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# **CARDIOLOGY**

**TRAINING MANUAL  
TO INTEGRATED PRACTICAL ORIENTED  
STATE EXAMINATION FROM “INTERNAL DISEASE”**

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The manual outlines the basics of modern cardiology knowledge that are subject to control during the development of a practically-oriented state examination on internal medicine.For students of higher medical educational institutionsIII – IVlevels of accreditation.

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## INTRODUCTION

Practically oriented state graduation exam conducted after obtaining the results of the license integrated examination "Step2". Students who have fully met all the requirements of the curriculum and the educational-professional program in the specialty, regardless of the outcome of the licensed integrated examination "Step2", are admitted to the compilation of a practical state-of-the-art examination.

The manual presents educational and methodological materials for the preparation of graduates of the medical faculty for a practical - oriented state examination on internal diseases (cardiology). Information on requirements to the level of theoretical knowledge, practical skills and abilities of graduates concerning the clinical, laboratory and instrumental examination of the patient, differential diagnosis, formulation of the diagnosis, treatment, provision of urgent help to patients with cardiovascular diseases is systematized. The stages of the practical-oriented state examination, protocols and evaluation criteria are clearly presented. Methods of measuring blood pressure, recording and analyzing electrocardiograms, estimating the results of echocardiography, bicycle ergometry are described in detail. The criteria for diagnosing the main cardiac arrhythmias and conduction disorders, determination, classification, diagnosis of arterial hypertension, coronary heart disease, myocardial infarction, heart failure, dyslipoproteinemia, cardiac reanimation methods are given. Sufficient number of samples of interpretation of laboratory and instrumental studies, situational problems with decision benchmarks.

## **LIST OF CARDIOLOGY THEORETICAL ISSUES, KNOWLEDGE OF WHICH SHOULD EVALUATE AT THE TIME OF PRACTICAL AND ORIENTIVE PUBLIC EXAM[1]**

**List 1 (syndromes and symptoms).**1.Arterial hypertension(essential hypertension, secondary arterial hypertension, renal - renovascular, renoparenhimal, endocrine - disease and syndrome Cushing, pheochromocytoma, primary hyperaldosteronism, diffuse toxic goiter, coarctation of the aorta, isolated systolic hypertension, hypertension in pregnancy).2.Andblood hypotension and fainting(vaso-depressant hypotension/fainting, postural orthostatic, iatrogenic hypotension, fainting in cardiovascular diseases: valvular heart disease, acute coronary syndrome, hypertrophic cardiomyopathy, aortic dissection, heart rhythm disturbance and conduction disorders: sinus node dysfunction, violations of atrioventricular conduction, supraventricular and ventricular tachycardia; thromboembolism of the pulmonary artery, nerve and endocrine diseases, metabolic disorders and hysterical neurosis, abdominal and rash typhus, menongococcal infection, hemorrhagic fever).3.Ascites(cirrhosis and liver tumors, right ventricular heart failure, including constrictive pericarditis, hepatic vein thrombosis, portal vein thrombosis or its branches, thrombosis, stenosis, obliteration of the lower vena cava at or above the liver veins, etc.).4. Chest pain (acute coronary syndrome, angina pectoris, stenosis of the aortic mouth, hypertrophic cardiomyopathy, mitral valve prolapse, coronary, aortic, myocarditis, acute pericarditis, aortic dissection, pleurisy, pulmonary artery thromboembolism, pneumothorax, mediastinal tumor, gastroesophageal reflux disease, cardiospasm, esophageal spasm, apple esophagus keel, peptic ulcer and other ulcers of the stomach and duodenum, pancreatitis, osteochondrosis of the thoracic spine, scapular lichen, myositis, chest pain, intercostal spacefor neuralgia, neurocirculatory dystonia syndrome, panic attacks, pulmonary plague, enterovirus disease).5.Shortness of breath(with heart failure with preserved and reduced systolic function of the left ventricle; respiratory failure due to bronchodilation and diseases of the lungs and pleura, including pneumonia, tuberculosis and pneumothorax; pulmonary vascular pathology, in particular pulmonary thromboembolism and chest or respiratory diseases muscles, hyperventilation syndrome with neurosis and neurocirculatory dystonia, defeat of the respiratory center for organic brain diseases,

anemia, bottlechange). 6. Cardiomegaly (acquired heart defects, in particular mitral valve failure, stenosis and aortic valve insufficiency, combined mitral and aortic heart defects, myocarditis and dilated cardiomyopathy, ischemic heart disease, exudative pericarditis). 7. Edema syndrome (venous edema: chronic venous insufficiency, venous outflow disturbance, deep vein thrombophlebitis, lymphatic edema: inflammatory, obstructive, fatty, orthostatic and idiopathic, with bone marrow disorders: arthritis, tendonitis, nephrotic syndrome, cardiovascular disease systems with the development of heart failure, liver disease, in particular, liver cirrhosis and other hypoproteinemic conditions: exudative enteropathy, malabsorption syndrome, alimentary and cachet edema, edema, and endocrine diseases: hypothyroidism). 8. Violation of the cardiac rhythm (extrasystole, fibrillation and atrial flutter, paroxysmal tachycardia). 9. Cyanosis diffuse and local (capture of the lungs and heart, including congenital heart defects in the condition of the Eisenmenger syndrome and acquired heart defects - mitral stenosis, tricuspid valve deficiency, cardiac and respiratory failure, and in the formation of pathological hemoglobin, true cereals, diphtheria, generalized forms of infectious diseases). 10. Heart murmurs (congenital heart defects, defect ventricular septal defect atrial septum, ductus arteriosus, coarctation of the aorta, acquired heart disease, mitral stenosis, mitral valve (organic and relative), mitral valve stenosis mouth aortic insufficiency aortic valve, hypertrophic cardiomyopathy, trilateral valve failure (organic and relative), innocent systolic noise in young people). 11. Shock (cardiogenic).

**List 2 (illness). Diseases of the circulatory system**

1. Essential arterial hypertension (hypertonic disease).
2. Secondary (symptomatic) arterial hypertension:
  - renal (renovascular, renoparenchymatous);
  - endocrine (syndrome and illness of Itsenko-Cushing's disease, pheochromocytoma, primary hyperaldosteronism, diffuse toxic goiter);
  - Coarctation of the aorta;
  - isolated systolic arterial hypertension;
  - arterial hypertension during pregnancy;
3. Neurocirculatory dystonia.
4. Atherosclerosis.
5. Chronic forms of coronary heart disease.
6. Acute myocardial infarction. Unstable angina.
7. Pericarditis.
8. Pulmonary heart.
9. Acquired heart disease: mitral, aortic and tricuspid valve, combined mitral and aortic defects.
10. Congenital heart defects: defecation of the atrial space, interventricular septum, open arterial duct, coarctation of the aorta.
11. Infectious endocarditis.
12. Myocarditis and

cardiomyopathy.13.Pulmonary artery thromboembolism.14.Violation of the heart rate.15.Violation of conduction of the heart.16.Heart failure.

**List 3(urgent states).**1.Stopping blood circulation and breathing 2.Acute coronary syndrome. 3. Acute heart failure 4. Shock 5.Acute respiratory failure. 7. Cardiac tamponade. 8. Pulmonary artery thromboembolism.9. Hypertensive crisis.10.Paroxysmal violations of the heart rhythm and cardiac conduction disturbances (paroxysmal tachycardia and atrial fibrillation/atrial flutter, high-level atrioventricular block, Morgagny-Adam-Stokes syndrome) 11.Komas.14. Fainting.

**List 4 (laboratory and instrumental research methods).**1.Analysis of the pleural fluid. 2. Analysis of ascitic fluid. 3. Analysis of synovial fluid. 4. Analysis of urine for diastase. 5. Urine analysis by Nechyporenko. 6. Analysis of urine by Zimnitsky. 7. Acute blood parameters, total protein of blood and its fractions.8. General blood test.9.General urine analysis. 10. Glucose tolerance test, glycemic and glucosuric profile, C-peptide, HbA1c. 11. Biochemical indices of blood serum iron exchange.12. Transaminases of blood, total bilirubin and its fractions. 13. Coagulogram. 14. Biochemical markers of myocardial necrosis, D-dimer. 15. Lipid blood spectrum. 16. Creatinine and urea of blood, velocity of glomerular filtration. 17. Uric acid of blood. 18. Electrolytes of blood. 19. General immunological blood profile.21 Serological response in autoimmune diseases. 23. Microbiological study biological fluids and excretions. 24.Enzymatic, immunochemical, molecular biological studies of blood 31. Hormonal examination adrenal glands, pituitary gland, thyroid gland. 32. Studies of lung function study of electrocardiographic. 34. Echocardiography 35. Samples with dosed physical activity. 36. Sonography, computer and magnetic resonance tomography of the thyroid gland, adrenal glands. 37. X-ray contrast angiography. 38. Examination of the abdominal cavity. 39. Radiation examination of the chest.

**List 5 (medical manipulations).**1.Measure arterial pressure. 2. Register electrocardiogram in 12 leads. 3. Perform artificial ventilation of the lungs and conduct indirect heart massage.4. Conduct calculation of the body mass index.

**Know the clinical pharmacology of the main groups of medicines.**1. $\alpha$ -and $\beta$ -adrenostimulants. 2. Antianginal. 3. Antiarrhythmic. 4. Antibacterial. 6. Antihypertensive. 7. Anticoagulants. 9. Expectorant. 10.Hemostatic. 11.

Glucocorticoids and cytostatic immunosuppressants. 12. Diuretics. 13. Proton pump inhibitors. 14. H<sub>2</sub>-histamine blockers. 15. Nonsteroidal anti-inflammatory drugs. 16. Oral hypoglycemic agents and insulin preparations. 17. Antiviral drugs. 18. Anti-acid drugs. 19. Solutions for detoxification therapy. 20. Solutions for rehydration therapy. 21. Cholinolytics, anesthetics, prokinetics.

**To evaluate the clinical data of the patient on the diagnostic, treatment and preventive scales.**

1. Scale of CHA<sub>2</sub>DS<sub>2</sub>-VASc Stroke risk assessment in patients with atrial fibrillation.
2. Scale of risk of bleeding in patients with atrial fibrillation HAS-BLED.
3. Scale of estimation of the total risk of complications in arterial hypertension.
4. GRACE scale of mortality risk assessment in patients with acute coronary syndrome without elevation of the ST segment at the hospital stage and after 6 months.
5. Geneva scale for calculating the clinical probability of pulmonary thromboembolism.
6. Wells scale for predicting the clinical risk of pulmonary artery thromboembolism.
7. Scale of estimation of the degree of prognostic risk in thromboembolism of the pulmonary artery PESI.

## **LIST OF PRACTICAL WORKS AND OBJECTIVES FROM CARDIOLOGY FOR PUBLIC PRACTICAL ORIENTED EXAM [1]**

List of Typical Tasks and Skills Examination Examples:

### **Work with the sick**

- To collect complaints, anamnesis of illness, anamnesis of life;
- To collect information about the general condition of the patient (consciousness, constitution, fatness) and to evaluate the appearance (examination of the skin, subcutaneous fat layer, palpation of the lymph nodes, thyroid and mammary glands), to examine the condition of the bone and muscle system, joints;
- **To** examine the state of the respiratory organs (examination of the chest, palpation of the chest, percussion and auscultation of the lungs);
- **To** examine the state of the system of blood circulation organs (examination and palpation of the area of the heart and vessels, percussion of the heart and auscultation of the heart and blood vessels);
- Inspect the state of the digestive system (examination, percussion, superficial and deep palpation);



-Examining the state of the genitourinary system (examination of the lumbar region, palpation of the kidneys).

- Highlight a Leading Clinical Symptom or Syndrome (List 1)
- Provide a probable (prior) or syndromic diagnosis of the disease (List 2).
- Assign and substantiate a laboratory and / or instrumental examination of a patient (List 2).
- Perform differential diagnosis for major symptoms and syndromes (List 1).
- Interpret the results of laboratory and instrumental studies (List 4)
- Make a clinical diagnosis (List 2).
- Identify the principles and nature of treatment (conservative, operative) disease (List 2).
- Determine the desired mode and diet of the patient (List 2).
- Determine the tactics of secondary prevention of patients who are subject to dispensary supervision.
- Conduct patient medical records.
- Diagnosis of Emergency Conditions (List 3)
- Definition of emergency medical care tactics (List 3)
- Emergency care (List 3)
- Performing medical manipulations (List 5)

## **STRUCTURE OF PRACTICAL ORIENTED PUBLIC EXAM AND THE CRITERIA OF ASSESSMENT OF KNOWLEDGE AND LEARNING LEVELS[1]**

**Practically oriented comprehensive state examination** consists of two parts:

- 1) the **first** part of the exam: direct work with patients.
- 2) the **second** part of the exam: Demonstration of basic skills and practical skills in accordance with the Educational Qualification Specification (EQS) of a specialist in specialty 7.12010001 "Therapeutic Case" (therapeutic profile) using phantoms, dummies, teaching materials, solving situational problems.

The result of a practically oriented state examination is estimated at two scales: in the points of a multi-point scale and in estimates of the traditional 4- ball scales (5 - "excellent", 4 - "good", 3 - "satisfactorily", 2 - "unsatisfactory").

All results are determined based on the primary scores recorded in individual protocols for conducting and evaluating the exam, approved by the order of the Ministry of Health of Ukraine of 31.01.2005 No. 53 on the approval of "Regulations on the organization and procedure for conducting state attestation of students who study in higher educational institutions and I – IV levels of accreditation in the field of training" Medicine " and the Instruction of the Ministry of Health of Ukraine dated April 15, 2014 "On the evaluation of educational activities of students in the context of the implementation of the European Credit Transfer System for the organization of the educational process"[2, 3].

Primary points entered into protocols are defined as follows:

The implementation of typical tasks and skills that are checked during the **first** part of the exam is evaluated with scores: "1", "0.5" and "0" (completed, not fully executed, not completed). Points are added to the individual protocols for conducting and evaluating the first part of the exam.

Execution of situational tasks, basic skills and skills, which are checked during the **second** part of the exam, is evaluated with scores of "1" and "0" (completed, not fulfilled). Points are added to the individual protocols for conducting and evaluating the second part of the exam.

The points for the first (second) part of the exam are determined as the arithmetic mean of all the points recorded in the individual protocols for conducting and evaluating the relevant part of the exam. Primary points for the first (second) part of the exam lie in the range from 0 to 1 and rounded to 2 (two) marks after the comma.

The resultant score for a practically oriented state exam is defined as the arithmetic mean of the first (*CA1*) and second (*CA2*) parts of the exam multiplied by the factor of 200, rounded to the integer value. **Such a score is an assessment of the student on a 200-point scale.**

Formula for recalculation:  $(CA1+CA2)/2*200$

Points for a Practical-oriented State Exam on a 200-point scale are converted to a four-point scale using the following criteria. Grades in multi-point and four-point scale are recorded in the exam group.

<b>Score on a multi-point (200) scale (when applying conversion factor "200")</b>	<b>Score on a four-point scale</b>
<b>From 180 to 200 points</b>	<b>5, "excellent"</b>
<b>From 140 to 179 points</b>	<b>4, "good"</b>
<b>From 101 to 139 points</b>	<b>3, "satisfactory"</b>
<b>100 points or less</b>	<b>2, "unsatisfactory"</b>

### **Criteria for assessing the score on a traditional 4-point scale[1]**

#### **Converting to the ECTS scale[3]**

During the ranking of students, the points from the state final exams on the 200-point scale are converted to the ECTS scale. Scores of the ECTS scale on a four-point scale are not converted and vice versa.

A, B, C, D, and E ratings are ranked by dean's office for all students of the same year who study in one specialty and successfully passed a practical exam. Students who scored 100 points or less (score "2") are not included in the list of ranked students. The ranking for determining the assessment of the ECTS is carried out by the dean's office on the number of points scored from the practical exam (according to the chosen form of conducting a practical exam can be one ranking or several). The results are entered into the relevant information and protocols.

<b>Estimation of ECTS</b>	<b>The statistical indicator</b>
<b>"A"</b>	<b>The best 10% of students</b>
<b>"B"</b>	<b>The next 25% of students</b>
<b>"C"</b>	<b>The next 30% of students</b>
<b>«D»</b>	<b>The next 25% of students</b>
<b>"E"</b>	<b>The last 10% of students</b>

### **PROCEDURE FOR PRACTICAL ORIENTED STATE EXAM [3]**

**The first part of the practical-oriented comprehensive state examination is working with the patient**

Student gets in SEC referral stating therapy department, ward, name and surname of the patient, is sent to the appropriate department, which in the presence of examiners and members of the SEC conducts examination of the patient and is responsible bedside examiners and members of the SEC.

It assesses the skills and abilities of each graduate to conduct a survey and objective examination of patients, establish and substantiate the previous clinical diagnosis, and determine the treatment tactic in the case of diseases in accordance with the EQCh specialty 7.12010001 "Medical Case". Examiners and members of the SEC evaluate the skills and abilities of the graduate and add ratings to the individual protocols of conduct (protocol number 1) and evaluation of the first part of the exam.

**Work with the patient is carried out according to the protocol No. 1**

No. of p	Typical tasks of the activity and the skills being tested are estimated by the points "1", "0,5", "0" except for the numbers 10, 11	Points
1	Collection of complaints, anamnesis of disease and life	
2	Review of the patient, collecting information about the general condition of the patient and his assessment.	
3	Physical examination of the cardiovascular system.	
4	Blood pressure test	
5	Physical examination of the respiratory organs.	
6	Physical examination of the abdominal cavity (digestive system and urogenital system)	
7	Physical examination of the bone and muscular system.	
8	Isolation of the leading syndrome, the most probable or syndromic diagnosis.	
9	Estimation of the appointment of laboratory and instrumental methods for studying a patient with whom the student works.	
10	Intra-syndrome differential diagnosis (estimated by the points "1" and "0").	
11	Preliminary clinical diagnosis (estimated at "1" and "0").	
12	Preparation of the survey plan.	
13	Defining the principles of treatment and tactics of patient management, including necessary mode and rest, diet.	
14	Determination of prognosis and prevention measures in this patient.	
15	A typical task (skill) audited by choice in HSE	

	Overall result	
	Average points	
	Assessment of ECTS	
	Traditional evaluation	

Optional higher education institution, students must complete two practical skills: (calculation of body mass index and evaluate it, to measure blood pressure).

**The second part of a practical state-of-the-art exam** is to assess the level of development of abilities and skills that does not involve direct work with patients. The exam is conducted in specially equipped training rooms and involves work with dummies, phantoms, educational materials and solving situational tasks, Evaluation of laboratory results and interpretations in instrumental studies.

The protocol of the second part of the practical-oriented state examination includes:

- Assess students' skills to diagnose emergency conditions and provide emergency medical care (situational tasks 1 and 2) that the student receives from the examiners.
- Evaluation of the results of laboratory and instrumental studies of therapeutic diseases (situational tasks 3 and 4).
- Students' ability to perform medical manipulations. Control 5 medical manipulations from the List of Skills and Practical Skills, according to the curriculum.

This part is assessed by examiners and members of the DEC. The results are recorded in the individual protocols (protocol # 2) for conducting and evaluating the second part of the exam.

**The evaluation of the results of the laboratory and the interpretation of the results of the instrumental research methods are carried out according to the protocol №2**

In the internal diseases			
No. of n	Situational tasks, basic skills and skills that are rated by "1" and "0"	Score	Signature
1	Diagnosis, analysis and evaluation based on ECG		

2	Diagnosis based on the results of R- grams		
3	Diagnosis based on the general results of sputum research		
4	Diagnosing, analyzing and evaluating the results of laboratory blood tests		
5	Diagnosing, analyzing and evaluating the results of laboratory urine tests		
6	Diagnosis based on the results of laboratory examination of duodenal sensing		
7	Situational task on diagnosing and treatment of tuberculosis		
8	Practical skills in tuberculin diagnostics		
9	Practical skills in preventing AIDS in healthcare workers		
10	Situational problem in diagnosing and preventing internal diseases №		
	Overall result		
	Average results		
	Assessment of ECTS		
	Traditional evaluation		

### **METHOD OF VIEWING OF PATIENTS[4]**

The main clinical methods of research are: questioning, review, palpation, percussion and auscultation[4].

Query consists of four sections:

1. Passport data
2. Complaints at the time of receipt
3. Anamnesis morbi
4. Anamnesis vitae

Passport data includes information about the age of the patient, his occupation, place of work and residence.

After obtaining passport data, find out the main complaints of the patient, carry out their details.

This section shows the complaints that occurred during a hospital admission to the clinic. It is necessary to make the details of them (character, degree of expressiveness, causes of them, duration, etc.), if there is an anastrophic course of the disease, it should detail the beginning of the onset of the attack, its course, duration, which factors or drugs will facilitate or stop attack.

#### **Scheme of purposeful patient survey on systems[4]**

##### **Respiratory system**

**Cough:** dry or spit; when it appears: in the morning, in the evening, at night; permanent or periodic; character of the cough: loud, strong, unconscious, barking; Conditions of appearance of a cough: in connection with a certain position of the body (what exactly), after eating, etc.

**Sputum:** daily quantity; how to crimp: easily, with the strength, in which position is better; character and color of sputum; the smell of sputum; consistence; number of layers and their characteristics.

**Haemorrhage:** intensity - streaks or pure blood; blood color: red, dark; frequency.

**Chest pain:** the nature of the pain: dull, acute, absent, prickly, with breathing; which relieves pain; during the pressure on the thoracic cavity, during the tilt of the trunk in different directions.

**Choking:** constant, at rest, during exercise, walk, depending on the position in bed, during a conversation; inspiratory, expiratory, mixed.

##### **Cardiovascular system[4]**

**Pain in the region of the heart:** permanent or paroxysmal localization (in the sternum, in the region of the heart, in the region of the apical pulse, etc.); irradiation; character: achiers, prickly, compression, stupid; What is accompanied by - a feeling of anxiety and fear, weakness, cold sweat, dizziness, etc.; intensity; duration; frequency of pain attacks; Causes and circumstances of occurrence of pain (during exercise, excitement, during sleep, etc.); handling and out of the patient during a pain attack; which has a therapeutic effect.

**A feeling of heartbeat.**

**Heartbeat:** heartbeat: permanent, attacks (intensity, duration, frequency); Conditions of appearance: during physical activity, at rest, during body change, during

excitement, etc .; what is accompanied (suffocation, pain in the heart, etc.), from which they pass. edema: on legs and other places, the time of their appearance (in the morning, at evening). The sensation of ripple: in which parts of the body, what are caused, from what passes. signs of spasm of peripheral vessels: intermittent lulling, feeling of a "dead finger"; what are they called from, what are they going to do?

#### **Digestive system[4]**

**Appetite:** good, low, elevated, perverted, aversion to food (which). Saturation: normal, fast, constant feeling of hunger. thirst: how many drinks a day, dry mouth. taste in the mouth: sour, bitter, metallic, sweet, dull or loss of taste. smell from the mouth: repellent (rotten), sweet, ammonia, sour, fecal, the smell of rotten apples, etc.. swallowing and passing food: painful, difficult, what food does not pass? salivation. dislocation: what, time of appearance, expressiveness, volume. Heartburn: A connection with food that facilitates heartburn? Nausea: Dependence on food and its nature.

**Vomiting:** on an empty stomach, after a meal (now or after a certain period of time); what feelings precede vomiting, whether it relieves the patient's state of health; the nature of vomiting: eaten food, bile, color of coffee density, with fresh blood clumps, etc .; their smell (rotten, sour, etc.), odorless.

**Abdominal pain:** localization and irradiation of pain; when and in what circumstances it occurs, before eating, after eating (at what time), night pain. Does pain decrease immediately after eating? Other factors that relieve pain (vomiting, medication, heat, etc.); Depending on the nature of the food (coarse, fat, acute, etc.) or its amount; character of the white: sharp, dull, aching, in the form of an attack or gradually increasing; duration of pain; what is accompanied; does not appear jaundice, dark urine, discolored feces after an attack of pain. breakdown and abdominal difficulty. abdominal distension, gas outflow, abdominal rumbling.

**Vaginal discharge:** regularly, irregularly, independently or after an enema, laxatives. Fascicles, several days. breaks: what is associated with how many times a day; are there tenesmus; the nature of feces (liquid watery, porcine, type of rice broth, etc.); color and smell of feces; admixture: blood, manure, residues of undigested food, worms; Blood allocation (before, during, or at the end of the bowel movement). Heartburn, itching, pain in the anus. Rejection of the rectum.



## Urinary system[4]

**Pain in the lumbar region:** the nature (dull, acute, anaphylactic), irradiation, duration, from which appear or amplify, than accompanied, that relieves pain.**Urination:** free, with force, normal jet, thin, intermittent, hanging down (question only men).rigor, heartburn, pain during urination;frequency of urination, especially at night;amount of urine per day.urine color: normal, dark, color of "meat fog", beer, etc.presence of blood during urination: at the beginning, in all portions, at the end.The presence of uncontrolled urination.

## Supporting device[4]

**Pain**in the limbs, joints.The nature of the pain, volatility, the connection with the change of weather, with the load, with excitement;the appearance of pains at rest, at night.

- **Swollen**joints, their redness (which ones).
- **Difficulty**during movements (in which the joints), stiffness in the morning, its duration.
- **Pain**and difficulty during movements in the spine (in which sections), irradiation of pains.

## Endocrine system[4]

- Violations of the Constitution and the Constitution.Violation of weight (obesity, weight loss).Changes in the skin (excessive sweating or dryness, rude it, the appearance of red leather strings, changes in color).Violation of primary and secondary sexual characteristics;dysmenorrhea and infertility in women;impotence in men. Hair stroke (excessive development, appearance on non-intrathoracic areas, hair loss).

## Nervous system, sensory organs[4]

- Night rest (deep sleep, superficial, with frequent waking, persistent, without dreams, with dreams, colored dreams, etc.) Condition after sleep (cheerfulness, improvement of well-being, wrinkling, weakness, "fragmentation") Memory (excellent, good, normal, low, very bad). The mood is in the morning, in the first, in the second half of the day (excellent, good, satisfactory, bad, very bad). Attention (excellent, good, satisfactory, bad, very bad) Headache (localization, nature, what is its origin, periodicity, duration, accompanying symptoms: ear tiredness, dizziness) Pain disorders, trembling of the limbs, seizures, violations skin sensitivity. Fever. Increase of temperature and its oscillations during the day (character of the curve). The rate of temperature increase and the duration of the fever. What lowers the temperature?Whether chills precede an increase in temperature, whether there is sweating after its decrease, the intensity of sweating, night sweats.

### **And the page of the current disease (anamnesis morbi)[4]**

This section shows the beginning of the disease and its dynamics until the time of admission to the clinic (hospital).

In the process of questioning you need to get answers to the following questions:

When, where and during which circumstances it was ill. As the disease began (acutely, gradually). What are the causes of the disease (in the opinion of the patient). The possible influence on the occurrence and course of the disease of the conditions of the external environment (professional, domestic, climatic and weather factors), physical or psycho-emotional strain, intoxication, errors in the diet is established. Infectious diseases (adenovirus infection, influenza, angina). What are the first signs of the disease. When and what is the first medical aid provided, its effectiveness. What changes in the patient's condition occurred from the moment of the onset of the disease to this time (the dynamics of patient complaints). In the case of a chronic course of the disease in a chronological sequence to reveal the relapses of the disease and their manifestations, as well as the periods of remission, their duration. What studies were conducted on the patient, their results. If available, an outpatient card, extracts from the history of the disease, radiographs, spirograms, ECG and other documents are used. What treatment was used at different stages of the disease, its effectiveness. What became the reason for this deterioration, describe in detail the main symptoms of its manifestation. How did the patient's condition change during the stay in the hospital until the patient's discharge (specifically, on the severity and characterization of the symptoms).

### **And the life story of the patient (anamnesis vitae)[4]**

Brief biographical data (place of birth, which on account of the child, as grew and developed, study, specialty, marriage, pregnancy, childbirth). Labor history (beginning of work, occupation, change, working conditions, industrial hazards, use of leave, service in the ranks of the armed forces, participation in the war). Housing and living conditions in different periods of the patient's life, composition of the family. Eating (mode, regularity, nature of food - its variety, caloric content). Carrying out diseases, injuries, operations, contusions, injuries, tuberculosis, sexually transmitted diseases: indicate the severity and duration of the disease, complications, medical treatment; parenteral interventions (subcutaneous, intramuscular, intravenous,

blood transfusions, treatment and tooth extraction), contact with patients with viral hepatitis B and C. Epidemiological history, contact with infectious patients. Harmful habits: smoking, from which age, smoking, number per day; alcohol from which age, in what quantity, how often; Other bad habits (drugs, strong coffee or tea). Family history and heredity (parents, brothers, sisters, children - their health, causes of death), hereditary diseases (congenital anomalies of development, mental illness, syphilis, illness of exchange, etc.), burden of anamnesis (alcoholism, malignant neoplasms, endocrine diseases and mental illness). Allergic history: the presence of allergic diseases in the patient, his relatives and children; reactions to blood transfusion, the administration of serums, vaccines and the administration of medicines (which and when); reactions to various foods, beverages (food allergy), cosmetics, odors, and pollen from various plants. Clear the reaction to contact with a variety of animals, clothes, wool, household dust, bed linen. Influence on the course of diseases, working conditions, professional factors, various factors (cooling, overheating, insolation). Meteorability and seasonality. To determine the impact on the course of the disease climatic-weather conditions, magnetic perturbations. Describe seasonal exacerbations, their cause (infection, atypia, weather, etc.). Disability: the number of days of incapacity during the year, the presence of a disability group. their cause (infection, atypia, weather, etc.). Disability: the number of days of incapacity during the year, the presence of a disability group. their cause (infection, atypia, weather, etc.). Disability: the number of days of incapacity during the year, the presence of a disability group.

#### **About the condition of the patient(status praesens)[4]**

General condition of the patient: satisfactory, moderate, heavy. Consciousness: clear, suppressed, stupor, sopor, coma, excitement, euphoria, delusions, hallucinations. Patient: active, passive, forced. Facial expression: calm, excited, awkward, suffering, masked. Procession: free, skeletal, cheerful, duct, specific (hemiparesis, parkinsonism, etc.). Body: right, wrong. Constitutional type (normosthenic, asthenic, hypertensive), height, weight. Kettle Index ( $\text{kg}/\text{m}^2$ ). Skin and visible mucous membranes: color (pale, pale pink, red, bluish, icteric, earthy, pigmentation, depigmentation); rash (erythema, roseola, papule, pustule, vesicula, bulla, petechiae, scab, bruising, erosion, cracks, ulcers, scabies); scarring, vascular asterisks, xanthomas, xanthelases; skin moisture; turgor skin; Hovel breeding type.

Subcutaneous fat cell: development is weak, moderately, excessively; the place of the largest fat deposits; presence of pastoseness, characteristic of edema by localization and prevalence (general, local); color of the skin in the area of edema (pallor, bluech, hyperemia), quality (mobile, soft, etc.). Lymph nodes: submandibular, cervical, supra- and subclavicular, elbow, and inguinal. Determination of their size, consistency, pain, mobility, adhesion between themselves and with the skin; tonsils, their size, color, the presence of purulent cork in the gaps. Muscles: Degree of development (normal, excessive, weak, muscle atrophy - general or local), tone (elevated, lowered, normal); pain during palpation and movements; tremor or tremor of individual muscles; paresis, paralysis of extremities. Bones: examine the bones of the skull, thorax, pelvis and extremities in order to detect deformation, periostitis, curvature, acromegaly, changes in the ultimate phalanges of the fingers and toes, thumb fingers, pain during palpation. Joints: configuration (normal, swelling, deformation); hyperemia of the skin and local temperature increase in the joint area; the volume of active, passive movements (free or limited); pain during palpation and during movements; crunch, fluctuation, contracture, ankylosis. Degree of development (normal, excessive, weak, atrophy of muscles - general or local), tone (elevated, lowered, normal); pain during palpation and movements; tremor or tremor of individual muscles; paresis, paralysis of extremities. Bones: examine the bones of the skull, thorax, pelvis and extremities in order to detect deformation, periostitis, curvature, acromegaly, changes in the ultimate phalanges of the fingers and toes, thumb fingers, pain during palpation. Joints: configuration (normal, swelling, deformation); hyperemia of the skin and local temperature increase in the joint area; the volume of active, passive movements (free or limited); pain during palpation and during movements; crunch, fluctuation, contracture, ankylosis. Degree of development (normal, excessive, weak, atrophy of muscles - general or local), tone (elevated, lowered, normal); pain during palpation and movements; tremor or tremor of individual muscles; paresis, paralysis of extremities. Bones: examine the bones of the skull, thorax, pelvis and extremities in order to detect deformation, periostitis, curvature, acromegaly, changes in the ultimate phalanges of the fingers and toes, thumb fingers, pain during palpation. Joints: configuration (normal, swelling, deformation); hyperemia of the skin and local temperature increase in the joint area; the volume of active, passive movements (free

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#### **Respiratory system[4]**

Overview (inspectio) (in the presence of strangulation - its character, type of respiration, number of breaths per minute), chest shape, abdominal distension or supra-subclavicular destruction); palpation (palpia) (resistance, chest pain, voice tremor); percussion: comparative percussion of the lungs, determination of zones of bluntness, tympanitis, etc. indicating their size and precise localization, determining the nature of percussion sound (clear pulmonary sound, dullness, dullness, box). Topographic percussion is the determination of the height of the standing of the tops of the lungs at the front and back, the lower boundaries, the excursion of the edges of the lungs in cm, auscultation: the nature of the respiration (vesicular, bronchial, rigid, etc.), wheezing (dry and wet, large, middle and smallpox, sonorous, not sonorous, crepitation, pleura friction noise, their exact localization), bronchophonia.

#### **Cardiovascular system[4]**

Overview (visual pulsations of vessels, "dance carotid", cardiac hump, apical and heart stimulation);palpation (apical and cardiac stimulation, its localization, systolic and diastolic trembling);percussion (the borders of the heart - relative and absolute dullness, configuration of the heart, width of the vascular beam in cm);auscultation (tones of the heart - clear, deaf, noises, their characteristic, noise of pericardial

friction);examination of vessels: visual examination (visual pulsation) and palpation of available arteries, vorticity and density of the vascular walls of the temporal, lumbar and shoulder arteries, auditory carotid, femoral arteries, the phenomenon of Traube-Vinogradova-Dyurizieva, listening of the cervical veins (noise of the wolf);Pulse: frequency, filling, tension, rhythm, shape;the presence of asymmetry of the pulse; during arrhythmia simultaneously (with the calculation of pulse strokes) listening to the heart (determination of so-called pulse deficit); capillary pulse; Blood pressure on both hands: during arterial hypertension - BP on the lower extremities.

#### **Digestive system[4]**

Overview: oral cavity, mucous membrane, tongue, its layers, the state of papillae, cracks, ulcers, gums, teeth; Abdomen (form, participation in the act of breathing, expansion of subcutaneous veins), visual peristalsis of the stomach and intestine; Palpation - superficial (strain of the abdominal wall, Schotkin-Blumberg symptom, pain, its localization, distinction of direct abdominal muscles); deep (for Obratsovy-Strazhesko). Detection of ascites percutaneously and by determining fluctuations.Extinction: regularity and character; Liver: Percutaneous determination of liver size by reference lines (sizes for Kurllov). If the liver is achievable palpation, that is, the protrusion from the edge of the edge arch - size, pain, surface (smooth, buggous), edge (sharp, rounded), consistency (dense, soft). Special study of the area of the gall bladder.Pancreas. Palpation in the Growth of the Spleen: palpation in different positions of the patient (on the back, on the right side), its size, shape, consistency and condition of the surface; percussion of the spleen-sizes in cm (length and intersection).

#### **Urinary system[4]**

Review of the lumbar region;Palpation of the kidneys (size, shape, consistency, posture).The Pasternatsky Symptom.Urination (free, painful, etc.).

#### **Nervous-endocrine system [4]**

Mood, sleep, memory, hypnotic reflexes, symptom of Romberg, character of dermatographism, exophthalmos (one-way or bilateral), presence of eye symptoms, examination and palpation of the thyroid gland.Vision.Hearing.

#### **Second-line diagnosis and its justification[4]**

The preliminary diagnosis is based on complaints, anamnesis and objective data, which directly confirm the presence of this disease (only the features that are characteristic of the disease are used), and the effectiveness of the therapy is being taken into account. If possible, the form, phase, stage, course of the disease, etc. are displayed and substantiated in the diagnosis. Substantiation of the main, concomitant (therapeutic) diseases and complications is carried out separately.

It is necessary to highlight subjective and objective symptoms, to formulate syndromes and to establish a nosological diagnosis. **The diagnosis should include:**

- **The main illness that has become the cause of hospitalization;**
- **Complications caused by the underlying disease;**
- **Functional diagnosis of the underlying disease, which should reflect the state of the function of the affected organ: compensation or decompensation, its degree.**
- **Concomitant disease that is not pathogenetically associated with the underlying disease;**
- **Complications due to concomitant illness;**
- **Functional diagnosis of concomitant illness.**

The justification of the preliminary diagnosis should be written based on the analysis of complaints, data on the history of disease and life, data of an objective review on the following points:

- list complaints that allow us to conclude that the primary defeat of one or another body or system (for example, typical pain syndrome, the presence of fever, shortness of breath, etc.)
- to list the data of anamnesis of the disease, on which one can draw a conclusion about the predicted diagnosis (for example, an indication of a previously transferred myocardial infarction, an analysis of available electrocardiograms, an indication of the renal colic transmitted, an indication of the performed operation, and so on)
- to list the data of anamnesis of life, allowing to assume the factors of the given disease (for example, the burdened family history, the presence of professional influences, bad habits - alcohol abuse, etc.)
- to list the data of objective research that revealed a deviation from the norm in the physic status, or any symptoms (for example, obesity, cardiomegaly, wheezing in the lungs, cyanosis, and so on), suggesting the disease

- in addition to the formulation of the diagnosis of the main nosological form it is necessary to give data on which it is possible to specify the diagnosis with indication of the stage and form of the course of the disease, the phase, the degree of activity, the degree of functional disorders, and so on.
- list the data indicating the presence of complications of this disease
- to formulate the diagnosis of concomitant pathology, which can affect the existing underlying disease.

#### **Plan examination of the patient[4]**

Based on the previous diagnosis, the student outlines an individual plan for the supervision of the patient and his examination, consultations of other specialists.

Additional research methods should aim at addressing the issues of diagnosis, functional state of organs and systems that are involved in the pathological process, the degree of activity and the severity of the disease.

The plan of laboratory and instrumental research methods should include:

- Clinical blood test every 7-10 days;
- Total urinalysis every 7-10 days;
- Stool on eggs of worms;
- Blood studies on AIDS, syphilis;
- Determination of blood group and Rh factor;
- Blood sugar;
- Fluorography of the chest (if during the last year it was not conducted);
- Electrocardiogram;
- Observation patients every 10 days.
- A list of special laboratory and instrumental studies that need to be conducted when detected in a patient's pathology (indicate which).

