Ministry of Health of Ukraine
Zaporizhzhya State Medical University

URGENT CONDITIONS IN CARDIOLOGY

Textbook for 6th course students of medical university

Zaporizhzya-2014

УДК: 616.43-083.98(075.8)=20
ББК 54.15я73
Е 50

Recommend by Central Methodical Council of Zaporizhzhya State Medical University as a study guide for students and teachers of higher educational medical institutions (protocol № 1 since 25.09.2014.)

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This textbook content methods of educational process organization by Module 4 «Urgent conditions in clinic of internal medicine», content Module 1 „Urgent conditions in cardiology" in accordance with the Program of Educational Discipline "Internal Medicine", specialties: "Medical business" and "Pediatrics".

In textbook there are methodical guidelines for practical lessons and individual work for 6th course students; protocols of examination, diagnosis, treatment and prevention; clinical protocols providing medical care, tests for evaluation of students' initial knowledge level and clinical tasks for self-study.

Contents

Module 4

«Urgent conditions in clinic of internal medicine»

Final aims of Module

Students must be:

- Definite level of examination and treatment of inpatients with urgent conditions.
- Use in practice algorithms of examination and management in patients with urgent conditions.
- Perform in practice differentiate diagnosis of basic syndromes in clinic of urgent conditions.
- Master methods of treatment of urgent conditions in accordance with evidence medicine data.
- Use in practice standards of diagnostic and treatment of inpatients with urgent conditions.

The organization of educational process should ensure participation of students in management at least 2/3 of inpatients. If there isn't possibility to gain access to patients of any category, students fill the educational history with appropriate diagnoses. Need of writing of medical history is defined by assistant/docent (head of the chair curriculum department) in accordance with weekly data of presence of appropriate patients in hospital departments.

Course conducted in hospitals with emergency departments. Each student must work in a hospital weekly with 3 new/nondifferentiated patients. During course weekly students examined 6-10 patients with dynamic observation and permanent patients. If for any reason a student inspects less than 2 patients per day, will studied treatment of patients who were hospitalized before. Quantity of patients for student curationn is defined by complexity of cases and possibility of students to work with additional patients. Didactic classes conduct during the morning inspections, lections and practical lessons. Assessment of administration of history case is conducted by teacher in time of work with patient.

Duties of the teacher

The teacher must constantly communicate with the student during the day. After examining the patient he must hold a conversation with the student and state that:

- Main complains were reviewed
- Anamnesis collected carefully organized, logically, consistently, methodically correct
- Physical examination completed methodically correct without violation of diagnostic procedures
- Student demonstrates correct technic in definition of pathognomonic symptoms
- He describes symptoms with usage of appropriate pathophysiologic terms
- Student findings are correct
- The student determined preliminary diagnosis correct and noted diseases for perform of differentiate diagnosis
- Student completed correct plan of examination and used the most informative diagnostic methods
- He wrote correct administration list, noted doses of drugs, duration of treatment
- He is able to ground reasonability of specified drugs
- He is able to attach to patient for treatment, shows positive side of treatment and risk of absence of treatment, demonstrates respect to patient and makes of patient active participant in treatment process
- He is able to report professionally about clinical condition of patient during 3-5 min
- He make correctly short, professionally note in diary
- He demonstrates responsibility in treatment of patients independently from age, sex, race, social status, sexual orientation and financial resources
- He may work in staff effectively
- He demonstrates respective term to all members of medical staff (faultiness, responsibility and fairness
- He accepts the terms that different minds and treatment features may be used.

Content Module 1.
Urgent conditions in cardiology
(30 h/1.0 credit).

Topic plan of practical classes

№	Topic	Hours
1	Management of patient with the complicated hypertensive crisis	5
2	Management of patient with paroxysmal arrhythmias and conduction	2
3	Management of patient with acute coronary syndrome	7
4	Management of patient with acute myocardial infarction. Management of patient with cardiogenic shock. Tactic of treatment at sudden cardiac death.	7
5	Management of patient with pulmonary embolism. Final control.	2 2
Total		25

Concrete aims:

Students must:

- Definite level of examination and treatment of inpatients with urgent conditions
- Use in practice algorithms of examination and management of inpatients with urgent conditions
- Perform in practice differentiate diagnosis of basic syndromes in clinic of urgent conditions
- Master methods of treatment of urgent conditions in accordance with evidence medicine data
- Use in practice standards of diagnostic and treatment of inpatients with urgent conditions

Topic 1. Management of patient with the complicated hypertensive crisis. Current standards of diagnostic and management of patients. Tactic of management of patients due to defeat of organs-targets. Futher management of patients.

Management of patient with cardiac asthma and pulmonary edema. Current standards of diagnostic and management of patients. Tactic of management of patients due to blood pressure level. Futher management of patients.

Topic 2. Management of patient with acute coronary syndrome. Current standards of diagnostic and management of patients. Tactic of management of patients due to ST segment elevation. Futher management of patients.

Topic 3. Management of patient with acute myocardial infarction. Current standards of diagnostic and management of patients. Tactic of management of patients due to ST segment elevation and pathological Q-wave. Futher management of patients.

Management of patient with cardiogenic shock. Current standards of diagnostic and management of patients. Tactic of management of patients due to blood pressure level and stage of shock. Futher management of patients.

Topic 4. Management of patient with pulmonary embolism. Current standards of diagnostic and management of patients. Tactic of management of patients due to embolization level. Futher management of patients.

Management of patient with sudden cardiac death. Current standards of diagnostic and management of patients. Technique of resuscitation. Defibrillation. Futher management of patients.

Topic 5. Management of patient with paroxysmal arrhythmias and conduction. Current standards of diagnostic and management of patients. Tactic of management of patients due to type of arrhythmia or block. Electropulse treatment and electrostimulation. Futher management of patients.

Topic 1.

Management of patient with the complicated hypertensive crisis. Curation of patient with cardiac asthma and pulmonary edema.

Actuality. The issue of the day of modern cardiology are hypertensive crises (HC). A hypertension crisis is a acute increase of blood pressure (systolic and diastolic) higher individual norm, which, as a rule, is accompanied by appearance or strengthening of disorders of function of organs-targets or vegetative nervous system. Hypertension crises are observed in 20-35% of patients with hypertensive disease, can arise up at all stages of it motion, and sometimes is it by an only display. For patients with hypertensive disease to the origin of hypertensive crisis all promote those factors which in ordinary terms cause the increase of blood pressure and change reactivity of organism; the last is related to the type of higher nervous activity, state of pressing-depressing and humoral mechanisms. Pathogenesis of hypertensive crisis is difficult and finally not studied, mechanism of their origin for patients with hypertensive disease and symptomatic hypertensions are different. For patients with hypertensive disease of crises it is arisen up mostly on a background the protracted overstrain of the central nervous system and hyperfunction of some systems of organism (simpatico-adrenal, renin-angiotensin-aldosteron and other) with violation of metabolism by exhaustion of compensatory possibilities. A hypertension crisis is examined as a clinical syndrome which is characterized by the stormy, sudden intensifying of hypertensive illness or symptomatic hypertension and shows up the sharp increase of blood pressure and row of general (excitation of the vegetative nervous system, hormonal and humoral violations) and regional symptoms with advantage of cerebral and cardiovascular disorders. Without regard to that diagnostics of hypertension crisis is conducted easily and this question the enormous amount of scientific-practical works is sanctified to, death rate at development of hypertension crisis for patients with hypertensive illness very high. It is known that a death rate at hypertension crisis in Ukraine among the entire countries of Europe remains most high. Hypertensive crises are uncomplicated and complicated.

Uncomplicated hypertension crisis is not accompanied by the defeat of organs of targets, it can be without symptoms. The risk of heavy complications is small, that is why blood pressure can be reduced during a few hours and days. Preparations mainly apply perorally, quite often such patients are treated by ambulatory.

Complicated hypertension crisis accompanied by acute violation of functions vitally important organs as a result of high blood pressure. It needs to be reduced immediately, preparations mainly enter parenterally, a patient is looked after in the block of intensive therapy. Among complications of hypertension crisis sufficiently often there is left ventricular failure (cardiac asthma and pulmonary edema).

Aim of employment: to teach students to the modern tactic of conduct patients with the complicated hypertensive crises, especially at development of acute left ventricular failure (cardiac asthma and pulmonary edema), diagnostics, differential diagnostics and prophylaxis at hypertension crisis, to determine the tactic of treatment at complicated by cardiac asthma and pulmonary edema hypertension crisis.

A student must know:

1. Determination of concept is a hypertensive crisis
2. Classification of hypertensive crisis (uncomplicated, complicated)
3. Concept of syndrome of left ventricular failure (cardiac asthma, pulmonary edema)
4. Pathogenesis of left ventricular failure at hypertensive crisis
5. Tactic of conduct (examination, treatment) of patients with hypertensive crises depending on complications
6. Principles of medicinal treatment of patients are with a hypertension crisis, complicated by left ventricular failure
7. Primary and secondary prophylaxis at arterial hypertension

A student must be able:

1. To conduct physical examination of patient with hypertensive crisis
2. To measure and interpret blood pressure
3. To expose the different variants of complications of hypertensive crisis
4. Fold the plan of inspection sick with hypertensive crisis, to ground application of basic invasion and non-invasive methods of examination, to determine indication and contra-indication to it realization and possible complications
5. On the basis of analysis of data of laboratory and instrumental inspection to conduct a differential diagnosis, ground and formulate a diagnosis
6. To diagnose and give help at the complicated hypertensive crisis
7. To demonstrate possessing moral-deontological principles of medical specialist and principles of professional deference to rank.

A study is held as work of students in composition small brigades at a bed of patient with hypertensive crisis.

During curation of patients a student uses corresponding Protocol and fills the cards of patients (see addition 1).

Protocol of inspection, establishment of diagnosis, treatment and prophylaxis at arterial hypertension and complicated hypertensive crisis in accordance with the standards of evidential medicine on the basis of recommendations of European society of cardiologists/of European society of hypertension (2013), Ukrainian association of cardiologists (2011) and national Clinical protocols (2008)

DIVISION OF ACTIVITY	GUIDANCE TO ACTIONS
Greeting	Greet and will appear to the patient
Acquaintance	Collect passport data of patient (name, surname, sex, age, residence, job and speciality)
Complaints of patient are in the moment of inspection	Define and go into detail the complaints of patient. At determination of complaints of patient pay attention to presence:

	1) headache 2) dizziness 3) murmur in ears 4) dyspnea 5) cough Define the presence of connection between complaints and level of blood pressure
Individual and domestic anamnesis, questioning by organs and systems	Find out the dynamics of disease, conduct questioning by organs and systems At questioning pay attention on a list relatively: 1. duration and previous levels of increase of blood pressure. 2. to the presence of symptoms which can testify to secondary character of arterial hypertension: - domestic anamnesis of kidneys disease; - illnesses of kidneys, infections of urinary ways, hematuria, abuse of analgetic facilities; - to the reception of medicine (perorally contraceptives, drops against rhinitis, cocaine, amphetamines, steroids, unsteroidal anti-inflammatory preparations, erythropoietin's, cyclosporine); - attacks of diaphoresis, headache, horror, palpitation (signs of pheochromocytoma); - attacks of muscular weakness and tetania (signs of aldosteronism). 3. To the presence of risk factors : - Arterial hypertension and cardiovascular diseases for family members; - dyslipidemias in domestic or individual anamnesis; - diabetes mellitus in domestic or individual anamnesis; - smoking; - obesity (will define food habits also); - level of the physical loading (amount of physical exercises, hours in twenty-four/hours week); - snoring, nightly apnoea. 4. To the presence of symptoms of defeat of organs-targets: - brain and vision: headache, dizziness, loop of vision, transitory ischemic attacks, sensory or motor insufficiency; - hearts: palpitation, pain in a thorax, shortness of breath, edema of shins; - kidneys: thirst, polyuria, nycturia, hematuria; - peripheral artery: cold extremities, remittent lameness. 5. Previous antihypertension treatment: - preparations; - efficiency of previous antihypertension treatment; - side effects at previous therapy. 6. The presence of hypertensive crises (periodicity, level of increase of blood pressure, clinical displays)
Measuring of blood pressure	1. Before the beginning of measuring of blood pressure give to the patient to rest a few minutes 2. Conduct measuring twice at intervals 1-2 min If there will be a wide difference between measuring - repeat measuring once again 3. Use a standard cuff (12-13 sm of x a 35 sm). For obese or thin patients it follows to use corresponding cuffs 4. Set a cuff at the level of heart regardless of pose of patient 5. Use I and V tones of Korotkov for authentication of systolic and diastolic blood pressure 6. Measure blood pressure on both hands (take into account higher indexes) 7. Measure blood pressure on I and V min after passing of patient to vertical position (at a concomitant diabetes mellitus, for older persons and at suspicion in the presence of postural hypotension)

Physical examination	At the inspection of patient estimate: 1. State of patient 2. Position at bed At the inspection of patient pay attention to presence: Signs of defeat of organs-targets : - peripheral arteries (absence, decrease or asymmetry of pulse, cold extremities, ischemic defeats of skin); - hearts (localization and description of apex push, violation of cardiac rhythm, wheezes in lungs, peripheral edemata); - brain (noises are on the arteries of neck, motor and sensory violations); - retinas of eye (from data of fundoscopy, at possibility).
Plan of examination	Made the plan of inspection sick taking into account the conservative, recommended methods, and also extended methods of examination, shown to the concrete patient (see farther) Some inspections are conducted in parallel with a grant to the patient of the first aid (ECG, X-ray of organs of thorax), other - after stabilizing of the patient state.
Laboratory and instrumental research (conservative methods)	1. Estimate levels: - glucose on an empty stomach; - general cholesterol, low density lipoproteins (LDLP), high density lipoproteins (HDLP) and triglycerides in the serum of blood; - to potassium in the serum of blood; - urinary acid and kreatinine in the serum of blood; - to haemoglobin and hematokrit. 2. Analyse an urine. 3. Analyse an electrocardiogram, expect the criteria of hypertrophy of the left ventricle (LV) of heart of Sokolov-Layone (SV1(2) +RV5(6)) 4. Analyse X-ray of organs of thorax
Laboratory and instrumental research (recommended methods)	Conduct the analysis of recommended at the inspection of patient with hypertensive crisis of methods of laboratory and instrumental inspection (at presence of data in a hospital chart): 1. ECHO-cardiography - estimate the presence of signs of hypertrophy of LV of heart, as its remodelling, presence of systolic and/or diastolic dysfunctions of LV of heart 2. Ultrasonic research of carotids - estimate the presence of atherosclerotic plaque and hypertrophy of wall (thickness of complex "intima-media") 3. Quantitative determination of proteinuria - at microalbuminuria (30-300 mg/24 hours), expect the size of correlation "albumen/kreatinine" 4. To monitoring of domestic and ambulatory (24-hours) blood pressure - educe day's type of increase of propulsion MODULE
Laboratory and instrumental research (extended methods)	Estimate expediency of application for the concrete patient of the extended methods of diagnostics : 1) defeat of heart, vessels, cerebrum (at complicated arterial hypertension); 2) researches for clarification of secondary arterial hypertension (determination of levels of renin, aldosteron, corticosteroids, catecholamins in plasma and/or urine, arteriography, ultrasonic research of kidneys and suprarenal glands, computer tomography, magnetic resonance therapy of organs of abdominal region and other)
Determination of prognosis of patient	To define prognosis of patient with arterial hypertension on the basis of stratification of risk by SCORE scale: - the sick groups of subzero risk have authenticity of fatal cardiovascular event during 10 years <of 4%; - the sick groups of moderate risk have authenticity of fatal cardiovascular event during 10 years of 4-5%; - the sick groups of high risk have authenticity of fatal cardiovascular event during 10 years of 5-8%; - patients with the complicated hypertensive crisis have very high authenticity of fatal cardiovascular event.
Estimation of capacity	Estimate the capacity of patient, coming from recommendations of Clinical protocol of conduct of patients with arterial hypertension: 1. Patients with the complicated hypertensive crisis are temporally disabled. In future, after the leading out of sore from crisis subject of capacity decides depending on the stage of hypertensive disease and consequences of carried crisis. A. Patients with arterial hypertension of I and II stages capable of working, subject to the ambulatory inspection and treatment in policlinics domiciliary. In difficult cases hospitalizations are subject to the cardiologic, nephrological, endocrinology separations of permanent establishments on the period of clarification f diagnosis and/or choice of therapy

