ENDOCRINE SYSTEM

1. How can endocrine disorders be classified?
    1. Hereditary, acquired
    2. Active, passive
    3. Uncompensated, overcompensated
    4. Primary, secondary
    5.1, 4
    6.1, 2, 3, 4.

2. Which is the MAIN pathogenic unit in endocrine disorders?
    1. Changed level of a certain hormone
    2. Disbalance between hormones and receptors
    3. Uncontrolled hormone function
    4. Increased or decreased “life” of a certain hormone
    5. Changes in the hormonal sensitivity

3. Which endocrine disorder is defined as secondary/tertiary?
    1. Pathological process in a certain endocrine gland
    2. It is a result of an increased releasing factors level
    3. It is a result of an impaired tropic hormones regulation
    4. It is a result of genetic defect in the gland
    5. 2. 3
    6. 1, 2, 3, 4

4. The increased metabolism in hyperthyroidism is a result of:
    1. High iodine plasma levels
    2. Stimulation of the oxygen consumption
    3. Suppressed oxygen consumption
    4. Impaired extramitochondrial oxygen use
    5. Decreased oxygen diffusion through the cellular membrane

5. Hyperthermia in hyperthyroidism is a result of:
    1. Activate the Krebs cycle
    2. Activate the Pentose phosphate pathway
    3. Decoupling of oxidative phosphorylation
    4. Increased mitochondrial redox-potential
    5. Primary mitochondrial hyperplasia.

6. Which of the following factors is NOT associated with thyrotoxicosis development?
    1. Genetic predisposition
    2. Obesity
    3. Psychic trauma
    4. Hormonal disbalance
    5. Increased levels of TSH

7. The pathogenesis of Graves-Basedow disease is associated with:
    1. Autoimmune stimulation of the gland.
    2. Toxic-dystrophic stimulation of the gland
    3. Increased affinity of the gland to iodine ions
    4. Primary hypersensitivity of the gland to catecholamine hormones
    5. 1, 3

8. What is the TSH level in primary hyperthyroidism?
    1. Constantly increased
    2. Decreased
    3. Unchanged
    4. With circadian fluctuations
    5. It does not concern the thyroid gland function

9. Tachycardia in hyperthyroidism is a result of:
    1. Blocked parasympathetic activation
    2. Increased cardiac afterload
    3. Decreased cardiac preload
    4. Increased sympathetic activation
    5. Malignant hyperthermia

10. Hypofunction of the thyroid gland could be a result of:
    1. Decreased TSH levels
    2. Insufficient iodine consumption
    3. Increased levels of thyroid-stimulating antibodies
    4. Long-term anti-thyroid function treatment
    5. 1, 2, 3
    6. 1, 2, 4

11. Hypothyroidism in children and adults leads to different pathologies. Which are they?
    1. Cretinism/Myxedema
    2. Gigantism/Acromegaly
    3. Dwarfism/Hypopituitarism
    4. Turner’s syndrome/Adipose-genetalia dystrophy
    5. Diabetes insipidus/Addison's disease

12. Which is NOT part of myxedema symptoms?
    1. Decreased metabolism
    2. Decreased psychic activity
    3. Tachycardia
    4. Inclination to hypothermia
    5. Obesity

13. In primary hypothyroidism:
    1. TSH is increased
    2. TSH is decreased
    3. Thyreotoxin is increased
    4. Iodine accumulation in the gland is increased
    5. Thyreoglobulin levels are increased

14. Which is NOT a cause for primary hypothyroidism?
    1. Congenital gland hypoplasia
    2. Thyroid stimulating growth factors
    3. Congenital defects in hormone synthesis
    4. TSH receptors resistance
    5. Long-term X-ray radiation

15. The pathogenesis of chronic hypocorticism is associated with decreased levels of:
    1. GCS
    2. MCS
    3. Suprarenal sex hormones
    4. Catecholamines
    5. 1, 2
    6. 1, 2, 3, 4

16. Inclination to hypoglycemia in Addison disease is a result of:
    1. Increased insulin secretion
    2. Renal diabetes
    3. Decreased gluconeogenesis and glycogenosynthesis
    4. Pathological glycogen synthesis in the liver
    5. Increased glycogen synthesis in the muscles

17. Arterial hypotension in Addison disease is associated with:
    1. Decreased catecholamine levels
    2. Impaired sensitivity of the arterial baroreceptors
    3. Decreased levels of GCS and hypovolemia
    4. Genetic predisposition
    5. Decreased central stimulation

18. Skin hyperpigmentation in chronic hypocorticism is a result of:
    1. Increased sun sensitivity
    2. Increased levels of ACTH and MSH
    3. Increased vitamin D synthesis in the skin
    4. Hereditary increased melanocytes
    5. Secondary siderosis

19. Which are the water-electrolyte disorders in hypocorticism:
    1. Hypernatremia, hyperkalemia, normovolemia
    2. Hyponatremia, hyperkalemia, hypervolemia

3. There are no changes in the electrolytes
4. Hypertonic hydration, cellular edema
5. Hyponatremia, hyperkalemia, dehydration.