#### **All laboratory and instrumental research methods, expert consultations[4]**

This section presents the results of compulsory and additional studies, conclusions of consultants. It is advisable to bring the normal parameters, and units of measurement in the additional column of laboratory and instrumental studies. Interpretation of the received data is carried out.

The same type of study is better located in the table, which will allow to allocate the dynamics of the indicator of peripheral blood leukocytes against the



background of pneumonia therapy with antibacterial drugs, or, for example, hemoglobin in anemia patient receiving iron preparations.

Also, an ECG analysis of a patient with myocardial infarction should not be formal. It will be substantiated if you bring the dynamics of wave and segments in specific leads (the presence of pathological Q, ST segment elevation, in which leads, and so on).

So you will be able to confirm your assumptions made as a concept of the diagnostic conclusion in the previous section.

### **Differential diagnostics[4]**

Differential diagnosis is performed by comparing the most important symptoms of the underlying disease in a patient with similar signs of other diseases.

This section begins with the justification of the choice of the disease, which will be differentiated. From the beginning, general manifestations of the illness of a supervised patient with a similar illness are described. The following is a comparison of each symptom in a given patient with a similar symptom of another illness with a reflection of the peculiarities (differences) of their manifestations.

It is necessary to take into account the absence of those symptoms in the curator's patient, which are characteristic of another disease, and vice versa, the presence of those symptoms that are not characteristic of another disease.

Differential diagnosis is performed in the same manner in which the patient was examined: from the beginning compares the complaints, then the data of the disease history and life, the results of objective examination and, finally, additional research methods that confirm the disease

Remark: only the symptoms and results of additional research methods that are present in this patient are used.

### **With an acute clinical diagnosis and its justification[4]**

In this section, the diagnostic version should be fully disclosed and validated as far as possible, since the selected diagnosis will depend on the treatment chosen.

Indicate which survey data confirmed your previous diagnosis, which clarified the form, phase, degree of activity and complication. It is possible that the diagnostic presentation after the examination had to be reviewed in favor of another diagnosis. This does not contradict the principles of medical thinking and does not

diminish your ability to meditate and interpolate information. Absence of doubt - often a satellite of limited horizons and dangerous self-confidence.

All changes and refinements of the diagnosis should be reflected in the text of the history of the disease: diaries, stage epicrises and so on.

The summary of your presentation could look like this:

The justification for the final diagnosis should be written by repeating the analysis of the complaints, data on the history of the disease and life, the objective review data, and completing the data of the survey, which confirmed it. When substantiating the clinical diagnosis reference is made to the previous diagnosis and differential diagnosis; Further, the data of additional research methods confirming this disease is used. It is necessary to separate the substantiation of the main, concomitant diseases and complications, justifying each position of the diagnosis.

Clinical diagnosis is formulated in accordance with the requirements of the classifications approved by the Ministry of Health of Ukraine or the congresses of doctors. Diagnosis displays the following sections:

- Etiology (if known);
- Clinical (clinical-morphological) variant of the disease;
- Phase (remission or exacerbation);
- Stage of flow;
- Some of the most distinct syndromes (the result of inclusion in the pathological process of various organs and systems);
- Complication.

#### **Treatment [4]**

Present the modern principles of treatment of the underlying disease according to the following plan:

- Regime;
- Diet;
- Psychotherapy;
- Drug treatment;
- Physiotherapy;
- Therapeutic physical training and massage;

- Sanatorium and spa treatment;
- Operative treatment (indications);
- Dispensary supervision and antiretroviral therapy.

#### **Prevention[4]**

Primary - the prevention of a disease, secondary - the prevention of exacerbations, relapses of the chronic process.

#### **Prognosis and examination of disability[4]**

The forecast is substantiated in relation to disease, life and work capacity. The forecast can be favorable, questionable and unfavorable.

The prognosis for the disease is considered favorable if there is confidence that the curative patient will recover; questionable - if there is no confidence in complete recovery and unfavorable if the disease is incurable and has a chronic progressive course.

The prognosis for life can be favorable in the event that the patient is not threatened with complications that are life-threatening; questionable - if under certain circumstances in a patient (taking into account his age, course of disease, progression, complications, treatment efficacy, etc.) may occur a fatal case, and unfavorable if the patient is a lethal accident is inevitable.

The prognosis concerning the ability to work is solved in the plan of temporary or permanent loss of it (disability group) taking into account the degree of functional disorders and occupation of the patient.

### **EXAMPLES OF FORMULATION OF DIAGNOSIS IN CARDIOLOGICAL CLINICS[5, 6]**

- Adenoma of the right adrenal gland, primary hyperaldosteronism (Conns disease). Secondary (symptomatic and chronic) arterial hypertension III st. Residual effects of ischemic cerebrovascular accident in the basin of the left carotid artery (January, 2015). Right hemiparesis. HF I st. with preserved EF.
- Arterial hypertension, 1 st., high additional risk.
- Arterial hypertension II st., 2 degree, very high additional risk. Hypertensive angiopathy of the retina (generalized narrowing of arteries).

- Arterial hypertension II st., 3 degree, high additional risk.Hypertensive heart (LVH) and HF with preserved EF.
- Arterial hypertension II st.,2 degree, high additional risk, hypertensive kidney damage (in the presence of proteinuria)
- Arterial hypertension II st., 3 degrees, a very high additional risk.CHD: stable angina pectoris, II fc. HF with preserved EF.
- Arterial hypertension III st, 3 degrees, a very high additional risk.Hypertensive retinopathia (hemorrhage in the fundus).
- Arterial hypertension III, 3 degrees, a very high additional risk.Hypertensive heart, HF (or IIB, III st.) with systolic dysfunction of LV, III FC NYHA.
- Arterial hypertension III, 2 degree, very high additional risk.Residual effects of acute cerebrovascular disorder
- Arterial hypertension III, 3 degrees, a very high additional risk.IHD, postinfarction cardiosclerosis (specify date infarction). HF with preserved EF.
- Arterial hypertension III, 2 degree, very high additional risk. Primary nephrosclerosis, CKD I st.
- Arterial hypertension III, 3 degrees, a very high additional risk.Transient ischemic attack (indicate vascular pool)
- Arterial hypertension III, 2 degree, very high additional risk.Chronic hypertensive encephalopathy III st.(or circulatory hypertensive encephalopathy III)
- Arterial hypertension III st.,3 degrees, very high additional risk.Hypertensive retinopathy (swelling of the optic nerve hemorrhage in the fundus)
- Acute rheumatic fever, activity of the III st., Carditis, polyarthritis, anulus erythema, HF II A st.FC II
- Acute viral (influenza) diffuse myocarditis, extrasystoly, HF IIwith low EF.
- Acute exudative streptococcal (sero-fibrinos) pericarditis, the average severity (Echo from 18.10.16). HF II A with preserved EF.
- Dilatational cardiomyopathy.Atrialfibrillation, permanent form. HF II B with low EF.FC IV.
- Closed head injury.A brain shaft (15th ofSeptember20<sup>th</sup>).Secondary (symptomatic) arterialglanI st.Uncomplicated hypertensive crisis (August 20, 2016).

- Coronary heart disease. Acute Q myocardial infarction of the anterior-lateral wall left ventricle (11.01.15). PCILDA(11.01.15). HF with preserved EF.
- CHD: inferior Q-Myocardial Infarction (from 20.11.17). Acute heart failure (Killip I c.) (21.11.17). CH I. FC I.
- CHD: Q anterior myocardial infarction (from 10.11.15). Acute heart failure (Killip III) (11.11.15). Post-infarction angina (15.11.15). Postinfarction cardiosclerosis (06.03.03). HF II A ct. FC I.
- Coronary artery disease, progressive angina (from 21.10.15). HF I. FC I.
- CHD: angina pectoris II FC, HF I.
- CHD: angina, which arose for the first time, HF 0 st.
- Neurocirculatory dystonia for hypertensive type, moderate severity.
- NCD, dyshormonal, moderate, hypertensive, tachycardial, depressive syndromes. Panic Attacks.
- NCD, moderate, with cardiac, respiratory and neurotic syndrome in acute phase.
- Staphylococcal endocarditis III activity; aortic lesions (valve insufficiency), mitral; HF II B, systemic vasculitis, nephritis with isolated urinary syndrome, splenomegalia.
- Congenital heart defect: ASD. Secondary infectious (fungi) endocarditis, III activity aortic lesions at the first valve (valve insufficiency), HF II st.
- Urolithiasis. Secondary chronic pyelonephritis secondary (symptomatic) arterial hypertension III st. Hypertensive heart. Permanent atrial fibrillation. HF IIA with preserved EF, II FC.
- Stenosis of the right renal artery. Condition after balloon angioplasty (January 2003). Secondary (symptomatic and chronic) arterial hypertension II st. Hypertensive heart, LV hypertrophy. Monotopic ventricular extrasystoly. HF I.
- Feochromocytoma of the right adrenal gland. Secondary arterial hypertension II st. Hypertensive heart. Paroxysmal form of atrial fibrillation. Uncomplicated hypertension and crises (September 2, 2014). HF I.
- Chronic rheumatic heart disease: mitral valve disease with a predominance of stenosis, II st. HF II A ct. FC III
- Chronic (idiopathic) constrictive pericarditis, HF II B st. FC IV
- Chronic post-tuberculous adhesive pericarditis, HF I. FC I.

## **CALCULATION OF THE BODY MASS INDEX[7, 8]**

The problems of obesity reached such importance in the world that now is regular in the twenty-first century. People who are overweight, 3 times more likely to suffer from arterial hypertension, 2.5 times more likely to develop carbohydrate metabolism, including - diabetes. It is known that 90% of all cases of diabetes are type 2 diabetes, and among patients 80-90% of patients have obesity. In young people with overweight, a high level of cholesterol is found 2.1 times more often[7].

According to WHO, Body Mass Index (BMI) is the most effective way to measure overweight and obesity at the population level, as it can be applied to all adults regardless of gender and age. At the same time, it should be considered only as an approximate benchmark, because it may not correspond to equal manifestations of obesity in different people. As well as for an individual, for the population there are their normal average levels of BMI. At the same time, this indicator may differ for a specific ethnic group. This is due to the social and economic, but, to a greater extent, ethnic characteristics of the populations. For most of them, the average normal BMI is about 22 kg/m<sup>2</sup>. In this case, the normal values of BMI for the population of developed countries correspond to an interval of 20-25 kg/m<sup>2</sup>, and for the population of developing countries - 18,5-25 kg/m<sup>2</sup>. As for individuals, 3 levels of BMI are distinguished for populations[8].

In the mortality forecast, a large number of indicators and techniques are used, among which one of the key places is the BMI. This is due, above all, to the existence of a positive correlation between mortality and obesity in virtually all populations. In turn, BMI is a simple, informative, but at the same time the most popular indicator for assessing the level of overweight and the degree of obesity. Studies in recent years have revealed the association of BMI with mortality from diseases such as ischemic heart disease, stroke, intestinal cancer, stomach. An important aspect of obesity is its relationship with hypertension. According to research data, arterial hypertension in persons with BMI > 30 kg/m<sup>2</sup> occur 3 times more frequently, hyperlipidemia is 1.5 times more frequent than among the general population. The risk of developing these diseases increases significantly with an increase in BMI > 35 kg/m<sup>2</sup>. According to modern beliefs, obesity is one of the factors of the development of inflammatory

processes of different localization and nature. Even a slight excess of BMI > 30 kg/m<sup>2</sup> greatly increases the risk of developing diseases such as diabetes mellitus, coronary heart disease, and many other diseases [7, 8].

Rarely, researchers are involved in the study of the risk of developing pathologies at low BMI values. Although it should be noted that these risks are quite consistent with the risks of developing diseases with BMI > 30 kg/m<sup>2</sup>. The results of an analysis conducted by an international team of researchers have shown that in patients with low BMI associated with osteoporosis, the risk of bone fractures is higher than in patients with a similar pathology and normal BMI. Body mass index (BMI) is a value that allows you to evaluate the degree of conformity of a person's mass and its height, and thereby indirectly assess whether the mass is inadequate, normal, excessive (obesity). The index of body mass index was developed by the Belgian sociologist and statistician Adolphe Quetelet (1869). The body mass index is calculated according to the formula: you need to divide your weight in kilograms by square of height in meters.  $BMI = \text{weight (kg)} : [\text{height (m)}]^2$  and measured in kg/m<sup>2</sup> [7, 8].

Interpretation of BMI:

- 20-25- normal weight, risk for health is absent;
- 25-30- the excess weight of the body, the risk for health is elevated;
- 30-35 – Obesity – Risk for health is high;
- 35 and more – sharply expressed obesity, risk for health is very high.

**Classification of obesity under the BMI (WHO) [7, 8]**

<b>Classification</b>	<b>Body mass index, kg/m<sup>2</sup> (BMI by Kettle)</b>	<b>Risk village in Putnam diseases</b>
Body mass deficiency	less than 18.5	Low (increased risk of other diseases)
Normal body mass	18.5-24.9	Usual
Excessive body weight (nursing)	25.0-29.9	Elevated

Obesity I degrees	30.0-34.9	High
Obesity II degrees	35.0-39.9	Very tall
Obesity III degree	40.0 and more	Extremely high

## **METHOD OF MEASUREMENT OF ARTERIAL PRESSURE[9, 10]**

### **Auscultatory method for measuring blood pressure**

Indirect measurement (auscultative method) of arterial pressure (AP), if properly performed, is a safe, painless procedure and provides reliable information. Diagnosis of hypertension in children and adolescents is based solely on the accuracy of blood pressure measurement by this method[9, 10].

### **Equipments[9, 10]**

Blood pressure is usually measured with a sphygmomanometer (mercury or aneroid) and a phonendoscope (stethoscope). The price of dividing the scale of the sphygmomanometer should be 2 mmHg. Indications of the mercury gauge are estimated at the upper edge (meniscal) of the mercury column. Mercury gauge is considered as a "gold standard" among all devices used to measure blood pressure, since it is the most accurate and reliable tool. An aneroid gauge consists of metal blends, which expand with increasing air pressure in the cuff, and the pressure value is estimated from the mark on the scale indicated by the gauge's arrow. The phonendoscope (stethoscope) should have a nozzle or membrane nozzle for listening to low frequency sounds. Headphones of the phonendoscope (stethoscope) must correspond to the dimensions of the researcher's external auditory passage and to block external noise[9, 10].

### **The situation[9, 10]**

Measurement of blood pressure should be carried out in a quiet, calm and comfortable environment at a comfortable temperature. Directly in the room where the blood pressure is measured, there should be a couch, table, place for the researcher, a chair for the patient with a straight back and, if possible, an adjustable seat height, or a device to maintain the patient's hand at the heart. When using a mercury sphygmomanometer, the meniscus of the mercury column should be at the



level of the eye of the researcher. The patient should sit on the chair next to the table. To measure the blood pressure in the standing position, a rack with an adjustable height and a supporting surface for the patient's hand is used. The height of the table and the racks should be such that when measuring the blood pressure of the middle of the cuff imposed on the patient's shoulder, it is at the heart level, that is, approximately at the level of the fourth interlaboratory 'I am sitting in a position or at the level of the middle axillary line in a lying position. The deviation of the midpoint of the cuff, applied to the shoulder or thigh of the patient, from the heart rate, may alter the blood pressure level on 0.8 mmHg. for every 1 cm deviation of the cuff: to inflate the AT at the position of the cuff below the heart rate and to reduce the blood pressure at the position of the cuff above the heart rate. Supporting the back of the patient on the back of the chair and hands on the supportive surface excludes increased blood pressure due to isometric muscle contraction[9, 10].

#### **Preparation of the patient for the measurement of blood pressure[9, 10]**

Measurement of blood pressure should be carried out not earlier than 1 hour after eating, drinking coffee, stopping physical activity, smoking, staying in the cold. The shoulder of the patient should be released from the clothes, the hand should conveniently lie on the table (when measuring blood pressure in the sitting position) or on the couch (when measuring blood pressure in the lying position), the palm up. Before measuring blood pressure, the patient needs to rest for 5 minutes. Measuring blood pressure should briefly explain to the patient the procedure for measuring, in order to avoid a negative reaction from the patient, which can lead to increased blood pressure. During the measurement, the patient should sit on the back of the chair, with relaxed, unbroken legs, not to change the position and not to speak during the entire blood pressure measurement procedure[9, 10].

#### **The size and position of the cuff[9, 10]**

It is necessary to correctly pick up the cuff, corresponding to the circumference of the patient's shoulder (child, adolescent or adult). The width of the inner (rubber) cuff of the cuff should be at least 40% of the shoulder circumference. The length of the rubber cuff of the cuff should cover 80% to 100% of the shoulder circumference. The circumference of the shoulder is measured with a centimeter tape with an accuracy of 0.5 cm in the middle of the distance between the elbow and the acromial appendix of

the shoulder blade. The cuff is superimposed so that the center of the rubber chamber is located above the shoulder artery on the inner surface of the shoulder, and the lower edge of the cuff was at 2-2.5 cm higher than elbow bend. The density of the cuff is determined by the possibility of holding one finger between the cuff and the surface of the patient's shoulder. Rubber tubes connecting the cuff with a pressure gauge should be medial (on the inner surface of the shoulder)[9, 10].

When measuring blood pressure on the lower limbs, the cuff of the appropriate size is superimposed on the thigh so that the center of the rubber chamber is located above the femoral artery on the inner surface of the thigh, and the lower edge of the cuff is 2 to 2.5 cm higher than the popliteal hollow[9, 10].

**Determination of the maximum level of airflow in the cuff (palpation method for assessing the level of arterial pressure systole)[9, 10]**

This procedure is necessary to establish the level of systolic blood pressure (SBP) to ensure adequate injection of air into the cuff with minimal discomfort for the patient with an auscultation method for determining the blood pressure.

Palpation method also allows to exclude "sound pass", to determine SBP in young children and at very low blood pressure (shock). The level of SBP at the same time is 5-10 mmHg below in comparison with the indications of the auscultatory measurement method[9, 10].

To evaluate the level of the SAT by the palpation method, it is necessary:

- to determine the pulsation of the shoulder artery in the elbow flexion (typical artery position - the medial tendon of the two-head muscle), the nature and the rhythm of the pulse;
- palpating the radius artery (or shoulder artery), quickly pump air into the cuff to 60-70 mmHg afterwards, observing the manometer readings, continue to slowly (at a rate of 10 mmHg/sec) to inflate the air until the pressure in the cuff exceeds 30 mm the level at which the rupture of the beam or shoulder is not determined arteries;

- slowly releasing the air from the cuff (at a rate of 2 mmHg/sec) to indicate, at the readings of the pressure gauge, the moment of restoration of the ripple. An indication of a manometer at the moment of the disappearance of the pulsation when the air is injected into the cuff and its recovery during the slow evaporation of the air from the cuff will correspond to the approximate level of the SBP;
- completely release the air from the cuff.

For further measurements, the air in the cuff should be pressurized at 30 mmHg higher than the SBP level, is evaluated palpated.

### **The position of the stethoscope [9, 10]**

The nozzle of a stethoscope with a bell or a low-frequency membrane is installed with a slight push over the area of maximum pulsation of the brachial artery in the elbow flexion so as not to touch the lower edge of the cuff or tubes. The collision with them violates the perception of Korotkov's tones. When measuring blood pressure on the lower extremities, the nozzle of the stethoscope with a rupture or low-frequency membrane is installed in the popliteal fossa, above the region of maximum pulsation of the popliteal artery [9, 10].

### **Pumping and blending cuffs [9, 10]**

The injection of air into the cuff to the maximum level (30 mmHg above the SBP level, evaluated palpated) should be performed quickly. Slow injection of air into the cuff leads to a violation of venous outflow, increased pain and "smearing sound." The air from the cuff is released at a rate of reduction of the mercury column of 2 mmHg per second, and with the appearance of Korotkov's tones - 2 mmHg for every pulse beat. If the meniscus of a mercury column at the moment of appearance or disappearance of Korotkov's tones is between two divisions of the manometer scale, then the readings of the SBP or DBP are evaluated at the nearest upper value. In case of bad hearing it is necessary to quickly release air from the cuff, check the position of the stethoscope and repeat the procedure in 2-3 minutes. Knowledge of the distinctive features of different phases of tones of Korotkov allows the most accurate determination of the level of SBP and DBP. The accuracy of the determination of BP

also depends on the decompression rate: the higher the decompression rate, the lower the accuracy of the measurement[9, 10].

### **Characteristic of Korotkov's tones[9, 10]**

<b>Phase</b>	<b>Characteristic of Korotkov's tones</b>
I (KI)	Sounds are weak, knocking, with gradually increasing intensity
I (KII)	The sounds are longer, muffled, cluttered
III (KIII)	Sounds again become expressive and loud
IV (KIV)	The sounds are soft, muted, less noticeable
V (KV)	Complete disappearance of sounds

### **Systolic blood pressure[9, 10]**

The SBP level is determined by the beginning of Phase I of the Korotkov tones - on the first of a series of tones that follow one another, that is, the first tone must follow the second tone in the first tone. A single tone at the beginning of the phase (when the first tone is silent - the audio pass) is ignored.

### **Diastolic blood pressure[9, 10]**

The values of DBP determine the beginning of the V phase of the tone Korotkov - in the silence, which occurs in the last tone of phase IV. The last tone at the end of the phase, even if it is single (when the last tone is preceded by an auscultation failure) is always taken into account. Auscultation should be continued for 20 mm after the last tone disappears, and at DAT above 90 mmHg – within 40 mm, since after the sound pass, the tones can resume. Compliance with this rule will prevent the definition of a mistakenly increased DBP [9, 10].

Definition of DBP in the fourth phase of tones (at the time of their sharp decrease) is recommended for the measurement of blood pressure in children under 12 years of age, pregnant women, as well as in patients with high-minute heart rate due to physical activity, disease or physiological characteristics. The transition to the fourth phase is necessary also in the absence of a clearly expressed fifth phase - the phenomenon of "infinite tone". Ability to correctly evaluate the beginning and end of the I - IV phases of tone Korotkov has a great clinical significance, especially when

measuring blood pressure in patients with heart rhythm disorders. Absence of the V phase, that is, when the tones of Korotkov listen to the end of the reduction of the mercury column ("the phenomenon of an infinite tone") can be observed with high cardiac output (in children, with thyrotoxicosis, fever, aortic insufficiency, during pregnancy). In these cases, the level of DBP is estimated at the beginning of phase IV - in the first of a series of Korotkov's tones that go one after the other [9, 10].

#### **Registration of measurement results [9, 10]**

In the history of the disease or questionnaire it is recommended to write on which hand the measurement was made, in which position (lying, standing or sitting), the size of the cuff. Measurement results are written in the form of KI/KV (see Table 1) (for example: 120/70); if the IV phase of Korotkov's tones was determined - in the form of KI/KIV/KV (for example: 120/78/70); with the "phenomenon of an infinite tone" when the V phase of the tone Korotkov is equal to 0 in the form of KI/KIV/KV (for example: 120/78/00).

#### **Repeated measurements [9, 10]**

Repeated measurements are carried out not earlier than 2-3 minutes after the complete release of air from the cuff. If the SBP or DBP indices differ by more than 5 mmHg, then an additional measurement is carried out. The average of two or more measurements taken in one hand is taken into account [9, 10].

#### **Measurement of blood pressure in other positions [9, 10]**

Measurement of blood pressure in the sitting and standing position can be carried out at the age of 2 years of the child. Up to 2 years of age, blood pressure is measured in the lying position [9, 10].

#### **Special situations when measuring blood pressure [9, 10]**

- The auspicious failure is a period of temporary lack of sound between the phases I and II of the tone of Korotkov. It can last up to 40 mmHg. Observed with high systolic blood pressure.
- Absence of the V phase of the tones of Korotkov (phenomenon of "endless tone"). It is observed at high cardiac output: in children, with thyrotoxicosis, fever, aortic insufficiency, in pregnant women. Tony Korotkiv listened to the zero division of the scale of the sphygmomanometer. In these cases, for the diastolic blood

pressure, the beginning of the IV phase of the tone of Korotkov is taken and the BP is recorded in the form of KI/KIV/K0.

- Measurement of blood pressure in the elderly. With age, thickening and compaction of the wall of the brachial artery are observed; it becomes rigid. Need higher (above intraarterial) level of pressure in the cuff to achieve compression of the rigid artery, resulting in a false overshoot of the level of AT (the phenomenon of "pseudohypertension"). Palpation of the pulse on the radial artery at the pressure level in the cuff, which exceeds the SAT, helps to recognize this error. It should palpate to determine the blood pressure on the forearm. With the difference between the SAT, defined palpated and ausculturally, greater than 15 mmHg, only direct invasive measurement can determine the true level of blood pressure in the patient. You should inform the patient about the problem and make an appropriate record in the history of the disease to avoid measurement errors in the future.
- Very large shoulder circle (obesity, very developed musculature), conical arm. In patients with a shoulder circle of more than 41 cm or with a conical shoulder shape, when it is not possible to achieve the normal position of the cuff, accurate measurement of blood pressure may not be possible. In such cases, using a cuff of the appropriate size, you should try to measure the PA palpatorily and auscultation on the shoulder and forearm. With a difference of more than 15 mmHg, blood pressure, defined palpatorily on the forearm, more accurately reflects the true blood pressure.

**Factors that determine the occurrence of errors when measuring blood pressure [9, 10]**

Factors that determine the occurrence of errors in the measurement of blood pressure can be related to equipment, a researcher who performs blood pressure measurements, and a patient. Because of limitations in the reliability of the results, repeated measurements of blood pressure are performed.

**Causes of possible errors in measuring blood pressure [9, 10]**

Environment and equipment	Researcher	Patient
<ul style="list-style-type: none"> <li>• Uncomfortable indoor temperature</li> <li>• Non-standard equipment</li> </ul>	<ul style="list-style-type: none"> <li>• Wrong fitted cuff</li> <li>• Failure to observe the measurement technique</li> </ul>	<ul style="list-style-type: none"> <li>• Wrong patient position</li> <li>• "Negative" reaction to blood pressure</li> </ul>

<ul style="list-style-type: none"> <li>• Malfunction of the pressure gauge</li> <li>• Tightness of the system</li> <li>• Defect of the phonendoscope (stethoscope)</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of vision</li> <li>• Hearing loss</li> <li>• Psychological factor (giving preference to the numbers ending at "0" and "5")</li> </ul>	measurement <ul style="list-style-type: none"> <li>• Smoking</li> <li>• Eating</li> <li>• The use of coffee</li> <li>• Drinking alcohol</li> <li>• Changes in body temperature</li> <li>• Physical load</li> <li>• Pain</li> </ul>
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The auscultative technique is currently recognized by the WHO as a reference method for noninvasive determination of AT, despite somewhat lower values for SAT and overestimated - for GAT compared with figures obtained from invasive measurements[9, 10].

The advantages of the Korotkov method are: higher resistance to cardiac arrhythmias and hand movements during measurement. Disadvantages of the method are related to high sensitivity to noise in the room, obstacles that arise when rubbing cuffs about clothing, as well as the need for accurate placement of the microphone over the artery. Accuracy of registration of blood pressure significantly decreases at low intensity of tones, the presence of "auscultative failure" or "endless tone".

### **Daily monitoring of arterial pressure[9, 10]**

Daily Blood Pressure Monitoring (DBPM) is a method for assessing daily rhythm in a natural environment using portable monitors of blood pressure.

#### **Indications for the conduct of the DBPM:**

- significant fluctuations of blood pressure during one or several visits;
- suspicion of "white coat hypertension";
- symptoms that make it possible to suspect hypotonic episodes;
- AH, resistant to pharmacological treatment;
- evaluation of the effectiveness of medication therapy.

#### **Contraindications to DBPM:**

There are no absolute contraindications to the use of DBPM in adults, children and adolescents.

#### **Possible complications:**

- swelling of the forearm and brush;

- petechiae hemorrhages;
- contact dermatitis.

### **Programming of monitors (measurement plan)[9, 10]**

The measurement plan involves setting daily (06.00-24.00) and night (00.00-06.00) periods. The frequency of measurements in the daytime - 1 time in 15 minutes, in the night period - 1 time in 30 minutes.

The periods of sleep and wakefulness are recorded by the patient by pressing the "event" button on the monitor. The beginning of the night period (for calculations) - approximately 1 hour after the "event", and the day - one hour before the "event". When statistical processing of data, the shift of the daily period to  $\pm 1$  hour does not affect the average. Editing the duration of day and night intervals is also possible after transferring data from the monitor to a computer.

When programming monitors you need to select the appropriate size of the cuff, disable the warning sound, turn off the display, so that the results of planned measurements (for reducing the increased attention of children to the device and the prevention of excitement from the high values of blood pressure).

### **Monitor installation method[9, 10]**

The monitor is placed in a case and secured to the body of the patient. The cuff is fixed so that the pipe fitting or the label "arteria" are approximately over the brachial artery. The outlet tube should be directed upwards so that the patient, if necessary, could put other clothes on top of the cuff.

After the monitor is installed, it is necessary to explain the behavior at the moment of measurement and demonstrate one measurement. During DMBP, the rhythm of life should be normal, and with an intense physical, emotional or mental load, the patient should make an appropriate entry in the diary. The patient begins to measure the measurement by compression of the shoulder as a result of an increase in pressure in the cuff. At this moment, if the patient is walking or running, it is necessary to stop, lower the arm with the cuff along the body, relax the muscles as much as possible, do not move your fingers. If the patient is sitting or lying, it is necessary to leave the hand in the position in which it was at the moment the device was turned on. Planned measurements are accompanied by a smooth inflow of air into the cuff and a gradual decrease in pressure. When the measurement failed, the monitor



again pumping air into the cuff after lowering the pressure. The monitors are equipped with the "Extra Measurement" button, which the patient can press when there is a headache attack, pain in the heart, dizziness, and so on. At this moment, an extraordinary measurement of blood pressure and heart rate will take place[9, 10].

### **Assessment of monitoring data[9, 10]**

In the analysis of data obtained at DMBP, the following informational groups are the following parameters:

- average values of BP (systolic, diastolic, pulse and average hemodynamic) per day, day and night;
- maximum and minimum values of blood pressure in different periods of the day;
- indicators of "pressure load" (hypertension index, index of hypertension) per day, day and night;
- variability of blood pressure;
- daily index (the degree of nightly decrease in blood pressure);
- morning rise of BP (magnitude and speed of morning rise BP);
- Duration of hypotonic episodes (index of time and index of hypotension area) at different periods of the day.

**The average values of BP** (systolic, diastolic, mean hemodynamic, pulse) give an idea of the level of blood pressure in the patient and most accurately reflect the true level of hypertension.

**The index of time (IR)** of hypertension or "proportion of high blood pressure" allows you to estimate the time of increase in blood pressure overnight. This indicator is calculated on the basis of the percentage of measurements that exceed the normal BP for 24 hours or separately for each time of day. As a criterion for hypertension in children and adolescents during the daytime period, they take the value of the 95th percentile for the corresponding sex, age and height, and during the night period, the blood pressure is 10% less than in the daytime. Inflammation of hypertension in healthy children and adolescents should not exceed 25%. In labile hypertension, hypertension ranges from 25 to 50%. Stable hypertension is diagnosed at a hypertension index of at least 50% at daytime and at night[9, 10].

**The area index** is calculated as the area of the figure limited by the curve of elevated blood pressure and the level of normal blood pressure.

**The variability of blood pressure** is calculated according to the standard deviation of the mean value of blood pressure. For children and adolescents, the norms of the variability of blood pressure are not established. For teenagers 16 years of age and older, you can use the variability standards that are currently available for older age groups: for SAT in daytime and nighttime -15 mmHg for DBP during the daytime -14 mmHg and at night -12 mmHg.

**The daily index (CI - the degree of nightly decrease in blood pressure)** shows the difference between the average daily and nightly values of blood pressure in percentage of daily average.

Optimum is the degree of night-time reduction of blood pressure from 10 to 20% in comparison with the daily indicators.

The size of the DI is divided into four groups of patients [9, 10]:

- normal (optimum) degree of nightly decrease in blood pressure (in the English literature - "dippers") - DI 10-20%;
- insufficient degree of night-time reduction of blood pressure ("non-dippers") -  $0 < DI < 10\%$ ;
- increased level of night-time reduction of blood pressure ("over-dippers") -  $DI < 20\%$ ;
- steady increase of night-time ("night-peakers") -  $CI < 0$ .

Morning elevated BP (magnitude and speed of morning rise AT). The magnitude of the morning rise of blood pressure is estimated by the difference between the maximum and minimum blood pressure in the period from 4.00 to 10.00 am in the morning. The speed of morning rise of blood pressure is estimated by the ratio of magnitude and time of lifting BP [9, 10].

## METHOD OF REGISTRATION AND EVALUATION OF ELECTROCARDIOGRAMS[11, 12, 13]

Electrocardiography (from the Greek "cardia" - the heart and "grapho" - write) - a method of graphically registering the difference in heart potentials in different phases of his activity. Electrocardiography allows you to study the basic functions of the heart: automatism, conductivity, excitability, refractoriness and aberrantness[11, 12, 13].

An electrocardiogram (ECG) is a graphical representation of oscillations of electrical potentials taken from the body surface.

ECG recording is performed using electrocardiographs - instruments that record changes in the potential difference between two points in the electric field of the heart (for example, on the surface of the body) during its excitation. Modern electrocardiographs are of high technical perfection and allow the implementation of both single-channel and multichannel ECG record[12].

Changes in the potential difference on the surface of the body that occur during the operation of the heart are recorded using various ECG system. Each discharge identifies the potential difference between two specific points of the electric field of the heart in which electrodes are installed. The last connect to an electrocardiogram electro galvanometer: one of the electrodes is connected to the positive pole of the galvanometer (this is a positive or active electrode of the discharge), the second electrode - to its negative pole (negative or indifferent, discharge electrode). In the international clinical practice, the most frequently used system of 12 leads, consisting of three standard, three reinforced unipolar limbs and six thoracic channels, was introduced[12].

**Standard bipolar leads**, proposed in 1913 by ECG detector Einthenhom, register the potential difference between two points distant from the heart and located in the frontal plane of the body, on the extremities (Fig. 1).

To record these assignments adopted colored markings of electrodes:

right hand - electrode of red color;

left hand - electrode of yellow color;

left leg - electrode of green color;

right leg - electrode of black color

Coupling of these electrodes (except for the black, connected to the grounding wire) forms a system of three standard assignments.

I lead - right hand (minus) and left hand (plus)

II lead - right hand (minus) and left leg (plus)

III draw - left hand (minus) and left leg (plus)

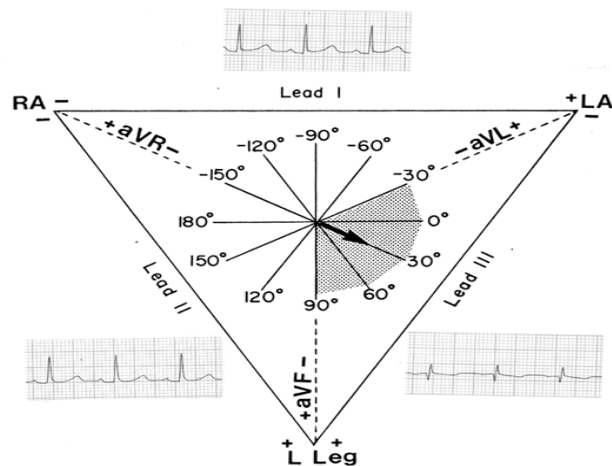


Fig.1. Scheme of standard and strengthened leads from the extremities.

The axes of the three standard leads form an equilateral triangle (the Einthoven triangle), whose vertices are the right hand, the left hand and the left foot with the electrodes installed there. In the center of the equilateral Einthoven triangle there is an electric heart center, or a single point cardiac dipole, which is equally distant from all three standard assignments. The hypothetical line, which combines two electrodes involved in the formation of electrocardiographic drainage, is called the axis of withdrawal. The axes of standard assignments are the sides of the Einthoven Triangle. Perpendiculars drawn from the center of the heart (from the location of a single cardiac dipole) to the axis of each standard lead, divide each axis into two equal parts: a positive, inverse in the direction of the positive (active) electrode (+) lead, and a negative, turned to a negative electrode (-) [12, 15].

**The reinforced unipolar distances from the extremities** proposed in 1942 by Goldberger record the potential difference between one of the extremities, on which the positive electrode is established, and the average potential of the other two extremities. The method has a standard marking (a is an augmented letter augmented, V - Wilson, right - right, left - left, foot - leg):

avR - enhanced unipolar drainage from the right arm  
 avL - reinforced unipolar withdrawal from the left hand  
 avF - reinforced unipolar withdrawal from the left leg.

The axes of reinforced unipolar leads from the extremities are obtained by connecting the electric center of the heart with the place where the active electrode of the given line is applied, that is, in fact, **with** one of the vertices of the Einthoven triangle. The electric heart center seems to divide the axis of these leads into two equal parts: a positive, directed to the active electrode, and a negative one facing the combined electrode of Goldberger (Fig.2).

**Standard and enhanced unipolar distances from the extremities allow you to register changes in the heartbeat of the frontal plane**, that is, in the plane in which the Einthoven Triangle is located. For a more accurate and clearer definition of the various deviations of the heart rate EMF in the frontal plane, a six-axis coordinate system of Bayley was proposed (Figure 2.2). It comes out when combining the axes of the three standard and three reinforced leads from the limbs, conducted through the electric heart center. The latter divides the axis of each lead into the positive and negative parts, respectively, to the active (positive) or negative electrode [12].

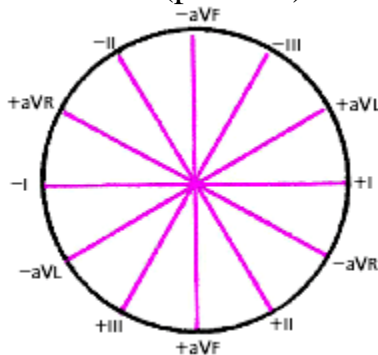


Fig.2. Bayley's six-axis coordinate system.

The wave of an electrocardiogram in various leads from the extremities can be considered as different projections of the same heart EMF on the axis of these assignments. Therefore, by comparing the amplitude and polarity of electrocardiographic complexes in different leads that are part of the six-axis coordinate system, it is possible to accurately determine the magnitude and direction of the heart EMF vector in the frontal plane [12].

