STATE UNIVERSITY
«UZHHOROD NATIONAL UNIVERSITY»
MEDICAL FACULTY N 2
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PATHOPHYSIOLOGY OF EXTERNAL BREATHING AND CARDIOVASCULAR SYSTEM

METHODICAL INSTRUCTIONS

for practical classes and self-study on Pathophysiology
for 3rd year students
of medical faculty №2, specialty 222 “Medicine”

Uzhhorod 2023

PATHOPHYSIOLOGY OF EXTERNAL BREATHING AND CARDIOVASCULAR SYSTEM. Methodical instructions for practical classes and self-study on Pathophysiology for 3rd year students of medical faculty №2, specialty 222 “Medicine” / Sheiko N.I., Slyvka Y.I. Uzhhorod: 2023. 58 p.

Methodological instructions for practical classes on Pathophysiology for students of the Medical faculty № 2 from the section “Pathophysiology of external breathing and cardiovascular system" have been prepared in accordance with the requirements of the Syllabus on Pathophysiology for students of the medical faculty of higher medical educational institutions of the III-IV levels of accreditation.

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Methodological instructions recommended for publication at the meeting of the Department of Physiology and Pathophysiology (Minutes № 13 of February 10, 2023) and at the Academic Council of the Medical Faculty № 2 of the State University "UzhNU" (Minutes № 7 of February 15, 2023)

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Methodological instruction to practical lesson № 23
Module 2. Pathophysiology of organs and systems

Theme: PATHOPHYSIOLOGY OF THE EXTERNAL BREATHING. RESPIRATORY FAILURE. INTERPRETATION OF SPIROGRAM

Student should know:

- Classifications, causes and pathogenesis of respiratory failure
- Reasons of origin and pathogenesis of asphyxia

Student should be able to:

• Analyze the role of disorders of ventilation, diffusion of gases through an alveolo-capillary membrane, perfusion in the lung circulation in development of respiratory failure.

• Explain reasons and mechanisms of development of restrictive and obstructive disorders of alveolar ventilation.

LIST OF CONTROL QUESTIONS

1. Determination of concept of failure of the external breathing, criteria, principles of classification.
2. Extra pulmonary and pulmonary disorders of alveolar ventilation: central, nerval-muscular, thoracodiaphragmatic, diminishing of communicating of airways, elastic properties of pulmonary tissue, amount of functioning alveoli.
3. Mechanisms of disorder of alveolar ventilation: dysregulatory, restrictive, obstructive.
4. Reasons and mechanisms of disorders of diffusion of gases in lungs.
5. Disorder of pulmonary circulation of blood. Disorder of general and regional ventilation- perfusion relations in lungs.
6. Disorders of non-respiratory functions of lungs, their influence on hemodynamics and hemostasis. Pathological breathing. Types of the periodic and terminal breathing.
7. Changes of indexes of gas composition of blood and acid-basic state at the different types of respiratory failure, their role for an organism.
8. Pathogenesis of basic clinical signs of failure of the external breathing. Shortness of breath: types, reasons, mechanisms of origin and development.
9. Asphyxia, reasons of origin and mechanisms of development.

Respiratory insufficiency is a pathologic process, which develops as a result of external respiration disorder, when the gas content of the blood does not correspond to organism requirements at rest and under physical load.

Classification

Pathology of the respiratory system may be classified according to different principles. Etiological classification divides it into *acquired and hereditary*

(congenital), as well as *infectious and noninfectious* (including environmental pathology caused by the pathogenic effect of environmental factors).
Topographic classification divides it into *extrapulmonary and pulmonary* according to localization of the initial cause.
Pathogenetic classification divides it into *primary* (pathology begins in the respiratory system) *and secondary* (complication of other diseases — arterial hypertension, heart insufficiency, cardiac asthma, etc.) as well as *total and partial* (disorder of all or one physiological process in the lungs). The latter in its turn is subdivided into ***ventilative, diffusive, perfusive and combined***. In its turn, ventilative pathology is subdivided into *disregulative, obstructive and restrictive*.
Depending on the kind of a typical pathological process it may be divided into *inflammatory, allergic, tumorous, and vascular*. Clinical classification divides it into *acute and chronic*.

Physical factors are mechanical traumas, foreign bodies obstructing the lumen of the respiratory tract, barotrauma leading to increased or decreased solubility of gases in the blood, electrotrauma (if electrical current passes through the respiratory center).
Chemical factors are poisons (muscarine), which lead to respiratory arrest or lung edema, narcotics, side effects of drugs, smoking, harmful industrial factors leading to environmental diseases, chemical warfare agents (phosgene), which cause pulmonary edema when inhaled.
Biological factors are infectious with tropism to the organs of the respiratory (pneumococcus, tubercle bacillus, adenoviruses) and exogenous immune factors (vaccines, heterogenous immune serum).
Endogenous factors are autoimmune and genetic.

Alteration of the ventilation of the alveoli

Increasing ventilation may be at increased its needs as *physiological (muscle work) and pathological* (metabolic acidosis), as well as hyperactivity of the respiratory center neurons. Reducing ventilation is not only a decrease in its needs, but if damaged neurons of the respiratory center, in violation of the nervous and neuromuscular transmission, respiratory muscles, decreased mobility of the chest, increasing the pleural space and restrictive and obstructive lung diseases.
There are ***extrapulmonary and pulmonary*** causes of ventilatory insufficiency of external respiration.
Extrapulmonary include:
1) dysfunction of the respiratory center;
2) violation of the function of spinal cord motor neurons;
3) dysfunction of the neuromuscular system;
4) violation of the mobility of the chest;
5) alteration of the integrity of the chest and pleural cavity
Pulmonary causes include:
1) conduction abnormalities of the airway;
2) alteration of the elastic properties of lung tissue;
3) reducing the number of functioning alveoli.

The mechanisms of disorders of alveolar ventilation can be divided into 3 groups:
1) dysregulation;
2) restrictive, and
3) obstructive.
Dysregulation is the mechanism of ventilation observed during such processes, for which there is a malfunction of the respiratory center.
Restrictive mechanism disorders of pulmonary ventilation called the anatomical or functional loss of gas exchange area of the lungs. Anatomical loss occurs as a result of removal (resection) or replacement of lung tissue (tumor). Atelectasis (alveoli collapse) can also lead to a reduction of the diffusion surface.
Obstructive mechanism is characterized by increased resistance to air flow. Narrow airway lumen may be narrowed by bronchial secretion, contraction or hypertrophy of bronchial muscles, loss of elastic properties of lung tissue that supports the bronchioles in the open state or compression from the outside.

Disorder of diffusion exists when the ratio of diffusion capacity to pulmonary perfusion (cardiac output) is reduced. Diffusion capacity decreases with increasing diffusion distances. In the event of pulmonary edema fluid plasma goes into the interstitial space or into the alveoli and thus the diffusion distance increases. Inflammation also increases the diffusion distance, because in the space between the alveoli and capillaries increases because of the swelling and the formation of connective tissue. Interstitial pulmonary fibrosis and connective tissue separates the alveoli and capillaries. Since diffusion distance - the distance between hemoglobin and alveolar gas, the anemia diffusion capacity is also reduced. Reduced diffusion capacity can also be caused by reduction of the diffusion area, for example, after unilateral pulmonary resection, a decrease in the number of alveolar septa (emphysema), a decrease in the number of alveoli (pneumonia, tuberculosis), pulmonary fibrosis. The diffusion area is reduced by alveoli collapse, intraalveolar pulmonary edema and myocardial lungs. Violation of diffusion is observed with an increase in cardiac output (such as during exercise), blood flows rapidly through the lungs and the contact time of blood and the alveoli decreases.

Disorder of perfusion of single alveoli in relation to its ventilation occurs in the occlusion of blood vessels, such as pulmonary embolism. In addition, the capillaries can be separated from alveolar by proliferating connective tissue in the case of pulmonary fibrosis. Finally, contact with the capillary can be lost if the alveolar septa are destroyed, for example, in the case of emphysema. Alteration of the ventilated alveoli perfusion improves the functional dead space, because the air in the alveoli of such does not participate in gas exchange. This condition can be compensated by deep breathing. If a large volume of the alveoli are not supplied with blood, a decrease in the area of diffusion and perfusion to reduce the compensation can not be guaranteed an increase in depth of breathing.

Clinical manifestations of respiratory failure

Asphyxia - inability to make breathing movements. It is one of the extreme conditions and always develops acutely. During asphyxia, there are three periods:
In the first period of asphyxia is an activation of the respiratory center, which manifests a rapid increase in respiratory rate and depth with a predominance of the inspiratory phase of the phase of expiration. Disorder of oxygen supply of vital centers of the brain is in the first period of the development of anxiety, fear, general excitation, euphoria, different motor responses (before seizures). Developing stress increases the tone of the sympathetic nervous system, which ensures the development of mydriasis, tachycardia, hypertension.
In the second period the respiratory rate gradually decreases with continued maximum amplitude of respiratory movements, increased expiratory phase. This is due to inhibition of the respiratory center under the influence of cerebral hypoxia and severe hypercapnia narcotic effect. Sympathetic hypertonicity followed by parasympathetic.
In the third period of asphyxia, a decrease of the amplitude of respiration, its frequency and finally stopped breathing. Blood pressure is greatly reduced. After a brief cessation of breathing usually occurs a few rare convulsive respiratory (gasping breath), followed by paralysis of respiration.

Pain in lesions of the respiratory system occurs only in those cases where the process involves the pleura, which are a large number of pain receptors. Lung tissue does not have pain receptors.

Cough — a reflex that protects the lungs from the accumulation of secretions and penetration of irritating and damaging agents. Cough is initiated by irritant receptors located in the walls of the tracheobronchial tree, they are extremely sensitive to irritants, and excessive accumulation of secretions. Afferent impulses are transmitted via the vagus nerve in the center of the medulla oblongata responsible for the formation of the cough response. Cough reflex is a protective reaction of the body, weakening it leads to increased likelihood of damage to lung tissue pathogens and exacerbate already developed disease. A prolonged and unproductive cough frequent exhausts the patient and has a negative impact on hemodynamics and respiratory function.

The pathophysiology of the some syndromes and diseases of respiratory systems

Pulmonary edema In the pulmonary capillary filtration pressure is determined by the effective filtration (difference between the gradients of hydrostatic and oncotic pressures). Increase the effective filtration pressure in the pulmonary vessels leads to pulmonary stasis, water filtration of plasma in the interstitial space leads to interstitial

pulmonary edema, and water penetration of plasma into the alveoli is the alveolar pulmonary edema. Increased hydrostatic pressure in the pulmonary capillaries is observed at an inadequate pumping function of the left ventricle. The reason for this may be a decrease in myocardial force of contraction or excessive demand in contraction (heart failure), mitral valve stenosis or regurgitation. Increased pressure in the left ventricle is transmitted to the pulmonary vessels. Increased permeability of pulmonary capillaries may lead to pulmonary edema. Increased permeability of the pulmonary capillary wall to plasma proteins reduces the oncotic pressure gradient and thus increases effective filtration pressure. Permeability of pulmonary capillaries is increased by inhaled corrosive gases (ammonia, chlorine compounds, formaldehyde), prolonged inhalation of pure oxygen. The effect of congestion in the pulmonary circulation is to reduce pulmonary perfusion, and reduction of oxygen absorption. Expansion of stagnant pulmonary vessels prevents alveolar distention and reduces the elasticity of lung tissue, but this advanced congestive capillaries compressing the bronchial tubes, leading to increased resistance to breathing.

Bronchial asthma Bronchial asthma (BA) is the most common chronic lung disease in children. Bronchial asthma is characterized by episodic reversible episodes of bronchospasm, resulting in excess bronchoconstrictor response to various stimuli. Since asthma is a heterogeneous disease and its cause may be a variety of factors, one universal classification does not exist. Nevertheless, we can distinguish two major categories of asthma:

Allergic asthma, in which episodes of bronchospasm is induced with hypersensitivity reactions of type I in response to contact with the lung tissue of endogenous and exogenous allergens. One type of asthma is acquired, atopic asthma, which occurs predominantly during the first two decades of life and is associated with other allergic manifestations in the patient. In allergic asthma serum IgE levels increased, there is eosinophilia.

Non-allergic asthma in which the mechanisms that trigger bronchospasm are not immune. In this form of asthma attack is caused by the influence of a small number of factors that a normal person does not cause bronchospasm. These factors include aspirin, viral respiratory infections, cold, mental stress, exercise, inhaled irritants such as ozone, SO2, NO2. In these patients, plasma levels of IgE is normal. Lower airway obstruction may be caused by a number of changes, including acute bronchospasm, swelling of the airway wall, chronic mucus plugging of the lumen and reorganization of the respiratory tract.

Acute bronchospasm is the result of IgE-dependent release of mediators under the influence of aeroallergens, and is the primary component of early asthmatic response. Bronchospasm also may be the result of the elimination PGE2 in blocking cyclooxygenase (aspirin asthma). Swelling of the walls of the alveoli occurs 6-24 hours after exposure to the allergen and belongs to the late asthmatic response. Formation of chronic airway obstruction caused by mucus exudation of plasma proteins and the accumulation of fragments of necrotic tissue. Resolution of blockage of the lumen can take weeks. Reorganization of the respiratory tract is associated

with a change in its structure as a result of prolonged inflammation and may damage the reversibility of airway obstruction.

Pneumonia The term "pneumonia" is described by the inflammation of lung parenchyma (alveoli and bronchioles). The etiology of pneumonia involves the action of both infectious (bacteria, mycoplasma, viruses, protozoa), and non-infectious agents (aspiration of gastric contents, inhalation of irritating gases and mists). A simple classification of pneumonia divides them into two groups: typical and atypical pneumonia.

Typical pneumonia is the result of infection by bacteria (streptococci, staphylococci, Legionella), which multiply in the extracellular space and causing inflammation and exudation of fluid into the cavity of the respiratory alveoli. Clinic of typical pneumonia severe, the disease is accompanied by a massive intoxication, high fever, profuse discharge of purulent sputum.

The cause of atypical pneumonia are largely intracellular bacteria (mycoplasma, viruses, chlamydia), which cause inflammatory changes mainly in the interstitial space of the lung parenchyma. Lack of full contact with the body's immune system pathogen causes poverty and persistent symptoms for this one.

Pathology of the pleura Inflammation of the pleura (pleuritis) is a common complication of respiratory infections and pneumonia. The main symptom of pleurisy is pain that occurs acutely. Pain limits the movement of the chest, coughing and deep breathing becomes impossible. Breathing becomes shallow and frequent. There is a restrictive type of respiratory failure. The accumulation of fluid in the pleural cavity (pleural effusion) significantly reduces respiratory function. Normally, the visceral and parietal pleural layers are separated with a thin layer of serous fluid. As in any other transcellular space, pleural effusion occurs in the case where the formation of liquid exceeds its absorption.

Five mechanisms associated with abnormal accumulation of fluid in the pleural cavity: 1) increasing the capillary pressure (heart failure),

2) increased capillary permeability (inflammatory changes of the pleura),

3) reduction of colloid osmotic pressure (hypoalbuminemia in the pathology of the lungs and kidneys) 4) increase in negative intrapleural pressure (atelectasis in),

5) disorders of the lymph drainage of pleural space (mediastinal carcinoma).

The accumulation of serous transudate in the pleural cavity is called hydrothorax. The most common cause is heart failure hydrothorax. Among other reasons, is isolated renal failure, nephrosis, liver failure and cancer. Exudate pleural fluid is called with a relative density greater than 1.020 and contains inflammatory cells. States in which the exudate is formed: infection, pulmonary infarction, tumors, rheumatoid arthritis and systemic lupus erythematosus.

Empyema — pus-filled pleural cavity occurs by direct infection of the pleural space. Found in bacterial pneumonia, lung abscess rupture into the pleural cavity, infiltration of subdiaphragmatic disease outbreaks or when infected as a result of trauma.

Chylothorax — effusion of lymph in the pleural cavity. Chylothorax is the result of injury, inflammation or tumor infiltration, lymph transport of violating the thoracic duct into the central circulation. It can also occur as a complication of intrathoracic surgical procedures and the use of large veins for parenteral nutrition and monitoring of central hemodynamics.

Hemothorax — blood in the pleural cavity. Bleeding may result from trauma, surgery on the organs of the chest, swelling or rupture of large vessels, such as aortic aneurysm.

