SPECIAL PATHOPHYSIOLOGY

CARDIO-VASCULAR SYSTEM

1. Myocardia lischemia is mainly a result of:
   1.Coronary hypoxemia.
   2. Coronary artery disease (CAD).
   3. Acute coronaritis.
   4. Coronary anemia.
   5. Heart remodelling.

2. The essence of myocardial ischemia is:
   1.Mismatch between import and export ofoxygen in the myocardium.
   2. Inadequacy between arterial and venous circulation of the heart
   3. Mismatch between coronary blood flow and myocardial demands.
   4. Inactivated myocardial oxygen.
   5. Circadian oxyge nimbalance in themyocardium.

3. Which of the following causes refer to the "risk factors" for CAD (coronary artery disease):
   1. Atherogenic dyslipoproteinemia, smoking.
   2. Arterial hypertension, diabetes.
   3. Fasting and increased physical activity.
   4. Obesity, decrease physical activity.
   5. 1, 2, 4.
   6. 2, 3, 4.

4. The main cause of CAD (coronary artery disease)is:
   1.Coronary atherosclerosis.
   2. Platelet aggregation in the coronary vessels.
   3. Impaired oxygen transport function of the blood.
   4. Coronary spasm induced by gastro-coronary reflex.
   5. Variations in oxygen demands of the myocardium.

5. For which forms of CAD coronary artery spasm is the major pathogenetic mechanism:
   1.Myocardial sclerosis.
   2. Sudden cardiac death.
   3. Prinzmetal's angina pectoris.
   4. Myocardial infarction.
   5. Stable angina pectoris.

6. What is angina?:
   1.Respiratory-related chest pain.
   2. Transient ischemic chest pain.
   3. Neuralgic chest pain.

4. Chest pain which depends on the position of the body.
5. Neuralgic pain in a fixed location.

7. The clinical manifestation of stable angina is provoked by:
   1.Physical effort.
   2. Mental stress.
   3. Arterial hypertension.
   4. Prolonged sleep.
   5. 1, 2, 3.
   6. 1, 2, 4.

8. The main pathogenetic mechanism of stable angina pectorisis:
   1.Increased oxygen requirements of the myocardium.
   2. Rapidly progressive coronary stenosis.
   3. Coronary thrombosis.
   4. Coronary dissection.
   5. Acute coronaritis.

9. The main pathogenetic mechanism of unstable angina pectoris is:
   1.Intramural myocardial coronary compression.
   2. Fixed coronary stenosis with primary arterial hypotension.
   3. Rapid onset and /or progressive narrowing of the coronary vessel.
   4. Hormonal oxidative phosphorylation explosion in myocardium.
   5. Exacerbated coronaritis.

10. Increased oxygen requirements of the myocardium, can be satisfied primarily by:
    1.Increasing oxygen capacity.
    2. Facilitated hemoglobin oxygen release.
    3. Internal reallocation of the coronary blood flow.
    4. Adequately increased coronary blood flow.
    5. Compensatory polycythemia.

11. Each coronary stenosis (at any time and/or degree) leads to decrease of:
    1.Myocardial oxygen demands.
    2. Deposited myocardium oxygen.
    3. Oxygen content in the coronary sinus.
    4. Collateral coronary blood flow.
    5. Coronary heart reserve.

12. What is acute myocardial infarction?:
    1.Myocardial dystrophy.
    2. Ischemic necrosis of the myocardium.
    3. Myocardial fibrosis.
    4. Acute inflammatory process.
    5. Spontaneous apoptosis of myocardiocytes.

13. What is the main pathogenetic unit in myocardial infarction formation?:
    1.Fixed coronary stenosis.
    2. Turbulence in the coronary blood flow.
    3. Acute ventricle-coronary reflux.
    4. Coronary thrombosis.
    5. Hyperemic coronary shunt.

14. Which pathogenetic mechanisms in the cell lead to irreversible ischemic necrosis?
    1.Lack of ATP.
    2. Lactic acidosis and cytosolic Ca^{2+} accumulation.
    3. Direct ischemic genomic paralysis.
    4. Rapid accumulation of free radicals.
    5. 1, 2, 3, 4.
    6. 1, 2, 4.

15. The most severe complication of acute myocardial infarction is:
    1.Pulmonary embolism.
    2. Cardiogenic shock.
    3. Acute aneurysm.
    4. Pericardial effusion.
    5. Ventrical thrombosis.

16. The most common complications of acute myocardial infarction are:
    1.Thromboembolism of cerebral vessels.
    2. Chronic left-sided heart failure.
    3. Heart aneurysm.
    4. Rhythm and conduction disorders.
    5. Acute pericarditis.

