

c. excitability increase caused by hyperpolarization

\*d. excitability decreases caused by more negative threshold potential

\*e. excitability decreases caused by hyperpolarization

56. How does the cessation of Ca, Mg membrane pumps act on intracellular Ca homeostasis?

A. calcium accumulate in the hyaloplasm of the cell due to impaired transport into Golgi

B. calcium ions level inside the cell decrease caused bu impaired transport outside the endoplasmic reticulum

C. calcium ions level inside the cell decrease caused by impaired inflow from outside the cell

D\*.calcium ions accumulate in the hyaloplasm of the cell due to impaired transport into endoplasmic reticulum

E\*.calcium accumulates in the hyaloplasm of the cell due to impaired transport outside the cell

57. How does the skeletal muscles weight change in diabetes mellitus type I?

A. muscle weight decreases caused by intensified lypolysis in insulin defficiency

B. muscle weigh decreases caused by diminished glycogenogenesis in insulin deficiency

C\*.muscle weight decreases caused by enhanced proteolysis which is the effect of glucagon

D. muscle weigh decreses caused by dehydration of muscles in hyperosmolarity caused by hyperglycemia

E\*.muscle weight decreases caused by impaired aminoacid uptake in lack of insulin

58. How does the structure of hypertrophic myocardium change?

A. increase number of cardiomyocytes

B. increase number of cardiomyocytes with decreasing their volume

C. increase abundantly the connective tissue

D. hypertrophy of cardiomyocytes with decreasing their number

E\*.hypertrophy of cardiomyocytes but their number remains constant\*

59. How does the systolic volume and cardiac output change in heart failure?

A. the systolic volume decreases, but cardiac output increases

B\*.both indices decrese

C. the systolic volume increases, but cardiac output decreases

D. both indices increase

E. both indices are maintain constant

60. How does thermoregulation change in hypothyroidism?

A. increases thermogenesis; thermolysis increases; body temperature is constant

B. thermogenesis increases; thermolysis unchanged, decreased body temperature

C. thermogenesis and thermolysis are reduced with decreased body temperature

D. thermogenesis is unchanged; enhanced thermolysis; decreased body temparature

E\*.thermogenesis decreases; thermolysis increases, decreased body temperature

61. How is cholemia reproduced in the frog?

A\*.thereby of bile administration in the spinal lymphatic duct

B. thereby of oleos suspension injection in heart

C. thereby intraperitoneal administration of HCl

D. thereby of bile acids administration in the spinal lymphatic sac

E. thereby of cholesterol administration in the spinal lymphatic sac

62. How is frog cardiac activity modified in experimental cholemic syndrome?

A. tachycardia

B. fibrillation

C. no change

D. extrasystole

E\*.cardiac stop

63. How is heart function modified in the frog due to KCl solution administration?

A. tachycardia appears

B\*.bradycardia appears

C. extrasystole appears

D\*.cardiac stop in diastole

E. cardiac stop in systole

66. How is hyperaldosteronism manifested?

A. increased resistance of peripheral vessels

B. excretory acidosis

C. urine hyperosmolarity

D\*.urine hypoosmolarity

E\*.excretory alkalosis

67. How is hyperaldosteronism manifested?

A. muscle hypotonus caused by hypercloremia

B. muscle hypotonus caused by hypernatremia

C. muscle hypotonus caused by hypercalcemia

D\*.muscle hypotonus caused by hypokalemia

E. muscle hypotonus caused by hyperkalemia

68. How is hyperaldosteronism manifested?

A. arterial hypertension which is in relation with izotonic hyperhydration

B. arterial hypertension which is in relation with hypotonic hyperhydration

C. arterial hypertension due to increased sensibility of the heart to catecholamines

D\*.arterial hypertension which is in relation with hypertonic hyperhydration

E\*.arterial hypertension caused by increased preload

69. How is hypoaldosteronism manifested?

A\*.arterial hypotension caused by hypoosomolar dehydration

B. arterial hypotension caused by hyperosmolar dehydration

C\*.hyperosmolarity of the urine

D. hypoosomolarity of the urine

E. cell dehydration

70. How is hypoaldosteronism manifested?

A. arterial hypotension caused by reduced sensitivity of the heart and blood vessels to CA

B\*.arterial hypotension caused by hypoosmolar dehydration

C. arterial hypotension caused hyperosmolar dehydration

D. arterial hypotension caused by izoosmolar dehydration

E\*.arterial hypotension caused by decreased preload

71. How is intestinal transit affected in case of stomach hypersecretion with hyperacidity?

A. increases

B\*.decreases

C. doesn't change

D\*.frequent constipation

E. diarrhea

72. How is manifested sinus bradycardia on ECG in the frog?

A. enlargement of QRS complex

B\*.increased R-R interval

C. decreased R-R interval

D. increased P-Q interval

E. elevation of S-T segment

73. How is manifested sinus tachycardia on ECG in the frog ?

A. enlargement of QRS complex

B. increased R-R interval

C\*.decreased R-R interval

D. increased P-Q interval

E. elevation of S-T segment

74. How is manifested ventricular extrasystole on ECG in the frog ?

A\*.enlargement of QRS complex

B\*.deformation of QRS complex

C\*.appearance of premature QRS complex

D. increased P-Q interval

E. elevation of S-T segment

75. How is mouse behavior changed in parenteral administration of gastric juice?

A. sleepiness

B. agitation

C\*.not change

D. seizures

E. hypodynamics

76. How is mouse behavior changed in parenteral administration of large gut content?

A. motor inhibition

B. motor activation

C. sleepiness

D\*.seizures

E\*.coma

77. How is mouse behavior changed in parenteral administration of small intestine content?

A\*.sleepiness

B. seizures

C. not change

D. motor excitation

E\*.motor inhibition

78. How is mouse diuresis modified in pituitary extract administration?

a. polyuria

\*b. oliguria

\*c. anuria

\*d. hypersthenuria

e. isosthenuria

79. How is performed predominant homeometric hyperfunction of the myocardium?

a. by tachycardia

b. by Bainbridge reflex

c. by increasing systolic pressure

\*d. by increasing contractility of the myocardium

e. by increasing excitability of the myocardium

80. How is the frog heart function modified due to KCl solution administration?

a. tachycardia appears

\*b. bradycardia appears

c. extrasystole appears

d. paroxysmal tachycardia appears

e. cardiac stop in systole

81. How is the frog heart function modified due to KCl solution administration?

a. tachycardia appears

b. paroxysmal tachycardia appears

c. extrasystole appears

\*d. cardiac stop in diastole

e. cardiac stop in systole

82. How mouse diuresis is modified in hyperglycemic model?

\*a. polyuria

b. oliguria

c. anuria

d. isosthenuria

e. hyposthenuria

83. How mouse diuresis is modified in hyperglycemic model?

\*a. polyuria

b. oliguria

c. anuria

d. isosthenuria

\*e. hypersthenuria

84. How mouse diuresis is modified in hyperglycemic model?

a. hyposthenuria

b. oliguria

c. anuria

d. isosthenuria

\*e. hypersthenuria

85. How mouse diuresis is modified in hyperosmolar model?

\*a. it decreases initially

b. it increases initially

c. no any changes

\*d. it increases after 1 hour

e. it decreases after 1 hour

86. How the frog breathing is modified in experimental cholemic syndrome?

a. respiratory frequency increases

\*b. respiratory frequency decreases

c. respiration amplitude rises

\*d. respiration amplitude falls

e. both amplitude and frequency do not change

87. How the frog cardiac activity is modified in experimental cholemic syndrome?

a. tachycardia

\*b. bradycardia

c. no change

d. extrasystole

\*e. cardiac stop

88. How the frog motor spinal reflexes change in experimental cholemia?

\*a. time of reflexes appearance increases

b. reflex speed increases

c. no any changes

\*d. sensibility to painful stimuli decreases

e. sensibility to painful stimuli increases

89. How the frog motor spinal reflexes change in experimental cholemia?

a. sensibility to painful stimuli does not change

b. reflex speed increases

c. no any changes

\*d. sensibility to painful stimuli decreases

e. sensibility to painful stimuli increases

90. How the mouse diuresis is modified in pituitary extract administration?

a. polyuria

b. hyposthenuria

\*c. anuria

\*d. hypersthenuria

e. isosthenuria

91. How the mouse diuresis is modified in pituitary extract administration?

a. polyuria

\*b. oliguria

c. hyposthenuria

\*d. hypersthenuria

e. isosthenuria

92. How the mouse diuresis is modified in pituitary extract administration?

a. polyuria

\*b. oliguria

\*c. anuria

d. hyposthenuria

e. isosthenuria

93. How the stomachal tonus and motility is changed in hyperchlorhydria?

a. reduced tonus

\*b. increased tonus

\*c. stomachal chymostasis

d. vomiting

e. accelerated stomachal evacuation

94. How the stomachal tonus and motility is changed in hypochlorhydria?

A\*.reduced tonus

B. increased tonus

C. stomachal chymostasis

D. vomiting

E\*.accelerated stomachal evacuation

95. How was acute tubular necrosis modulated in mice?

A. thereby subcutaneous administration of HCl solution

B. thereby subcutaneous administration of NaCl solution

C. thereby subcutaneous administration of AgNO3 solution

D\*.thereby intraperitoneal administration of sublimate solution

E. thereby intraperitoneal administration of AgNO3 solution

96. How was heart function changed in experimental infarction?

A\*.tachycardia followed by bradycardia

B. bradycardia followed by tachycardia

C\*.decreased contractile force

D. connective tissue developed

E. increased contractile force

97. How was heart function changed in experimental infarction?

A\*.tachycardia followed by bradycardia

B. bradycardia followed by tachycardia

C. increased contractile amplitude

D. developed connective tissue

E. increased contractile force

98. How was intestinal autointoxication reproduced in the mouse?

A. thereby of small intestine content administration

B\*.thereby of large intestine content administration

C. thereby of stomach juice administration

D. thereby of bile administration

E. thereby of pancreatic juice administration

99. How was Kratschmer protecting reflex demonstrated in rabbit?

A. thereby of both nostrils closing

B. thereby of dioxide carbon inhalation

C\*.thereby of ammonia inhalation

D. thereby of monoxide carbon inhalation

E. thereby of one nostril closing

100. How was modeled acute adrenocortical insufficiency in rats?

A. due to ligation of suprarenal arteries

B. due to administration of drugs that block cholesterol metabolism

C\*.due to bilateral surgical removal of adrenal glands

D. due to unilateral surgical removal of adrenal glands

E. due to administration of cytostatics

101. In what anatomical area venous stasis is developed in case of left ventricular failure?

A. in portal vein

B. in inferior members

C. in the liver

D. in systemic circulation

E\*.in pulmonary circulation

102. In what cases are develop porto – caval anastomosis?

A. systemic hypertension

B\*.portal vein hypertension

C. hypovolemic shock

D. hypervolemia

E. chronic arterial hypotension

103. In what disorder can be attested leucocyturia?

\*a. urethritis

b. glomerulonephritis

c. acute tubular necrosis

d. congenital tubulopathy

e. nephritic syndrome

104. In what disorder can be attested leucocyturia?

\*a. inflammation of the pelvicalyceal system

b. glomerulonephritis

c. acute tubular necrosis

d. congenital tubulopathy

e. nephritic syndrome

105. In what disorder can be attested leucocyturia?

\*a. inflammation of the urinary bladder

b. glomerulonephritis

c. acute tubular necrosis

d. congenital tubulopathy

e. nephritic syndrome

106. In what disorders can be attested hyposthenuria?

a. diabetes mellitus

\*b. diabetes insipidus

c. acute glomerulonephritis

d. dehydration

\*e. hyperhydration

107. In what disorders can be attested isosthenuria?

\*a. chronic renal failure

b. acute glomerulonephritis

c. cystitis

d. urethritis

e. hypoaldosteronism

108. In what disorders can be found hypersthenuria?

a. diabetes mellitus

b. diabetes insipidus

c. tubular necrosis

\*d. dehydration

e. hyperhydration

109. In what disorders can be found hyposthenuria?

a. diabetes mellitus

\*b. diabetes insipidus

\*c. tubular necrosis

d. acute glomerulonephritis

e. dehydration

110. In what disorders can develop dysmetabolic heart failure?

a. insufficiency of tricuspidian valve

\*b. myocardial necrosis

\*c. inflammation of the myocardium

d. aortic stenosis

e. hypovolemia with hemoconcentration

111. In what pathologic condition can be attested overload of the heart with resistence?

a. mitral valve insufficiency

b. aortic valve insufficiency

\*c. hemoconcentration

d. insufficiency of pulmonary valve

e. hypervolemia with hemodilution

112. In what pathologic conditions can be attested overload of the heart with volume?

a. mitral stenosis

\*b. pulmonary valve insufficiency

c. hypovolemic shock

d. hypercatecholaminemia

\*e. hypervolemia

113. In what state is found the primary absolute erythrocytosis?

1. anemia
2. incoercible vomiting
3. kidneys diseases
4. hypoxia
5. \*erythroblastic leukemia

