ADRENAL DISEASE

1. In the cortex of the adrenal hormones are produced by all except:
1. cortisol
2. progesterone
3. adrenaline
4. aldosterone

2. With the absence of or a significant reduction in any of the adrenal cortex hormone for the emergence of skin pigmentation?
1. Cortisol
2. testosterone
3. adrenaline
4. aldosterone

3. Development of achlorhydria and anorexia due to the lack or decline:
1. cortisol
2. testosterone
3. adrenaline
4. aldosterone

4. Secondary adrenal insufficiency caused by:
1. autoimmune processes in the adrenal
2. the destruction of the adrenal tuberculous process
3. iatrogenic effects (bilateral adrenalectomy, prolonged steroid therapy)
4. pituitary pathology

5.Reason for primary adrenal insufficiency is all, except:
1. autoimmune processes in the adrenal
2. the destruction of the adrenal tuberculous process
3. iatrogenic effects (bilateral adrenalectomy, prolonged steroid therapy)
4. pituitary pathology

6. How healthy adrenal glands respond to the test with ACTH?
1. to increase urinary excretion of 17-KS and 17-OCS and increased serum cortisol levels
2. the absence of a response in the form of increased urinary cortisol excretion and increase serum cortisol levels

7. What ACTH test results are typical for primary adrenal insufficiency?
1. increase urinary excretion of 17-KS and 17-OCS and increased serum cortisol levels
2. the absence of a response in the form of increased urinary cortisol excretion and increase serum cortisol levels

8. Which symptom is determined in patients with primary adrenal insufficiency and absent in patients with secondary adrenal insufficiency:
1. weight loss
2. hyperpigmentation of the skin and mucous
3. reduction of blood pressure
4. marked weakness

9. What imaging studies adrenal is the most informative?
1. X-ray
2. computed tomography
3. radioisotope scanning

10. Typical complaints of patients with chronic adrenal insufficiency includes everything except:
1. general weakness
2. loss of appetite
3. weight loss
4. increased appetite
5. the craving for salty
6. abdominal pain

11. In acute adrenal insufficiency, patients should be entered in the first place:
1. sodium chloride solution
2. B vitamins
3. hydrocortisone
4. norepinephrine
5. ascorbic acid

12. Anabolic agents are derived:
1. glucocorticosteroids
2. estrogen
3. mineralocorticosteroid
4. androgens
5. progestins

13. The cause of diabetes disease Cushing is:
1. primary degradation of b-cells of the pancreas
2. disturbance of insulin sensitivity
3. obesity
4. increased gluconeogenesis
5. inactivation of insulin

14. Adaptation to external influences on adrenal tumor depends on adequate compensation:
1. cortisol
2. ACTH
3. adrenaline
4. Prolactin
5. aldosterone

15. With long-term administration of prednisone, hyperglycemia begins to develop as a result of:
1. destruction of b-cells of the pancreas
2. enhance gluconeogenesis
3. inhibition of insulin secretion
4. decrease in glucose utilization tissues

16. Hypothalamic-pituitary-adrenal system responds in a "feedback":
1. on aldosterone
2. for cortisol
3. to ACTH
4. by dehydroepiandrosterone
5. to progesterone

17. What is the most characteristic symptom is pain in the stomach in patients with acute adrenal insufficiency?
1. flatulence
2. vomiting
3. diarrhea

4. tachycardia
5. drop in blood pressure

18. Primary osteoporosis in patients with Cushing's disease is mainly due to:
1. Disturbance of the protein matrix of bone
2. with dysfunction of the parathyroid glands
3. in violation of the secretion of mineralocorticoids
4. with an increase in urinary calcium excretion
5. malabsorption of calcium in the gastrointestinal tract

19. In Addison's disease is affected:
1. Beam layer of the adrenal cortex
2. glomerular layer of the adrenal cortex
3. plexiform layer of the adrenal cortex
4. all the layers of the adrenal cortex
5. all the layers of the adrenal cortex and medulla

20. Patients within 3 months of receiving dexamethasone about systemic lupus erythematosus in a dose of 2.5 mg / day. What is the adrenal glands produce cortisol?
1. increased
2. reduced
3. not changed
4. violation can only be detected during the test with sinaktenom
5. reduced the half-life