	B. Patients with arterial hypertension of III stage must be directed on MSEC in connection with the loss of capacity. Thus, the temporal loss of capacity for patients on arterial hypertension comes at hypertensive crisis, appearance of acute coronary failure, origin of attacks of cardiac asthma and pulmonary edema, sharp violations of cerebral circulation of blood. In case of necessity of realization of MSEC at formulation of clinic-expert diagnosis it follows to specify the stage of hypertensive disease, features of flow of illness, height and stability of blood pressure, character and frequency of crisis, presence of complications such as ischemic heart disease (IHD), heart failure (HF), chronic kidney failure, violation of cerebral circulation of blood. It is necessary to have the detailed description of the state of eye ground /presence of haemorrhage, efficiency of hypotension therapy, functional class of stabile angina pectoris of tension, presence of concomitant diseases,. The defined value belongs to character of profession, terms of labour. At presence of IHD it follows to consider heart trouble a basic disease. Without the symptoms of heavy heart failure a capacity is determined by the state of cerebral circulation of blood, in such cases a dominant role in examination belongs to the neurologist.
Formulation of diagnosis	Standards of diagnoses : - Hypertensive disease II stage, 2 degree. Hypertrophy of the left ventricle of heart. Hypertensive crisis, complicated by acute left ventricular failure (pulmonary edema), HF II-A, with preserved ejection fraction, II FC (NYHA). Risk 4. - Hypertensive disease III stage, Cardiac complicated crisis (date), 3 degree, IHD. Diffuse cardiosclerosis. Paroxismal form of ventricular tachycardia (date). Medicinal cardioversion (date), HF II-A, with preserved ejection fraction, II FC (NYHA). Risk 4. - Hypertensive disease III stage. Cerebral complicated криз (date). Acute hypertension encephalopathy (date), HF II-A, with preserved ejection fraction, II FC (NYHA). Risk 4.
Plan of treatment	Conduct the plan of treatment, which must include nonpharmacological and pharmacologicaltherapy
Diagnostics of type of hypertensive crisis	A kind will define crisis: 1. Criteria of crisis: outbreak, considerable increase of blood pressure, appearance or strengthening of symptoms from the side of organs-targets 2. Criteria of uncomplicated crisis: the acute making progress defeat of organs-targets absents, strengthening of symptoms takes place from the side of organs-targets (headache, pains in area of heart, extrasystoly) or from the side of the vegetative nervous system (vegetative violations, shaking, polakiuria) 3. Criteria of complicated crisis: acute making progress the defeat of organs-targets with development: - acute myocardial infarction - stroke - transitory ischemic attack - acute dissection aneurysm of aorta - acute left ventricular failure - unstable angina - eclampsias - acute hypertension encephalopathy - bleeding
Conduct of patients with complicated hypertensive crisis Medicinal therapy	
Choice of tactic of medicinal therapy	By the mean of choice for purchased of heavy hypertensive crisis /GC of second type by N.A.Ratner/ Clophelinum continues to remain. At intravenous slow introduction 0,5-1 мl of a 0,01 % solution of Clophelinum on 10-15 ml of isotonic solution of chloride of sodium of BP goes down through 5-15 min. At the same time such rapid decline of BP does not worsen the state of cerebral and coronal circulation of blood. If to enter Clophelinum in a muscle, then the decline of BP is marked on 30-60-th min. For warning of orthostatic collapse it is necessary to adhere to the bed mode during 2 hours after parenterally introduction of preparation. In stationary terms with cerebral symptoms at hypocinetic GC/of the second type/ appoint a 0,25 % solution of droperidol a patient for 4-6 ml in a vein on 20 ml of a 0,9 % soluble-sodium of chloride or 20 ml of a 5 % solution of glucose. At slow introduction to the vein of droperidol already on 2-5-th min gets better feel sick and BP goes down. However hypotension effect of short duration, somewhere near one hour. That is why it is expedient to combine droperidol with Lasix for 2-4 ml. At the expressed psycho motor excitation and appearance of cramps droperidol combine with

	seduxen or elenium. For older patients with the expressed cerebral and coronal atherosclerosis, and also an III stage of hypertensive disease BP it is expedient to reduce slowly, during 20-40 min, to warn sharp violations of cerebral and coronal circulation of blood. If GC flows with the minimum symptoms of sharp violation of cerebral circulation of blood, defeat of eyes and hypertensive encephalopathy, then Clophelinum or droperidol /better droperidol/ combine with euphylin. 10-15 ml of a 2,4 % solution of euphylin enter in a vein on 10 ml of a 0,9 % solution of chloride of sodium. In the complement of the combined therapy it costs to plug 2-4 ml of Lasix in a vein. For patients with sympathoadrenal GC marked sine tachycardia, extrasystoly, high systolic and pulse pressure, excitation, fear. In such cases appoint in a vein 3-5 ml of a 0,1 % solution of obzidan /anaprilin/ on 10-15 ml of a 0,9 % solution of chloride of sodium. The decline of BP and deceleration of heart rate comes through 5-10 min. The hypotension action of medicine proceeds a few hours. For warning of relapse GC appoint anaprilin for 80-120 mg/twenty-four hours. If acute left ventricular failure is diagnosed on a background of GC, then preparations of choice – ganglion blockersof rapid action, intravenouse introduction of nitroglycerine. In muscles enter 0,5-1 ml of a 5 % solution of pentamin. BP goes down through 15-30 min and remains mionectic during 3-4 hours For achievement of more rapid and expressed hypotension effect combine 0,5-1-2 ml of a 5 % solution of pentamin with 2 ml of a 0,25 % solution of droperidol on 100-150 ml of a 0,9 % solution of chloride of sodium, medicine enter in a vein with speed 15-30 drops for a minute under control of BP. In the first 10-20 min BP goes down on 25-30 % as compared to a weekend. At the clinical displays of cardiac asthma simultaneously appoint Lasix for 40-80 mg and inhalations of oxygen with foam extinguisher(ethyl spirit,). Instead of pentamin use arfonad or benzohexoniy. The rapid decline of BP, especially for older people, can result in a collapse and violation of regional hemodynamics. With a prophylactic aim a patient must be abed 2-3 hours. At treatment GC take into account clinical symptoms and impression of internalss. For therapy of the urgent states, hypertensive encephalopathy, acute left ventricular and kidney insufficiency, dissection of aneurysm, eclampsia ideal hypotension drug is remained nitroprussid of sodium at 0,25-10 mcg/kg/min intravenously at drops with beginning of action at once. Duration of introduction from a few hours to twenty-four hours, but the maximal dose of drug is entered during 3-7 min Diazoxyd (hyperstat) enter stream intravenously 50-150 mg or for 15-30 mg/min, beginning of action through 1-2 min, and a hypotension effect proceeds a few hours. Preparation is appointed at hypocinetic GC, malignant arterial hypertension, hypertensive ancephalopathy, GC with sharp kidney insufficiency. GC at phochromocytoma take off by phentolaminum, tropafen or nitroprusside. From physical methods assign mustard plasters, hot baths of feet, cold for a head. On occasion for liquidation of the phenomena of cardiac asthma and pulmonary edema use bloodletting for 300-500 ml. Such treatment is shown to the patients of young and middle age. To the older persons with the expressed cerebral atherosclerosis assign medical leech on processus mastoideus, for 2-4 leeches from every side. Reason of GC are psycho-emotional overstrains with blowing off adaptation mechanisms, meteorological factors, hormonal disorders. In declining and senile years the sudden increase of BP can be provoked by a sharp cerebral ischemia as a result of cerebral atherosclerosis, neck osteochondrosis or chronic circulator cerebral insufficiency. Finely, GC can cause acute myocardial ischemia with reflex excitation of CNS and sudden extrass of catecholamins in blood. Thus, during realization of prophylactic measures it costs to take into account the mechanism of development of GC, adjust the rhythm of life. Pyrroxan in a dose 0,045-0,06 g/24 hours is caused by a positive effect at the diencephalic crises of sympathoadrenal genesis from hyperadrenalinemia, reduces excitability and paroxysmal activity of cortical and subcortical structures of brain. In menopause appoint a correcting hormonotherapy which quite often results in disappearance of GC. Women with progesteron insufficiency 6-8 days prior to menstruation accept diuretics: triampur, hypothiasid. If appearance of GC is predefined by the sharp ischemia of brain, then medicinal therapy is sent to the improvement of cerebral circulation of blood. Next to the antagonists of calcium and facilities which improve microcirculation, patients get cardiotonics, that promotes tone. In the first half of day cordiamin is appointed them, adonizid and others like that. Thus, morning orthostatic low blood pressure diminishes with the sharp vibrations of BP.

Unmedicinal therapy

Modification of life style	Give to recommendation in relation to modification of life style according to the risk of cardiovascular complication, which it is possible to modify factors educed for a patient : - to decrease a bodyweight at presence of obesity or surplus mass of body; - to decrease the use of alcohol; - regularly to execute dynamic physical exercises; - to limit the use of kitchen salt a to 5 g/twenty-four hours; - to limit the use of the saturated fats and cholesterol; - to give up smoking; - to use foods with enhanceable maintenance of potassium, calcium and magnesium.
Prophylaxis of hypertensive disease and crisis	The primary prophylaxis of hypertensive disease includes a fight against risk factors. It is sent to making healthy of population with the observance of healthy way of life. Component parts of primary prophylaxis : 1. normalization of body mass with limitation of calorie content of meal, 2. feed with limitation of kitchen salt a to 4-6-10 g/ twenty-four hours, 3. physical loading at a hemodynamic situation 4. on possibility exception of stresses with modification of behaviour of people of psychological type. An especially important value a primary prophylaxis has for people with frontier arterial hypertension. The secondary prophylaxis of hypertensive disease carries massive character. A fight proceeds against risk factors. Patients with hypertensive disease are subject to the clinical supervision with the dynamic watching their health each 3-6 months by the Important component of fight from hyperlipidemia and obesity, is limitation of calorie content of food, foods with large maintenance of cholesterol and adipose. In a meal it is expedient to include foods, rich polyunsaturated fat acids and potassium, sea fish, vegetables, fruit. An amount of kitchen salt must be no more than 4-6 gs/twenty-four hours. Psychotherapy includes autotrening, reflexotherapy, volitional respiratory gymnastics, rational rest. Sanatorium-resort treatment and stay have a prophylactic value in preventive clinics at a production To the secondary prophylaxis take rational employment with the exception of terms of labour, which assist the increase of BP /nightly changes, vibration, noise/. Depending on a level and stability of BP in ambulatory terms appoint hypotension medicinal therapy, more frequent as monotherapy. The review of patient in such case is conducted one time in 2-3 weeks, and at normalization of BP - one time in 1-2 months it is Necessary to provide control by lipidogramm , ECG, state of eyeground, functions of kidneys. A secondary prophylaxis detains progress of hypertensive disease, passing of it to the malignant variant warns the origin of such complications, as a haemorrhage in a brain, cardiac asthma, pulmonary edema, kidney insufficiency, CHF and other

Independent work

А. Study of the special literature

Guidelines for the management of arterial hypertension // European Heart Journal (2013) - doi:10.1093/eurheartj/eht151

- Mayo Clinic Cardiology – third edition (2007)
- Valentine Fuster – The HEART 11-edition (2007)
- Braunwald – Heart Diseases 8th edition (2008)
- Ragavendra R. Baliga - Practical Cardiology Evaluation and Treatment of Common Cardiovascular Disorders, 2nd Edition (2008)
- Swanton R.H. – Swanton's cardiology – 6th edition (2008)
- Mohammad Shenasa – Cardiac Mapping Third Edition (2009)

1. Preparing of summary due to the topic of the lesson by the content of magazines:

- American College of Cardiologists Journal
- American Heart Journal
- American Journal of Cardiology
- American Journal of Hypertension
- British Heart Journal
- The Canadian Journal of Cardiology
- Cardiology
- Circulation
- European Heart Journal
- Heart
- Hypertension
- International Journal of Cardiology

Б. Educational-methodical manuals.

В. Preparation of abstract on the topic of employment after materials of magazines.

Decision of tests and situational tasks of Step 2.
Writing of protocols of clinical analysis of patients (see Appendix 1).

Marks of mastering of practical skills

№ from/п	Skills and manipulations	Signature student/teacher
1.	Practical skills	
1.1.	Able to conduct, physical examination of patient with a hypertensive crisis.	
1.2.	Able to analyse data of laboratory examination	
1.3.	Able to appoint base therapy to the patient with the complicated hypertensive crisis	
2.	Urgent states	
2.1.	Able to give help at appearance of left ventricular failure	

Tests for control of initial level of knowledge

1. To the group of very high risk (4) of cardiovascular complication take patients with arterial hypertension, which have:
   A. 1-2 risk factors on a background I-II degree of increase of blood pressure
   A. plural risk factors are at normal blood pressure
   B. 1-2 risk factors are at high normal blood pressure
   C. *Set cardiovascular disease
   D. A defeat of organs-targets is at high normal blood pressure.
2. Crisis motion of arterial hypertension is characteristic for:
   A. Renovascular arterial hypertension
   B. Renoparenhimal arterial hypertension
   C. Isolated systolic arterial hypertension
   D. *Pheochromocytoma
   E. Itsenko-Kushing syndrome.
3. Medicinal antihypertensive therapy needs to be begun immediately for patients with:
   A. 1-2 risk factors on a background I-II of degree of increase of blood pressure
   B. plural risk factors at normal propulsion module
   C. 1-2 risk factors at high normal blood pressure
   D. * set cardiovascular disease
   E. Defeat of organs-targets at high normal blood pressure.
4. At treatment of uncomplicated hypertensive crisis it is recommended way of conduct of preparations:
   A. Perorally
   B. *Perorally and/ore intramuscular
   C. Intramuscular and/or intravenous
   D. Intravenous.
5. What sign is pathognomonic for left ventricular failure?
   A. Jugular venous distention
   B. Hydroperitoneum
   C. Hepatomegaly
   D.*Orthopnoe
   E. Edemata on feet .
6. For the hypertensive crisis of the first type characteristically:
   A. Rapid beginning
   B. Mainly increase of systolic blood pressure
   C. Expressed vegetative symptoms
   D. *All transferred
   E. None of transferred.
7. For the hypertensive crisis of the second type characteristically:
   A. Slow development
   B. Repressing increase of diastolic blood pressure
   C. A threat of development of complications from the side of organs-targets
   D. *All transferred
   E. None of transferred.
8. It is known that for the rapid decline of blood pressure in case of hypertensive crisis it is necessary to apply preparations of short action of parenterally. Name them:
   A. Nitroprusside of sodium
   B. Nitroglycerine
   C. Esmolol
   D. *All are transferred
   E. None of transferred.
9. For the prophylaxis of the repeated increase of blood pressure during treatment of hypertensive crisis apply preparations of long duration action inward or parenterally. Name them:

A. Enalapril
B. Labetolol
C. Corinfar-retard
D. Clonidin
E. *All transferred.

10. To what safe level it is necessary to reduce blood pressure on the first stage of treatment of hypertensive crisis:
A. On 50%
B. On 40%
C. On 30_35%
D. *On 20_25%
E. Has no value.

11. At a pulmonary edema which runs across with the increased blood pressure preparations of choice is:
A. Morphin, prednisolon, uregit, corglicon;
B. Dofamin, euphylin, nitroglycerine;
C. *Nitroprusside of sodium, Lasix
D. Euphylin
E. Strofantin.

12. In an increase of blood pressure next mechanisms participate except:
A. increase of cardiac ejection
B. delay of sodium
C. increase of activity of renin
D. increase of production of catecholamine's
E. *increase of venous pressure.

13. What disease characterised an paroxysmal type of hypertension?
A. aldosteroma
B. nodular periarteritis
C. *pheochromocytoma
D. Itsenko-Kushing syndrome
E. Acromegaly.

Tests for control of eventual level of knowledge of students

1. Patient V., 42 years., complain about a stuffiness, spastic pain in region of heart, which arose up suddenly. In anamnesis hypertensive disease. Objectively:: hyperaemia and humidity of skin, pulse 82 at min, BP 240/120 mmHg. On ECG: a rhythm is a sine, correct. What complication did develop for a patient?
A. *Uncomplicated cardiac crisis
B. Complicated cardiac crisis
C. Uncomplicated cerebral crisis
Д. Complicated cerebral crisis
E. Hypothalamic paroxysm.

2. For the woman of 50 years, which is ill hypertensive illness of over 10 years, on a background stress, suddenly an BP rose to 200/110 mmHg. The state was accompanied by shaking of body, headache, tachycardia, general excitation, feeling of heat and dryness in to the mouth. Setting of what preparations is most reasonable in this case?
A. Blockers of receptors of angiotensin -II
B. Antagonists of calcium
C. *Beta-blockers
D. Diuretic
E. ACE inhibitors.