Pleural effusion acts as a stopper for lungs movement, it causes a decrease in their unfolding. Effusion can cause the displacement of mediastinal structures to the opposite side of the chest. Compression of the lungs reduces their ventilation (restrictive type).

Pneumothorax. Normally, the air in the pleural cavity is absent. The flow of air into the pleural cavity is called pneumothorax. Pneumothorax causes partial or complete atelectasis on the affected side. Pneumothorax may occur without apparent cause (spontaneous pneumothorax) or as a result of direct damage to chest or upper respiratory tract infections (traumatic pneumothorax). Tension pneumothorax occurs when the pressure in the pleural cavity above atmospheric pressure. This is a life-threatening condition is the result of trauma chest, at which air can enter but can not leave the pleural space. In this case the trachea and the mediastinum deviates, it causes massive afferent impulses from the nerve trunks of the mediastinum and the development of cardiopulmonary shock.

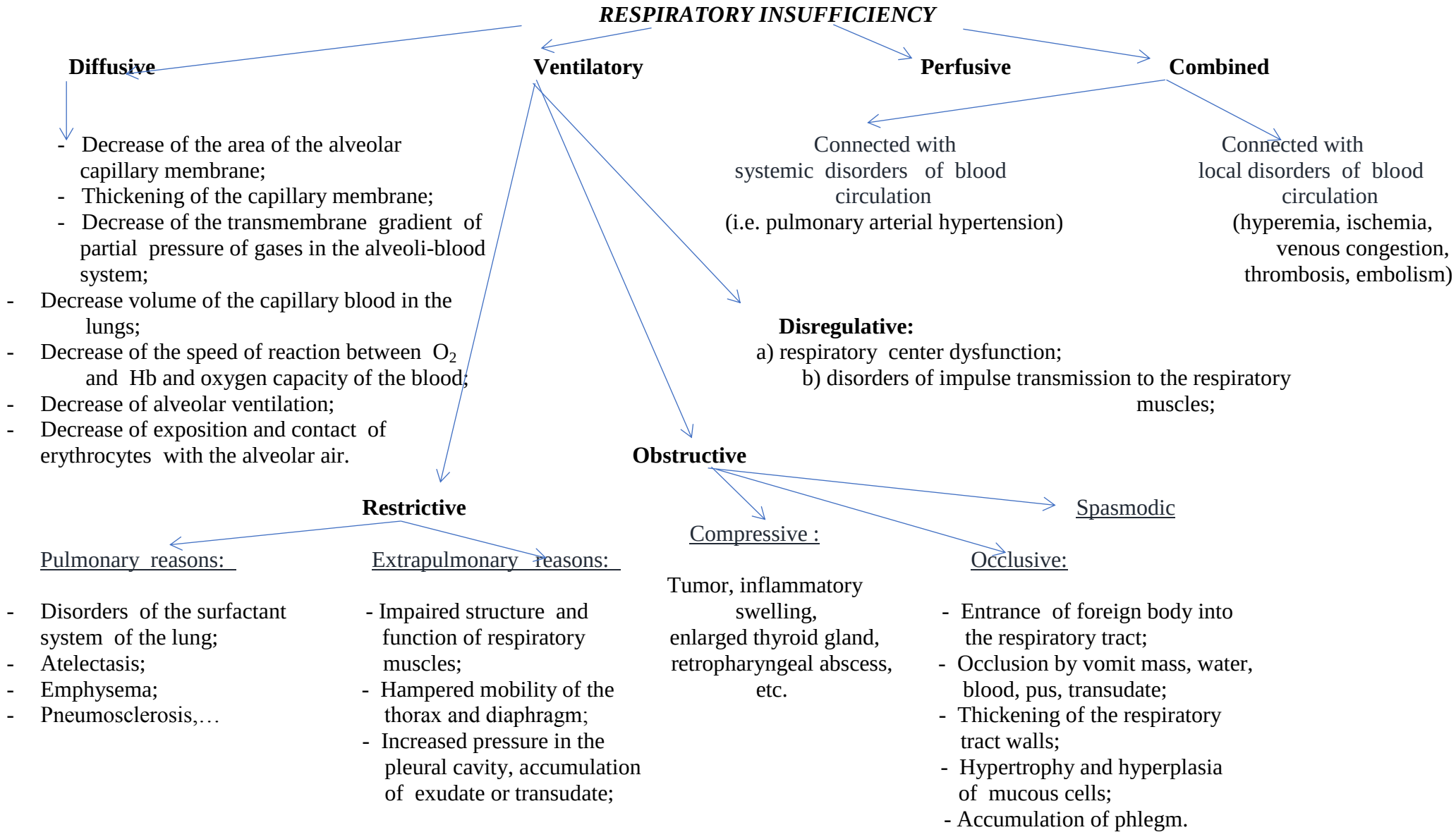

Fig. 12 Respiratory insufficiency

KROK 1 mcqs_A is correct answer

1. A 62-year-old patient was admitted to the neurological department due to cerebral haemorrage. Condition is grave. There is observed progression of deepness and frequency of breath that turnes into reduction to apnoea, and the cycle repeates. What respiration type has developed in the patient?
A Cheyne-Stockes respiration
B Kussmaul respiration
C Biot's respiration
D Gasping respiration
E Apneustic respiration
2. A group of mountain climbers went through the blood analysis at the height of 3000 m. It revealed decrease of HCO3 to 15 micromole/l (standard is 22-26 micromole/l). What is the mechanism of HCO3 decrease?
A Hyperventilation
B Intensification of acidogenesis
C Hypoventilation
Decrease of ammoniogenesis
E Decrease of bicarbonate reabsorption in kidneys
3. A 62-year-old patient was admitted to the neurological department due to cerebral haemorrage. His conditionis grave. There is evident progression of deep and frequent breath that turns into reduction to apnoea and the cycle repeats. What respiration type has developed in the patient?
A Cheyne-Stockes respiration
B Kussmaul respiration
C Biot's respiration
D Gasping respiration
E Apneustic respiration
4.While having the dinner the child choked and aspirated the food. Meavy cough has started, skin and mucose are cyanotic, pulse is rapid, respiration is infrequent, expiration is prolonged. What disorder of the external respiration has the child?
A Stage of expiratory dyspnea on asphyxia
B Stage of inspiratory dyspnea on asphyxia
C Stenotic respiration
D Alternating respiration
E Biot's respiration
5. A 23-year-old patient has been admitted to a hospital with a cranio-cerebral injury. The patient is in a grave condition. Respiration is characterized by prolonged convulsive inspiration followed by a short expiration. What kind of respiration is it typical for?
A Apneustic
B Gasping breath
C Kussmaul's
D Cheyne-Stokes
E Biot's
6. A child was born asphyxiated. What drug must be administered to the newborn to stimulate breathing?
A Aethimizolum
B Lobeline
C Prazosin
D Atropine
E Proserine
7. A female patient, having visited the factory premises with lots of dust in the air for the first time, has got cough and burning pain in the throat. What respiratory receptors, when irritated, cause this kind of reaction?
A Irritant receptors
B Juxtacapillary (J) receptors
C Stretch receptors of lungs
D Proprioceptors of respiratory muscles
E Thermoreceptors
8.While having the dinner the child choked and aspirated the food. Meavy

cough has started, skin and mucose are cyanotic, rapid pulse, rear breathing, expiration is prolonged. What disorder of the external breathing developed in the child?
A Stage of expiratory dyspnea on asphyxia
B Stage of inspiratory dyspnea on asphyxia
C Stenotibreathing
D Alternating breathing
E Biot's breathing
9. A 12 y.o. boy who suffers from bronchial asthma has an acute attack of asthma: evident expiratory dyspnea, skin pallor. What type of alveolar ventilation disturbance is it?
A Obstructive
B Restrictive
C Throraco-diaphragmatic
D Central
E Neuromuscular
10. A patient staying in the pulmonological department was diagnosed with pulmonary emphysema accompanied by reduced elasticity of pulmonary tissue. What type of respiration is observed?
A Expiratory dyspnea
B Inspiratory dyspnea
C Superficial respiration
D Infrequent respiration
E Periodic respiration
11. An unconscious young man with signs of morphine poisoning entered admission office. His respiration is shallow and infrequent which is caused by inhibition of respiratory centre. What type of respiratory failure is it?
A Ventilative dysregulatory
B Ventilative obstructive
C Ventilative restrictive
D Perfusive
E Diffusive
12.The alveolar ventilation of the patient is 5 L/min, the breath frequency is 10per/min, and the tidal volume is 700 ml. What is the patient's dead space ventilation?
A 2,0 L/min
B 0,7 L/min
C 1,0 L/min
D 4,3 L/min
E –
13. X-ray examination discovered lungs emphysema in the patient. What is the reason of short breath development in this case?
A Decreased lungs elasticity
B Increased lungs elasticity
C Inhibition of respiratory center
D Excitation of respiratory center
E Decreasing of alveoli receptors sensitivity
14. A patient after pathological process has a thickened alveolar membrane. The direct consequence of the process will be the reduction of:
A Diffuse lung capacity
B Oxygen capacity of blood
C Minute respiratory capacity
D Alveolar lung ventilation
E Reserve expiratiory capacity
15. A patient has got a spasm of smooth muscles of bronchi. Activators of what membrane cytoreceptors are physiologically reasoned to stop an attack?
A β-adrenoreceptors
B α-adrenoreceptors
C α- and β-adrenoreceptors
D H-cholinoreceptors
E M-cholinoreceptors
16. A12 y.o. boy who suffers from bronchial asthma has an acute attack of asthma: evident expiratory dyspnea, skin pallor. What type of alveolar ventilation disturbance is it?

A Obstructive
B Restrictive
C Throracodiaphragmatic
D Central
E Neuromuscular

17. Examination of a miner revealed pulmonary fibrosis accompanied by disturbance of alveolar ventilation. What is the main mechanism of this disturbance?
A Limitation of respiratory surface of lungs
B Constriction of superior respiratory tracts
C Disturbance of neural respiration control
D Limitation of breast mobility
E Bronchi spasm

18. A man took a quiet expiration. Name an air volume that is mean while contained in his lungs:
A Functional residual capacity
B Residual volume
C Expiratory reserve volume
D Respiratory volume
E Vital lung capacity

19. A man's intrapleural pressure is being measured. In what phase did the man hold his breath, if his pressure is 7,5 cm Hg?
A Quiet inspiration
B Quiet expiration
C Forced inspiration
D Forceexpiration
E –

20. If a man has an attack of bronchiospasm it is necessary to reduce the effect of vagus on smooth muscles of bronchi. What membrane cytoreceptors should be blocked for this purpose?
A M-cholinoreceptors
B N-cholinoreceptors
C α-adrenoreceptors
D β-adrenoreceptors
E α- and β-adrenoreceptors

21. Vagi of an experimental animal were cut on both sides. What respiration changes will be observed?
A It will become deep and infrequent
B It will become shallow and frequent
C It will become deep and frequent
D It will become shallow an infrequent
E No changes will be observed

22. A patient with bronchial asthma has developed acute respiratory failure. What kind of respiratory failure occurs in this case?
A Obstructive disturbance of alveolar ventilation
B Restrictive ventilatory defect
C Perfusion
D Diffusion
E Dysregulation of alveolar ventilation

23. To assess the effectiveness of breathing in patients, the indicator of functional residual capacity is used. It includes the following volumes:
A Expiratory reserve volume and residual volume
B Inspiratory reserve volume and residual volume
C Inspiratory reserve volume, tidal volume, residual volume
D Expiratory reserve volume and tidal volume
E Inspiratory reserve volume and tidal volume

24. A 26-year-old female patient with bronchitis has been administered a broad spectrum antibiotic as a causal treatment drug. Specify this drug:
A Doxycycline
B Interferon
C BCG vaccine
D Ambroxol
E Dexamethasone

25. A 12-year-old child has a viral infection complicated by obstructive bronchitis. Bronchospasm can be eliminated by inhalations of a drug from the following pharmacological group:
A β2-agonists
B M-anticholinergics
C N-cholinomimetics
D β2-adrenergic blockers
E Analeptics
26. Analysis of the experimental spirogram of a 55-year-old person revealed a decrease in tidal volume and respiratory amplitude compared to the situation of ten years ago. The change in these indicators is caused by:
A Decreased force of respiratory muscle contraction
B Gas composition of the air
C Physical build of a person
D Height of a person
E Body mass of a person
27. A patient has increased thickness of alveolar-capillary membrane caused by a pathologic process. The direct consequence will be reduction of the following value:
A Diffusing lung capacity
B Oxygen capacity of blood
C Respiratory minute volume
D Alveolar ventilation of lungs
E Expiratory reserve volume
28. A patient has a traumatic injury of sternocleidomastoid muscle. This has resulted in a decrease in the following value:
A Inspiratory reserve volume
B Expiratory reserve volume
C Respiratory volume
D Residual volume
E Functional residual lung capacity
29. When studying the signs of pulmonary ventilation, reduction of forced expiratory volume has been detected. What is the likely cause of this phenomenon?
A Obstructive pulmonary disease
B Increase of respiratory volume
C Increase of inspiratory reserve volume
D Increase of pulmonary residual volume
E Increase of functional residual lung capacity
30. A 26-year-old female patient with bronchitis has been administered a broad spectrum antibiotic as a causal treatment drug. Specify this drug:
A Doxycycline
B Interferon
C BCG vaccine
D Ambroxol
E Dexamethasone
31. A 28-year-old patient undergoing treatment in the pulmonological department has been diagnosed with pulmonary emphysema caused by splitting of alveolar septum by tissular tripsin. The disease is cased by the congenital deficiency of the following protein:
A α1-proteinase inhibitor
B α2-macroglobulin
C Cryoglobulin
D Haptoglobin
E Transferrin
32. 14 days after quinsy a 15-year-old child presented with morning facial swelling, high blood pressure, "meat slops"urine. Immunohistological study of a renal biopsy sample revealed deposition of immune complexes on the basement membranes of the capillaries and in the glomerular mesangium. What disease developed in the patient?
A Acute glomerulonephritis

B Acute interstitial nephritis
C Lipoid nephrosis
D Acute pyelonephritis
E Necrotizing nephrosis
33. Urine analysis has shown high levels of protein and erythrocytes in urine. This can be caused by the following:
A Renal filter permeability
B Effective filter pressure
C Hydrostatiblood pressure in glomerular capillaries
D Hydrostatic primary urine pressure in capsule
E Oncotic pressure of blood plasma Intrapleural pressure of an individual is being measured.
34. In what phase did he hold his breath if the pressure is - 25 cmH2O?
A Forced inspiration
B Quiet expiration
C Quiet inspiration
D Forceexpiration
E –
35. A female patient suffering from bronchial asthma had got a viral infection that provoked status asthmaticus with fatal outcome. Histological examination of lungs revealed spasm and edema of bronchioles, apparent infiltration of their walls with lymphocytes, eosinophils and other leukocytes; labrocyte degranulation. What mechanism of hypersensitivity underlies the described alterations?
A Reagin reaction
B Immune cytolysis
C Inflammatory
D Autoimmune
E Immune complex
36. Lungs of a preterm infant have areas of atelectasis (pulmonary collapse). The main cause is:
A Surfactant deficiency
B Increased viscous resistance
C Underdeveloped inspiration muscles
D Diminisheforce of surface tension of lungs
E Surfactant excess
37. A patient with marked pneumofibrosis that developed after infiltrating pulmonary tuberculosis has been diagnosed with respiratory failure. What is its pathogenetic type?
A Restrictive
B Obstructive
C Dysregulatory
D Reflex
E Apneistic
38. When studying the signs of pulmonary ventilation, reduction of forced expiratory volume has been detected. What is the likely cause of this phenomenon?
A. Obstructive pulmonary disease
B. Increase of respiratory volume
C. Increase of inspiratory reserve volume
D. Increase of pulmonary residual volume
E. Increase of functional residual lung capacity
39. A 30-year-old man has sustained an injury to his thorax in a traffic incident, which caused disruption of his external respiration. What type of ventilatory difficulty can be observed in the given case?
A. Restrictive extrapulmonary ventilator impairment
B. Restrictive pulmonary ventilatory impairment
C. Obstructive ventilatory impairment
D. Impaired ventilation regulation dysfunction
E. Cardiovascular collapse

40. A patient demonstrates sharp decrease of pulmonary surfactant activity. This condition can result in:
A. Alveolar tendency to recede
B. Decreased airways resistance
C. Decreased work of expiratory muscles
D. Increased pulmonary ventilation
E. Hyperoxemia
41. A woman, who has been suffering from marked hypertension for 15 years, has lately developed dyspnea, palpitations, slightly decreased systolic pressure, while diastolic pressure remains the same. What is the main mechanism of heart failure development in this case?
A. Cardiac overload due to increased vascular resistance
B. Cardiac overload due to increased blood volume
C. Damage to the myocardium
D. Disorder of impulse conduction in the myocardium
E. Dysregulation of cardiac function
42. During training session in the laboratory the students were performing spirography on themselves. What indicator CANNOT be measured with this method?
A. Functional residual capacity
B. Vital capacity
C. Respiratory minute volume
D. Respiration rate
E. Maximal breathing capacity

Tests for Self-Control

1.A 23-year-old patient was hospitalized with a craniocerebral trauma in a serious condition. Respiration is characterized by prolonged convulsive inspiration and short expiration. What type of respiration is it?
A. Kussmaul's.
B. Gasping.
C. Apneustic.
D. Cheyne-Stokes'.
E. Biot's.
2. Cutting of both vagus nerves was reproduced under experimental conditions. What type of respiration will the experimental animal develop?
A. Frequent and shallow.
B. Frequent and deep.
C. Infrequent and shallow.
D. Infrequent and deep.
E. Periodic.
3. 5 ml of air was injected into the pleural cavity of a rat. What type of respiration failure develops in this case?
A. Restrictive disorder of alveolar ventilation.
B. Obstructive disorder of alveolar ventilation.
C. Perfusive.
D. Diffusive.
E. Disregulatory impairment of alveolar ventilation.
4. A patient has been delivered to a hospital in diabetic coma. Respiration is noisy and frequent. Forced expiration follows deep inspiration. What type of respiration is it?
A. Apneustic.
B. Cheyne-Stokes'.
C. Gasping.
D. Stenotic.
E. Kussmaul's.
5. A diphtheria patient developed edema of the larynx. In addition, infrequent and deep respiration with

labored inspiration is observed. What is this respiration called?
A. Apneustic.
B. Kussmaul's.
C. Cheyne-Stokes'.
D. Stenotic.
E. Gasping.