The direction of the axes of assignments is taken to determine in degrees. At the beginning of the reference (0), the radius taken strictly horizontally from the electric center of the heart to the left in the direction to the positive pole I of the standard lead is taken conditionally. The positive pole II standard lead angle is  $+60^\circ$ , abduction aVF-angle of  $+90^\circ$ , III standard lead -angle of  $+120^\circ$ , aVL -at an angle of  $-30^\circ$ , and aVR-- $150^\circ$  angle to the horizontal. The lead aVL is perpendicular to the standard drive axis II, the standard drive axis I is perpendicular to the aVF axis, and the aVR axis is perpendicular to the axis III of the standard drive[12].

The system of standard and enhanced from the limbs of deliveries is supplemented by one-pole delivery from the chest, proposed in 1934 by Wilson. They record the potential difference between the positive chest electrode located at different points and the combined Wilson electrode, combining three electrodes from the extremities[12, 13].

Breast electrodes are marked with the letter V (from Wilson) and are located in the following positions (Figure 3):

- V1 - the fourth inter-choice item to the right of the sternum;
- V2 is the fourth intervertebral left of the sternum;
- V3 - intermediate position V2 and V4;
- V4 - the fifth intervertebral area on the left medullary clavicular line;
- V5 - the fifth intervertebral line on the left anterior axillary line;
- V6 - the fifth intervertebral line on the left middle armpit.

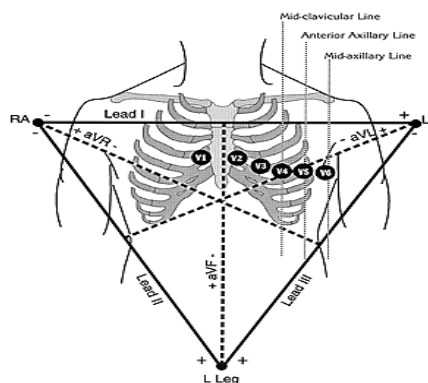


Fig.3. Chest drawings.

**Chest extraction registers changes in heartbeat mainly in the horizontal plane.** The axis of each chest is formed by a line connecting the electric center of the heart with the location of the active electrode on the chest. The axes of the leads V1 and V5, as well as V2 and V6, are approximately perpendicular to each other[12, 13].

#### **Additional divisions[12]**

In order to expand the diagnostic capabilities of 12 commonly used leads, an additional placement of Wilson lead electrodes is used:

V7 - the fifth intervertebral line on the left posterior axillary line;

V8 - the fifth intervertebral line on the left line of the shoulder blade;

V9 - the fifth intervertebral line on the left side of the retinal line.

**Sky system:** the electrodes of the system of three standard assignments are used to record entries.

- red electrode - the second intermeasurement to the right of the sternum;
- green electrode - fifth intervertebral in the median-clavicular line;
- yellow electrode - fifth intervertebral in the posterior axillary line.

With such a placement of electrodes on the electrocardiogram, in the position of the switch on the first standard drain, the discharge "Dorsalis" (D) is recorded; the second - registers the allocation "Anterior" (A), the third - "Inferior" (I).

The system of heavily drafts allows us to clarify the changes in the ECG, which are observed in damage to the lateral and posterior walls of the left ventricle.

**Discharge V3R-V6R,** active electrodes which are placed on the right half of the chest, are used to diagnose hypertrophy of the right heart and focal changes in the psoriasis[12, 13].

**Proliferation for Slapak-Partil:** a kind of special bipolar thoracic dilutions with a high position of electrodes. For registration of an electrocardiogram, an electrode with the left arm should be installed on the posterior axillary line at the level of the apical impulse, the electrode on the right arm - in the second intervertebral column on the left side along the abdominal line (lead S1), on the median-clavicular line (S2), then this electrode successively move in the direction to the left shoulder at 1.5-2cm(lead S3,S4).The left hand electrode remains at the starting point - the projection of the apical impulse on the posterior axillary line.The ECG is written in the first

position of the assignment switch. ECG registration for Slapak-Partil has advantages in diagnosing myocardial infarction of the back-basal localization [12, 13].

### **Method of registration of an electrocardiogram**[12, 15]

ECGs are registered in a room remote from possible sources of electrical interference: physiotherapeutic and X-ray rooms, distribution boxes, etc. The couch should be at least 1,5-2 m from the wiring of the power grid. ECG recording is performed after a 10-minute rest period and not earlier than 2 hours after food, usually in the patient's position lying on his back with a quiet breath. Four lamellar electrodes are applied to the inner surface of the legs and the forearm in the lower third of them using rubber belts or special clamps, and one or several (with multichannel recording) chest electrodes are mounted on the chest, using a rubber piston-sucker or disposable glutinous electrodes that are glued. To improve the contact of the electrodes with the skin and to reduce the obstacles and guiding currents in the places of application of electrodes, it is necessary to degrease the skin alcohols in advance and cover the electrodes with a layer of a special conductive paste that allows the intermediate electrode resistance to be reduced as much as possible [12, 13].

Each electrode, installed on the limbs or on the surface of the chest, is connected to the wire coming from the electrocardiogram, marked with a certain color. The following is commonly used for marking electrodes: **the right hand is red; left hand - yellow; left leg - green; right leg (grounding of the patient) - black color; thoracic electrode – white** [12].

In the presence of a 6-channel electrocardiogram, which allows simultaneously to register the ECG in 6 chest leads, a wire with a red marking of the tip is connected to the electrode V1; to electrode V2-yellow, V3-green, V4-brown, V5-black and V6-blue or violet. The marking of the remaining wires is the same as in single-channel electrocardiograms [12].

Before starting an ECG record, you must **set the same electrical signal strength (control milivolt)** on all channels of the electrocardiogram. For this, in each electrocardiograph, the possibility of supplying a galvanometer with a standard calibration voltage equal to 1 mV is provided. The gain of each channel is selected so that the voltage of 1 mV causes the deviation of the galvanometer and the recording system, equal to 10 mm. To do this, in the position of the switch selector "0" regulate



the gain of the electrocardiogram and record the calibration millivolt. If necessary, you can change the gain: decrease with a very large amplitude of the ECG wave (1 mV=5 mm) or increase with a small amplitude (1 mV = 15 or 20 mm)[12, 13].

In modern electrocardiographs, automatic gauge amplification is provided.

#### **ECG recording algorithm[12, 15]**

- Record a control millivolt, the standard of which confirms the serviceability of the equipment.
- Standard millivolt has an amplitude of 10 mm and straight corners.
- The line of recording should not be thicker than 1 mm, there should be no cues.
- The standard list is 12 entries: three standard, three reinforced, and 6 chest.
- At least 3 cardiac cycles are recorded for each withdrawal.
- The ECG must be labeled with the common label: I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, V5, V6.
- There must be the date and time of recording, marked age, gender, predicted diagnosis.
- The standard recording speed is 50 mm/s, recording is possible at 25 mm/s, 100 mm/s or more, but then the recording speed should be indicated on the film.

#### **Basic rules for designating ECG elements[12, 15]**

- The wave with an amplitude of 5 mm and more are indicated by the capital letters of the Latin alphabet, and less than 5 mm are capitalized.
- Intervals and segments are calculated with accuracy up to 0.01 s.
- For the Q wave take the first negative wave of the ventricular complex, preceding the wave R; the rest of all negative waves are affected by the wave S.
- The wave R is always positive, and the wave Q and S are negative.
- If the wave R or S are multiple, then the further ones are indicated by numbers near and above the wave. For example: qRsr<sup>1</sup>s<sup>1</sup>.
- When decoding the wave P and T, the sign (+, - + -, - +), amplitude, form is taken into account.
- The segment ST is taken into account with respect to the isoline: on the isoline, higher or lower than the isoline, by how many millimeters.

**ECG analysis protocol.**The protocol for ECG analysis usually consists of 4 parts: the passport part, the calculation data, the ECG description and the clinical and electrocardiographic conclusion.

**A. Passport part.**Surname, name, patronymic, gender, age of the patient is indicated; anticipated diagnosis; date and time of the recording of the ECG.

**B. Calculated part.**Information is given on the duration of the main wave and intervals (Fig. 4), the results of calculating the formulas of hypertrophy of the heart, and others.The calculation of the duration of the main wave and intervals is usually carried out according to the II standard withdrawal, but if in this withdrawal the wave and intervals are badly expressed, one can count them on another assignment.

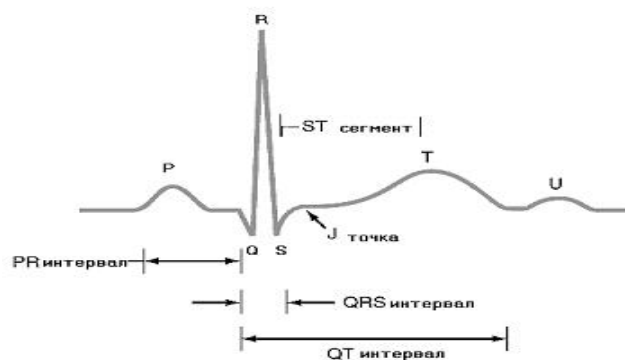


Fig.4. Elements of the ECG.

Calculated:

- wave P (from the beginning of the wave to its end; in healthy individuals it is within 0.06 - 0.1 sec);
- interval PQ (from the beginning of the wave P to the beginning of the Q or R wave in the absence of Q; in healthy individuals ranges from 0.12 to 0.20, depends on the heart rate, reflects atrial-ventricular conductivity);
- QRS complex (normally ranging from 0.06 to 0.11 sec, reflecting intravaginal conductivity);
- QRST interval (from the beginning of the wave Q to the end of the T wave, reflects the electric systole of the heart, norms are calculated according to special formulas, in healthy individuals usually ranges from 0.36 to 0.42 seconds).
- interval R - R;
- heart rate (heart rate). Heart rate =  $60/RR$ .

- the ratio of the wave of the QRS complex in the standard leads is determined (the Einthoven formula);
- the position of the electric axis of the heart is determined.
- calculate other indices (hypertrophy of the heart organs, etc.).

### **B. Descriptive part[12]**

The wave and intervals in all 12 ECG leads are described.

- P wave: Normally, its amplitude ranges from 0.5 to 2.5 mm; it is positive in all leads, except aVR and sometimes V1. The record is made in the form of a formula. For example, P I, II, III, V1-V6+
- QRS ventricular complex. In general, V1 has the formula rS, V5-6 is the formula qR, the transition zone is V3. The record is made in the form of formulas: V1 - rS, V5 - qR, transition zone - V3.
- Particularly, the wave Q is analyzed. For healthy individuals, it should not exceed 1/3 of the tone R, with which it is recorded, and should not be longer than 0.04 s (except aVR). **This is the most significant place in the ECG, because Q, exceeding the norm, is a necrosis tuck!** When a pathological tone Q is detected, it is denoted by a large letter and an exclamation point is placed, for example: QV4-5 (!). If the ventricular complex is represented by one negative tone, it is denoted by QS.
- Segment ST. Normally located on isolation or deviating from it no more than by + -0,5 mm. **This is the second most significant place in the ECG because the deviation of the ST segment from the isolation is characteristic of myocardial damage.**

Sample record: ST I, II, III, V<sub>1-6</sub> on isolation.

- The wave T in healthy individuals is positive (with the exception of aVR), unequivocal with a rounded vertex. The appearance of negative equilibrated sharp (coronary) wave T is characteristic for myocardial ischemia (ischemic wave).

Sample recording: T I, II, III, V<sub>1-6</sub>+

### **G. The final part (conclusion)[12]**

The electrocardiographic conclusion indicates:

- the main driver of the rhythm in the sinus or non-sinus (which is exactly);
- regularity of heart rhythm: correct or wrong;



Fig.5. Characteristics of ECG elements (scheme).

**P wave.**- Atrial Complex, reflecting the process of depolarization of the atrium. The first 0.02-0.03 sec, the wave of excitation spreads only to the right atrium, the further 0,03-0,06 sec - simultaneously on both atriums. In the final 0.02-0.03 sec, excitement extends only to the left atrium, since the entire myocardium of the right atrium is already in an excited state. The polarity of the wave P in the leads P I, II, aVF, V3-V6 is always positive. The pin R aVR is always negative. The pin P III can be positive, biphasic or negative at the horizontal position of the electric axis of the heart. The pin P aVL is positive, two-phase or negative in the vertical electric position of the heart. The pin V V1 is more likely to be two-phase, it can be registered in the form of a low positive pin. Occasionally, the same polarity has a prick P V2. The amplitude of the wave P is 0.5-2.5 mm. Its duration does not exceed 0.1 sec (varies from 0.07 to 0.1 sec).

**PQ segment.**Excitation of the atrioventricular joint, the His bundle, the bundle of the His beam, Purkinje fibers creates a very small potential difference, which on the ECG is represented by an isoelectric line, located between the end of the wave P and the beginning of the ventricular complex. The PQ interval corresponds to the time of propagation of excitation from the sinus node to the contractile ventricle myocardium. This index includes the P wave and segment PQ and is measured from the beginning of the wave P to the beginning of the ventricular complex. The duration of the interval PQ is normally 0.12-0.20 sec (up to 0.21 seconds for bradycardia) and depends on the frequency of heart rate, increasing with shuffling the sinus rhythm. An increase in the interval PQ of more than 0.20 (0.21 sec) is characterized as a violation of the conductivity A.

**Complex QRS** - ventricular complex, is formed in the process of depolarization of the ventricles. For greater clarity of the explanation of the origin of the individual wave of this complex, the continuous process of excitation along the ventricles can be divided into 3 main stages.

**The initial stage of depolarization of the ventricles** corresponds to the first 0.02-0.03 sec proliferation of ventricular myocardial excitation and is due, primarily, to excitation of the interventricular septum, and, to a lesser extent, to the right ventricular myocardium. The total (momentary) initial vector is directed to

the **right and forward** and has a small value. The projection of this vector on the drive axis determines the direction and magnitude of the initial wave of the ventricular complex in most electrocardiographic distributions. Since the initial moment vector of the depolarization of the ventricles is projected onto the negative parts of the axes of leads I, II, III, aVL, aVF, then in these leads a small negative deviation is recorded – the **wave q**. Its direction is from the electrodes  $V_5 - V_6$  also explains the appearance of a small **q in these leads**. Simultaneously, the vector oriented from the electrodes  $V_1 - V_2$ , where he formed a small action initial positive amplitude **wave R** [12, 13].

**The main stage of depolarization of the ventricles** corresponds to a further 0.04-0.07 sec, when excitation is distributed along the free walls of the ventricles. The total head vector is directed from right to left, correspondingly to the orientation of the total vector of the more powerful left ventricle. The projection of the main vector on the drive axis defines the main ventricle of the ventricular complex in each of them. The vector is projected into positive portions of the axes I, II, III, aVL, aVF leads, where the **wave R** and the negative part of the aVR are formed, which leads to the simultaneous registration of the **wave S**. The main moment vector is oriented to the electrodes  $V_5 - V_6$  and causes the formation positive wave – the **wave R**. The same vector has a direction from the electrodes  $V_1 - V_2$ , so at the same time period in them a negative wave is formed – the **wave S** [12, 13].

**The final stage of depolarization of the ventricles.** The process of depolarization of the ventricles ends with the spread of excitement on their basal divisions. This happens at 0.08-0.10 seconds. The total (momentary) terminal vector has a small value and varies considerably in direction. However, more often it is oriented right and back. In a number of leads from the limbs, in the leads  $V_4 - V_6$  under its action, formed terminal negative wave - the wave S. In leads  $V_1 - V_2$  his vector, merging with the main, contributes to the formation of deep wave S. Thus, the same electrical processes, recorded simultaneously with the propagation of excitations in the ventricles, in different directions can be represented by the wave of different polarity and magnitude. This is determined by the projection of the corresponding moment vectors on the drive axis. In other words, depending on the position of the electrodes, the wave reflecting the initial, main and final stages of depolarization of the ventricles may have different directions and different amplitudes [12, 13].

With the amplitude of the Q wave of the ventricular complex, exceeding 5 mm, it is indicated by a large letter. If the amplitude of the wave is less than 5 mm - small. QRST ventricular complex reflects the process of spreading and stopping excitation in the ventricular myocardium [12].

**Q wave.** The first negative wave of the ventricular complex. The ventricular complex may have only one Q. It can be registered in leads I, II, III, aVL, aVF, aVR. Its presence is mandatory in leads  $V_4$ - $V_6$ . The presence of this wave in the leads  $V_1$ - $V_3$  is a sign of pathology. Criteria for normal Q: duration not more than 0,03 sec; the depth is not more than 25% of the amplitude of the wave R in the same cut (except for the allocation aVR, which can normally register a complex of the form of QS or Qr). Caution should be taken when evaluating Q in QIII. The pathological nature of the Q-wave is likely to be probable if it is accompanied by a deep Q-II and Q-aVF duct exceeding 25% of the wave R. When inhaling breath, the Q-wave of the throat, connected with the transverse location of the heart, disappears or decreases [12].

**R wave-** any positive wave of the ventricular complex. In the presence of several positive waves, they are designated, respectively, as R, R', R, etc. R can have a very small amplitude in the leads aVR, aVL (at the vertical position of the electric axis of the heart) and in the release  $V_1$ . In this ventricular complex looks like QS. The amplitude of the wave R does not exceed 20 mm in the leads from the extremities and 25 mm thoracic. In practical electrocardiography, the ratio of the amplitudes of the wave R in different directions is often more important than its absolute value. This is due to the influence of extracardiac factors on the amplitude characteristics of the ECG (emphysema, obesity). The ratio of the height of the wave R in the leads from the extremities is determined by the position of the electric axis of the heart. In the thoracic nodes in normal, the amplitude of the wave R is gradually increasing from  $V_1$  to  $V_4$ , where its maximum height is usually recorded. From  $V_4$  to  $V_6$ , there is a gradual decrease in the amplitude of the wave R. The dynamics of the amplitude of the wave R in the chest leads can be described by the formula:  $R_{V_1} < R_{V_2} < R_{V_3} < R_{V_4} > R_{V_5} > R_{V_6}$ . [12].

**S wave** is a negative wave that follows the wave R. The wave S can be several and then they are designated as S', S, etc. If the ventricular complex is represented by one negative wave (in the absence of a wave R), it is denoted as QS. S wave is a

non-permanent wave of the ventricular complex. It has the maximum amplitude in  $V_1$  or  $V_2$  and gradually decreases to the  $V_5$ - $V_6$  directions (which may be absent normally) [12].

The ratio of the wave S in the chest leads is the formula:  $SV_1 > SV_2 > SV_3 > SV_4 > SV_5 > SV_6$ . In the leads from the extremities, the presence and depth of this wave depend on the position of the electric axis and the turns of the heart. Typically, in these leads, the amplitude of the wave S does not exceed 5-6 mm. Its width is within 0.04 mm. The described dynamics of the R and S wave in the chest leads corresponds to a gradual increase in the ratio of the R/S amplitudes from the right leads, where it is  $< 1.0$ , to the left, in which the ratio is  $> 1.0$ . Breast withdrawal with equal amplitudes of the wave R and S ( $R/S = 1.0$ ) is called the transition zone. Often in healthy people this is a  $V_3$  release [12].

**The total duration of the QRS complex**, representing the time of intraventricular conduction, is 0.07-0.1 sec. Equally important for intraventricular conductivity is ventricular activation time or **intrinsicoid deflection (ID)**. Indicator characterizes the time of excitation from the endocardium to the epicardium of the ventricle wall located below the electrode. The internal deviation is determined for each ventricle separately. For the right ventricle, this index (IDd) is measured at the  $V_1$  distance from the beginning of the ventricular complex to the vertex of the wave R (or the vertices of the last wave R in the RSR complex). Normally IDd = 0.02-0.03 sec. Internal deviation for the left ventricle (IDs) are evaluated in  $V_6$  at a distance from the beginning of the ventricular complex to the vertex of the wave R (or the vertices of the last wave R during its cleavage). Normally IDs = 0.04-0.05 seconds. [12].

**Segment ST** is a line from the end of the ventricular complex to the beginning of the T wave. It corresponds to the period of full coverage of ventricular myocardial arousal. At the same time, the potential difference in the cardiac muscle is absent or very small. Therefore, the ST segment is isolated or slightly biased toward it. In the leads from the extremities and the left thoracic leads, normalization of the ST segment shift downwards and upwards from the isoline is not more than 0.5 mm. In the right chest leads it is possible to shift it upwards by 1.0 to 2.0 mm (especially at high T-waves in the same leads). The downward movement of the ST segment in the left thoracic nodes is not normal [12].



**T-wave** shows the process of rapid end-to-end ventricular myocardial repolarization. The total vector of ventricular repolarization, extending from subepicardial layers to subendocardial, has the same direction as the principal moment vector of depolarization. In this regard, the polarity of the wave T in most leads coincides with the polarity of the main wave of the QRS complex [12].

Normally, the wave T I, II, aVF,  $V_3$ - $V_6$  is always positive, the T aVR wave is always negative. The T-wave in the III lead can be positive, two-phase and even negative at the horizontal position of the electric axis of the heart. T aVL is both positive and negative - with the vertical position of the axis of the heart.  $TV_1$  (less  $TV_2$ ) can be either positive or biphasic or negative. It is asymmetric, has a smoothed top. The amplitude of the T wave in the leads  $V_5$ - $V_6$  is 1/3-1/4 of the height of the wave R in these leads. In retraction  $V_4$  ( $V_3$ ), it can reach 1/2 of the amplitude of the wave R. Usually in the leads from the extremities, it does not exceed 5-6mm, in the chest - 15-17 mm [12].

**The QT interval** is an electric systole of the ventricles, which depicts depolarization and ventricular repolarization processes. This indicator is measured from the beginning of the ventricular complex to the end of the T wave. The duration of the QT interval is influenced by the heart rate, the patient's weight, which is taken into account when evaluated [12].

The proper value can be calculated according to Bazett's formula:  $QT = k \cdot \sqrt{RR}$ , where k is the coefficient equal to 0.37 for men and 0.40 for women. A proper QT value corresponding to the given heart rate and the patient's gender can be established on a special nomogram. The QT interval is considered normal if its actual value does not exceed its value by more than 0.04 seconds [12].

**U wave.** There is no single thought about the origin of this ECG trace. The appearance of it is associated with the potential arising from the stretching of the ventricular myocardium during the period of rapid filling, with the repolarization of the papillary muscles, Purkinje fibers. This is a small positive amplitude wave that goes through 0.02-0.03 sec per wave T. Often it is registered in leads II, III,  $V_1$ - $V_4$  [12].

**T-P interval.** This isoelectric line, which serves as the starting point for determining the interval PQ and segment ST.

**RR interval**- the duration of one heart cycle in seconds. The duration of the heart cycle is measured between the vertices of R in two adjacent complexes. The rhythm is considered correct if the fluctuation of the RR interval in different cycles does not exceed 10%. Usually measure 3-4 intervals, of which the average value is written. For the calculation of heart rate (HR) at regular rhythm use the formula: heart rate =  $60/RR$ , where 60 - number of seconds per minute. There is a special table, which specifies the duration of RR and, accordingly, the frequency of heart rate[12].

### **Electric heart axis[12, 13]**

The genesis of normal ECG, the origin and nature of its pathological changes most clearly explains the vector theory of cardiac dipole. The electromotive force (EMF) of a dipole (a cell) can be conventionally represented as a vector, that is, a straight line with an arrow directed from the negative to the positive pole of the dipole. The value of the segment of a straight line, taken in scale units, reflects the value of the EMF, and the orientation is direction. Under certain assumptions, the heart can be considered as one point source of current - the total single heart dipole, which produces a total EMF. In strictly consistent distribution of myocardial excitation, the total EMF consistently and naturally changes in magnitude and direction. Each individual moment of the heart cycle corresponds to its own total momentum EMF. In this case, two unidirectional vectors are summed up, with a divergent value of the EMF decreases, and the vector of the EMF is directed toward the largest one. If two vectors are directed to each other at an angle, the resulting vector is a diagonal of the parallelogram. If two vectors are equal in magnitude, directed to the opposite sides, then their sum is equal to zero. When depolarizing the ventricles of the heart formed an integral vector, which Einthoven called the electric axis of the heart (EBC). The electric axis of the heart is directed from the base of the heart to its top[12].

In order to facilitate understanding of the change in the electric axis of the heart in standard leads, Einthoven also introduced the concept of the angle of the alpha. The angle of the alpha is the angle formed between the line of the first lead and the electric axis of the heart. Normally, the alpha angle is in the range from zero to 90 degrees (Figure 6). With pathology, the angle of the alpha can go beyond these limits.



Fig.6.Electric Heart Ax.

**Determination of the electric axis of the heart by the algebraic sum of the wave of the QRS complex(Fig. 7):**

- the algebraic sum of the wave R and S in the first standard release is applied to the axis of the first drain;
- the algebraic sum of the wave R and S in the third standard release is applied to the third axis;
- perpendiculars from the points obtained;
- The line drawn from the center of the six-axis coordinate system to the point of intersection of the perpendiculars is the electric axis of the heart; its direction is determined in a circle divided by degrees.

The direction of the electric axis of the heart approximately corresponds to the direction of the largest total vectors depolarization ventricles.

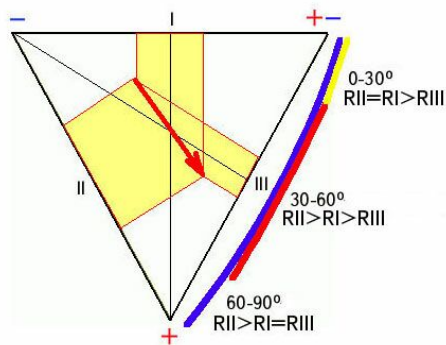


Fig.7. Normal type of ECG.

Depending on the position of the electric axis (integral vector), three main types of electrocardiogram are distinguished on the front plane: normal type (angle of alpha

from 0 to 90 degrees), left type (angle of alpha less than zero degrees) and right type (angle of alpha more than 90 degrees).

As can be seen from the diagram, the normal electric heart axis (integral vector) can be in the range from 0 to 90 degrees, and in the case of a normal type, the angle of the alpha varies from 30 to 60 degrees. The position of the vector in the range from 0 to 30 degrees is called a horizontal tendency, and from 60 to 90 degrees - the vertical trend[12, 13].

### **Visual determination of angle $\alpha$ [12]**

The method is based on two principles:

- the maximum positive (or negative) value of the algebraic sum of the wave of the QRS complex is recorded in the electrocardiographic excision whose axis approximates to the location of the electric axis of the heart, and the resulting resulting QRS vector is postponed to a positive (or, correspondingly, negative) part of the axis of this lead;
- A complex of type RS, where the sum of algebraic wave is equal to zero ( $R = S$  or  $R = Q + S$ ), is written in the withdrawal, whose axis is perpendicular to the electric axis of the heart.

When visualizing the position of the EMU, it is necessary to find a lead in which the highest wave R (with minimum or no wave S).

At the maximum wave RI - determine the horizontal position of the EBS (angle  $\alpha = 0^\circ$ ).

At the maximum wave R in aVL - EBS is rejected to the left (angle  $\alpha = -30^\circ$ )

Maximum wave R in II release - EVS has normal position (angle  $\alpha + 60^\circ$ ).

The maximum R to produce aVF is the vertical position of the EBS (angle  $\alpha + 90^\circ$ ).

The maximum torque R in retraction III - EVS is set to the right (angle  $\alpha + 120^\circ$ ).

If the maximum R in aVR - the position of the EMU is impossible to determine.

### **Criteria for a normal electrocardiogram(Figure 8)[12, 13, 15]**

- Rhythm is sinusoid.
- The wave and ECG intervals are within normal limits.
- Heart rate (HR) in the range of 60-100 per 1 minute.
- The ECG type is normal, the alpha angle is between 0 and 90 degrees.

- There are no signs of rhythm disturbance, conduction.
- There are no signs of hyperfunction, hypertrophy of the heart.
- There are no metabolic disorders, signs of ischemia, damage, necrosis.

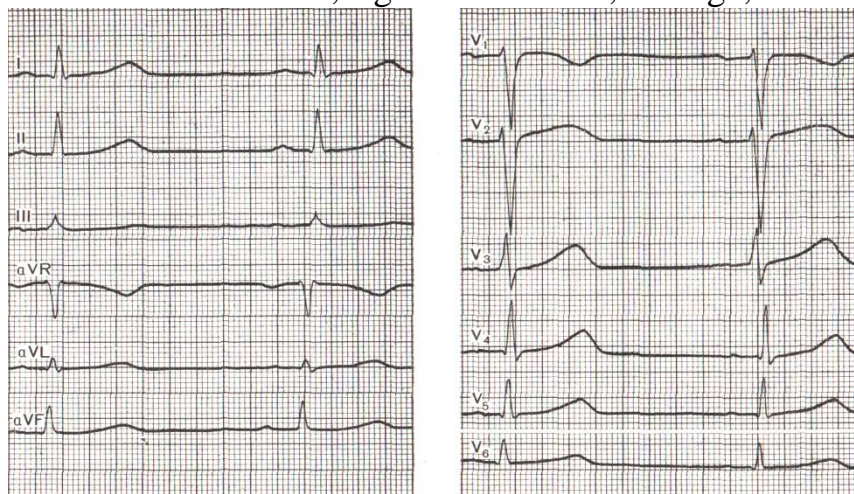


Fig.8. Normal ECG

#### **ECG criteria for sinus rhythm [12, 13]**

- Before QRS complex is positive P wave in all leads except aVR, where negative P wave (sometimes V<sub>1</sub> wave R may be biphasic or negative).
- PQ interval is not less than 0.12 seconds.
- Intervals of RR are equal to or vary by no more than 10%.
- At normal frequency, heart contractions are in the range of 60 to 100 per min.

### **ELECTROCARDIOGRAM IN RISK AND CONSEQUENCES OF DISEASE [12, 13, 14, 15]**

Arrhythmias include changes in the frequency, sequence, or force of heart contraction. Electrophysiological causes of arrhythmias [15]:

- Changing the automatism of the sinoatrial node.
- The emergence in any section of the myocardium of a source with increased activity, which can produce pulses to reduce heart rate.
- Violation of conduction pulses from the atrium to the ventricles.

**Sinus tachycardia** is an increase in cardiac activity in a state of rest of up to 90 - 150-160 in 1 minute with the correct rhythm, when the rhythm driver has a sinus node.

### **Mechanism of sinus tachycardia[15]:**

- Increased sympathetic tone (increased production of catecholamines).
- Reduced tone of the vagus nerve (depression of acetylcholinergic effect).
- Influence on the cells of the sinus node (hypoxia, acidosis, elevated body temperature, infection).

The most important diseases that cause sinus tachycardia: rheumatic heart disease, myocardial infarction, thyrotoxicosis.

Sinus tachycardia is more common in people with a healthy heart, it refers to neurogenic tachycardia with a disturbance of the balance of the tone of the autonomic nervous system with a sympathetic advantage and a weakening of the tone of parasympathetic inertia.

### **ECG - criteria for sinustachycardia[15]:**

- Wave R. sinus origin.
- Permanent normal interval P-P with a duration of 0.12-0.2 seconds.
- Reduced RR intervals (less than 0.60 s) due to reduced diastolic intervals of TP.
- There are no complaints or: heartbeat, feeling of heaviness or pain in the heart.
- Slow down the rhythm when irritating the vagus nerve - pressing on a sinocaroid sinus, a Valsalva test.

**Sinus bradycardia** is a slowdown in the heartbeat rhythm (less than 60 beats per minute). The driver of the rhythm remains the sinus node. Sinus bradycardia is rarely less than 40 beats per minute.

Mechanism of sinus bradycardia: tone of the vagus nerve, tone of the sympathetic nerve and direct action on the cells of the sinus node. The cause of sinus bradycardia can be physiological, vagal bradycardia, toxic and medication bradycardia and bradycardia in myocardial pathology[12, 13]. Often it is such diseases as: myocardial infarction, coronary atherosclerosis, rheumatic carditis.

Complaints with sinus bradycardia are absent, sometimes it is palpitations, dizziness, syncopal conditions. Objectively, the correct rhythm (less than 60 beats per minute) is determined, the force of heart tones is not changed. Very pronounced arrhythmia can cause cerebral ischemia with syncope phenomena[13].

### **ECG signs of sinus bradycardia[15]:**

- Wave P of sinus origin.

- Intervals R-R to 0,20 s and more.
- Heart rate is less than 60 beats per minute.

**Sinus arrhythmia** is characterized by an intermittent increase in frequency and number of abnormalities due to the uneven generation of pulses of excitation in the sinus node itself. Respiratory arrhythmias depend on the phase of breathing. It is caused by reflex changes in the tone of n.vagus and n.sympaticus due to the phase of breathing. This occurs in the paths of several reflexes that affect the function of the sinus node during respiration[15].

**Reflection Bainbridge.** During the inspiration, a large influx of blood into the heart increases pressure on the baroreceptors of the right atrium and hollow veins. This leads to stimulation of the sympathetic nerve and acceleration of the heart rate. Exposure leads to the reverse effect[12].

**Goering Brayer Reflex.** During the inspiration, the end of afferent nerve fibers is irritated and the vagus nerve is suppressed, which leads to more frequent cardiac contractions. The main diagnostic criteria for respiratory arrhythmia is the detection of a connection with the breathing and disappearance phases after respiratory depression or the use of atropine[12].

**Extrasystolic arrhythmias** is one of the most common cardiac arrhythmias. In healthy persons, extrasystole is functional and can be provoked by various vegetative reactions, smoking, taking alcohol, coffee. Extrasystoles of organic origin are more serious in the prognostic plan. Their appearance indicates profound changes in the heart muscle in the form of foci of ischemia, dystrophy, necrosis or cardiosclerosis. Most often, extrasystole is observed in acute myocardial infarction, coronary artery disease, rheumatic heart defects, myocarditis. Extrasystoles - this is an extraordinary excitation of the heart, due to the following mechanisms [12].

**I. Re-entry** – the main mechanism for the emergence of extrasystole. Circulating wave of myocardial excitation and fiber of the conduction system of the heart. Necessary conditions for its occurrence[15]:

- At least two ways of holding.
- The appearance of a one-sided block in one of the ways.
- Slow down of another path.

- Retrograde return of excitation by blocked before this path to the point of depolarization. The emergence of this mechanism is possible in the conditions of the anatomical base (**macro re-entry** with additional paths) and functional myocardial heterogeneity (**micro re-entry**).

**Criteria**[12, 15]:

- Called and removed by programmable electrical stimulation.
- Reciprocal ratio between the interval between the electrical stimulation and the interval to the first ECG complex.
- Ability to depolarize both ventricles with the help of electrical stimulation of the heart during ventricular tachycardia without its termination.
- Ability to increase the heart rate during paroxysm tachycardia with programmable stimulation.

**II. Ectopic automatism.** The emergence of rhythmic spontaneous depolarization of cell membranes during diastole (at the highest rate of spontaneous depolarization of sinus node cells as the dominant driver of the rhythm). Fibrillation, AV connection, bundle branch block, fiber Purkin'ye - latent pacemakers and can activate its function in terms of reducing depolarizing role sinus (vago-tonia) while increasing the speed of spontaneous activation of latent pacemakers (ischemia , acidosis, hypokalemia, toxic effects of cardiac glycosides, etc.). Pathological myocardial cell automatism occurs near the ischemia zone due to increased concentration of extracellular potassium, increased local release of catecholamines, etc.[15, 16].

**Criteria**[15]:

- Tachycardia is not provoked or stopped by a programmable electrical stimulus.
- Tachycardia can be provoked by frequent stimulation at a frequency of stimuli equal to the frequency of ventricular tachycardia.
- Extra stimulus on paroxysm leads to a complete compensatory pause.
- Infusion of catecholamines leads to tachycardia.

**III. Trigger activity.** Ectopic excitation when generating oscillatory trace potentials. It differs from ectopic automation in the absence of a spontaneous phase of depolarization and the onset of the functioning of the focus of automatism only after extrastimulus or frequent stimulation[15, 16].



High-amplitude stimuli may exceed the threshold potential, cause a reduction in myocardium. Postpotential enhancement occurs when overdose of glycosides, the administration of catecholamines, and frequent stimulation. Potassium salts, reducing the amplitude of trace potential, lead to therapeutic effect.

**Criteria[15]:**

- The emergence of catecholamine tachycardia.
- Electric stimulation with premature extramasses - with the reduction of the interval of adhesion, the interval between stimulus and ventricular response decreases.
- Gradual increase in the frequency of ventricular contractions in paroxysm.
- With an increase in the number of premature extramutations, the number of causes of myocardial contractions increases.

With the development of individual areas of the heart ischemia, dystrophy, necrosis, the electrical properties of different parts of the myocardium and the conduction system of the heart can differ significantly from one another. There is a so-called electrical non-homogeneity of the heart muscle, which manifests itself at an uneven rate of conducting an electrical impulse in different parts of the heart and the development of unidirectional blockades of conduction. At the same time, some parts of the heart will be excited by an unusual way, but with a great delay, when all other parts of the heart managed to get out of the state of refractoriness. In this case, the excitation of this area can re-spread to the areas of the heart before they reach the pulse of the sinus node[12, 13].

**Distinguish the extrasystole of the atrial, with AV-connection and ventricular[12, 15].**

Early extrasystoles are aligned on the wave T or are from the T wave not more than 0.04 s. The distance from the extrasystole to the next cycle is called a compensatory pause. There are incomplete and full compensatory pauses[15, 16].

If the extrasystole occurs in the atrial or AV-connection, the ectopic pulse extends not only to the ventricles, but also to the retrograde at the atria. Having reached the sinus node, the ectopic impulse discharges it, so the pause, which is after the extrasystole, includes the time "discharge" of the sinus node, as well as the time of preparing for it another sinus pulse[15].

With the ventricular extrasystoles, the ectopic pulse does not reach retrograde to the atrium. In this case, the sinus pulse stimulates the atrium, passes through the AV node, but can not cause ventricular excitement, because they are in a state of refractoriness. Conventional ventricular excitement will occur only at the expense of the next sinus pulse[15].

The distance between the normal QRS complex before the extrasystole and the normal QRS complex after the extrasystole is equal to the doubling RR interval and indicates a complete compensatory pause[15].

Extrasystoles can be monotonous, originating from one part of the myocardium, and polythymic - from different parts of the myocardium.

#### **ECG signs of extrasystoles[15, 16]:**

##### **Ancestral extrasystoles**

- Extraordinary appearance of the wave P and the QRS complex.
- Deformation or change in the polarity of the wave P of extrasystoles.
- Unchanged QRS extrasystoles complex.
- Having an incomplete compensatory pause after the atrial extrasystoles.

##### **Nodes of extrasystole**

- Extraordinary appearance of an ECG of the unchanged QRS complex.
- Negative wave P is absent or there is a negative P wave after the QRS complex.
- The presence of an incomplete compensator pause.

##### **Ventricular extrasystoles**

- Extraordinary appearance of the modified QRS complex.
- Expansion and deformation of the extrasystolic complex QRS.
- The location of the RS-T segment and the extrasistal T-axis is discordant to the direction of the main line of the QRS complex.
- The absence of a pre-existent throne R.
- Having a complete compensatory pause after the ventricular extrasystoles.