3. Woman of 57-th years is ill hypertensive disease during a few years. During the physical loading there were a headache, nausea, vomiting, weakness of left arm and leg. PBP 230/120 mmHg. Is there what tactic of doctor in relation to the terms of grant of help?
A. Treatment is in daily permanent establishment at a policlinic
B. Treatment is in ambulatory terms
C. *Hospitalization is to the neurological separation
D. Hospitalization is to the therapeutic separation
E. Hospitalization is to the cardiologic separation.

4. For a patient which suffers on hypertensive disease, after a stress situation suddenly there was an attack of dyspnea. Objectively: orthopnoe, breath rate 36 at min., moist cough with the selection of foamy sputum, breathing it is weak above the bottom departments of lungs, moist wheezes, pulse- 128 at min, BP 220/130 mmHg, I tone above the apex of heart is weak, accent of II tone above a pulmonary artery. What complication did arise up for a patient?
A. *Acute left ventricular failure
B. Sharp respiratory insufficiency.
C. Pulmonary embolism
D. Pneumonia
E. Pneumothorax.

5. What reason of high blood pressure for a patient with next clinical signs: sudden appearance of headache on a background the sharp increase of blood pressure, which is accompanied by nausea, tachycardia, pallor of skin covers, at finish accompanied with polyuria?

A. Kon syndrome
B. Itsenko-Kushing syndrome
C. Climacteric syndrome
D. * *Pheochromocytoma
E. Thyrotoxicosis.

6. At 59 years patient with hypertensive disease after stress suddenly appeared main pain, palpitation, pain in area of heart, feeling of alarm. Objectively: a patient is excited, heart rate - 120 at min, BP 240/120 mm Hg. Tones of heart are rhythmic, accent of II tone above an aorta. Setting of what preparations is most expedient in this case ?

A. Papaverin.
B. Raunatin
C. *Beta-blockers
D. Alpha-blockers
E. Adelphan.

7. High blood pressure with 10-years-old experience marked worsening of feel in the last a 2 twenty-four hours, when an increasing for intensities headache, somnolence, muscular weakness, appeared; binds it to that which accepted many rich and salt food. A kind diures marked after the reception of a 1 pill of triampur. At a review: a face is puffy, heart rate 62 at minute, BP 190\130 mmHg. The limits of heart are extended to the left on a 1 sm, neurological status without rough symptoms. A thyroid is not megascopic. Choose the most exact variant of conclusion on character of hypertension:

A. Crisis at pheochromocytoma
B. Crisis at Kon syndrome
C. Neuro-vegetative crisis
D. **Hydro-salt crisis
E. Progressive flow of hypertensive disease.

8. A patient of 65 years complaince on the attack of difficulty in breathing, pain in a heart, palpitation after the physical loading. 3 months ago carried Q-myocardial infarction. A patient is covered by a death-damp, acrocyanosis, the veins of neck filling out, pulse 110 at minute, BP 100/60 mmHg. Tones of heart are deaf. Breathing is heavy, perceptible in the distance. During a cough foamy sputumis distinguished , which is painted in a pink color. What starting mechanism of development of this state?

A. *Acute left ventricular failure
B. Sharp vascular insufficiency
C. Congestion in the organism of water and sodium
D. Acute pulmonary heart
E. Increase of selection of catecholamins.

9. A patient of 50 years, got in a hospital with complaints about great head pain, which is accompanied by nausea, palpitation, prickly pain in area of heart. At night the shortness of breath of the mixed type appeared suddenly, cough with the selection of pink foamy sputum. Objectively: the state is heavy, acrocyanosis, breath rate 36 at minute. In lungs there are soundings wheezes on all slowness. The left limit of heart is displaced outside on a 3 sm, accent of II tone above an aorta. BP 240/120 mmHg, pulse 120 at minute. What complication of hypertensive disease take place:

A. *Acute left ventricular failure
B. Acute right ventricular failure
C. Myocardial infarction
D. Pulmonary embolism
E. Hypertensive encephalopathy.

10. A woman of 65 years complains about the attack of difficulty in breathing with repressing difficulty of inhalation, feeling of raging in breasts, cough. During 25 years marks the high numbers of blood prssure, carried the Q-myoardial infarction, the state became worse hour ago after the physical loading. A patient is in position of orthopnoe, cyanochroic. BP 220/110 mmHg, pulse of 96 at min, rhythmic. Tones of heart are weak, accent of II tone and systolic noise above an aorta. In the lungs of breathing hard, weak, there are unsoundings різнокаліберні wheezes in bottom departments. Peripheral edemata are absent. These hearing of heart can testify to the presence of:

A. Atherosclerosis of aorta
B. * Heart failure
C. Insufficiency of aortic valve
D. Pulmonary heart
E. Insufficiency of mitral valve.

11. Man, 66 years, during 15 years suffers on hypertensive disease, did not treat oneself systematic. On a background the increase of BP to 200/110 mmHg a headache appeared, pain in the thorax of aching character without irradiation. On ECG - rejection of electric to the landmark of heart on the left, diffuse changes of myocardium, criterion of Sokolov-Layon a 40 mm of .Change of neurological status and sight is not educed. What complication at patient?

A. Myocardial infarction
B. Complicated hypertensive crisis
C. *Uncomplicated hypertensive crisis

D. Sharp hypertension encephalopathy.

12. A patient, 46 years, complains about the attacks of headache, feeling of pulsation in temples, dizziness, which are accompanied by palpitation, sweat, hypersalivation, muscular weakness, pain behind breastbone. During an attack a patient is pale, sharply BP rises to 280/160 mmHg. Attacks arise up spontaneously, often at night. What from the brought preparations over most effective in this case?

A. Dibasol
B. *Phentolamin
C. Furosemid
D. Metoprolol
E. Nitroglycerine.

Topic 2.
Management of patient with acute coronary syndrome.

Actuality. Ischemic heart disease (IHD) is the most frequent reason of death in Europe which stipulates almost 2 million deaths of its habitants annually. In halves of all deaths from cardiovascular diseases reason of death is IHD (on the second place is a stroke which stipulates third of all of cases of cardiovascular death. From IHD 21% men and 22% women perish annually. Pathology of coronary arteries is reason of death approximately 17% men under age 65 and 12% women of that age. Near the third of all of acute coronary events are ST-elevated Myocardial Infarction. 30-50% persons with an acute coronary syndrome (ACS) perish yet on the prehospital stage – as a rule in the first minutes after the appearance of symptoms. Thus the level of prehospital lethality of such patients in the last few years did not change substantially, while hospital lethality at ACS in the developed countries of the world considerably diminished for the last decades and survivability of patients in permanent establishment grew from 75% to 96% in a present tense. It happened due to a number of achievements in intensive cardiology, such as embodiment in practice of the special separations for intensive treatment of ACS, development of littleinvasive technologies, appearance and successful use of modern thrombolytics. Together with it given receipt in the large registers of patients with acute coronary pathology, specify on that 20 – 30% all of patients on ACS are not got reperfusion therapy in no shape or form, and for many persons which get reperfusion therapy it is not in time. Therefore actual is activation of fight against these failings for the sake of meaningful increase of survivability of patients.

Purpose of the lesson: to teach students to modern tactic of management of patient with an acute coronary syndrome, in practice to apply the modern standards of diagnostics, treatment and prophylaxis of ACS, on the basis of management of patients with ACS in the conditions of permanent establishment and policlinic.

Student must know:

1. Definition of term ACS with and without ST segment elevation.
2. Etiology and pathogenesis of ACS.
3. Key clinical signs and criteria of diagnostics of ACS.
4. Electrocardiography signs of acute coronary failure.
5. Role of instrumental methods of research in diagnostics of ACS (echocardiography, diurnal monitoring of ECG and BP, loading tests).
6. Biochemical markers of myocardial damage, inflammatory activity and thrombosis in patient with acute coronary syndrome.
7. Risk stratification of development of acute myocardial infarction and death.
8. Indications and contra-indication to performing of coronarography.
9. Recommendations for selection of modern method of treatment of patient with ACS with and without ST segment elevation.
10. Main statements of protocols of Medicare by specialty of «Cardiology».

A student must be able to:

1. Perform interview and physical examination of patients with ACS.
2. Create the plan of examination of patient with ACS, to ground application of basic methods of examination, to define indications to their performing.
3. To perform registration and interpretation of ECG in patient with ACS.
3. To estimate the results of echocardiography, diurnal monitoring of ECG and BP, loading tests.
4. To estimate the results of laboratory tests on the biochemical markers of damage of myocardium, inflammatory activity and convolutional system of blood.
5. To estimate the results of coronarography.
6. To perform a differential diagnosis with pathology accompanied by acute pain in thorax.
7. On the basis of analysis of data of laboratory and instrumental inspections, to ground and formulate the diagnosis of ACS.
8. To prescribe the proper treatment the patient with ACS with or without ST segment elevation.
9. To demonstrate the domain of medical specialist deontological principles.

Employments pass as work of students in composition small brigades near a bed sick on ACS. In obedience to a through on-line tutorial "Internal medicine" for higher medical establishments of III-IV levels of accreditation, organization of curriculum must provide participating of student in the conduct of 3- of 4 patients from ACS. During curationn of patients a student uses the proper Protocol and fills the cards of patients.

Study-time of practical lesson (5,5 hours):

1. Morning medical conference – 30 min;
2. Curation of patients in department – 2 hours;
3. Clinical analysis of case history – 1,5 hours;
4. Independent work (a study of the special literature, articles from magazines for the last 2 years, registration of diary, decision of tasks to the step 2) – 1,5 hours.

Protocol of inspection, establishment of diagnosis, treatment and prophylaxis

SECTION OF ACTIVITY	GUIDLINE TO ACTIONS
Greeting	Greet and appear a patient
Acquaintance	Collect passport information of patient (LNS, sex, age, residence, job and specialty)
Complaints of patient are in the moment of inspection	Define and detail the complaints of patient. At determination of complaints of patient pay a regard to presence: - intensive pain in a thorax - to character of pain – pressing, squeezing or burning - to duration of pain – more than 15 minutes - to the pallor of skin and mucus shells - death-damp - expressed weakness - tachi- or bradicardia - high or low level of blood pressure
Individual and domestic anamnesis, questioning after organs and systems	Find out the dynamics of disease, conduct questioning after organs and systems. 1. To the presence of risk factors. At questioning pay attention on list relatively: - overweight persons (25 kg/m^2); - tobacco smoking; - not mobile way of life; - age over 45 years; - arterial hypertension; - hypercholesterolemia; - atherosclerosis, diabetes mellitus, gout, chronic diseases of liver, buds, cardio-vascular system (ischemic diseases, myocardial infarction, strokes in anamnesis); - metabolic syndrome; - next-of-kins have illnesses of the cardio-vascular system. 2. To the presence of specific facts of anamnesis of disease: - an origin of the protracted and intensive anginal pain is first in life; - destabilizations of clinical symptoms of angina pectoris; - system displays of atherosclerotic process.
Physical examination	At the inspection of patient estimate: 1. Color and humidity of skin and mucus layers 2. Level of pulse and BP 3. Localization and description of apex push 4. Violation of cardiac rhythm 5. Presence of pathological noises and tones of heart 6. A presence of noises is on the vessels of neck 7. Wheezes are at auscultation of lungs 8. Presence of peripheral edema
Plan of inspection	General analysis of blood and urine, level of blood glucose, electrolytes, transaminases, lipidogramme, coagulograme, platelet aggregation, level of troponin I or T, MB faction of CPK, myoglobin, protein C, ANUP; ECG, EchoCG, diurnal monitoring of ECG and BP, coronarography, loadings tests (VEM, treadmill)
Laboratory and instrumental researches	Estimate levels of: - hemoglobin, WBC, leucocytes, ESR - glucose on an empty stomach - potassium, sodium, calcium, magnesium and chlorine in blood - ASAT, ALAT - protrombine, fibrinogen, other indexes of coagulation and anticoagulation systems of blood - ability of platelet aggregation - troponins I or T, MB faction of CPK - myoglobin, protein C, ANUP - total cholesterol (CS), lipoproteins of high and low density (HDL, LDL cholesterol) and triglycerides (TG) in blood serum; - urinary acid and kreatinine in blood serum; - microalbuminurea To interpret information got during a leadthrough:

	- ECG - EchoCG - Diurnal monitoring of ECG and BP - Coronarography - Loadings tests (VEM, treadmill)
Formulation of diagnosis	- Acute coronary syndrome with/without ST segment elevation - Complication (violation of rhythm and conductivity, AHF – Killip I-III, pericarditis, myocardial infarction in anamnesis with indication of date) - Heart failure (to specify the stage and functional class by NYHA). - Arterial hypertension (to specify the stage, degree and risk group) - Concomitant diseases.
Plan of treatment	To work out a plan of treatment, which must include non-medicinal and medicinal therapy
Nonmedicinal therapy	
	Give recommendations in relation to modification of way of life: - to decrease a bodyweight at presence of obesity; - to limit the use of light carbohydrates; - to decrease the use of alcohol; - regularly to execute dynamic physical exercises; - to limit the use of kitchen salt till 3 g/day in AH; - to limit the use of the saturated fats and cholestrol; - to stop smoking; - to plug in the ration of products, rich in vegetable fibers.
Medicinal therapy	
Methods of medicinal therapy	1. On the basis of interpretation of ECG to define belonging of patient to the certain type of acute coronary syndrome: with or without segment ST elevation 2. Depending on the type of ACS and possibility of performing of urgent coronarography, to define the method of recanalization of the staggered coronal vessel (thrombolysis, PCI, heparin therapy) 3. At the choice of tactic of treatment it is necessary to take into account an evidential base in obedience to these controlled researches in relation to every group of medicines, that it is needed to appoint.
Thrombolytics	
Streptokinase	*Mechanism of action:* instrumental in a system fibrinolysis and does not own bring together with a fibrin; *Data the controlled researches:* level of evidence is 1A. *It is indicated* at the acute myocardial infarction with ST segment elevation at first 6 o'clock from the beginning of disease at impossibility of leadthrough of coronarography and angioplasty. *Contra-indicated* at a hemorrhagic stroke, to the trauma of head during 3 weeks, to the gastroenteric bleeding during the last month, acute myocardial infarction without ST segment elevation, dissection of aorta.
Alteplasa	*Mechanism of action:* recombinante human plasminogen activator to of tissue type; converts plasminogen into plasmin which results in dissolution fibrinous clot; *Data the controlled researches:* level of e evidence is 1. *Indicated* at the acute myocardial infarction with ST segment elevation at first 6 hours from the beginning of disease at impossibility to perform coronarography and angioplasty. *Contra-indicated* at brain hemorrhages in anamnesis, skull mass, bleeding of any localization, suspicion on dissection of aortal aneurysm, acute myocardial infarction without ST segment elevation.
Anticoagulants	
Unfractionated heparin	*Mechanism of action*: inhibit coagulation factors directly in blood, make intensive antithrombine action, brakes formation of