6. A 30-year-old man complains of dyspnea, sensation of heaviness in the right part of the chest, general weakness. Body temperature is 38.9°C. Objectively: during respiration the right part of the thorax is behind the left one. Exudate has been found by means of pleurocentesis in the right part of the chest. What is the main cause of exudation?
A. Decreased resorption of the pleural fluid.
B. Increase of blood pressure.
C. Hyperproteinemia.
D. Aggregation of erythrocytes.
E. Increase of vessel wall permeability.

7. A 12-year-old teenager has developed a serious attack of bronchial asthma with such symptoms: pronounced expiratory dyspnea, pale skin. What kind of alveolar ventilation disorder is taking place?
A. Neuromuscular.
B. Restrictive.
C. Perfusive.
D. Central.
E. Obstructive.

8. A patient was hospitalized to the otolaryngologic department with a foreign body in the upper respiratory tract. What kind of pathological respiration is observed in this case?
A. Frequent, shallow.
B. Frequent, deep.
C. Infrequent.
D. Kussmaul's.
E. Periodic.

9. An X-ray examination revealed diffuse atelectasis in a newborn. What is the most possible cause of this condition?
A. Bronchopneumonia.
B. Bronchial asthma.
C. Occlusion of the pulmonary artery.
D. Surfactant deficiency.
E. Pulmonary tuberculosis.

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 391-407 pp.
2. Victor N. Jelski, Svetlana V. Kolesnikova. Handbook Of Pathophysiology Part 2: Pathophysiology of organs and systems. - Donetsk, Ukraine. – 2011. – 36-50 pp.
3. Krishtal N.V. Pathophysiology: textbook/ N.Krishtal et al.// Kyiv: AUS Medicine Publishing, 2017. - 440-460 pp.

Additional

4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. —3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L.

Methodological instruction to practical lesson № 24
Module 2. Pathophysiology of organs and systems

Theme: PATHOPHYSIOLOGY OF SYSTEMIC CIRCULATION. PATHOPHYSIOLOGY OF HEART. INSUFFICIENCY OF HEART. CORONARY HEART DISEASE.

Student should know:

- Typical pathological states and disorder in the systemic circulation: heart failure; heart failure, arrhythmias of heart; arterial hypertension, arterial hypotension; arteriosclerosis, atherosclerosis.
- Classifications of typical disorders in the systemic circulation.

Student should be able to:

- Analyze the changes of basic parameters of cardio- and hemodynamics at insufficiency of heart (frequency and force of heart-contractions, minute and systolic volumes of blood, systolic, diastolic, mean and pulse arterial blood pressure, venous blood pressure).
- Analyze causative-consequence relationships, to be able to separate pathological and adaptive-compensatory changes, local and systematic processes in pathogenesis of insufficiency of blood circulation, heart failure, myocardial infarction, shock states (cardiogenic shock).

LIST OF CONTROL QUESTIONS

1. Determination of concept of heart failure, principles of its classification, characteristic of disorders of cardio- and hemodynamics. A concept of acute and chronic ("stagnant", congestive) heart failure. Etiology, pathogenesis, stages of chronic heart failure. Mechanisms of development of basic clinical signs of chronic heart failure of blood (shortness of breath, cyanosis, edema).
2. Acute heart failure: etiology, pathogenesis, changes, pathological and adaptive-compensatory. Collapse, shock as variants of acute heart failure .
3. Determinations of concept of insufficiency of heart, principles of classification.
4. Heart failures a result of overload. Reasons of overload of heart by volume and by resistance. Mechanisms of immediate and long duration adaptation of heart to the surplus loading: tachycardia, hyperfunction (hetero-, homeometric), hypertrophy of myocardium. Hypertrophy of heart: types, reasons, mechanisms of development, stage (by Meerson). Features of the hypertrophied myocardium, reasons and mechanisms of its decompensation. Myocardial form of cardiac insufficiency.
5. Coronarogenic damages of myocardium. Insufficiency of coronary circulation (relative and absolute; acute and chronic), mechanisms of development. A concept of "critical stenosis". Consequences of ischemia of myocardium:

depression of retractive activity, electric instability, damage/necrosis of cardiomyocytes, additional damage at reperfusion. Ischemic illness of heart as example of coronary heart disease, its varieties. Clinical-laboratory criteria, signs and complications of myocardial infarction. Pathogenesis of cardiogenic shock. Principles of prophylaxis and treatment of ischemic heart disease.

6. Etiology and pathogenesis of noncoronarogenic damages of myocardium, Cardiomyopathies. Classification. Characteristic of reasons and mechanisms of origin, clinical signs.
7. Extramyocardial insufficiency of heart. Disorders of pericardium. Acute tamponade of heart. Principles of cardioprotection and treatment of insufficiency of heart/ blood circulation.

Cardiac insufficiency is a pathological process, which is characterized by impairment of heart functioning as a pump to move the blood through the vessels at a proper speed and inability to supply tissues with the necessary amount of blood at rest and under physical load.

There are three adaptive mechanisms, which provide the maintenance of the heart minute volume and a proper speed of blood flow:

• *Enforcement of heart contractions.*

• *Acceleration of heart contractions (tachycardia).*

• *Enlargement of heart (hypertrophy).*

Under increased heart load the disorders are divided into two stages.

Stage of compensation takes place, when in spite of the harmful effect of the etiological factor the heart minute volume and blood flow speed are kept at the normal level.

Stage of decompensation develops, when the heart minute volume decreases, congestion in the circulatory system develops.

Etiology of heart failure

Heart failure may be caused by a variety of conditions including:

- conditions that impair the contractility of the heart owing to primary damage to myocardium (e.g., cardiomyopathies);

- conditions that produce a volume overload on the heart (e.g., hypervolemia, valvular insufficiency);

- conditions that generate a pressure overload on the heart (e.g., hypertension, valvular stenosis); - conditions that restrict diastolic filling of the heart (e.g., mitral stenosis, cardiac tamponade);

- conditions that reduce chamber size (e.g., myocardial hypertrophy)

-conditions that decrease venous return of blood to the heart (hypovolemia, collapse).

Classification

The pathophysiological classification of cardiac insufficiency (according to the mechanisms of development) is of special attention. There are three pathophysiological types of cardiac failure.

1. *Cardiac insufficiency due to overload* of the heart occurs, when a healthy heart performs hard work for a long time.
2. *Myocardial type* of cardiac insufficiency results from primary myocardium pathology. It may be caused by disorders of coronary circulation, autoimmune aggression against the heart, infection, intoxication, hypoxia, avitaminosis, systemic hormonal and electrolyte imbalance, some hereditary disturbances of metabolism.
3. *Mixed type* of cardiac insufficiency develops, when damage of the myocardium is combined with heart overload (for example, in rheumatism, when inflammatory lesion of the heart is combined with disorders of the valve apparatus).

Depending on the clinical course, cardiac disturbances are divided into:

a) acute and chronic;
b) right-side and left-side;
c) primary and secondary (as an associated symptom of other diseases — fever, anemia, hyperthyroidism, etc.).

Heart insufficiency manifestations are divided into acute and chronic. Further manifestations are divided into local (in the myocardium) and systemic (in the whole organism). Local manifestations are subdivided into biochemical, morphological and functional changes in the myocardium.

Changes in the Myocardium

Biochemical changes in the myocardium are the following:
damage of the enzyme composition; reduction of oxygen intake (hypoxia);
disorder of oxidative phosphorylation;
reduction in the synthesis of macroergic compounds (ATP);
loss of glycogen; disturbance of protein synthesis; electrolytic imbalance:
•• accumulation of sodium and calcium ions in cells;
•• loss of potassium from cells;
•• hampered reverse transport of Ca^{2+} ions from mitochodria into the sarcoplasmic reticulum;
acidosis in the intracellular media.

Morphological changes in the myocardium include:
• destructive changes in the mitochondria;
• swelling or pyknosis of the nuclei;
• disappearance of transverse striation in the muscle fibers;
• disorders of the nervous apparatus of cells;
• dilatation of the heart cavities;
• substitution of cardiomyocytes by connective tissue (cardiosclerosis);

• cardiomyocyte death.

Functional disturbances of the myocardium are the following:
• cardiac rhythm disorders resulting from disturbance of automatism, excitability, conductivity, contractility;
• impairment of the process of contraction and relaxation of the cardiac muscle fibers;
• reduction of the force and speed of cardiac muscle contraction;
• local contractions of separate cardiomyocytes;
• reduction of the systolic heart volume;
• increase of the residual systolic volume and diastolic pressure;
• changes in ECG.

Systemic Changes in the Organism

Acute Cardiac Insufficiency

If increased heart load is excessive, compensatory mechanisms fail to manage the overload, and acute cardiac insufficiency develops. It is accompanied by significant changes in blood circulation:
• acute decrease of arterial blood pressure;
• increase of venous pressure;
• significant reduction of the minute blood volume;
• circulatory and tissue hypoxia and systemic metabolic acidosis connected with it;
• acute pain;
• disorders of breathing;
• ischemia of the brain, loss of consciousness, convulsions;
• severe changes resembling shock.

Together with metabolic there may be structural changes in the cardiac muscle, so that even under further load reduction heart activity may not be normalized. Acute cardiac insufficiency can develop in myocardial infarction, myocarditis, ventricular fibrillation, paroxysmal tachycardia, heart tamponade, thrombosis and embolism of the pulmonary artery.

Chronic Cardiac Insufficiency

Some manifestations of chronic cardiac insufficiency have been described in the previous chapters, and the pathogenesis has been discussed in details. They are *edema, dyspnea, venous congestion, chronic circulatory hypoxia, cyanosis, basal metabolism increase.* Chronic or congestive cardiac insufficiency develops due to metabolic disturbances in the myocardium in prolonged hyperfunction of the heart or different kinds of myocardium pathology.

HEART FAILURE

Failure due to overload – results of great volume or pressure loads on the heart with normal contractivity	**Myocardial failure** – primary damage to myocardium → decreased myocardial contractivity	**Extramyocardiac failure** – occurs due to conditions which are extrinsic to myocardium
1) **Volume overload** (increased blood filling – *preload*): • Incompetence of the heart valves; • Intracardiac shunts (e.g. in atrial or ventricular septal defect); • Increased venous return (hypervolemia, strenuous physical activity). 2) **Pressure overload** (increased resistance to ejection of the blood from the heart – pressure *afterload*): • Increased pressure in the systemic or pulmonary circulation – systemic or pulmonary hypertension; • Stenosis of the aortic or pulmonary valves.	1) **Coronarogenic damages of myocardium** – Ischemic heart disease: • *Obstructive ischemia* by thrombi, emboli or atherosclerotic plaques; • *Angiospastic ischemia* due to functional disturbances of the vasocnstrictive and vasodilative apparatus of coronary arteries; • *Compressive ischemia* by a tumor. 2) **Non-coronarogenic damages of myocardium**: • *Alterations in the conductive system* of the heart. • *Alterations in myofibers* – cardiomyopatic form: o Hypertrophic – characterized by thickened ventricular muscle mass. Is transmitted genetically in an autosomal dominant pattern. It may be asymptomatic or may be associated with symptoms of ventricular outflow obstruction or impaired diastolic filling. o Dilated (congestive) – characterized by dilation of one or both ventricular chambers. Etiology: alcohol toxicity, genetic abnormality, pregnancy, postviral myocarditis. Clinical picture: slowly progressing biventricular heart failure with low ejection fraction. o Restrictive – characterized by stiff, fibrotic ventricle with impaired diastolic filling. Can be attributed to specific clinical disorders, i.e. amyloidosis, sarcoidosis, genetically inherited diseases,… Restricted diastolic filling and resultant low stroke volume.	1) **Conditions that restrict diastolic filling** (accumulation of fluid in pericardium): • Pericarditis (Idiopathic, radiation induced, infectious, immune-inflammatory [e.g., Dressler syndrome], …); • Neoplastic diseases (mesothelioma, breast or lung carcinoma,…); • Hemorrhage into pericardium (trauma, post cardiopulmonary resuscitation,...). 2) **Conditions that decrease venous return of blood to the heart** (hypovolemia, collaps)

Fig. 13 Heart Failure

KROK 1 mcqs_ *A is correct answer*

1. Dystrophic changes of the heart muscle are accompanied with cardiac cavity enlargement, decrease of the strength of heart contraction, increased amount of blood, which remains in the heart during systolic phase, overfilled veins. For what state of heart is it characteristic?
A Myogenic dilatation
B Tonogenic dilatation
C Emergency stage of hyperfunction and hypertrophy
D Cardiosclerosis
E Tamponage of the heart
2. Transmural myocardial infarction in the patient was complicated with progressive acute left ventricle insufficiency. What is the most typical for this state?
A Edema of the lungs
B Edema of the extremities
C Cyanosis
D Ascites
E Arterial hypertension
3. After a serious psycho-emotional stress a 45-year-old patient suddenly felt constricting heart pain irradiating to the left arm, neck and left scapula. His face turned pale, the cold sweat stood out on it. The pain attack was stopped with nitroglycerine. What process has developed in this patient?
A Stenocardia
B Myocardial infarction
C Stroke
D Psychogenic shock
E Stomach ulcer perforation
4. An animal with aortic valve insufficiency got hypertrophy of its left heart ventricle. Some of its parts have local contractures. What substance accumulated in the myocardiocytes caused these contractures?
A Calcium
B Potassium
C Lactic acid
D Carbon dioxide
E Sodium
5. A 59 year old patient is a plant manager. After the tax inspection of his plant he felt intense pain behind his breastbone irradiating to his left arm. 15 minutes later his condition came to normal. Which of the possible mechanisms of stenocardia development is the leading in this case?
A High catecholamine concentration in blood
B Coronary atherosclerosis
C Intravascular aggregation of blood corpuscles
D Coronary thrombosis
E Functional heart overload
6. The patient with acute miocardial infarction was given intravenously different solutions during 8 hours with medical dropper 1500ml and oxygen intranasally. He died because of pulmonary edema. What caused the pulmonary edema?
A Volume overload of the left ventricular
B Decreased oncotic pressure due to hemodilution
C Allergic reaction
D Neurogenic reaction
E Inhalation of the oxygen
7. A patient who suffers from acute myocarditis has clinical signs of cardiogenic shock. What of the under-mentioned pathogenetic mechanisms plays the main part in shock development?
A Disturbance of pumping ability of heart
B Depositing of blood in organs

