DISTURBANCES IN THE MICROCIRCULATION - HYPEREMIAS, EMBOLISM AND THROMBOSIS

1. Arterial hyperemia is:
    1. Increased incoming amount of blood to a certain organ with decreased outflow.
    2. Increased incoming amount of blood to a certain organ with normal outflow.
    3. Decreased incoming amount of blood with normal outflow.
    4. Decreased or blocked incoming of blood.
    5. Disturbed perfusion of a tissue.

2. Venous hyperemia is:
    1. Localized increase of blood amount due to impaired outflow.
    2. Slowing or blocking of the blood flow in capillaries, small arteries and veins.
    3. Increased incoming blood flow and more rapid outflow.
    4. Redistribution of the blood flow.
    5. Increased tissue perfusion due to larger amount of blood coming.

3. From functional point of view, the arterial hyperemia is a sign of:
    1. Filling of blood with storing it.
    2. Hyperperfusion of the area.
    3. Compensatory shunting of the blood flow.
    4. Edema formation.

5. Lymph production.

4. Arterial hyperemia could be a result of:

1. Increased function of an organ.

2. Inflammation, fever.

3. The action of thermal, chemical irritants, etc.

4. Thrombosis and embolism of the vessel.

5. 1, 2, 3.

6. 2, 3, 4.

5. The mechanisms taking part in arterial hyperemia are:

1. Neurogenous.

2. Humoral.

3. Cellular-inductive.

4. Immunogenous.

5. 1, 2.

6. 2, 3, 4.

6. Neurotonic arterial hyperemia is mainly the result of:

1. Increased tone of the vasodilatatory nerves.

2. Increased tone of the vasoconstrictive nerves.

3. Periodic change of the tone of n. vagus.

4. Decreased vasoconstrictive tone.

5. 1, 4

7. Which of the following factors take place in the mechanism of arterial hyperemia:

1. Bioactive substances /histamine, bradykinin, etc./

2. Metabolites /lactic acid, adenosine, CO2 etc./

3. Changes in pH (acidosis).

4. 1, 2.

5. 1, 2, 3.

8. In case of rapid evacuation of a liquid out of the abdominal or pleural cavity the result could be:

1. Reflex decompression hyperemia.

2. venous hyperemia.

3. Ischemia.

4. Working hyperemia.

5. None of the above.

9. Point out the clinical and functional signs of arterial hyperemia:

1 Redness and increase of temperature.

2. Cyanosis and decrease of temperature.

3. Edema.

4. Increased turgor.

5. 1, 4.

6. 1, 3, 4.

10. The changes in blood flow in a prominent constriction of the veins are:

    1. Increased incoming blood, normal outflow.
    2. Decreased incoming amount of blood, disturbed outflow.
    3. Normal incoming amount of blood, disturbed outflow.
    4. Decreased collateral circulation.
    5. Compensatory shunting of the blood flow.

11. Reasons for observing venous hyperemia could be:

    1. Blocked vessel.
    2. Compressed vessel.
    3. Constitutional weakness of the venous apparatus.
    4. 1, 2.
    5. 1, 2, 3.

12. From microscopic point of view arterial hyperemia is characterized by:

    1. Increased diameter of the vessels and more rapid blood flow.
    2. Increased diameter of the vessels and slowed down blood flow.
    3. Normal diameter of the vessels and slowed down blood flow.
    4. Reduced vascular network.
    5. 3, 4.

13. From microscopic point of view venous hyperemia is characterized by:

    1. Increased diameter of the vessels and more rapid blood flow.

2. Increased diameter of the vessels and slowed down blood flow, sludge phenomenon, diapedesis of RBC.

3. Reduced vascular diameter, slowed down blood flow.

4. Increased vascular network.

5. Reduced vascular network, “steal phenomenon”.

14. In what type of hyperemia hypoxia, disturbed metabolism, metabolic acidosis and dystrophy are present:

1. Arterial hyperemia.

2. Venous hyperemia.

3. Reactive hyperemia.

4. Working hyperemia.

5. 3, 4.