Enoxaparin	thromboplastin and fibrin. *Data the controlled researches:* level of evidence is 1. *Indicated* at a coronal thrombosis at ACS, for warning and treatment of thrombotic complications of AMI, prophylaxis of reocclusion during the performing of TLT, prophylaxis of relapses of ischemia at Unstable Angina and AMI. *Contra-indicated* in internal bleeding, hemorrhagic diathesis, hemorrhagic stroke, uncontrolled hypertension, heavy kidney or hepatic insufficiency. Mechanism of action: antithrombotic effect by a inhibition of factor Xa and IIa. *Given the controlled researches:* level of evidence is 1. *Indicated* at a coronary thrombosis at ACS, for warning and treatment of thrombotic complications at AMI, prophylaxis of reocclusion during the performing of TLT, prophylaxis of relapses of ischemia at Unstable Angina and AMI. *Contra-indicated* at the decline of coagulation of any genesis, erosive-ulcerative process in GIT in the phase of exacerbation, especially with a tendency to bleeding, septic endocarditis, spinal or epidural puncture, traumas of CNS, organs of sight, ear, surgical operations on these organs.
Antiagregants	
Aspirin	*Mechanism of action:* represses formation of thromboxane A2 and reduces the capacity of platelets to agregation. *Indicated* at acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the internal bleeding, erosive diseases of GIT, enhanceable sensitiveness to preparation.
Klopidogrel	*Mechanism of action:* inhibit ADP-induced platelet aggregation and influences on the receptors of thrombocytes of IIb/IIIa. *Indicated* at acute coronary syndrome and heart attack of myocardium. *Contra-indicated* at hemorrhagic diathesis, propensity to bleeding (ulcer disease, hemorrhagic strokes), leucopenia, thrombopenia, agranulocytosis.
Statins	
Simvastatin	*Mechanism of action:* make hypolipidemic effect due to reverse depression of activity of GMG-KoA-Reductase in a liver and ileum intestine and depression of formation in the liver of lipoprotieds of very high-density. *Indicated* at a acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the severe diseases of liver, pregnancy and lactation.
Atorvastin	*Mechanism of action:* make hypolipidemic effect due to reverse depression of activity of GMG-KoA-Reductase in a liver and ileum intestine and depression of formation in the liver of lipoproteins of very high-density. *Indicated* at a acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the severe diseases of liver, pregnancy and lactation.
Beta-adrenoblockers	
Atenolol	*Mechanism of action:* make negative inotropic and chronotropic action on an account due to what the requirement of myocardium goes down in oxygen and the risk of myocardial ischemia goes down during loading. *Indicated* at a hyperpiesis, for the prophylaxis of attacks of stenocardia; heart attack of myocardium; violation of cardiac rhythm: at sinus tachicardia. *Contra-indicated* at chronic heart failure of IIB-III stage, AV-blockade of II and III degree, SA blockade, syndrome of weakness of sinus node; to acute heart failure; to cardiogenic shock; bradicardia, arterial hypotension, metabolic acidosis, bronchial asthma, hypersensitiveness to preparation.
Nebivolol	*Mechanism of action:* a beta-1-adrenoreceptor blocks selectively,

	stimulate the synthesis of endothelial relax factor, reduces cardiac extrass, BCV, ZPSO, inhibit formation of renin. *Indicated* at essential hypertension, IHD. *Contra-indicated* at chronic heart failure of IIB-III stage, AV-blockade of II and III degree, SA blockade, syndrome of weakness of sinus node; to acute heart failure; to cardiogenic shock; bradicardia, arterial hypotension, metabolic acidosis, bronchial asthma, hypersensitiveness to preparation.
Inhibitors of ACE	
Ramipril	*Mechanism of action:* convert angiotensin I to angiotensin II, that results in expansion of vessels and brings in the deposit in cardioprotective and endoteliprotective action of Ramipril, causes the decline of secretion of aldosteron, results in the considerable decline of peripheral resistance, diminishes the hypertrophy of myocardium. *Indicated* at hypertension, CHF, myocardial infarction, for the decline of risk of development of AMI, stroke. *Contra-indicated* at an enhanceable sensitiveness, heavy kidney insufficiency, hemodialisis, pregnancy, lactation.
Perindopril	*Mechanism of action:* convert angiotensin I to angiotensin II, that results in expansion of vessels and brings in the deposit in cardioprotective and endoteliprotective action of perindopril, causes the decline of secretion of aldosterone, results in the considerable decline of peripheral resistance, diminishes the hypertrophy of myocardium, reduces the surplus laying of collogen in the subendocardial layers of myocardium and proceeds in the isoensime type of miosin. *Indicated* at a hyperpiesis, CHF, myocardial infarction, for the decline of risk of development of IM, stroke. *Contra-indicated* at an enhanceable sensitiveness, heavy kidney insufficiency, pregnancy, lactation.

Independent work

2. Studying of special issues:

- ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation // European Heart Journal (2012) 33 , 2569–2619

- ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation // European Heart Journal (2011) 32 , 2999–3054

- Third universal definition of myocardial infarction // European Heart Journal (2012) 33 , 2551–2567

- Mayo Clinic Cardiology – third edition (2007)
- Valentine Fuster – The HEART 11-edition (2007)
- Braunwald – Heart Diseases 8th edition (2008)
- Ragavendra R. Baliga - Practical Cardiology Evaluation and Treatment of Common Cardiovascular Disorders, 2nd Edition (2008)
- Swanton R.H. – Swanton's cardiology – 6th edition (2008)
- Mohammad Shenasa – Cardiac Mapping Third Edition (2009)

3. Preparing of summary due to the topic of the lesson by the content of magazines:

- American College of Cardiologists Journal
- American Heart Journal
- American Journal of Cardiology
- American Journal of Hypertension
- British Heart Journal
- The Canadian Journal of Cardiology
- Cardiology
- Circulation
- European Heart Journal
- Heart
- Hypertension
- International Journal of Cardiology

Answering of tests and situation tasks by STEP2

Writing of protocols of patient management.

Marks are about mastering of practical skills

№ з/п	Skills and manipulations	Signature student/leader
1	Practical skills	
1.1	Able to canvass, physical inspection of patient on GKS.	
1.2	Able to register and interpret EKG	
1.3	Able to analyze information of laboratory inspection	
1.4	Able to analyze information of instrumental researches	
2	Exigent states	
2.1	Able to give a help at to sharp pain in a thorax	
2.2	Able to give a help at sharp cardiac insufficiency	
2.3	Able to give a help at violations of rhythm and conductivity	

Tests of initial level of knowledge

1) Factors which reduce the risk of development of ACS:
*increased level of high-density lipoproteins
diabetes mellitus
arterial hypertension
slow-moving way of life
tobacco smoking
2) What is contra-indication for prescription of beta-blockers?
sinus tachycardia
ventricular tachycardia
paroxysmal supraventricular tachycardia
*obstructive bronchitis
arterial hypertension
3) In case of acute pain in epigastric area and behind the sternum in man of middle age it is necessary to begin examination from:
sounding of stomach
X-ray of gastrointestinal tract
*ECG
gastroduodenoscopy
urine test for uropepsin
4) Which from the listed preparations is most effective antiplatelet agent?
cardicet
*aspirin
phenilin
hydrochlorothiazide
nifedipine
5) In case of local stenosis of large coronary artery the optimum method of treatment of ACS is:
conservative therapy by coronary active drugs
*percutaneous transluminal coronary angioplasty
destruction of atheromatous plaque cutting balloon
coronary bypass surgery
heart transplantation
6) The optimum method of treatment of ACS in case of defeat of three and more coronary arteries by atherosclerosis is:
coronary angioplasty
conservative therapy by nitrates and beta-blockers
conservative therapy by amiodaronum and calcium antagonists
*coronary bypass surgery
implantation of artificial pacemaker
7) The most pathognomonic ECG-sign of ACS without segment ST elevation is:
*horizontal or downsloping depression of ST segment
ST segment depression protuberant up to its top and asymmetric T-wave
ST segment elevation
deep Q-waves
QS-waves

Clinical tasks for individual education

1) Man 59 years old suffers from pressing pain behind sternum at the physical loading, which is relieved by nitroglycerine, during 5 years. The frequency of attacks was variable depending on physical activity from 1 to 4 per day. Patient regularly got beta-blockers, nitrates, aspirin. In spite of the fact of regular reception of drugs, during last three weeks patient noticed changes of characteristics of angina: they began to disturb more frequently – 10 – 12 attacks per day, became more prolonged, began to appear in night-time. On ECG – there were no significant abnormalities in comparison with previous ECG strips. Formulate a correct diagnosis.
*IHD. Unstable angina.

Acute pericarditis
IHD. Acute myocardial infarction
Hypertensive crisis
Dissection of aneurism of aorta
2) A man, 40 years old, delivered by ambulance to ICU after 2 hours from the beginning of intensive anginal attack which developed at first in life. Objective status: the state is severe, anginal pain is not relieved. BP=110/70 mmHg, HR=88 beats/min. Heart sounds are muffled, on apex – there is systolic murmur. In lungs – crackles are absent. A liver is not enlarged, there is no peripheral edema. ECG: elevation of ST segment in leads I, AVL, V1 - V4 till 8 mm, depression of ST segment in leads II, III, AVF on 2 mm.
ACS without ST segment elevation
Prinzmetal angina
Hypertensive crisis
Dissection of aneurism of aorta
*ACS with ST segment elevation
3) Patient, 68 years old, during 4 years suffers from typical stable angina. On ECG at rest changes were absent. During the last two weeks there were 6 attacks of pain, lasts one was today, very intensive, appeared at rest and lasted 2 hours, not relieved by nitrates. Ambulance was called, narcotic analgetics were applied. Objectively: condition is moderate. HR – 80/min. Heart sounds are muffled. In lungs – without abnormalities. BP is 120/65 mmHg On ECG: negative equal side T waves in leads V2-V5; horizontal depression of ST in taking of I, V5-6. Patient was admitted with the diagnosis ACS without ST segment elevation. What method will allow to verify diagnosis?
Reopletizmographia
*Blood test on Troponin
Diurnal monitoring of ECG
Blood test for glycosylated hemoglobin
Lipid profile
4) Patient U., 61 y.o., admitted to the hospital with the diagnosis ACS with ST segment elevation. First ECG was registered during typical angina status, which lasts during 1.5 hours: high ST-segment elevation in leads II, III, aVF and discordant changes in leads I, aVL i V 1-4. Thrombolysis was performed. What from the listed is criterion of efficiency of thrombolysis?
Pathological Q-wave
*Isoelectric ST-segment
Prolongation of PQ-interval
Appearance of U-wave
Prolongation of QT-interval

MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHYA STATE MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL DISEASE-1

«RATIFIED»
Methodic commission of therapeutic disciplines
Protocol № __dated _____2013

«RATIFIED»
on a methodical conference of departments of internal diseases-1
Protocol № __dated _____2013
«RATIFIED»
Dean of the medical faculty № 1 ZSMU
Docent Kompaniets V.M._____
Date 29/08/2013

METHODICAL RECOMMENDATIONS

for independent work of students and practical lessons.
Discipline of "Internal Medicine", Module 4; Content module 1

Management of patient with Acute myocardial infarction

Methodical recomendations prepared by
Doc. Kiselov S.M.

Zaporizhzhya, 2013

Topic 3

Management of patient with acute myocardial infarction.

Actuality. By the indexes of mortality rate from cardio-vascular diseases (CVD) Ukraine occupies the non-prestigious first place among the countries of European Society. Annually prevalence and morbidity of CVD grows up. Great attention demand mortality index from CVD for men of young and middle age which in 3–5 times exceeds analogical indexes in the most developed countries. In the structure of death rate of population of Ukraine CVD occupy the first place. They are reason of death of almost 60% of patients. Significant part of lethal cases, related to pathology of cardio-vascular system, is stipulated by Acute Myocardial Infarction (AMI). In spite of the certain successes in treatment of AMI, in most industrialized countries this pathology remains among major reasons of morbidity and death rate. It is known that significant part of patients which had AMI, has a high risk of complications in postinfarction period, and mortality in this group is about 20% per year. Therefore prophylactic and therapeutic measures in an early postinfarction period must be used, in the first turn, for patients with the high risk of complications. Last years the most expressed positive influence on the results of treatment of AMI had improvement of algorithms of treatment, directed recanalization of coronary artery, connected with AMI. In large randomized trials with application of early thrombolytic therapy for patients with AMI, mortality rate during 30 days was 6–10%, while in researches with application of percutaneus transluminal coronary angioplasty – 2,5%.

The above-mentioned information stipulate importance and actuality of study of etiology, key points of pathogenesis, typical and atypical clinical forms, standards of diagnostics of AMI with the purpose of optimization of medical tactic of adjusted on an early exposure and adequate treatment of patients with AMI.

Purpose of employment: to teach students to modern tactic of management of patient with acute myocardial infarction, to apply the modern standards of diagnostics, treatment and prophylaxis of AMI, on the basis of curationn patients with AMI in the conditions of hospital and policlinic.

Student have to know:

11. Determination of concept of myocardial infarction.
12. Etiology and pathogenesis of myocardial infarction.
13. Key clinical signs and criteria of diagnostics of myocardial infarction.
14. Electrocardiography signs of acute myocardial infarction.
15. Place of instrumental examination methods in diagnostics of AMI (echocardiography, diurnal monitoring of ECG and BP, loadings tests).
16. Biochemical markers of myocardial damage, inflammatory activity and thrombosis in patient with acute myocardial infarction.
17. Risk stratification of development of acute myocardial infarction and death.
18. Indications and contra-indication to coronarography.
19. Recommendation due to selection of modern method of treatment of patient with acute myocardial infarction.
20. Main statements of protocols of Medicare in specialty «Cardiology» .

A student must be able to:

4. Perform interview and physical examination of patients with AMI.
5. Create examination plan for patient with AMI, to ground application of main examination methods, to determine indications for them.
6. Perform registration and interpretation of ECG in AMI.
10. Estimate the results of echocardiography, diurnal monitoring of ECG and BP.
11. Estimate the results of laboratory tests on the biochemical markers of myocardium damage, inflammatory activity and coagulation system of blood.
12. Estimate the results of coronaroangiography.
13. Perform differential diagnosis with pathological conditions accompanied by acute pain in thorax.
14. On the basis of analysis of data of laboratory and instrumental examination, to ground and formulate the diagnosis of AMI.
15. Prescribe proper treatment to patient with AMI with or without segment ST elevation.
16. Demonstrate the ethical and moral principles of medical specialist.

Classes have to be performed as work of students in small brigades near bed of patient with ACS. Due to educational program "Internal medicine" for higher medical establishments of III-IV levels of accreditation, organization of curriculum must provide participating of student in the curation of 3 to 4 patients with ACS. During patient curation student uses the proper Protocol and fills in the cards of patients.

Time-study of practical lesson (5,5 hours):

5. Morning medical conference – 30 min;
6. Curation of patients in department – 2 hours;
7. Clinical analysis of case history (seminary) – 1,5 hours;
8. Individual work (study of special literature, articles in magazines for last 2 years, registration of diary, answering questions for STEP 2) – 1,5 hours.

Protocol of examination, formulation of diagnosis, treatment and prophylaxis

SECTION OF ACTIVITY	GUIDELINE FOR ACTIONS
Greeting	Greet and introduce yourself to patient
Acquaintance	Collect passport information of patient (LFS, sex, age, address, job

	and specialty)
Complaints of patient in the moment of examination	Determine and detail the complaints of patient. In complaints of patient pay attention to presence of: - Intensive pain in thorax - character of pain – pressing, squeezing or burning - to duration of pain – more than 15 minutes - to the pallor of skin and mucus layers - cold sweat - acute weakness - tachi- or bradicardia - high or low level of blood pressure
Individual and family anamnesis, questioning by organs and systems	Find out the dynamics of disease, perform questioning by organs and systems. 1. Presence of risk factors. During questioning pay attention on the facts of: - overweight persons ($\geq$ 25 kg/m^2); - tobacco smoking; - passive way of life; - age over 45 years; - arterial hypertension; - hypercholesterolemia; - atherosclerosis, diabetes mellitus, gout, chronic diseases of liver, kidney, cardio-vascular system (ischemic heart disease, myocardial infarction, strokes in anamnesis); - metabolic syndrome; - cardio-vascular diseases in most close relatives. 2. Presence of specific facts in anamnesis of disease: - appearance of prolonged and intensive anginal pain at first in life; - destabilizations of clinical signs of angina pectoris; - system signs of atherosclerotic process.
Physical examination	During examination of patient to estimate: 9. Color and humidity of skin and mucus layers 10. Level of pulse and BP 11. Localization and description of apex shove 12. Violation of heart rhythm 13. Presence of pathological murmurs and heart tones 14. Presence of murmurs on the neck vessels 15. Crackles in auscultation of lungs 16. Presence of peripheral edema
Plan of examination	General analysis of blood and urine, level of blood glucose, electrolytes, transaminases, lipid profile, coagulation profile, platelet aggregation, level of troponin I or T, MB fraction of CPK, myoglobin, protein C, ANUP; ECG, EchoCG, diurnal monitoring of ECG and BP, coronarography
Laboratory and instrumental researches	Estimate levels of: - hemoglobin, RBC, WBC, ESR - fasting glucose - potassium, sodium, calcium, magnesium and chlorine in blood - ASAT, ALAT - protrombine, fibrinogen, other indexes of coagulation and anticoagulation system of blood - aggregation ability platelets - troponin I or T, CPK-MB - myoglobin, protein C, ANUP - general cholesterol, cholesterol lipoproteins of high and low density (HDL, LDL) and triglycerides (TG) in blood serum; - uric acid and kreatinine in blood serum; - microalbuminuria To interpret information got from: - ECG - EchoCG - Diurnal monitoring of ECG and BP - Coronarography