C Reduction of diastolic flow to the heart
D Decrease of vascular tone
E Increase of peripheral vascular resistance
8. A 45 year old patient was admitted to the cardiological department. ECG data: negative P wave overlaps QRS complex, diastolic interval is prolonged after extrasystole. What type of extrasystole is it?
A Atrioventricular
B Sinus
C Atrial
D Ventricular
E Bundle-branch
9. A patient suffering from stenocardia was taking nitroglycerine which caused restoration of blood supply of myocardium and relieved pain in the cardiac area. What intracellular mechanism provides restoration of energy supply of insulted cells?
A Intensification of ATP resynthesis
B Reduction of ATP resynthesis
C Increased permeability of membranes
D Intensification of oxygen transporting into the cell
E Intensification of RNA generation
10. In course of a preventive examination of a miner a doctor revealed changes of cardiovascular fitness which was indicative of cardiac insufficiency at the compensation stage. What is the main proof of cardiac compensation?
A Myocardium hypertrophy
B Tachycardia
C Rise of arterial pressure
D Dyspnea
E Cyanosis
11. A patient ill with essential arterial hypertension had a hypertensic crisis that resulted in an attack of cardiac asthma. What is the leading mechanism of cardiac insufficiency in this case?
A Heart overload caused by high pressure
B Heart overload caused by increased blood volume
C Absolute coronary insufficiency
D Myocardium damage
E Blood supply disturbance
12. A 60-year-old patient with a long history of stenocardia takes coronarodilator agents. He has also been administered acetylsalicylic acid to reduce platelet aggregation. What is the mechanism of antiplatelet action of acetylsalicylic acid?
A It reduces the activity of cyclooxygenase
B It reduces the activity of phosphodiesterase
C It enhances the activity of platelet adenylate cyclase
D It enhances the synthesis of prostacyclin
E It has membrane stabilizing effect 6 hours after the myocardial infarction
13. A patient was found to have elevated level of lactate dehydrogenase in blood. What isoenzyme should be expected in this case?
A LDH1
B LDH2
C LDH3
D LDH4
E LDH5
14. A patient with extensive myocardial infarction has developed heart failure. What pathogenetic mechanism contributed to the development of heart failure in the patient?
A Reduction in the mass of functioning myocardiocytes
B Pressure overload
C Volume overload

D Acute cardiac tamponade
E Myocardial reperfusion injury
15. Autopsy of the dead patient who died from pulmonary edema revealed a large yellowgrey nidus in the myocardium, and a fresh thrombus in the coronary artery. What is the most likely diagnosis?
A Myocardial infarction
B Cardiosclerosis
C Myocarditis
D Amyloidosis
E Cardiomyopathy
16. Experimental stimulation of the sympathetic nerve branches that innervate the heart caused an increase in force of heart contractions because the membrane of typical cardiomyocytes permitted an increase in: A Calcium ion entry
B Calcium ion exit
C Potassium ion exit
D Potassium ion entry
E Calcium and potassium ion exit
17. For biochemical diagnostics of myocardial infarction it is necessary to measure activity of a number of enzymes and their isoenzymes. What enzymatic test is considered to be the best to prove or disprove the diagnosis of infarction in the early period after the chest pain is detected?
A Creatine kinase isoenzyme CK-MB
B Creatine kinase isoenzyme CK-MM
C LDH1 lactate dehydrogenase isoenzyme
D LDH2 lactate dehydrogenase isoenzyme
E Aspartate aminotransferase cytoplasmic isoenzyme
18. A patient in three weeks after acute myocardial infarction has pain in the heart and joints and pneumonia. What is the main mechanism of development of postinfarction Dressler's syndrome?
A Autoimmune inflammation
B Ischemia of myocardium
C Resorption of enzymes from necrotized area of myocardium
D Secondary infection
E Vessels ' thrombosis
19. The high level of Lactate Dehydrogenase (LDH) isozymes concentration showed the increase ofLDH-1 and LDH-2 in a patient's blood plasma. Point out the most probable diagnosis:
A Myocardial infarction
B Skeletal muscle dystrophy
C Diabetes mellitus
D Viral hepatitis
E Acute pancreatitis
20. Marked increase of activity of MB-forms of CPK (creatinephosphokinase) and LDH-1 was revealed by examination of the patient's blood. What is the most probable pathology?
A Miocardial infarction
B Hepatitis
C Rheumatism
D Pancreatitis
E Cholecystitis
21. The calcium canals of cardiomyocytes have been blocked on an isolated rabbit's heart. What changes in the heart's activity can happen as a result?
A Decreased rate and force of heart beat
B Decreased heart beat rate
C Decreased force of the contraction
D Heart stops in systole
E Heart stops in diastole
22. Examination of a person revealed that minute volume of heart is 3500 mL ,systolic volume is 50 mL. What is the frequency of cardiac contraction?
A 70 bpm
B 60 bpm
C 50 bpm

D 80 bpm
E 90 bpm
23. Dystrophic alterations of heart are accompanied with dilation of heart cavities, decreased force of heart contractions, increased blood volume that remains during systole in the heart cavity, vein overfill. What heart condition is it typical for?
A Myogenic dilatation
B Tonogenic dilatation
C Emergency stage of hyperfunction and hypertrophy
D Cardiosclerosis
E Cardiac tamponade
25.While preparing a patient to the operation the heart chambers' pressure was measured. In one of them the pressure changed during one heart cycle from 0 to 120 mm Hg. What chamber of heart was it?
A Left ventricle
B Right ventricle
C Right atrium
D Left atrium
E –
24. A patient presents high activity of LDH1,2, aspartate aminotransferase, creatine phosphokinase. In what organ(organs) is the development of a pathological process the most probable?
A In the heart muscle (initial stage of myocardium infarction)
B In skeletal muscles (dystrophy, atrophy)
C In kidneys and adrenals
D In connective tissue
E In liver and kidneys
25. 12 hours after an acute attack of retrosternal pain a patient presented a jump of aspartate aminotransferase activity in blood serum. What pathology is this deviation typical for?
A Myocardium infarction
B Viral hepatitis
C Collagenosis
D Diabetes mellitus
E Diabetes insipidus
26. Blood minute volume of a 30 year old woman at rest is 5 l/m. What blood volume is pumped through the pulmonary vessels per minute?
A 5 l
B 3,75 l
C 2,5 l
D 2,0 l
E 1,5 l
27. A 38 year old patient suffers from rheumatism in its active phase. What laboratory characteristic of blood serum is of diagnostic importance in case of this pathology?
A C-reactive protein
B Uric acid
C Urea
D Creatinine
E Transferrin
28. An animal with aortic valve insufficiency got hypertrophy of its left heart ventricle. Some of its parts havelocal contractures. What substance accumulated in the myocardiocytes caused these contractures?
A Calcium
B Potassium
C Lactic acid
D Carbon dioxide
E Sodium
29. A 59 year old patient is a plant manager. After the tax inspection of his plant he felt intense pain behind his breastbone irradiating to his left arm.15 minutes later his condition came to normal. Which of the possible mechanisms of stenocardia development is the leading in this case?
A High catecholamine concentration in blood
B Coronary atherosclerosis

C Intravascular aggregation of blood corpuscles
D Coronary thrombosis
E Functional heart overload
30. In course of a preventive examination of a miner a doctor revealed changes of cardiovascular fitness which was indicative of cardiac insufficiency at the compensation stage. What is the main proof of cardiac compensation?
A Myocardium hypertrophy
B Tachycardia
C Rise of arterial pressure
D Dyspnea
E Cyanosis
31. ECG of a 44-year-old patient shows signs of hypertrophy of both ventricles and the right atrium. The patient was diagnosed with the tricuspid valve insufficiency. What pathogenetic variant of cardiac dysfunction is usually observed in case of such insufficiency?
A Heart overload by volume
B Coronary insufficiency
C Cardiac tamponade
D Heart overload by resistance
E Primary myocardial insufficiency
32. A 56 year old patient suffering from cardiac insufficiency has edema of feet and shins, edematous skin is pale and cold. What is the leading mechanism of edema pathogenesis?
A Rise of hydrostatic pressure in venules
B Drop of oncotic pressure in capillaries
C Increase of capillary permeability
D Disorder of lymph outflow
E Positive water balance
33. ECG of a 44-year-old patient shows signs of hypertrophy of both ventricles and the right atrium. The patient was diagnosed with the tricuspid valve insufficiency. What pathogenetic variant of cardiac dysfunction is usually observed in case of such insufficiency?
A Heart overload by volume
B Heart overload by resistance
C Primary myocardial insufficiency
D Coronary insufficiency
E Cardiac tamponade
34. A 50 year old patient suffers from essential hypertension. After a physical stress he experienced muscle weakness, breathlessness, cyanosis of lips, skin and face. Respiration was accompanied by distinctly heard bubbling rales. What mechanism underlies the development of this syndrome?
A Acute left-ventricular failure
B Chronic right-ventricular failure
C Chronileft-ventricular failure
D Collapse
E Cardiac tamponade
35. After a serious psychoemotional stress a 48 year old patient suddenly developed acute heart ache irradiating to the left arm. Nitroglycerine relieved pain after 10 minutes. What is the leading pathogenetic mechanism of this process development?
A Spasm of coronary arteries
B Dilatation of peripheral vessels
C Obstruction of coronary vessels
D Compression of coronary vessels
E Increase in myocardial oxygen consumption
36. A 49 year old woman spent a lot of time standing. As a result of it she got leg edema. What is the most likely cause of the edema?
A Increase in hydrostatic pressure of blood in veins
B Decrease in hydrostatic pressure of blood in veins
C Decrease in hydrostatic pressure of blood in arteries

D Increase in oncotic pressure of blood plasma
E Increase in systemic arterial pressure.
37. Patient's systolic blood pressure is 90 mmHg, diastolic-70mmHg.Such blood pressure is caused by decrease of the following factor:
A. Pumping ability of the left heart
B. Pumping ability of the right heart
C. Aortic compliance
D. Total peripheral resistance
E. Vascular tone
38. Due to blood loss the circulating blood volume of a patient decreased. How will it affect the blood pressure in this patient?
A. Systolic and diastolic pressure will decrease B. Only systolic pressure will decrease
C. Only diastolic pressure will decrease
D. Systolic pressure will decrease, while diastolic will increase
E. Diastolic pressure will decrease, while systolic will increase

39. A man presents with glomerular filtration rate of 180 ml/min., while norm is 125±25 ml/min. The likely cause of it is the decreased: A. Plasma oncotic pressure
B. Effective filtration pressure
C. Hydrostatic blood pressure in the glomerular capillaries
D. Renal blood flow
E. Permeability of the renal filter

A 67-year-old man was delivered to the cardiology unit with complaints of periodical pain in the heart, dyspnea after even insignificant physical exertion, cyanosis, and edemas. ECG revealed additional contractions of the heart ventricles. Name this type of rhythm disturbance:
A. Extrasystole
B. Bradycardia
C. Tachycardia
D. Flutter
E. Fibrillation

Tests For Self-Control:

1. Acute failure of the mitral valve was experimentally reproduced in an animal. The heart adapted by activation of the heterometric mechanism. What is the essence of this mechanism?
A. Compensatory hypertrophy of the myocardium.
B. The law of Frank—Starling.
C. Decreased formation of calcium-troponin complexes.
D. Intensification of protein biosynthesis.
E. Intensification of conductivity.
2. A 41-year-old patient with signs of pulmonary edema and left ventricular heart failure was given a diagnosis of aortic stenosis. What is the cause of heart failure development?
A. Increased volume of the vascular bed.
B. Damage of the myocardium.
C. Decreased volume of the circulating blood.
D. Cardiac overload due to increased blood volume.
E. Cardiac overload due to increased blood outflow resistance.
3. A 37-year-old man who had suffered from mitral valve failure for many years developed acute cardiac decompensation. What

pathophysiological variant of cardiac failure is observed in this case?
A. Neurogenic heart damage.
B. Hypoxic heart damage.
C. Coronary heart damage.
D. Cardiac volume overload.
E. Cardiac resistance overload.
4. A patient has mitral valve regurgitation. As a result, cardiac overload by blood volume developed. What is the main mechanism of immediate compensation?
A. Effect of catecholamines.
B. Homeometric.
C. Intensification of protein biosynthesis.
D. Heterometric.
E. Hypertrophy of the myocardium.
5. A woman has suffered from arterial hypertension for 15 years. Now dyspnea and palpitation appeared; systolic pressure decreased a little. What is the basic mechanism of heart failure in this case?
A. Disturbance of conductivity.
B. Cardiac overload with increased blood volume.
C. Damage of the myocardium.
D. Cardiac overload due to increased blood outflow resistance.
E. Disturbance of cardiac activity regulation.
6. A patient demonstrates abrupt arterial pressure increase due to changes of the vascular tone. What compensatory mechanism provides an increased force of myocardial contraction in this case?
A. Renin-angiotensin system activation.
B. Influence of the sympathetic nervous system on the heart.
C. Influence of the parasympathetic nervous system on the heart.
D. Homeometric.
E. Heterometric.
7. A patient has arterial hypertension. As a consequence of hypertensic crisis, acute heart failure developed. What is the main mechanism of heart failure onset in this case?
A. Absolute coronary failure.
B. Cardiac volume overload.
C. Damage of the myocardium.
D. Cardiac resistance overload.
E. Relative coronary failure.
8. A 51-year-old patient complains of dyspnea, palpitation, pain in the right hypochondrium, edema on the legs. ECG shows hypertrophy of both ventricles and the right atrium. Regurgitation of the tricuspid valve is diagnosed. What pathogenetic variety of heart failure is it?
A. Arrhythmic.
B. Cardiac resistance overload.
C. Initial myocardial failure.
D. Cardiac volume overload.
E. Extramyocardial.
9. In an 18-year-old man mitral valve insufficiency without circulation disturbance is revealed. What type of adaptive reaction takes place?
A. Homeometric.
B. Heterometric.
C. Myogenic dilatation.
D. Hypertrophy of the heart.
E. Intensification of conductivity

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 338-354 pp.
2. Victor N. Jelski, Svetlana V. Kolesnikova. Handbook Of Pathophysiology Part 2: Pathophysiology of organs and systems. - Donetsk, Ukraine. – 2011. – 68-80 pp.
3. Krishtal N.V. Pathophysiology: textbook/ N.Krishtal et al.// Kyiv: AUS Medicine Publishing, 2017. - 367-382 pp.

Additional

4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. —3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. - 2000.

Methodological instruction to practical lesson № 25
Module 2. Pathophysiology of organs and systems

Theme: PATHOPHYSIOLOGY OF BLOOD VESSELS

Student should know:

- Modern criteria for diagnostics of arterial hypertension.
- Classifications of arterial hypertension.

Student should be able to:

- Characterize the features of different forms of arteriosclerosis, explain the modern theories of pathogenesis of atherosclerosis.
- Interpret a primary arterial hypertension as multifactor disease.
- Differentiate the role of volume changes and peripheral resistance of blood flow in development of different haemodynamic variants of arterial hypertension.
- Genetic defects as basis of pathogenesis of primary arterial hypertension.
- Explain the role of kidneys in pathogenesis of primary and secondary arterial hypertension.
- Apply knowledge about the experimental models of typical disorders in the system of blood circulation (coronary heart disease, arteriosclerosis, arterial hypertension) for the analysis of their pathogenesis.
- Explain reasons and mechanisms of development of arterial hypotension.

LIST OF CONTROL QUESTIONS

1. A concept of vascular insufficiency. Types, reasons and mechanisms of its development. Arteriosclerosis: determination of concept, classification. Basic forms of arteriosclerosis:

atherosclerosis, mediacalcinosis, arteriolosclerosis, their general characteristics (typical localization, signs, complications).