20. Which of the following is typical for secondary chronic hypocorticism?
1. Arterial hypertension
2. No skin pigmentation
3. Diabetes mellitus type II
4. Fat tissue redistribution
5. Malignant hypothermia

21. Primary hypercorticism could be a result of:
1. Pathological process in the hypothalamus
2. Eosinophilic adenoma of the adenohypophysis
3. Cortex hyperplasia of the adrenal gland
4. Long-term Cortison treatment
5. Hyperplasia of the adrenal medulla

22. Secondary hypeglucocorticism could be a result of:
1. Increased levels of ACTH
2. Autoimmune lesions in zona glomerulosa
3. Basophilic adenoma of the adenohypophisis
4. Benign tumor of the adrenal cortex
5. 1, 3
6. 1, 2, 4

23. The pathogenesis of hypercorticism is **mainly** associated with increased levels of:
1. GCS
2. MCS
3. Catecholamines
4. Dopamine
5. Suprarenal sex hormones

24. Protein metabolism disturbances in hypercorticism are associated with:
1. Delayed transport of aminoacids in the cells
2. Augmented catabolic processes
3. Change in the primary polypeptides structure
4. Locally increased anabolism in the limbs
5. Redistribution of the muscle tissue

25. In primary hyperaldosteronism:
1. Aldosterone does not affect renin secretion
2. There is increased level of aldosterone and increased level of renin
3. There is decreased level of aldosterone and decreased level of renin
4. There is increased level of aldosterone and decreased level of renin
5. Aldosterone augments the circadian rhythm of renin secretion

26. What are the disturbances in water-electrolyte balance in primary hyperaldosteronism?
    1. Hypernatremia, hyperkalemia, hypovolemia
    2. Hyponatremia, hypokalemia, hypovolemia
    3. Hypernatremia, hypokalemia, hypervolemia
    4. Hypernatremia, hyperkalemia, hypervolemia
    5. Hypercalcemia, hypokalemia, hypervolemia

27. Which of the following is a common cause for adrenal medulla hyperfunction?
    1. Increased stimulation of the adenohypophysis
    2. Pheochromocytoma
    3. Increased activity of RAAS
    4. Electrolyte stimuli – hyponatremia, hyperkalemia
    5. 1, 3

28. Pheochromocytoma leads to:
    1. Elevated catecholamines
    2. Elevated aldosterone
    3. Increased level of tropic hormones
    4. Decreased level of catecholamines
    5. Overactivated parasympathetic nervous system

29. The biological effects of STH could be diminished in a deficiency of:
    1. Somatostatins
    2. Somatoliberins
    3. Somatomedins
    4. Prostaglandins
    5. Leukotriens

30. The growing effect of STH is associated with:
    1. Increased activity of peptide hydrolases
    2. Positive nitric and phosphorous balance
    3. Decreased catabolic processes
    4. Stimulated lipogenesis
    5. Increased appetite

31. Which factor plays a major role in the pathogenesis of pituitary dwarfism?
    1. Decreased production of TSH
    2. Decreased production of ACTH
    3. Decreased production of FSH
    4. Decreased secretion of ADH
    5. Decreased secretion of STH

32. Panhypopituitarism is a result of damaged:
    1. Adenohypophysis

2. Supraoptic nucleus of the hypothalamus
3. Neurohypophysis
4. Epiphysis
5. 3, 4

33. Which are the causes of panhypopituitarism?
1. Pituitary gland damage during pregnancy
2. Tumor or inflammation that affects the pituitary gland
3. Craniocerebral trauma, massive hemorrhage
4. 1, 3
5. 1, 2, 3

34. The pathogenesis of panhypopituitarism is associated with decreased levels of:
1. GTH, TSH, ACTH
2. ADH, oxytocin
3. MSH, GCS, catecholamines
4. Somatomedins, melatonin
5. Thymosins, substance P

35. What is the cause of primary diabetes insipidus?
1. Damage in the hypothalamus and hypophysis
2. Hereditary defect of the ADH receptors in the renal tubules
3. Acquired defect of the ADH receptors in the renal tubules
4. Hyperplasia of the epiphysis
5. Atrophy in substantia nigra