114. Storage of which acids develop acidosis in patients with type I diabetes mellitus?

1. \* acetone
2. pyruvic acid
3. \* acetoacetic acid
4. oxalacetic acid
5. acetic acid
6. To what stressful factor were subjected laboratory animals with acute experimental hypocoticism?
7. hypobaric hypoxia
8. artificial hypervolemia
9. inanition
10. \* cold water
11. electrical shock
12. To what stressful factor were subjected laboratory animals with acute experimental hypocoticism?
13. hypobaric hypoxia
14. \* physical effort
15. inanition
16. artificial hypervolemia
17. electrical shock
18. To what stressful factors were subjected laboratory animals in experimental hypocoticism?
19. hypobaric hypoxia
20. \* physical effort
21. inanition
22. \* cold water
23. electrical shock

118. What alveolar air parameters reduce alveolar capillary diffusion?

A. oxygen partial pressure increases

B\*.oxygen partial pressure decreases

C\*.dioxide carbon partial pressure increases

D. dioxide carbon partial pressure decreases

E. nitrogen partial pressure increases

1. What are the biochemical changes in the blood in liver failure?

A\*.reduced prothrombin level

B. increased albumin level

C. reduced uric acid level

D. increased HDL level

E. reduced ammonia level

1. What are biochemical changes in the blood in liver failure?

A\*.hyperammoniemia

B\*.reduced albumin level

C\*.reduced prothrombin level

D. reduced urea level

E. reduced ammonia level

1. What are biochemical changes in the blood in liver failure?

A\*.increased level of aromatic aminoacids

B. increased albumin level

C. reduced uric acid level

D. increased HDL level

E. reduced ammonia level

1. What are biochemical changes in the blood in liver failure?

A\*.reduced albumin level

B. increased albumin level

C. reduced uric acid level

D. increased HDL level

E. reduced ammonia level

1. What are blood biochemical changes in cholestasis?

A\*.increased level of conjugated bilirubin

B\*.increased level of biliary salts in the blood

C\*.decreased prothrombin level

D. hyperbilirubinemia with unconjugated bilirubin

E. hyperlipidemia

1. What are blood biochemical changes in cholestasis?

A\*.hyperbilirubinemia with conjugated bilirubin

B. reduced level of biliary salts in the blood

C. increased level of ALAT and ASAT

D. hyperbilirubinemia with unconjugated bilirubin

E. hyperlipidemia

1. What are blood biochemical changes in cholestasis?

A\*.hypercholesterolemia

B. reduced level of biliary salts in the blood

C. increased level of ALAT and ASAT

D. hyperbilirubinemia with unconjugated bilirubin

E. hyperlipidemia

1. What are causes of eosinophilia?

A\*.insufficiency of adrenal glands

B. allergic diseases

C. cocci infection

D\*.parasites

E. chronic specific infections

127. What are causes of erectile dysfunction in men in type I diabetes mellitus?

A. high level of HDL which lead to atherosclerosis of pudenda artery

B. high level of free fatty acids in the blood which lead to atherosclerosis of pudenda artery

C. hypersecretion of FSH and LH caused by high level of corticosteroids in insulin deficiency

D\*.high level of VLDL and LDL which lead to atherosclerosis of pudenda artery

E. hyposecretion of FSH and LH caused by high level of corticosteroids in insulin deficiency

128. What are causes of hypokalemia in chronic liver affection?

a. disruption of protein synthesis

b. deregulation of glycolysis

\*c. aldosterone metabolism disorder

d. impaired glycogenolysis

e. disturbances in the metabolism of glucocorticoids

129. What are causes of intestinal auto-intoxications?

\*a. excessive formation of products of putrefaction in the gut

\*b. hepatic failure

c. kidney failure

d. enteritis

e. constipation

130. What are causes of neutrophilia?

a. insufficiency of adrenal glands

b. allergic diseases

\*c. cocci infection

d. parasites

e. chronic specific infections

1. What are causes of polydipsia in diabetes mellitus type I?
2. \* hypovolemia with hyperosmolar dehydration
3. hypovolemia with izoosmolar dehydration
4. hypovolemia with hypoosmolar dehydration
5. direct excitation of osmoreceptors in hypothalamus caused by hyperglycemia
6. low level of ADH in insulin deficiency
7. What are causes of secondary endocrine disorders?
8. disorders of endocrine hypothalamus
9. \* adenohypophysis disorders
10. neurohypophysis disorders
11. disorders of peripheral endocrine glands
12. disorders of peripheral hormonal reception
13. What are causes of tertiary endocrine disorders?
14. \* disorders of endocrine hypothalamus
15. adenohypophysis disorders
16. neurohypophysis disorders
17. disorders of peripheral endocrine glands
18. disorders of peripheral hormonal reception
19. What are the causes of the deep and slow (stenotic) respiration?
20. bronchial epithelium edema
21. spasm of lower air ways
22. \* compression of upper air ways
23. \* larynx edema
24. bronchial asthma
25. What are causes of the deep and slow (stenotic) respiration?
26. \* obturation of the superior air ways
27. spasm of lower air ways
28. bronchial epithelium edema
29. \* larynx edema
30. bronchial asthma
31. What are causes of the deep and slow (stenotic) respiration?
32. \* obturation of upper air ways
33. spasm of lower air ways
34. \* compression of upper air ways
35. \* larynx edema
36. bronchial asthma
37. What are causes of the deep and slow (stenotic) respiration?
38. \* obturation of upper air ways
39. spasm of lower air ways
40. \* compression of upper air ways
41. bronchial epithelium edema
42. bronchial asthma
43. What are causes of the deep and slow (stenotic) respiration?
44. bronchial epithelium edema
45. spasm of lower air ways
46. \* compression of upper air ways
47. \* larynx edema
48. bronchial asthma
49. What are changes in carbohydrate metabolic processes in liver failure?
50. \* increased glucose level after meal
51. \* reduced glucose level in fasting
52. development of fructosemia
53. \* reduced glycogen storages in the liver
54. increased glycogen storages in the liver
55. What are changes in lipid metabolic processes in hepatic failure?
56. increased lipolysis in the liver
57. \* liver steatosis
58. \* hyperlipidemia with VLDL
59. hyperlipidemia with HDL
60. \* development of hyperlipidemia with non-esterified free fatty acids
61. What are changes in protein metabolic processes in liver failure?
62. \* development of hyperglobulinemia
63. \* development of hypoalbuminemia
64. disturbances in synthesis of gama-globulins
65. increased urea level in the blood
66. \* increased level of aminoacids in the blood
67. What are changes of the peripheral blood smear in iron deficiency anemia?
68. megalocytosis
69. \* hypochromic erythrocytes
70. \* microcytosis
71. \* anulocytosis
72. Drepanocytosis
73. What are characteristic manifestations for left heart failure?
74. splenomegaly
75. hepatomegaly
76. \* tachycardia
77. venous stasis in systemic circulation
78. \* reduced cardiac output
79. What are characteristic manifestations for left heart failure?
80. bradycardia
81. hepatomegaly
82. \* tachycardia
83. \* venous stasis in pulmonary circulation
84. venous stasis in systemic circulation
85. What are characteristic manifestations for right heart failure?
86. pulmonary edema
87. \* accumulation of transudate in abdominal cavity
88. venous stasis in pulmonary circulation
89. \* venous stasis in systemic circulation
90. pulmonary hypertension
91. What are characteristic manifestations for right heart failure?
92. pulmonary edema
93. \* hepatomegaly
94. venous stasis in pulmonary circulation
95. \* venous stasis in systemic circulation
96. reduced cardiac output
97. What are endocrine functions of the kidneys?

A\*.release of erythropoietin

B\*.paracrine secretion of vasodilator prostaglandins

C. release of angiotensin

D\*.local activation of kallikrein system

E\*.renin production

1. What are the main syndromes in acute renal failure?

A\*.urinary syndrome

B\*.humoral syndrome

C\*.clinical syndrome

D. nephrotic syndrome

E. nephritic syndrome

149. What are manifestations of clinical syndrome in acute renal failure?

A. anemia

B\*.hypertension

C\*.disturbances of breathing rhythm

D\*.neuropsychic disorders

E. hypothermia

1. What are manifestations of gastrointestinal autointoxication?

A\*.arterial hypotension

B. arterial hypertension

C. diarrhea

D\*.depression

E. hypoglycemia

1. What are manifestations of humoral syndrome in acute renal failure?

A\*.hyperazotemia

B\*.hyperhydration

C. acidosis

D. anemia

E. alkalosis

1. What are manifestations of urinary syndrome in acute renal failure?

A\*.oliguria

B\*.isosthenuria

C. hyposthenuria

D. hypersthenuria

E. hematuria

1. What are the mechanisms of frog motor activity disorders in experimental cholemia?

A\*.inhibition of the spinal motor neurons

B. excitation of the spinal nervous centers

C. increase of receptor sensitivity

D. decrease of receptor sensitivity

E. neuromuscular junction blocking

154. What are mechanisms of frog respiration change in experimental cholemic syndrome?

A. excitation of the respiratory center do to bile acids action

B\*.inhibition of the respiratory center do to bile acids action

C. CO2 blood pressure elevation

D. blood PO2 reducing

E. blood PO2 increasing

1. What are mechanisms of mouse diuresis change in experimental hydremia?

A\*.effective filtration pressure increases

B\*.capillary hydrostatic pressure increases

C. oncotic pressure enhancement in glomerular capillaries

D. hydrostatic pressure diminution in afferent arteriole

E. tubular absorption decrease

1. What are mechanisms of polyuria in a mouse hyperglycemic model?

A\*.effective filtration pressure enhancement

B\*.increase of osmotic pressure in primary urine

C. increase of osmotic pressure in secondary urine

D. intensification of the tubular absorption

E\*.insufficient reabsorption of the filtrated glucose

1. What are mechanisms of polyuria in a mouse hyperglycemic model?

A\*.effective filtration pressure enhances

B\*.increase of osmotic pressure into primary urine

C. increase of osmotic pressure into secondary urine

D. intensification of tubular absorption

E. increase of osmotic pressure in tubular interstitium

1. What are mechanisms of polyuria in a mouse hyperglycemic model?

A\*.effective filtration pressure enhances

B. increase of osmotic pressure in tubular interstitium

C. increase of osmotic pressure into secondary urine

D. intensification of tubular absorption

E\*.insufficient reabsorption of the filtrated glucose

158. What are metabolic effects of glucocorticoids?

a. enhanced glycogenogenesis with hyperglycemia and insulin resistence

b. enhanced lypolysis leading to transport hyperlipidemia with HDL

\*c. enhanced lypolysis leading to transport hyperlipidemia with free fatty acids