#### **Classification of ventricular extrasystoles**

**(B.Lown, M.Wolf, 1971)[15]:**

- 0 - there are no ventricular extrasystoles;
- 1- years of extrasystoles (less than 30 per hour);
- 2- frequent extrasystoles (more than 30 per hour);

3- polymorphic ventricular ectasy stalks;

4a- Parents Extrasystoles;

4b- short episode of ventricular tachycardia (three and more complexes);

5- early ventricular extrasystoles.

### **Paroxysmal tachycardia[15, 16]**

Paroxysmal tachycardia is an attack of increased cardiac activity of the correct rhythm (more than 140 per minute), with a sudden onset and end. The origin of paroxysmal tachycardia is explained by the theory of "unit ectopic center". An ectopic focus in the heart muscle is the result of a change in cellular metabolism, biochemical and electrolyte impairment[12, 13].

Transitions of one arrhythmia to another depend on the frequency of impulses that emanate from the ectopic focus. An important mechanism is the re-entry wave re-entry[12, 15].

These mechanisms are not by accident similar to the mechanisms of the emergence of extrasystoles, since the attack of paroxysmal tachycardia can be considered as a long series (one for the second) of the extrasystole, the source of which is the ectopic center located in the atria, AV node or ventricles[15].

### **ECG signs of atrial paroxysmal tachycardia[15]**

- Heart beat up to 140-250 per minute while maintaining the correct rhythm.
- Presence in front of each ventricular complex of reduced, deformed, biphasic or negative wave R.
- Normal unchanged QRS complexes.

**Reciprocal AV-node tachycardia.** Predefined mechanized IOM re-entry (retrograde P wave often than not you are denoted, coinciding with the complex QRS, sometimes recorded after the QRS short intervals RP (RP < 50% RR). Momentum is anterograde to slow way and retrograde bottom - Quick, atrium and the ventricles are excited simultaneously[12, 14].

**An orthodromic tachycardia** occurs with the presence of an additional pathway (WPW syndrome) with an anterogenic pulse through an AV node on the ventricles with subsequent retrograde returns through an additional pathway to the atrium. Registered retrograde wave P with a short interval of RP (RP < 50% RR),

negative P to I excretion, delta wave is not recorded, since ventricular activation goes through the AV region[12, 15].

**Antidromic tachycardia** occurs rarely and with the presence of a significant additional pathway (WPW syndrome) with an impulse inflow anterograde through an additional pathway to the ventricles with subsequent retrograde returns through the AV node to the atrium, occasionally, the anterograde wave P, necessarily delta wave, are registered, therefore that the activation of the ventricles goes through an additional pathway, the ECG is similar to the ventricular tachycardia[12, 15].

**Nodular paroxysmal tachycardia[15, 16]**

- A sudden attack of tachycardia to 140-220 per minute.
- Presence of leads II, III, avF of P-wave, located behind QRS complexes, or overlapped by QRS.
- Normal QRS complexes.

**Atrial flutter** is a frequent atrial contraction of up to 200-400 in 1 minute while maintaining the correct regular atrial rhythm[12].

Atrial flutter is observed with organic changes in the atria of the myocardium and is due to the presence of electrical inhomogeneity there. At a young age, this happens in patients with rheumatism, myocarditis, mitral heart disease, and in the elderly - with CHD, MI.

At the heart of this arrhythmia lies the mechanism of increasing the automatism of the cells of the leading atrial system or the mechanism of re-entry of the wave of re-breathing (re-entry).

**ECG-signs of atrial flutter[15, 16].**

- Presence of frequent (up to 200-400 per minute) regular, similar atrial waves of the electrocardiogram, which has a characteristic ankle shape.
- Same regular ventricular rhythm.

**Atrial fibrillation-** frequent, from 350 to 700 per minute, chaotic arousal and reduction of individual atrial muscle fibers, each of which is in fact an ectopic pulse center, is observed throughout the entire cardiac cycle. In this case, the excitation and reduction of the atrium, as a whole, is absent. Not all of these chaotic excitations can pass through the AV node, as many of them find the ventricles in a state of

refractoriness. Flicker arrhythmia is observed in coronary artery disease, mitral stenosis, thyrotoxicosis [12, 15].

In all cases, there is a significant electrical inhomogeneity of the atrium myocardium, which forms the basis of the formation of the circular motion of the wave of excitement in the atria due to the mechanism of re-entry. But in contrast to the atrial flutter, when the frequent motion of the wave of excitation passes along one and the same path, with the flashing arrhythmia, the direction of the excitation wave is constantly changing [15, 16].

At first, flashing arrhythmia can be paroxysmal, and in the future it becomes permanent.

#### **ECG signs of atrial fibrillation [15, 16]**

- Absence in all leads of the wave R.
- The presence of waves f.
- Irregularity of the ventricular complexes.

#### **Ventricular paroxysmal tachycardia [12, 14]**

- Heartbeat attack to 140-220 per minute.
- The deformation and expansion of the QRS complex is more than 0.12 s with a discondantly located RS-T segment and a T wave.
- Presence of atrioventricular dissociation, different rhythm of the ventricles and atria.

**Ventricular fibrillation** is often, up to 200-500 per minute, irregular, irregular excitement and reduction of individual muscle fibers of the ventricles [12].

Ventricular fibrillation arises as a result of rapid circular motion of the vibrational myocardial excitation wave through the mechanism of re-entry. The direction of motion of the wave is constantly changing, which leads to irregular chaotic excitation and reduction of individual groups of muscle fibers of the ventricles [12, 15].

#### **ECG signs of ventricular fibrillation [12, 15]**

The presence of frequent, up to 200-500 irregular waves per minute, differing in shape and amplitude from each other.

## **Hypertrophy of the heart[12, 16]**

Hypertrophy of the heart is a compensatory reaction of the myocardium, which is manifested in increasing the mass of the heart muscle. Hypertrophy develops in response to increased cardiac load in the presence of valve heart defects or with increased pressure in the large or small circle of blood circulation. Hypertrophy of the left atrium is more common in patients with mitral heart disease, predominantly with mitral stenosis[12, 16].

### **ECG signs of hypertrophy of the left atrium[12, 16]**

- Decompression and increase of the amplitude of the wave P, II, avL, V5, V6 (P-mitral)
- Increase the amplitude and duration of the second negative phase of the wave P in the release of V1 (less often V2) or the formation of a negative wave P in V1.
- Negative or two-phase (+ -) wave P III.
- Increase of the width of the wave P over 0,1 s.

Compensatory hypertrophy of the right atrium develops in diseases that are accompanied by an increase in pressure in the pulmonary artery, most often in the chronic pulmonary heart.

### **ECG-signs of hypertrophy of the right atrium[12, 16]**

- In highways II, III, avF, P high-amplitude tops (P-pulmonale).
- In V2 leads, the P wave is positive with a pointed tip (P-pulmonale).
- In the leads I, avL, V5, V6 is a low amplitude, and in avL may be negative.
- The duration of the wave P does not exceed 0.10 s.

Compensatory left ventricular hypertrophy develops in hypertension, aortic heart defects, mitral valve failure, and other diseases that are accompanied by left ventricular overload.

### **ECG signs of left ventricular hypertrophy[12, 16]**

- Increase in the amplitude of the R wave in the left chest (V5, V6) and the amplitude of the S-wave in the right chest (V1, V2). At the same time,  $R_{V1} < R_{V5}; R_{V5.6} > 25 \text{ mm}$  or  $R_{V5.6} + S_{V1} > 35 \text{ mm}$ .
- Signs of a rotation of the heart around the longitudinal axis: shifting the transition zone to the right

- Displacement of the electric axle to the left. In this case,  $R_I > 15 \text{ mm}$ ,  $R_{avL} > 11 \text{ mm}$ ,  $R_I + S_{III} > 25 \text{ mm}$ .
- Shifting the RS-T segment in leads V5,6, avL below isolation and forming a negative or two-phase (+ -) pin T in leads I, avL, V5, V6
- Increasing the length of the QRS interval in the left breast (V5, V6) for more than 0.05 s.

**Sokolov-Layon index**[12, 15]:

- $S_{V1} + R_{V5 \text{ OR } V6} > 35 \text{ MM}$ ,
- $R_{avL} > 11 \text{ mm}$ .

**Cornell criterion**[12, 15]:

- woman  $S_{V3} + R_{avL} > 20 \text{ mm}$
- men  $S_{V3} + R_{avL} > 28 \text{ mm}$ .

**Lewis's index (Gubner-Ungerleider)**[12, 15]:

- $R_I + S_{III} > 25 \text{ mm}$ .

**Framingham criteria**[12, 15]:

- $R_{avL} > 11 \text{ mm}$ ,
- $R_{V4-6} > 25 \text{ mm}$ ,
- $S_{V1-3} > 25 \text{ mm}$ ,
- $S_{V1 \text{ OR } V2} + R_{V5 \text{ OR } V6} > 35 \text{ MM}$ ,
- $R_I + S_{III} > 25 \text{ mm}$

**Criteria for the Minnesota Code (at least one)**[12, 15]:

- $R_{V5 (V6)} > 26 \text{ mm}$  or
- $R_{I (or II, III, avF)} > 20 \text{ mm}$  or
- $R_{avL} > 12 \text{ mm}$
- In the absence of a criterion:
- $15 \text{ mm} < R_I < 20 \text{ mm}$
- $R_{V5 (V6)} + S_{V1} > 35 \text{ mm}$

**Hypertrophy of the right ventricle** develops with mitral stenosis, chronic pulmonary heart and other diseases that lead to prolonged overload of the right ventricle[12, 15].

**ECG-signs of right ventricular hypertrophy**[12, 15]

- Offset of the electric heart axis to the right (angle of alpha more than 100 degrees)

- Increase the amplitude of the R wave in the right chest (V1, V2) and increase the S wave in the left chest (V5, V6). At the same time,  $R_{V1} > 7 \text{ mm}$ ,  $R_{V1} + S_{V5.6} > 10.5 \text{ mm}$ .
- Appearance in V1 assignment of the QRS type rSR' or QR.
- Moving the transition zone to the left until the V5-V6 leads.
- Offset RS-T segment down and the appearance of negative wave E in leads III, avF, V1, V2.
- Increase the duration of the interval in the right thoracic lead (V1) greater than 0.03 sec.

#### **Blocks of the heart [12, 15, 16]**

- **sinus node blocks** (1st st, 2nd st and type I, type II, 3rd st);
- **atrioventricular blocks** (1st st, 2nd st type I, type II, 3rd st);
- **intraventricular blocks** (single-strike, duplex, triplex).

#### **Sick sinus node syndrome [15, 16]**

Frequent infringements of the rhythm, often supraventricular, are SSS, (synonyms: sinus node dysfunction syndrome (SNDS), inert CA-node, bradycardias). At the heart of the pathogenesis of the syndrome is the reduction of the formation of pulses in the CA-node or violation of the pulse from the sinus node to the atrium [15].

Mechanisms of the violation of the function of the SA-node [15]:

1. Temporary or permanent loss of the ability to host SA impulses or reduce the strength of sinus impulses to sub threshold of depolarization inability fibrillation.
2. Blockade of SA-node with the impossibility of propagation of impulses.
3. Loss of myocardial atrial capability to perceive impulses of the SA-node.

SSS - deviation from the norm, regardless of the mechanism of development, based on pathophysiological and clinical symptoms, may be organic (primary SSS) and functional (secondary SSS), occurs under the influence of external factors and violations of vegetative innervation [15].

Most often it is bradycardia or SA-blockade. Since the SA-node - the main driver of the rhythm in physiological conditions - is ineffective, conditions for the emergence of ectopic lesions, including paroxysmal rhythm disturbances, appear [12, 15].



The assumption of SSS as the cause of relapsing paroxysms of arrhythmias should occur if out of attacks in patients, there is persistent bradycardia, dizziness, episodes of fainting, unfolding picture of the Morgan-Edams-Stokes syndrome. Necessary careful ECG analysis outside the attack, testing with atropine and isoproterenol, Holter monitoring of the ECG. On the ECG, registered outside the attack, there are sinus bradycardia and various variants of the SA-blockade. When injected 1-2 mg of atropine intravenously into a healthy person, the pulse rate is increased to 100 per min or more; in patients with CTSR not more than 90. Holter monitoring allows to record all ECG changes that are characteristic of the SSS and to document the association of these ECG-signs with such manifestations of syndrome as dizziness, fainting. The most accurate method for diagnosing a syndrome is an electrophysiological study[15].

### **Classification of sinus node dysfunction [15, 16]:**

#### **I. Organic (primary):**

##### **A. Acute.**

- Ischemic necrosis:

- 1) acute myocardial infarction;
- 2) disseminated intravascular coagulation;
- 3) thrombocytopenic purpura;
- 4) pheochromocytoma.

- Transient ischemia:

- 1) spasm of the coronary vessels;
- 2) coronary atherosclerosis;
- 3) transient thrombotic formation;
- 4) fibrous-dysplasia of the artery of the SA-node.

- Traumatic.

- Postoperative.

- Acute inflammation (diphtheria, collagenosis, vasculitis).

- Pericarditis.

- Invasions (tumors, abscesses, hemorrhages).

##### **B. Chronic.**

- Sclerogenic-fibrinogenic-degenerative disease.

- Summer age.

- Infiltration:

- 1) amyloidosis;
- 2) fat degeneration;
- 3) myxedema;
- 4) tumor infiltration.

- Ischemia:

- 1) coronary atherosclerosis;
- 2) spasm of the coronary arteries;
- 3) embolization of the arteries of the SA-node.

- Calcification.

- Inflammation:

- 1) infectious;
- 2) bacterial (typhoid, diphtheria);
- 3) parasitic (Chagas disease);
- 4) immunological (rheumatism).
  - Collagen vascular disease.
  - Friedreich progressive muscular dystrophy.
  - Endocrine diseases:

- 1) myxedema;
- 2) pheochromocytoma;
- 3) hyperthyroidism;
- 4) massive rapid loss of body weight.

- Postoperative (correction of the defect atrial SD, Tetralogy of Fallot, transposition of great vessels).

- Congenital:

- 1) hypoplasia of the SA-node;
- 2) coarctation of the aorta;
- 3) calcination of the mitral ring;
- 4) family-hereditary.

## **II. Functional (secondary)**

### **A. Acute**

- Increased vagus tone:

- 1) vasovagal syncope episodes;
- 2) sensitive carotid sinus (cardiodepressor type);
- 3) myocardial ischemia (acute myocardial infarction of the back wall of the lungs);
- 4) training;
- 5) vomiting;
- 6) glossopharyngeal dysplasia;
- 7) swallowing;
- 8) sleep;
- 9) hypercalcemia;
- 10) anesthesia;
- 11) Valsalva's test;
- 12) electric cardioversion;
- 13) subarachnoid haemorrhages and increased intracranial pressure;
- 14) hypothermia;
- 15) obstructive jaundice;
- 16) accelerating stimulation and removal of paroxysms of ultraviolet tachycardia;
- 17) medicated (beta-blockers, calcium antagonists, lidocaine);
- 18) metabolic;
- 19) hyperkalaemia.

B. Chronic.

- Increased vagus tone.

- 1) syndrome of sensitive carotid sinus;
- 2) athletes;
- 3) intracranial hypertension;
- 4) childhood;
- 5) medication (digitalis, methyldopa, novocainamid, calcium antagonists, amiodarone, disopyramides);
- 6) nicotine.

### **Distribution of patients with dysfunction SA node into groups[15]:**

1. Symptomatic DSN, clinical signs caused spontaneous arrhythmias caused by DSN.
2. Probably symptomatic DSN, the link of the clinic with DSN has not been proved.
3. DSN asymptomatic, clinical absent determined attack economies.

### **By clinical manifestations[12, 15]:**

- I. Latent form of DSN: characteristic bradycardia, migraine of the rhythm of the atrial, arrhythmias.
- II. Manifestation of the DSN: bradycardia, sinus arrhythmia, extrasystole, dizziness, asystole and Morgan-Edams-Stokes attacks.

### **In the course of[12, 15]:**

- I. Functional DSN, after the disappearance of etiological factor in improving the condition of patients, sometimes there is a need to restore autonomic balance vagolitical (drugs, atropine) or sympatonic (apresyn, korinfar, aminophylline, izadryn).
- II. Organic DSN (ie, SS syndrome).

In the case of ineffective drug correction, implantation of a pacemaker is indicated, especially in cases of severe clinical condition: pulse less than 40 beats per minute, Morgany-Edams-Stokes attacks.

### **EKG manifestations of SSS [15]:**

1. Sinus bradycardia (less than 50 beats per minute).
2. Periods of failure SA-node, asystole.
3. SA-blockades.
4. paroxysmal atrial arrhythmias with fast and slow ventricular rhythm.
5. Inefficiency recovery rate during atrial fibrillation and long pauses after recovery.
6. Tachy and bradycardia syndrome.

EKG-signs of SSS.

### **Forms of the SSS [15]:**

1. Latent.
2. Manifest.

3. Brady-tachyform.

4. Chronic bradyform of flashing arrhythmia, which is not treated by electropulse therapy.

**Non-invasive diagnosis of SSS** with the use of transvaginal electrocardiostimulation[15, 16]:

1. Determination of own cardiac rhythm after pharmacological denervation with atropine and propranolol with sympathetic and parasympathetic SA-node blockade - distribution of organic and functional damage of SA-node (DSN and SSS).

2. Estimation of automatism of the CA-node in the conditions of the trans esophageal electrocardiostimulation during 30-60 s with the measurement of post-stimulation pause with an increase in the frequency of the trans esophageal electrocardiostimulation at 10 imp/min until the development of the AV-block II of the (point Wenckbaha). Accelerating stimulation suppresses the automation of the SA-node, and we determine the following parameters:

- restoring the function of SA-node - interval between last stimulus that caused the contraction and the beginning of the first spontaneous P wave (normally less than 1400 ms);
- Adjusted time for the recovery of the SA-node function is the difference between the FVSFR and the ascending RP interval (normally less than 525 ms);
- sinoatrial time - transient stimulation of the atrial with a frequency of 6-8 impulses higher than the ascending frequency of heart rate (normally 200-240 msec).

The CSTR combines the following clinical disturbances of the rhythm:

- pronounced sinus bradycardia (50 in 1 min or less), constant or episodic;
- "Refusal" of the CA-node (including atrial stop) with periods of asystole or compensated slip rhythms;
- SA-blockade, not due to drugs;
- repeated alternation of sinus bradycardia (sometimes normal sinus rhythm) with attacks of atrial fibrillation, atrial tachycardia ("syndrome of bradykardia" - Shorter syndrome);

- slow recovery of the CA-node function after electrical cardioversion, electrical stimulation of the atrial, or spontaneous stop of ultrasound ventricular tachyarrhythmia.

### **Atrioventricular blocks [14, 15, 16]**

AB-blockade - slowing or stopping impulses from the atrial to the ventricles. AV-blocks, like other conduction disturbances, are characterized by degree, constancy or variability, a place of origin. Localization of AB-blockades may be as follows[15]:

- in inter-node tract between PP and AV-node;
- in the AB-node;
- at the level of the general trunk of the His beam;
- simultaneously at several levels.

AV-blockade is divided into proximal - there is a higher common trunk of the His bundle (in these cases, the ventricle complex lasts more than 0.11 s) and distal - at the level of the beam His (ventricular complex more than 0.12 s).

### **AB blockade I degree[12, 15]**

Most often characterized by extension of the interval P-Q more than 0,20 s and sometimes reaches 0,5-0,7 s. Ventricular complexes are of the usual form. With significant prolongation of the interval P-Q, the wave P can be placed on the previous tine T and poorly differentiated. Occasionally for blockade at the inter-node level, the wave P is expanded, having two vertices and a lowered amplitude. At distal level, blocking of the ventricular complexes is expanded due to intravaginal conduction disorder. AV-blockades of the I degree may be functional, associated with vagotonia. The development of organic blockade is observed in myocarditis of different etiology, with AMI of the lower back localization, with numerous myocardial dystrophies. Distal blockade may be due to postinfarction cardiosclerosis.

Clinically, AV-blockades do not show the degree I, but they may have an adverse prognostic value. For example, in the case of AMI, the nodal AV-blockade of the 1st degree passes into the AB-blockade of the II and III degrees.

**AV blockade of the 2nd degree of the I type of Mobits (periodical Samoylov-Venkybah)[12, 15]**

The interval P-Q progressively increases from cycle to cycle with the loss of one ventricular complex at the end of periodicals. The period of periodicals can be characterized by the ratio of the number of atrial complexes to the number of ventriculars - 4: 3, 5: 4, etc. Distal AV-blockades of the second degree of type I are combined with violations of intraventricular conduction - the ventricular complexes are widespread, having the appearance of a complete blockade of one of the legs of the His bundle. Nodule blockades that occur on the background of acute pathology are temporary and disappear in the background of treatment. The prognosis for these blockades is favorable, but one must take into account their possible transition to a full AB blockade. Ventricular contractions, especially in bradycardia, may be accompanied by lethargy, dizziness and other symptoms of cerebral ischemia[12].

In this type of blockade there is no progressive prolongation of intervals P-Q, which can be normal or prolonged. Death of the ventricular system occurs suddenly. The length of the pause is equal to the double-spaced P-P. Sometimes the atrial pulse locking is repeated with a defined sequence of 3: 2, 4: 3, etc.

Block II of the Mobitz type arises at a distal level - at the legs of the beam of His. Ventricular complexes are often expanded. Prognostically, this blockade is unfavorable and often becomes a complete AV block with the threat of MES syndrome development[12].

AV blockade of the second type of type 2: 1 and 3: 1. Such an option for blocking atrial pulses may appear against the background of block I and II types. If there is an expansion of the ventricular complexes, such blockade is formed on a distal level and is considered as variant of type II. Narrow ventricular complexes more often reflect the proximal level of blockade, that is, it belongs to Type I with favorable prognosis. Substations of the second degree are characterized by a ratio between the atrial and ventricular complexes type 4: 1, 5: 1, etc. Ventricular complexes can be narrow (proximal blockade) or wide (distal blockade). Distal blockade type 2: 1 and 3: 1 and subtotal can be the cause of severe cerebral ischemia, congestive insufficiency of circulation[12].

**AV-blockade of the III degree (full of AB-blockade)[12, 15]**

This blockade is characterized by loss of connection between activation of the atrium and ventricles, rhythms of which are autonomous. Atrial rhythm is more common than ventricular. The driver of the atrial rhythm is the SA-node, the ventricles are activated by automatic AV-cells or by the cells of the legs of the His beam and Purkinje fibers. The ECG records a distal blockade with wide ventricular complexes and a ventricular rhythm of less than 40 in 1 min. Complete AB blockade can be acute, transient, chronic. Acute AB-nodal blockade of the third degree arises in patients with the first day of AMI, more often posteriorly localization.

She is sensitive to atropine, which emphasizes her vagal genesis. The development of a blockade in later periods of the MI is associated with ischemia of the AV node. Chronic full AB blockades can be congenital and acquired. The most common causes of chronic full-AB blockade are severe myocardial damage (myocarditis, MI) and their consequences (cardiosclerosis). In some cases, a complete AB blockage runs asymptomatic or with little symptomatology. This applies, first of all, to blockages of paroxysmal type with a frequency of ventricular automatism about 40 pulses in 1 min. Most patients complain of an enlarged heartbeat, the frequency of which is practically unchanged in physical and psycho-emotional stresses. Ventricular bradysystole with full AB blockade in the background of severe myocardial damage is accompanied by the development of congestive insufficiency of the circulatory system, deterioration of cerebral and coronary circulation[12].

If there is a proximal full AV block of the III st., The rhythm driver of the ventricles, as a rule, is located in the AV-connection, below the blockade. Therefore, excitation is distributed over a leading system by the usual route and the QRS complexes are unchanged (heart rate is 45-60 per 1 minute). In complete distal (triphasicular) AB blockade, the source of the ventricular rhythm is located in one of the branches of the Gissan beam. The course of vaginal excitation is severely impaired and QRS complexes are widespread and deformed (heart rate not more than 40-45 per 1 minute)[12].

AV blockade of the 2nd and 3rd centuries, especially the distal form of complete AV blockade, is often accompanied by pronounced hemodynamic disorders with the development of Morgan-Adams-Stokes attacks[12].



**Blockade of the bundle of the His beam** -slow or complete termination of conduction of excitation by one, two or three branches of the His beam (single-headed, two-and three-pointed blockades).**In the complete blockade of the right bundle of the His beam** in the V1 exclusion, the QRS complex of the type rSR or rsR is registered, that is, a complex that has a characteristic M-shaped appearance. The duration of the QRS complex exceeds 0.12 sec. In the leads II and aVR, M-like QRS complexes (rSR', rsR' or rR') are also recorded, but the R' wave is generally low. In leads I, aVL, V<sub>5-6</sub>, QRSs of the type qRs are registered with a common and often stabbed S[15, 16].

**Brugada's syndrome** is characterized by a blockage of the right leg of the His beam with the rise of the segment ST in leads V<sub>1-3</sub> and a high probability of ventricular fibrillation[12, 15].

**The main ECG-sign of the complete blockade of the left bundle of the His beam** is the presence of distributed deformed complexes of type R (more than 0.12 s) with a split or broad tip at the leads V<sub>5-6</sub>, I, aVL, and in V<sub>1-2</sub>, III, aVF - common deformed ventricular complexes such as QS or rS with a split or wide tip of the wave S[12, 15].

In addition to the complete blockade of the left leg of the His beam, which consists of the anterior and posterior branches, the blockade of the right leg and the left front branch of the His beam, as well as the blockage of the right leg and the left posterior branch of the His beam, are related to the two-bulb blockade.

**For the blockade of the front branch of the left leg of the His beam,** the characteristic deviation of the electrical axis of the heart to the left is more than -30. The ram R in aVL exaggerates the R wave in the excerpts, and in the second, third, and aVF leads the ventricular complex of the type rS is registered. In the aVR assignment a QR complex is registered, where R is Q. QRS is less than 0.11 s[12, 15].

**When blocking the posterior branch of the left leg of the His beam,** the deviation of the electrical axis of the heart to the right is determined to be greater than +90. The QRS complex is equal to or less than 0.11 s. In the leads I and aVL are complexes of the type rS, in III and aVF are qR or R[12,15].

**For the complete blockage of the right leg and the front branch of the left leg of the His beam,** the width of the QRS complex is greater than 0.12 s, the

deflection of the electrical axis of the heart to  $-30^\circ$  with the presence of ECG signs of the blockade of the right leg in the thoracic nodes[12, 15].

**At full blockade of the right leg and the rear branch of the left leg of the His beam**, the width of the QRS complex is greater than 0.12 s, the deviation of the electric axis to the right is greater than  $+90^\circ$ . In leads I and aVL, QRS type rS or RS with wide and deep wave S. The QRS complex in leads III, aVF has type qR with wide R. In the chest positions, signs of the blockade of the right leg of the His beam[12, 15].

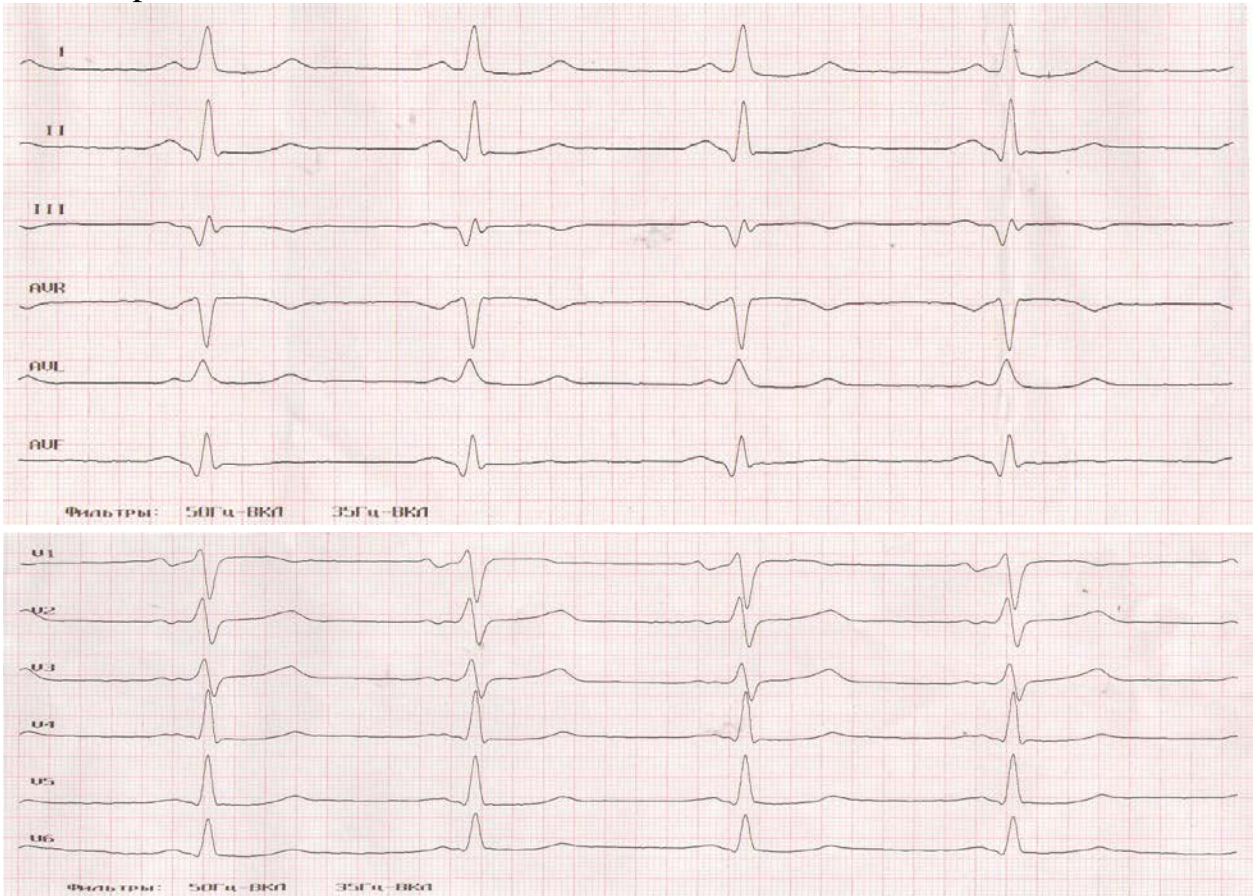
**At an incomplete three-point blockade** on an ECG, violations of AV-conduction by the type of incomplete AV-blockade of degrees I and II and common, deformed QRS complexes are recorded. An electrical pulse from the atrium is carried out to the ventricles in one, less damaged, branch of the His beam. In contrast to the complete three-way blockade, each of the QRS complexes registers the wave P[12, 15].

**In the presence of a complete three-arm blockade**, the electrical impulse does not generally be performed from the atrium to the ventricles, that is, there is a complete AV block of the III, with complete separation of the atrial and ventricular rhythms[12, 15].

## Electrocardiogram(Example of Interpretation)

Date of research \_\_\_\_\_

Name of patient \_\_\_\_\_



### Etalon response

PQ	0.14
QRS	0.10
QT	0.38
RR	0.85
Heart rate	70

Rhythm sinus, regular. The position of the EMU is horizontal. Pathological wave Q II, III, aVF is recorded, which may be a sign of Q-myocardial infarction of the posterior-diaphragmatic wall of the left ventricle.

## **ECHOCARDIOGRAPHY[17, 18]**

**Echocardiography** (ECHO) is a method of visualizing cavities and structures of the heart using ultrasound waves. Echocardiological examination should be carried out in the position of the patient on the left side. Accesses: left parasternal, apical, subcostal, supersonic, right parasternal and supraclavicular. Parasternal position is the long axis of the left ventricle (LV). This position is convenient for standard measurements and calculations. To obtain a position, the sensor is set in the IV or V intercostals at the left edge of the sternum. In the case when in the parasternal position the cursor of the M-mode is located strictly perpendicular to the image of the heart, measurements can be carried out with great accuracy. If the heart and cursor images are located at an angle, all the dimensions of the heart chambers will be significantly overestimated. EchoCG conclusion is to begin with indications of the size of the walls and the diameters of the cavities of the heart and trunk vessels[17, 18].

Standard echocardiographic measurements should be performed in the parasternal position on the long axis in the M- or B-mode at the end of the diastole[17, 18].

### **Standards for parasternal position[17, 18].**

Right ventricle (RV) - no more than 30mm

The thickness of the wall of the RV - not more than 5 mm

The thickness of the interventricular membrane is not more than 12 mm

The ultimate diastolic size of the LV is not more than 56 mm

The ultimate systolic LV size is not more than 40mm

The thickness of the back wall of the LV is not more than 12 mm

Left atrium (LA) - no more than 40mm

Aorta (Ao) - no more than 40mm

Pulmonary artery (PA) - no more than 28 mm

The final diastolic volume (EDV) is  $119 + 7$  ml

The final systolic volume (ESV) is  $45 + 3$  ml

Evaluation of systolic and diastolic LV function.

Stroke volume (SV) is the volume of blood that is thrown out in one abbreviation(70-100 ml).

$SV = EDV - ESV$ , where

EDV - ultimate diastolic volume

ESV is the ultimate systolic volume

The minute volume of blood (MVB) is the volume of blood pumped by the heart in one minute (5-7 ml/min).

$$\text{MVB} = \text{SV} * \text{HR}$$

Calculation of Myocardial Fibers Reduction Fraction (FS):

$$\text{FS} = (\text{EDS} - \text{ESS}) / \text{EDS}, \% \text{ FS} = \text{FS} \times 100\%$$

(in N% FS > 30%)

The degree of systolic thickening of the back wall of the heart and the interventricular membrane is determined by the formula [17, 18]:

- $T = T(s) - T(d) / T(d)$ , where
- T - degree of thickening

T(c) and T(d) - thickness of the wall of the LV at the end of diastole and systole.

**The ejection fraction (EF, EF) is most often used to assess the state of systolic function of the heart [17, 18].**

$$\text{EF} = (\text{EDV} - \text{ESV}) / \text{EDV}, \% \text{ EF} = \text{FS} \times 100\%, (\text{at N } 50\text{-}70\%).$$

**To assess the state of diastolic function, LV is used [17, 18]:**

- the rate of pressure increase in the LV in the beginning of the systole (dP/dt)
- time constant of myocardial relaxation during isovolumic diastole
- rigidity of the chamber of LV
- time isovolumic relaxation
- time isovolymic reduction
- transdermal diastolic flow
- half time peak acceleration E AT1/2

in the norm AT1/2 = 62+18 m/s

- half delayed peak E DT1/2
- in the normal range DT1/2 = 73 + 24 m/s
- fraction 1/2 filling
  - fraction 1/3 filling
  - time constant of myocardial relaxation.

Physiological agents that affect the diastolic function: age, heart rate, systolic function of the lungs, atrial function, respiration, load test.

**Echocardiography plays a leading role in the diagnosis of coronary artery disease and its complications[17]:**

- Estimation of total myocardial contractility;
- Assessment of local myocardial contractility;
- Assessment of diastolic function;
- Diagnosis of CHD complications.

**Allocate the following variants of contractility[17, 18]:**

- normokinesis - all areas of the endocardium in the systole uniformly thicken
- hypokinesis - reduction of thickening of the endocardium in one of the zones in the systole compared with other areas. It can be diffuse and local. Local hypokinesis is associated with small or intracervical damage to the myocardium.
- Akinesis - the absence of thickening of the endocardium in the systole in one of the sites is associated with major-head injury.
- dyskinesis - the paradoxical movement of the regions of the heart muscle in the systole is characteristic of the aneurysm. The paradoxical movement of the ventricular membrane can be observed:
  - a) against the background of the blockade of branches of the His beam
  - b) in the presence of an electrocardiostimulator
  - c) Against the background of pulmonary hypertension
  - r) against the background of pericarditis.

**Map of echocardiographic examination(example of interpretation)**

Surname \_\_\_\_\_ person . \_\_\_\_\_ Age \_\_\_\_\_ 40 y . \_\_\_\_\_

	<b>gradient pressure</b>	<b>area</b>	<b>regurgitation</b>	<b>other</b>
<b>Mitral valve</b>			II degree	No special
<b>Aortic valve</b>	2.4 mmHg		absent	No special
<b>Tricuspid valve</b>			1st degree	No special
<b>Pulmonary valve</b>			absent	No special

Aortic root (N <3.5 cm) 3.5 cm Left atrium (N <4 cm) 4.8 cm  
 Left ventricle: EDV (N f: 56-104 ml, m: 67-155 ml) 215 ml  
 ESV (N f: 19-49 ml, m: 22-58 ml) 147 ml EF 31.6%  
 Right atrium (N <2.5 cm) 3.5 cm Right ventricle (N <2.5 cm) 3.1 cm  
 Systolic pressure in the pulmonary artery 35 mmHg; \_\_\_\_\_ degree  
 The thickness of the walls and the region's contractile capacity  
 IBP 0.8 cm \_\_\_\_\_  
 Rear 0.8 cm \_\_\_\_\_  
 Lateral \_\_\_\_\_ cm \_\_\_\_\_ diffuse hypokinesis  
 Front \_\_\_\_\_ cm \_\_\_\_\_  
 Top \_\_\_\_\_ cm \_\_\_\_\_  
 Aneurysm \_\_\_\_\_  
 Other: separation of the pericardium leaves to 0.4 cm in diastole.

### **Etalon response**

Significant dilatation of the cavity of the left atrium, left ventricle, right ventricle.

Significant diffuse decrease in the contractile capacity of the left ventricle.

Relative failure of the mitral valve of the II degree.

Pulmonary hypertension of the 1st degree.

These changes are possible in dilated cardiomyopathy, acute diffuse myocarditis.

**Stress Echocardiography** is an ultrasound examination of the heart that is used to record myocardial ischemia with the help of physical, pharmacological and other stress agents. Stress-echocardiography can determine the ischemic reaction of the myocardium to stressful effects, which manifests itself by the pathological kinetics of the walls of the lungs, which is recorded by two-dimensional echocardiography in real time. In most cases, regional contraction occurs immediately after a violation of myocardial perfusion, but before the onset of ECG depression, the segment of ST and rebound pain. Thus, disturbance of regional contractility is one of the earliest signs of myocardial ischemia. Stress Echocardiography distinguishes high sensitivity, specificity, availability of the study, the possibility of multiple repetition. The test is

reliable, non-invasive, safe, easy to execute, more economical, requiring less time than radionuclide methods of diagnosing myocardial ischemia.[19].

In healthy individuals, the normal reaction of LV to stress-test load causes hyperkinetic movement of all walls of the lungs; increases systolic thickening of the walls of the lungs, increases the PV, decreases the size of the lungs. Pathological response of LV to stress test[19]:

- violation of local movement of the walls of the lungs in the form of hypokinesis, akinesis, dyskinesis;
- reduction of systolic thickening of LV walls;
- reduction of EF, increase of EDP in LV and LA;
- increase in size of LV;
- increase in size of RV;
- the lack of adequate hyperkinetic and hyperdynamic cardiac responses to stress-test effects.