2. Atherosclerosis. Factors of risk of atherosclerosis. Experimental models. Modern and historical theories of atherogenesis. A role of damage of endothelium, inflammation, inherited and acquired disorders of receptor-mediated transport of lipoproteins (LP) (disorder of receptors of LP, defects of molecules of LP, modification of LP) in atherogenesis. Disorders of transport of lipids in blood. Hyper-, hypo-, dyslipoproteinemias. Dependence of development of dyslipoproteinemias on the factors of environment (diet), heredity and concomitant diseases. Modern classifications of dyslipoproteinemias (primary and secondary; according to the phenotype of LP; with the high or low risk of atherosclerosis), criteria of hypercholesterolemia, hypertryglyceridemia, low level of LPHD.
3. Etiology, pathogenesis of primary (inherited, familial) and secondary (at disorders of feeding, obesity, diabetes mellitus, illnesses of kidneys, hypothyreosis, cirrhosis of liver, influence of medicinal drugs), dyslipoproteinemias. Consequences/complications of dyslipoproteinemias. Principles and aims of renewal of normal lipid composition of blood.

4. Arterial hypertension (AH), determination of concept, principles of classification. Hemodynamic variants of AH. A role of disorders of pressor and depressor systems in development of AH.
5. Primary and secondary arterial hypertension. Etiology, pathogenesis. Experimental models.
6. Primary AH as multifactor disease: a role of factors of heredity and external factors in development of primary AH. Theories of pathogenesis of primary AH (dysregulatory, membrane etc).
7. Mechanisms of development of primary and secondary hypertension of small circle of circulation of blood.
8. Arterial hypotension: determinations of concept, criteria. Etiology and pathogenesis of acute and chronic arterial hypotensions. Collapse. Reasons and mechanisms of development, signs.

Vascular insufficiency is a disorder of blood circulation and blood supply of organs resulting in impairment of substance and oxygen exchange between the blood and tissues.

Atherosclerosis is a pathological process, which is characterized by infiltrative-proliferative changes of the inner layer of elastic-type arteries with deposition of lipids, fibrin and calcium accompanied by elasticity impairment and vessel lumen narrowing.

Endogenous factors play a more decisive role in atherosclerosis development:
• pathological heredity (enzymopathy);
• elderly age (hyperlipoproteinemia and hypercholesterolemia are more frequently observed in elderly people);
• hormonal insufficiency (hypothyroidism, DM, hypogonadism, Cushing's syndrome);
• arterial blood hypertension («the higher the blood pressure, the greater the risk»);
• diastolic hypertension is a more important correlate;
• disorders of metabolism (obesity, gout, xanthomatosis, liver pathology).

Risk factors are:
• hypodynamia;
• overeating, alcohol abuse, smoking;
• stress, which may lead to vessel wall trophicity disorder.

Pathogenesis. Some concepts of atherosclerosis pathogenesis have been proposed:
1. Concept of primary systemic disorders of lipid metabolism and secondary damage of the vascular wall.
2. Concept of primary damage of the vascular wall and its secondary lipoidosis («reaction to injury» hypothesis).

From the point of view of the main link of atherosclerosis pathogenesis there are three concepts distinguished:
1. Hypercholesterolemic.
2. Thrombogenic.
3. Genetic.

Morphological changes in vessels and the dynamics of their atherosclerotic damage proceed in some stages with the following order of events.

1. *Infiltration* of the vessel intima by native or modified lipoproteins of the blood plasma. Lipid deposition is an early event in atherogenesis. Lesions occur primarily within the tunica intima. Excessive capture of lipids by macrophages and infiltration of the arterial wall with macrophages containing low-density lipoproteins. Transformation of macrophages into foam cells, which are the base of lipid stain formation. It leads to endothelial injury. Lipid capture by smooth muscle cells. Lipid-filled smooth muscle cells lose contractility.
2. *Proliferation* is local irritation and multiplication of histiocytes, fibroblasts and smooth muscle cells of vessels, which capture lipids. Connective tissue excrescence. Consolidation of the connective fibers. Thickening of the subendothelium, deformation of the elastic tissue. Formation of atherosclerotic fibrous plaques on the endothelium, which consists of lipid-laden smooth muscle cells surrounded by a fibrous matrix. If a lesion is in progress, it occludes the arterial lumen.
3. *Degeneration* and destruction of the intima and vascular wall. Destruction of foam cells, their lysis, fragmentation of fibrous structures. Formation of lipid stains. Formation of ulcers, which can perforate. Progressing atherosclerotic plaques. The core of fibrous plaques consists of lipids and debris of cells necrotized as a result of insufficient blood supply.
4. *Sclerotization* (calcification) of vessels. The lumen of the atherosclerotically changed vessels narrows as a result of atherosclerotic plaque formation. When the altered complex structure becomes rigid, it causes vascular occlusion.
Atherosclerotic changes in vessels predispose to thrombogenesis. Blood clots are formed in the intima layer. Ischemia (infarction) develops in the region of the damaged vessels. Fibrous plaques are altered by hemorrhage. Functional disturbances of vessels consist in:
• disorders of vessel elasticity;
• vessel incapability of dilatation;
• tendency of the damaged vessels toward spasm.

REGULATION OF BLOOD PRESSURE

Mechanisms	**Vasoconstriction**	**Vasodilation**
Nervous	***Autonomous nervous system:*** SNS: stimulation of α-receptors – increase arteriole resistance to blood flow; $ß_1$-receptors – increased heart rate. ***Baroreceptors (carotid sinus and aortic arch)*** – sensitive to mean arterial pressure. ***Hypothalamus:*** vasopressin (ADH)	***Autonomous nervous system:*** PSNS: vagotonus; SNS: $ß_2$-receptors – vasodilation ***Baroreceptors (carotid sinus and aortic arch)*** – sensitive to mean arterial pressure.

Hormonal	***ADH*** – reabsorption of water; ***Catecholamines*** – vasoconstriction , increased heart rate; ***Mineralocorticoinds (aldosterone)*** – increased sodium absorption, increased sensitivity of blood vessels to vasoconstrictive effects; ***Glucocorticoids*** – increase responsiveness to catecholamines and increased cardiac output. ***Thyroid hormone*** – raise sensitivity to catecholamine sans increases the force of heartbeat.	***Atrial natriuretic hormone*** – excretion sodium in urine; – suppression of aldosterone production; – antagonism against angiotensin; – removal of water from the organism; – increase of glomerular filtration; – inhibition of Ca^{2+} entry into smooth muscle cells and its release from the intracellular depot. ***Progesterone*** –
Renal	***RAAS*** – Vasoconstriction; – Sodium and water retention; – Vessel wall hypertrophy. *Result: increased arterial pressure.*	– Secretion of *phospholipid inhibitor* of renin; – Secretion of *angiotensinase* (inactivation of angiotensin II); – Excretion of sodium in the urine; – Removal water from organism; – Excretion of hormones and other substances with vasoactive effect; – Secretion of *prostaglandins.*
Electrolytes	Na^+ – increased vessel resistance, – retains water elevating circulating blood volume. Ca^{2+} – raises the tonus of contractile fibers.	***Prostaglandins and vagus nerve*** promote entry of K^+ into the cells (maintains the rest potential of the membranes of excitable cells)
Local metabolic and endothelial	———————	***Monooxide of nitrogen (NO)*** contributes to arterial vessel dilation. ***Lactate, bradykinin, histamine*** cause vasodilation.

Fig 14. Mechanisms of blood pressure regulation

Hypertension is a sustained elevation of the systemic arterial pressure.
Primary and secondary hypertensions are distinguished.
Primary hypertension is a sustained elevation of the arterial BP which is not connected with certain diseases or pathological processes in body‘s organs. The cause of primary hypertension is unknown, hence essential hypertension (EH). In essential hypertension increased BP is main and often the only sign of disease.
Secondary hypertension is a sustained elevation of the arterial BP which results from pathological processes in body‘s organs, hence symptomatic hypertension. Secondary hypertension occurs at the following conditions:
-renal diseases (glomerulonephritis, pyelonephritis, polikistosis of kidneys);
-endocrine diseases (mainly at adrenal tumors: pheochromocytoma, Cushing‘s syndrome, Conn‘s disease (primary hyperaldosteronism));
-diseases of the heart and vessels (some valve diseases, coarctation of the aorta, atherosclerosis);
- the nervous system diseases (encephalitis, trauma, hemorrhage in the brain).
In all symptomatic hypertensions the etiology is known. Cessation of the cause leads to normalization of the arterial BP.
Risk factors of essential hypertension:
-age – EH is more common in adults than in adolescents and children;
-sex – men are affected more often than women;
-race – EH is more common among blacks than among whites;
-heredity – in some families EH develops several times more often than in remaining population; -high-sodium diet – EH is widely spread in countries (Japan, Chine) where people have a high salt intake;
-obesity;
-physical inactivity;
-hyperinsulinemia;
-oral contraceptive drugs- prolonged treatment with oral contraceptives containing estrogens produces significant hypertension in some women(due to an increase in circulating levels of angiotensinogen);
-stress.
At present there are two concepts in ***pathogenesis of essential hypertension***: the dysregulation theory and the membrane theory.
The dysregulation theory explains an origin of EH by disturbances of the mechanisms which control blood pressure. The membrane theory explains an origin of EH by primary disturbances in smooth-muscle cells of arterioles.
The dysregulation theory According to this theory there are three stages in pathogenesis of essential hypertension including:
1. The first stage is due to the sympathetic system overactivation caused by frequent and prolonged stresses. Persistent negative emotions cause overstrains of the higher parts of central nervous system, especially the cerebral cortex. It results in development of a stable focus of excitation (―pathological dominant‖) in the vasomotor center of the subcortical region. In its turn, the latter causes an increase in excitability of the sympathetic division of the nervous system. It leads to a release a lot of catecholamines into the blood. Arterial blood pressure increases due to the following effects of catecholamines:

-an increase in the cardiac output, -an increase in the peripheral resistance,
-a spasm of afferent arterioles of the kidneys with a resultant release of renin by the juxtaglomerular cells
2. The second stage is due to an activation of the renin-angiotensin system. Renin cleaves angiotensinogen, a circulating α2-globulin produced in the liver, to form angiotensin I which is further modified by converting enzyme to the angiotensin II. Role of angiotensin II in an increase in arterial blood pressure :
-angiotensin II is a potent vasoconstrictor,
-angiotensin II i stimulates the production of aldosterone by the adrenal cortex.
3. The third stage is due to an activation of the aldosteronevasopressin system. Adrenal secretion of aldosterone in response to renin stimulation, results in increased renal absorption of sodium. Increased concentration of Na^+ in the blood causes the excitation of osmoreceptors with consequent production of vasopressin by nuclei of hypothalamus. Increase in arterial blood pressure is due to the following effects of vasopressin:
-it causes an increase in renal absorption of water that leads to increased total blood volume and, accordingly, to increased arterial BP,
-vasopressin possesses the direct vasoconstrictory action. Later in the cause of EH the stage of in reversible structural changes occurs.
Firstly sustained spasm of the arterioles leads to hypertrophy of their musculature, and there is some organic narrowing of the vessels.
Then hypertrophied smooth muscle cells are subjected to dystrophy changes and necrosis. Replacement of them by connective tissue occurs (arteriosclerosis). Arteriolar walls become rigid and incapable to constriction and dilatation. Total PR increases causing sustained increased arterial blood pressure.
The membrane theory According to this theory essential hypertension develops due to disturbances of ion pumps of the smooth muscle cells membranes:
Inhibition of the work of Na^+-K^+ pumps leads to an accumulation of Na^+ in endotheliocytes of arterioles. It results in the following:
-swelling of the walls of arterioles and their narrowing,
-increased sensitivity of smooth muscle cells to the action of catecholamines,
-damage and necrosis of cells with consequent replacement of them by connective tissue and development arteriosclerosis
Disorder of the work of Ca^{++} pumps leads to impaired removal of Ca^{++} from cells. Accumulation of Ca^{++} into cytoplasm of cells results in the following:
-sustained contraction of smooth muscle of arterioles that causes increased total peripheral resistance,
-damage of cells with consequent development of arteriosclerosis.

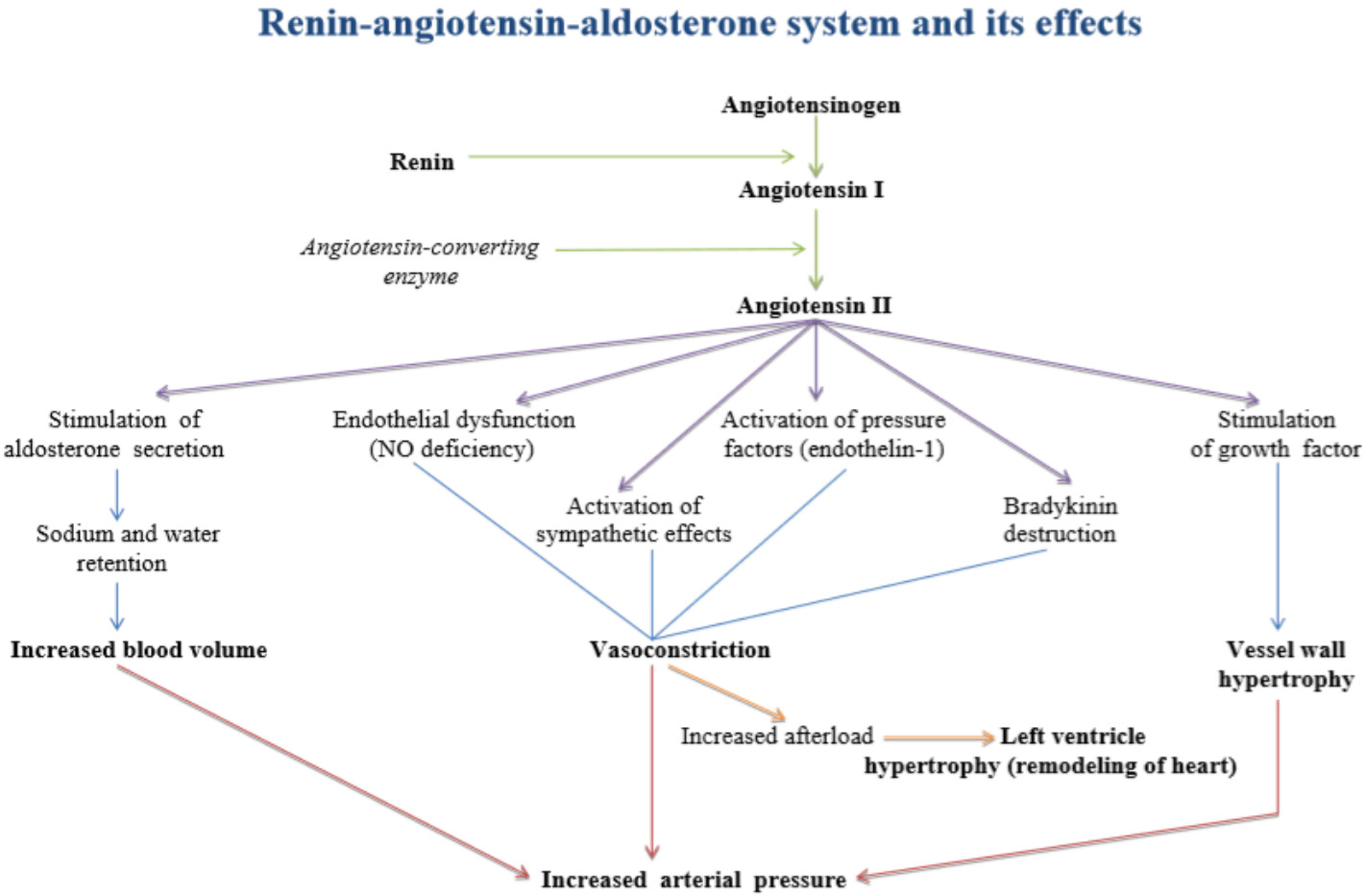
Renin-angiotensin-aldosterone system and its effects
Angiotensinogen
Renin
Angiotensin I
Angiotensin-converting enzyme
Angiotensin II
Stimulation of aldosterone secretion
Endothelial dysfunction (NO deficiency)
Activation of pressure factors (endothelin-1)
Stimulation of growth factor
Activation of sympathetic effects
Bradykinin destruction
Sodium and water retention
Increased blood volume
Vasoconstriction
Vessel wall hypertrophy
Increased afterload
Left ventricle hypertrophy (remodeling of heart)
Increased arterial pressure