d. enhanced lypolysis leading to transport hyperlipidemia with VLDL and LDL

\*e. reduced peripheral up-take of glucose with insulin resistence

159. What are metabolic effects of glucocorticoids?

a. enhanced glycolysis with hyperglycemia

b. enhanced gluconeogenesis from free fatty acids with hyperglycemia

\*c. enhanced lypolysis with transport hyperlipidemia

d. intense oxidation of free fatty acids in the liver with high level of HDL and LDL in the blood

e. enhanced lypolysis with retention hyperlipidemia

160. What are metabolic effects of glucocorticoids?

a. enhanced glycogenogenesis with hyperglycemia

b. diminished proteosynthesis with positive nitrogen balance

\*c. intensified protein breakdown with negative nitrogen balance

\*d. intensified gluconeogenesis from glycerol with hyperglycemia

e. intensified gluconeogenesis from free fatty acids with hyperglycemia

161. What are pathogenic factors of respiratory distress syndrome in adults?

a. pulmonary arterial hypertension

\*b. alveolar-capillary membrane permeability increase

c. pulmonary parenchyma elasticity increase

\*d. alveolar edema

\*e. intra-alveolar liquid coagulation

162. What are postrenal factors of acute renal failure?

a. action of nephrotoxic factors

b. massive hemolysis

\*c. obstruction of urinary pathway

d. constriction of renal artery

e. dilation of renal artery

163. What are prerenal causes of acute renal failure?

\*a. shock of any origin

b. reduced oncotic pressure in the blood

c. dilation of renal artery

d. hyperhydration

e. hemodilution

164. What are prerenal causes of acute renal failure?

\*a. stenosis of renal artery

b. reduced oncotic pressure in the blood

c. dilation of renal artery

d. hyperhydration

e. hemodilution

165. What are prerenal causes of acute renal failure?

\*a. severe hypovolemia

\*b. obstruction of renal artery

c. dilation of renal artery

d. hyperhydration

e. hemodilution

166. What are prerenal causes of acute renal failure?

\*a. heart failure

\*b. reduced peripheral vascular resistence

c. dilation of renal artery

d. hyperhydration

e. hemodilution

167. What are prerenal causes of acute renal failure?

\*a. severe hypovolemia

b. reduced oncotic pressure in the blood

c. dilation of renal artery

d. hyperhydration

e. hemodilution

168. What are signs of absolute leucocytosis?

A. proportional increase in all leucocytes in the blood

B\*.increased leucocyte number in a unit of blood

C. increased percents of leucocytes in the leucogramm

D\*.increased number of young form of leucocytes in the blood

E. concomitant increase in erythrocytes and leucocytes number in the blood

169. What are somatic manifestations GH hypersecretion in children?

A. gigantism, acromegaly, splanhnomegaly

B\*.gigantism, splanhnomegaly

C. acromegaly, splanhnomegaly

D. gigantism, acromegaly

E. acromegaly, atrophy of internal organs

170. What are the biochemical changes of cholestasis in the blood?

A\*.decreased level of prothrombin

B. hyperbilirubinaemia with free bilirubin

C. hyperlipidemia

D. higher levels of chylomicrons in the blood

E. increase activity of aminotransferases

171. What are the causes of antidiuretic hormone hypersecretion?

A. izotonic hyperhydration

B. hypotonic hyperhydration

C\*.hypertonic dehydration

D\*.izotonic dehydration

E. low level of potasium in the blood

172. What are the causes of chronic renal failure?

A. primary and secondary glomerular disorders

B. tubulo-interstitial disorders

C\*.vascular renal disorders

D\*.severe hypovolemia

E\*.acute renal failure

173. What are the causes of GH-releasing hormone hypersecretion?

A. basophilic adenoma of pituitary

B\*.acidophilic adenoma of pituitary

C. chromophobe adenoma of pituitary

D. adenoma of posterior lobe of pituitary

E\*.hypersecretion of somatostatin-releasing factor

174. What are the causes of GH-releasing hormone hyposecretion?

A. atrophy of basophilic pituitary cells

B. atrophy of acidophilic pituitary cells

C. atrophy of chromophobe pituitary cells

D. atrophy of posterior pituitary lobe

E\*.ischemic stroke

175. What are the causes of impaired function of anterior pituitary?

A. disruption of release and transport of liberins and statins to adenohypophysis through systemic circulation

B. disruption of release and transport of liberins and statins to adenohypophysis and neurohypophysis through systemic circulation

C\*.disruption of release and transport of liberins and statins to adenohypophysis through portal circulation

D. disruption of release and transport of liberins and statins to neurohypophysis through systemic circulation

E. disruption of release and transport of liberins and statins to neurohypophysis through portal c.

176. What are the causes of insufficient exocrine secretion of the pancreas?

A\*.obstruction of the pancreatic duct

B\*.sympathicotonia

C. duodenal ulcer

D. hyperplasia of alpha cells

E. hyperplasia of beta cells

177. What are the causes of insufficient exocrine secretion of the pancreas?

A\*.abusive alcohol consumption

B\*.occlusion of pancreatic duct

C\*.activation of sympathetic tone

D. vagotonia

E. duodenal ulcer

178. What are the causes of insufficient exocrine secretion of the pancreas?

A\*.stricture of the pancreatic duct

B\*.pancreatic hypoperfusion

C. duodenal ulcer

D. pancreatic alpha cell hyperplasia

E. beta cell hyperplasia

179. What are the causes of insufficient exocrine secretion of the pancreas?

A\*.stricture of the pancreatic duct

B\*.pancreatic hypoperfusion

C. duodenal ulcer

D. pancreatic alpha cell hyperplasia

E. beta cell hyperplasia

180. What are the causes of insufficient exocrine secretion of the pancreas?

A\*.ahlorhidria

B. acetilcholine hyposecretion

C\*.vagotomia

D. hyperplasia of alpha cells

E. hyperplasia of beta cells

181. What are the causes of intrinsic acute renal failure?

A\*.action of nephrotoxic factors

B\*.massive hemolysis

C\*.Crush syndrome

D. constriction of renal artery

E. dilation of renal artery

182. What are the causes of left heart failure?

A. pulmonary emphysema

B\*.aortic stenosis

C\*.systemic hypertension

D. pneumosclerosis

E. pulmonary hypertension

183. What are the causes of left heart failure?

a. pulmonary emphysema

\*b. aortic stenosis

\*c. systemic hypertension

d. pneumosclerosis

e. pulmonary hypertension

184. What are the causes of portal hypertension in hepatic cirrhosis?

a. right ventricular failure

b. hepatomegaly

c. insufficiency of porto – cava collaterals

\*d. capillarisation of hepatic sinusoids

\*e. increases the intrahepatic vessels resistance

185. What are the causes of prolactin hypersecretion?

a. pituitary adenoma from basophilic cells

b. primary hyperthyroidism

c. pituitary adenoma from cromofobe cells

d. tertiary hypothyroidism

\*e. primary hypothyroidism

186. What are the causes of secondary hyperaldosteronism?

\*a. ADH deficiency

b. ADH excess

c. hyposecretion of atrial natriuretic hormone

d. aldosteron-secreting tumor in adrenal cortex

\*e. hypersecretion of atrial natriuretic hormone

187. What are the causes of steatorrhea?

\*a. obstruction of choledocus

\*b. deficiency of pancreatic lipase

\*c. liver cirrhosis

d. cholemia

e. hyperlipidemia

188. What are the changes of blood cells counts in hyperproliferation of red bone marrow?

a. increasing the number of erythroblasts

b. increasing the number of stem cells

\*c. increasing number of young red blood cells

d. intensification of ineffective erythropoiesis

e. erythrocytosis with hemodilution

189. What are the changes of myelogram in hyperproliferation of red bone marrow?

\*a. increased number of erythroblasts and normoblast

b. increased number of erythroblasts and megaloblasts

\*c. substitution of yellow bone marrow with red bone marrow

d. substitution of red bone marrow with fat

e. increased number of mature erythrocytes

190. What are the consequences of choledocus obstruction?

\*a. acholia

\*b. lipid maldigestion

c. hyperlipidemia

d. higher level of chylomicrons in the blood

e. increased level of aminotransferase

191. What are the consequences of choledocus obstruction?

\*a. acholia

\*b. higher level of conjugated bilirubin

c. hyperlipidemia

d. increased level of aminotransferases

e. higher level of free bilirubin

192. What are the consequences of disaccharide maldigestion?

\*a. hypovolemia

\*b. pancreatic insufficiency

c. constipation

d. hyperhydration

\*e. hypoglycemia

193. What are the consequences of lipid maldigestion?

\*a. steatorrhea

\*b. blood hypocoagulation

\*c. defficiency of vitamin K

d. hyperlipidemia

e. constipation

194. What are the consequences of protein maldigestion?

\*a. hypoalbuminemia

b. proteinuria

c. reduced creatinine level in the blood

d. reduced urea level in the blood

e. reduced aminoacid concentration in the blood

195. What are the hallmarks of absolute secondary erythrocytosis?

\*a. the count of reticulocyte above 2%

\*b. erythrocytes count greater than 5X1012 / L

c. lack of reticulocytes

d. the total volume of blood less than 7% of body weight

e. erythroblastic presence in the blood

196. What are the manifestations of ADH hypersecretion?

a. hypertonic dehydration; urine hyperosmolarity

b. hypotonic hyperhydration; urine hypoosmolarity

\*c. hypotonic hyperhydration; urine hyperosmolarity

d. izotonic hyperhydration; urine hyperosmolarity

e. hypotonic dehyration; urine hypoosmolarity

197. What are the manifestations of glucocorticoid hypersecretion?

\*a. increased neutrofil count

b. reduced neutrofil count

\*c. lymphocytopenia

d. lymphocytosis

e. anemia

198. What are the manifestations of glucocorticoid hypersecretion?

A. truncal obesity due to enhanced lypogenesis which is a direct effect of high level of cortisol in hypercorticosolism

B\*.truncal obesity due to enhanced lypogenesis which is effect of high insulin level in hypercorticosolism

C. truncal obesity due to enhanced lypogenesis which is a direct effect of high level of androgen in hypercorticosolism

D. predisposition to viral infection caused by lymphocytosis

E\*.predisposition to viral infection caused by lymphocytopenia

199. What are the manifestations of glucocorticoid hypersecretion?

A. anemia

B. increased resistance to viral infections

C\*.predisposition to allergic reactions

D. susceptibility to parasitic diseases

E\*.erythrocytosis

200. What are the manifestations of glucocorticoid hypersecretion?

A. general obesity caused by increased lipogenesis which is effect of insulin

B\*.muscular hypotonus caused by hypokalemia

C. muscular hypotonus caused by hyperkalemia

D\*.excretory alcalosis

E. metabolic alkalosis

201. What are the manifestations of glucocorticoid hypersecretion?

A. hyperglycemia caused by enhanced glycolysis

B. hyperglycemia caused by increased gluconeogenesis from free fatty acids

C\*.insulin-resistence

D\*.reduced peripheral up-take of glucose

E. increased periperal up-take of glucose

202. What are the manifestations of glucocorticoid hypersecretion?

A\*.high level of VLDL and LDL and susceptibility to atherosclerosis

B. high level of HDL and LDL and susceptibility to atherosclerosis

C. high level of VLDL,LDL and HDL and susceptibility to atherosclerosis

D\*.insulin-resistence caused by reduced peripheral uptake of glucose

E. insulin resistence caused by increased peripheral uptake of glucose

203. What are the manifestations of glucocorticoid hypersecretion?

A. increased eozinofil count

B\*.reduced eozinophil count

C. lymphocytosis due to reduced apoptosis in lymphoid tissue

D\*.lymphocytopenia due to increased apoptosis in lymphoid tissue

E. anemia

204. What are the manifestations of glucocorticoid hyposecretion?

A. skin depigmentation caused by low level of POMC

B. arterial hypotension with compensatory tachycardia

C\*.skin hyperpigmentation caused by high POMC level

D. skin hyperpigmentation caused by low level of POMC

E\*.arterial hypotension with bradycardia

205. What are the manifestations of glucocorticoid hyposecretion?

A. fast sexual development in female teenages

B. hyposexuality in males

C. hypersexuality in females

D\*.hyposexuality in females

E. hypersexuality in males

* 1. What are the manifestations of glucocorticoids hypersecretion?