21. Bilateral adrenal hyperplasia is caused by:
1. increased secretion of ACTH
2. increased secretion of CRH
3. reduced secretion of ACTH
4. increased secretion of TSH
5. increased secretion of somatostatin

22. The biological action of glucocorticoids:
1. increased potassium reabsorption in the distal tubules of the kidney
2. anti-inflammatory effect
3. catabolic
4. increase the utilization of glucose by peripheral tissues
5. activation of hepatic gluconeogenesis

23. For acute adrenal insufficiency is characterized by:
1. fever
2. abdominal pain
3. nausea
4. arterial hypertension

24. For Addison crisis is characterized by:
1. anacatharsis
2. drop in blood pressure
3. prostration
4. acetonuria

25. Skin pigmentation in Addison's disease requires a differential diagnosis with the following conditions:
1. toxic goiter
2. hemachromatosis

3. pellagra
4. scleroderma

26. Cushing's syndrome is caused by excessive secretion of:
1.ACTH
2. cortisol
3. cateholamino
4. androgen
5. aldosterone

27. Cushing's disease is caused by excessive secretion of:
1. ACTH
2.cortizol
3.cateholamine
4.androgen
5.aldosteron

28. Part of the central mechanism in the development of Cushing's disease is to:
1. disturbance in the rate of secretion of ACTH and cortisol;
2. increased prolactin;
3. decrease GH;
4. lower TSH;
5. reduced gonadotropins.

29. The clinical symptoms of Cushing's can be anything other than:
1. obesity
2.arterial hypertension
3.bluish striae
4. hypoglycaemia
5.disturbance in menstrual cycle
6.virilization
7.hypotrophy muscles

30. For Cushing's disease is characterized by:
1. A thin, dry skin
2. excessive deposition of fat in the neck, trunk, abdomen, face in the form of a "full moon"
3. the presence of "menopausal hump"
4. reduction in breast volume
5. humidity of the skin

31. Hypertension in Cushing's disease is caused by:
1. central mechanisms of regulation of vascular tone;
2. enhance the function of the adrenal cortex;
3. water retention and sodium;
4. decreased renin release;
5. renal artery stenosis.
6. by renal tubular

32. In the clinical analysis of blood disease Cushing's notes:
1. lymphocytopenia;
2. decreased hemoglobin and lymphocytosis;
3. eosinopenia, granulocytopenia;
4. polycythemia;
5. eosinophilia, and granulocytopenia.

33. For Cushing's disease is most characteristic:
1. hyperkalemia
2. hyponatremia;
3. hypercalcemia;
4. hypokalemia;
5. increase in alkaline phosphatase activity.

34. The most common bone changes pituitary Cushing are:
1. deformation and fracture;
2. osteoporosis;
3. growth retardation in children;
4. differential acceleration and growth of the skeleton;
5. hyperostosis.

35. In severe disease Cushing there is:
1. uniform distribution of subcutaneous fat;
2. pathological fractures;
3.transitional hypertension;
4. skeletal growth;
5. preserve mestrualny cycle.

36. Carbohydrate metabolism in Cushing's syndrome due to:
1. insulin resistance
2. hyperinsulinemia
3. hyperglycagonnemia
4. glycogenolysis
5. gluconeogenesis

37. A negative result in a large sample with dexamethasone can be deleted:
1. Cushing's disease;
2. adenomatoz adrenal cortex;
3. glucosterone
4. ectopic AKTT syndrome;
5. corticosterone

38. Remission of Cushing's disease after radiation therapy comes in:
1. 1-2 months;
2. 3-4 months;
3. 5-6 months;
4. 7-8 months;
5. more than 1 year.

39. Therapeutic effect hloditana (mitotane)is:
1. destruction of the adrenal cortex;
2. suppressing CRH secretion;
3. suppression of ACTH secretion;
4. In the inhibition of the enzyme 11-b-hydroxylase;
5. In the suppression of the enzyme 17-a-hydroxylase.