Formulation of diagnosis	- Acute Q-wave (non-Q-wave) myocardial infarction - Complication (violation of rhythm and conduction, acute HF – Killip I-III, pericarditis, myocardial infarction in anamnesis with notification of date) - Heart failure (to specify the stage and functional class by NYHA) - Arterial hypertension (to specify the stage, degree and risk group) - Concomitant diseases
Plan of treatment	To work out a plan of treatment, which must include nonmedicinal and medicinal therapy
Nonmedicinal therapy	
	Give recommendations about modification of way of life: - to decrease a bodyweight at presence of obesity or in overweight patients; - to limit the use of easy digestible carbohydrates; - to decrease the use of alcohol; - to do regularly dynamic physical exercises; - to limit the use of kitchen salt till 3 g/day in hypertensive patients; - to limit the use of the saturated fats and cholesterol; - to give up smoking; - to include to ration products, which are rich of vegetable fibers.
Medicinal therapy	
Methods of medicinal therapy	1. On the basis of interpretation of ECG to define belonging of patient to the certain type of acute myocardial infarction: with the indent of Q or without the indent of Q 2. Depending from the type of AMI and possibility to perform urgent coronarography, to define the method of recanalization of defeat coronary vessel (thrombolysis, PCI, heparin-therapy) 3. In selection of treatment tactic it is necessary to take into account an evidential base in obedience to these controlled researches in relation to every group of medicines, that it is needed to be prescribed.
Thrombolytics	
Streptokinase	*Mechanism of action:* instrumental in a system fibrinolysis and does not own bring together with a fibrin; *Data the controlled researches:* level of evidence is 1A. *It is indicated* at the acute myocardial infarction with ST segment elevation at first 6 o'clock from the beginning of disease at impossibility of leadthrough of coronarography and angioplasty. *Contra-indicated* at a hemorrhagic stroke, to the trauma of head during 3 weeks, to the gastroenteric bleeding during the last month, acute myocardial infarction without ST segment elevation, dissection of aorta.
Alteplasa	*Mechanism of action:* recombinante human plasminogen activator to of tissue type; converts plasminogen into plasmin which results in dissolution fibrinous clot; *Data the controlled researches:* level of e evidence is 1. *Indicated* at the acute myocardial infarction with ST segment elevation at first 6 hours from the beginning of disease at impossibility to perform coronarography and angioplasty. *Contra-indicated* at brain hemorrhages in anamnesis, skull mass, bleeding of any localization, suspicion on dissection of aortal aneurysm, acute myocardial infarction without ST segment elevation.
Anticoagulants	
Unfractionated heparin	*Mechanism of action*: inhibit coagulation factors directly in blood, make intensive antithrombine action, brakes formation of thromboplastin and fibrin. *Data the controlled researches:* level of evidence is 1. *Indicated* at a coronal thrombosis at ACS, for warning and treatment of thrombotic complications of AMI, prophylaxis of

Enoxaparin	reocclusion during the performing of TLT, prophylaxis of relapses of ischemia at Unstable Angina and AMI. *Contra-indicated* in internal bleeding, hemorrhagic diathesis, hemorrhagic stroke, uncontrolled hypertension, heavy kidney or hepatic insufficiency. Mechanism of action: antithrombotic effect by a inhibition of factor Xa and IIa. *Given the controlled researches:* level of evidence is 1. *Indicated* at a coronary thrombosis at ACS, for warning and treatment of thrombotic complications at AMI, prophylaxis of reocclusion during the performing of TLT, prophylaxis of relapses of ischemia at Unstable Angina and AMI. *Contra-indicated* at the decline of coagulation of any genesis, erosive-ulcerative process in GIT in the phase of exacerbation, especially with a tendency to bleeding, septic endocarditis, spinal or epidural puncture, traumas of CNS, organs of sight, ear, surgical operations on these organs.
Antiagregants	
Aspirin	*Mechanism of action:* represses formation of thromboxane A2 and reduces the capacity of platelets to agregation. *Indicated* at acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the internal bleeding, erosive diseases of GIT, enhanceable sensitiveness to preparation.
Klopidogrel	*Mechanism of action:* inhibit ADP-induced platelet aggregation and influences on the receptors of thrombocytes of IIb/IIIa. *Indicated* at acute coronary syndrome and heart attack of myocardium. *Contra-indicated* at hemorrhagic diathesis, propensity to bleeding (ulcer disease, hemorrhagic strokes), leucopenia, thrombopenia, agranulocytosis.
Statins	
Simvastatin	*Mechanism of action:* make hypolipidemic effect due to reverse depression of activity of GMG-KoA-Reductase in a liver and ileum intestine and depression of formation in the liver of lipoprotieds of very high-density. *Indicated* at a acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the severe diseases of liver, pregnancy and lactation.
Atorvastin	*Mechanism of action:* make hypolipidemic effect due to reverse depression of activity of GMG-KoA-Reductase in a liver and ileum intestine and depression of formation in the liver of lipoproteins of very high-density. *Indicated* at a acute coronary syndrome and acute myocardial infarction. *Contra-indicated* at the severe diseases of liver, pregnancy and lactation.
Beta-adrenoblockers	
Atenolol	*Mechanism of action:* make negative inotropic and chronotropic action on an account due to what the requirement of myocardium goes down in oxygen and the risk of myocardial ischemia goes down during loading. *Indicated* at a hyperpiesis, for the prophylaxis of attacks of stenocardia; heart attack of myocardium; violation of cardiac rhythm: at sinus tachicardia. *Contra-indicated* at chronic heart failure of IIB-III stage, AV-blockade of II and III degree, SA blockade, syndrome of weakness of sinus node; to acute heart failure; to cardiogenic shock; bradicardia, arterial hypotension, metabolic acidosis, bronchial asthma, hypersensitiveness to preparation.
Nebivolol	*Mechanism of action:* a beta-1-adrenoreceptor blocks selectively, stimulate the synthesis of endothelial relax factor, reduces cardiac extrass, BCV, ZPSO, inhibit formation of renin. *Indicated* at essential hypertension, IHD. *Contra-indicated* at chronic heart failure of IIB-III stage, AV-blockade of II and III degree, SA blockade, syndrome of weakness of sinus node; to acute heart

	failure; to cardiogenic shock; bradicardia, arterial hypotension, metabolic acidosis, bronchial asthma, hypersensitiveness to preparation.
Inhibitors of ACE	
Ramipril	*Mechanism of action:* convert angiotensin I to angiotensin II, that results in expansion of vessels and brings in the deposit in cardioprotective and endoteliprotective action of Ramipril, causes the decline of secretion of aldosteron, results in the considerable decline of peripheral resistance, diminishes the hypertrophy of myocardium. *Indicated* at hypertension, CHF, myocardial infarction, for the decline of risk of development of AMI, stroke. *Contra-indicated* at an enhanceable sensitiveness, heavy kidney insufficiency, hemodialisis, pregnancy, lactation.
Perindopril	*Mechanism of action:* convert angiotensin I to angiotensin II, that results in expansion of vessels and brings in the deposit in cardioprotective and endoteliprotective action of perindopril, causes the decline of secretion of aldosterone, results in the considerable decline of peripheral resistance, diminishes the hypertrophy of myocardium, reduces the surplus laying of collogen in the subendocardial layers of myocardium and proceeds in the isoensime type of miosin. *Indicated* at a hyperpiesis, CHF, myocardial infarction, for the decline of risk of development of IM, stroke. *Contra-indicated* at an enhanceable sensitiveness, heavy kidney insufficiency, pregnancy, lactation.

Management of patient with cardiogenic shock
(European Recommendations – 2008)

Heart failure (HF) – complex of syndromes which is accompanied by breathlessness at rest or during physical load ad/or fatigue, crackles as a result of lung edema, edema of legs, objective evidence of pathological changes in myocardium or heart function at rest.

Clinical effect from treatment of HF is not enough for diagnostics but may be useful whn diagnosis is unclear in after diagnostic procedures.

Acute and chronic heart failure

Classification of heart failure based on clinical symptoms, explains the differences between first HF attack, transient and chronic heart failure. Transient HF is symptomatic, which occurred within a short period of time, although that could be prolonged treatment.

Differences between systolic and diastolic HF

Most patients with heart failure have manifestations of systolic and diastolic dysfunction at rest or during exercise. Patients with diastolic heart failure have a complaint and / or symptoms of HF and preserved left ventricular ejection fraction exceeding 45-50%.

Epidemiology

Prevalence of PA in the population ranges from 2 to 3% and increases dramatically among people aged over 75 years. Thus, the prevalence of heart failure among persons aged 70-80 years ranges 10-20%.

50% of patients die within 4 years from the date of diagnostics of HF. 40% of patients hospitalized with heart failure will die, or re-admitted to the hospital during a year.

Diastolic HF (EF> 45-50%) observed in half of patients with heart failure.

According to the latest research, the prognosis for these patients is the same as for patients with heart failure with systolic dysfunction.

Etiology of heart failure

The most frequent causes of worsening cardiac function: myocardial injury due to acute or chronic ischemia, increase of vascular resistance with blood pressure elevation or development of tachyarrhythmia (such as atrial fibrillation). Ischemic heart disease (IHD) is the cause of myocardial injury and development of heart failure in more than 70% of patients. Violation of the valve accounts 10% of HF cases, cardiomyopathy - 10% more.

Classification of HF by structure changes and (ACC/AHA) and symptoms (NYHA)

Stages of HF (ACC/AHA)

Stage A – high risk of HF development. Changes of structure and function are absent; complaints and symptoms are absent.

Stage B – there are structure changes in myocardium connected with development of HF, but without visible signs or symptoms.

Stage C – symptomatic HF, connected with structural changes of the heart.

Stage D – there are progressive structural changes of myocardium and symptoms of HF at rest, in spite of treatment.

Functional class of HF (NYHA)

Class I – no limitation in physical activity. Usual physical activity does not lead to excessive fatigue, palpitation or breathlessness.

Class II – mild limitation of physical activity. Usual physical activity leads to excessive fatigue, palpitation or breathlessness.

Class III – moderate limitation of physical activity. Less then usual physical activity leads to excessive fatigue, palpitation or breathlessness.

Class IV – severe limitation of physical activity. Symptoms at rest. In any physical activity discomfort sensation increases.

Algorithm of HF diagnostics

In general, patients with LV dysfunction or HF present to the healthcare provider in 1 of 3 ways:

1) **With a syndrome of decreased exercise tolerance.** Most patients with HF seek medical attention with complaints of a reduction in their effort tolerance due to dyspnea and/or fatigue. These symptoms, which may occur at rest or during exercise, may be attributed inappropriately by the patient and/or healthcare provider to aging, other physiological abnormalities (e.g., deconditioning), or other medical disorders (e.g., pulmonary disease). Therefore, in a patient whose exercise capacity is limited by dyspnea or fatigue, the healthcare provider must determine whether the principal cause is HF or another abnormality. Elucidation of the precise reason for exercise intolerance can be difficult because several disorders may coexist in the same patient. A clear distinction can sometimes be made only by measurements of gas exchange or blood oxygen saturation or by invasive hemodynamic measurements during graded levels of exercise (see ACC/AHA 2002 Guideline Update for Exercise Testing).

2) **With a syndrome of fluid retention.** Patients may present with complaints of leg or abdominal swelling as their primary (or only) symptom. In these patients, the impairment of exercise tolerance may occur so gradually that it may not be noted unless the patient is questioned carefully and specifically about a change in activities of daily living.

3) **With no symptoms or symptoms of another cardiac or noncardiac disorder.** During their evaluation for a disorder other than HF (e.g., abnormal heart sounds or abnormal electrocardiogram or chest x-ray, hypertension or hypotension, diabetes mellitus, an acute myocardial infarction (MI), an arrhythmia, or a pulmonary or systemic thromboembolic event), patients may be found to have evidence of cardiac enlargement or dysfunction.

A variety of approaches have been used to quantify the degree of functional limitation imposed by HF. The most widely used scale is the NYHA functional classification, but this system is subject to considerable interobserver variability and is insensitive to important changes in exercise capacity. These limitations may be overcome by formal tests of exercise tolerance. Measurement of the distance that a patient can walk in 6 minutes may have prognostic significance and may help to assess the level of functional impairment in the very sick, but serial changes in walking distance may not parallel changes in clinical status. Maximal exercise testing, with measurement of peak oxygen uptake, has been used to identify appropriate candidates for cardiac transplantation, to determine disability, and to assist in the formulation of an exercise prescription, but its role in the general management of patients with HF has not been defined.

Identification of a Structural and Functional Abnormality

A complete history and physical examination are the first steps in evaluating the structural abnormality or cause responsible for the development of HF. Direct inquiry may reveal prior or current evidence of MI, valvular disease, or congenital heart disease, whereas examination of the heart may suggest the presence of cardiac enlargement, murmurs, or a third heart sound. Although the history and physical examination may provide important clues about the nature of the underlying cardiac abnormality, identification of the structural abnormality leading to HF generally requires invasive or noninvasive imaging of the cardiac chambers or great vessels.

The single most useful diagnostic test in the evaluation of patients with HF is the comprehensive 2-dimensional echocardiogram coupled with Doppler flow studies to determine whether abnormalities of myocardium, heart valves, or pericardium are present and which chambers are involved. Three fundamental questions must be addressed: 1) Is the LV ejection fraction (EF) preserved or reduced? 2) Is the structure of the LV normal or abnormal? 3) Are there other structural abnormalities such as valvular, pericardial, or right ventricular abnormalities that could account for the clinical presentation? This information should be quantified with a numerical estimate of EF, measurement of ventricular dimensions and/or volumes, measurement of wall thickness, and evaluation of chamber geometry and regional wall motion.

Right ventricular size and systolic performance should be assessed. Atrial size should also be determined semiquantitatively and left atrial dimensions and/or volumes measured. All valves should be evaluated for anatomic and flow abnormalities to exclude the presence of primary valve disease. Secondary changes in valve function, particularly the severity of mitral and tricuspid valve insufficiency, should be determined.

Noninvasive hemodynamic data acquired at the time of echocardiography are an important additional correlate for patients with preserved or reduced EF. Combined quantification of the mitral valve inflow pattern, pulmonary venous inflow pattern, and mitral annular velocity provides data about characteristics of LV filling and left atrial pressure. Evaluation of the tricuspid valve regurgitant gradient coupled with measurement of inferior vena caval dimension and its response during respiration provides an estimate of systolic pulmonary artery pressure and central venous pressure. Stroke volume may be determined with combined dimension measurement and pulsed Doppler in the LV outflow tract. However, abnormalities can be present in any of these parameters in the absence of HF. No single parameter necessarily correlates specifically with HF; however, a totally normal filling pattern argues against clinical HF.

A comprehensive echocardiographic evaluation is important, because it is common for patients to have more than 1 cardiac abnormality that contributes to the development of HF. Furthermore, the study may serve as a baseline for comparison, because measurement of EF and the severity of structural remodeling can provide useful information in patients who have had a change in clinical status or who have experienced or recovered from a clinical event or received treatment that might have had a significant effect on cardiac function.

Other tests may be used to provide information regarding the nature and severity of the cardiac abnormality. Radionuclide ventriculography can provide highly accurate measurements of LV function and right ventricular EF, but it is unable to directly assess valvular abnormalities or cardiac hypertrophy. Magnetic resonance imaging or computed tomography may be useful in evaluating chamber size and ventricular mass, detecting right ventricular dysplasia, or recognizing the presence of pericardial disease, as well as in assessing cardiac function and wall motion.

Magnetic resonance imaging may also be used to identify myocardial viability and scar tissue. Chest radiography can be used to estimate the degree of cardiac enlargement and pulmonary congestion or to detect the presence of pulmonary disease. A 12-lead electrocardiogram may demonstrate evidence of prior MI, LV hypertrophy, cardiac conduction abnormality (e.g., left bundle-branch block), or a cardiac arrhythmia. However, because of their low sensitivity and specificity, neither the chest x-ray nor the electrocardiogram should form the primary basis for determining the specific cardiac abnormality responsible for the development of HF.

Laboratory Testing

Laboratory testing may reveal the presence of disorders or conditions that can lead to or exacerbate HF. The initial evaluation of patients with HF should include a complete blood count, urinalysis, serum electrolytes (including calcium and magnesium), glycohemoglobin, and blood lipids, as well as tests of both renal and hepatic function, a chest radiograph, and a 12-lead electrocardiogram. Thyroid function tests (especially thyroid-stimulating hormone) should be measured, because both hyperthyroidism and hypothyroidism can be a primary or contributory cause of HF. A fasting transferrin saturation is useful to screen for hemochromatosis; several mutated alleles for this disorder are common in individuals of Northern European descent, and affected patients may show improvement in LV function after treatment with phlebotomy and chelating agents. Magnetic resonance imaging of the heart or liver may be needed to confirm the presence of iron overload. Screening for human immunodeficiency virus (HIV) is reasonable and should be considered for all high-risk patients. However, other clinical signs of HIV infection typically precede any HF symptoms in those patients who develop HIV cardiomyopathy. Serum titers of antibodies developed in response to infectious organisms are occasionally measured in patients with a recent onset of HF (especially in those with a recent viral syndrome), but the yield of such testing is low, and the therapeutic implications of a positive result are uncertain (see a recent review of the role of endomyocardial biopsy, Evaluation of the Possibility of Myocardial Disease, in the full-text guideline. Assays for connective tissue diseases and for pheochromocytoma should be performed if these diagnoses are suspected, and serum titers of Chagas disease antibodies should be checked in patients with nonischemic cardiomyopathy who have traveled in or emigrated from an endemic region.