**Stress agents[19]:**

- Physical exercise tests;
- Pharmacological stress tests (with dobutamine);
- Hyperventilation test and cold test;
- Stress tests with electrostimulation of the heart: through the esophageal electrocardiostimulation of the atrial, endocardial stimulation of the atrium and ventricles;
- Psychological tests;
- Sample with lifting of the lower extremities.

**All stress-echocardiography studies are mandatory[19]:**

- in the original position;
- during stress testing;
- in the recovery period.

Stress echocardiography allows to predict the course of chronic forms of CHD, the prognosis (estimation of probability) for the development of acute coronary insufficiency or myocardial infarction, to assess the degree of risk of developing cardiac complications in surgical operations on the heart and blood vessels, to predict the development of complications in non-cardiac operations[19].



## **EXERCISE STRESS TEST [20]**

Veloergometry (BEM) - an ECG-study method in conditions of muscular work on a bicycle lens that allows adjusting the size of loads by changing the resistance of the pedal's rotation. It is most expedient to use continuous operation to increase power without rest periods. In this case, the power is set depending on the state of health, sex, age, weight and physical fitness [20, 21].

Conducting VEM research is recommended for diagnostic purposes for the detection of latent coronary insufficiency, in the examination of persons with deviations in the state of health, in specifying the genesis of changes in the terminal part of the ventricular complex of the ECG. Begin the procedure not earlier than 2 hours after eating. The speed of pedaling is 60 revolutions per minute. Record the output values of the ECG, remove at the end of each minute of the sample, after its termination, as well as in the recovery period at 2-, 3-, 5-, 10-minute rest. Measures blood pressure.

Another variant of stress testing is the treadmill test [20]. The Treadmill test report must match the physical capabilities of the patient and meet the test tasks. For healthy individuals, the standard Bruce protocol is also popular. A multi-stage Bruce protocol with maximum physical activity includes 3-minute periods to achieve steady state before the next increase in load. In older people or those with limited physical abilities through heart disease, the protocol can be modified to include two 3-minute steps on the tape speed of 2.7 km/h and at 0% and 2.7 treadmill km/h with inclination of the track in 5%. Limiting the Bruce protocol is a relatively large increase in  $Vo_2$  between the stages. If the patient performs the load > 3rd degree, he spends extra energy. Protocols using Naughton and Weber use 1-2-minute intervals with a load increase between them at 1 MET. These protocols are more suitable for patients with low TFA, for example, with compensated CHF. Implementation of the protocol with the inclination of the treadmill begin with a relatively slow speed of the track, which gradually accelerates until the course of the patient becomes fast. The angle of inclination progressively increases due to fixed time intervals (for example, 10-60 seconds) starting from the zero level and with the estimated increase in the basis of the expected functional endurance of the patient so that the entire protocol is completed within 6-12 minutes. With such a protocol, the rates of FN increase

continuously, and the stable state can not be achieved. During a Treadmill test, it is important that the patient does not hold on the rails, especially in the front. In bench tests, the reevaluation of functional reserves may reach 20%, and  $VO_2$  - significantly reduced. Due to the fact that resistance to handrails is difficult to quantify and take into account during multiple testing, more stable results can only be obtained if the patient does not hold on to them [20, 21].

Objective physiological indicators that reflect the human capabilities during loading are presented in the form of dual product (DP) and metabolic units (ME). Doubles, or Robinson's index [20]:

$DP = BP \times HR$ ; This is the value of systolic blood pressure, multiplied by the number of cardiac contractions at the height of the load. According to some authors, this value accurately characterizes the possibility of coronary blood flow [20].

ME is proposed for measuring the aerobic capacity of the myocardium. 1 ME corresponds to the consumption of oxygen in the amount of 1.5 ml per 1 kg of body weight per minute. ME index indicates how much oxygen consumption increases along with increasing load compared to the rest. During loading, the maximum consumption of oxygen may exceed  $21 \text{ cm}^3/(\text{kg}/\text{min})$  [20, 21].

**Criteria for termination of a test [20]:**

- increase in pulse to the submaximal value, which is 75% of the maximum permissible ( $220 - \text{age}$ );
- depression of segment ST at 0,2 mV and more;
- disturbance of the rhythm of high gradations;
- changes in atrioventricular and intraventricular conduction
- increase of blood pressure to 220/120 mm Hg.;
- the onset of angina pectoris;
- significant choking
- dizziness;
- sharp changes in the color of the face;
- expressed general weakness;
- severe muscle pain;
- failure of the patient;

The test is considered to be positive in case of angina, ECG signs of myocardial ischemia (reduction of the S-T segment by 1 mm or more, the migration of the S-T segment by more than 1 mm, the emergence of threatening rhythm disturbances (frequent or polytopian ventricular extrasystoles, paroxysmal tachycardia, flashing arrhythmia, the presence of violations of atrioventricular and intraventricular conduction, from the time of the QRS complex: a sharp decrease in the voltage of the wave R, the deepening and expansion of the previously existing Q and Q wave, the transition of the Q to QS), with a decrease in AT of 25-30% of the output that can be the beginning of ischemic collapse, and the increase in blood pressure up to 230/130 mm Hg and more, dyspepsia (often breathing more than 30 minutes per minute), the appearance of a general abrupt weakness, the emergence of symptoms of cerebral insufficiency (dizziness, headache, nausea, visual impairment)[20, 21].

### **TRANSESOPHAGEAL ELECTRIC STIMULATION OF THE HEART [15]**

The method of transesophageal electrical stimulation of the heart (TEES) is based on endocardial heart stimulation using an artificial rhythm driver - an external stimulant. The proliferation of endocardial stimulation has led to the introduction of a non-invasive version of the method - via spasmodic pacing. To date, this method is used to diagnose and treat violations of rhythm and conduction, in particular with syndrome of weakness of sinus node, supraventricular paroxysmal rhythm disturbances, atrial fibrillation, for the diagnosis of coronary artery disease and evaluation of LV function[15].

The method of the TEES disaster is characterized by certain advantages compared to VEM, which is due to higher security in the non-invasive nature, lack of significant effect on blood pressure. The TEES is conducted according to the generally accepted method in the patient's position on the back. The test begins after an intranasal injection of an electrode probe with a distance between the poles of 2.5 to 3 cm under the control of the esophageal ECG, and the registration of the initial parameters of hemodynamics. Localization of the probe is determined by the maximum amplitude of the wave P of the esophageal ECG. Stimulation begins with a

frequency rhythm of 80-100 to 160 imp/min with a stepwise increasing interval of 20 imp/min at 2 minutes of each degree. According to the results of the TEES, the following indicators of the function of the sinus node and atrioventricular conductivity are determined[15]:

- synaptic time as the difference in the length of intervals between atrial potentials;
- Time to restore the function of the sinus node;
- the interval from the last cardiovascular to the own wave P;
- Adjusted time to restore the sinus node function;
- Difference between and average output cardiocycle;
- Wenckebach's point is the minimum frequency of stimulation, which leads to a functional atrioventricular blockade of the second degree.

The trial is terminated in the development of myocardial ischemia, as evidenced by the appearance of pain and/or depression of the ST segment of 0.08 s after the J point of the horizontal or ciliated-type type with an amplitude of 0.1 mV or more, or a transient decrease in the ST segment with an amplitude of 0.2 mV or more. In the absence of ECG changes, the sample is brought to 160 imp/min[15].

## ARTERIAL HYPERTENSION[10,21]

### Terminology:

- ✓ **Arterial hypertension** -constantly elevated systolic and/or diastolic blood pressure (SBP and/or DBP).
- ✓ **Essential hypertension**(primary hypertension, or hypertonic disease) - elevated blood pressure in the absence of an obvious cause of its increase.
- ✓ **Secondary hypertension (symptomatic)** -hypertension, the cause of which can be detected.
- ✓ **Resistant(refractory)hypertension**- in the setting of  $\geq 3$  antihypertensive drugs in adequate doses, it is not possible to reach the target blood pressure level.
- ✓ **Malignant hypertension**is a syndrome with high blood pressure ( $\geq 220/120$  mm Hg) with hemorrhages and exudates in the retina, often with edema of the optic nerve.
- ✓ **Hypertensive crisis**- a sudden significant increase in blood pressure, which is accompanied by the appearance/increase of disorders of the target organs or the autonomic nervous system.

### Classification AH [10]

#### Indicators of BP

Indicator	SBP	DBP
Optimal blood pressure	<120	<80
Normal blood pressure	<130	<85
Increased normal blood pressure	130-139	85-89

#### Degree of severity or form of arterial hypertension

1 degree (mild hypertension)	140-159	90-99
2 degree (moderate hypertension)	160-179	100-109
3 degree (severe hypertension)	$\geq 180$	$\geq 100$
Isolated systolic hypertension	$\geq 140$	<90
subgroup: marginal	140-149	<90

## Stages of arterial hypertension[10,21]

### Stage I

Objective signs of organic damage to target organs are absent

### Stage II

There are objective signs of damage to target organs without symptoms on their part or disruption of the function:

or Left ventricular hypertrophy (according to ECG, ECG, radiography)

or Generalized narrowing of the arteries of the retina,

or Microalbuminuria or proteinuria and/or a slight increase in plasma creatinine concentrations (1.2-2.0 mg / dl or 177  $\mu$ mol/L)

### Stage III

Objective signs of damage to target organs with symptoms on their part and impaired function.

Heart - Myocardial infarction.

- Cardiac insufficiency of IIA-III st.

Brain - Insult.

- Transient ischemic attack.

- Acute hypertensive encephalopathy.

- Chronic hypertensive encephalopathy of stage III.

- Vascular dementia.

Eyes - Hematopoiesis and exudates in the retina with or without edema of the visual disc (these signs are pathognomonic also for the malignant phase of hypertension)

Kidneys - Creatinine plasma concentration > 2.0 mg/dL (> 177  $\mu$ mol / L)

Vessels - Layer aortic aneurysm.

### Target organs damage [10]

• **Left ventricular hypertrophy-ECG criteria:** Sokolow-Lyon criterion ( $S_{v1} + R_{v5}$  or  $v_6 \geq 38$  mm), Cornell criterion in the form of modified Cornell voltage index [ $R^{avL}$ (mm) +  $S^{v3}$ (mm)]  $\times$  QRS (at the level of  $\geq 2440$  mm/ms are determined by LVH). **Echocardiographic criteria** index left ventricular mass to men  $\geq 125$  g/m<sup>2</sup> for women  $\geq 110$  g/m<sup>2</sup>.

The recognized distribution of **left ventricular hypertrophy** depending on the **types of LV geometry** consists in conducting an echocardiographic study with the estimation of the following indices:

1) the **mass of the myocardium of the LV** according to the ASE (American Society of Echocardiography) by the  $MM = 1.04 \cdot \{ [EDS + PWLVd + IVSd]^3 - EDS^3 \} - 13.6$  (g), where the EDS is the terminal diastolic size, PWLVd-thickness of the back wall of LV in diastole, IVSd-thickness of IVS in diastole.

2) The **index of mass of myocardial infarction (IMMLM)** according to the formula  $IMMLSH = MM/S$  (g/m<sup>2</sup>), where MM is the mass of the myocardium LV, S is the area of the body surface (m<sup>2</sup>).

3) the **relative thickness of the wall of the LV** by the formula  $= [2 \cdot PWLVd] / EDS$ , where PWLVd-thickness of the back wall of LV in diastole, EDS – the ultimate diastolic size.

**The geometry of the LV** in the beginning depends on the IMMLV (Ganau A. et al., 1992) in the following distribution: normal geometry (IMMLV < 125 g/m<sup>2</sup>, OTH < 0.45); concentric remodeling (IMMLV < 125 g/m<sup>2</sup>, OTH > 0.45); eccentric hypertrophy (IMMLV > 125 g/m<sup>2</sup>, OTH < 0.45); concentric hypertrophy (IMMLV > 125 g/m<sup>2</sup>, OTH > 0.45).

**Types of left ventricular geometry by A. Ganau et al. (1992)**

<b>Types of geometry LV</b>	<b>OTH</b>	<b>IMMLV</b>
Normal geometry of LV	< 0.45	< 125 g/m <sup>2</sup>
Concentric remodeling	> 0.45	< 125 g/m <sup>2</sup>
Concentric hypertrophy	> 0.45	> 125 g/m <sup>2</sup>
Eccentric hypertrophy	< 0.45	> 125 g/m <sup>2</sup>

- **Ultrasound signs of vessel wall thickening** (intima/carotid artery thickness  $\geq 0.9$  mm) or available atherosclerotic plaque.

- A slight increase in the concentration of **creatinine** (in men, 115-133  $\mu\text{mol/l}$  or 1,3-1,5 mg/dl, in women - 107-124  $\mu\text{mol/l}$  or 1,2-1,4 mg/dl)

**-Microalbuminuria**(30-300 mg/24 h, albumin/creatinine ratio  $\geq 22$  mg/g or  $\geq 2.5$  mg/mmol in men and  $\geq 31$  mg/g or  $\geq 3.5$  mg/mmol in women).

### **Concomitant diseases[10]**

**-Diabetes mellitus-** plasma glucose uptake  $\geq 7.0$  mmol/l (126 mg/dl); plasma glucose 2 years after meal  $\geq 11.00$  mmol/l (198 mg/dl).

**-Cerebrovascular diseases**(ischemic stroke, cerebral hemorrhage, transient ischemic attack). Diseases of the heart - myocardial infarction, angina pectoris, postoperative revascularization, heart failure.

**-Kidney disease-** diabetic nephropathy, renal insufficiency (serum creatinine  $> 133$   $\mu$ mol/l or  $> 1.5$  mg/dl, in women  $> 124$   $\mu$ mol/l or  $> 1.4$  mg/dl). Damage to peripheral arteries.

**-Severe retinopathy-** hemorrhages, exudates, swelling of the optic nerve.

## **ISCHEMIC HEART DISEASE[21,22]**

Classification of coronary heart disease

1. Sudden coronary death:

1.1. Sudden clinical coronary death.

1.2. Sudden coronary death (fatal case).

2. Angina pectoris:

2.1.1. Stable stress angina (indicating the functional class (FC), as shown in the table; for III and IV FC it is possible to join the angina of rest, which is a stenocardia of small strains).

2.1.2. Stable angina pectoris with angiographically intact vessels (coronary syndrome X).

2.2. Vasospastic angina (angiospastic, spontaneous, variant, Prinzmetal).

2.3 Unstable angina:

2.3.1. Angina, which arose for the first time to 28 days (first angina attacks with transient changes in ECG-rest).

2.3.2. Progressing stenocardia (appearance of angina in rest or night attacks in a patient with angina tension, change in FC angina, progressive decrease in exercise tolerance, transient changes in ECG-rest).



2.3.3. Early post-infarction angina (3 - 28 days).

3. Acute myocardial infarction.

The diagnosis is indicated with an indication of the date (up to 28 days), localization (the anterior wall, anterior, prefabricated, anterior, septal, diaphragmatic, lower limb, lower limb, lower limb, upper limb, basal-lateral, upper side, lateral, posterior, posterior, posterior, septal, right ventricular ), recurrent (from 3 to 28 days), primary, repeated (indication of size and localization is not necessary, if there are difficulties in ECG-diagnostics):

3.1. Acute myocardial infarction with the presence of Q (transmural, large)

3.2. Acute myocardial infarction without Q (small-throat).

3.3. Acute subendocardial myocardial infarction.

3.4. Acute myocardial infarction (undetermined).

3.5. Recurrent myocardial infarction (from 3 to 28 days).

3.6. Repeated myocardial infarction (after 28 days).

3.7. Acute coronary insufficiency - a diagnosis of the previous one - elevation or depression of the segment of ST, which reflects acute ischemia before the development of signs of myocardial necrosis or sudden coronary death.

Some complications of acute myocardial infarction are indicated by their time of occurrence:

acute HF (Classes by Claire I-IV);

violations of heart rhythm and conduction;

rupture of the outer heart (with hemopericardium, without haemopericardium and internal (defect of interventricular membrane, torsion of tendon chord, rupture of papillary muscle)

thromboembolism of different localization;

thrombosis in the cavity of the heart;

acute aneurysm of the heart;

Dressler syndrome;

postinfarction angina (from 72 hours after the development of AMI and up to 28 days).

4. Cardiosclerosis:

4.1. focal cardiosclerosis;

4.1.1.postinfarction cardiosclerosis with indication of the form and stage of HF, the nature of the violation of rhythm and conduction, the number of transferred heart attacks, their location and time of occurrence);heart aneurysm is chronic;

4.1.2.focal cardiosclerosis, not caused by MI;

4.2.diffuse cardiosclerosis (indicating the stage of HF, violation of rhythm and conduction).

5. Impotence CHD.

The diagnosis is based on the detection of signs of myocardial ischemia by means of a physical activity test, Holter ECG monitoring with QC data verification, Tl/Tc myocard scintigraphy, stress-echocardiography (stress-echocardiography) [21].

**Classification of angina according to the division into functional classes for the classification of the Canadian cardiovascular society[21, 22]**

<b>FC angina</b>	<b>Canadian ClassificationFCangina</b>	<b>Domestic modification</b>	<b>Veloergometry</b>
<b>I FC</b>	The appearance of angina when load is of high duration and intensity.	Great load	More than 100 W
<b>II FC</b>	Slight limitation of daily physical activity, development of angina during the course of a distance of more than 2 quarters.	More than 500 m above the 1st floor	75-100 W
<b>III FC</b>	Angina pectoris of moderate severity, pain in the course of a distance of 1-2 quarters, on the 1st floor.	100 m, 1 floor	50 W
<b>IV FC</b>	Heavy angina with pain in everyday work, often alone.	Less than 100 m	25 W or less

**Acute coronary syndrome (ACS)** - a group of symptoms and signs acute myocardial infarction (AMI) or unstable angina [11, 12]. The term ACS is used at first contact with patients as a preliminary diagnosis and includes ACS with ST-segment elevation on ECG and without such. First, as a rule, AMI with Q wave on an electrocardiogram, the second - AMI without Q and unstable angina (final clinical diagnoses) [23, 24, 27].

**Classification of ACS [24, 27]:**

- Acute coronary syndrome with the rise of the ST segment or acute blockage of the left (right) leg of the His bundle. Sustained ST segment elevations indicate acute complete occlusion of the coronary artery. The purpose of treatment in this situation is the rapid restoration of vascular patency with thrombolytic drugs (in the absence of contraindications) or percutaneous angioplasty. ACS with ST elevation in most cases is preceded by MI with Q wave.
- Acute coronary syndrome without ST segment elevation. Changes in ECG are characterized by persistent or transient depression of the ST segment, inversion, smoothness, or pseudonormalization of the T wave. ECG in the first hour may be normal. Emergency care with ACS without ST segment elevation is to eliminate pain and myocardial ischemia using aspirin, heparin,  $\beta$ -blockers, nitrates. Thrombolytic therapy is ineffective and may even worsen the prognosis of patients. The diagnosis of ACS at the pre-hospital stage is based on clinical manifestations (angina status) and ECG-diagnosis (Figure 9).

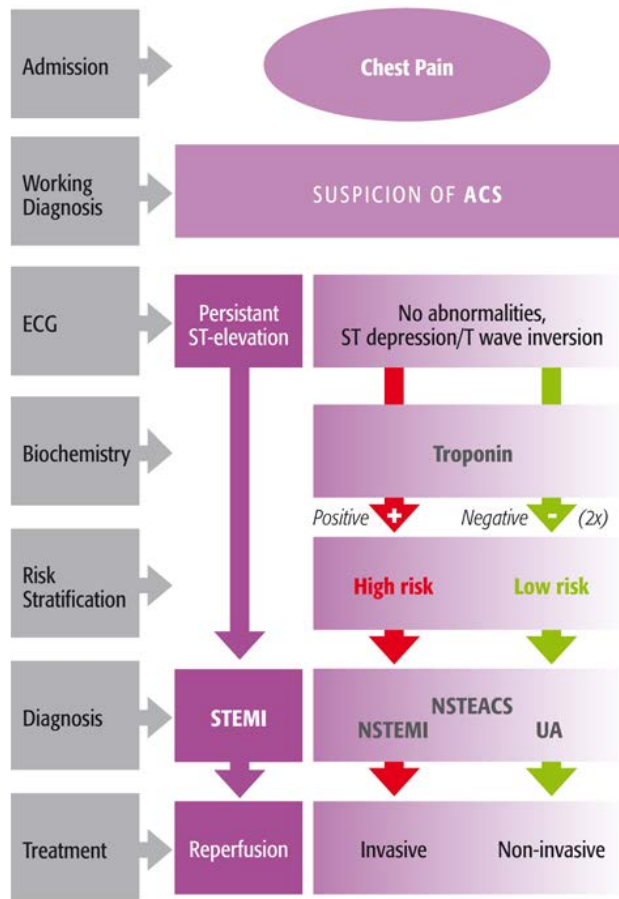


Fig.9. Algorithm of diagnostics of ACS

### Electronic diagnosis of myocardial infarction[12, 14, 25]

Acute myocardial infarction (AMI) reflects the death of cardiomyocytes, due to the preservation of ischemia in conditions of prolonged acute coronary insufficiency and restriction of coronary blood supply. AMI is diagnosed in the presence of clinical symptoms of myocardial ischemia and ST segment elevation, namely, a new ST segment elevation at J point of at least 0.2 mV in the leads  $V_1$ - $V_3$  and 0.1 mV in other leads or without elevation of the ST segment with depression of ST or T wave disorders. These ECG changes are accompanied by the dynamics of markers of myocardial necrosis and ECH by cardiographic signs[12, 13, 14].

The center of myocardial damage in the IM consists of a zone of necrosis and adjacent to it a zone of damage that goes into the zone of ischemia. The area of **necrosis** expressed in ECG by changes in the QRS complex, the zone of damage - the displacement of the ST segment, the zone of ischemia - by the change in the T

wave. The diagnosis is indicated with a date (up to 28 days), localization, recurrent (from 3 to 28 days), primary, repeated (specify size and localization is not necessary, if there are difficulties in ECG-diagnostics)[12, 14].

The main ECG-sign of MI(**necrosis**) is the appearance of a wide and deep **Q**. As a rule, the wave **Q** appears a few hours after the onset of MI.

The zone of **damage** is characterized by an ECG arcuate rise of the **ST** segment, which merges with the **T** wave (**monophasic curve**). The shift of the **ST** segment is a very characteristic and early ECG-sign of the MI, which precedes the appearance of the **Q**-wave.

The **ST** segment **elevation** is maintained for **3-5 days**, after which it gradually decreases to isolation and a deep negative "coronary" wave **T** forms. With common MI elevations, the **ST** segment may be seen for a longer time - up to **1-2 weeks**. In some cases, prolonged **ST** segment **elevation** may be a reflection of concomitant pericarditis. It should also be borne in mind that the presence of ECG-signs of heart aneurysms ("**frozen monophasic curve**").

Consequently, the nature of the ECG changes may be the **most acute** (hours, rarely days), **acute** (up to 10 days), **subacute** (up to 4-8 weeks) and **scarring** (8 weeks or more) periods of MI.

**There are three main localizations of MI[12, 14]:**

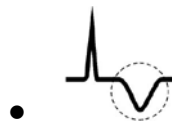
- Implantation of the anterior wall of the left ventricle.
- MI of the posterior diaphragmatic region of the left ventricle.
- MI of the posterior basal division of the left ventricle.

**Classification of myocardial infarction by ECG signs[12, 14]:**

- Acute myocardial infarction with the presence of **Q** (transmural, focal)



- Acute myocardial infarction without **Q** (small-grained).



- Acute subendocardial myocardial infarction.



In case of damage to the anterior part of the interventricular membrane, characteristic ECG changes are observed in V<sub>1</sub>-V<sub>2</sub>.

When localization of the MI in the area of the anterior wall of the left ventricle (including the tops), the corresponding ECG changes are recorded in V<sub>3</sub>-V<sub>4</sub>, lateral wall - in leads I (less often than II), aVL, V<sub>5</sub>, V<sub>6</sub>, high departments the side wall - in the leads I and aVL, and in V<sub>4</sub>-V<sub>6</sub> in the III interdiscipline.

**Reciprocal (discordant) changes in the segment of ST are observed in the MI of the anterior wall of the left ventricle in leads II, III, and VF.**

**The posterior diaphragmatic (lower) MI is accompanied by corresponding ECG changes in leads II, III, aVF and discordant in I, aVL.**

For **inferobasal MI**, the appearance of only reciprocal ECG changes in the leads V<sub>1</sub>-V<sub>2</sub> (high wave **R** and **T**), as well as the appearance of the **Q** wave at the corresponding dynamics **T** in the leads V<sub>7</sub>-V<sub>9</sub> is **characteristic**. Reciprocal changes while logged in leads I, V<sub>1</sub>-V<sub>4</sub>.

At the same time, the occurrence of MI in the opposite sections of the left ventricle may not be accompanied by appropriate reciprocal ECG changes.

The presence of an electrocardiogram **QS** complex is characteristic of a transmural MI.

Preservation of the wave **R** in the presence of pathological **Q** indicates a large-area MI.

A characteristic feature **and intramural MI** is the formation of deep "coronary" T wave in multiple chest leads.

Possible decrease of amplitude R, rise or depression of segment **ST**.

Subendocardial MI is characterized by depression of the segment of **ST** in several chest leads, the wave **T** may be negative, two-phase or positive, **the amplitude of the wave R**-lowered.

### **AMI stages for ECG dynamics[12, 14]**

1. Ischemic stage with the development of subendocardial ischemia with depression of the segment ST and an increase in the growth of the wave T, the appearance of "coronary" wave T (15-30 min).

2. Stage of damage with the transition of subendocardial ischemia into subepicardial, the development of elevation and depression of the segment ST.

3. The acute stadium is accompanied by the rise of the segment ST, its merger with a positive T wave, when it moves from the topline of the wave R, the appearance of the pathological wave Q (several hours - 2-3 days).

4. Subcategory of the stadium - the ST segment returns to the isolinium, the negative wave T forms, the formation of the pathological Q wave (up to 3-4 weeks) ends.

5. The stage of the "scar" is the pathological wave Q, the negative wave T, the ST segment on the isoline, and eventually the changes may disappear (1-6 months to a year).

### **Localization of MI according to ECG data[12, 14]**

1. AnteriorAMI (lead I, II, aVL).

2. Antero-septalAMI (withdrawal I, II, aVL,  $V_{1-2}$ ).

3. Antero-septal-apical MI (I, II, aVL,  $V_{1-4}$ ).

4. Antero-septal-apical -lateral MI (I, II, aVL,  $V_{1-6}$ ).

5. PosteriorMI (II, III, aVF).

6. Postero-lateral AMI (II, III, aVF,  $V_{5-6}$ ).

7. Postero-basal AMI (a resin of a wave R in  $V_{1-2}$ , pathological changes in additional leads D,  $S_{1-4}$ ).

The standard of diagnosis of myocardial damage and the main criterion for diagnosing an MI without elevation of the ST segment are biochemical markers of damage. Determination of the content of cardiac troponins I, or T twice, after 6 - 12 hours. Criteria necrosis - increased Troponin, at least in one of the samples CK and/or MB - CK to the diagnostic level[21, 23, 27].

### Release of cardiac markers after acute myocardial infarction (AMI).

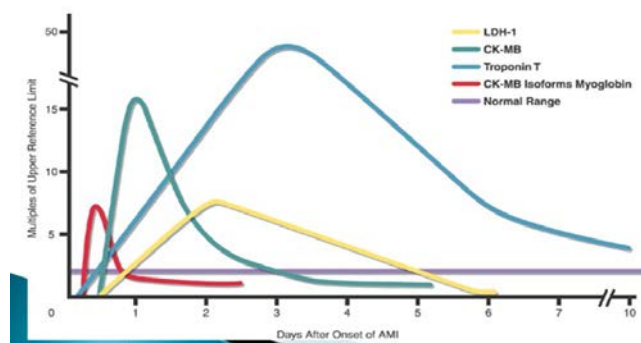


Fig. 10. Dynamics of markers of myocardial necrosis in patients with acute myocardial infarction

Increasing the content of markers of necrosis of cardiomyocytes in ACS should be differentiated with pulmonary artery thromboembolism[25, 26]. Recently, one of the most valuable in terms of diagnosis of thrombosis is the method used to determine the level of blood D-dimers. This fibrin, which is the ultimate product of blood coagulation, acts as a substrate for plasmin - the main enzyme of fibrinolysis, which causes its asymmetric sequencing to occur. The split factor XII and fibers formed fibrin-D-dimer and to a lesser extent trimers (as plasmin is not able to cut formed a covalent bond between the strings)[26].

In the blood of healthy people, the concentration of D-dimers does not exceed 500 ng of FEU (fibrinogen-equivalent units)/ml. Excess indicates an activation of fibrinolysis, which is preceded by increased coagulation with excessive formation of insoluble fibrin[26].

In the case of TELL, the determination of D-dimers is characterized by high sensitivity (96-99%), but low specificity (no more than 50%). This method of research has a high negative diagnostic value, that is, it is possible to exclude in the patient a pulmonary disorder with a normal content of D-dimers (<500 µg/l). However, an increase in the level of D-dimers may also be due to other causes associated with active thrombotic events - sepsis, acute myocardial infarction, malignant tumors, inflammation, surgical interventions, some systemic diseases, pregnancy, etc. (a slight increase in the level of D-dimers can also be observed in the elderly). Consequently,



when an increased concentration of D-dimers is detected, a patient needs to be screened to confirm the presence of a blood clot in the pulmonary vessels. However, the beginning of the diagnostic algorithm precisely from the definition of D-dimers allows at this stage to exclude pulmonary embolism without further examination in a third of patients. It should be noted that in many patients post-carotid artery disease, the level of D-dimer remains elevated for several months, despite anticoagulant therapy. Therefore, when recurring pulmonary embolism, increasing the level of D-dimers can not be considered as a marker for recurrent thrombotic formation, although the normal value of the indicator allows eliminating the relapse of the disease (sensitivity of about 100%) Therefore, when recurring pulmonary embolism, increasing the level of D-dimers can not be considered as a marker for recurrent thrombotic formation, although the normal value of the indicator allows eliminating the relapse of the disease (sensitivity of about 100%). Therefore, when recurring pulmonary embolism, increasing the level of D-dimers can not be considered as a marker for recurrent thrombotic formation, although the normal value of the indicator allows eliminating the relapse of the disease (sensitivity of about 100%) [26].

### **Blood Troponin I(Examples of Interpretation)**

Data Research \_\_\_\_\_

Name patient \_\_\_\_\_

<b>Indicator</b>	<b>The values of the indicators in the patient</b>	<b>Norm</b>
TroponinI	19.0	to 0.5 ng / ml

### **Etalon response**

Myocytes staff taking part in the regulation of muscle contraction. Increasing the level of troponin in the blood plasma reflects the presence of myocardial necrosis, regardless of the mechanism and cause of its occurrence. The degree of its elevation is

high, which may be a sign of development of acute myocardial infarction (normalization of troponin I level occurs after 10-12 days).

### **HEARTFAILURE[21, 25,28]**

**Acute HF** (AHF) is a hemodynamic state with a rapid onset due to heart dysfunction and the development of clinical signs of HF, which manifests itself in the following clinical forms: 1) acute decompensated HF (originated for the first time or as manifestation of decompensation of CHF); 2) pulmonary edema; 3) hypertensive GSN, 4) cardiogenic shock, 5) HF with high cardiac output, 6) acute right heart failure, classified according to the most commonly used classification of AHF for T.Killip (1967)[25].

#### **Classification of acute HF (AHF) by T.Killip (1967)[21, 25]**

**I st.**- without signs of HF;

**II st.** -with weak or moderate cardiac insufficiency, with moist wheezes no more than 50% of the area of the lungs, and/or the third tone on the apex of the heart;

**III st.** -with a clinic of pulmonary edema, with wet wheezing more than 50% of the surface of the lungs, III ton at the apex of the heart;

**IV st.** -with cardiogenic shock, peripheral vasoconstriction, cyanosis, cold sweat, oliguria, periodic "eclipse" of consciousness.

#### **Classification of pulmonary edema by origin[21, 25]**

**1. Cardiogenic pulmonary edema**(with the increase of hydrostatic pressure in the pulmonary arterial capsules when the blood flow from the small circle decreases or its inflow into the pulmonary artery).

**2. Toxic pulmonary edema**(damage to the alveolar capillary membranes with increased penetration and production of alveolar-bronchial secretion).

**3. Neurogenic pulmonary edema**(central nervous system disease).

**4. Swelling of the lungs with a change in the pressure gradient** in pulmonary capillaries and alveoli during respiration versus inspiratory resistance (laryngospasm, stenotic hypostasis of the larynx, artificial ventilation of the lungs with negative pressure on the exudation).

**5. Swelling of the lungs with decreased oncotic pressure**(hypoalbuminemia).

**6. Swelling of the lungs with insufficiency of the lymphatic vessels.**

**7. Swelling of the lungs with an increase in negative intracranial pressure**(rapid removal of pleural effusion).

**8. Swelling of the lungs with uremia with an increase in the sensitivity of the capillaries and heart failure with the phenomena of congestion.**

**9. Swelling of the lungs with overdose of narcotic substances**(heroin, methadone).

**Cardiogenic shock, classification according to the mechanism of development[21, 25]:**

- **Reflex** related to pain;
- **"True"** due to violation of myocardial contractility;
- **irreducible**, the most severe, associated with significant metabolic disturbances, disseminated intravascular coagulation;
- **Arrhythmic** on the background of reducing the minute volume of blood in arrhythmias.

**Cardiogenic shock, classification by gravity of the course[21, 23, 25]:**

**I degree** -middle form, accompanied by AT 90/50-60/40 mm Hg, heart rate 100-110 for 1 min, cardiac index (CI) > 1.8 L/min/m<sup>2</sup>, diastolic pressure of the pulmonary artery (PAP) <24 mmHg, central venous pressure (CVP) <150 mmHg, diuresis > 20 ml/h; arterial hypotension is maintained for 3-5 hours.

**II degree** is a severe form, the level of AT 80/50-40/20 mm Hg, heart rate - 110-120 for 1 min, CI- 1,5-1,8 l/min/m<sup>2</sup>, PAP - 24-30 mmHg, CVT increases to 240 mmHg, diuresis <20 ml/h with anuria phenomena; Hypotension is maintained > 5-10 hours.

**III degree** -a reactive form is associated with the significant deterioration of the forecast as very difficult patients, blood pressure you detect right way, CI <1.5 L/min/m<sup>2</sup>, HR > 120 for 1 min, PAP >30 mmHg, CVP >250 mmHg, anuria, short unsteady pressure reaction, irradiation, and further development of acute cardiac insufficiency with alveolar pulmonary edema are determined.

## Classification of chronic heart failure (Adapted Clinical Practice 2016)[28]

### *Basic terms:*

- Clinical stage of HF
- Option HF
- Functional class (FC)

**Codes for ICD-10: I 50, I 50.0, I 50.9.**

**Clinical stages:**I;II A;II B;III

**HF<sub>I</sub>, HF<sub>II A</sub>, HF<sub>II B</sub> and HF<sub>III</sub>** meet the criteria of I, II A, II B and III stages of chronic blood circulation insufficiency according to classification Strazheska and V.H.Vasilenko (1935):

**I**- initial blood circulation insufficiency; Is detected only when physical activity (shortness of breath, tachycardia, fatigue); At rest, hemodynamics and organ function are not affected.

**II**- expressed prolonged circulatory failure; violation of hemodynamics (stagnation in the small and large circulations of blood circulation, etc.), disorders of the function of organs and metabolism expressed in peace;

**stage A**- the beginning of the stage; the violation of hemodynamics is expressed moderately; there is a disturbance of the function of the heart or only one of its departments;

stage **B**- deep disorders of hemodynamics, affects the entire cardiovascular system.

**III**- ultimate, dystrophic insufficiency of blood circulation; severe violation of hemodynamics, persistent changes in metabolism and organ functions, irreversible changes in the structure of tissues and organs.

### **Options for heart failure[11,28]:**

- with a lower left ventricular ejection fraction (LV EF  $\leq$  40%);
- with a preserved ejection fraction of LV (LV EF > 40%).

### **Complaints and objective evidence typical of CH**

<i>Complaints</i>		<i>Objective signs</i>	
<i>Typical</i>	<i>Less typical</i>	<i>More specific</i>	<i>Less specific</i>
Dyspnea	Night cough	Increased pressure in jugular veins	Weight gain (>2 kg/week)
Orthopnea	Whistling wheezing	Third tone of the	Weight loss (with
Paroxysmal			

nocturnal dyspnea Reduced tolerance to physical activity Weakness, fatigue, increase the time required for recovery after exercise Edema of the ankles	The feeling of breaking Loss of appetite Confusion of consciousness (in particular, in the elderly) Depression Palpitation Dizziness Faint Bondopnea [40]	heart (rhythm of the gallop) Lateral displacement of apical impulse	severe HF) Loss of peripheral tissues (cachexia) Heart noise Peripheral edema (bone, lumbar, scrotum) Pelenary crepitation Air flow limitation and percussion dullness in the basal lung (pleural effusion) Tachycardia Irregular pulse Tahifone Cheney-Stokes breathing Hepatomegaly Ascites Cold limbs Oliguria Low pulse pressure
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### **Classification of CHF by functional classes [11, 28]**

**FC I-** Shortness of breath and palpitation are absent under ordinary load, physical activity tolerance meets the standards, maximum oxygen consumption is more than 21 ml/kg (7-16 Mt.Od).

**FC II-** Moderate physical activity restriction, shortness of breath and heartbeat arise at ordinary load, exercise tolerance corresponds to 75-100 W in a sample with dosed physical activity - bicycle ergometry, maximum oxygen intake of 14-21 ml/kg (4-7 Met.Od ), patients with CHF I and CHF IIA after adequate treatment.

**FC III-** Significant limitation of physical activity, shortness of breath and heartbeat arise with insignificant loading, physical tolerance corresponds to 50 W, maximum oxygen intake is 7-14 ml/kg (2-4 Mets.Od), patients with CHF IIA before or without treatment special treatment effect, IIB after adequate treatment.

**FC III-** Shortness of breath with insignificant loading and rest, exercise tolerance corresponds to 25 W, maximum oxygen consumption is less than 7 ml/kg (2 Met.Od), patients with CHF IIB without a special effect from treatment, III st.

### **DYSLIPOPROTEIDEMIA[29]**

**Dyslipidemia** should be considered a violation of the function and/or composition of blood lipids and lipoproteins, which may be a consequence of many causes and can either alone or in conjunction with other risk factors, to induce an atherosclerotic process manifestation[29]. First of all, the increase in total cholesterol (TC) and low density lipoprotein cholesterol (LDL) should be taken into account, as it is these disorders that are associated with an increase in cardiovascular risk (CVR).