40. What are the clinical symptoms of Cushing, not characteristic of the early stages of Cushing's syndrome:
1. displastic obesity
2. bluish striae
3.virilization

4. pathological fractures due to osteoporosis, diffuse
5. arterial hypertension

41. Differential diagnosis of Cushing's syndrome and Cushing disease:
1. short dexamethasone
2. long dexamethasone
3. uroven 17-GCS in the daily urine
4. uroven cortisol in daily urine
5. ACTH level in the serum
6. sutochny rhythm of cortisol secretion

42. When exogenous (iatrogenic) Cushing syndrome cortisol levels in the blood are:
1. increase
2. decrease
3.do not changed

43. What tumor can cause of ectopic ACTH-dependent Cushing's syndrome:
1. tumor of liver
2. oat cell cancer
3. tumor of testicles
4. medullary thyroid cancer
5. follicular thyroid cancer
6. tumor of parathyroid gland

44. What are the clinical features, not characteristic of the ectopic ACTH-dependent Cushing's syndrome variants:
1. slow progression
2. increase in liver
3. ascitis
4. psychological disturbances
5. circulation insufficiency

45. What clinical symptoms hypercorticotism not characteristic of hypothalamic syndrome of puberty period
1. hyperglycemia
2. stria of the skin
3. arterial hypertension
4. osteoporosis
5. muscle hypotrophy

46. What method of therapy is preferred in the case of Cushing central origin?
1. medical therapy aimed at inhibition of the secretion of corticotropin and CRH
2. surgical correction adrenal hyperplasia
3. radiotherapy therapy
4. selective adenomectomy transphenoidal methold
5. medical therapy aimed at inhibition of the biosynthesis

47. For ectopic ACTH syndrome is characterized by:
1. positive large dexamethasone
2. increase in the excretion of 17-ACS (test with metiraponom)
3. central obesity
4. very high levels of ACTH
5. hyperpotassiumia

48. Typical manifestations of increased production of glucocorticoids are:

1 weight loss
2. stretch marks on the skin
3. arterial hypotension
4. increase moisture of the skin
5. decrease glucose

49. Nelson's syndrome is manifested:
1. low levels of ACTH in the blood;
2. increase in moisture of the skin;
3. tuberculose adrenal
4. high cortisol levels in the blood;
5. chronic adrenal insufficiency.

50. What are the most common cause of chronic adrenal insufficiency:
1. destruction of adenalectomy
2. autoimmune destruction of the adrenal cortex
3. tumor pituitary
4. tumor adrenal
5. exogenous glucocorticoids

51. Pigmentation of the skin in patients with Addison's disease is particularly pronounced in
1. Exposed areas of the body (face, hands, fold the back of the hands and feet, etc.);
2. Areas subject to friction (axilla and groin, knees, etc.);
3. Areas of postoperative scarring;
4. places of natural pigmentation (nipple milk and mammary glands, sex organs);
5. mucous membranes (lips, gums, tongue, etc.).

52. Manifestations of Addison's disease are
1. hyperpigmentation of the skin;
2. abdominal pain (Addison gastrointestinal crises);
3. hypotension;
4. hypertension;
5. cardialgia

53. With moderate to severe adrenal insufficiency mean
1. replacement therapy and glucose mineralcorticoid
2. ascorbic acid and anabolic steroids;
3. nicotinic acid;
4. spironolactone, veroshpiron;
5. surgery.

54. Chronic adrenal insufficiency are all of the following except:
1. weakness
2. loss of weight
3. arterial hypotension
4. hyperglycemia
5. hyperpigmentation skin

55. What are the specific symptoms of Chronic adrenal insufficiency
1. muscular weakness
2. the need for salty foods
3. arterial hypotension
4. tendency to hypoglycemic conditions
5. pigmentation of the skin and mucous

56. For deficiency of glucocorticoids is not typical clinical symptom following:
1. hypotension
2. tendency to hypoglycemic conditions
3. muscle weakness
4. loss of weight
5. dispeptic disorders

57. For mineralocorticoid deficiency characterized by the following clinical signs:
1. hypotension
2. tendency to hypoglycemic conditions
3. muscular weakness
4. loss of weight
5.the need for salty foods

58. What are the main laboratory diagnostic criteria for Chronic adrenal insufficiency
1. high levels of potassium in the blood serum
2. low levels of serum cortisol
3. a low level of sodium in the blood serum
4. normal corticotropin levels in serum
5. low or high levels of corticotropin in serum