17. Which of the following are complications in chronic phase of myocardial infarction:
    1. Heart aneurysm.
    2. Chronic heart failure.
    3. Cardiogenic shock.
    4. Rhythm and conduction disorders.
    5. 1, 2, 4.
    6. 1, 3, 4.

18. Which clinical type of CAD is associated with apoptosis and loss of myocardial cells?:
    1.Acute myocardial infarction.
    2. Atherosclerotic myocardiosclerosis.
    3. Stable angina.

4. Ischemic sudden cardiac death.
5. Progressive unstable angina.

19. What is the main cause of atherosclerotic myocardiosclerosis?
    1.Acute myocardial ischemia.
    2. Postischemic immune response against the heart cells.
    3. Steady chronic myocardial hypoperfusion.
    4. Progressive inflammatory process in the myocardium.
    5. Continuous spasm of the coronary vessels.

20. Which is the most common mechanism of sudden cardiac death?
    1.Ventricular fibrilation.
    2. Ventricular rupture.
    3. Cardiac tamponade.
    3. Electromechanical ventricular dissociation.
    5. Sudden block in ventrical contractions.

21. The lack of pain during myocardial ischemia could be caused by:
    1. Decreased pain sensitivity.
    2. Ischemic generation of high-frequency pain impulses.
    3. Mild ischemia, that does not reach the pain threshold.
    4. Ischemic block of the pain impulses conduction.
    5. 1, 3, 4.
    6. 1, 2, 3, 4.

22. Main pathogenetic factor for the diastolic dysfunction in pericardial disorders is:
    1.Pericardial-myocardial pathological reflex.
    2. Presence of fluid in the pericardium.
    3. Increased intrapericardial pressure.
    4. Reduced pericardial-epicardial contact.
    5. Dysfunction between the left and right side of the heart.

23. Main pathogenetic mechanism in cardiac tamponade is:
    1.Reduced myocardial contractility.
    2. Impaired diastolic filling of the heart.
    3. Reflex tachycardia.
    4. Increased afterload.
    5. Increased blood accumulation in the heart chambers.

24. Pathophysiological manifestations of pericardial effusions are:
    1. Diastolic dysfunction and central venous stasis.
    2. Systolic dysfunction and central venous stasis.
    3. Diastolic dysfunction with empty central circulation.
    4. Diastolic dysfunction with arterial hypertension.
    5. Decompensated ventricular diastolic dysfunction.

25. Main pathogenetic mechanisms imparing contractility of cardiomyocytes are:
    1.Reduced capacity of ion-transport mechanisms.
    2. Reduced effectiveness of cardiac adrenergic regulation.
    3. Damage to cell membranes and cardiomyocyte enzymes.
    4. Genomic blocking of myocyte hypertrophy.
    5. 1, 2, 3.
    6. 1, 2, 3, 4.

26. What kind of changes are induced by the mechanism of Frank-Starling?
    1.Increasing the strength, speed and amplitude of contraction.
    2. Increasing the primary length of myofibres and thediastolic volume.
    3. Increasing the contact area actin/myosin (S/N) and the number of A-N bridges.
    4. Increasing the amount of Ca^{2+} in cardiomyocytes.
    5. 1, 2, 3.
    6. 1, 2, 3, 4.

27. Conditions leading to activation of the Frank-Starling mechanism are::
    1. Increased afterload.
    2. Increased preload.
    3. Sinus tachycardia.
    4. Physical exercise.
    5. Increased myocardium contractility.

28. Compensatory character of myocardial hypertrophy depends on:
    1.Increased number of sarcomeres.
    2. Increasing the amount and size of the sarcomeres.
    3. Adaptive reordering of the sarcomeres.
    4. Hyperplasia of the myocardiocytes, not associated with the sarcomeres.
    5. 1, 3.
    6. 1, 2, 4.

29. Pathological hypertrophy is characterized by:
    1.Excessive hypertrophy of organelles, cells and capillaries.
    2. Hyperplasia of cardiomyocytes.
    3. Cardiomyocyte polyploidy.
    4. Multiplication of cardiac connective tissue elements.
    5. Massive intramyocardial hemorrhage.

30. What is the main purpose of hypertrophy?
    1.To progressively increase hyperfunction.
    2. Attempt to reduce hyperfunction.
    3. To relatively stabilize the hyperfunction.

4. To compensate hyperfunction.
5. To provide better functional status of the heart.

31. Power and rate of contraction and relaxation are compensatory increased via massive Ca^{2+} release in :
    1.Increased length of cardiomyocytes.
    2. Simpathetic adrenal stimulation of the heart.
    3. Increased force of contraction with unchanged length of cardiomyocytes.
    4. Increased volume of cardiomyocytes.
    5. Vagal stimulation.