For analyzes, standardized, accessible methods are used. Test methods can be widely used with test strips to determine the level of general X-rays and other lipids and lipoproteins. Most often, only three components of the lipid spectrum are determined by the laboratory, namely, cholesterol, triglycerides (TG) and high density lipoprotein cholesterol (HDL cholesterol). Prognostically significant LDL cholesterol (LDL cholesterol) in this situation is calculated by the formula Friedewald \*[29]:

- in mmoles/liter:

$$\text{Cholesterol LDL} = \text{total cholesterol} - \text{HDL cholesterol} - (0.45 \times \text{TG})$$

- in mg/dl:

$$\text{Cholesterol LDL} = \text{total cholesterol} - \text{HDL cholesterol} - (0.2 \times \text{TG})$$

\* The calculation is valid only if the TG concentration is less than 4.5 mmol/l (400 mg/dl). Error in the determination of HDL cholesterol or in the determination of TG in patients who have violated dietary recommendations before donating blood can steadily lead to an error in the calculation of the most prognostically significant LDL cholesterol!

Non-HDL cholesterol is calculated by simply deducting HDL cholesterol from the total cholesterol and, unlike LDL chole, does not require TG to be less than 5 mmol/L. To evaluate SSR, non-HDL cholesterol <4 mmol/l (150 mg / dL) should be considered as the target level of therapy[29].

For the characterization of hyperlipoproteinemia the most common is the WHO classification.

**Classification by hiperlipoproteynemiyD.Fredrickson(1970)[29]**

<b>Phenotype</b>	<b>Plasma cholesterol</b>	<b>Triglycerides</b>	<b>LP changes</b>	<b>Atherogenicity</b>
I	Increased	Increased or normal	Improvement of CM	Non-atherogenous phenotype
IIa	Increased	Normal	Increase LDL	High
II b	Increased	Increased	Increase LDL and LDL	High
III	Increased	Increased	Increased hypertension	High
IV	More often than normal	Increased	Increase LDL	Moderate
V	Increased	Increased	Increase of CM and VLDL	Low

Clinical classification of dyslipidemias of the Ukrainian Scientific Society of Cardiology (2011)[29]:

1. Hypercholesterolemia (type IIa responsible forD.Fredrickson).
2. Combined dyslipidemia (corresponding to type IIband III for typeD.Fredrickson).
3. Hypertriglyceridemia (corresponding to the type andVbyD.Fredrickson).

For the implementation of measures for primary and secondary prevention of cardiovascular complications, in the treatment of dyslipidemia, target levels are primarily based on the results of clinical trials. Lately, in all lipid lowering studies, the level of LDL cholesterol is used as an indicator of response to therapy. Therefore, the level of LDL cholesterol remains the primary objective in most strategies for the treatment of dyslipidemia.

### Recommendations on target levels of LDL cholesterol [29]

Recommendations	Class of recommendations	Level of evidence
In patients with VERY HIGH CVR (CHF, type 2 diabetes, type 1 diabetes with target organ damage, moderate or severe chronic kidney disease or SCORE risk $\geq 10\%$ ) target LDL cholesterol level $<1,8$ mmol/L (less than 70 mg/dL) and/or $\geq 50\%$ reduction if the target levels were not achieved	<b>I</b>	<b>A</b>
In patients with HIGH CVR (a significant increase in one risk factor, a risk of SCORE $\geq 5$ to $<10\%$ ), it is advisable to reach the target LDL level $<2.5$ mmol/L (less than 100 mg/dL)	<b>IIa</b>	<b>A</b>
In patients with MODERATE CVR (SCORE <sub>risk</sub> $\geq 1$ to $<5\%$ ), it is advisable to achieve a target LDL level of $<3$ mmol/L (less than 115 mg/dL)	<b>IIa</b>	<b>C</b>

If it is not possible to detect LDL cholesterol, the following target levels of general TC should be used according to European guidelines (2016): less than 5 mmol/l for the general population, less than 4.5 mmol/l for high-risk patients and less than 4 mmol/L for patients with very high risk.

• **Assessment of the SCORE scale[29, 30].**

Cardiovascular Risk (CVR) should be considered an individual risk of developing atherosclerotic cardiovascular events for a specified time. The risk assessment principles can be defined as follows:

1. Patients with established cardiovascular disease (CVD), type 2 diabetes mellitus, type 1 diabetes with microalbuminuria, with very high manifestations of only one risk factor (total cholesterol  $> 8$  mmol/l (320 mg/dL), LDL cholesterol  $> 6$  mmol/l (240 mg/dl), arterial pressure (AT)  $> 180/110$  mm Hg), chronic kidney disease (CKD) are classified as patients with high and very high cardiovascular risk and require the active modification of all risk factors.



2. For all other patients, you should use the SCORE scale. Score (SCORE) (Systematic Coronary Risk Assessment) has been developed to assess this risk over a 10-year period. Cohort studies were conducted on the basis of the scale in 12 European countries, with a total of 205178 people. Ukraine belongs to a group of high-risk countries. The individual 10-year risk of cardiovascular events is determined by the SCORE tables according to the patient's gender, smoker status, blood pressure and total cholesterol level. The resulting figure is expressed as a percentage and represents the probability of occurrence of cardiovascular mortality for 10 years. Depending on the patient's risk, the patient should be classified into one of the categories:

- Low risk - less than 5%
- High risk - 5% or more

It is necessary to consider the presence of additional factors that increase the level of cardiovascular risk. It can be higher than the one defined by SCORE:

- in socially vulnerable groups of the population;
- In sedentary patients with a central type of obesity;
- in patients with diabetes mellitus, which is three times higher than men and five times among women, than determined by SCORE;
- there are signs of subclinical atherosclerosis or thickening of the intima-media complex (KIM) according to ultrasound examination of common carotid arteries, computed tomography;
- left ventricular hypertrophy (according to ECG or echocardiography);
- Early development of cardiovascular complications in the immediate family;
- low HDL cholesterol and hypertriglyceridemia;
- increase the level of inflammation markers (C - reactive protein (CRP), fibrinogen, homocysteine).

In the general recommendations of the European Society of Cardiologists and the European Society for Atherosclerosis, devoted to dyslipidemia, four variants of the SCORE table are proposed for the determination of the individual CVR in this patient population, depending on the level of HDL - 0.8 mmol/l; 1.0 mmol/L; 1.4 mmol/L and 1.8 mmol/L [30].

Definition of an individual CVR is necessary for carrying out of the full complex of diagnostic and medical measures. Taking into account all factors, the following levels of the general SSR were proposed[30]:

### Level of totalcardiovascular risk

Total CVR (SCORE),%	Level of LDL cholesterol				
	<70 mg/dl, <1.8mmol/l	70-100 mg/dl, 1.8-2.5 mmol/L	100-155 mg/dl, 2.5-4.0mmol/L	155-190 mg/dl, 4.0-4.9mmol/L	>190 mg/dl, >4.9 mmol/l
<1	No need to interfere	Does not require interferences	Lifestyle change	Lifestyle change	Lifestyle changes, with inefficiencies - apply medications
Class A/Level b	I/C	I/C	I/C	I/C	IIA/A
≥1 to <5	Lifestyle change	Lifestyle change	Lifestyle changes, with inefficiencies - apply medications	Lifestyle changes, with inefficiencies - apply medications	Lifestyle changes, with inefficiencies - apply medications
Class A / Level b	I/C	I/C	IIA/A	IIA/A	I/A
≥ 5 to <10 or high risk	Changing lifestyle, applying medication	Changing lifestyle, applying medication	Changing lifestyle and immediate use of medication	Changing lifestyle and immediate use of medication	Changing lifestyle and immediate use of medication
Class A / Level b	IIA/A	IIA/A	IIA/A	I/A	I/A
≥ 10 or very high risk	Changing lifestyle, applying medication	Changing lifestyle and immediate use of medication	Changing lifestyle and immediate use of medication	Changing lifestyle and immediate use of medication	Changing lifestyle and immediate use of medication
Class A / Level b	IIA/A	IIA/A	I/A	I/A	

#### 1. Very high risk

Installs in patients who have: documented CVD according to invasive or noninvasive testing (coronary angiography, registration of atherosclerotic plaques in ultrasonography of common carotid arteries), history of myocardial infarction, coronary revascularization (percutaneous coronary intervention (PCI), aorto coronary artery bypass graft (CABG) and other arterial revascularization, ischemic stroke, peripheral arterial disease;

Type 2 diabetes, type 1 diabetes with the presence of organ damage; moderate and severe (GFR<60 ml/min/1.73 m<sup>2</sup>);calculated using tables for 10 years risk SCORE ≥ 10%.

**2. High risk**

It is established in patients with: a significant increase in only one risk factor (total cholesterol> 8 mmol/l (320 mg/dl); LDL cholesterol> 6 mmol/l (240 mg/dL); AT> 180/110 mmHg. Art.), family dyslipidemia; calculated using tables for 10 years risk 5% ≤ SCORE <10%).

**3. Moderate risk**

Installed in patients who have a calculated risk score of 1% ≤ SCORE <5%).This risk is subject to change with regard to the early onset of CVD in the family history, abdominal obesity, physical activity, HDL cholesterol, TG, CRP, lipoprotein, fibrinogen, homocysteine, apolipoprotein B, and social class.

**4. Low risk**

Installed in patients with a calculated risk of SCORE for 10 years less than 1%.

In the future, the patient management strategy is determined depending on the general CVR and LDL cholesterol levels.

**Biochemical studies of blood(sample interpretationlipidspectrum)**

**Date of study** \_\_\_\_\_

**Name patient** \_\_\_\_\_, male,

	<b>Biochemical indicators</b>	<b>Patient Indicators</b>	<b>Target levels</b>
1	Cholesterol is common	5.3 mmol/l	
2	Triglycerides	1,09 mmol/l	<1.7 mmol/l
3	Cholesterol is a high density lipoprotein	1,32 mmol/l	-For men>1,0 mmol/l -For women>1,2 mmol/l
4	Cholesterol is a low density lipoprotein	3.48 mmol/l	

### **Etalon response**

The level of total cholesterol exceeds the target levels for both the general population (<5.0 mmol/L) and for patients with high cardiovascular risk (<4.5 mmol/L) and very high cardiovascular risk (<4,0mmol/L).

Elevated LDL cholesterol levels for patients with moderate cardiovascular risk (<3 mmol/l) and for patients with high cardiovascular risk (<2.5 mmol/l) and very high cardiovascular risk (<1,8mmol/L).

Determining the level of HDL cholesterol and triglycerides is important in determining cardiovascular risk.

### **CARDIO-PULMONARY RESUSCITATION[9, 31, 32]**

All states requiring cardio-pulmonary resuscitation measures are united by the concept of "clinical death" characterized by stopping of breathing and circulation [31]. This is understood not only by the complete mechanical stop of the heart, but also by the kind of cardiac activity that does not provide the minimum required level of blood circulation. Such a condition can develop with various life-threatening cardiac arrhythmias: ventricular fibrillation, complete transverse (atrial-ventricular) blockade, accompanied by Morgani-Edem-Stokes attacks, paroxysmal ventricular tachycardia, electro-mechanical dissociation, and others.

The cause of the primary stop of blood circulation can be acute myocardial infarction, electrolyte imbalance, thromboembolism of the pulmonary artery, rupture and stratification of the aneurysm of the aorta, etc. Other diseases - cardiomyopathy (diseases of the heart muscle), bronchial asthma, hemorrhagic stroke (cerebral hemorrhage) - lead to sudden death more rarely. One of the causes of sudden cardiac arrest in people of middle age is alcohol abuse. The defeat of the heart, which occurs at the same time, is caused by prolonged toxic effect of the alcohol metabolite product—acetaldehyde[31, 32].

The immediate cause of sudden cardiac arrest is the ventricular fibrillation, much less often the reason is the asystole (no shortening) of the ventricles. Ventricular fibrillation is manifested by non-rhythmic uncoordinated ineffective contractions of individual muscle fibers or their groups. Ventricular fibrillation leads to a sudden

cessation of blood circulation, manifested by the onset of clinical death - in most patients instantaneously, within a few seconds, without prior complaints[9, 31].

Some patients soon before loss of consciousness pay attention to sharp weakness, acute pain in the area of the heart, shortness of breath, which suddenly arose, interruptions in the area of the heart. Loss of consciousness may be accompanied by seizures, involuntary urination. Breathing becomes liquid, then stops. The skin is initially pale gray, then becomes cyanotic. Pupils expand, their reaction to light is absent. Pulse and arterial pressure are not determined, tones of the heart are not heard. Sudden death often occurs during wakefulness - in the morning or evening hours. Signs of a stroke: loss of consciousness; absence of pulse on the carotid arteries; stop breathing; convulsions; the expansion of the pupils and their lack of reaction to light; change in the color of the skin. For the confirmation of cardiac arrest there is sufficient presence of the first three signs[9, 31].

Spend time on measuring blood pressure, for auscultation of the heart should not be - irreversible changes in the higher parts of the central nervous system come already in 3-4 minutes after the cessation of ventricular contraction and blood circulation. Therefore, resuscitation measures should be started immediately, at the time of sudden death. Cardiopulmonary resuscitation (CPR) is not shown, and it can not be started in cases: if it has been determined that more than 25 minutes have elapsed since the heart stop (at normal ambient temperature); at the terminal stages of oncological disease. CPR does not take place if patients have legally recorded their refusal of resuscitation measures in advance[9, 31].

### **Cardiopulmonary resuscitation[31, 32]**

It includes elementary life support and consists of the following activities:

- 1) indirect heart massage;
- 2) restoration of airway patency;
- 3) artificial ventilation of lungs (ventilation of lungs) and oxygenation.

In addition, there are activities of a specialized reanimation complex (for AP Zilber), which includes:

- electrocardiography and defibrillation;
- provision of venous access and the introduction of medicines;

- tracheal intubation.

**Emergency care algorithm (CAB-algorithm) [31, 32]**

Across the globe, the CAB algorithm is used to provide emergency care, - abbreviations from English words:

**Circulation-** blood circulation, (C)

**Airway-** passage of the airways, (A)

**Breathing-** breathing, (B).

According to the recommendations of the AHA(2017) and the European Resuscitation Council (2017), a change in the sequence of actions confirmed when a CPR is performed by a resuscitation unit: first, compression chest compressions must be performed before performing artificial respiration (C-A-B instead of AB- C) to reduce the delay before the first compression. One resuscitator should start CPR with 30 compression chest compressions, followed by 2 breaths. CPR should be performed qualitatively: Compressive compression of the chest should be performed with proper frequency and depth of depression with full chest straightening after each compression, with minimum intervals between compressions and no loss of lung ventilation[31, 32].

The recommended compression rate is from 100 to 120 per minute.

The recommended depth of compressive compression of the chest for adults is not less than 5 cm but not more than 6 cm.

When performing artificial respiration, you should spend about 1 second on breathing, providing sufficient volume for the visual lift of the chest. The ratio of compressions with inhaled remains 30: 2. You should not interrupt chest compressions for more than 5 seconds. for breathing [31, 32].

- Defibrillation, performed 3-5 minutes after the development of cardiac arrest, increases survival rate to 50-70%. Early defibrillation is possible if an auto external defibrillator (AED) is available in public places [31, 32].

- The CPR sequence in adults can be safely applied to children with heart failure. The depth of compression of the chest in children should be not less than one third of its depth (for infants it is up to 4 cm, for children up to 5 cm).

- Severe obstruction of the respiratory tract, caused by foreign bodies, refers to urgent conditions. You should start to render assistance immediately by striking the back, and if this is ineffective, then do a thrust into the stomach. If the victim has lost consciousness, it is necessary to immediately start CPR, simultaneously calling for help [31, 32].

**Carrying out an indirect heart massage[31, 32]**

Before CPR begin in the absence of the affected ripple on the sleep or femoral artery.

The victim should be in a horizontal position on the back, on a solid and level basis. His head should not be higher than the level of the chest, legs should be raised. The position of the rescuer's hands is on the sternum of the victim (two transverse fingers from the base of the bellied appendage upwards), then both hands are parallel to each other, one on the other ("in the castle") located in the lower third of the sternum (Fig.11,12,13).Then it is necessary to begin compression compression of the chest with a frequency of 100-120 per minute to a depth of 5-6 cm[31, 32].

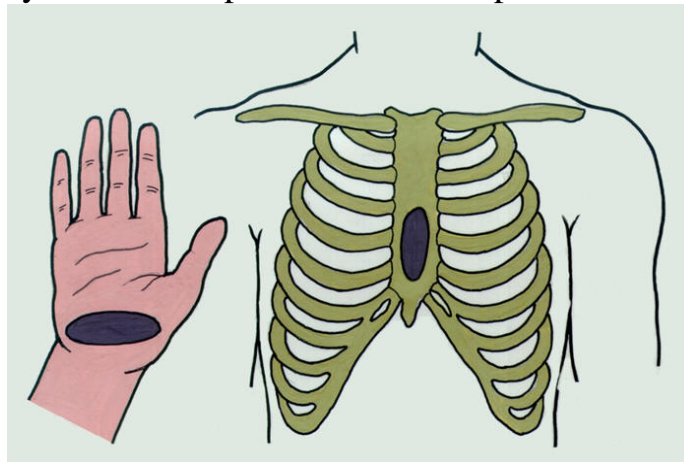


Fig.11.Place of collision of the arm and sternum with indirectheart massage.

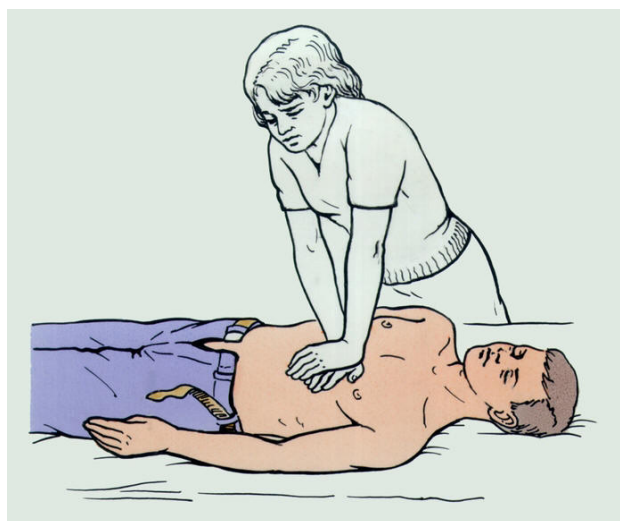


Fig.12.Situation of the patient and the resuscitation in the indirect massage of the heart.

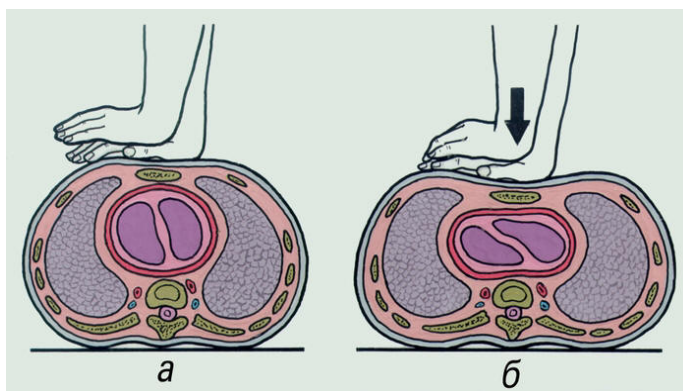


Fig.13.Scheme of indirect heart massage:  
 and -imposing hands on the sternum;  
 b -pressing on the sternum.

The ratio of the number of compressions to the frequency of breathing without intubation for both one and for two resuscitators should be 30: 2 (ie, after 30 compressions, 2 injections are necessary) and synchronized if the trachea is intubated - compression of the chest should be performed at a frequency of 100 -120/min, ventilation with a frequency of 10/min, asynchronously (since compression of the chest with simultaneous pulmonary stimulation increases coronary perfusion pressure)[31, 32].

When conducting indirect cardiac massage, some rules should be observed.First press on the chest cavity smoothly, try to determine its elasticity.Do not do jerky



movements, this is the right way to break the chest. Try to work with hands completely straightened in the elbow joints perpendicular to the chest, using not the strength of the hands, but the mass of the trunk. This saves strength and increases the effectiveness of the massage. If everything is done correctly, a pulse should appear on the sleepy and femoral arteries[31, 32].

The effectiveness of the massage is determined by the release of blood from the heart as a result of compression between the sternum and vertebral column and extrusion of blood from the lungs.

**Complications of indirect (external) heart massage** - fractures of the ribs - can occur in elderly patients and when pushed to the upper third of the sternum; If you rely on the area of the bellies, then you can cause ruptures of the abdominal cavity[31, 32].

The heart massage is necessarily combined with artificial breathing in the "mouth to mouth" or "mouth to nose" manner. Heart massage and artificial respiration are more convenient for two people[9, 31].

#### **Extensive instruction for conducting heart massage[31, 32]**

1. Put the victim on the back, unclutter the collar or strap.
2. Stand before the knees and again check your heartbeat.
3. Put your wrists on one hand in the area of the heart. The fingers and palms should not push the chest.
4. Fetch the second arm in the back of the first arm (if the injured adult).
5. Do not bend your hands, swing your body forward and backward. Do not bend your hands to click on the heart, otherwise you will immediately get out of power.

**For adults.** When pressing the chest should flex 5-6cm. Make 100-120 impulses per minute.

**For children.** Use only one arm and do approximately 100-120 cautious impulses per minute, pushing the chest 4-5 cm.

**For infants.** Very careful! Make a massage with two fingers, squeezing the chest 2-4 cm at a speed of 100-120 beats per minute. To avoid damage to the liver, click above.

6. Carry a heart massage until the pulse appears or the face and face changes to normal, or until the victim comes to the memory.

## **Restoration of the passage of the respiratory tract[31, 32]**

### **Causes of respiratory passage:**

- Tongue is the most common cause of obstruction of the respiratory tract of the victim in an unconscious state;
- Injury - violation of anatomy, blood, fractures of wave;
- Edema of the larynx or laryngospasm, thermal burn;
- Alien body - the most common cause of obstruction of the respiratory tract in children;
- Infections - films with diphtheria, abscesses.

In case of urgent conditions, respiratory tract permeability is often disturbed as a result of the tongue's passing, aspiration by vomiting, blood. It is necessary to clear the mouth and throat and perform the "triple reception Safar" - to open the head in the cervical spine; push the lower jaw forward and upward; open your mouth. If it is impossible to exclude a fracture of the cervical spine and can not extend the head, they are limited to the protrusion of the jaw and the opening of the mouth[9, 31].

If a denture is an integral part, it is left in the oral cavity, as it retains the contour of the mouth and facilitates the conduct of mechanical ventilation.

When obstructing the respiratory tract, the victim's body is stacked to the side and carried 3-5 sharp blows to the lower part of the palm in the interlopathic area, and then they try to remove the foreign body from the oropharynge with their finger. If this method is ineffective, then follow Haymlykmethod[9].

### **Haymlyk Reception [9]**

If an injured alien body is stuck in the throat and interferes with breathing, and it is in the mind, subdiaphragmatic and abdominal shocks should be performed.

Similar events are also referred to as manual impulses (pneumatic shock method), or Haymlyk's technique.

### **Affected in the mind[9, 31]**

- The victim is in position sitting or standing (fig.14).
- Stand behind the victim and place your foot between the stop of the victim.
- Reach him with his hands behind his waist.

- Squeeze the brush of one hand into a fist, press it with a thumb to the abdominal of the victim in the middle line, slightly above the navel hole, and much lower than the end of the bovine appendix (the rib angle).
- Grasp the hand compressed in your fist with the brush of the other hand and a fast jerk movement directed upwards, press on the stomach of the victim.
- Shocks should be carried out separately and definitely until the alien body is removed or the injured person can not breathe and talk, or until the victim does not fade.
- If the victim is unconscious, lower it to the floor on the leg and follow the next manipulation.

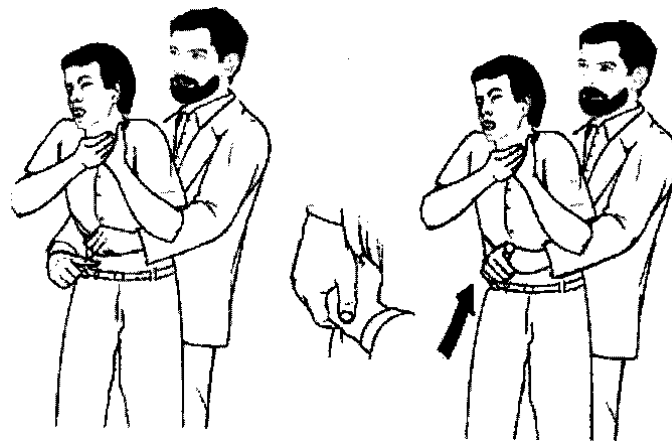


Figure14.Haymlyk's admission to the victimin consciousness

### **The victim is unconscious[9, 31]**

If the victim is unconscious (Figure15), do the following:



Fig.15.Abdominal shock in the unconscious victim

- Put the victim on the back.
- Sit upwards across the hips of the victim, kneeling on the floor, and put one hand on the palm base on his stomach along the middle line, slightly above the navel hole, far enough from the end of the bellied appendix.
- From the top, put the brush of the other hand and press on the abdomen with sharp jerk movements directed to the head, 5 times.
- Check the CAB (blood circulation, airway passage, breathing,).

**Attention:** the contents of the stomach can enter the mouth and then into the respiratory tract, leading to severe pneumonia. To prevent this, after every 5 tapping, check the mouth for the presence of vomiting and remove them.

If the victim was unconscious, the passage of the respiratory tract could not be restored, one should perform a conicotomy after one series of 5 impulses.

Conicotomy (cryocytreatment) consists in the breakdown (puncture) of the rectal membrane with the inability to intubate the trachea or the presence of obstruction in the larynx. The main advantages of this method are the simplicity of the technical execution and the speed of execution (in comparison with tracheostomy).

The glandular membrane is located between the lower edge of the thyroid and the upper edge of the rectal cartilage of the larynx. In this area there are no large vessels and nerves. Conicotomy is performed in the position of maximal extension of the head back. It is better to put a small roller in the hanging area. The large and middle finger must fix the larynx for the lateral surfaces of the thyroid cartilage. A cross-section of the skin is made above the rectangular membrane. By the nail of the index finger, the scalpel perforates the membrane itself, after which a plastic or metal cannula is carried through the opening in the trachea [9].

To facilitate conicotomy special devices are created - konicotomy. The "Partex" disposable sets for konikotomii consist of a knife for opening the skin, trocar and cannula [9].

In children under 8, conicotomy of the puncture is performed.

After that an artificial ventilation of the lungs begins.

### **Artificial lung ventilation[31, 32]**

Indications for artificial ventilation (auxiliary and artificial respiration) of the lungs are a sharp weakening or the absence of independent breathing, which usually arise in the terminal states.

The task of artificial ventilation is the rhythmic injection of air into the lungs in sufficient volume, while the exhalation is carried out by the elasticity of the lungs and chest, that is, passively [9].

The most accessible and widespread in pre-medical resuscitation is a simple method of artificial respiration "mouth to mouth" or "mouth in the nose" (Fig. 16). At the same time in the lungs of the patient can be blown by a double "physiological norm" - up to 1200 ml of air. This is quite enough, as a healthy person breathes in the air for about 600-700 ml of air. Air injected by the helping person is quite suitable for alive because it contains 16% of oxygen (at 21% in the air)[9].

### **The technique of conducting artificial respiration "mouth to mouth" and "mouth in the nose" [9, 31]**

When carrying out artificial ventilation, "mouth to mouth" or "mouth to nose", the patient's head should be thrown back as far back as possible (fig. 16).

At the same time, the position of the head due to the displacement of the root of the tongue and the epiglottis in front opens the larynx and provides free access to the air through it in the trachea[9].

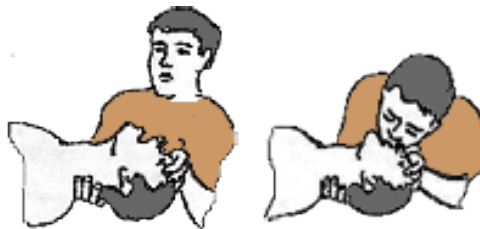


Fig.16. Artificial ventilation "mouth to mouth"

The medical worker, who carries out artificial respiration, is located on the side of the victim, one hand grips his nose, and the other opens his mouth, gently pressing the chin of the patient. It is advisable to cover the mouth of a patient with a gauze or a bandage, after which the medical worker, who conducts artificial ventilation, takes a deep breath, tightly presses his lips to the mouth of the victim and makes a vigorous

exhalation. Then he removes his lips from the patient's mouth and draws his head to the side. Artificial breath is well controlled. Initially, air intake passes easily, but as the lungs fill and stretch, the resistance increases[9].

In the same way as the mouth-to-mouth method, mouth-to-mouth breathing is performed, with the mouth closed by the palm of the patient or by pressing the lower lip to the upper finger[9].

With effective artificial respiration, it is clearly seen how the stomach grows during "breathing"[9].

The most effective mechanical ventilation, carried out with the help of respiratory equipment [9].

The mechanical ventilation is usually carried out through an intubation tube or a tracheotomy cannula, with the help of special respirator devices [9].

Respirators can be divided into three groups [9]:

- respirators regulated by pressure. The devices operate from compressed gas and are used mainly for short-term artificial respiration during transportation of the patient.
- respirators regulated by frequency.
- respirators regulated by volume.

In the event of an emergency, it is necessary to have a laryngoscope, a set of intubation tubes, a bag "Ambu" (RDA-1), a set for tracheostomy.

### **Advanced resuscitation in adults [9, 31, 32]**

The emphasis is on the use of rapid response systems and the prevention of internal hospital stopping of the heart.

- It is necessary to minimize breaks between high-performance chest compressions when performing intensive reanimation: only a brief pause is allowed to perform a specific action. This refers to the execution of defibrillation, for which we suppose to be interrupted for no more than 5 seconds.
- Focus on using self-cleaving electrodes for defibrillation and a defibrillation strategy that minimizes pause before applying. It should not be forgotten that handheld electrodes are used in some situations.

- In the modern recommendations, a new monitoring section was added during intensive intensive care, with an emphasis on the use of a capnography to confirm the correct installation and monitoring of the intubation tube position, assessing the quality of CPR, and timely detection of signs of spontaneous blood circulation regeneration.
- There are different approaches to ensuring respiratory tract compliance during CPR. It is recommended to use a step-by-step approach, taking into account the peculiarities of the clinical situation and the experience of the intensive care unit.
- Recommendations for drug therapy during CPR have not changed, but there is a balance between the role of drugs in improving cardiovascular outcomes.
- Routine use of mechanical devices to compress the chest cells and slime is not recommended, but their use is possible in situations where continued performance quality compresses impossible or threatens the safety reanimator.
- Ultrasound examination during cardiac arrest may play a role in identifying its reverse causes.
- Extracorporeal life-support methods may be effective in a number of patients in the event of ineffectiveness of standard measures of extended reanimation.

### **Heart stop in special circumstances [9, 31, 32]**

This section has been compiled to cover the potentially reversible causes of cardiac arrest that need to be identified or excluded during any CPR. They are divided into two groups of 4G and 4T: hypoxia, hypo / hyperkalemia (or other electrolyte disturbances, hypo- / hyperthermia, hypovolemia, intense (Tension) pneumothorax, cardiac tamponade, thrombosis (coronary or pulmonary artery); toxin and (poisoning) [9, 31].

- Survival after cardiac arrest after asphyxiation is rare. For those who survived the typical severe neurological deficiency. During CPR, it is critically important to have the most effective ventilation of the lungs using oxygen as soon as possible.

A high level of caution and aggressive treatment can prevent heart failure due to electrolyte disturbances.

- Patients with hypothermia and without symptoms of hemodynamic instability should be warmed up with the help of minimally invasive techniques. Patients with

unstable hemodynamics should be sent directly to centers with the ability to perform extracorporeal livelihoods.

- Early recognition and urgent intramuscular use of adrenaline remains the basis of treatment for anaphylaxis.
- Continuation of CPR during transport may be appropriate for a number of patients requiring immediate delivery to the catheterization laboratory and urgent percutaneous coronary intervention (PCI).
- Recommendations for the use of fibrinolytics in pulmonary artery thromboembolism remained unchanged.

### **Special conditions of the place of the event [9, 31, 32]**

This section includes recommendations for the treatment of cardiac arrest, which occurred in some kind of special places. These include health care facilities (for example, surgery, cardiac surgery, catheterization laboratory, dialysis department, jaw-facial surgery), passenger aircraft or sanitation, football field, environmental conditions (eg, drowning, hard to reach places, high mountains, bangs under an avalanche, a lightning strike, an electric trauma) or a catastrophe with a large number of injuries.

- In patients undergoing major heart surgery, the key to the epHu resuscitation is timely recognition of the need for immediate resternotomii, especially in the context of tamponade or bleeding when external chest compressions may be ineffective.
- At cardiac arrest, as a result of arrhythmias that are subject to de-fibrillation (ventricular fibrillation or ventricular tacha-cardiac failure without pulse), during catheterization of the heart, immediately apply up to 3 consecutive times before starting chest compression. In order to provide highly effective compression and reduce the radiation load on personnel during angiography, it is recommended to use devices for mechanical CPR.
- Automatic external defibrillators and equipment necessary for CPR must necessarily be on board all commercial flights in Europe, including regional and budget airlines. If limited access will prevent the implementation of traditional techniques, it should be anticipated that the CPR should be "through the head".



- A sudden cardiac arrest at an athlete on the playing field is likely to have a cardiac origin and requires prompt recognition and prompt defibrillation.
- Immersion in water for more than 10 minutes, and it is associated with a non-favorable result. The role of others in removal of the water and started CPR cover including but important. Priority of reanimation strategies in such situations remain oxygenation and ventilation.

Access difficult and long transportation reduces the chances of a successful outcome of cardiac arrest in hard-to-ground or in the mountains. The role of sanitary care and the availability of ADAs in remote, but often visited places.

- Criteria for the termination of long and extracorporeal CPR at first warming patients with cardiac arrest victims when avalanches become more stringent in order to reduce the number of bad cases of extracorporeal techniques.
- The importance of safety measures when performing CPR for victims of electric shock is emphasized.
- During the events with a large number of victims, rescuers excess resources, individuals who do not serve UT signs of life, CPR is performed.

### **Special patients[9, 31, 32]**

This section contains recommendations for CPR in patients with severe concomitant diseases (eg, asthma, heart failure with ventricular maintenance devices, neurological diseases, obesity) and specific physiological conditions (pregnancy, old age).

- Patients with ventricular maintenance devices may not be able to verify heart failure. If the heart stop occurred within 10 days after surgery and defibrillation is ineffective, rearterotomy should be performed immediately.
- In patients with subarachnoid hemorrhage, ECG changes that are similar to acute coronary syndrome can be recorded. Clinical conclusion will depend on whether a computer tomography was performed before or after coronary angiography.

There is no change in the CPR algorithm in patients with obesity, but in this situation, the effective performance of CPR may require significant physical stress. It should be considered more frequent than the standard recommended 2 minutes, change the resuscitation. It should be traced as soon as possible to intubation.

- With cardiac arrest in pregnant women, high-performance CPR with manual ablation of the uterus remains key, as early as possible transition to advanced reanimation and delivery if there is no rapid recovery of spontaneous blood circulation.

### **Treatment in the postreassonary period [31]**

This is a new section in the Recommendations of the European Council on Resuscitation in 2017. The recommendations of this section have been drawn up by the European Resuscitation Council in cooperation with the European Intensive Care Society, bearing in mind that quality post-transplant care is a vital link in the survival chain.

- Emphasis is placed on the need for urgent coronary catheterization and PCI after an internal hospital stopping of the heart, if its cardiac origin is assumed.
- Support for optimum temperature remains important, but at present the temperature is 36 ° C instead of the previously recommended range of 32-34 ° C. The warning of hyperthermia remains very important.
- Currently, the prediction of the effects of treatment is carried out through a multimodal strategy. It is necessary to give sufficient time for a neurological restoration, waiting for a complete cessation of sedation.
- A new section dedicated to the rehabilitation of survivors after cardiac arrest has been added. The recommendations include a system for treatment in the post-remission period, and should include screening for potential violations of the cognitive and emotional areas and informing the patients.

### **Reanimation measures in pediatrics[31, 32]**

#### **Basic resuscitation measures**

- The duration of artificial inspiration should be about 1 second, as in adults.
- When compressing the chest, the lower part of the sternum should compress at least one-third of the anterior-posterior chest diameter (up to 2-4 cm for infants and up to 4-5 cm for children).

#### **Curation children in critical condition**

- In the absence of signs of septic shock, a child with an illness that is accompanied by hyperthermia should be infused with caution and under constant control. In some

forms of septic shock, the restriction of infusion with isotonic solutions of crystalloids may be better than free use of liquids.

- For cardioversion of ultraviolet tachycardia, the initial dose is revised to 1 J/kg.

### **An algorithm for treating cardiac arrhythmias in pediatrics**

- Most of the provisions coincide with those for adults.

### **Treatment in the postreanimation period**

- After the rehabilitation of spontaneous blood circulation in community-based conditions, prevention of fever is necessary.
- When maintaining temperature, the goal should be either normometry or moderate hypothermia.
- There is no clear prognostic criterion for termination of resuscitation.

### **Reanimation measures in newborns at birth[31, 32]**

- Support in childbirth: the child's situation at birth is unique - it rarely requires resuscitation, but sometimes there is a need for medical care in the immediate post-natal period. The term "childbirth support" was introduced to emphasize the difference between the interventions necessary to restore the functions of vital organs (resuscitation) and support during childbirth.

•**Clamping of the umbilical cord:** at present, uncomplicated newborns are advised to delay the cord clamping from full birth for at least 1 min., Both in term and premature infants.

• **Body temperature:** The temperature of newborns born without asphyxiation should be maintained between 36.5 C and 37.5 C after delivery. The high significance of this position indicates its distinct connection with lethality and complications.

The temperature immediately after birth should be recorded as a prognostic criterion for the result and as a quality indicator.

•**Body temperature support:** at delivery for a term of pregnancy less than 32 weeks. To maintain the temperature between 36.5 C and 37.5 °C in the period from the actual birth to stabilization, a complex of additional interventions may be required. It may include the supply of warmed and wet gases, an increase in the temperature of the room in the room, plus the wrap of the head and body with plastic material, plus the heating of the mattress. Each of these techniques can reduce hypothermia by itself.

- **Optimal estimation heart rate:** in newborns requiring resuscitation, an electrocardiography can provide a quick and accurate estimate of the heart rate.