Several recent assays have been developed for natriuretic peptides (BNP and NT-proBNP). Several of the natriuretic peptides are synthesized by and released from the heart. Elevated plasma BNP levels have been associated with reduced LVEF, LV hypertrophy, elevated LV filling pressures, and acute MI and ischemia, although they can occur in other settings, such as pulmonary embolism and chronic obstructive pulmonary disease.

Natriuretic peptides are sensitive to other biological factors, such as age, sex, weight, and renal function. Elevated levels lend support to a diagnosis of abnormal ventricular function or hemodynamics causing symptomatic HF. Trials with these diagnostic markers suggest use in the urgent-care setting, where they have been used in combination with clinical evaluation to differentiate dyspnea due to HF from dyspnea of other causes, and suggest that its use may reduce both the time to hospital discharge and the cost of treatment. BNP levels tend to be less elevated in HF with preserved EF than in HF with low EF and are lower in obese patients. Levels of natriuretic peptides may be elevated meaningfully in women and in people over 60 years of age who do not have HF, and thus these levels should be interpreted cautiously in such individuals when distinguishing between cardiac and noncardiac causes of dyspnea. Elevated natriuretic peptide levels may lend weight to a suspected diagnosis of HF or trigger consideration of HF when the diagnosis is unknown but should not be used in isolation to confirm or exclude the presence of HF.

Physical examination, ECG, x-ray examination of thorax, EchoCG → BNP

	BNP	NT-proBNP
Unlikely HF	< 100 pg/ml	< 400 pg/ml
Doubtful HF	100 - 400 pg/ml	400 - 2000 pg/ml
Probably HF	> 400 pg/ml	> 2000 pg/ml

Laboratory Assessment

Serum electrolytes and renal function should be monitored routinely in patients with HF. Of particular importance is the serial measurement of serum potassium concentration, because hypokalemia is a common adverse effect of treatment with diuretics and may cause fatal arrhythmias and increase the risk of digitalis toxicity, whereas hyperkalemia may complicate therapy with angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and aldosterone antagonists. Worsening renal function may require adjustment of the doses of diuretics, renin-angiotensin-aldosterone system antagonists, digoxin, and noncardiac medications. Development of hyponatremia or anemia may be a sign of disease progression and is associated with impaired survival.

Serum BNP levels have been shown to parallel the clinical severity of HF as assessed by NYHA class in broad populations. Levels are higher in hospitalized patients and tend to decrease during aggressive therapy for decompensation. Indeed, there is an increasing body of evidence demonstrating the power of the addition of BNP (or NT-proBNP) levels in the assessment of prognosis in a variety of cardiovascular disorders. However, it cannot be assumed that BNP levels can be used effectively as targets for adjustment of therapy in individual patients. Many patients taking optimal doses of medications continue to show markedly elevated levels of BNP, and some patients demonstrate BNP levels within the normal range despite advanced HF. The use of BNP measurements to guide the titration of drug doses has not been shown

conclusively to improve outcomes more effectively than achievement of the target doses of drugs shown in clinical trials to prolong life. Ongoing trials will help to determine the role of serial BNP (or other natriuretic peptides) measurements in both diagnosis and management of HF.

Serial chest radiographs are not recommended in the management of chronic HF. Although the cardiothoracic ratio is commonly believed to reflect the cardiac dilatation that is characteristic of HF, enlargement of the cardiac silhouette primarily reflects changes in right ventricular volume rather than LV function, because the right ventricle forms most of the border of dilated hearts on radiographs. Similarly, changes in the radiographic assessment of pulmonary vascular congestion are too insensitive to detect any but the most extreme changes in fluid status.

Repeat assessment of EF may be most useful when the patient has demonstrated a major change in clinical status. Both improvement and deterioration may have important implications for future care, although the recommended medical regimen should be continued in most cases. Improvement may reflect recovery from a previous condition, such as viral myocarditis or hypothyroidism, or may occur after titration of recommended therapies for chronic HF. Thus, it is appropriate to obtain a repeat EF after some period of optimal medical therapy, typically 4 to 6 months, to decide about the implantation of an implantable cardioverter-defibrillator (ICD). Deterioration may reflect gradual disease progression or a new event, such as recurrent MI. Routine assessment of EF at frequent, regular, or arbitrary intervals is not recommended.

There has been no established role for periodic invasive or noninvasive hemodynamic measurements in the management of HF. Most drugs used for the treatment of HF are prescribed on the basis of their ability to improve symptoms or survival rather than their effect on hemodynamic variables. Moreover, the initial and target doses of these drugs are selected on the basis of experience in controlled trials and are not based on the changes they may produce in cardiac output or pulmonary wedge pressure. Nevertheless, invasive hemodynamic measurements may assist in the determination of volume status and in distinguishing HF from other disorders that may cause circulatory instability, such as pulmonary diseases and sepsis. Measurements of cardiac output and pulmonary wedge pressure through a pulmonary artery catheter have also been used in patients with refractory HF to assess pulmonary vascular resistance, a determinant of eligibility for heart transplantation. Cardiac output can also be measured by noninvasive methods.

Assessment of Prognosis

Although both healthcare providers and patients may be interested in defining the prognosis of an individual patient with HF, the likelihood of survival can be determined reliably only in populations and not in individuals. However, some attempt at prognostication in HF may provide better information for patients and their families to help them appropriately plan for their futures. It also identifies patients in whom cardiac transplantation or mechanical device therapy should be considered.

Multivariate analysis of clinical variables has helped to identify the most significant predictors of survival, and prognostic models have been developed and validated. Decreasing LVEF, worsening NYHA functional status, degree of hyponatremia, decreasing peak exercise oxygen uptake, decreasing hematocrit, widened QRS on 12-lead electrocardiogram, chronic hypotension, resting tachycardia, renal insufficiency, intolerance to conventional therapy, and refractory volume overload are all generally recognized key prognostic parameters, although the actual prognostic models incorporating them are not widely used in clinical practice. Although elevated circulating levels of neurohormonal factors have also been associated with high mortality rates, the routine assessment of neurohormones such as norepinephrine or endothelin is neither feasible nor helpful in clinical management. Likewise, elevated BNP (or NT-proBNP) levels predict higher risk of HF and other events after MI, whereas marked elevation in BNP levels during hospitalization for HF may predict rehospitalization and death. Nonetheless, the BNP measurement has not been clearly shown to supplement careful clinical assessment for management.

Because treatment of HF has improved over the past 10 years, the older prognostic models need to be revalidated, and newer prognostic models may have to be developed. Outcomes have been improved for most high-risk patients, which has resulted in a shift in the selection process for patients referred for heart transplantation. Routine use of ambulatory electrocardiographic monitoring, T-wave alternans analysis, heart rate variability measurement, and signal-averaged electrocardiography have not been shown to provide incremental value in assessing overall prognosis, although ambulatory electrocardiographic monitoring can be useful in decision making regarding placement of ICDs.

Treatment

Measures listed as Class I recommendations for patients in stage A or B are also appropriate for patients with current or prior symptoms of HF. In addition, moderate sodium restriction, along with daily measurement of weight, is indicated to permit effective use of lower and safer doses of diuretic drugs, even if overt sodium retention can be controlled by the use of diuretics. Immunization with influenza and pneumococcal vaccines may reduce the risk of a respiratory infection. Although most patients should not participate in heavy labor or exhaustive sports, physical activity should be encouraged (except during periods of acute exacerbation of the signs and symptoms of HF, or in patients with suspected myocarditis), because restriction of activity promotes physical deconditioning, which may adversely affect clinical status and contribute to the exercise intolerance of patients with HF.

Three classes of drugs can exacerbate the syndrome of HF and should be avoided in most patients:

1) Antiarrhythmic agents can exert important cardiodepressant and proarrhythmic effects. Of available agents, only amiodarone and dofetilide have been shown not to adversely affect survival.

2) Calcium channel blockers can lead to worsening HF and have been associated with an increased risk of cardiovascular events. Of available calcium channel blockers, only the vasoselective ones have been shown not to adversely affect survival.

3) Nonsteroidal anti-inflammatory drugs can cause sodium retention and peripheral vasoconstriction and can attenuate the efficacy and enhance the toxicity of diuretics and ACE inhibitors. A discussion of the use of aspirin as a unique agent is found later in this section.

Patients with HF should be monitored carefully for changes in serum potassium, and every effort should be made to prevent the occurrence of either hypokalemia or hyperkalemia, both of which may adversely affect cardiac excitability and conduction and may lead to sudden death. Activation of both the sympathetic nervous system and renin-angiotensin system can lead to hypokalemia, and most drugs used for the treatment of HF can alter serum potassium. Even modest decreases in serum potassium can increase the risks of using digitalis and antiarrhythmic drugs, and even modest increases in serum potassium may prevent the use of treatments known to prolong life. Hence, many experts believe that serum potassium concentrations should be targeted in the 4.0 to 5.0 mmol per liter range. In some patients, correction of potassium deficits may require supplementation of magnesium and potassium. In others (particularly those taking ACE inhibitors alone or in combination with aldosterone antagonists), the routine prescription of potassium salts may be unnecessary and potentially deleterious.

Of the general measures that should be used in patients with HF, possibly the most effective yet least used is close observation and follow-up. Nonadherence with diet and medications can rapidly and profoundly affect the clinical status of patients, and increases in body weight and minor changes in symptoms commonly precede by several days the occurrence of major clinical episodes that require emergency care or hospitalization. Patient education and close supervision, which includes surveillance by the patient and his or her family, can reduce the likelihood of nonadherence and lead to the detection of changes in body weight or clinical status early enough to allow the patient or a healthcare provider an opportunity to institute treatments that can prevent clinical deterioration. Supervision need not be performed by a physician and may ideally be accomplished by a nurse or physician's assistant with special training in the care of patients with HF. Such an approach has been reported to have significant clinical benefits.

Recommendations Concerning Aldosterone Antagonists. The addition of low-dose aldosterone antagonists is recommended in carefully selected patients with moderately severe or severe HF symptoms and recent decompensation or with LV dysfunction early after MI. These recommendations are based on the strong data demonstrating reduced death and rehospitalization in 2 clinical trial populations. The entry criteria for these trials describe a broader population than was actually enrolled, such that the favorable efficacy/ toxicity ratio may not be as applicable to patients at the margins of trial eligibility. For both of these major trials, patients were excluded for a serum creatinine level in excess of 2.5 mg per dL, but few patients were actually enrolled with serum creatinine levels over 1.5 mg per dL. In the trial of patients after MI, there was a significant interaction between serum creatinine and benefit of eplerenone. The average serum creatinine of enrolled patients was 1.1 mg per dL, above which there was no demonstrable benefit for survival.

To minimize the risk of life-threatening hyperkalemia in patients with low LVEF and symptoms of HF, patients should have initial serum creatinine less than 2.0 to 2.5 mg per dL without recent worsening and serum potassium less than 5.0 mEq per liter without a history of severe hyperkalemia. In view of the consistency of evidence for patients with low LVEF early after MI and patients with recent decompensation and severe symptoms, it may be reasonable to consider addition of aldosterone antagonists to loop diuretics for some patients with mild to moderate symptoms of HF; however, the writing committee strongly believes that there are insufficient data or experience to provide a specific or strong recommendation. Because the safety and efficacy of aldosterone antagonist therapy have not been shown in the absence of loop diuretic therapy, it is not currently recommended that such therapy be given without other concomitant diuretic therapy in chronic HF. Although 17% of patients in the CHARM (Candesartan in Heart Failure: Assessment of Reduction in Mortality and Morbidity) add-on trial were receiving spironolactone, the safety of the combination of ACE inhibitors, ARBs, and aldosterone antagonists has not been explored adequately, and this combination cannot be recommended.

Ventricular Arrhythmias and Prevention of Sudden Death

Patients with LV dilation and reduced LVEF frequently manifest ventricular tachyarrhythmias, both nonsustained ventricular tachycardia (VT) and sustained VT. The cardiac mortality of patients with all types of ventricular tachyarrhythmias is high. The high mortality results from progressive HF, as well as from sudden death. Sudden death is often equated with a primary arrhythmic event, but multiple causes of sudden death have been documented and include ischemic events such as acute MI, electrolyte disturbances, pulmonary or systemic emboli, or other vascular events. Although ventricular tachyarrhythmias are the most common rhythms associated with unexpected sudden death, bradycardia and other pulseless supraventricular rhythms are common in patients with advanced HF.

Sudden death can be decreased meaningfully by the therapies that decrease disease progression, as discussed elsewhere in these guidelines. For instance, clinical trials with beta blockers have shown a reduction in sudden death, as well as in all-cause mortality, in both postinfarction patients and patients with HF regardless of cause. Aldosterone antagonists decrease sudden death and overall mortality in HF early after MI and in advanced HF. Sudden unexpected death can be decreased further by the use of implanted devices that terminate sustained arrhythmias. Even when specific antiarrhythmic therapy is necessary to diminish recurrent ventricular tachyarrhythmias and device firings, the frequency and tolerance of arrhythmias may be improved with appropriate therapy for HF. In some cases, definitive therapy of myocardial ischemia or other reversible factors may prevent recurrence of tachyarrhythmia, particularly polymorphic VT, ventricular fibrillation, and nonsustained VT. Nonetheless, implantable defibrillators should be recommended in all patients who have had a life-threatening tachyarrhythmia and have an otherwise good prognosis.

The absolute frequency of sudden death is highest in patients with severe symptoms, or Stage D HF. Many patients with end-stage symptoms experience "sudden death" that is nonetheless expected. Prevention of sudden death in this population could potentially shift the mode of death from sudden to that of progressive HF without decreasing total mortality, as competing risks of death emerge. On the other hand, prevention of sudden death in mild HF may allow many years of meaningful survival. This makes it imperative for physicians to not only assess an individual patient's risk for sudden death but also assess overall prognosis and functional capacity before consideration of device implantation.

Secondary Prevention of Sudden Death. Patients with previous cardiac arrest or documented sustained ventricular arrhythmias have a high risk of recurrent events. Implantation of an ICD has been shown to reduce mortality in cardiac

arrest survivors. An ICD is indicated for secondary prevention of death from ventricular tachyarrhythmias in patients with otherwise good clinical function and prognosis, for whom prolongation of survival is a goal. Patients with chronic HF and a low EF who experience syncope of unclear origin have a high rate of subsequent sudden death and should also be considered for placement of an ICD. However, when ventricular tachyarrhythmias occur in a patient with a progressive and irreversible downward spiral of clinical HF decompensation, placement of an ICD is not indicated to prevent recurrence of sudden death, because death is likely imminent regardless of mode. An exception may exist for the small minority of patients for whom definitive therapy such as cardiac transplantation is planned.

Primary Prevention of Sudden Death. Patients with low EF without prior history of cardiac arrest, spontaneous VT, or inducible VT (positive programmed electrical stimulation study) have a risk of sudden death that is lower than for those who have experienced previous events, but it remains significant. Within this group, it has not yet been possible to identify those patients at highest risk, especially in the absence of prior MI. Approximately 50% to 70% of patients with low EF and symptomatic HF have episodes of nonsustained VT on routine ambulatory electrocardiographic monitoring; however, it is not clear whether the occurrence of complex ventricular arrhythmias in these patients with HF contributes to the high frequency of sudden death or, alternatively, simply reflects the underlying disease process. Antiarrhythmic drugs to suppress premature ventricular depolarizations and nonsustained ventricular arrhythmias have not improved survival, although nonsustained VT may play a role in triggering ventricular tachyarrhythmias. Furthermore, most antiarrhythmic drugs have negative inotropic effects and can increase the risk of serious arrhythmia; these adverse cardiovascular effects are particularly pronounced in patients with low EF. This risk is especially high with the use of class IA agents (quinidine and procainamide), class IC agents (flecainide and propafenone), and some class III agents (D-sotalol), which have increased mortality in post-MI trials. Amiodarone is a class III antiarrhythmic agent but differs from other drugs in this class in having a sympatholytic effect on the heart. Amiodarone has been associated with overall neutral effects on survival when administered to patients with low EF and HF. Amiodarone therapy may also act through mechanisms other than antiarrhythmic effects, because amiodarone has been shown in some trials to increase LVEF and decrease the incidence of worsening HF. Side effects of amiodarone have included thyroid abnormalities, pulmonary toxicity, hepatotoxicity, neuropathy, insomnia, and numerous other reactions. Therefore, amiodarone should not be considered as part of the routine treatment of patients with HF, with or without frequent premature ventricular depolarizations or asymptomatic nonsustained VT; however, it remains the agent most likely to be safe and effective when antiarrhythmic therapy is necessary to prevent recurrent atrial fibrillation or symptomatic ventricular arrhythmias. Other pharmacological antiarrhythmic therapies, apart from beta blockers, are rarely indicated in HF but may occasionally be used to suppress recurrent ICD shocks when amiodarone has been ineffective or discontinued owing to toxicity.