32. Prolonged tachycardia has negative effects on the heart due to:
    1.Shorten diastolic filling.
    2. Increased oxygen consumption of the myocardium.
    3. Impaired synchronization between the left and right ventricle.
    4. Dysfunctionof the valve apparatus.
    5. 1, 2.
    6. 1, 2, 3, 4.

33. Pathological consequences of long-standing myocardial hypertrophy are:
    1. Reduced ventricular compliance.
    2. Completely suppressed mechanism of Frank-Starling.
    3. Decreased myocardial contractility.
    4. Cardiac chronotropic hypersensitivity.
    5. 1, 3.
    6. 1, 2, 3, 4.

34. The most important non-cardiac compensations in heart failure are related to:
    1.Stimulation of the renin-angiotensin-aldosterone system.
    2. Increased secretion of ADH.
    3. Increasing the concentration of 2,3-DPG in erythrocytes.
    4. Increased oxygen affinity of hemoglobin.
    5. 1, 2, 3.
    6. 1, 2, 3, 4

35. Heart failure is a clinical manifestation of:
    1.Ventricular contractile dysfunction.
    2. Pumping heart failure.
    3. Electrical instabilityof the heart.
    4. Decentralization of circulation.
    5. Inappropriate centralization of blood flow.

36. Which are the main causes for development of cardiogenic pump deficiency?
    1.Impaired, reduced and limited ventricular filling.
    2. Excessively increased resistance in systole.

3. Critically lowered blood volume.
4. Functionally inefficient cardiac contractions.
5. 1, 2, 4.
6. 1, 2, 3, 4.

37. Which are the main causes for development of hemodynamic heart failure (HF)?
    1. Aortic and pulmonary stenosis.
    2. Pulmonary and systemic hypertension.
    3. Insufficient valvular defects.
    4. Myocardial ischemia, myocarditis, cardiomyopathy.
    5. 1, 2, 3.
    6. 1, 2, 3, 4.

38. Energetic type heart failure develops in:
    1. Coronary artery disease, myocarditis,myocardiodystrophy.
    2. Stenotic valvular defects.
    3. Systemic and pulmonary arterial hypertension.
    4. Congenital valvular defects without cyanosis.
    5. Prolonged exercise.

39. What is the main pathogenetic unit of cardiogenic pulmonary edema?:
    1.Decreased pulmonary lymphatic drainage.
    2. Increased hydrostatic pressure in pulmonary capillaries.
    3. Low counter pressure in pulmonary interstitium.
    4. Increased permeability of the alveoli-capillary barrier.
    5. Reduced plasma concentration.

40. Which is the most common cause of right-sided heart failure development?:
    1. Chronic obstructive pulmonary disease (COPD).
    2. Arterial hypertension.
    3. Congenital and acquired defects of the mitral valve.
    4. Rhythm and conduction disorders.
    5. 1, 3.

41. What is the main pathogenetic factor of "cor pulmonale" development:
    1.Arterial hypertension.
    2. Pulmonary hypotension.
    3. Pulmonary hypertension.
    4. Stagnant bloodflow in the pulmonary circulation due to weakness of the left ventricle.
    5. Arterial hypercapnia.

42. Which of the following could be a cause of rhythm and conduction heart disorders?
    1.Intoxication.
    2. Metabolic disorders.
    3. Emotional stress.

4. Electrolyte imbalance.
5. Excessive physical exercise.
6. All stated.

43. What are the basic mechanisms for arrhythmias development?
1.Increased automaticityof the heart.
2. Occurrence of re-entry mechanism.
3. Occurrence of ectopic excitatory pulse.
4. Ventricular hypo- and akinesia.
5. 1, 3, 4.
6. 1, 2, 3

44. Pathogenesis of reumocarditis is based on:
1. Streptococcal induced myocardial intoxication.
2. Crossed (streptococcus A / cardiomyocytes) immunological reactivity.
3. Autoimmune reaction against cardiac immunologically privileged antigens.
4. Generalized immune deficiency.
5. Genetic defects in myocardium.

45. Etiology is still unclear in:
1.Acute bacterial myocarditis.
2. Ischemic myocardial damage.
3. Cardiomyopathy.
4. Myocardiodystrophy.
5. Tezaurismozes of the myocardium.

46. Hemodynamic disorders in acute myocarditis are closest to those of:
1.Acute myocardial infarction.
2. Dilated cardiomyopathy.
3. Acute pericardial injury.
4. Chronic atherosclerotic cardiomyopathy.
5. Obstructive-restrictive cardiomyopathy.