**Meconium:** tracheal intubation should not be the standard in the presence of meconium, it should only be used in case of suspicion of obstruction of the trachea. Attention should be paid to the initiation of ventilation in the first minute of life in newborns who do not breathe or do it inefficiently - there should be no delay.

- **Air/oxygen:** ventilatory support for newborn infants should begin with air. In prematurely initially, use air or oxygen at low concentrations (up to 30%). If, despite effective ventilation, oxygenation (ideally monitored by oximetry) remains unacceptable, it is advisable to apply oxygen to a higher concentration.

- **Permanent positive pressure in the respiratory tract:** starting respiratory support of the preterm with self-breathing and respiratory distress is better than permanent positive pressure, but not from intubation of the trachea.

**EXAMPLES OF INTERPRETATION OF LABORATORY STUDIES AND  
RENGENOGRAMS [33, 34]**

General blood test (example of interpretation)

Date \_\_\_\_\_

Name:

Indexes	Result	Norm
Leukocytes	5.8	4.0-9.0 x10 /liter
Hemoglobin	86	H: 130-160 g/l G: 120-140 g/l
Red blood cells	2.2	H: 4.0 - 5.0 x <sup>10</sup> <sup>12</sup> /liter G: 3.7- 4.7 x <sup>10</sup> <sup>12</sup> / l
Color index	1.2	0.85-1.05
Reticulocytes	0.5	0.5-1.0%
The average amount of red blood cells(MCV)	113	81-99 μm <sup>3</sup>
Average hemoglobin content (MCH)	57.4	27.0-36.0 pg
The average concentration of hemoglobin in erythrocytes(MCHC)	43	32.0-36.0 g/dl
Hematocrit	30	43-54% J.36-47%
Platelets	160	150-390 x10 /liter
ESR	30	B: <10 F: <15 mm/h

Leukocytes formula

Indexes	Result%	Rate %	Absolute value	Norm
Eosinophils	1	0.5 - 5.0	0,06	0-0.5x <sup>10</sup> <sup>9</sup> /l

Basophils	1	0-1	0,06	0-0.2x10 <sup>9</sup> /l
Myelocytes	0	0	0	0
Metaemyelocytes	0	0	0	0
Core neutrophils	4	1-6	0,23	0.1- 0.6x10 <sup>9</sup> /liter
Segmented Ntrophils	68	50-70	3.94	2.0- 7.2x10 <sup>9</sup> /l
Lymphocytes	17	19-40	0.99	1.2- 3.2x10 <sup>9</sup> /liter
Monocytes	9	3-10	0.52	0.3- 0.8x10 <sup>9</sup> /liter

Etalon response:

In the blood test, decrease in hemoglobin, erythrocytes, increase in color index, erythrocytic indices (MSV, MCH, MCHC) and normal level of reticulocytes are determined. The revealed changes indicate that the patient: hyperchromic, normoregeneratory B<sub>12</sub>-folio deficiency anemia. It can be a consequence of diseases of the gastrointestinal tract (peptic ulcer of the stomach and duodenum, gastritis, gastroduodenitis, etc.), dysbiosis, helminthiasis, oncological disease.

#### Biochemical blood test (example of interpretation)

Date of study \_\_\_\_\_

Name. \_\_\_\_\_

Indicator	Result	Norm
Total protein	48 g/l	66-87 g/l
Albumini	32 g/l	36-50 g/l
Creatinine	200 μmol/l	G: 44-80 μmol/L; H: 62-106 μmol/L
Cholesterol	7.8 mmol/L	< 5.2 mmol/L
Potassium	4.9 mmol/L	3.5-5.3 mmol/l

Sodium	135mmol/l	135-148 mmol/L
Calcium	2.2mmol/l	2.2-2.75 mmol/L
Phosphorus	1.23mmol/l	0.81-1.55 mmol/L
Glomerular filtration rate	55ml/min/1.73 m <sup>2</sup>	≥ 90mL/min/1.73m <sup>2</sup>

Etalon response:

Changes of biochemical parameters characteristic of chronic kidney disease III level, as evidenced by an increase in serum creatinine 200  $\mu\text{mol/l}$  decrease in glomerular filtration rate 55mL/min/1.73m<sup>2</sup>. Reducing the level of total protein and albumin with increasing cholesterol levels is a sign of the presence of nephrotic syndrome in a patient that is inherent in chronic glomerulonephritis, possibly amyloidosis, Kimmelsthyl-Wilson syndrome.

### Chest X-ray (an example of interpretation)



#### **Etalon response.**

Pulmonary fields of normal transparency. Strengthening the pulmonary vascular pattern. Sinuses are free.

The shadow of the heart is typical. Signs of enlargement of the left atrium and left ventricle. Dilation of the right heart. Explosion of the trunk of the pulmonary artery, dilatation of the right heart of the heart.



## **Examples of X-ray conclusion formulation research in some pathological conditions [33,34]**

### **1. Cardiomegaly**

Cardiomegaly is an enlargement of all the parts of the heart, manifested by the expansion of the shadow of the heart. This term includes dilatation (increasing the size by dilatation ventricles) and hypertrophy (increase myocardial mass).

Example of conclusion: the heart is enlarged in the width (all sections, vascular bundle extended).

### **2. Upper lobe Pneumonia.**

Pneumonia is an infectious disease of a variety of etiologies, the morphological substrate of which is infiltration of the pulmonary tissue by the inflammatory element. This is due to the appearance on the X-ray of infrared shadows of various shapes and sizes with fuzzy contours.

Example of conclusion: in the upper pulmonary field on the right there is a section of infiltration with a clear lower and fuzzy upper contour, the average intensity of the inhomogeneous structure, the right root of the extended, high density.

### **3. Hydropneumothorax.**

Hydropneumotorx - the presence in the pleural cavity of air and liquids (exudate, blood). There in connection with the trauma of the chest, but may be due to inflammatory tumors.

Example of conclusion: to the right, the transparency of the pulmonary field is elevated. Pulmonary picture is not defined. In the root zone of the eclipse of an oval shape with clear equal contours, the lung is colligated. At the level of the 5th rib is the horizontal level of the liquid.

### **4. Exudative pleurisy.**

Exudative pleura - inflammation of the pleura, which is accompanied by accumulation of fluid in the pleural cavity. Radiologically detected fluid level with oblique upper limit.

Example of the conclusion: to the left of the level of the 4th rib is an intensive homogeneous eclipse with a oblique border above which merges with the dome of the diaphragm. The right diaphragm dome is clear, sinuses are free.

## **5. Myeloma disease.**

Systemic lesion of bones with formation of foci of myeloid tissue in flat bones (skull bones, pelvic bones, sternum).Diagnosis - X-ray, puncture biopsy of the bone marrow, urine analysis on the Ben Jones protein.

Example of conclusion: in the bones of the skull there are multiple areas of enlightenment - the centers of destruction with clear, even contours.

## SITUATION CASES WITH ANSWERS ATTACHMENTS [34, 35]

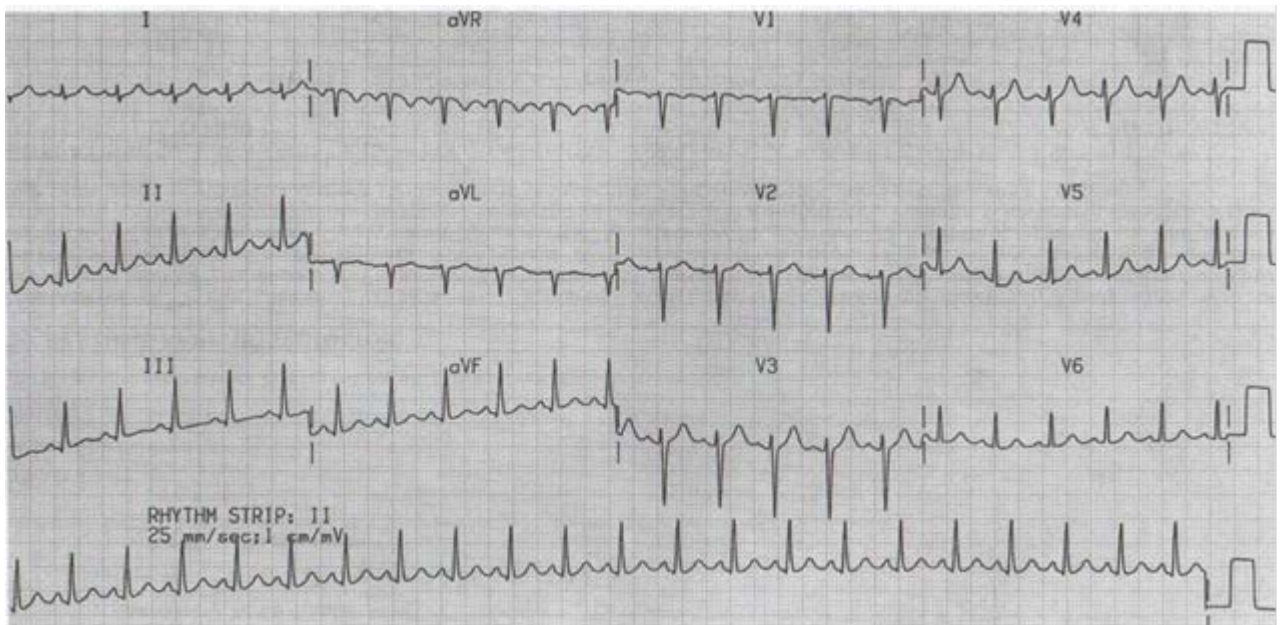
### CASE 1

Patient G., 48, turned to the clinic doctor with complaints of a sharp compression of the sternum, with irradiation in the left shoulder. Such a pain arose for the first time on the way to work. Smokes, alcoholic drinks do not abuse. Objectively: high nutrition. Skin is pale, damp. In the lungs, vesicular breathing, no wheezing. Pulse 92 per minute, rhythmic, satisfactory filling. AT - 155/80 mm Hg. Art. Borders of the heart: right - on the right edge of the sternum, left - 1 cm outside of the left medullary clavicular line. The tones of the heart are muffled, no noise. Abdomen is soft, painless. The liver and spleen are not palpable.

1. To diagnose.
2. Perform differential diagnostics.
3. Design a survey plan.
4. To design a treatment plan.

The results of additional tests to CASE 1:

ECG - attached.



1. General blood test: er.-  $4,5 \times 10^{12}$ , leuc.-  $10,5 \times 10^9$ , e. - 0, n. - 6, segm.-65, l.-22, m.- 7, ESR - 10 mm/h.

2.MB-CFK - 24 OD/L (norm - up to 25 OD/l).Troponin I-0.8 ng/ml (norm to 1.0 ng/ml).

#### Example response to CASE 1

1. CHD: the first occur angina
2. Differential diagnosis is performed between angina pectoris, aortic aneurysm, myocarditis, pericarditis, pleurisy, pneumothorax.
3. The survey plan includes: general analysis of blood in dynamics, ECG in dynamics, blood test for markers of myocardial damage (after 12-24 hours), blood coagulation, chest x-ray, echocardiography, test and exercise, coronary angiography.
4. Plan of treatment: antiagregants, statins, beta-blockers, antihypertensive suppressants.

#### CASE 2

Patient K., 57 years old, was taken to hospital with complaints of intensive compression of the pain behind the breasts with irradiation in the left shoulder, which lasts for 1.5 hours, is not removed by taking nitroglycerin, heart failure, a sharp general weakness, cold sticky sweat. The day before worked physically in the country. In anamnesis - during 4-5 years, notes of compression pain for the sternum during fast walking, which lasts 3-5 minutes, are held calmly and from taking nitroglycerin.

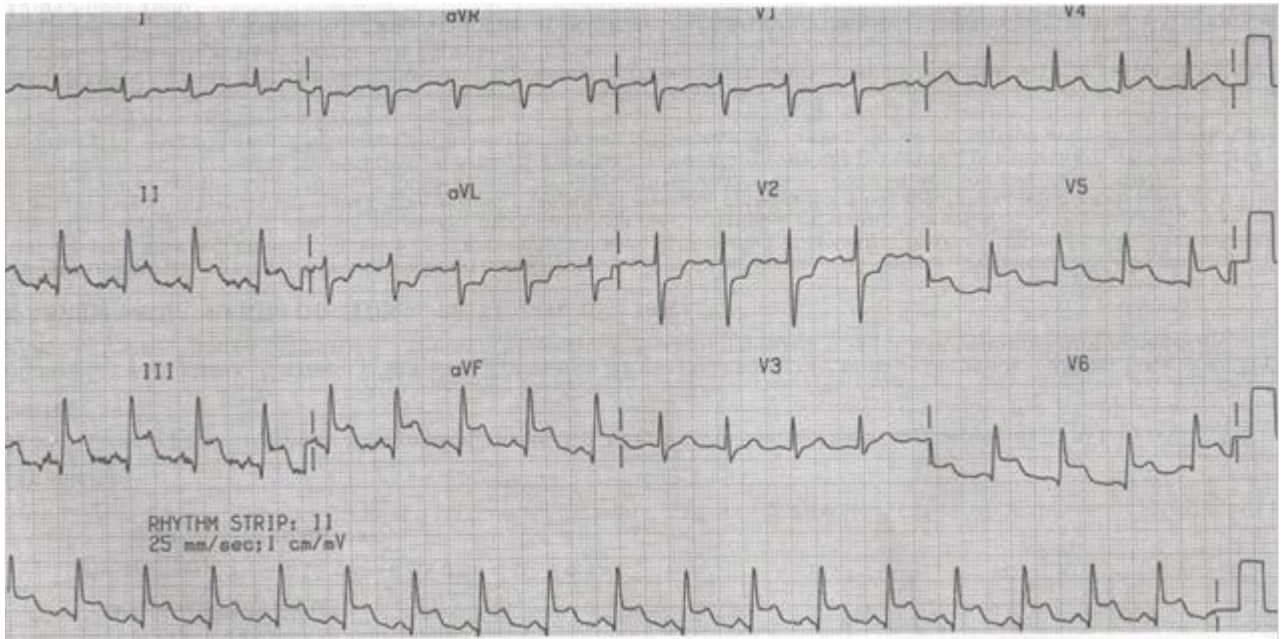
Objectively: the skin is pale, acrocyanosis, palms of the wet. Pulse 96 per minute, single extrasystoles. AT - 90/60 mm Hg. Art. The borders of the heart are enlarged to the left by 1.5 cm. The tones are deaf, single extrasystoles. In the lungs, vesicular breathing. Abdomen is soft, painless. The liver is not palpable.

General blood test: RBC-  $4.3 \times 10^{12}$ , WBC-  $9.2 \times 10^9$ , n. - 4, segm.-66, l.-23, m.-7, ESR - 10 mm/h.

1. Make a diagnosis.
2. Conduct a differential diagnosis.
3. Identify the plan for the follow-up.
4. Assign treatment.

The results of further examination of the CASE 2:

ECG - attached.



2. Serum of blood CK-MB 68 UD/l (norm to 25 OD/l), Troponin I - 158 ng/ml (norm to 1.0 ng/ml).

3. General blood test on the sixth day after hospitalization: WBC-  $6,0 \times 10^9$ , e.- 1, n.- 2, segm.-64., l.- 24, m.- 9, ESR -24 mm/hour.

Examples answers to CASE 2

1. CHD: acute Q myocardial infarction of the inferior wall LV.

2. Differential diagnosis is performed between angina pectoris, pericarditis, myocarditis, cardiomyopathy, aortic aneurysm, pneumothorax, pleurisy, pulmonary embolism.

3. The survey plan includes: general analysis of blood in dynamics, ECG in dynamics, blood test for markers of necrosis of cardiomyocytes, radiography of chest organs, echocardiography, coronary angiography.

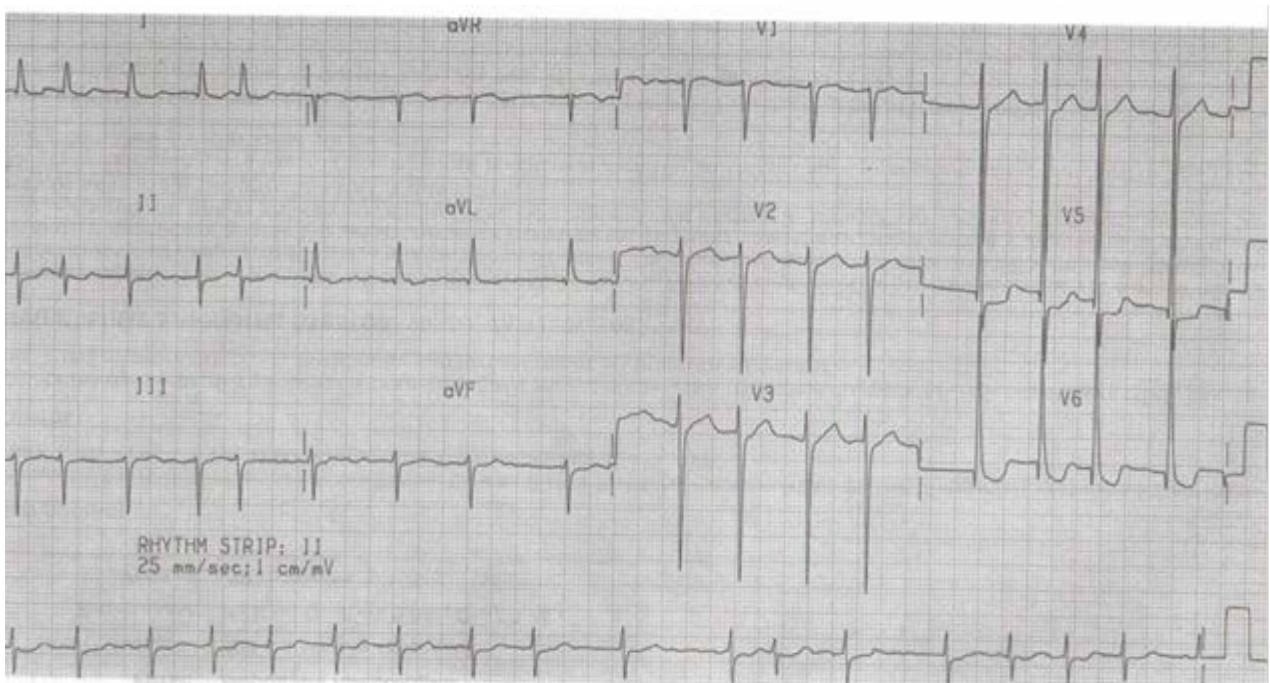
4. Treatment: relief of pain syndrome - narcotic analgesics, oxygen therapy, antiaggregants, anticoagulants, reperfusion therapy, anti-ischemic therapy.

### CASE 3

Patient V., 58 years old, 2 years ago during work in a country house suddenly there was a feeling of frequent disordered palpitation, accompanied by weakness, unpleasant sensations in the heart. Delivered to the hospital admissions office. These feelings of heartbeat, more often during exercise, are noted during the last year. These episodes were short-lived and passed on their own in a state of rest. In the analysis of an outpatient card over the past 2 years, there has been a marked increase in blood pressure and cholesterol (7.6 mmol/L - dominated by low density lipoprotein).

Objectives: pale skin, giperstemic type. In the lungs, vesicular breathing, no wheezing. Left end of heart - on median-clavicular line. AT - 150/100 mm Hg. Art. Pulse on the arteries - frequent, arrhythmic, frequency - 102 in 1 minute. Tones of the heart at the apex have incongruous melodies, arrhythmic, heart rate - 112 in 1 minute. Abdomen is soft, painless. The liver is not enlarged.

1. Establish a preliminary diagnosis.
2. To design a plan for patient examination.
3. Perform differential diagnosis.
4. Define treatment tactics.



Results of additional survey to CASE 3:

ECG - attached.

2. ECHO-CG is a small extension of the left atrium cavity (4.8 cm), signs of LVH. Blood on cholesterol - 7.6 mmol/l, TTG - 2.1 ng/ml (norm 0.1 - 4.1 n/ml), INR - 1.0 OD, PTI - 100%.

3. Eye condition - atherosclerosis of the retina.

4. General urine analysis - weight - 1020, no protein, no sugar, 1 - 1-2 in n/s.

5. Blood analysis for sugar - blood glucose - 4.5 mmol/l.

6. Total blood test: Hb - 140 g/l, RBC-  $4.5 \times 10^{12}/\mu$ , WBC-  $6,0 \times 10^9/l$ , ESR - 6 mm/year.

#### Example response to CASE 3

1. Preliminary diagnosis - Hypertension II stage, 2 degree, very high additional risk. Paroxysmal form of atrial fibrillation.

2. Patient follow-up plan: ECG, Holter monitoring, ECO-CG, electrophysiological examination of the heart, laboratory parameters: electrolytes, thyroid hormones, coagulogram, ocular bottom.

3. Atrial fibrillation as a syndrome with valvular heart defects, thyrotoxicosis, cardiopathy.

4. Treatment: the main task - to treat paroxysm and anticoagulant therapy. Restoration of sinus rhythm: preparations of group 1 (propafenone, flekainid); electropulse therapy. Parenteral anticoagulants (heparin, LMWH) with subsequent conversion to vitamin K antagonists (warfarin) or direct oral anticoagulants (dabigatran, rivaroxaban, apixaban). Treatment for arterial hypertension.

#### CASE 4

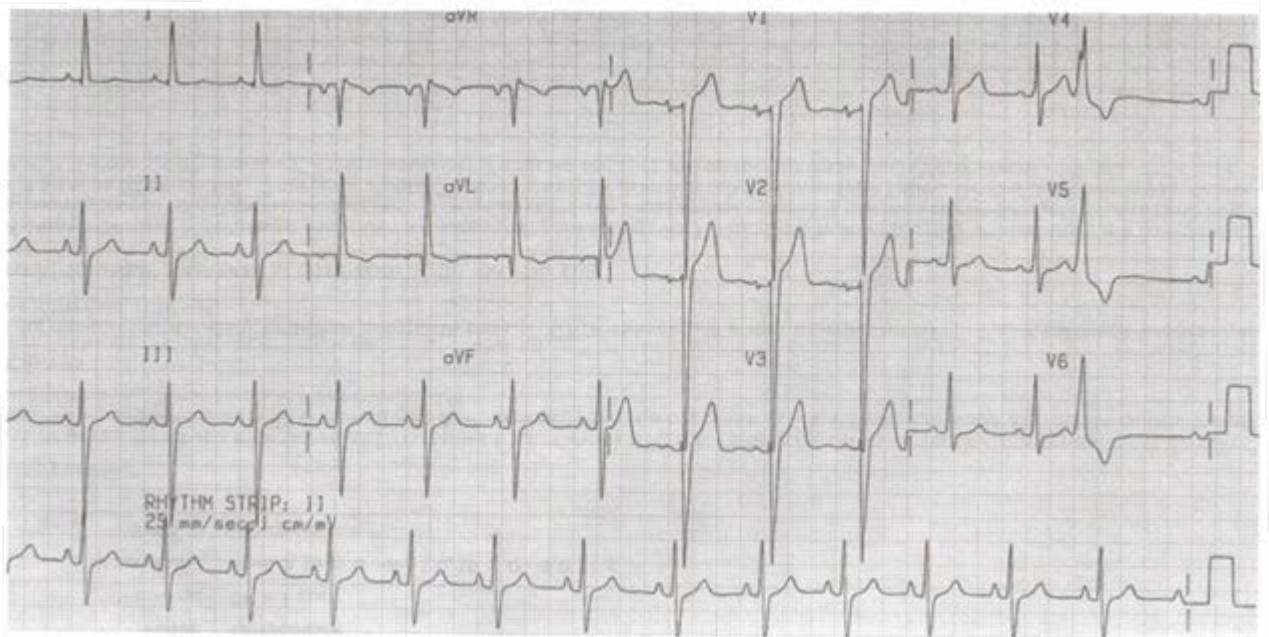
Patient K., 58 years old, admitted with complaints of severe headache in the occipital area of pulsating nature, accompanied by nausea, disposable vomiting, dizziness, the appearance of a "net" in front of the eyes. Headaches occurred earlier, often in the morning or after psycho-emotional stress. For medical help did not address. The last attack of pain arose suddenly against the background of a satisfactory state of health. Before that he was on a business trip, worked hard.

Objectively: a state of moderate severity. The patient is somewhat excited, frightened. The skin covers are clean, high humidity, and there is hyperemia of the face and neck. In the lungs vesicular breathing, no wheezing. Pulse - symmetrical, intense, frequent - 92 in 1 min. AT - on the Ave. Hands - 195/100 mm Hg. Art., on the left - 200/100 mm Hg. Art. The borders of the heart are left - 1.5 cm outside of the left medullary clavicular line. Heart tones are sound, rhythmic, accent P ton on the aorta. Heart rate - 92 in 1 min. Abdomen is soft, painless. The liver is not enlarged. The symptom of Pasternatsky's negative. No edema.

1. Establish a preliminary diagnosis.
2. Design a survey plan.
3. Perform differential diagnosis.
4. Define treatment tactics.

The results of further investigation CASE 4:

1. ECG - attached.



2. The bottom of the eye - the narrowing of the arteries and veins, vessels Salus -II.
3. Urine analysis - weight - 1018, no protein, no sugar, 1 - 1-3 in n/s.



4. Echocardiography - Left ventricular hypertrophy, signs of a hyperkinetic type of hemodynamics.
5. General blood test: RBC -  $132 \text{ g/l}$ , WBC-  $4,5 \times 10^{12} / \text{l}$ , l-  $6.0 \times 10^9 / \text{l}$ , CI- 0.9; e - 1, n - 4, s -66, l-24, m- 5, ESR - 6 mm/hour.
6. Blood glucose - 4.5 mmol/L, Troponin I - 0.08 ng/ml (norm 0.1-1.0 ng / ml).

#### Standards for answers to CASE 4

1. Preliminary diagnosis: Hypertensive disease stage II, 3 degrees, very high additional risk. Uncomplicated hypertensive crisis.
2. Survey plan: ECG, ophthalmology, urinalysis general, ECO-CS, total blood test, blood glucose.
3. Differential diagnosis - the exclusion of secondary arterial hypertension (primarily renal origin, as the most frequent).
4. Treatment: therapy of a hypertensive crisis; therapy for hypertension (diuretics, beta-blockers, ACE inhibitors, calcium antagonists).

#### CASE 5

In the maternity department of M. 35 years, during the first birth, severe pain in the chest appeared, a sharp shortness of breath of mixed nature, lost consciousness.

Objectively: the general condition is heavy, no consciousness, there is a blue-crimson cyanosis of the upper body. Breathing superficial to 50 in 1 minute. When auscultation is in the right half of the chest abruptly weakened, isolated dry wheezing, in the lower parts of the uneven wheezing. The neck veins are swollen, pulse is filiform 100 in 1 minute. AT - 90/40 mm Hg. Hearty tones are deaf, splitting the second tone over the pulmonary artery. Abdomen enlarged, palpation is not available.

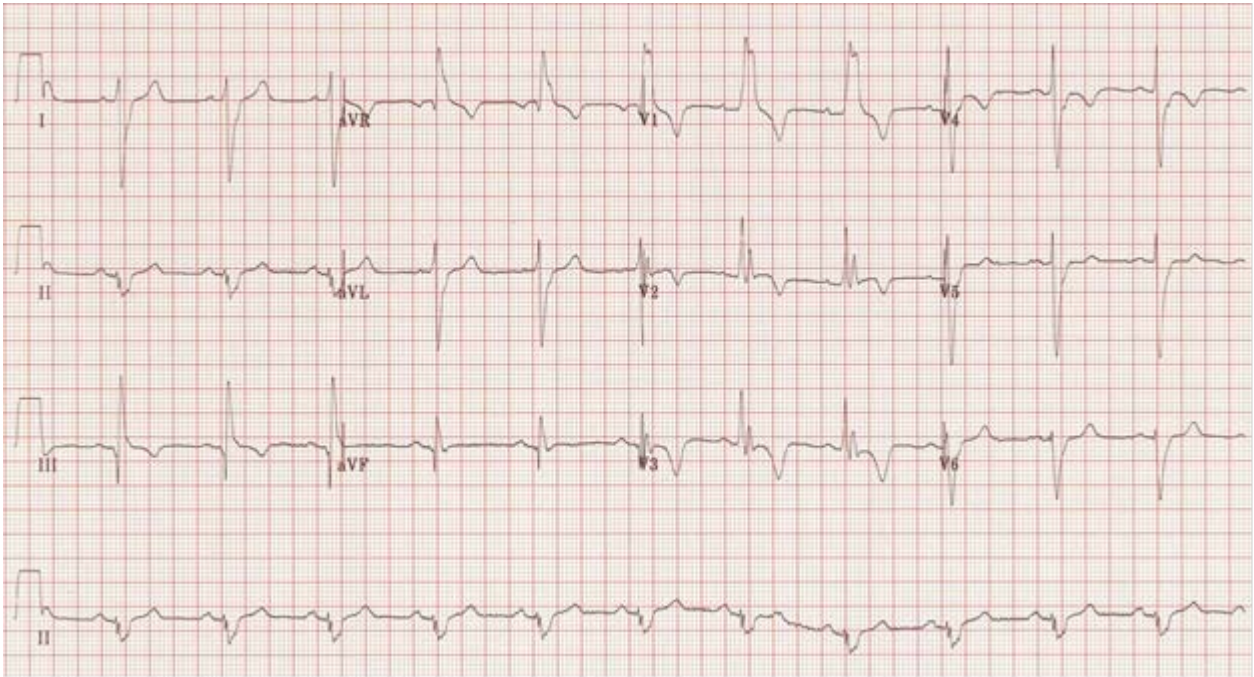
1. Establish a preliminary diagnosis.
2. Plan an additional survey.
3. Perform differential diagnosis.
4. Define treatment tactics.

The results of further examination of the CASE 5:

Blood test: RBC-  $4,5 \times 10^{12}/l$ , HB - 135 g/l, ESR - 15 mm/h, WBC -  $9,5 \times 10^{12}/l$ , p - 2%, s - 65%, e - 2%, m - 10% , l - 21%, protein -80 g/l, albumin - 42%, PTI - 105%.

1. Urine analysis: straw yellow, acid reaction, weight - 1016, leukocytes - 1-2 in n/, ep.cells - 1-2 in n/s.

2. ECG - attached.



Example response to CASE 5

1. Preliminary diagnosis: thromboembolism of the pulmonary artery.

2. General blood test, determination of markers of necrosis of cardiomyocytes, cerebral sodiumuretic peptide, determination of anticoagulant system of blood, examination chest X-ray, electrocardiography, echocardiography, multispiral computed tomography.

3. It is necessary to conduct differential diagnostics with: an attack of angina, myocardial infarction, aneurysm of the aorta, pleurisy, pneumothorax, circopic pneumonia.

4. Fibrinolytic and anticoagulant therapy (heparin). Shock treatment, treating heart failure. Release of pain syndrome. Reduced pressure in the pulmonary artery.

CASE 6

The patient 48 years old complains about pain in the sternum with irradiation in the left shoulder blade when walking in a flat area of more than 500 m. After taking nitroglycerin, the pain goes through 2 minutes. He is ill for about 5 years.

Objectively: BR - 17 for 1 min., Pulse - 76 beats/min., Rhythmic, blood pressure - 135/85 mmHg. The activity of the heart is rhythmic, the accent of the second tone above the aorta. The ECG has not been altered beyond the attack.

Your previous diagnosis?

Therapeutic tactics?

### Electrocardiogram

Date of study \_\_\_\_\_

Name \_\_\_\_\_



### The answer to the CASE 6

RR	0.60 - 0.65 s
Heart rate	92 - 100 per min
PQ	0.18 sec
QRS	0,10 sec
QT	0.36 sec

Rhythm sinus, regular. Sinus tachycardia. The electric heart axis is tilted to the left. Significant deviation of the electrical axis of the heart to the left and the QRS complex in the release of aVL of type qR indicate the presence of a blockade of the anterior-upper branch of the left leg of the His beam. Horizontal depression of the segment ST up to 5mm in leads V1 - V6, which is a sign of subendocardial ischemia in the anterior, medial, apical, anterior-lateral margin of the left ventricle.

In a patient with coronary heart disease: stable angina pectoris II FC, HF-0.

Diagnostic program:

1. Mandatory research:

- Definition of complaints and anamnesis.
- Clinical review.
- measurement of blood pressure.
- laboratory examination (blood and urine tests, serum glucose, chest X, TG, LDL cholesterol, LDL cholesterol, potassium, sodium, creatinine, ALT, AST, bilirubin and its fractions)

- ECG in 12 leads.

- EchoCG

- X-ray of lungs.

- exercise test.

2. Additional studies:

- coagulogram

- Holter ECG.

- Coronary angiography.

- Echocardiography with dobutamine and dipyridamole (in the presence of contraindications for a test with metered-dose exercise).

Treatment of patients with stable angina pectoris consists in reduction of clinical manifestations and prevention of cardiovascular complications.

Compulsory components of treatment for such patients are lifestyle modification (smoking cessation, rational nutrition, regular exercise, body weight control, blood pressure, lipid levels and blood glucose levels), control of risk factors, patient training and pharmacotherapy.

To remove an angina attack, commonly used short-acting nitrates - nitroglycerin 0.5 mg under the tongue and termination of physical activity.

The recommended administration of beta-blockers is preferred over the long-term selective beta-1 blockers, taking into account the need for 24-hour myocardial protection against ischemia (bisoprolol 5-10 mg once daily) or calcium channel blockers that reduce heart rate (diltiazem 60 mg 3 p / d). The dose of drugs is determined by heart rate in a state of rest (the recommended reduction in heart rate to 55-60 beats/min).

All patients who do not have contraindications should take acetylsalicylic acid (ASA) at a dose of 75-150 mg/day. With contraindications or intolerance of ASA, clopidogrel (75 mg 1 day/day) is indicated.

All patients are recommended for taking statins (atorvastatin 20 mg once daily) under the control of lipidograms.

The use of ACE inhibitors (or ARIs) is indicated in the presence of additional indications (such as HF, AH, diabetes, etc.).

## CASE 7

A 57-year-old patient complains of compressive pain over the sternum for more than 30 minutes, which does not diminish after sublingual administration of nitroglycerin. The last 5 days marked the compression of the sternum with irradiation in the left shoulder with considerable physical activity. Doctors did not address. At inspection: the condition is heavy, heart rate - 92 for 1 min, AT - 110/75 mm Hg. ECG: increase of the segment ST in  $V_1$ - $V_3$  leads.

1. Formulate and justify the previous diagnosis.
2. Draw up and validate the inspection and treatment plans.

### Key Answer CASE7

1. CHD. Acute myocardial infarction without Q-wave on the front wall of the left ventricle. The diagnosis is based on the presence of a patient with angina pain lasting more than 15 minutes, which does not decrease after sublingual administration of nitroglycerin, data of the anamnesis (the last 5 days marked the compression of the pain behind the breast with irradiation in the left shoulder under physical activity), the data of the patient's examination, the data of the ECG (arcuate increase of the segment ST in  $V_1$ - $V_3$  leads).

2. Laboratory examination: general tests of blood and urine, CFK in dynamics 3 times, MB-CFK or troponin T or I in dynamics 2 times, ALT, AST, potassium, sodium, bilirubin, creatinine, total cholesterol, triglycerides, blood glucose, coagulogram Echocardiography, ECG in 12 leads in dynamics, coronary angiography.

Treatment includes anesthetics (narcotic analgesics), thrombolytic therapy (actelisis), dual antiplatelet therapy (aspirin and clopidogrel), anticoagulants (enoxaparin), nitrates (with pain syndrome, under the control of AT), statins (atorvastatin), beta-blockers (from 2 days under the control of AT), ACEI (appointment depends on the stabilization of blood pressure), metabolic therapy.

### CASE 8

A 65-year-old patient complains of shortness of breath, general weakness that occurs after exercise, which has been well tolerated before. For 30 years, he has an arterial hypertension with a periodic increase in blood pressure above 180/110 mm Hg. 3 years ago, suffered myocardial infarction. Objectively: the condition of the patient is relatively satisfactory, AT - 150/90 mm Hg., heart rate 88 /min. The left border of the relative dullness of the heart is shifted to the left, an increase of the tone II above the aorta. The arteries of the fundus are narrowed and twisted. ECG: signs of post-myocardial infarction and left ventricular hypertrophy.

1. Formulate a preliminary diagnosis. Substantiate
2. Modify the patient's echocardiography.

3. Draw an ECG according to the description.
4. What is the secondary organ damage in arterial hypertension?
5. Make and justify the patient's treatment plan.

Key answer CASE 8

1. Preliminary diagnosis: AH 3 stage, III degree, hypertensive heart. CHD: postinfarction atherosclerosis. HF. Substantiation:

- Hypertensive disease - a patient 30 years old suffering from arterial hypertension;
- The third stage of hypertension is confirmed by a transient myocardial infarction in the history and ECG-sign of a suffered heart attack;
- About the third degree is shown by the level of arterial pressure over 180/110 mm Hg.;
- About hypertensive heart can be said on the basis of objective data - the left border of the relative dullness of the heart is shifted to the left, in the II intercostal space to the right of the sternum, systolic noise, an increase of II tone over the aorta and ECG-sign of left ventricular hypertrophy;
- Cardiac insufficiency - shortness of breath, general weakness occur after exercise, which has been well tolerated before.

**2. Modify the patient's echocardiography.**

Echocardiography is an important method for diagnosing heart muscle damage in patients with arterial hypertension, since it allows you to evaluate the state of the chambers of the heart, the valvular apparatus, and the functional capacity of the myocardium. During the echocardiography of this patient, it is possible to detect thickening of the walls of the left ventricle, impaired contractility of the myocardium of the lungs in the form of hypokinesia in the area of the rumen on the back wall; The ejection fraction of LV is within the normal range (57%).

**3. Draw an ECG according to the description.**

On paper, you need to display the following changes:

- Signs of left ventricular hypertrophy
  - the left-hand side ( $R_I > R_{II} > R_{III}$  or high  $R_I$ , deep  $S_{III}$ );
  - $R_I + S_{III} \geq 25$  mm;
  - $S_{V_2} + R_{V_5} \geq 35$  mm;

-Transition zone shift in  $V_5$ .

- Signs of myocardial infarction

abnormal wave Q and smoothed wave T in leads II, III, aVF

#### **4. What are the secondary lesions of the target organ in arterial hypertension.**

- from the side of the cardiovascular system there is a development of myocardial infarction, heart failure, layering aneurysm of the aorta;
- the defeat of the nervous system is manifested as a stroke, transient ischemic attacks, acute and chronic hypertensive encephalopathy, vascular dementia;
- renal failure develops from the kidneys;
- At the fundus there are sclerotic changes in arterioles and venules, hemorrhages, exudates in the retina, edema of the optic nerve.

#### **5. Make and justify the patient's treatment plan.**

The purpose of treatment for a patient with hypertension is to reduce the risk of cardiovascular complications, disability and mortality.