A\*.hypercalcemia with calciuria

B. hypocalcemia which leads to hyperexcitability

C. blood and urine hypoosmolarity

D\*.blood hyperosmolarity

E. urine hyperosmolarity

207. What are the manifestations of hyperaldosteronism?

A. increased peripheral vascular resistence caused by hypokalemia

B\*.on ECG low amplitude of T wave; additional U wave

C. on ECG high amplitude of T wave; additional U wave

D. desensitization of vascular myocytes to catecholamines with vasodilation

E\*.increased peripheral vascular resistence caused by hypernatremia

208. What are the manifestations of primary hyposecretion of glucocorticosteroids?

A. skin hyperpigmentation caused by low level of POMC

B. hypertonic dehydration

C\*.skin hyperpigmentation caused by high level of POMC

D\*.hypotonic dehydration

E. insufficiency of melanin caused by low MSH

209. What are the manifestations of tertiary hyposecretion of glucocorticosteroids?

A. hypertonic dehydration

B\*.skin depigmentation caused by low level of POMC

C\*.hypotonic dehydration

D. skin hyperpigmentation caused by high level of POMC

E. skin depigmentation caused by high level of POMC

210. What are the mechanisms of functional exhaustion and cardiosclerosis of hypertrophied myocardium?

A\*.relative coronary insufficiency

B\*.hypermetabolic hypoxia of the myocardium

C\*.overloading of intact cardiomyocytes

D. increased pressure within pericardial cavity

E. decreased perfusion pressure in coronary

211. What are the mechanisms of increased blood pressure in painful stimulation?

A\*.increased renin secretion

B. increased number of adrenoreceptors

C\*.increased peripheral vascular resistence

D. activation of MAO

E. activation of kallikrein-kinin system

212. What are the metabolic effects of glucocorticoids?

A. increases protein breakdown with positive nitrogen balance

B\*.increased lipolysis with transport hyperlipidemia

C. intensifies glycogenesis with hyperglycemia

D. intensifies gluconeogenesis from free fatty acids leading to hyperglycemia

E. increased lipolysis leading to high level of HDL in the blood

213. What are the mouse diuresis changes in acute tubular necrosis?

A\*.initial polyuria

B\*.late oliguria

C. initial oliguria

D. late polyuria

E. no any changes

214. What are the principal pathogenetical links of pulmonary emphysema?

\*a. alveoli elasticity diminution

b. alveoli compliance diminution

c. collagen decrease in the alveolar walls

\*d. elastic fibers diminution in the alveolar wall

\*e. collagen enhancement in the alveolar walls

215. What are the signs of primary absolute erythrocytosis (erythremia)?

\*a. neutrophilia

\*b. erythrocytes greater than 6x1012 / L

\*c. reticulocyte count exceed 2.5%

d. thrombocytopenia

e. lack of reticulocytes

216. What are the signs of prolactin hypersecretion at women?

a. low level of estrogen caused by hyposecretion of FSH

\*b. low level of estrogen caused by hyposecretion of LH

\*c. reduced peripheral up-take of glucose with insulin resistance

d. increased peripheral up-take of glucose and sensitization of insulin receptors