The role of ICDs in the primary prevention of sudden death in patients without prior history of symptomatic arrhythmias has been explored recently in a number of trials. If sustained ventricular tachyarrhythmias can be induced in the electrophysiology laboratory in patients with previous MI or chronic ischemic heart disease, the risk of sudden death in these patients is in the range of 5% to 6% per year and can be improved by ICD implantation.

The role of ICD implantation for the primary prevention of sudden death in patients with HF and low EF and no history of spontaneous or inducible VT has been addressed by several large trials that used only readily available clinical data as entry criteria. The first of these demonstrated that ICDs, compared with standard medical therapy, decreased the occurrence of total mortality for patients with EF of 30% or less after remote MI. Absolute mortality was decreased in the ICD arm by 5.6%, a relative decrease of 31% over 20 months. In a second trial, a survival benefit was not demonstrated with devices implanted within 6 to 40 days after an acute MI in patients who at that time had an EF less than 35% and abnormal heart rate variability. Although sudden deaths were decreased, there was an increase in other events, and ICD implantation did not confer any survival benefit in this setting. A third trial examining the benefit of ICD implantation for patients with EF less than 35% and NYHA functional class II to III symptoms of HF included both ischemic and nonischemic causes of HF; absolute mortality was decreased by 7.2% over a 5-year period in the arm that received a simple "shock-box" ICD with backup pacing at a rate of 40 bpm. This represented a relative mortality decrease of 23%, which was a survival increase of 11%. There was no improvement in survival during the first year, with a 1.8% absolute survival benefit per year averaged over the next 4 years. The DEFINITE (Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation) trial compared medical therapy alone with medical therapy plus an ICD in patients with nonischemic cardiomyopathy, NYHA class I to III HF, and an LVEF less than 36%. The ICD was associated with a reduction in all-cause mortality that did not reach statistical significance but was consistent in terms of magnitude of effect (30%) with the findings of the MADIT II (Multicenter Automatic Defibrillator Implantation II) and the SCD-HeFT (Sudden Cardiac Death in Heart Failure: Trial of prophylactic amiodarone versus implantable defibrillator therapy).

There is an intrinsic variability in measurement of EF particularly shortly after recovery from an acute coronary syndrome event. Moreover, as reviewed earlier, the pivotal primary prevention trials used a variable inclusion EF, ranging below 30% or 36%. Given the totality of the data demonstrating the efficacy of an ICD in reducing overall mortality in a population with dilated cardiomyopathy of either ischemic or nonischemic origins, the current recommendation is to include all such patients with an LVEF of less than or equal to 35%.

ICDs are highly effective in preventing death due to ventricular tachyarrhythmias; however, frequent shocks from an ICD can lead to a reduced quality of life, whether triggered appropriately by life-threatening rhythms or inappropriately by sinus or other supraventricular tachycardia. For symptoms from recurrent discharges triggered by ventricular arrhythmias or atrial fibrillation, antiarrhythmic therapy, most often amiodarone, may be added. For recurrent ICD discharges from VT despite antiarrhythmic therapy, catheter ablation may be effective.

It is important to recognize that ICDs have the potential to aggravate HF and have been associated with an increase in HF hospitalizations. This may result from right ventricular pacing that produces dyssynchronous cardiac contraction; however, the occurrence of excess nonsudden events with ICDs placed early after MI suggests that other factors may also

limit the overall benefit from ICDs. Careful attention to the details of ICD implantation, programming, and pacing function is important for all patients with low EF who are treated with an ICD. The ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities provides further discussion of the potential problem of worsening HF and LV function in all patients with right ventricular pacing.

The decision regarding the balance of potential risks and benefits of ICD implantation for an individual patient thus remains a complex one. A decrease in incidence of sudden death does not necessarily translate into decreased total mortality, and decreased total mortality does not guarantee a prolongation of survival with meaningful quality of life. This concept is particularly important in patients with limited prognosis owing to advanced HF or other serious comorbidities, because there was no survival benefit observed from ICD implantation until after the first year in 2 of the major trials. Furthermore, the average age of patients with HF and low EF is over 70 years, a population not well represented in any of the ICD trials. Comorbidities common in the elderly population, such as prior stroke, chronic pulmonary disease, and crippling arthritic conditions, as well as nursing home residence, should be factored into discussions regarding ICD. Atrial fibrillation, a common trigger for inappropriate shocks, is more prevalent in the elderly population. The gap between community and trial populations is particularly important for a device therapy that may prolong survival but has no positive impact on function or quality of life. Some patients may suffer a diminished quality of life because of device-site complications, such as bleeding, hematoma, or infections, or after ICD discharges, particularly those that are inappropriate.

Consideration of ICD implantation is thus recommended in patients with EF less than or equal to 35% and mild to moderate symptoms of HF and in whom survival with good functional capacity is otherwise anticipated to extend beyond 1 year. Because medical therapy may substantially improve EF, consideration of ICD implants should follow documentation of sustained reduction of EF despite a course of beta blockers and ACE inhibitors or ARBs; however, ICDs are not warranted in patients with refractory symptoms of HF (Stage D) or in patients with concomitant diseases that would shorten their life expectancy independent of HF. Before implantation, patients should be fully informed of their cardiac prognosis, including the risk of both sudden and nonsudden mortality; the efficacy, safety, and risks of an ICD; and the morbidity associated with an ICD shock. Patients and families should clearly understand that the ICD does not improve clinical function or delay HF progression. Most important, the possible reasons and process for potential future deactivation of defibrillator features should be discussed long before functional capacity or outlook for survival is severely reduced.

Hydralazine and Isosorbide Dinitrate

In a large-scale trial that compared the vasodilator combination with placebo, the use of hydralazine and isosorbide dinitrate reduced mortality but not hospitalizations in patients with HF treated with digoxin and diuretics but not an ACE inhibitor or beta blocker. However, in another large-scale trial that compared the vasodilator combination with an ACE inhibitor, the ACE inhibitor produced more favorable effects on survival, a benefit not evident in the subgroup of patients with Class III to IV HF. In both trials, the use of hydralazine and isosorbide dinitrate produced frequent adverse reactions (primarily headache and gastrointestinal complaints), and many patients could not continue treatment at target doses.

Of note, a post hoc retrospective analysis of both vasodilator trials demonstrated particular efficacy of isosorbide dinitrate and hydralazine in the African American cohort. A confirmatory trial has been done. In that trial, which was limited to the patients self-described as African American, the addition of hydralazine and isosorbide dinitrate to standard therapy with an ACE inhibitor and/or a beta blocker was shown to be of significant benefit. The benefit was presumed to be related to enhanced nitric oxide bioavailability. Accordingly, this combination is recommended for African Americans who remain symptomatic despite optimal medical therapy. Whether this benefit is evident in other patients with HF remains to be investigated. The combination of hydralazine and isosorbide dinitrate should not be used for the treatment of HF in patients who have no prior use of an ACE inhibitor and should not be substituted for ACE inhibitors in patients who are tolerating ACE inhibitors without difficulty.

Despite the lack of data with the vasodilator combination in patients who are intolerant of ACE inhibitors, the combined use of hydralazine and isosorbide dinitrate may be considered as a therapeutic option in such patients. However, compliance with this combination has generally been poor because of the large number of tablets required and the high incidence of adverse reactions. For patients with more severe HF symptoms and ACE inhibitor intolerance, the combination of hydralazine and nitrates is used frequently, particularly when ACE inhibitor therapy is limited by hypotension or renal insufficiency. There are, however, no trials addressing the use of isosorbide dinitrate and hydralazine specifically in the population of patients who have persistent symptoms and intolerance to inhibitors of the renin-angiotensin system.

Cardiac Resynchronization Therapy

Approximately one-third of patients with low EF and Class III to IV symptoms of HF manifest a QRS duration greater than 0.12 seconds. This electrocardiographic representation of abnormal cardiac conduction has been used to identify patients with dyssynchronous ventricular contraction. While imperfect, no other consensus definition of cardiac dyssynchrony exists as yet, although several echocardiographic measures appear promising. The mechanical consequences of dyssynchrony include suboptimal ventricular filling, a reduction in LV dP/dt (rate of rise of ventricular contractile force or pressure), prolonged duration (and therefore greater severity) of mitral regurgitation, and paradoxical septal wall motion Ventricular dyssynchrony has also been associated with increased mortality in HF patients. Dyssynchronous contraction can be addressed by electrically activating the right and left ventricles in a synchronized manner with a biventricular pacemaker device. This approach to HF therapy, commonly called cardiac resynchronization therapy (CRT), may enhance ventricular contraction and reduce the degree of secondary mitral regurgitation. In addition, the short-term use of CRT has been associated with improvements in cardiac function and hemodynamics without an accompanying increase in oxygen use, as well as adaptive changes in the biochemistry of the failing heart.

To date, more than 4000 HF patients with ventricular dyssynchrony have been evaluated in randomized controlled trials of optimal medical therapy alone versus optimal medical therapy plus CRT with or without an ICD. CRT, when added to optimal medical therapy in persistently symptomatic patients, has resulted in significant improvements in quality of life,

functional class, exercise capacity (by peak oxygen uptake) and exercise distance during a 6-minute walk test, and EF in patients randomized to CRT or to the combination of CRT and ICD. In a meta-analysis of several CRT trials, HF hospitalizations were reduced by 32% and all-cause mortality by 25%. The effect on mortality in this meta-analysis became apparent after approximately 3 months of therapy. In 1 study, subjects were randomized to optimal pharmacological therapy alone, optimal medical therapy plus CRT alone, or optimal medical therapy plus the combination of CRT and an ICD. Compared with optimal medical therapy alone, both device arms significantly decreased the combined risk of all-cause hospitalization and all-cause mortality by approximately 20%, whereas the combination of a CRT and an ICD decreased all-cause mortality significantly by 36%. More recently, in a randomized controlled trial comparing optimal medical therapy alone with optimal medical therapy plus CRT alone (without a defibrillator), CRT significantly reduced the combined risk of death of any cause or unplanned hospital admission for a major cardiovascular event (analyzed as time to first event) by 37%. In that trial, all-cause mortality was significantly reduced by 36% and HF hospitalizations by 52% with the addition of CRT.

Thus, there is strong evidence to support the use of CRT to improve symptoms, exercise capacity, quality of life, LVEF, and survival and to decrease hospitalizations in patients with persistently symptomatic HF undergoing optimal medical therapy who have cardiac dyssynchrony (as evidenced by a prolonged QRS duration). The use of an ICD in combination with CRT should be based on the indications for ICD therapy.

With few exceptions, resynchronization trials have enrolled patients in normal sinus rhythm. Although the entry criteria specified QRS duration only longer than 0.12 seconds, the average QRS duration in the large trials was longer than 0.15 seconds, with less information demonstrating benefit in patients with lesser prolongation of QRS. Two small studies, one randomized and the other observational, evaluated the potential benefit of CRT in HF patients with ventricular dyssynchrony and atrial fibrillation. Although both studies demonstrated the benefit of CRT in these patients, the total number of patients examined (fewer than 100) precludes a recommendation for CRT in otherwise eligible patients with atrial fibrillation. To date, only a small number of patients with "pure" right bundle-branch block have been enrolled in CRT trials. Similarly, the prolonged QRS duration associated with right ventricular pacing has also been associated with ventricular dyssynchrony that may be improved by CRT, but no published studies have addressed this situation as yet. Recommendations regarding CRT for patients with LVEF of less than or equal to 35%, NYHA functional class III, and ambulatory class IV symptoms or dependence on ventricular pacing have been updated to be consistent with the ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities.

Ten studies have reported on CRT peri-implant morbidity and mortality. There were 13 deaths in 3113 patients (0.4%). From a pooled assessment of 3475 patients in 17 studies, the success rate of implantation was approximately 90%. Device-related problems during the first 6 months after implantation reported in 13 studies included lead malfunction or dislodgement in 8.5%, pacemaker problems in 6.7%, and infection in 1.4% of cases. These morbidity and mortality data are derived from trials that used expert centers. Results in individual clinical centers may vary considerably and are subject to a significant learning curve for each center; however, as implantation techniques evolve and equipment improves, complication rates may also decline.

Intermittent Intravenous Positive Inotropic Therapy

Although positive inotropic agents can improve cardiac performance during short- and long-term therapy, long-term oral therapy with these drugs has not improved symptoms or clinical status and has been associated with a significant increase in mortality, especially in patients with advanced HF. Despite these data, some physicians have proposed that the regularly scheduled intermittent use of intravenous positive inotropic drugs (e.g., dobutamine or milrinone) in a supervised outpatient setting might be associated with some clinical benefits.

However, there has been little experience with intermittent home infusions of positive inotropic agents in controlled clinical trials. Nearly all of the available data are derived from open-label and uncontrolled studies or from trials that have compared one inotropic agent with another, without a placebo group. Most trials have been small and short in duration and thus have not been able to provide reliable information about the effect of treatment on the risk of serious cardiac events. Much, if not all, of the benefit seen in these uncontrolled reports may have been related to the increased surveillance of the patient's status and intensification of concomitant therapy and not to the use of positive inotropic agents. Only 1 placebo-controlled trial of intermittent intravenous positive inotropic therapy has been published, and its findings are consistent with the results of long-term studies with continuous oral positive inotropic therapy in HF (e.g., with milrinone), which showed little efficacy and were terminated early because of an increased risk of death.

Given the lack of evidence to support their efficacy and concerns about their toxicity, intermittent infusions of positive inotropic agents (whether at home, in an outpatient clinic, or in a short-stay unit) should not be used in the long-term treatment of HF, even in its advanced stages. The use of continuous infusions of positive inotropic agents as palliative therapy in patients with end-stage disease (Stage D) is discussed later in this document.

Independent work

Studying of special issues:

- ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation // European Heart Journal (2012) 33 , 2569–2619

- ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation // European Heart Journal (2011) 32 , 2999–3054

- Third universal definition of myocardial infarction // European Heart Journal (2012) 33 , 2551–2567

- Mayo Clinic Cardiology – third edition (2007)
- Valentine Fuster – The HEART 11-edition (2007)
- Braunwald – Heart Diseases 8th edition (2008)

- Ragavendra R. Baliga - Practical Cardiology Evaluation and Treatment of Common Cardiovascular Disorders, 2nd Edition (2008)
- Swanton R.H. – Swanton's cardiology – 6th edition (2008)
- Mohammad Shenasa – Cardiac Mapping Third Edition (2009)

Preparing of summary due to the topic of the lesson by the content of magazines:

- American College of Cardiologists Journal
- American Heart Journal
- American Journal of Cardiology
- American Journal of Hypertension
- British Heart Journal
- The Canadian Journal of Cardiology
- Cardiology
- Circulation
- European Heart Journal
- Heart
- Hypertension
- International Journal of Cardiology

Answering of tests and situation tasks by STEP2

Writing of protocols of patient management.