59. What are the main laboratory parameters, to distinguish primary from secondary Chronic adrenal insufficiency
1. level serum cortisol
2. level cortisol in daily urine
3. ACTH level in the serum
4. level serum aldosterone
5. level of 11 deoxycoritizola serum

60. Name the most physiological rhythm of receiving glucocorticoid for substitution therapy:
1. early morning and evening
2. early morning and afternoon
3. full dose in the morning after breakfast
4. all dose in 24 hours
5. afternoon and evening

61. What are the main criteria for selection of the dose Glucocorticoid in Chronic adrenal insufficiency
1. weight gain
2. increase in blood pressure
3. the disappearance of the skin hyperpigmentation
4. level glycemia
5. level of serum cortisol

62. What are the main criteria for clinical compensation Chronic adrenal insufficiency
1. stabilization of body weight
2. normal blood pressure
3. normalization of appetite
4. recovery of muscle strength
5. elimination of pigmentation of the skin and mucous

63. What are the main criteria for selection of the dose mineralocorticoid when Chronic adrenal insufficiency
1. blood pressure

2. the level of sodium in the blood serum
3. serum potassium level
4. level of serum aldosterone
5. edema

64. What you do in the case of concomitant disease or stress at Chronic adrenal insufficiency
1. increase dose GC and MC
2. increase the dose of GC
3. increase dose of MK
4. increase dose of GC and reduce the dose of MC
5. decrease dose and increase the dose of GC MC

65. In secondary adrenal insufficiency have the following clinical signs, except for:
1. hypotension
2. hypooglicemiya
3. tendency salt intake
4. loss of weight
5. hyperpigmentation of skin
6. decrease axillary and pubic hair distribution

66. Name drugs are not recommended for infants and young children to correct glucocorticoid insuffiency
1. hydrocortizone
2. prednisolone
3. cortizone
4. dexamethasone

67. Factors causing the development of acute adrenal insufficiency, can not be:
1. severe infection in a patient with Chronic adrenal insufficiency
2. stress patient with Chronic adrenal insufficiency
3. stress in a healthy person
4. trauma
5. surgical intervention
6. double sided adrenal hemorrhage in humans, which has no Chronic adrenal insufficiency

68. For the clinical picture of the acute adrenal insufficiency characterized by the following features:
1. hypothermia
2. nausea, vomiting
3. a drop in blood pressure
4. edema
5. spasm

70. What are regulators of aldosterone secretion:
1. ACTH
2. potassium
3. cortizol
4. vazopressin
5. renin-angiotensin system

71. What are the most common cause of primary hyperaldosteronism
1. double sided diffuse adrenal hyperplasia
2. congenital adrenal hyperplasia
3. Conn's syndrome (aldosteronoma)
4. tumor of adrenal

72. What are the clinical symptoms are not specific to primary hyperaldosteronism
1. arterial hypertension
2. poliuriya, polydipsia
3. edema
4. symtoms of hypokalemia
5. disturbance of glucose tolerance

73. Classical biochemical criteria for primary hyperaldosteronism of the following except:
1. hypokalemia
2. decrease plasma renin activity (PRA)
3. increase aldosterone
4. hyponatremia
5. increase levels of potassium in the urine

74. What is the most common cause of hypoaldosteronism:
1. congenital adrenal hyperplasia
2. pseudohypoaldosteronizm
3. primary adrenal insufficiency
4. one sided adrenalectomy
5. diabetic nephropathy

75. Pheochromocytoma - is a tumor originating from:
1. glomerular zone of the adrenal cortex
2. facicular zone of the adrenal cortex
3. reticular zone of the adrenal cortex
4. adrenal medulla

76. Pheochromocytoma can secrete hormones listed below, except:
1. aldosterone
2. adrenalin
3. noradrenalin
4. dopamin
5. serotonin
6. neuropeptide Y

77. Name the clinical symptom that is not characteristic of adrenal localization of pheochromocytoma:
1. bradycardia
2. tremor
3. increase systolic blood pressure
4. increase in pupils
5.emortional excitement