15. Define blood stasis:

1. Normal incoming of blood with disturbed outflow.

2. Increased incoming blood with normal outflow.

3. Decrease to full blocking of the blood flow to a certain area.

4. Blocking of the blood flow in capillaries, small arteries and veins.

5. Redistribution of the blood flow in a certain area.

16. Ischemia is a state of:

1. Decreased or completely missing arterial blood supply.

2. Normal amount of incoming blood with disturbed outflow.

3. Increased arterial blood flow with normal outflow.

4. Increased “pumping out” of the blood from an area.

5. Centralisation of the blood flow.

17. Depending on the causing factor, microcirculatory ischemia could be:

1. Due to compression.

2. Due to obturation.

3. Angiospastic.

4. Due to redistribution.

5. 1, 2, 3.

6. 1, 2, 3, 4.

18. The most common reason for infarction is:

1. A sudden, critical reduction of the blood flow.

2. Venous obstruction.

3. Capillary blood stasis.

4. Arterial hyperemia.

5. Collateral hypocirculation.

19. Thrombosis is a process of:

1. Formation of a blood clot in a vessel of a living organism.

2. Formation of a blood clot in the tissues of a living organism.

3. Formation of blood clots “post mortem” or “in vitro”.

4. Formation of posttraumatic hematoma.

5. Spontaneous hemoconcentration.

20. The main factors leading to blood clotting (the so called Virchow's triad) are:

1. Changes in the endothelium.
2. Changes in the diameter of the vessel.
3. Disturbance in the blood flow (speed, character).
4. Changes in the composition of the blood.
5. 1, 3, 4.
6. 1, 2, 3.

21. The triggering factor for a thrombus formation is usually:

1. Activation of the platelets.
2. Damage to the endothelium.
3. Changes in the velocity or characteristics of the blood flow.
4. Primarily activated fibrinolysis.
5. Obligatory activated leucocytes.

22. Which factors determine the antithrombogenic qualities of the endothelium:

1. Prostacyclin, adenosine, nitric oxide.
2. Heparin, protein C, AT-III.
3. Endocapillary glycocalyx.
4. 1, 2.
5. 1, 2, 3.

23. Aggregation of platelets is stimulated by:

1. Increased level of heparin.

2. Increased ratio **TxA**2 / PG-I2.

3. Equal deficiency of TxA2 and PG-I2.

4. Decreased ratio TxA2 / PG-I2.

5. Calcium deconjugation.

24. Embolism is a process of:

1. Blocking of a vessel by abnormal matter, travelling with the blood flow.

2. Disturbing the rheological characteristics of the blood.

3. Formation of a blood clot due to overactivation of the coagulation system in a living organism.

4. Local formation of a blood clot.

5. Premature activation of fibrinolysis.

25. Which of the following could be attributed to endogenous embolism:

1. Thromboembolism.

2. Air / fat / bacterial embolism.

3. Gas / parasitic embolism.

4. Gas / fat / hard foreign bodies embolism.

5. None of the above.

26. Thromboembolism could be observed in:

1. Anaerobic gas gangrene, caisson disease.

2. Rupture of subcutaneous fat tissue, fracture of long bones.

3. Entering of amniotic fluid into the uterine arteries.

4. Trauma to the lungs, artificial pneumothorax.

5. Detachment of parts of the thrombus or its aseptic / putrid decomposition.

27. Gas embolism could be observed in:

1. Injury of big vessels and air entering inside them.

2. Artificial pneumothorax.

3. Caisson disease, gas gangrene.

4. Thrombophlebitis.

5. Fracture of a long bone.

28. The most common reason for air embolism is:

1. Laceration wounds in the region of v. jugularis and the venae cavae.

2. Artificial pneumothorax.

3. A breach in the hermetization of an aircraft.

4. Caisson disease.

5. 3, 4.

6. 1, 2.

29. When a long bone fracture is present in an adult, there is a risk of:

1. Thromboembolism.

2. Bacterial embolism.

3. Gas embolism.

4. Air embolism.

5. Fat ebmolism.

30. Embolism in the small circulatory system emerges as a result of:

1. Left ventricle aunerysm.
2. Blocking of **a**. pulmonalis by a thromboembolism.
3. Blocking of art. carotis by a thromboembolism.
4. Acute left ventricle insufficiency.
5. Progressive mitral stenosis.