Fig 15. RAAS

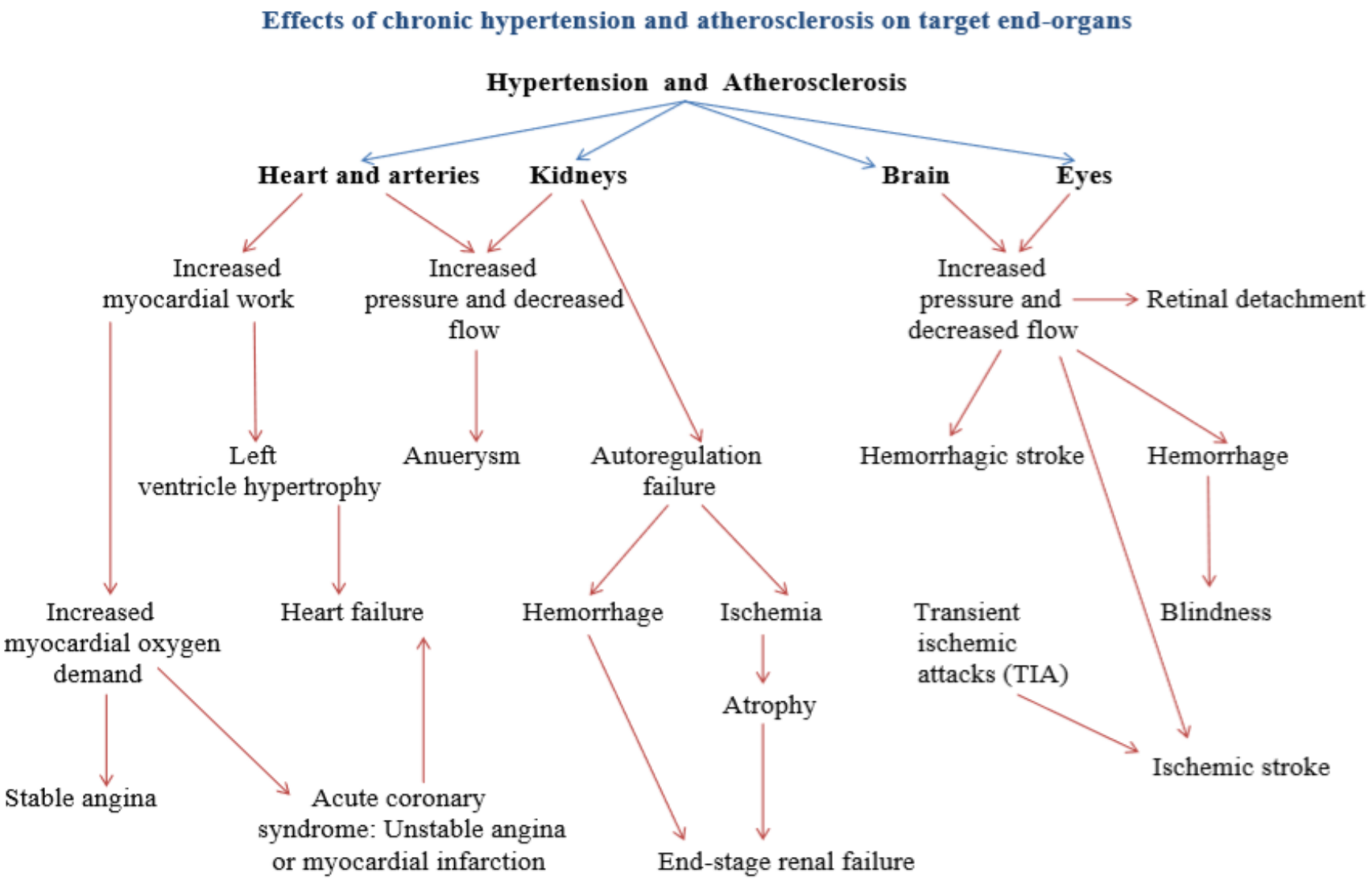

Fig. 16 Effects of atherosclerosis and hypertension on humans' body

KROK 1 mcqs_A is correct answer

1. Arterial hypertention is caused by the stenosis of the renal arteries in the patient. Activation of what system is the main link in the pathogenesis of this form of hypertension?
A Renin-angiotensin
B Sympathoadrenal
C Parasympathetic
D Kallikrein-kinin
E Hypothalamic-pituitary
2. Prophylactic medical examination of a 36 year old driver revealed that his AP was 150/90 mm Hg. At the end of working day he usually hears ear noise, feels slight indisposition that passes after some rest. He was diagnosed with essential hypertension. What is the leading pathogenetic mechanism in this case?
A Neurogenetic
B Nephric
C Humoral
D Endocrinal
E Reflexogenic
3. Arterial pressure of a surgeon who performed a long operation rised up to 140/110 mm Hg. What changes of humoral regulation could have caused the rise of arterial pressure in this case?
A Activation of sympathoadrenal system
B Activation of formation and excretion of aldosterone
C Activation of renin angiotensive system
D Activation of kallikrein kinin system
E Inhibition of sympathoadrenal system
4. A driver who got a trauma in a road accident and is shocked has reduction of daily urinary output down to 300 ml. What is the main pathogenetic factor of such diuresis change?
A Drop of arterial pressure
B Drop of oncotic blood pressure
C Increased vascular permeability
D Decreased number of functioning glomerules
E Secondary hyperaldosteronism
5. A 70 year old man is ill with vascular atherosclerosis of lower extremities and coronary heart disease. Examination revealed disturbance of lipidic blood composition. The main factor of atherosclerosis pathogenesis is the excess of the following lipoproteins:
A Low-density lipoproteins
B Cholesterol
C High-density lipoproteins
D Intermediate density lipoproteins
E Chylomicrons
6. A patient has arterial hypertension. What long-acting drug from the group of calcium channel blockers should be prescribed?
A Amlodipine
B Octadine
C Pyrroxanum
D Atenolol
E Reserpine
7. A patient with constant headaches, pain in the occipital region, tinnitus, dizziness has been admitted to the cardiology department. Objectively: AP- 180/110 mm Hg, heart rate - 95/min. Radiographically, there is a stenosis of one of the renal arteries. Hypertensive condition in this patient has been caused by the activation of the following system:
A Renin-angiotensin
B Hemostatic
C Sympathoadrenal
D Kinin
E Immune
8. A 43-year-old-patient has arterial hypertension caused by an increase in

cardiac output and general peripheral resistance. Specify the variant of hemodynamic development of arterial hypertension in the given case:
A Eukinetic
B Hyperkinetic
C Hypokinetic
D Combined
E ----
9. A 16-year-old female patient has fainted after quickly changing her body position from horizontal to vertical one. Which process from the ones listed below has caused the loss of consciousness in the first place?
A Decreasing venous return
B Increasing venous return
C Increasing central venous pressure
D Decreasing oncotic pressure of blood plasma
E Increasing arterial pressure
10. A patient has insufficient blood supply to the kidneys, which has caused the development of pressor effect due to the constriction of arterial resistance vessels. This is the result of the vessels being greately affected by the following substance:
A Angiotensin II
B Angiotensinogen
C Renin
D Catecholamines
E Norepinephrine
11. A 40-year-old patient complains of intensive heartbeats, sweating, nausea, visual impairment, arm tremor, hypertension. From his anamnesis: 2 years ago he was diagnosed with pheochromocytoma. Hyperproduction of what hormones causes the given pathology?
A Catecholamines
B Aldosterone
C Glucocorticoids
D ACTH
E Thyroidal hormones
12. Arterial pressure of a surgeon who performed a long operation rised up to140/110mm Hg. What changes of humoral regulation could have caused the rise of arterial pressure in this case?
A Activation of sympathoadrenal system
B Activation of formation and excretionof aldosterone
C Activation of renin angiotensive system
D Activation of kallikrein kinin system
E Inhibition of sympathoadrenal system
13. An aged man had raise of arterial pressure under a stress. It was caused by activation of:
A. Sympathoadrenal system
B Parasympathetic nucleus of vagus
C Functions of thyroid gland
D Functions of adrenal cortex
E Hypophysis function
14. Prophylactic medical examination of a 36 year old driver revealed that his AP was 150/90 mm Hg. At the end of working day he usually hears ear noise, feels slight indisposition that passes after some rest. He was diagnosed with essential hypertension. What is the leading pathogenetic mechanism in this case?
A Neurogenetic
B Nephric
C Humoral
D Endocrinal
E Reflexogenic
15. Systemic arterial pressure of an adult dropped from 120/70 to 90/50 mm Hg that led to reflectory vasoconstriction. The vasoconstriction will be maximal in the following organ:
A Bowels
B Heart
C Brain

D Kidneys
E Adrenals
16. A patient ill with essential arterial hypertension had a hypertensic crisis that resulted in an attack of cardiac asthma. What is the leading mechanism of cardiac insufficiency in this case?
A Heart overload caused by high pressure
B Heart overload caused by increased blood volume
C Absolute coronary insufficiency
D Myocardium damage
E Blood supply disturbance
17. In response to a change in body position from horizontal to vertical blood circulation system develops reflectory pressor reaction. Which of the following is its compulsory component?
A Systemic constriction of the venous vessels
B Systemic dilatation of the arterial resistive vessels
C Decrease in the circulating blood volume
D Increase in the heart rate
E Weakening of the pumping ability of Heart
18. A patient with hypertensic crisis has increased content of angiotensin II in blood. Angiotensin pressor effect is based on:
A. Contraction of arteriole muscles
B. Activation of biogenic amine synthesis
C. Prostaglandin hyperproduction
D. Vasopressin production stimulation
E. Activation of kinin–kallikrein system
19. A 43-year-old-patient has arterial hypertension caused by increase in cardiac output and general peripheral resistance. Specify the variant of hemodynamic development of arterial hypertension in the given case:
A. Eukinetic
B. Hyperkinetic
C. Hypokinetic
D. Combined
E. –
20. A patient has insufficient blood supply to the kidneys, which has caused the development of pressor effect due to constriction of arterial resistance vessels. This condition results from the vessels being strongly affected by the following substance:
A. Angiotensin II
B. Angiotensinogen
C. Renin
D. Catecholamines
E. Norepinephrine
21. Patient's systolic blood pressure is 90 mmHg, diastolic-70mmHg.Such blood pressure is caused by decrease of the following factor:
A. Pumping ability of the left heart
B. Pumping ability of the right heart
C. Aortic compliance
D. Total peripheral resistance
E. Vascular tone

22. A 14-year-old adolescent has diphtheria. During the peak of the disease against the background of acute drop in body temperature and tachycardia the blood pressure is 70/50 mm Hg. What type of vascular tone disturbance is it?
A. Acute hypotension
B. –
C. Chronic hypotension
D. Somatoform autonomic dysfunction
E. Essential hypotension

Tests for Self-Control

1. A 60-year-old man has blood vessel atherosclerosis. Excess of what substances plays a leading role in the pathogenesis of this disease?
A. Histic lipoprotein lipase.
B. High-density lipoproteins.
C. Chylomicrons.
D. Low-density lipoproteins.
E. Fatty acids.

2. Rabbits were fed with addition of cholesterol. In 5 months atherosclerotic changes in the aorta were found. What is the main cause of atherogenesis in this case?
A. Endogenic hypercholesterinemia.
B. Overeating.
C. Inactivation of receptors to low-density lipoproteins.
D. Hypodynamia.
E. Exogenous hypercholesterinemia.

3. In a patient with persistent arterial hypertension angiography showed atherosclerotic lesion of both renal arteries. What is the initial mechanism of arterial hypertension development?
A. Increased secretion of vasopressin.
B. Increased level of catecholamines.
C. Increased level of hydrocortisone.
D. Increased cardiac outflow.
E. Increased production of renin.

4. A patient suffers from hypertensic crisis. An increased level of angiotensin II was found in the blood. What is the pressure effect of angiotensin associated with?
A. Activation of biogenic amine synthesis.
B. Contraction of the arteriole muscles.
C. Hyperproduction of prostaglandins.
D. Stimulation of vasopressin formation.
E. Activation of the kallikrein-kinin system.

5. A patient has arterial hypertension caused by renal artery stenosis. Activation of what system is the main mechanism of this form of hypertension?
A. Sympatho-adrenal.
B. Renin-angiotensin.
C. Parasympathetic.
D. Kallikrein-kinin.
E. Hypothalamo-pituitary.

6. A patient with idiopathic hypertension revealed increased concentration of vasopressin in the blood. The function of what organ does this hormone influence directly?
A. Liver.
B. Kidneys.
C. Heart.
D. Lungs.
E. Epinephral glands.

7. A 43-year-old patient suffers from arterial hypertension caused by a moderate increase of cardiac outflow and general peripheral resistance. What hemodynamic variant of arterial hypertension has developed in this case?
A. Eukinetic.
B. Hyperkinetic.
C. Hypokinetic.
D. Akinetic.
E. Combined.

8. A 65-year-old man has been suffering from arterial hypertension for 15 years. Now systolic pressure is decreasing and diastolic pressure remains increased. What hemodynamic type of arterial hypertension is it?
A. Hyperkinetic.
B. Normokinetic.
C. Hypokinetic.
D. Eukinetic.
E. Dyskinetic.

9. Arterial hypertension was reproduced by narrowing the renal arteries in a dog. Activity of the renin-angiotensin-aldosterone system increased. What component of this system has the strongest pressor effect?
A. Renin.
B. Angiotensin II.
C. Angiotensin I.
D. Kinins.
E. Aldosterone.

10. A patient with alcoholic cirrhosis of the liver dyspnea. Decrease of arterial pressure, ascites, dominal veins, and splenomegaly have been hemodynamics is observed?
A. Failure of the left ventricle.
B. Failure of the right ventricle.
C. Total heart failure.
D. Collapse
E. Portal hypertension.

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 364-387 pp.
2. Victor N. Jelski, Svetlana V. Kolesnikova. Handbook Of Pathophysiology Part 2: Pathophysiology of organs and systems. - Donetsk, Ukraine. – 2011. – 59-67 pp.
3. Krishtal N.V. Pathophysiology: textbook/ N.Krishtal et al.// Kyiv: AUS Medicine Publishing, 2017. – 390-416 pp.

Additional

4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. —3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. - 2000.

Methodological instruction to practical lesson № 26
Module 2. Pathophysiology of organs and systems

Theme: HEART ARRHYTHMIAS. PATHOPHYSIOLOGIC BASICS OF ECG ANALYSIS

Student should know:

- Mechanisms of development of arrhythmias.

Student should be able to:

- Apply knowledge about typical disorders of heart rhythm (disorders of automatism, excitability, conductivity and combined) for the analysis of electrocardiogram.

LIST OF CONTROL QUESTIONS

1. Arrhythmias of heart: classification, reasons, mechanisms, typical electrocardiographic signs.
2. A role of additional conduction ways of heart in development of arrhythmias.
3. Reasons and mechanisms of origin of ectopic foci of excitation in myocardium, mechanisms of the repeated entrance and recirculation of excitation.
4. Fibrillation and defibrillation of heart.
5. Disorders in conductive system of a heart.
6. ECG signs of ischemic heart disease.

Disorders of cardiac rhythm — arrhythmias are the most often heart disease. It can varied from non-dangerous altered heart rhythm till dramatically drop of blood pressure and death. In general arrhythmia can arise from disorder of impulse formation or impulse conduction.

Formation of heart rhythm is based on automaticity — ability of pacemakers to generate impulse itself. Disorders of automaticity are classified:

1. Increase of automaticity

a. Increase of automaticity of sinus node
b. Increase of automaticity of latent pacemakers
c. Ectopic sources of automaticity

2. Trigger activity

3. Decrease of automaticity

Under normal conditions heart rate in *nomotopic pacemaker* (sinus node) and in *latent pacemakers* (sinoatrial node, Purkinje cells) is controlled by neuro-endocrine factors — acetylcholine, norepinephrine, epinephrine. So in case of emotional stress, physical activity or other stress reactions release of catecholamines leads to increase

of heart rate (*nomotopic tachycardia*) and play important role in adaptation and compensatory reactions. The same effect can be in case of action of catcholamine mimetics or acetylcholine blockers. These factors not only maintain increase of automaticity of nomotopic pacemaker and but also they can cause activity of latent pacemakers. If a
rate of depolarization of latent pacemaker more than a rate of depolarization of nomotopic pacemaker ectopic impulse *(extrasystole*) or ectopic rhythm (*paroxysmal tachycardia*) occurs. Sometime in damage of myocardium (hypoxia, acidosis, toxins) some cardiomyocytes which are not belonged to conductive heart system can get pathological ability for spontaneous depolarization of membrane and so they become sources of *ectopic rhythm*.
Trigger activity is the term which explain occurrence of tachycardia in some cases when normal action potential causes additional depolarization and new action potentials (depolarizing after-potentials). There are two types of these depolarizing after-potentials — *early depolarizing afterpotentials and late depolarizing after-potentials.* The early depolarizing after-potentials occur in case of prolonged action potential (in bradycardia, hypokaliemia, use of antiarrhythmic drugs). The late depolarizing afterpotentials are generated after post-hyperpolarization and caused by tachycardia, digitalis intoxication, and hypercalcaemia.