e. hyperglycemia caused by enhanced glycogenolysis

218. What are the signs of prolactin hypersecretion in men?

\*a. decreased libido caused by hyposecretion of LH

\*b. oligozoospermia caused by hyposecretion of FS

c. enhanced glycogenolysis with hyperglycemia

d. oligozoospermia caused by hyposecretion of LH

e. decrease of libido caused by hyposecretion of FSH

219. What are the sources of proteolytic enzymes which lyse pulmonary alveoli?

a. mastocytes

\*b. polymorphonuclear cells

\*c. macrophages

\*d. exocrine pancreas

e. hepatocytes

220. What are the traits of the pulmonary emphysema?

\*a. increased residual volume

\*b. vital lung capacity decrease

c. residual volume diminution

\*d. expiratory dyspnea

e. pulmonary hyperventilation

221. What bacteriostatic factor is generated by neutrophil leukocytes?

\*a. lactoferrin

b. antibacterial antibodies

c. activated complement

d. histaminase

e. hyaluronidase

222. What can be a cause of left heart failure?

a. pulmonary emphysema

\*b. aortic stenosis

c. dehydration

d. pneumosclerosis

e. pulmonary hypertension

223. What can be causes of gastrointestinal autointoxication?

\*a. intensification of intestinal putrefaction

\*b. excessive intake of protein

\*c. excessive bacterial colonization in the intestine

\*d. liver insufficiency

e. diarrhea

234. What can be the cause of aclorhydria?

\*a. somatostatin hypersecretion

b. somatostatin hyposecretion

\*c. enteroglucagon hypersecretion

d. hypersecretion of gastrin

e. pancreatic hypersecretion

235. What can be the cause of aclorhydria?

\*a. neurotensin hypersecretion

b. hypertrophic gastritis

c. gastric ulcer

\*d. secretin hypersecretion

e. pancreatic hypersecretion

236. What can be the consequences of incorrective vomiting?

\*a. hypokaliemia

b. hyprekaliemia

c. metabolic acidosis

\*d. hypovolemia

\*e. activation of the renin-angiotensin-aldosterone system

237. What causes aclorhydria?

a. gastrinoma

\*b. atrophic and degenerative changes of the gastric mucosa

\*c. infiltrative forms of gastric cancer

d. hypertrophic gastritis

e. gastric ulcer

238. What can be the cause of aclorhydria?

A\*.neurotensin hypersecretion

B. hypertrophic gastritis

C. gastric ulcer

D\*.secretin hypersecretion

E. pancreatic hypersecretion

239. What can be the consequences of incoercitive vomiting?

A\*.hypopotasaemia

B. hypopotasaemia

C. metabolic acidosis

D\*.hypovolemia

E\*.activation of the renin-angiotensin-aldosterone system

240. What causes aclorhydria?

A. gastrinoma

B\*.atrophic and degenerative changes of the gastric mucosa

C\*.infiltrative forms of gastric cancer

D. hypertrophic gastritis

E. gastric ulcer

241. What causes aclorhydria?

A\*.gastrointestinal polypeptide hypersecretion

B. hypertrophic gastritis

C\*.creatine hypersecretion

D. hypersecretion of gastrin

E. pancreatic hypersecretion

242. What cells have Glut insulin-dependent receptors?

a. adipose cells which are equipped with GLUT- 2 receptors

b. skeletal muscle cells which are equipped with GLUT-2 receptors

c. renal epithelial cells which have GLUT-4 receptors

d. smooth muscle cells which have GLUT-2 receptors

\*e. skeletal muscles which have GLUT-4 receptors

243. What cells have Glut insulin-dependent receptors?

a. smooth myocytes which are endowed with GLUT-4 receptors

b. hepatocytes which are endowed with GLUT-4 receptors

c. adipocytes which are endowed with GLUT-2 receptors

\*d. adipocytes which are endowed with GLUT-4 receptors

e. hepatocytes which are endowed with GLUT-2 receptors

244. What cells have Glut insulin-dependent receptors?

\*a. adipocytes

b. neurons

c. renal epithelial cells

d. enterocytes

e. hepatocytes

245. What cells have insulin-dependent hexokinase?

a. leukocytes

b. adipocytes

c. renal epithelial cells

d. enterocytes

\*e. hepatocytes

246. What changes in hemogram can be attested in B12 deficiency anemia?

\*a. hemoglobin level in erythrocytes above 100 pg

\*b. average diameter of erythrocytes above 8 mkm

c. thrombocytosis

d. neutrophilia with left nuclear shift

e. neutrophilia with right nuclear shift

247. What changes in stomachal digestion can be attested in gastric hypochlorhydria?

a. polysaccharide maldigestion

b. lipid maldigestion

\*c. protein maldigestion

d. maldigestion of starch

e. improvement of stomachal digestion

248. What changes of lung ventilation refer to hypoventilation?

a. vital lung capacity increases

b. respiratory minute-volume increases

\*c. the rate of death anatomic space from respiratory minute-volume increases

d. the rate of death anatomic space from respiratory minute-volume decreases

\*e. respiratory minute-volume decreases

249. Which of the following can be pathogenetic mechanisms of cardiac edema?

a. hyperpermeability of biological membranes

\*b. activation of renin-angiotensin-aldosterone mechanism

c. hypoproteinemia

d. hypernatriemia

\*e. venous hyperemia

250. What changes of lung ventilation refer to hyperventilation?

A. vital lung capacity increases

B\*.respiratory minute-volume increases

C. the rate of death anatomic space from respiratory minute-volume increases

D\*.the rate of death anatomic space from respiratory minute-volume decreases

E. respiratory minute-volume decreases

251. What changes of lung ventilation refer to hyperventilation?

A. vital lung capacity increases

B\*.respiratory minute-volume increases

C. the rate of death anatomic space from respiratory minute-volume increases

D\*.the rate of death anatomic space from respiratory minute-volume decreases

E. respiratory minute-volume decreases

252. What changes of lung ventilation refer to hypoventilation?

A. vital lung capacity increases

B. respiratory minute-volume increases

C\*.the rate of death anatomic space from respiratory minute-volume increases

D. the rate of death anatomic space from respiratory minute-volume decreases

E\*.respiratory minute-volume decreases

253. What changes of lung ventilation refer to hypoventilation?

A. vital lung capacity increases

B. respiratory minute-volume increases

C\*.the rate of death anatomic space from respiratory minute-volume increases

D. the rate of death anatomic space from respiratory minute-volume decreases

E\*.respiratory minute-volume decreases

254. What changes of the alveolar air composition refer to hyperventilation?

A. partial pressure of oxygen decreases

B\*.partial pressure of oxygen increases

C\*.partial pressure of dioxide carbon decreases

D. partial pressure of dioxide carbon increases

E. partial pressure of nitrogen increases

255. What changes of the alveolar air composition refer to hyperventilation?

A. partial pressure of oxygen decreases

B\*.partial pressure of oxygen increases

C\*.partial pressure of dioxide carbon decreases

D. partial pressure of dioxide carbon increases

E. partial pressure of nitrogen increases

256. What changes of the alveolar air composition refer to hypoventilation?

A\*.partial pressure of oxygen decreases

B. partial pressure of oxygen increases

C. partial pressure of dioxide carbon decreases

D\*.partial pressure of dioxide carbon increases

E. partial pressure of nitrogen increases

257. What changes of the alveolar air composition refer to hypoventilation?

A\*.partial pressure of oxygen decreases

B. partial pressure of oxygen increases

C. partial pressure of dioxide carbon decreases

D\*.partial pressure of dioxide carbon increases

E. partial pressure of nitrogen increases

258. What changes of the arterial blood composition refer to hypoventilation?

A\*.oxygen pressure decreases

B. oxygen pressure increases

C. dioxide carbon pressure decreases

D\*.dioxide carbon pressure increases

E. nitrogen pressure increases

259. What changes of the arterial blood refer to hyperventilation?

A. oxygen pressure decreases

B\*.oxygen pressure increases

C\*.dioxide carbon pressure decreases

D. dioxide carbon pressure increases

E. nitrogen pressure increases

260. What changes of the arterial blood refer to hyperventilation?

A. oxygen pressure decreases

B\*.oxygen pressure increases

C\*.dioxide carbon pressure decreases

D. dioxide carbon pressure increases

E. nitrogen pressure increases

261. What changes of the arterial blood refer to hyperventilation?

A. oxygen pressure decreases

B\*.oxygen pressure increases

C\*.dioxide carbon pressure decreases

D. dioxide carbon pressure increases

E. nitrogen pressure increases

262. What substance is formed from proteins putrefaction in the intestine?

\*a. hydrogen sulfide

b. lactic acid

c. uric acid

d. acetate

e. carbon dioxide

263. What conditions enhance glucose use by neurons in the lack of insulin?

a. insulin-dependent glucokinase

b. insulin-dependent Glut receptors

c. insulin-dependent phosphorylase

d. insulin-dependent phosphatease

\*e. insulin-independent GLUT receptors

264. What digestive changes can be found in bile deficiency?

a. polysaccharide maldigestion

\*b. steatorrhea

\*c. intestinal atonia

d. amylorrhea

e. creatorrhea

265. What digestive changes can be found in exocrine defficiency of the pancreas?

\*a. polysaccharide maldigestion

\*b. protein maldigestion

\*c. lipid maldigestion

d. starch maldigestion

e. improvement of intestinal digestion

266. What digestive changes can be found in small intestine mucosal dysfunction?

a. disorders in breakdown of polysaccharides

\*b. disorders in breakdown of disaccharides

c. disorders in breakdown of polypeptide

d. disorders in breakdown of lipids

\*e. disorders in breakdown of dipeptide

267. What disorder is manifested by tubular proteinuria?

\*a. amyloidosis

b. glomerulonephritis

c. urolithiasis

d. hydronephrosis

e. nephrosclerosis

268. What disorder is manifested by tubular proteinuria?

\*a. inflammatory tubulopathy

b. glomerulonephritis

c. urolithiasis

d. hydronephrosis

e. nephrosclerosis

269. What disorders are manifested by tubular proteinuria?

\*a. dystrophic tubulopathy

\*b. amyloidosis

c. glomerulonephritis

d. urolithiasis

e. hydronephrosis

270. What disorders involves nephritic syndrome?

\*a. hematuria

\*b. arterial hypertension

c. hyperlipidemia

d. hypolipidemia

e. lipiduria

271. What disorders involves nephrotic syndrome?

\*a. massive proteinuria

\*b. hyperlipidemia

c. arterial hypertension

d. hyposthenuria

e. leucocyturia

272. What does intraparenchymal pulmonary restriction represent?

\*a. loss of lung compliance and elasticity

b. thoracic cage compliance reducing

c. pleural compliance decrease

d. reducing of thoracic cage skeleton compliance

e. obturation of upper airways

273. What exogenous factor causes stomach hypersecretion?

A\*.ethanol

B\*.gastrin hypersecretion

C. vasoactive intestinal polypeptide

D. secretin

E. pancreatic hyposecretion

274. What exogenous factor causes stomach hypersecretion?

A\*.gastrin hypersecretion

B. pepsin hypersecretion

C\*.vagotonia

D. pancreatic hypersecretion

E. pancreatic hyposecretion

275. What factor leads to reduced sodium reabsorbtion in proximal renal tubes?

A\*.pathologies at the level of renal tubes

B. disturbance of glucose reabsorbtion

C. disturbance of aminoacids reabsorbtion

D. reduces aldosterone level

E. hyperaldosteronism

276. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

A\*.reduced ADH secretion

B. inflammation of the renal glomeruli

C. insufficiency of natriuretic peptide

D. hypersecretion of aldosterone

E. hypersecretion of rennin

277. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

A\* degenerescence of tubular epithelium

B. inflammation of the renal glomeruli

C. insufficiency of natriuretic peptide

D. hypersecretion of aldosterone

E. hypersecretion of rennin

278. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

A\*.Areactivity of tubular epithelium to vasopressin

B. inflammation of the renal glomeruli

C. insufficiency of natriuretic peptide

D. hypersecretion of aldosterone

E. hypersecretion of rennin

279. What exogenous factor causes stomach hypersecretion?

\*a. gastrin hypersecretion

b. pepsin hypersecretion

\*c. vagotonia

d. pancreatic hypersecretion

e. pancreatic hyposecretion

280. What factor leads to reduced sodium reabsorbtion in proximal renal tubes?

\*a. pathologies at the level of renal tubes

b. disturbance of glucose reabsorbtion

c. disturbance of aminoacids reabsorbtion

d. reduces aldosterone level

e. hyperaldosteronism

281. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

\*a. increased concentration of osmotic active substances in primary urine

b. inflammation of the renal glomeruli

c. insufficiency of natriuretic peptide

d. hypersecretion of aldosterone

e. hypersecretion of rennin

282. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

\*a. reduced ADH secretion

b. inflammation of the renal glomeruli

c. insufficiency of natriuretic peptide

d. hypersecretion of aldosterone

e. hypersecretion of rennin

283. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

\*a. degenerescence of tubular epithelium

b. inflammation of the renal glomeruli

c. insufficiency of natriuretic peptide

d. hypersecretion of aldosterone

e. hypersecretion of rennin

284. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

\*a. areactivity of tubular epithelium to vasopressin

b. inflammation of the renal glomeruli

c. insufficiency of natriuretic peptide

d. hypersecretion of aldosterone

e. hypersecretion of rennin

285. What factor leads to reduced water reabsorbtion in distal and collector renal tubes?

\*a. increased concentration of osmotic active substances in primary urine

b. inflammation of the renal glomeruli

c. insufficiency of natriuretic peptide

d. hypersecretion of aldosterone

e. hypersecretion of rennin

286. What factors can provoke high airways obstruction?

a. pulmonary artery thrombosis

\*b. bronch mucus edema

\*c. foreign bodies in airways

\*d. larynx edema

e. mediastinum tumors

287. What factors can provoke inferior airways obstruction?

\*a. inflammation with mucus hypersecretion

\*b. inflammation with bronchiole mucus edema

c. larynx edema

d. swelling of the nasal passages

e. spasm of smooth musculature of the principal bronchi

