Acid- Base Balance

1. Metabolic acidosisis a process of:
    1. Accumulation and/or excess ofnon-respiratoryH^+.
    2. Deficiency of non-respiratory H^+.
    3. DecrasedCO_2 partial pressure.
    4. Increased CO_2 partial pressure.
    5. Increased bicarbonate concentration.

2. Main pathogenetic mechanism for metabolic acidosis development is:
    1. Excess production of H^+ in the body.
    2. Decreased H^+ excretion by kidneys.
    3. Increased bicarbonate production by kidneys.
    4. Activated Darrow– Kotlov mechanism
    5. 1, 2.
    6. 1, 3, 4.

3. Which process is characterized with accumulation of metabolic H^+?
    1. Respiratory center depression.
    2. Diminished endogenous production of H^+ donors.
    3. Exogenous intake of acid products.
    4. Reduced buffer capacity of extracellular space.
    5. Disturbed Starling equation.
    6. 3, 4

4. In which case the metabolic acidosis is NOT caused by increased production of H^+?
    1. Diabetes mellitus.
    2. Prolonged fasting.
    3. Renal failure.
    4. Acute intoxication.
    5. Shock.
    6. Severe hypoxia.

5. Hypoxic type lactic acidosis occurs when tissue pO2 falls:
    1. Under 70 mmHg.
    2. Under 60 mmHg.
    3. Under 40 mmHg.
    4. Under 20 mmHg.
    5. Under 10 mmHg.

6. pO2 is not essential for lactic acidosis

6. Not-hypoxic lactic acidosis is most commonly observed in:
    1. Physical exercise.
    2. Enzyme defects in lactate metabolism.
    3. Hyperoxia.
    4. Respiratory acidosis.
    5. 1, 3.

7. Main pathogenetic unit in hypoalkaline metabolic acidosis is:
    1. Increased bicarbonate loss from extracellular space.
    2. Increased intracellular bicarbonates.
    3. Protein buffers deficiency.
    4. Disturbance in hemoglobin buffer system.
    5. Bicarbonates transfer into the cells.

8. Retention type metabolic acidosisis a result of:
    1. Retention of acid products.
    2. Impaired H^+ secretory capacityof the kidney.
    3. Increased absorption of H^+ ions.
    4. Development of renal H^+ generator.
    5. 1, 2.
    6. 1, 3, 4.

9. Metabolic alkalosis is a process of:
    1. Accumulation and/or excess of non-respiratory H^+
    2. H^+ reduction and/or non-respiratory H^+ deficiency .
    3. Cells and tissues H^+ imbalance.
    4. Organic anions accumulation.
    5. Impaired hemoglobin buffering.

10. Main pathogenetic unit for metabolic alkalosis development is:
    1. Decreased bicarbonate concentration in extracellular space.
    2. Increased bicarbonate concentration in extracellular space.
    3. Bicarbonate transfer between cells and extracellular space.
    4. Decreased CO_2 partial pressure.
    5. Increased CO_2 partial pressure.

11. Main pathogenetic mechanism for metabolic alkalosis development is:
    1. Suppressed renal bicarbonate generation.
    2. Activated renal bicarbonate generation.

3. Acids loss with subsequent chlor deficiency.
4. 1, 2.
5. 2, 3.

12. Trigger for metabolic alkalosis is:
    1.Synthesis or intake of bicarbonate.
    2. Increased renal bicarbonate affinity.
    3. Increased plasma-erythrocyte HCO_3 translocation.
    4. 1, 2.
    5. 1, 2, 3.

13. How can metabolic alkalosis be stabilized?
    1. Hypocapnia development.
    2. Compensatory glutamin lysis.
    3. Urea synthesis stimulation.
    4. Increased renal bicarbonate retention.
    5. Increased bicarbonate synthesis from monocyte-macrophage system.

14. How can metabolic alkalosis be divided?
    1. Hypoxic and non-hypoxic.
    2. Congenital and acquired.
    3. Calcium dependent andcalcium independent.
    4. Chlor dependent and chlor independent.
    5. Normo-, hypo- and hyperosmotic.