Increase of activity of parasympathetic system and release of acetylcholine lead to decrease of heart rate. A lot of toxins and drugs can mime or block action of the acetylcholine so they can decrease heart rhythm too.
Other cause of arrhythmia — disorders of impulse conductivity. They are classified:
1. Block of impulse conductance
2. Re-entry
3. Additional conducting pathway

Impulse conductivity is blocks when impulse achieves part of myocardium which is unable for electric excitation by its nature (scar) or functional state (refractory period). Block can be temporary or constant, one-sided or double-sided. Most common causes of heart block are myocardial ischemia, myocardial sclerosis, myocardial trauma, drugs. Conduction block leads to bradyarrhythmia and appearance of rhythm from latent pacemaker or ectopic rhythm.

Re-entry is the universal mechanism for the most of tachyarrhythmias. It is circulation of electrical circuit in part of myocardium which cases periodical depolarization of cardiomyocytes. The basis of this disorder is a change of local conductance when the impulse from one point of myocardial net is distributed with unequal velocity so the impulse can ―return‖ to the initial point and cause new wave of depolarization before a wave of depolarization from nomotopic pacemaker. Local ischemia or drugs can change features of some part of myocardial net (result in local block or slowing of impulse propagation) so are most common causes of this mechanism.

Additional conducting pathway (like Kent bundle) is the anatomic abnormality in some peoples. Abnormal location of these pathways causes too fast distribution of impulse between atrium and ventricles with no atrioventricular pause. It looks like giant re-entry mechanism where the role of slow part of conductive net is played by normal conductivity system. The results of this disorder are tachyarrhythmia and deficit of ventricle filling.

Clinically arrhythmias are divided into two groups: *bradyarrhythmias and tachyarrhytmias.*

Bradyarrythmia or bradycardia is decrease of heart rate less then 55 beats per minute. The decrease of heart rate lead to decrease of cardiac output, so it leads to decrease of blood pressure and as a result — circulatory hypoxia. Clinical signs are maintained by last one and include picture of brain hypoxia, activation of sympatho-adrenal system mostly.

Types of bradyarrhytmia:

1. Atrial bradycardia
a. Respiratory sinus arrhythmia
b. Sinus bradycardia
c. Sick sinus syndrome
2. Atrioventricular nodal bradycardia
a. Junctional rhythm
b. Sinoatrial block
3. Ventricular bradycardia

Respiratory sinus arrhythmia — occurs during respiration. Heart rate increases during inhalation and decreases during exhalation. Caused by changes in the vagal tone during respiration.

Sinus bradycardia — can be normal in athletes, caused by increase tone of parasympathetic nervous system, damage of sinus nod by ischemia, drugs, trauma.

Sick sinus syndrome — variability of sinus rhythm rate connected with malfunction of sinus node often due to ageing.

Junctional rhythm — rhythm from AV node in case of total damage of nomotopic pacemaker.

Sinoatrial block — most common cause of bradycardia, usual complication of ischemic heart disease. In the basis of this disorder — block of impulse propagation in AV node.

First degree AV block — on ECG PR interval greater than 0.20sec
Second degree AV block —
Type Mobitz I — on ECG there is progressive prolongation of PR interval (Wenckebach periods) with disappearance of QRS complex.
Type Mobitz II — on ECG there is prolonged PR interval and disappearance of QRS complex occurs suddenly without prolongation of PR interval. Disappearance of QRS

can be regular (drop of every 4th QRS complex) and clustered (drop of 2 or 3 QRS complexes in series).

Third degree AV block — on ECG there is connection between P waves and QRS complexes. QRS complexes very deformed and are manifestation of ventricular rhythm.

Ventricular rhythm — heart rate less than 45 beats per minute, source of impulses lies below Purkinje cells, occurs in inhibition of nomotopic pacemaker, third degree AV block. On ECG this one is characterized by deformed QRS complexes.

Tachyarrythmia or tachycardia is increase of heart rate more then 100 beats per minute (varies according to age and sex). The progressive high increase of heart rate lead to decrease of stroke volume due to shortening of ventricles filling time, so it leads to decrease of cardiac output, drop of blood pressure and as a result — circulatory hypoxia. In addition increase of heart rate increases oxygen demand of myocardium and make period of filling of coronary arteries shorter (they are filled during diastole). Both these phenomena lead to progressive heart ischemia and make worse circulatory hypoxia. The last one makes clinical picture as it makes in case of bradyarrhythmia.

Types of tachyarrhytmia:

1. Supraventricular tachyarrhythmia
a. Sinus tachycardia
b. Atrial tachycardia
c. Atrial flutter
d. Atrial fibrillation
e. Paroxysmal supraventricular tachycardia (AV nodal reentrant tachycardia)
f. Wolff-Parkinson-White syndrome
2. Ventricular tachyarrhythmia
a. Ventricular tachycardia
b. Torsades de pointes
c. Ventricular fibrillation

Sinus tachycardia — heart rate 100-180 bpm. Normal P wave and QRS complexes. Usually this is result of increase of sympathetic tone (stress, physical exertion, fever, hypethyreosis, hypoxemia).

Atrial tachycardia — heart rate 100-180 bpm. P wave is deformed.

Atrial flutter — on ECG P waves look like saw teeth their rate 250-300 bpm. High rate (>280) of impulses from sinus node doesn‘t allow AV node to pass excitation to ventricles, so ventricles has their own rhythm. Decrease of atrial rhythm to 200 bpm leads to propagation of this rhythm to ventricles and as a result heart rate increases.

Atrial fibrillation — on ECG P waves looks chaotic and often unrecognizable. It is potential life-threatening state because of decrease of stroke volume. Also in atrium in this condition there is high risk of thrombus formation and further arterial embolisation due to disorder of normal hemodynamics (local congestion of blood).

Most common cause of atrial fibrillation is dilatation of left or right atrium (in mitral stenosis or insufficiency).

Paroxysmal supraventricular tachycardia — abrupt attack of tachycardia 140-250 bpm with the same abrupt cessation of this one. Reentry — the main mechanism of this type of arrhythmia.

Wolff-Parkinson-White syndrome — result of presence of additional conducting pathway (most often bundle of Kent). On ECG PR interval shorter than normal, ascending part of R wave is deformed (delta-wave), QRS complex wide.

Ventricular tachycardia — sequence of ventricle extrasystoles with rate 100-200 bpm. On EKG QRS complexes wide and deformed, P waves absent. It can be result of increase of automaticity of ectopic pacemakers or due to re-entry mechanism. The most common cause of this type of arrhythmia is myocardial infarction with scarring. Often this one transforms to ventricular fibrillation.

Ventricle fibrillation — the most dangerous arrhythmia which demand emergency treatment. It is characterized by chaotic excitement of myocardial cells with non-effective contraction. It leads to crucial decrease of stroke volume and drop of blood pressure and cardiogenic shock. Ventricle fibrillation is the main cause of death in myocardial infarction, cardiomyopathies, cardial valve disease, congenital heart diseases, hyperkalemia, hypomagnesemia.

Tasks

Describe the changes on ECG:

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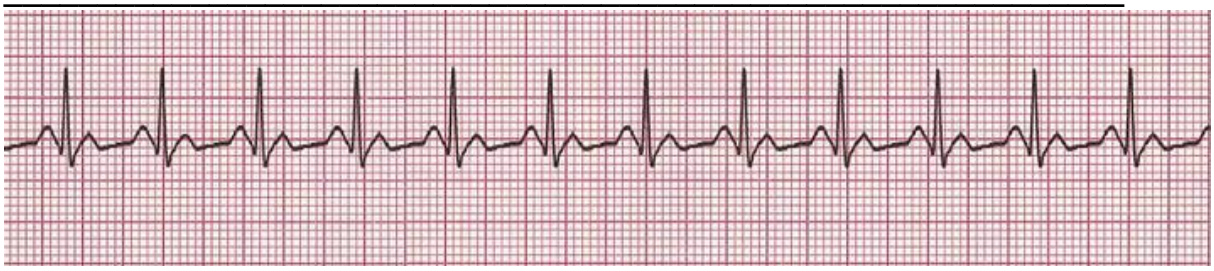

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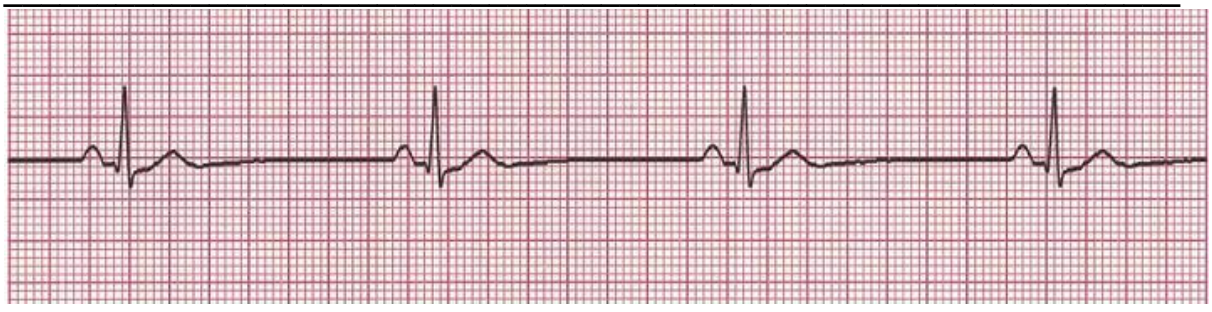

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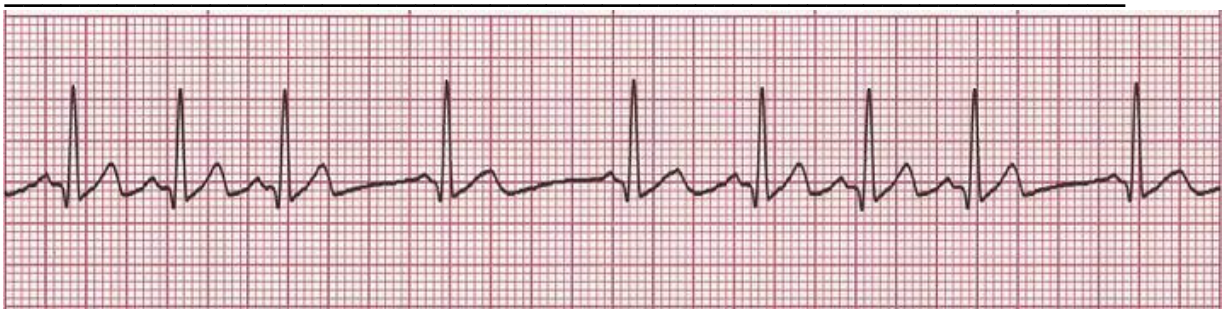

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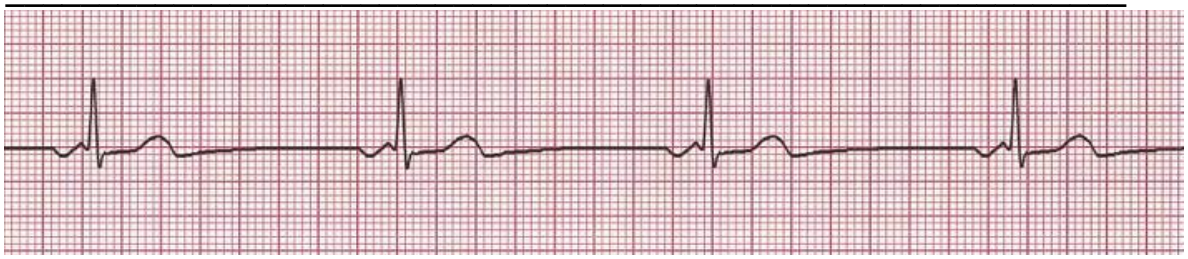

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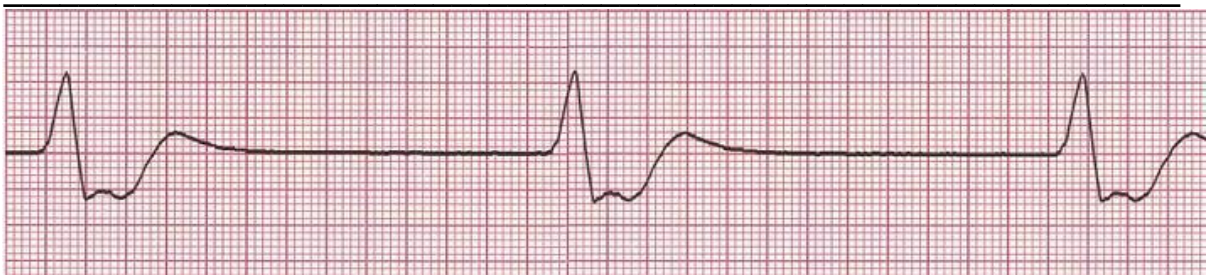

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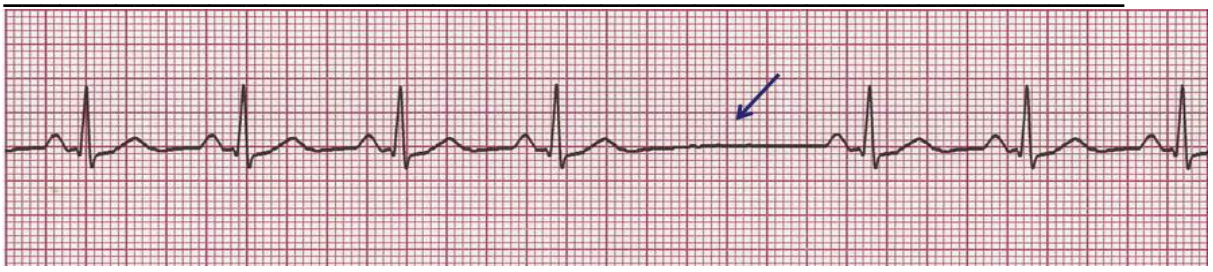

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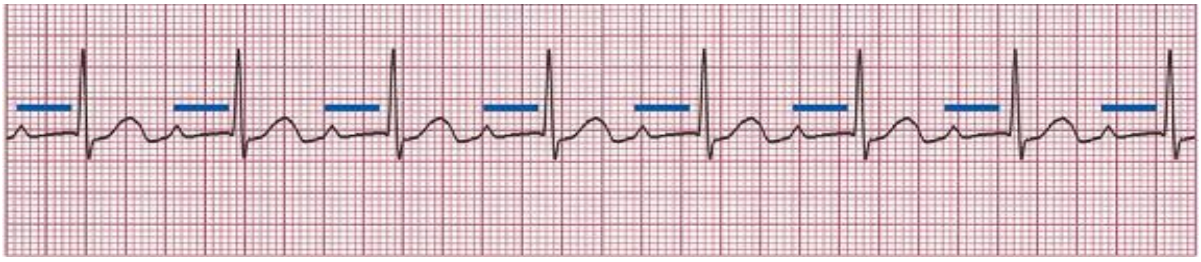

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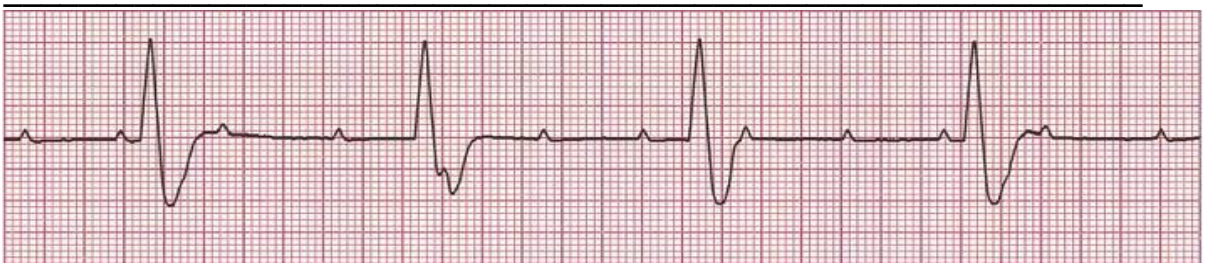