**Mode** - normal with the restriction of intense physical activity.

**Diet Therapy**- 4-6 meals a day (diet №10) with a restriction of the kitchen salt up to 3-6 g per day is foreseen; food should be rich in proteins and vitamins with a total caloric content of 1500-2000 calories; quantity of liquid, taking into account liquid dishes, fruits, vegetables - from 800 to 1500 ml/day; food should be consumed products rich in potassium and magnesium (prunes, dried fruits, baked potatoes, etc.).

**Lifestyle modification** by influencing "manageable" risk factors for arterial hypertension - normalization of body weight, reduction of alcohol consumption, smoking cessation.

#### **Drug treatment.**

In order to reduce the level of arterial pressure, this patient is prescribed drugs and series:

- hydrochlorothiazide 12, 5 mg per 1 tablet. day



Hydrochlorothiazide is thiazide diuretic, the basis of which is the hypotensive effect of reducing the reabsorption of sodium and water, and with prolonged use, the reduction of peripheral vascular resistance.

- lisinopril 20 mg per 1 tablet.day

This is a drug that belongs to the group of angiotensin-converting enzyme inhibitors that lower the blood pressure by blocking the activity of the enzyme that converts inactive angiotensin-I into active angiotensin-II, and also increases the concentration of vasodilating substances.

- Metoprolol 100 mg per 1 tablet.day

Metoprolol is a selective beta-blocker, which is based on reducing cardiac output and decreasing the activity of renin in blood plasma by reducing the activity of the sympathetic nervous system.

In order to prevent thromboembolic complications (development of repeated myocardial infarction, stroke), antiplatelet drugs are prescribed:

- Acetylsalicylic acid 100 mg per 1 ton per day after meals.

The patient must be on a clinical examination in order to correct the doses of prescribed drugs, the frequency of their administration, timely detection of complications of hypertension and concomitant illness.

### **Comments:**

1. To diagnose hypertension it is necessary to exclude symptomatic hypertension. It is important to determine the risk stratification factors for quantification of the prognosis, which include the level of blood pressure, sex, age, smoking, dyslipidemia, abdominal obesity, target organ damage, diabetes, and clinical conditions associated with arterial hypertension. This patient has a very high risk: a 65 year old man, an arterial hypertension of the third degree, a defeat of the target organs (LV hypertrophy, damage to the retina vessels), myocardial infarction. To assess the dynamic characteristics of heart failure, it is important to determine the functional class, which is established according to clinical criteria and may change under the influence of treatment.

2. Echocardiographic control is often used when performing functional tests. Stress Echocardiography (combining bicycle ergometry and echocardiography) is

based on the fact that in the case of ischemia, local contractility occurs earlier than ECG changes.

3. Among instrumental research methods for such a patient, it is important to conduct Holter monitoring, which allows to record episodes of rhythm disturbances, ischemia and prevent the development of complications in a timely manner.

4. In assessing the state of a hypertensive patient, an assessment of the category of hypertensive crisis is important, depending on the presence of damage to the target organs. Complicated hypertensive crisis is characterized by acute or progressive damage to target organs, has a direct threat to the life of the patient, requires an immediate, within 1 hour, reduction of blood pressure by parenteral administration of drugs. Such complications include myocardial infarction, stroke, acute scattering of aortic aneurysm, acute left ventricular failure, arrhythmias, transient ischemic attack, eclampsia, acute hypertensive encephalopathy, acute renal failure, and bleeding.

5. Lifestyle adjustment should be performed for all patients with elevated blood pressure in a complex treatment, as these measures contribute to the effectiveness of antihypertensive drugs and reduce their dose. As progression of chronic heart failure is increasingly limited to physical activity, the length of stay in bed increases.

This patient is subject to medical treatment, since in addition to arterial hypertension of the third degree, the defeat of the target organs.

The choice of drugs for this patient is justified, since ACE inhibitors contribute to the reduction of mortality in patients with post-infarction atherosclerosis and heart failure, beta-blockers reduce the excessive activity of the sympathetic nervous system (tachycardia) and are also indicated in patients with a history of myocardial infarction, those azide diuretics are prescribed to patients older age, with signs of heart failure.

**LIST OF BASIC PHARMACOLOGICAL PREPARATIONS  
TO A STATE PRACTICAL ORIENTED ISSUIT [1]**

	Ukrainian name	Latin name	Release form, input path
<b>Cardiology</b>			
1	Nitroglycerin	Nitroglycerinum	tabl.0.5 mg;aerosol 1 dose = 400 micrograms;amp10 ml (1 mg/ml) in/v drip
2	Novokainamid	Novocaineamidum	Amp5 ml 10% i/v
3	Lidocaine Hydrochloride	Lidocainum	Amp2% r 2 ml
4	Mezaton	Mesatonum	Amp 1% rn 1 ml intravenous
5	Digoxin	Digoxinum	Amp0.025% rn 1-2 ml, tabl.0.25 mg
6	Magnesium sulfate	Magnesium sulphate	Amp25% 10 ml I/I, I/m
7	Nitroprusid sodium	Sodium nitroprusside	fl.30 mg intravenous drops.(5 µg/kg min)
8	Esmolol	Esmololum	Amp5 ml/in pp.
9	Labetalol	Labetalolum	Amp1% vr 5 ml v/v slowly
10	Enalaprilat	Enalaprilat	Amp1 ml (1.25 mg) v/v slowly
11	Nimodipine	Nimodipinum	fl.0.02% 50 ml v/v slowly
12	Urapidil (errantyl)	Urapeded	Amp5 ml 25 mg/v
13	Morphine hydrochloride	Morphinum	amp1% rn 1 ml intravenous bolus
14	Heparin	Heparinum	fl.5000 IU/ml 5 ml (1000 IU / year intravenously via infusomat)
15	Enoxaparin	Enocsaparinum	syringe 0.4 ml, 0.8 ml / well
16	Alteplase (aclitis)	Alteplasmae	fl. 50 mg / v
17	Tenekteplasa (Metalize)	Tenecteplasmae	fl. 50 mg / v
18	Streptokinase	Streptocinase	fl. 1,5 million OD in / out drops
19th	Fondaparinux (arikstra)	Fondaparinux	syringe 2.5 mg p / w
20	Isosorbide Dinitrate (Isocet)	Isosorbide dinitrate	Amp 10 ml (0.01 g) in/v drip

21	Dopamine	Dopamine	Amp4% 2 ml (<3 µg/kg / min - diuretic effect; 3-5 - inotropic effect;> 5 - vasopressor effect)
22	Norepinephrine	Noradrenaline hydrotartra	Amp0.2% rn 2 ml intravenous drip
23	Levosimendan (Simdax)	I'm leaning	fl.5 ml (2.5 mg / ml) v / v
24	Atropine sulfate	Atropinum	Amp0.1% rn 1 ml / m, p / w
25	Adrenalin	Adrenaline hydrochloride	Amp0.18% 1 ml v / v
26	Adenosine triphosphate (ATP)	Adenosine	Amp 1% v-1 ml v / v
27	Methyldopa	Methyldopa	tabl. 250 mg inside
28	Moksonidine	Moxonidinum	tabl. 200mg, 300mg, 400mg inwards
29	Amiodarone	Amiodarone	tabl. 200 mg inwards
30	Amlodipine	Amlodipine	tabl.5;10mg inwards
31	Atenolol	Atenololum	tabl.40mg inwards
32	Verapamil	Verapamilum	tabl. 80mg inwards
33	Hydrochlorothiazide	Hydrochlorothiazide	tabl.12.5;25mg inwards
34	Spironolactone	Spironolactonum	tabl.25mg inwards
35	Furosemide	Furosemidum	tabl.40mg inwards
36	Digoxin	Digoxinum	tabl.0.25mg inwards
37	Dabigatran		
38	Enalapril	Enalapril	tabl. 10; 20mg inwards
39	Eplerenon		
40	Isosorbide dinitrate	Isosorbide dinitratum	tabl.5mg inwards
41	Carvedilol	Carvedilolum	tabl. 6.25;12.5;25mg inwards
42	Bisoprolol	Bisoprolol	tabl. 5;10mg inwards
43	The purpose of Mr rolol	Metoprololum	tabl. 50 mg inwards
44	Nitroglycerin	Nitroglycerin	tabl.5mg inwards
45	Simvastatin	Simvastatin	tabl. 20;40 mg inwards
46	Clopidogrel	Clopidogrelum	tabl. 75 mg inwards
47	Rivaroxaban	Rivaroxabanum	tabl.20 mg inwards
48	Warfarin	Warfarinum	tabl.5 mg inwards
49	Perindopril	Perindopril	tabl.5;10 mg inwards
50	Ramipril	Ramiprilum	tabl.5;10 mg inwards
51	Trimetazidine	Trimetazidine	tabl.35 mg inwards

52	Rosuvastatin	Rosuvastatinum	tabl.10;20;40 mg inwards
53	Atorvastatin	Atorvastatin	tabl.20; 40; 80 mg inwards
54	Ticagrelor	Ticagrelorum	tabl. 90 mg inside
55	Torasemid	Torasemidum	tabl. 10 mg inwards
56	Valsartan	Valsartan	tabl. 80mg inwards
57	Losartan	Losartanum	tabl. 50 mg inwards
58	Propafenone	Propaphenonum	tabl.150 mg in the middle

### EXAMPLES OF RECIPES[11]

1. Rp: lisinopril and 0.02  
D.t.d. N20 intab.  
S. Take the pill once a day after eating.
  
2. Rp: Tab. Enalapril 0.01 N 20  
DS Take 1 tablet twice a day after meals.
  
3. Rp: Amlodipine 0.005  
D. td N20 incaps.  
S. Take 1 capsule once a day before meals.
  
4. Rp:Tab.Bisoprolol0.01  
Dtd N 30  
S. Take 1 pill in the morning.
  
5. Rp:Tab.Prednisolone 0.005  
Dtd N 50  
S. Take 4 pills in the morning and 2 pills in the afternoon after meals.
  
6. Rp: Tab. Nitroglycerin 0.0005  
Dtd N 50  
S. Take 1 tablet under the tongue with an angina attack.
  
7. Rp: Tabulated "Allochololum" N 50

DS Take 1 pill Each day before meals.

8. Rp: Tab. Amoxicillin/Clavulanic Acid 875/125 mg

Dtd N 14.

S. Take 1 tablet twice a day.

9. Rp: "Cardiomagnyl" 0.075

Dtd N50 intab.

DS Take 1 tablet per night.

10.Rp.: Sol.Thiotriazolin 2.5% - 4.0

DtdN 10 in ampull.

S. Administer slowly at 4 ml once daily.

11.Rp: Sol.Glucose 5% - 200.0

Dtd N 5.

S. The contents of the vial should be administered intravenously.

12.Rp.:Sol.Enoxaparin Sodium 0.6

Dtd N 10.

S. Inject the contents of the syringe (0.6 ml) 2 times a day subcutaneously.

13.Rp: Seftriaxone 1.0

Dtd N 10.

S. The contents of the vial dilute 10 ml of water for injection, enter slowly.

14.Rp: Sol.Amiodarone 0.15

Dtd N 2.

S. 300 mg to dissolve in 250 ml of 5% glucose, enter duly dropwise.

15.Rp: Heparini 5 ml (25,000 OD)

Da tales doses No. 2Signa: Administer1 ml subcutaneously four times a day.

## **DIAGNOSTIC, TREATMENT AND PREVENTIVE SCORES IN CARDIOLOGY [24, 26, 27, 36]**

### **Atrial fibrillation**

#### ***Stratification of Stroke and Thromboembolism***

Identification of clinical risk factors for stroke has led to the development of various risk assessment schemes for its development. Modern protocols suggest a more detailed analysis of the risk factors for stroke and address the issue of antithrombotic therapy based on their presence (or lack of). The main risk factors (formerly referred to as high risk factors) are stroke or history of TIA, thromboembolism, and senile age ( $\geq 75$  years). The presence of certain heart valve defects (eg mitral stenosis or prosthetic of Old valve in heart) also indicates a high risk of stroke. Clinically significant non-essential risk factors (formerly referred to as middle-risk factors) are CH (especially moderate or severe LV systolic dysfunction, characterized by a decrease in erythropoiesis  $\leq 40\%$ ), hypertension and diabetes. To the other's do factors risk (previously they were considered less valid than them) include female gender, age 65-74 years, and vascular disease (myocardial infarction, plaque in the aorta and peripheral vascular disease). Risk factors are cumulative, and anticoagulant therapy is warranted in the presence of at least two clinically significant non-essential risk factors [36].

To characterize the risk assessment scheme in patients with non-valvular AF, one can use the acronym CHA<sub>2</sub>DS<sub>2</sub>-VASc [HF, AH, age  $\geq 75$  (double), diabetes, stroke (double), vascular disease, age 65-74, and feminine sex]. In applying this scheme awarded 2 points for stroke and TIA history and age  $\geq 75$  years, and 1 point - age 65-74 years, hypertension, diabetes, HF, vascular disease (myocardial infarction, plaque in the aorta and peripheral arterial disease, including revascularization, amputation or angiographic changes) and females [36].

### Risk Index CHA<sub>2</sub>DS<sub>2</sub>VASc [36]

Letter	Risk factor	Score
C	Congestive heart failure/LV dysfunction	1
H	Hypertension	1
A <sub>2</sub>	Age ≥75	2
D	Diabetes mellitus	1
S <sub>2</sub>	Stroke/TIA/thrombo-embolism	2
V	Vascular disease*	1
A	Age 65–74	1
S	Sex category (i.e., female sex)	1
	Maximum score	9

Congestive heart failure/LV dysfunction means LV ejection fraction ≤40%. Hypertension includes the patients with current antihypertensive medication. \*Prior myocardial infarction, peripheral artery disease, aortic plaque. LV: left ventricular, TIA: transient ischemic attack

The introduction of a CHA<sub>2</sub>DS<sub>2</sub>-Vas scale for stroke risk assessment has simplified the initial decision for the administration of oral anticoagulants (POAC) in patients with AF. It is recommended to assess the risk of stroke in patients with AF based on CHA<sub>2</sub>DS<sub>2</sub>-Vas.

Overall, patients without risk factors for stroke do not require anticoagulant therapy, whereas patients with risk factors for stroke, that is, the number of points on the CHA<sub>2</sub>DS<sub>2</sub>-Vas scale for men and 2 for women - it is advisable to consider the possibility of appointment, the number of points per CHA<sub>2</sub>DS<sub>2</sub>-Vas 2 or more for men and 3 or more for women - Vitamin K antagonist (warfarin) or a direct oral anticoagulant (rivaroxaban, apixacaban, dabigatran) should be prescribed [36].

#### ***Risk of bleeding***



Before the start of anticoagulation it is necessary to estimate the risk of bleeding. A new simple bleeding risk index has been developed - HAS-BLED (hypertension, kidney/liver, stroke, history of bleeding or predisposition to bleeding, labile INR, age > 65 years, drug/alcohol use)[36].

<b>HAS-BLED Heart Risk Index[36]</b>	
<b>Risk Factor</b>	<b>Score</b>
Hypertension	1
Abnormal renal/liver function	1 or 2
Stroke	1
Bleeding tendency	1
Labile INR	1
Age >65	1

A score of 0-2 indicates low risk of bleeding;  $\geq 3$  indicates high risk of bleeding  
Hypertension is defined as a systolic blood pressure > 160 mmHg  
1 point is awarded for each of abnormal renal or liver function, and drugs or alcohol.

The first letters of English words

*Hypertension – systolic blood pressure > 160 mmHg., infringement of function of kidneys-dialysis, transplantation of kidney or serum creatinine  $\geq 200$  mmol/l, violations of function of liver-a chronic disease of the liver (eg cirrhosis) or biochemical signs of serious lesions of the liver (eg, The level of bilirubin least at twice higher than the upper limits of the rules in combination with increased activity of AST and ALT and alkaline phosphatase more than a 3-fold compared with the upper limit of the norm, and so on hemorrhage – bleeding in the history and/or inclination to bleeding, for example, hemorrhagic diathesis, anemia, and so on., labile INR -unstable/high INR in or lack of term preservation INR in a target range (eg, < 60% of the time), drugs/alcohol – concomitant intake of drugs, such as antiplatelet drugs, non-steroidal anti-inflammatory drugs, oral alcohol abuse.*

This index should be used to assess the risk of bleeding in patients with AF, but the number of points is not a contraindication for the administration of anticoagulants. The value of the index  $\geq 3$  indicates a high risk of hemorrhagic complications, and for some patients some precautions and regular follow-up after anticoagulation therapy with correction of identified risk factors are indicated[15].

### **ACUTE CORONARY SYNDROME[24, 27]**

A decision on the need and emergency, and coronary angiography in patients with ACS with elevation ST defined after risk stratification on a scale GRACE[27].

GRACE (Global Registry of Acute Coronary Events) allows you to assess the mortality and mortality risk both at the hospital stage and during the next half-year, as well as to determine the optimal treatment for a particular patient [27].

At the time of admission of patients with NSTEMI using this scale evaluated the risk of immediate (during hospital treatment) adverse cardiovascular outcomes (death, myocardial infarction) when you select a conservative treatment strategy[27].

**Mortality in a hospital for 6 months in low, medium and high risk groups according to GRACE according to registry data[27]**

Risk Score for NSTEMI-ACS		
Probability of In Hospital Death		
Risk Category (tertiles)	GRACE Risk Score	Probability of Death In-Hospital (%)
Low	1 – 108	<1
Intermediate	109 – 140	1 – 3
High	141 – 372	>3

Probability of Death Post Discharge to 6 Months		
Risk Category (tertiles)	GRACE Risk Score	Probability of Death Post-discharge to 6 Months (%)
Low	1 – 88	<3
Intermediate	89 – 118	3 – 8
High	119 – 263	>8

*Comment: At GRACE <http://gracescore.org/>, you can download an automatic risk calculator for free, which is easy to use in clinical practice.*

High-risk patients should be transported to centers (outlets) that provide tertiary care, where invasive diagnosis (coronary angiography) is possible. If necessary, the transport of patients stabilized after percutaneous coronary interventions may be performed in the opposite direction according to the local protocol (clinical route of the patient) for rehabilitation in other hospitals (without the possibility of primary coronary interventions) or in cardiac surgery, if in the process of invasive diagnosis the patient is shown surgical intervention[27].

### **Risk of hemorrhage[27]**

Bleeding is associated with an unfavorable prognosis in ACS without elevation of the ST segment. CRUSADE's Blood Risk Scale ([www.crusadebleedingscore.org/](http://www.crusadebleedingscore.org/)) is based on data from 71,277 patients from the CRUSADE registry(Figure 22)[27].

**The algorithm is used to assess the risk of severe bleeding in the hospital according to CRUSADE[27]**

Predictor	Score
Baseline hematocrit (%)	
<31	9
31–33.9	7
34–36.9	3
37–39.9	2
≥40	0
Creatinine clearance (mL/min)	
≤15	39
>15–30	35
>30–60	28
>60–90	17
>90–120	7
>120	0
Heart rate (bpm)	
≤70	0
71–80	1
81–90	3
91–100	6
101–110	8
111–120	10
≥121	11
Sex	
Male	0
Female	8
Signs of CHF at presentation	
No	0
Yes	7
Prior vascular disease	
No	0
Yes	0
Diabetes mellitus	
No	0
Yes	6
Systolic blood pressure (mmHg)	
≤90	10
91–100	8
101–120	5
121–180	1
181–200	3
≥201	5

*CHF* congestive heart failure

Creatinine clearance was estimated with the Cockcroft-Gault formula

Prior vascular disease was defined as history of peripheral artery disease or prior stroke

Fig.22. The risk of developing large CRUSADE scale bleeds

No scale can replace clinical calculations, but they are an objective clinical method for assessing the risk of bleeding for individuals or for a specific group[27].

The interpretation is as follows: the number of points  $\leq 20$  the risk of large bleeding in the hospital is very low, 21-30 points of risk low, 31-40 risk moderate, 41-50 points risk high, more than 50 points risk is very high[27].

### **APE [26]**

#### Evaluation of clinical probability

Clinical evaluation of individual symptoms, signs and their combination or use forecasting rules allow to distribute patients with suspected pulmonary embolism in certain categories of clinical or pretest probability corresponding increase in actual prevalence of confirmed pulmonary embolism[26]. Since the index of post test likelihood of APE (which, in particular, it is determined by the results of computed tomography) depends not only on the characteristics of the diagnostic examination itself but also on the degree of pretest probability, this indicator has become a key element of all algorithms of diagnostic examinations for the presence of pulmonary embolism. One of the most commonly used prognostic rules is the rule proposed by Wells and co-authors (Wells et al.). This rule has undergone expanded validation using both a three-level classification (low, medium and high probability of APE), and a two-level classification (likely to have APE or no APE). Revised Geneva Scale is also a simple and standardized method. Recently mentioned two scales - Wells and Geneva - have been simplified to promote their use in clinical practice and externally validated [26]. So, according to Wells's simplified scale, with a score of 2 or more, the diagnosis of APE is probable, with a simplified Geneva scale of 3 or more points, the diagnosis of APE is probable[26].

## Clinical algorithms for estimating the probability of pulmonary embolism [26]

Revised Geneva score <sup>64</sup>		Wells score <sup>65</sup>	
Variable	Points	Variable	Points
<b>Predisposing factors</b>		<b>Predisposing factors</b>	
Age >65 years	+1	Previous DVT or PE	+1.5
Previous DVT or PE	+3	Recent surgery or immobilization	+1.5
Surgery or fracture within 1 month	+2	Cancer	+1
Active malignancy	+2	<b>Symptoms</b>	
<b>Symptoms</b>		Haemoptysis	+1
Unilateral lower limb pain	+3	<b>Clinical signs</b>	
Haemoptysis	+2	Heart rate	
<b>Clinical signs</b>		75–94 beats/min	+3
Heart rate		≥95 beats/min	+5
75–94 beats/min	+3	Pain on lower limb deep vein at palpation and unilateral oedema	+4
≥95 beats/min	+5	<b>Clinical judgement</b>	
Pain on lower limb deep vein at palpation and unilateral oedema	+4	Alternative diagnosis less likely than PE	+3
<b>Clinical probability</b>		<b>Clinical probability (3 levels)</b>	
Low	0–3	Low	0–1
Intermediate	4–10	Intermediate	2–6
High	≥11	High	≥7
		<b>Clinical probability (2 levels)</b>	
		PE unlikely	0–4
		PE likely	>4

Index severity of pulmonary embolism (PESI) is at present the most widely accepted method of assessing prognosis death for 30 days and allows detect of place, volume and treatment of pulmonary embolism[26].

Given the complexity of the original PESI index, which provides for different estimates of 11 variables, a simplified version, known as sPESI, was developed and approved. Patients with pulmonary embolism have reported that sPESI allows for a more accurate prediction of 30 days than the shock index (heart rate ratio to systolic blood pressure), and the zero result of the simplified PESI allows identification of patients with a low risk of stroke. The use of sPESI in combination with troponin tests allows for additional prognostic information, especially for identifying patients at low risk[26].

## The original and simplified version of the 30-day mortality risk assessment scale

Original PESI [24]		Simplified PESI [25]	
Variable	Points	Variable	Points
Age	1 per year	Age >80 yrs	1
Male sex	10		
History of cancer	30	History of cancer	1
History of heart failure	10	History of heart failure or chronic lung disease	1
History of chronic lung disease	10		
Pulse rate >110 beats·min <sup>-1</sup>	20	Pulse rate >110 beats·min <sup>-1</sup>	1
Systolic blood pressure <100 mmHg	30	Systolic blood pressure <100 mmHg	1
Respiratory rate ≥30 breaths·min <sup>-1</sup>	20		
Body temperature <36°C	20		
Altered mental status <sup>#</sup>	60		
S <sub>a</sub> O <sub>2</sub> <90%	20	S <sub>a</sub> O <sub>2</sub> <90%	1
<b>Risk classification*</b>		<b>Risk classification</b>	
Class I (<65 points): very low risk		0 points: low risk	
Class II (66–85 points): low risk		≥1 point: high risk	
Class III (86–105 points): intermediate risk			
Class IV (106–125 points): high risk			
Class V (>125 points): very high risk			

S<sub>a</sub>O<sub>2</sub>: arterial oxygen saturation. <sup>#</sup>: disorientation, confusion or somnolence; <sup>\*</sup>: patients in PESI classes I and II are collectively referred to as low-risk patients.

Patients with no signs of shock and hypotension does not belong to the high risk of adverse CV event. Further stratification of their risk should be made after confirmation of the diagnosis in the pulmonary artery bypass grafting, as this may influence the choice of treatment strategy and duration of hospitalization. In these patients, the risk assessment should begin with the scale PESI or simplified index - sPESI with a view to identify groups of middle and high risk. Approximately one-third of patients with pulmonary embolism are in the low-risk group of an early adverse clinical outcome defined by PESI class I or II, or 0 according to sPESI. Accordingly, normotensive patients ≥III classes by PESI or ≥1 by sPESI form a medium-risk group. Patients who have confirmed PFS dysfunction (with echocardiography or CT angiography) and increased levels of cardiac markers (especially a positive troponin test) should be classified as moderate to high risk. Patients who have a normal PMS image according to echocardiography or CT scan data and / or cardiac biomarkers are also normal, refer to low-risk subjects[26].

Confirmed high-risk APE. In the absence of absolute contraindications, thrombolytic therapy (TLT) is performed, with absolute contraindications or in the case of ineffectiveness of TLT - surgical embolectomy, in case of impossibility of its implementation - catheter embolectomy[26].



Confirmed low risk APE.In the case of low risk, drugs of choice are low molecular weight heparins (LMW) or fondaparinux, in the case of moderate risk, TLT (especially without the risk of bleeding). Patients with low risk carcinogenesis can be discharged home based on outpatient monitoring and treatment with vitamin K antagonists (warfarin). LMWH, fondaparinuxes or UFH therapy should be started as soon as possible and continue for at least 5 days. All other patients should be prescribed an antagonist of vitamin K (warfarin) within 24 hours of diagnosis, under the control of the international normalized ratio (INR).An alternative option is direct oral anticoagulants (rivaroxaban, apixaban, dabigatran).The therapy should be performed within 3 months, after which it is possible to consider the ablation of anticoagulant therapy or its continuation[26].

## REFERENCES

1. Внутрішня медицина. Наскрізна програма підготовки фахівців освітньо-кваліфікаційного рівня “Спеціаліст” для студентів вищих навчальних закладів МОЗ України. – Київ, 2016. – 307 с.
2. Наказом МОЗ України від 31.01.2005 № 53 «Про затвердження «Положення про організацію та порядок проведення державної атестації студентів, які навчаються у вищих навчальних закладах III-IV рівнів акредитації за напрямом підготовки «Медицина»»
3. Інструкція МОЗ України від 15.04.2014 р. «Щодо оцінювання навчальної діяльності студентів в умовах впровадження Європейської кредитно-трансферної системи організації навчального процесу»
4. Історія хвороби в терапевтичній клініці : навч.-метод. посіб./ В. Д. Сиволап, С. М. Кисельов, О. О. Солов'юк, О. В. Назаренко, Я. В. Земляний. - Запоріжжя : ЗДМУ, 2017. – 103 с.
5. Сиволап В.Д. Класифікація, приклади формулювання та кодування діагнозів захворювань серцево-судинної системи : навч. посіб. / В.Д. Сиволап, С.М. Кисельов, Д.А. Лашкул. – Запоріжжя, 2010. – 47 с.
6. Сучасні класифікації та стандарти лікування захворювань внутрішніх органів. Невідкладні стани в терапії : довід.-посіб. / за ред. Ю. М. Мостового. - 18-е вид., допов. і переробл. - К. : Центр ДЗК, 2015. - 680 с.
7. Ендокринологія: підручник / П.М. Боднар, Г.П. Михальчишин, Ю.І. Комісаренко [та ін.] ;за ред. П.М. Боднара. - Вид.3, перероб. та доп. – Вінниця : Нова книга, 2013. – 480 с.
8. Передерій В.Г. Основи внутрішньої медицини: підручник: у 3-х т. Т. 1 /В.Г.Передерій, С.М.Ткач. – Вінниця: Нова книга, 2009. – 636 с.
9. Техніка лікарських маніпуляцій в терапевтичній практиці : навч. посіб. / під ред. проф. В.Д. Сиволап. – Запоріжжя: ЗДМУ, 2008. – 134 с.

10. Наказ МОЗ України від 24.05.2012 р. №384 «Про затвердження та впровадження медико-технологічних документів зі стандартизації медичної допомоги при артеріальній гіпертензії». Уніфікований клінічний протокол первинної, екстреної та вторинної (спеціалізованої) медичної допомоги «Артеріальна гіпертензія».
11. Ведення хворих в кардіологічній клініці : навч.-метод. посіб. / В. Д. Сиволап [та ін.]. – вид. 2-ге доповн. та доопрац. – Запоріжжя : ЗДМУ, 2016. – 143 с.
12. Сиволап В.Д. Клиническая электрокардиография: учебник. – Запорожье, 2008. - 264 с.
13. Сиволап В.Д. Электрокардиографическая диагностика в неотложной кардиологии: учебное пособие / В.Д. Сиволап. – Запорожье, 2015. – 67 с.
14. Скибчик В.А. Електрокардіографічна діагностика і лікування в невідкладній кардіології / В.А.Скибчик, Я.В.Скибчик. – К: ТОВ “Люди в білому”, 2014. – 154 с.
15. Кушаковский М.С. Аритмии сердца. Издательство: “Фолиант”, 2004. – 672 с.
16. Практикум з електрокардіографії / Жарінов О.Й., Куць В.О., Вережнікова Г.П., Серова О.Д. – Львів-Київ, 2014. – 268 с.
17. Павлюк В. І. Практична ехокардіографія / В. І.Павлюк; за ред. проф. О. Й. Жарінова. – Львів : Видавництво МС, 2014. - 146 с.
18. Шиллер Н., Осипов М.А. Клиническая эхокардиография, второе издание. - М.: Практика, 2005. – 344 с.
19. Стаднюк Л.А., Лапшин О.В., Динник О.Б. та співавт. Стресс-ехокардіографія. – К., 2004. – 166 с.
20. Жарінов О.Й., Куць В.О., Тхор Н.В. Навантажувальні проби в кардіології. – К.: Медицина світу, 2006. – 90 с.
21. Руководство по кардиологии / под ред. В.Н.Коваленко. – К.:Морион. - 2008. – 875 с.

22. Наказ МОЗ України від 02.03.2016 №152. Стабільна ішемічна хвороба серця. Уніфікований клінічний протокол первинної, вторинної та третинної (високоспеціалізованої) медичної допомоги.
23. Сиволап В. Д. Гострий коронарний синдром : навч.-метод. посіб. до курсу за вибором «Актуальні питання кардіології» / В. Д. Сиволап, Д. А. Лашкул. - Запоріжжя : [ЗДМУ], 2016. – 198 с.
24. Наказ МОЗ України від 02.07.2014 № 455. Гострий коронарний синдром з елевацією сегмента ST. Уніфікований клінічний протокол екстреної, первинної, вторинної (спеціалізованої) та третинної (високоспеціалізованої) медичної допомоги та медичної реабілітації.
25. Сиволап В.Д. Невідкладні стани в кардіології: навч.-метод. посіб. до практичних занять та самостійної роботи студентів мед. та педіатр. ф-тів з дисципліни Внутрішня медицина / В.Д. Сиволап, С.М. Кисельов, Д.А. Лашкул, О.П. Бондаренко. – Запоріжжя, 2017. – 260 с.
26. Сиволап В. Д. Тромбоемболія легеневої артерії [Електронний ресурс] навч.-метод. посіб. до курсу за вибором «Актуальні питання кардіології» / В. Д. Сиволап, О. П. Бондаренко. - Запоріжжя : [ЗДМУ], 2016. – 78 с. - Електрон. текстові дані (1 файл: 472 КБ). - Назва з екрана.
27. Наказ МОЗ України від 03.03.2016 №164. Гострий коронарний синдром без елевації сегмента ST. Уніфікований клінічний протокол екстреної, первинної, вторинної (спеціалізованої), третинної (високоспеціалізованої) медичної допомоги та медичної реабілітації.
28. Уніфікований клінічний протокол первинної, вторинної (спеціалізованої) та третинної (високоспеціалізованої) медичної допомоги «Хронічна серцева недостатність» - 2016 (проект).
29. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias. European Heart Journal (2016) 37, 2999-3058.

30. Наказ МОЗ України від 13.06.2016 № 564 "Про затвердження та впровадження медико-технологічних документів зі стандартизації медичної допомоги в частині профілактики серцево-судинних захворювань".
31. Perkins G.D., Handley A.J., Koster R.W. et al. European Resuscitation Council guidelines for resuscitation 2015. Section 2. Adult basic life support and automated external defibrillation // Resuscitation. - 2015. - Vol. 95. - P. 81-99.
32. 2017 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations Summary Circulation. 2017 Dec 5;136(23):e424-e440.
33. Сиволап В.Д.  
Принципы дифференциальной диагностики и формулирования диагноза : учеб.-метод. пособие / В. Д. Сиволап. – Запорожье, 2009. – 135 с.
34. Клінічна оцінка результатів інструментальних досліджень в кардіології : навч.-метод. посіб. / В.Д. Сиволап, В.Х. Каленський, Д.А. Лашкул, О.П. Бондаренко. – Запоріжжя, 2016. – 105 с.
35. Сборник обучающих и контролирующих задач для самоподготовки к практическим занятиям по «Внутренним болезням : учеб.-метод. пособие для студентов, врачей-курсантов, интернов, клинических ординаторов/сост. В.Д.Сиволап. – Запорожье, 2011. – 196 с.
36. Наказ МОЗ України від 15.06.2016 № 597 "Про затвердження та впровадження медико-технологічних документів зі стандартизації медичної допомоги при фібриляції предсердь".

## **RECOMMENDED LITERATURE**

### **Basic:**

1. Davidson's Principles and Practice of Medicine, 23st Edition. Editors: Stuart Ralston, Ian Penman, Mark Strachan, Richard Hobson. Elsevier. – 2018. – 1440p.

2. Emergency Management of Internal Disease : textbook approved by the Ministry of Public Health of Ukraine and the Ministry of Education and Science of Ukraine / O. Babak [et al.] ; ed.: O. Babak, O. Bilovol. - Kyiv : AUS Medicine Publ, 2010. - 448 p
3. First Aid for the USMLE Step 2 CK Clinical Knowledge: a student-to-student guide / T. Le [et al.]. - 9th ed. - USA : McGraw-Hill Education, 2016. - 550 p.
4. USMLE. Step 2 CK. 2018. Internal Medicine : lecture notes / ed.: J. J. Lieber, F. P. Noto. - New York : Kaplan Medical USMLE, 2017. - 496 p.
5. Syvolap V.D., Kadzharyan V.G., Solov'yuk O.O. Urgent conditions in endocrinology : Textbook for 6-th course students of medical university. – Zaporizhzhia, 2018. – 108 p.
6. Syvolap V.D., Kadzharyan V.G., Solov'yuk O.O. Management of patients in the endocrinology clinic : Textbook for 6-th course students of medical university. – Zaporizhzhia, 2017. – 136 p.
7. Syvolap et al. Management of patients in the cardiology clinic. Textbook for 6-th course students of medical university.- Zaporizhzhia, 2018. – 240 p.
8. Syvolap et al. Urgent conditions in cardiology clinic. Textbook for 6-th course students of medical university.- Zaporizhzhia, 2018.–153 p.

**Additional:**

1. 2018 ESC Guidelines for the diagnosis and management of syncope European Heart Journal, ehy037. <https://doi.org/10.1093/eurheartj/ehy037>
2. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2017 Nov 13. pii: HYP.0000000000000065. doi: 10.1161/HYP.0000000000000065.

3. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC) *European Heart Journal*, Volume 39, Issue 2, 7 January 2018, Pages 119–177, <https://doi.org/10.1093/eurheartj/ehx393>
4. 2017 ESC/EACTS Guidelines for the management of valvular heart disease *European Heart Journal*, Volume 38, Issue 36, 21 September 2017, Pages 2739–2791, <https://doi.org/10.1093/eurheartj/ehx391>
5. 2017 ESC focused update on dual antiplatelet therapy in coronary artery disease developed in collaboration with EACTS: The Task Force for dual antiplatelet therapy in coronary artery disease of the European Society of Cardiology (ESC) and of the European Association for Cardio-Thoracic Surgery (EACTS) *European Heart Journal*, Volume 39, Issue 3, 14 January 2018, Pages 213–260, <https://doi.org/10.1093/eurheartj/ehx419>
6. 2017 ESC Guidelines on the Diagnosis and Treatment of Peripheral Arterial Diseases, in collaboration with the European Society for Vascular Surgery (ESVS): Document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries *European Heart Journal*, Volume 39, Issue 9, 1 March 2018, Pages 763–816, <https://doi.org/10.1093/eurheartj/ehx095>
7. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) *European Heart Journal*, Volume 37, Issue 27, 14 July 2016, Pages 2129–2200, <https://doi.org/10.1093/eurheartj/ehw128>

8. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS European Heart Journal, Volume 37, Issue 38, 7 October 2016, Pages 2893–2962, <https://doi.org/10.1093/eurheartj/ehw210>
9. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias European Heart Journal, Volume 37, Issue 39, 14 October 2016, Pages 2999–3058, <https://doi.org/10.1093/eurheartj/ehw272>
10. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) European Heart Journal, Volume 37, Issue 29, 1 August 2016, Pages 2315–2381, <https://doi.org/10.1093/eurheartj/ehw106>
11. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS) European Heart Journal doi:10.1093/eurheartj/ehv317
12. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC) European Heart Journal, Volume 36, Issue 41, 1 November 2015, Pages 2793–2867, <https://doi.org/10.1093/eurheartj/ehv316>
13. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC) European Heart Journal, Volume 37, Issue 3, 14 January 2016, Pages 267–315, <https://doi.org/10.1093/eurheartj/ehv320>



14. 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC) *European Heart Journal*, Volume 36, Issue 44, 21 November 2015, Pages 3075–3128, <https://doi.org/10.1093/eurheartj/ehv319>
15. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) *European Heart Journal*, Volume 36, Issue 42, 7 November 2015, Pages 2921–2964, <https://doi.org/10.1093/eurheartj/ehv318>
16. 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism: The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC) *European Heart Journal*, Volume 35, Issue 43, 14 November 2014, Pages 3033–3073, <https://doi.org/10.1093/eurheartj/ehu283>
17. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy The Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC) *European Heart Journal* (2014) 35, 2733–2779 doi:10.1093/eurheartj/ehu284
18. 2014 ESC/EACTS Guidelines on myocardial revascularization The Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS) *European Heart Journal* (2014) 35, 2541–2619 doi:10.1093/eurheartj/ehu278
19. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC) *European Heart Journal* (2014) 35, 2873–2926 doi:10.1093/eurheartj/ehu281