15. Which is the clinical breathing manifestations in metabolic alkalosis?
    1. Loud and deep breathing – Kussmaul's respiration.
    2. Agonal breathing.
    3. Slow and/ or shallow breathing.
    4. Biot's respiration.
    5. Breathing is not changed.

16. What is the characteristic of respiratory acidosis?
    1. Decreased pCO_2.
    2. Increased pCO_2.

3. Increased pO_2.
4. Decreased pO_2.
5. Decreased HbCO.

17. Main pathogenetic mechanism for respiratory acidosis development is:
    1. Alveolar hyperventilation.
    2. Alveolar hypoventilation.
    3. Disturbed gases diffusion in lungs.
    4. Isolated increase of CO_2 production.
    5. 2, 4.
    6. 1, 3, 4.

18. Which of the followed does NOT lead to development of primary respiratory acidosis?:
    1. Supressed respiratory center.
    2. Mechanical asphyxia.
    3. Inadequate dosed oxygen therapy.
    4. Airways spasm.
    5. Psycho-emotional excitement

19.What is the compensation of respiratory acidosis?
    1. Tubular bicarbonate excretion.
    2. Activated tubular acid- and ammonia genesis.
    3. Activated ketone synthesis.
    4. Suppressed ornithine cycle.
    5. Activated pentosephosphate cycle.

20. What is the characteristic of primary respiratory alkalosis?
    1. Increased bicarbonate level.
    2. Decreased bicarbonate level.
    3. Increased CO_2 partial pressure.
    4. Decreased CO_2 partial pressure.
    5. Mandatory presence of HbCO.

21. Main pathogenetic mechanism for respiratory alkalosis development is:
    1. Collateral alveolar ventilation.
    2. Alveolar hyperventilation.
    3. Alveolar hypoventilation.
    4. Decreased ventilation/perfussion ratio.
    5. Increased alveolar CO2 export.

22. What is the compensation of primary respiratory alkalosis?
    1. Hypercapnia development.
    2. Hypocapnia development.
    3. Chlorid deficiency development.
    4. Increased plasma bicarbonates.
    5. Decreased plasma bicarbonates.

23. What is the renal compensation of primary respiratory alkalosis?
    1. Decreased tubular H^+ secretion.
    2. Increased tubular H^+ secretion.
    3. Increased amino acid clearance.
    4. Activated renin-angiotensin system.
    5. Increased aldosteron effects.

24. Decreased plasma bicarbonate level in respiratory alkalosisis a result of:
    1. Supressed acido- and amonnia genesis.
    2. Hamburger's effect.
    3. Increased cellular lactate productionandsecretion.
    4. 1, 2.
    5. 1, 2, 3.

25. Symptoms of "dizziness" and muscles tremor in respiratory alkalosis are associated with:
    1. Increased sodium level.
    2. Increased calcium level.
    3. Decreased sodium level.
    4. Decreased calcium level.
    5. Increased urea level.

26. The inadequacy in which processes leads to acid-base balance distublance?
    1. Production, transport and excretion of organic anions.
    2. Production, transport and excretion of lactate.
    3. Production, transport and excretion of electrolytes.
    4. Production, transport and excretion of CO_2.
    5. Production, transport and excretion of H^+.

27. The term "Hypoprotonemia"describes the presence of:
    1. Alkalosis.
    2. Bicarbonatemia.
    3. Alkalemia

4. Hyposmia.
5. 1, 2, 3.

28. Which of the following is NOT a classification of acid-base disorders?
1. Compensated, subcompensated and uncompensated.
2. Mild, moderate, severe, life-threatening.
3. Respiratory, metabolic, mixed.
4. Local and general.
5. Hereditary, congenital, acquired.

29. Neutralization of H^+ excess/ or deficiency / in the extracellular space is achieved by:
1. Extracellular buffering
2. Ion exchange in renal tubules.
3. Metabolic transformations.
4. Alterations of pulmonary ventilation.
5. 1, 2, 3.
6. 1, 2, 3, 4.

30. When an acid-base balanace disturbance can be definied as compensated?
1. pH is in the references, but the other indexes are out if it.
2. pH is out of the references, but the other indexes are normal.
3. All the indexes are in the references.
4. All the indexes are out of the references.
5. All mentioned, but monitored in a timely manner.