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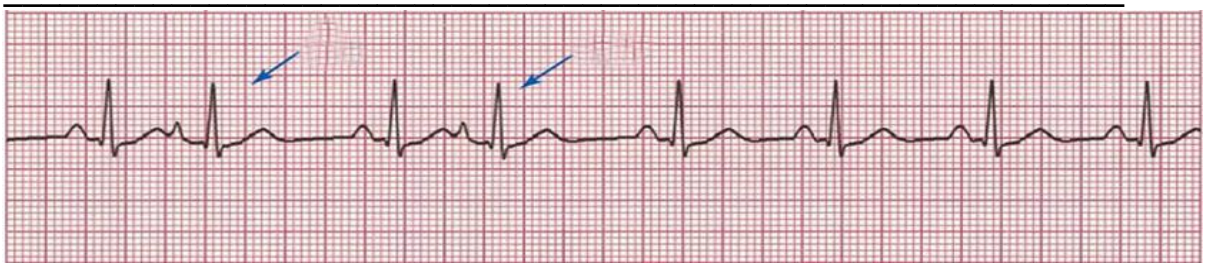

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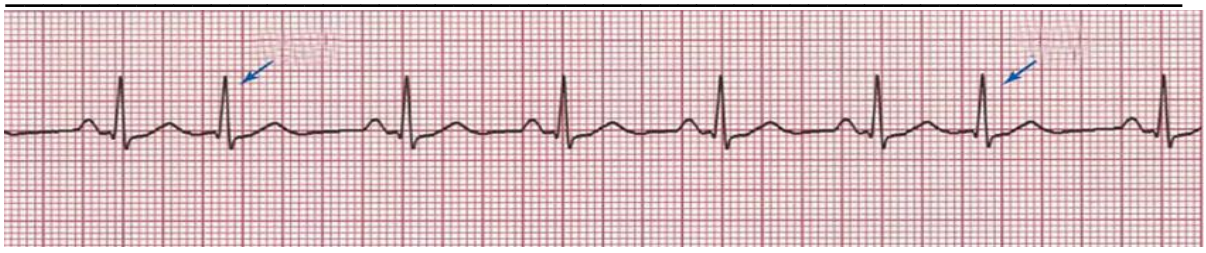

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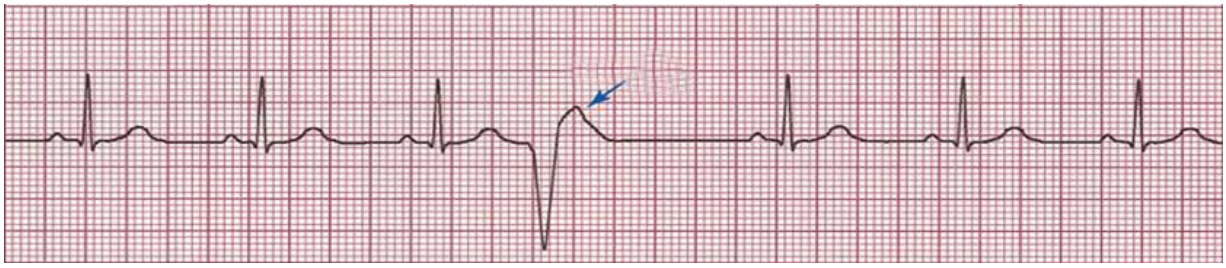

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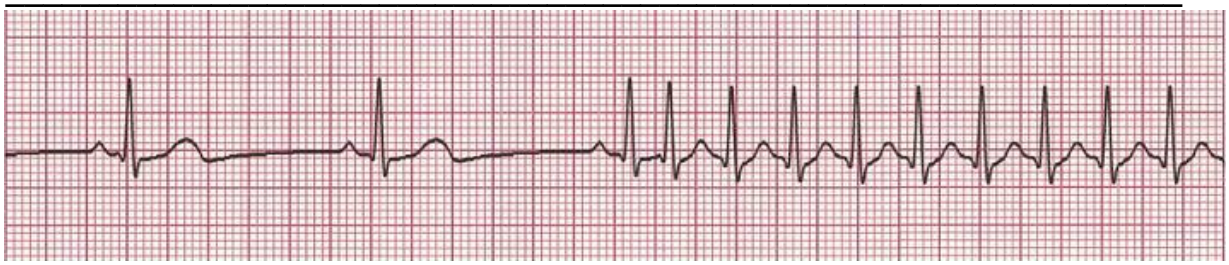

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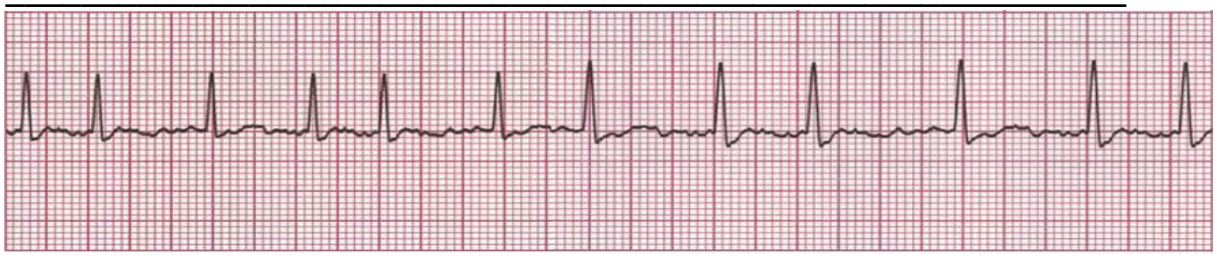

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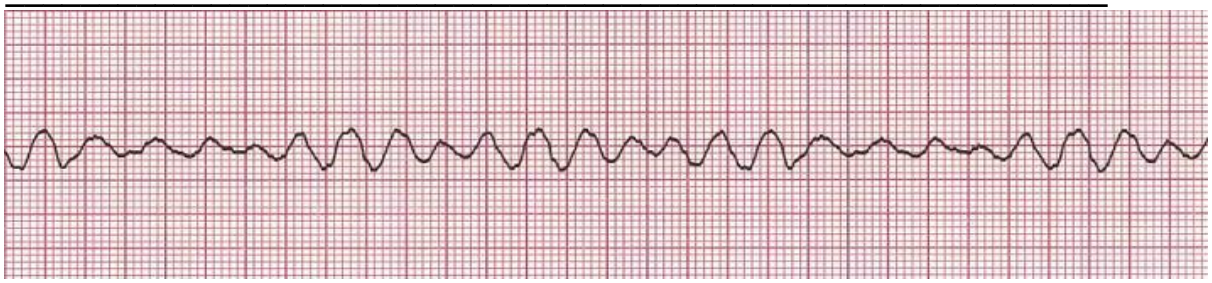

KROK 1 mcqs_A is correct answer

1. Person has stable HR, not more than 40 bpm. What is the pacemaker of the heart rhythm in this person?
A Atrioventricular node
B Sinoatrial node
C His' bundle
D Branches of His' bundle
E Purkinye' fibers
2. ECG of a 44-year-old patient shows signs of hypertrophy of both ventricles and the right atrium. The patient was diagnosed with the tricuspid valve insufficiency. What pathogenetic variant of cardiac dysfunction is usually observed in case of such insufficiency?
A Heart overload by volume
B Heart overload by resistance
C Primary myocardial insufficiency
D Coronary insufficiency
E Cardiac tamponade
3. In a 45-year-old patient on ECG it was revealed: sinus rhythm, the number of auricular complexes exceeds number of ventricular complexes; progressing extension of the P-Q interval from complex to complex; fallout of some ventricular complexes; P waves and QRST complexes are without changes. Name the type of heart rhythm disfunction.
A Atrioventricular block of the II degree
B Synoauricular block
C Atrioventricular blockade of the I degree
D Intraatrial block
E Complete atrioventricular block
4. Processes of repolarisation are disturbed in ventricular myocardium in examined person. It will cause amplitude abnormalities of configuration and duration of the wave:
A T
B Q
C R
D S
E P
5. ECG of a patient shows such alterations: P-wave is normal, P-Q-interval is short, ventricular QRST complex is wide, R-wave is double-peak or two-phase. What form of arrhythmia is it?
A WPW syndrome (Wolff-Parkinson-White)
B Frederick's syndrome (atrial flutter)
C Atrioventricular block
D Ventricular fibrillation
E Ciliary arrhythmia
6. A 49 y.o. woman consulted a doctor about heightened fatigue and dyspnea during physical activity. ECG: heart rate is 50/min, PQ is extended, QRS is unchanged, P wave quanity exceeds quantity of QRS complexes. What type of arrhythmia does the patient have?
A Atrio-ventricular block
B Extrasystole
C Sinus bradycardia
D Ciliary arhythmia
E Sinoatrial block
7. A patient has extrasystole. ECG shows no P wave, QRS complex is deformed, there is a full compensatory pause. What extrasystoles are these?
A Ventricular
B Atrial
C Atrio-ventricular
D Sinus
E –
8. Electrocardiogram of a 45-year-oldman showed absence of P-wave in all the leads. What part of the conducting system is blocked?
A Sino-atrial node

B Atrio-ventricular node
C Common branch of the bundle of His
D Branches of the bundle of His
E Purkinje's fibres

9. A person has steady HR not exceeding40 bpm. What is the pacemaker of the heart rhythm in this person?
A Atrioventricular node
B Sinoatrial node
C His' bundle
D Branches of His' bundle
E Purkinye' fibers

10. After the trauma, the patient's right n.vagus was damaged. Which violation of the cardiac activity is possible in this case?
A Violation of the automatism of Kiss-Fleck node
B Violation of the automatism of a atrioventricular node
C Violation of a conductivity in the right auricle
D Block of a conductivity in the atrioventricular node
E Arrhythmia

11. An isolated cell of human heart automatically generates excitation impulses with frequency 60 times pro minute. What structure does this cell belong to?
A Sino-atrial node
B Atrium
C Ventricle
D Atrioventricular node
E His' bundle

12. Examination of an isolated cardiomyocyte revealed that it didn't generate excitation impulses automatically. This cardiomyocyte was obtained from:
A Ventricles
B Sinoatrial node
C Atrioventricular node
D His' bundle
E Purkinje's fibers

13. A 49 y.o. woman consulted a doctor about heightened fatigue and dyspnea during physical activity. ECG: heart rate is 50/min, PQ is extended, QRS is unchanged, P wave quanity exceeds quantity of QRS complexes. What type of arrhythmia does the patient have?
A Atrioventricular block
B Extrasystole
C Sinus bradycardia
D Ciliary arrhythmia
E Sinoatrial block

14. A patient has extrasystole .ECG shows no P wave, QRS complex is deformed, there is a full compensatory pause. What extrasystoles are these?
A Ventricular
B Atrial
C Atrioventricular
D Sinus
E –

15. ECG of a patient with hyperfunction of thyroid gland showed heart hurry. It is indicated by depression of the following ECG element:
A R − R interval
B P − Q segment
C P − Q interval
D P − T interval
E QRS complex

16.Vagus branches that innervate heart are being stimulated in course of an experiment. As a result of it the excitement conduction from atria to the ventricles was brought to a stop. It is caused by electrophysical changes in the following structures:
A Atrioventricular node
B His' bundle
C Sinoatrial node
D Ventricles
E Atria

17. A cardiac electric stimulator was implanted to a 75 year old man with heart rate of 40 bpm. Thereafter the heart rate rose up to 70 bpm. The electric stimulator has undertaken the function of the following heart part:
A Sinoatrial node
B Atrioventricular node
C His' bundle branches
D His' bundle fibers
E Purkinje's fibers
18. Analysis of the ECG revealed the missing of several PQRST cycles. The remaining waves and complexes are not changed. Specify the type of arrhythmia:
A Sinoatrial block
B Atrial fibrillation
C Atrioventricular block
D Atrial premature beat
E Intra-atrial block
19. ECG of a patient displays an abnormally long R wave (up to 0,18 s). This is caused by a decrease in the conduction velocity of the following heart structures:
A Ventricles
B Atria
C Atrio-ventricular node
D Right ventricle
E Left ventricle
20. Since a patient has had myocardial infarction (type of block)
A Complete atrio-ventricular
B Partial atrio-ventricular
C Sino-atrial
D Intra-atrial
E Intraventricular
21. Since a patient has had myocardial infarction, his atria and ventricles contract independently from each other with a frequency of 60-70 and 3540 per minute. Specify the type of heart block in this case:
A Complete atrio-ventricular
B Partial atrio-ventricular
C Sino-atrial
D Intra-atrial
E Intraventricular
22. A patient complains of palpitation after stress. The pulse is 104 bpm, P-Q=0,12 seconds, there are no changes of QRS complex. What type of arrhythmia does the patient have?
A Sinus tachycardia
B Sinus bradycardia
C Sinus arrhythmia
D Ciliary arrhythmia
E Extrasystole
23. ECG of a patient shows such alterations: P-wave is normal, P-Q-interval is short, ventricular QRST complex is wide, R-wave is double-peak or two-phas e. What form of arrhythmia is it?
A WPW syndrome (Wolff-Parkinson-White)
B Fredericks syndrome (atrial flutter)
C Ventricular fibrillation
D Atrioventricular block
E Ciliary arrhythmia
24. A 45 year old patient was admitted to the cardiological department. ECG data: negative P wave overlaps QRS complex, diastolic interval is prolonged after extrasystole. What type of extrasystole is it?
A Atrioventricular
B Sinus
C Atrial
D Ventricular
E Bundle-branch
25. A patient has delayed conduction of excitement through the atrioventricular node. What changes of ECG will be observed?
A Prolongation of P − Q interval
B Prolongation of Q − S interval
C Negative T wave
D S − T-segment displacement

E Prolongation of Q − T interval

26. A patient complains of palpitations after stress. Pulse is104/min. ,P-Q=0,12 seconds, there are no changes in QRS complex. What type of arrhythmia does the patient have?

A. Sinus tachycardia
B. Sinus bradycardia
C. Sinus arrhythmia
D. Ciliary arrhythmia
E. Extrasystole

27. Electrocardiogramanalysis demonstrates that cardiac cycle of a human equals 1 second. It means that heart rate per minute equals:

A. 60
B. 50
C. 70
D. 80
E. 100

28. A 15-year-old teenager complains of lack of air, general weakness, palpitations. Heart rate is 130/min., BP is 100/60 mm Hg. ECG: QRS complex has normal shape and duration. The number of P waves and ventricular complexes is equal, T wave merges with P wave. What type of cardiac arrhythmia is observed in the teenager?

A. Sinus tachycardia
B. Sinus extrasystole
C. Atrial fibrillation
D. Atrial thrill
E. Paroxysmal atrial tachycardia

29. A 17-year-old girl suffers from periodical palpitations that last several minutes. Her heart rate is 200/min., rhythmic. What heart rhythm disorder developed in this patient?

A. Paroxysmal tachycardia
B. Sinus tachycardia
C. Sinus bradycardia
D. Extrasystole
E. Atrioventricular block

30. A 48-year-old man is unconscious. He has a history of several syncopal episodes with convulsions. ECG shows deformed QRS complexes unconnected with P waves, atrial contractions are approximately 70/min., ventricular contractions - 25-30/min. Name the type of arrhythmia in this case:

A. Complete atrioventricular block
B. First-degree atrioventricular block
C. Second-degree atrioventricular block
D. Intraatrial block
E. Intraventricular block

Tests For Self-Control (give correct answer)

1. Which of the following would represent left
or right atrial enlargement on a surface electrocardiogram?
A. Wide or tall P wave
B. Wide or tall T wave
C. A prominent U wave
D. An elevated J point
E. A large QRS voltage
2. Which of the following is often noted during hypokalemia?
A. Prominent P wave
B. Prominent QRS complex
C. Long Q–T interval
D. Prominent U wave
E. J point elevation
3. Which of the following associations is correct?
A. Hypokalemia: shortened Q–T interval
B. Hypercalcemia: long Q–T interval
C. Hypercalcemia: flattened T waves
D. Hypocalcemia: U waves
E. Hyperkalemia: peaked T waves

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 348-350 pp.
2. Victor N. Jelski, Svetlana V. Kolesnikova. Handbook Of Pathophysiology Part 2: Pathophysiology of organs and systems. - Donetsk, Ukraine. – 2011. – 51-58 pp.
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Additional

4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. —3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. – 2000.