288. What factors cause stomach hypersecretion?

\*a. ethanol

\*b. gastrin hypersecretion

c. calcium salts

d. pepsin hypersecretion

e. cholecystokinin hypersecretion

289. What factors decrease alveolar capillary diffusion?

A\*.pneumosclerosis

B\*.interstitial pulmonary edema

C. atherosclerosis of pulmonary circuit arteries

D\*.pulmonary circuit stasis

E. arterial hyperemia

290. What factors decrease alveolar capillary diffusion?

A\*.pneumonia

B\*.pulmonary edema

C. atherosclerosis of pulmonary circuit arteries

D. right heart failure

E. arterial hyperemia

291. What factors decrease oxygen capacity of the blood?

A\*.fetus hemoglobin

B\*.bivalent iron oxidation of hemoglobin

C. blood iron concentration decreases

D. blood ferritin concentration decreases

E. blood transferrin concentration decrease

292. What factors decrease oxygen capacity of the blood?

A\*.blood hemoglobin below 100 g/L

B\*.carboxy-hemoglobin formation

C. carbhemoglobin formation

D. blood ferritin concentration decreases

E. blood transferrin concentration decreases

293. What factors decrease oxygen capacity of the blood?

A\*.fetus hemoglobin

B\*.bivalent iron oxidation of hemoglobin

C. blood iron concentration decreases

D. blood ferritin concentration decreases

E. blood transferrin concentration decreases

294. What factors lead to aminoaciduria?

A. congenital insufficiency of the hexokinase in the renal epithelium

B. congenital distal tubulopathy

C\*.congenital proximal tubulopathy

D\*.liver disorders followed by aminoacidemia

E. glomerulopathy

295. What factors lead to intestinal maldigestion?

A. epithelial injury at the level of intestinal brush border

B\*.inflammatory processes in the small intestine

C\*.excessive bacterial colonization of small intestine

D. intestinal hyperperfusion

E. atrophy of small intestine mucosal layer

296. What factors lead to reduced glucose reabsorbtion in the kidneys?

A\*.congenital insufficiency of the hexokinase in the renal epithelium

B. glomerulopathy

C. distal tubulopathy

D\*.proximal tubulopathy

E. diabetes mellitus

297. What factors lead to reduced water reabsorbtion in distal and collector renal tubes?

A\*.increased concentration of osmotic active substances in primary urine

B\*.reduced ADH secretion

C. inflammation of the renal glomeruli

D. insufficiency of natriuretic peptide

E. hypersecretion of aldosterone

298. What factors lead to reduced water reabsorbtion in distal and collector renal tubes?

A\*.degenerescence of tubular epithelium

B\*.reduced reactivity of tubular epithelium to vasopressin

C. inflammation of the renal glomeruli

D. insufficiency of natriuretic peptide

E. hypersecretion of aldosterone

299. What factors lead to reduced water reabsorbtion in proximal renal tubes?

A\*.increased concentration of osmotic active substances in primary urine

B. insufficiency of antidiuretic hormone

C\*.dystrophy of tubular epithelium

D. areactivity of the tubular epithelium to the vasopressin

E. inflammation of the renal glomeruli

300. What factors provoke acute respiratory distress in adults?

A\*.traumatic shock

B. systemic arterial hypertension

C. pulmonary arterial hypertension

D. portal hypertension

E. bronchioles spasm

301. What factors provoke acute respiratory distress in adults?

A\*.bilateral inflammation of lungs

B\*.hemotransfusion shock

C. systemic arterial hypertension

D. pulmonary arterial hypertension

E. bronchiole spasm

302. What factors provoke acute respiratory distress in adults?

A\*.hypovolemic shock

B\*.disseminated intravascular coagulation

C. systemic arterial hypertension

D. pulmonary arterial hypertension

E. portal hypertension

303. What factors provoke pulmonary edema?

A\*.hypoproteinemia

B. pulmonary hypoperfusion

C. right-left vascular shunt

D. bronchial asthma

E. larynx stenosis

304. What factors provoke pulmonary edema?

A\*.left ventricle insufficiency

B. pulmonary hypoperfusion

C. right-left vascular shunt  
D. bronchial asthma

E. larynx stenosis

305. What factors provoke pulmonary edema?

A\*.hypoproteinemia

B. pulmonary hypoperfusion

C. right-left vascular shunt

D. bronchial asthma

E. larynx stenosis

306. What factors provoke pulmonary edema?

A\*.pulmonary circuit capillary stasis

B\*.pulmonary circuit capillary hyperpermeability

C. pulmonary hypoperfusion

D. bronchial asthma

E. larynx stenosis

307. What factors provoke pulmonary edema?

A\*.left ventricle insufficiency

B. pulmonary hypoperfusion

C. right-left vascular shunt

D. bronchial asthma

E. larynx stenosis

308. What factors provoke pulmonary edema?

A\*.lung lymphatic stasis

B\*.protein accumulation in the pulmonary interstitium

C. pulmonary hypoperfusion

D. bronchial asthma

E. larynx stenosis

309. What factors stimulate rennin production?

A\*.hypovolemia

B\*.increased sympathetic tonus

C. hyperaldosteronism

D. hypokalemia

E. hypernatremia

310. What factors stimulate rennin production?

\*a. renal hypoperfusion

\*b. hyponatremia

c. hyperaldosteronism

d. hypokalemia

e. hypernatremia

311. What factors stimulate rennin production?

\*a. hypovolemia

\*b. increased sympathetic tonus

c. hyperaldosteronism

d. hypokalemia

e. hypernatremia

312. What factors stimulate rennin production?

\*a. renal hypoperfusion

b. hypercalcemia

c. hyperaldosteronism

d. hypokalemia

e. hypernatremia

313. What factors stimulate rennin production?

\*a. hyponatremia

b. hypocalcemia

c. hyperaldosteronism

d. hypokalemia

e. hypernatremia

314. What factors stimulate rennin production?

\*a. hypovolemia

b. hypercalcemia

c. hyperaldosteronism

d. hypokalemia

e. hypernatremia

315. What heart compartments undergo hyperfunction in hypertensive disease?

a. right ventricle

b. left atrium and ventricle

c. right atrium

\*d. left ventricle

e. ight atrium and ventricle

316. What hemoglobin compounds decrease oxygen capacity of the blood?

a. association of oxygen with hemoglobin

b. reduced hemoglobin

c. association of dioxide carbon with hemoglobin

\*d. association of monoxide carbon with hemoglobin

\*e. hemoglobin with trivalent iron

317. What is a cause of coronary failure in type I diabetes mellitus?

A. energy deficiency in the myocardium caused by reduced up-take of glucose

B. reduced uptake of fatty acids by myocardium in lack of insulin

C. increased blood level of HDL and LDL with atherosclerosis of coronary artheries

D\*.increased blood level of VLDL and LDL with atherosclerosis of coronary artheries

E. increased lypolysis with high level of FFA in the blood

318. What is a cause of decreased bactericide activity of phagocytes reduction in patients with type I diabetes mellitus?

A. glucagon inhibits the production of free radicals in the cytoplasm of phagocytes

B. glucocorticosteroids which are increased in insulin deficiency induce apoptosis of neutrophils

C. glucagon which level are increased in insulin deficiency inhibits activity of lysosomal enzymes of phagocytes

D. lack of insulin inactivates lysosomal enzymes of phagocytes

E\*.insulin deficiency reduces microbicidal activity of phagocytic cells reducing availability of reactive oxygen species

319. What is a cause of hypersthenuria in diabetes mellitus type I?

A. increased urine density caused by presence of glucose in the urine when the renal threshold exceed 15 mmol/L

B. increased urine density caused by presence of glucose in the urine caused by increased filtration of glucose through renal filter

C. increased urine density caused by presence of glucose in the urine when the renal threshold exceed 7 mmol/L

D\*.increased urine density caused by presence of glucose in the urine caused by insufficient reabsorbtion of glucose in proximal convoluted tubules

E. increased urine density caused by presence of glucose in the urine caused by insufficient reabsorbtion of glucose in distal convoluted tubules

320. What is a cause of increased appetite in diabetes mellitus type I?

A. hyperglycemia

B. hyperlipidemia

C. glucagon

D. catecholamines

E\*.leptin

321. What is a cause of muscle atrophy in type I diabetes mellitus?

A. reduced glycogen storages caused by reduced glycogenogenesis in lack of insulin

B. reduced triglyceride storages caused by diminished lipogenesis in lack of insulin

C\*.reduced protein synthesis which is a direct consequence of insulin deficiency  
D. reduced protein synthesis which is a direct consequence of glucagon hypersecretion in insulin deficiency

E. reduced protein synthesis which is a direct consequence of cortisol hypersecretion in insulin deficiency

322. What is a cause of muscle atrophy in type I diabetes mellitus?

A. reduced glycogen storages caused by reduced glycogenogenesis in lack of insulin

B. reduced triglycerade storages by reduced lipogenesis in lack of insulin

C. excessive myolysis which is a direct consequence of glucagon hypersecretion in insulin deficiency

D. excessive myolysis which is a direct consequence of insulin deficiency

E\*.excessive myolysis which is a direct consequence of cortisol hypersecretion in insulin deficiency

323. What is a cause of predisposition to pyogenic infections in patients with type I diabetes mellitus?

A. humoral immunodeficiency type I

B. cellular immunodeficiency

C\*.reduced microbicidal activity of phagocytic cells caused by reduced ability to perform devitalization of bacteria

D. insufficient phagocyte activity of neutrophils caused by deficiency in free radicals

E. reduced activity of phagocytic cells caused by reduced expresion of opsonin receptors such reducing engulfing abilities

324. What is a cause of tertiary hypothyroidism?

a. pituitary disorders

b. inflammation of thyroid gland

\*c. hypothalamic disorders

d. iodine deficiency in the diet

e. thyroid removal

325. What is a cause of visual disturbances in I type diabetes mellitus?

a. microangiopathy at the level of lens

\*b. microangiopathy at the level of retina of the eyes

c. atherosclerosis of retinal artery caused by excessive blood level of VLDL and HDL

d. reduced up-take of glucose by retinal cells with energy deficiency

e. macroangiopathy at the level of retina of the eyes

326. What is a mechanism of albuminuria in patients with type I diabetes mellitus?

a. lack of insulin directly inhibits reabsorption of albumins from primary urine

\*b. microangiopathy in the renal glomeruli with increased albumin filtration through renal filter

c. excessive glucocorticoids increases glomerular filtration of albumins

d. increased basement membrane permeability of renal tubules with increased secretion of albumines in primary urine

e. disintegration of epithelial cells at the level of proximal tubes with reduced reabsorbtion of albumins

327. What is achlorhydria?

a. lack of Cl ions in the blood

\*b. absence of HCl in gastric juice

c. lack of enzymes in gastric juice

\*d. increase in stomach pH

e. decrease in stomach pH

328. What is cardiovascular system response to pain?

a. tachycardia and arterial hypo tension

b. bradycardia and arterial hypo tension

\*c. tachycardia and arterial hypertension

d. bradycardia and arterial hypertension

e. tachycardia and decreased resistance of peripheral circulation

329. What is characteristic for dyspnea?

\*a. breathing frequency change

\*b. breathing amplitude change

\*c. breathing rhythm change

d. blood gaseous change

\*e. subjective sensation of air lack

330. What is characteristic of pathological pain?

a. threshold of excitation is increased and the intensity of pain sensation decreases

\*b. excitation threshold is reduced and the intensity of pain sensation increases

c. threshold excitation is normal and intensity of pain sensation is appropriate

d. excitation threshold is increased and the intensity of pain sensation increases

e. excitation threshold is reduced and the intensity of pain sensation decreases

331. What is characteristic of pathological pain?

A. it occurs at a specific excitation of nociceptors

B. it occurs at a specific excitation of nociceptive system structures

C\*.it occurs at localization of pathological processes in structures of nociceptive system

D. it occurs at destruction of spinal posterior horns

E. it occurs at destruction of cortical post central gyrus

332. What is characteristic of physiological pain?

A. apparition of nocigen stimulus action on exteroreceptors

B\* Apparition of nocigen stimulus action on nocireceptors

C. apparition of nocigen stimulus action on proprioreceptors

D. it occurs spontaneously in the absence of nocigen stimulus

E. it occurs to physiological stimulus action on nociceptors

333. What is characteristic of physiological pain?

A. threshold of excitation is increased and decreases the sensation of pain intensity

B. excitation threshold is reduced and the intensity of the sensation of pain increases

C\*.threshold excitation is normal and intensity of the sensation of pain is appropriate to stimulus

D. excitation threshold is increased and the intensity of the sensation of pain increases

E. excitation threshold is reduced and decreases the intensity of pain sensation

334. What is characteristic of the pulmonary emphysema?

A\*.vital lung capacity decrease

B. inspiratory dyspnea

C. residual volume diminution

D. asphyxia

E. pulmonary hyperventilation

335. What is characteristic of the pulmonary emphysema?

A\*.expiratory dyspnea

B. inspiratory dyspnea

C. residual volume diminution

D. asphyxia

E. pulmonary hyperventilation

336. What is characteristically for dyspnea?

A\*.breathing frequency change

B\*.breathing amplitude change

C\*.breathing rhythm change

D. blood gaseous change

E\*.subjective sensation of air lack

337. What is endocrine system response to pain?

a. hyper secretion of adrenal medulla, adrenal cortex, thyroid gland and insulin

\*b. hypersecretion of adrenal medulla, adrenal cortex, and glucagon

c. hyper secretion of adrenal medulla, adrenal cortex, thyroid gland and insulin hypo secretion

d. hyper secretion of adrenal medulla, adrenal cortex , sex hormones and insulin

e. hyper secretion of adrenal medulla, adrenal cortex, reduced secretion of sex hormones

338. What is expiratory dyspnea?

a. inspiration prolongation

\*b. expiration prolongation

c. inspiration effort increasing with a passive expiration

\*d. forced expiration

e. concomitant prolongation of both inspiration and expiration

339. What is hypersalivation?

\*a. 2.5 L / 24 hours

b. 1L / 24 hours

c. 1.5L / 24 hours

d. 0.5L / 24 hours

e. 0.1 L / 24 hours

340. What is inspiratory dyspnea?

\*a. inspiration prolongation

b. expiration prolongation

\*c. inspiration effort increasing with a passive expiration

d. inspiration effort increasing with a forced expiration

e. concomitant prolongation of both inspiration and expiration

341. What is mechanism of hypothyroidism at methyluracil administration?

A. Intensification of iodine absorption from the blood

B. Intensification of iodine uptake by the thyrocytes

C\*.Inhibition of iodine uptake by the thyrocytes

D. Destruction of the thyroglobulin in thyrocytes

E. Inhibition of thyroid hormones exocytose

342. What is mechanism of oliguria in mouse pituitary extract administration?

A\*.water reabsorption increases in distal renal tubes

B. water reabsorption increases in proximal renal tubes

C. water reabsorption increases in whole canalicular system

D. plasma oncotic pressure increases

E. effective filtration pressure increases

343. What is one of immediate cardiac compensatory reaction in heart failure?

A. hydroelectrolytic retention

B. enhanced erythrocytopoiesis

C. myocardial hypertrophy

D\*.enhanced erythrocytopoiesis

E. bradycardia

344. What is one of late cardiac compensatory reaction in heart failure?

A. hydroelectrolytic retention

B. enhanced erythrocytopoiesis

C\*.myocardial hypertrophy

D. tachycardia

E. bradycardia

345. What is one of the consequences of portal hypertension?

A. development of cava – caval anastomosis

B\*.accumulation of transudate into the abdominal cavity

C. varicose dilation of leg veins

D. bleeding from hemorrhoidal veins

E. ischemia of the abdominal organs

346. What is one of the consequences of portal hypertension?

A. development of cava – cava anastomosis

B. ischemia of the abdominal organs

C\*.bleeding from inferior esophageal veins

D. bleeding from hemorrhoidal veins

E. varicose dilation of leg veins

347. What is one of the consequences of portal hypertension?

A. development of cava – caval anastomosis

B. ischemia of the abdominal organs

C. varicose dilation of leg veins

D. bleeding from hemorrhoidal veins

E\*.esophageal venous hyperemia

348. What is one of the consequences of portal hypertension?

A\*.development of porto – cava shunts

B. ischemia of the abdominal organs

C. varicose dilation of leg veins

D. bleeding from hemorrhoidal veins

E. development of cava – caval anastomosis

349. What is pancytopenia?

a. reduce the total number of red blood cells in peripheral blood

b. decrease the number of granulocytes in peripheral blood

c. decrease the number of platelets in peripheral blood

\*d. erythrocytopenia, granulocytopenia and thrombocytopenia

e. growth of all peripheral blood figurative elements

350. What is pathogenesis of hyperglycemia in diabetes mellitus type I?

a. inhibition of proteosynthesis from glucose in abcence of insulin

b. intense mobilization of glucose from glycogen of striated muscles in insulin deficiency

c. gluconeogenesis from fatty acids caused by high level of glucocorticosteroids

\*d. excessive glycogenolysis in hepatocytes induced by high level of glucagon

e. inability of the kidneys to remove excess glucose in the absence of insulin

351. What is pathogenesis of hyperglycemia in diabetes mellitus type I?

\*a. gluconeogenesis from amino acids caused by glucocorticosteroids

\*b. intense mobilization of glucose from glycogen of striated muscles

c. gluconeogenesis from fatty acids caused by glucocorticosteroids

d. inhibition of proteosynthesis from glucose without insulin

e. inability of the kidneys to remove excess glucose in the absence of insulin

352. What is pathogenesis of hyperglycemia in diabetes mellitus type I?

\*a. inhibition of glycogenogenesis in the lack of insulin

b. intense mobilization of glucose from glycogen of striated muscles

c. gluconeogenesis from fatty acids caused by glucocorticosteroids

d. inhibition of proteosynthesis from glucose without insulin

e. excessive gluconeogenesis from FFA caused by insulin deficiency

353. What is pathogenesis of primary hyperthyroidism?

A\*.autoantibodies against TSH receptors on thyrocytes

B. producing pituitary tumor of thyrotropin

C. overproduction of thyroid-releasing hormone

D. excessive consumption of iodine

E. autoantibodies against thyroglobulin

354. What is pathogenesis of tertiary hyperthyroidism?

A. producing thyroid tumor of thyroid hormones

B. producing pituitary tumor of thyrotropin

C\*.overproduction of thyroid-releasing hormone

D. excessive consumption of iodine

E. dietary iodine deficiency

355. What is pulmonary obstruction?

A\*.narrowing of airways by a foreign body

B. airways ectasia

C\*.increased resistance of airways

D. airways spasm

E. decreased resistance of airways

356. What is pulmonary obstruction?

A\*.narrowing of airways by a foreign body

B. airways ectasia

C\*.increased resistance of airways

D. airways spasm

E. decreased resistance of airways

357. What is somatic manifestation GH hypersecretion in adults?

A. gigantism, acromegaly, splanhnomegaly

B. gigantism, splanhnomegaly

C\*.acromegaly, splanhnomegaly

D. gigantism, acromegaly

E. acromegaly, atrophy of internal organs

358. What product is formed from bacterial fermentation of carbohydrates in the digestive tract?

a. alcohol

\*b. lactic acid

c. bytiric acid

\*d. carbon oxyde

e. pyruvic acid

359. What is the biological importance of glucocorticoids in the antenatal ontogenesis of the lungs?

A. stimulates development of bronchial tree

B. stimulates proliferation of alveolars type I and synthesis of surfactant

C\*.stimulates proliferation of alveolars type II and synthesis of surfactant

D. stimulates proliferation of alveolars macrophages

E. stimulates local immunity

360. What is the biological importance of glucocorticoids in the antenatal ontogenesis of the thyroid gland?

A. stimulates synthesis of thyroid hormone

B. stimulates iodine uptake by thyroid of fetus

C. stimulates the uptake of maternal thyroid hormone

D\*.contributes to the synthesis of enzymes of thyroid hormonogenesis

E. stimulates proliferation of fetal thyroid gland

361. What is the biological importance of glucocorticoids in the antenatal ontogenesis of gastrointestinal tract?

A. stimulates the proliferation of intestinal mucous

B\*.stimulates digestive enzymes

C. stimulates local immunity

D. stimulates development of autonomic nervous system

E. stimulates the formation of intestinal barrier

362. What is the biological importance of glucocorticoids in the antenatal ontogenesis of eyes?

A\*.stimulates enzymes that synthesize rhodopsin

B. stimulates network nerve development of retina

C. stimulates the development of optical media

D. stimulates the formation of blood-eye barrier

E. stimulates development of eye neuromuscular ends

363. What is the cause of GH hypersecretion?

A. basophilic adenoma of pituitary

B\*.acidophilic adenoma of pituitary

C. chromophobe adenoma of pituitary

D. adenoma of posterior lobe of pituitary

E. adenoma of intermediate lobe of pituitary

364. What is the cause of primary hyperaldosteronism?

A\*.tumor of glomerulosa layer of adrenal cortex

B. tumor of fasciculata layer of adrenal cortex

C. tumor of reticularis layer of adrenal cortex

D. tumor of anterior pituitary

E. tumor of hypothalamus

365. What is the cause of relative hypoxia in hypertrophied myocardium?

A\*.myocardial hyperfunction

B. formation into the vessels of atheromatous plaques

C. disturbance of oxygen using

D. disturbance of energogenesis

E. relative insufficiency of myocardial vasculature

366. What is the cause of secondary hyperaldosteronism?

A. renin hypersecretion in hypertonic hyperhydration

B\*.renin hypersecretion in renl hypoperfusion

C. hypersecretion of aldosteron from glomerular layer in adrenal cortex

D. hypersecretion of aldosteron from fascicular layer in adrenal cortex

E. renin hypersecretion in hypotonic hyperhydration

367. What processes lead to proximal canalicular acidosis?

a. disorders in H+ secretion

\*b. disorders in NAHCO3 reabsorbtion

c. disorders in ammonia reabsorbtion

\*d. administration of diuretics from carboanhydrase inhibitors

\*e. Fanconi syndrome

368. What processes is disturbed in chronic erythroblast leucosis?

a. hemoglobin synthesis

b. erythrocyte maturation

c. uncontrolled differentiation of erythroblast series

\*d. uncontrolled proliferation of erythroblasts series

e. reduced proliferation of erythroblast series

369. What is the digestive consequence of pancreatic secretion?

\*a. maldigestion of protein

b. maldigestion of dipeptides

c. maldigestion of fatty acids

d. maldigestion of disaccharides

e. malabsorption of monosaccharides

370. What is the digestive consequence of pancreatic secretion?

\*a. creatoree

\*b. steatoree

c. maldigestion of fatty acids

d. maldigestion of disaccharides

e. malabsorption of monosaccharides

371. What is the digestive consequence of pancreatic secretion?

\*a. polysaccharide maldigestion

b. maldigestion of dipeptides

c. maldigestion of fatty acids

d. maldigestion of disaccharides

e. malabsorption of monosaccharides

372. What processes determine the extraparenchymal pulmonary restriction?

\*a. pleura damage

\*b. thoracic cage alteration

c. pulmonary circuit disorders

\*d. neuro-muscular apparatus disorders

e. lung compliance change

373. What processes determine the extraparenchymal pulmonary restriction?

\*a. pleura damage

\*b. thoracic cage alteration

c.. pulmonary circuit disorders

d. neuro-muscular apparatus disorders

e. lung compliance change

374. What processes are disturbed in large intestine disorders?

a. digestion of polysaccharides

b. digestion of proteins

c. digestion of lipids

\*d. water reabsorbtion

\*e. synthesis of vitamins from group B

375. What processes are disturbed in B12 – deficiency anemia?

\*a. cell proliferation

b. cell differentiation

c. hemoglobin synthesis

\*d. intensifies lysis of erythrocytes

e. erythrocytes exit from bone marrow out

376. What process of bilirubin metabolism changes in premicrosomial hepatic jaundice?

\*a. uptake of free bilirubin from the blood

b. conjugation of free bilirubin captured from the blood

c. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

d. evacuation of the bile via the biliary intrahepatic ducts

e. evacuation of the bile via the extrahepatic biliary ducts

377. What process of bilirubin metabolism is disturbed in intrahepatic mechanical jaundice?

a. uptake of free bilirubin from the blood

b. conjugation of free bilirubin captured from the blood

c. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

\*d. evacuation of the bile via the biliary intrahepatic ducts

e. evacuation of the bile via the extrahepatic biliary ducts

378. What processes are characteristic for experimental cholemia?

\*a. vital centers inhibition in CNS

b. brain excitation

\*c. neuron inhibition in spinal cord

d. spinal cord excitation

e. spontaneous depolarization velocity decline of sinus node

379. What is the effect of postsynaptic receptor inhibition?

a. hyper function of innervated structures

b. atrophy of innervated structures

c. hypersensitivity of innervated structures

d. desensitization of innervated structures

\*e. paralysis of innervated structures

380. What is the function of the antinociceptive system?

A. annihilates any sensation of pain

B. decreases any sensation of pain

C. decreases only physiological sensation of pain

D\*.reduces only pathological sensation of pain

E. reduces chronic pain

390. What is the main pathogenetic link of pulmonary emphysema?

a. antiprotease excess in the blood

\*b. proteases excess in the blood

\*c. alpha-1 antitrypsin excess in the blood

d. alpha-1 antitrypsin excess

\*e. protease excess in the pulmonary parenchyma

391. What is the main pathogenetic link of pulmonary emphysema?

\*a. alveoli elasticity diminution

b. alveoli compliance diminution

c. collagen decrease in the alveolar wall

\*d. elastic fibers diminution in the alveolar wall

\*e. collagen enhancement in the alveolar wall

392. What is the manifestation of glucocorticoid hyposecretion?

a. arterial hypotension caused by hypernatremia with hyperhydartion

b. arterial hypotension caused by isotonic dehydration

c. arterial hypotension caused by excessive vasodilatation

\*d. arterial hypotension caused by hypotonic dehydration

e. arterial hypotension caused by hypertonic dehydration

393. What is the manifestation of hyperaldosteronism?

\*a. hyperhydration; hyperosmolarity; cell dehydration

b. hyperhydration; hyperosmolarity; cell overhydration

c. hyperhydration; hypoosmolarity; cell overhydartion

d. hypohydration; hyperosmolarity; cell dehydration

e. hypohydration; hypoosmolarity; cell dehydration

394. What is the manifestation of hyperaldosteronism?

a. intravascular dehydration

b. interstitial dehydration

\*c. interstitial hyperhydration

d. hyperkalemia

e. hypocalcemia

395. What is the manifestation of hyperaldosteronism?

a. urine hyperosmolarity

\*b. urine hypoosmolarity

c. poliuria

d. blood hypoosmolarity

e. hypokalaemia

396. What is the mechanism of Leydig cell atrophy in a prolonged administration of high doses of androgens?

a. exogenous androgens directly inhibit multiplication of Leydig cells

\*b. exogenous androgens inhibit secretion of LH

c. exogenous androgens inhibit secretion of FSH

d. exogenous androgens inhibit secretion of prolactin

e. exogenous androgenele desensitisate receptors for endogenous androgens

397. What is the mechanism of pain in anoxia?

a. synthesis of nocigenic kinins

\*b. accumulation of anaerobic metabolites

c. direct excitation of nocigenic receptor due to the lack of oxygen

d. threshold of nociceptors excitation decreases

e. nociceptors sensitization

398. What is the mechanism of Sertoli cells atrophy on a prolonged administration of high doses of androgens?

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b. exogenous androgens inhibit secretion of LH

\*c. exogenous androgens inhibit secretion of FSH

d. exogenous androgens inhibit secretion of somatotropin

e. exogenous androgenele desensitisate receptors for endogenous androgens

399. What is the mechanism of testosterone hyposecretion in a prolonged administration of high doses of androgens?

a. exogenous androgens directly inhibit secretion of Leydig cells

\*b. exogenous androgens inhibit secretion of LH which further inhibit secretion of Leydig cells

c. exogenous androgens inhibit secretion of FSH which further inhibit secretion of Leydig cells

d. exogenous androgens inhibit secretion of somatotropin which further inhibit secretion of Leydig cells

e. exogenous androgenele desensitisate receptors for endogenous androgens

400. What process of bilirubin metabolism changes in postmicrosomial hepatic jaundice?

a. uptake of free bilirubin from the blood

b. conjugation of free bilirubin captured from the blood

\*c. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

d. evacuation of the bile via the biliary intrahepatic ducts

e. evacuation of the bile via the extrahepatic biliary ducts

401. What is the normal sequence of stages in acute renal failure?

A\*.onset, phase of oligo-anuria, phase of polyuria, recovery

B. onset, phase of polyuria, phase of oligo-anuria, recovery

C. onset, phase of polyuria, recovery

D. onset, phase of oligo-anuria, recovery

E. onset, compensatory phase, phase of polyuria, recovery

402. What is the organotropic effect of glucocorticoids?

A. myocardial atrophy

B. fat atrophy

C. atrophy of nervous tissue

D. skin atrophy

E\*.atrophy of connective tissue

403. What is the organotropic effect of glucocorticoids?

A. myocardial atrophy

B. fat atrophy caused by enhanced lipolysis

C. atrophy of thymus caused by enhanced lypolysis

D. skin atrophy

E\*.atrophy of lymphoid tissue caused by enhanced apoptosis

404. What is the organotropic effect of glucocorticoids?

A. myocardial atrophy

B. fat storage by promoting lipogenesis

C. atrophy of skeletal muscle caused by enhanced lipolysis

D\*.atrophy of skeletal muscles caused by protheolysis

E. atrophy of skeletal muscle caused by enhanced glycogenolysis

405. What is the pathogenetic mechanism of glomerular hematuria?

A\*.passage of erythrocyte via the increased permeability of glomerular filter

B. passage of erythrocytes via the contort proximal tubes

C. passage of erythrocytes via the contort distal tubes

D. trauma of the urinary pathway by the renal stones

E. inflammation of the renal pelvis

406. What is the possible cause of tertiary hypercortisolism?

A. hypersecretion of ACTH by hypothalamus

B. hypersecretion of CRH by adenohypophysis

C. hypersecretion of CRH by neurohypophysis

D\*.hypersecretion of CRH by hypothalamus

E. hypersecretion of CRH by adrenal cortex

407. What is the pulmonary emphysema?

A. low airways narrowing

B\*.inflatable pulmonary alveoli

C. excessive dilation of terminal bronchioles

D. pulmonary parenchyma collapse

E. excessive persistent dilation of bronchi type II

What is the sign of prolactin hypersecretion in men?

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\*d. hypotrophic genitalia

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a. number of leucocytes in the blood above 9x109/L

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\*d. increased percents of one type of leucocytes due to reduction in other form of leucocytes

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411. What is the somatic manifestation of GH hyposecretion in adults?

a. dwarfism, hypotrophy of internal organs, osteoporosis, tissue atrophy

b. gigantism, hypotrophy of internal organs, osteoporosis , tissue atrophy

\*c. hypotrophy of internal organs, osteoporosis, tissue atrophy

d. acromegaly, hypotrophy of internal organs, osteoporosis , tissue atrophy

e. dwarfism, splanhnomegaly, osteoposis, tissue atrophy

412. What is the somatic manifestation of GH hyposecretion in children?

\*a. dwarfism, hypotrophy of internal organs, osteoporosis , connective tissue atrophy

b. gigantism, hypotrophy of internal organs, osteoporosis, connective tissue atrophy

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426. What process leads to distal canalicular acidosis?

\*a. disorders in H+ secretion

b. disorders in NaHCO3 reabsorbtion

c. disorders in ammonia reabsorbtion

d. administration of diuretics from carboanhydrase inhibitors

e. reduced glomerular filtration rate

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c. excessive dilation of terminal bronchioles

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e. excessive persistent dilation of bronchi type II

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\*b. inflatable pulmonary alveoli

c. excessive dilation of terminal bronchioles

d. pulmonary parenchyma collapse

e. excessive persistent constriction of bronchi type II

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b. increased number of young leucocytes in the blood

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\*d. increased percents of one type of leucocytes due to reduction in other form of leucocytes

e. increased percent of young form of leucocytes due to reduction in mature form of leucocytes

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\*c. hypotrophy of internal organs, osteoporosis, tissue atrophy

d. acromegaly, hypotrophy of internal organs, osteoporosis , tissue atrophy

e. dwarfism, splanhnomegaly, osteoposis, tissue atrophy

444. What is the somatic manifestation of GH hyposecretion in children?

\*a. dwarfism, hypotrophy of internal organs, osteoporosis , connective tissue atrophy

b. gigantism, hypotrophy of internal organs, osteoporosis, connective tissue atrophy

c. hypotrophy of internal organs, osteoporosis , connective tissue atrophy

d. acromegaly, hypotrophy of internal organs, osteoporosis , connective tissue atrophy

e. dwarfism, splanhnomegaly, osteoporosis, connective tissue atrophy

445. What process is disturbed in B12 deficiency anemia?

\*a. cell multiplication

b. cell differentiation

c. hemoglobin synthesis

d. release of erythrocytes from bone marrow

\*e. cell maturation

446. What mechanism reduces the resistance of the rat with experimental hyperthyroidism to hypoxia?

a. decreased rate of oxido- reduction reactions

\*b. increased O2 consumption

c. decreased rate of basal metabolic

d. increased membrane resting potential of the neuron

e. decreased O2 consumption

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c. Decreased rate of basal metabolic

d. Increased membrane resting potential of the neuron

e. Decreased O2 consumption

448. What mechanisms reduce the resistance of the rat with experimental hyperthyroidism to hypoxia?

\*a. intensification of basal metabolic rate

\*b. increased O2 consumption

c. decreased basal metabolic rate

\*d. exhaustion of neuronal metabolic substrate

e. decreased O2 consumption

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a. decreased rate of oxido- reduction reactions

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d. Increased membrane resting potential of the neuron

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\*b. increased O2 consumption

c. decreased basal metabolic rate

\*d. exhaustion of neuronal metabolic substrate

e. decreased O2 consumption

452. What mechanisms reduce the resistance of the rat with experimental hyperthyroidism to hypoxia?

a. Decreased oxido- reduction reactions

\*b. Increased O2 consumption

c. Decreased rate of basal metabolic

\*d. Exhaustion of neuronal metabolic substrate

e. Decreased O2 consumption

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\*a. Increased rate of basal metabolic

b. Decrease of oxido- reduction reactions

c. Decreased rate of basal metabolic

\*d. Exhaustion of neuronal metabolic substrate

e. Decreased O2 consumption

454. What pathological phenomena involves nephritic syndrome?

\*a. periorbital edema

\*b. oliguria

c. hypolipidemia

d. lipiduria

e. hyperlipidemia

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A\*.cell multiplication

B. cell differentiation

C. hemoglobin synthesis

D. release of erythrocytes from bone marrow

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A\*.disorders in H+ secretion

B. disorders in NaHCO3 reabsorbtion

C. disorders in ammonia reabsorbtion

D. administration of diuretics from carboanhydrase inhibitors

E. reduced glomerular filtration rate

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C. disorders in ammonia reabsorbtion

D. administration of diuretics from carboanhydrase inhibitors

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A. uptake of free bilirubin from the blood

B\*.conjugation of free bilirubin captured from the blood

C. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

D. evacuation of the bile via the biliary intrahepatic ducts

E. evacuation of the bile via the extrahepatic biliary ducts

459. What process of bilirubin metabolism changes in postmicrosomial hepatic jaundice?

A. uptake of free bilirubin from the blood

B. conjugation of free bilirubin captured from the blood

C\*.excretion of conjugated bilirubin from hepatocyte in biliary canalicules

D. evacuation of the bile via the biliary intrahepatic ducts

E. evacuation of the bile via the extrahepatic biliary ducts

460. What process of bilirubin metabolism changes in premicrosomial hepatic jaundice?

A\*.uptake of free bilirubin from the blood

B. conjugation of free bilirubin captured from the blood

C. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

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461. What process of bilirubin metabolism is disturbed in intrahepatic mechanical jaundice?

A. uptake of free bilirubin from the blood

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C. excretion of conjugated bilirubin from hepatocyte in biliary canalicules

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A\*.vital centers inhibition in CNS

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C\*.neuron inhibition in spinal cord

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E. spontaneous depolarization velocity decline of sinus node

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D\*.water reabsorbtion

E\*.synthesis of vitamins from group B

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482. What represent hyperpnea?

\*a. increased respiratory frequency

\*b. increased amplitude of breathing

c. shallow breathing

d. reduced frequency of breathing

e. deep and accelerated breathing

483. What represents bradypnea?

a. frequent breathing

b. profound breathing

c. superficial breathing

\*d. rare breathing

e. profound and accelerated breathing

484. What represents polipnea?

\*a. frequent breathing

b. profound breathing

c. shallow breathing

d. rare breathing

e. profound and accelerated breathing?

485. When accelerated and deep respiration occurs?

\*a. hypoxia

b. bronchial asthma

\*c. metabolic acidosis

d. pulmonary emphysema

e. pulmonary edema

486. When expiratory dyspnea occurs?

a. pneumonia

b. pulmonary hypertension

\*c. terminal bronchioles spasm

d. high airways stenosis

e. pulmonary atelectasis

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b. pulmonary hypertension

\*c. terminal bronchioles spasm

d. high airways stenosis

e. pulmonary atelectasis

488. When frequent shallow breathing- occurs?

a. hypoxia

b. bronchial asthma

c. metabolic acidosis

\*d. pulmonary emphysema

\*e. pulmonary edema

489. When shallow and deep respiration occurs?

a. physical effort

\*b. larynx edema

c. pneumonia

d. pulmonary edema

e. hypoxia of any origin

490. Which bile component determines the frog cardiac activity change in cholemia?

a. cholesterol

\*b. bile acids

c. phospholipids

d. glycoproteins

e. bile pigments