Marks are about mastering of practical skills

№ з/п	Skills and manipulations	Signature student/leader
1	Practical skills	
1.1	Able to canvass, physical inspection of patient with AMI.	
1.2	Able to register and interpret ECG	
1.3	Able to analyze information of laboratory inspection	
1.4	Able to analyze information of instrumental researches	
2	Exigent states	
2.1	Able to give a help at to acute pain in thorax	
2.2	Able to give a help at sharp cardiac insufficiency	
2.3	Able to give a help at violations of rhythm and conductivity	

Tests of initial level of knowledge

1) Changes of blood, not characteristic for the acute stage of myocardial infarction:
elevation of myoglobin
elevation of troponin I
appearance of C-reactive protein
*increased activity of alkaline phosphatase
elevation of MB-fraction of CPK
2) Most exact diagnostic ECG-sign of transmural myocardial infarction is:
negative T-wave
violation of rhythm and conductivity
*presence of QS-complex
ST-segment depression
decline of amplitude of R-wave
3) In case of occurring of acute pains in epigastric area and behind sternum in man of middle ages it is necessary to begin the examination from:
sounding of stomach
X-ray examination of gastro-intestinal tract
*ECG
gastroduodenoscopy
research of urine on uropepsin
) Which from the listed preparations is most effective antiplatelet agent?
cardicet
*aspirin
phenilin
hydrochlorothiazide
nifedipine
5) What is Dresler syndrome, in patients with acute myocardial infarction?
gap of interventricular septum
gap of interatrial septum
interruption of papillary muscle
*autoallergic reaction

nothing from listed
6) What echocardiographic signs are characteristic myocardial infarction?
diffuse hyperkinesia
diffuse hypokinesia
*local hypokinesia
local hyperkinesia
dyskinesia
7) What treatment is contraindicated at first 6 hours of myocardial infarction?
thrombolytic therapy
anticoagulation therapy
*digitalis therapy
therapy by calcium antagonists
therapy by peripheral vasodilators

Clinical tasks for individual education

1) Suddenly, in man, 55 y.o., after abundant feast with application of strong alcohol, appeared intensive pain in epigastrium and right subcostal space. There was single vomit. Pulse - 100/min, rhythmic, BP - 90/60 mmHg. Blood test: Hb - 152 g/l, WBC - $9,5x10^9$/l, ESR - 8 mm/h. ECG: ST-segment elevation up to 4 mm in leads III, aVF. What pathology can be assumed in patient?
Food intoxication
Acute cholecystitis
Acute pancreatitis
Perforation of gastric ulcer
*Acute myocardial infarction
2) Patient, 62 y.o., appealed to the cardiologist only four days after acute pain attack behind sternum, which lasted more than an hour. Objective status: patient condition is satisfactory, BP -120/75 mmHg, HR - 82/min. ECG: rhythm is sinus. Signs of complete left bundle branch block (not fixed before). What biochemical can prove the diagnosis of acute myocardial infarction?
ASAT, ALAT
Myoglobin
*Troponin I
MB-CPK
Alkaline phosphatase
3) In cardiologic department was hospitalized patient with complaints on pain behind sternum. Condition is severe, cold sweat, pulse is arrhythmic, BP -140/90 mmHg. On ECG: in V1-V3 – QS-wave. What disease is necessary to think about?
Myocardial infarction of lateral wall
*Myocardial infarction of anterior wall
Myocardial infarction of posterior-diaphragm wall
Non-Q-wave myocardial infarction
Myocardial infarction of right ventricle
4) Patient, 49 y.o., driver, admitted to cardiologic clinic with complaints on squeezing pain behind sternum with irradiation to the neck, which appeared two hours ago, weakness. Validol and nitroglycerine without effect, condition in admission is severe. A skin is pale, moisture. Heart sounds are muffled, HR - 96/min, BP - 110/70 mmHg. Abdomen is soft, a liver is not enlarged. Edema is absent. What is preliminary diagnosis?
Neuro-circulatory dystony
Angina pectoris
Pulmonary tromboembolism
*Acute myocardial infarction
Dissection of aorta
5) Patient, 54 y.o., complaints on elevation of temperature up to 37,8°C, weakness. Yesterday after work in-cold experienced pain between scapulas. Objectively: insignificant pallor and humidity of skin. HR - 62/min, I heart tone is weakened. BP - 110/70 mmHg. In lungs there is vesicular breathing, single wheezes (patient is smoker). Abdomen is soft, moderate flatulence. On ECG: rhythm is sinus, Q-wave in leads II, III, AVF - till 0,04 sec., with ST-segment elevation on 2 mm; depression of ST-segment in I lead on 1 mm. In blood: WBC -$10,0x10^9$/L, ESR - 12 mm/h. What is the diagnosis of patient?
Dry pleurisy
Prinzmetal angina
*Acute myocardial infarction
Respiratory infection
Acute pericarditis
6) Patient, 73 y.o., admitted to the hospital with acute myocardial infarction of left ventricle. Noticeable pericardial pulsation. Relapsing paroxysms of ventricular tachycardia. On ECG there is the acute phase of Q-wave myocardial infarction without dynamics. X-ray symptoms of congestive heart failure. On a 9th day in patient appeared dyaphoresis, weakness, prolonged tachycardia and subfebril fever. What is the most credible diagnosis?
Relapse myocardial infarction

*Trombendokarditis
Pulmonic tromboembolism
Internal heart rupture
Focal pneumonia
7) Man, 49 y.o., on 4th day of myocardial infarction complaints on the shortness of breath, breathlessness, expressed weakness. Objectively: cyanosis, PS - 110/min, BP-100/70 mmHg, BR - 34/min. Tones of heart are muffled, accent II heart sound above pulmonic artery. Under clavicula wet crackles are heard. What is the most probable complication?
Bilateral hospital-acquired pneumonia
Cardiogenic shock
Paroxysm of ventricular tachicardia
*Edema of lungs
Dressler syndrome
8) Patient, 52 y.o., complains of the intensive squeezing retrosternal pain during 4 hours. On ECG – horizontal depression of ST-segment is in leads III, aVF till 2 mm, high T-wave and ST-segment elevation is registered in leads V1-V4 till 3 mm. On the repeated ECG in 3 hours the above-described changes were saved. The diagnosis – acute myocardial infarction without complications. When the patient can be allowed to walk in limits of the ward?
*On a 4-5th day
From a 1th day
On a 10-12th day
On a 13-14th day
On a 15-16th day
9) Patient, 54 y.o., admitted to ICU department with diagnosis: acute Q-wave myocardial infarction. After 24 hours of hospital treatment patient state worsened considerably. Complaints on breathlessness. Objective status: skin is pale, wet, cold by touch. Breathing of vesicular, weakened, BR - 36/min, PS -110/min, rhythmic, heart tones are muffled, rhythm of gallop, BP - 80/40 mmHg, urination - 10 ml per 1 hour. What complication of acute myocardial infarction developed in this patient?
*Cardiogenic shock
Cardiac asthma
Edema of lungs
Acute heart aneurysm
Syndrome of Dresler
10) Patient, 70 y.o., hospitalized in infarction departmet in 4 hours after the beginning of retrosternal pain. Four months ago patient has suffered from stroke. His diagnosis: IHD, acute Q-wave myocardial infarction of anterior-septal area, apex and lateral wall of left ventricle. Complete AV-block. BP -160/80 mmHg. In lungs there are moist non-sonorous rales. What from the listed is absolute contra-indication to thrombolysis?
A presence of moist rales in lungs
Age of patient
High BP
Complete AV-block
*Stroke in anamnesis

MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHYA STATE MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL DISEASE-1

«RATIFIED»
Methodic commission of therapeutic disciplines
Protocol № __dated _____2013

«RATIFIED»
on a methodical conference of departments of internal diseases-1
Protocol № __dated _____2013
«RATIFIED»
Dean of the medical faculty № 1 ZSMU
Docent Kompaniets V.M._____
Date 29/08/2013

METHODICAL RECOMMENDATIONS

for independent work of students and practical lessons.
Discipline of "Internal Medicine", Module 4; Content module 1

Management of patient with
Acute pulmonary embolism
Management of patient with
Sudden cardiac death

Methodical recomendations prepared by
Doc. Lashkul D.A.

Zaporizhzhya, 2013

Topic 4.

Management of patient with pulmonary embolism. Management of patient with sudden cardiac death.

Actuality. Pulmonary embolism is a common and potentially lethal condition. Most patients who succumb to pulmonary embolism do so within the first few hours of the event. Despite diagnostic advances, delays in pulmonary embolism diagnosis are common and represent an important issue. javascript:showrefcontent('refrenceslayer');As a cause of sudden death, massive pulmonary embolism is second only to sudden cardiac death.

In patients who survive a pulmonary embolism, recurrent embolism and death can be prevented with prompt diagnosis and therapy. Unfortunately, the diagnosis is often missed because patients with pulmonary embolism present with nonspecific signs and symptoms. If left untreated, approximately one third of patients who survive an initial pulmonary embolism die from a subsequent embolic episode.

The incidence of pulmonary embolism in the United States is estimated to be 1 case per 1000 persons per year. Studies from 2008 suggest that the increasing use of computer tomography (CT) scanning for assessing patients with possible pulmonary embolism has led to an increase in the reported incidence of pulmonary embolism. Pulmonary embolism is present in 60-80% of patients with DVT, even though more than half these patients are asymptomatic. Pulmonary embolism is the third most common cause of death in hospitalized patients, with at least 650,000 cases occurring annually. Autopsy studies have shown that approximately 60% of patients who have died in the hospital had pulmonary embolism, with the diagnosis having been missed in up to 70% of the cases. Prospective studies have demonstrated DVT in 10-13% of all medical patients placed on bed rest for 1 week, 29-33% of all patients in medical intensive care units, 20-26% of patients with pulmonary diseases who are given bed rest for 3 or more days, 27-33% of patients admitted to a critical care unit after a myocardial infarction, and 48% of patients who are asymptomatic after a coronary artery bypass graft.

Target classes: to teach students modern tactics of a patient with pulmonary embolism, in practice, to apply current standards of diagnosis, differential diagnosis, prevention and treatment**.**

The student should know:

1. The definition of PE
2. Etiology and pathogenesis of pulmonary embolism
3. Risk factors for venous thromboembolism
4. Classification of pulmonary embolism
5. Clinical criteria for diagnosis of pulmonary embolism
6. Key markers of stratification PE
7. Therapeutic approach in pulmonary embolism
8. Standard first aid to patients with pulmonary embolism
9. Prevention of pulmonary embolism

The student should be able to:

1. To conduct survey and physical examination of the patient with pulmonary embolism
2. Identify various options for PE course
3. Draw up a plan of survey of patient with pulmonary embolism, justify application of the main invasive and noninvasive methods of examination, to determine the indications and contraindications for ix in, and possible complications
4. Based on the analysis of clinical, laboratory and instrumental examination to conduct a differential diagnosis, justify and formulate a diagnosis
5. Assign treatment, conduct primary and secondary prevention of pulmonary embolism in
6. To demonstrate the possession of moral and ethical principles of medical specialist and principles of professional subordination

Classes are held in the form of students' work within small teams in the bed of the patient with pulmonary embolism. During Supervision sick student enjoys Protocol and fills a patient (see Appendix 1).

Protocol examination, diagnosis, treatment and prevention of pulmonary embolism in accordance with the standards of evidence-based medicine on the basis of the European Society of Cardiology concerning acute pulmonary embolism and national clinical protocols (2007)

SECTION OF	GUIDE TO ACTION
Welcome	Introduce yourself
Acquaintance	Collect the passport data of the patient (name and patronymic surname., sex, age, residence, place of employment and specialization)
The complaints of the patient at the time of inspection	Identify and details a complaint a patient. In determining patient complaints, note the presence of: 6) pain (characterized by chest pain that is exacerbated during deep inspiration and coughing due to the development of heart attack, pneumonia and aseptic inflammation pleural leaves). The pain can be intense, radiate to the blade space as a result of acute pulmonary artery enlargement, ischemic lung tissue or irritation of nerve endings in the pericardium. 7) acute respiratory distress syndrome (characterized by the sudden appearance of expiratory dyspnea, cyanosis, bronchospasm). Shortness of breath is the result of irritation of the respiratory center in response to hypoxia, hypercapnia and pulmonary hypertension. In massive PE indicates the development of cyanosis

	face, neck, upper torso (iron color is severe weather). Sudden cyanosis in combination with shortness of breath, palpitations and pain in the chest is a reliable sign of PE. 8) The syndrome of acute circulatory failure (collaptoid) (resulting from the reflex fall in blood pressure in a large circle of blood circulation and reduce blood flow in the LV, due to a sharp increase in pressure in the lesser circulation). Progressive reduction of blood pressure can lead to death. 9) The syndrome of acute right ventricular failure (accompanied by the development of acute pulmonary heart by reflex spasm of blood vessels of the lungs and a sharp rise in diastolic pressure, resulting in a surge RV, reducing its contractile ability (as evidenced by the epigastric pulsation, displacement limits of the relative dullness of the heart to the right, swelling of the neck veins, venous positive pulse). Sometimes auscultated diastolic murmur Graham Still, due to the expansion of pulmonary artery rings, the return of blood from the pulmonary artery in RV. Rarely can develop acute left ventricular failure as a result of displacement interventricular septum into the cavity left ventricular dilation by RV. 10) The syndrome of acute cardiac arrhythmias (accompanied by the occurrence of sinus tachycardia, extrasystole, AF, blockade of the right bundle branch block, myocardial hypoxia caused by congestion and RV, which leads to the formation of ectopic foci and even ventricular fibrillation). 11) The syndrome of acute coronary insufficiency is secondary. It is caused by the mismatch between blood supply and metabolic needs RV, decreased blood pressure and arterial hypoxemia. In such cases, Electrocardiogram (ECG) ST segment shifts below the baseline and there is a negative spike T. At autopsy patients who died of myocardial necrosis in determining fire subendocardial layer resulting from myocardial ischemia. 12) Cerebral syndrome (characterized by psychomotor agitation, meningeal and focal symptoms, seizures and sometimes coma, caused by the development of hypoxic brain encephalopathy and cerebral edema). 13) Abdominal syndrome (characterized by pain in the liver, nausea, vomiting, flatulence (symptom of dynamic intestinal obstruction), resulting from increased liver , stretching hlison capsule and spasm of the arteries of the abdominal cavity). 14) Intoxication syndrome (characterized by fever at the onset to subfebrile figures sometimes - febrile). Its duration is from 2 to 12 days. 15) Immunological syndrome (occurs within 2-3 weeks, manifested rash like urticaria, pulmonitis, recurrent pleurisy, eosinophilia, appearance in the blood of circulating immune complexes).
Individual and family history, survey of the agencies and systems	Find out the dynamics of disease, conduct surveys for agencies and systems In the survey, note the information on: - **Risk factors:** - Phlebitis and thrombophlebitis of the lower (least upper) limb veins of the pelvis; - Tumors of various organs; - Heart disease with severe cardiomegaly, large cavities left and right ventricles (mitral stenosis, coronary heart disease [CHD], MI, dilated cardiomyopathy, diffuse myocarditis, infective endocarditis, atrial fibrillation [AF], chronic heart failure, etc.); - Surgery, prolonged immobilization of limbs, injuries. - **Availability of the symptoms of the target**: - Lung: pain, acute respiratory distress syndrome; - Vessels: collapse, resulting in reflex blood pressure fall; - Heart palpitations, arrhythmias and conduction, chest pain, shortness of breath, swelling of the legs; - Brain: psychomotor agitation, meningeal and focal symptoms, seizures and sometimes coma, caused by the development of hypoxia; - Abdomen: pain in the liver, nausea, vomiting, flatulence (symptom of dynamic intestinal obstruction)
Physical examination	On examination the patient with PE noteworthy characteristic pale ash hue of the skin with cyanosis of mucous membranes and nail lodges. When massive thromboembolism can develop pronounced cyanosis of the skin of the upper half of the body. Cough with a little phlegm, hemoptysis, which occurs in about 1/3 of cases, fever, pleural rub, wet rales in the lungs, chest pain pleural character associated with pulmonary infarction or heart attack, pneumonia and is often accompanied by

	thrombosis of small LA branches. In submassive PE sharp increase in pulmonary vascular resistance leads to severe dysfunction of the right ventricle (acute pulmonary heart), which shows clinical and electrocardiographic characteristics. Clinical symptoms include abnormal pulsation, tone accent II and noise on systolic pulmonary artery, protodiastolic gallop, swelling of the neck veins, enlarged liver.
Plan Survey	On examination the patient rate: 1. The severity 2. The situation in bed 3. Signs of destruction of the target 4. Electrocardiographic study 5. X-ray study 6. Clinical and biochemical laboratory tests
Laboratory and instrumental investigations (routine method)	Make a plan for patient examination including routine recommended methods and advanced methods of examination shown a particular patient
Laboratory and instrumental investigations (recommended method)	Evaluate test results 1. In the **analysis of blood** - leukocytosis with leukocyte possible shift to the left, increased erythrocyte sedimentation rate, eosinophilia, lymphopenia, monocytosis relative. 2. The level of **lactate dehydrogenase** (LDH-3) (rare LDH-1) increased, bilirubin in the blood increased. Increased troponin in the blood accompanied by a significant increase in hospital mortality (44% vs. 3% in patients without increasing concentrations of troponin). 3. Determination of blood by ELISA **D-dimer**, one of the degradation products of fibrinogen and fibrin from the activation of fibrinolysis (normal is 0.5 mg / ml blood), which is a favorable indicator and avoids nemasyvnu PE in the first phase of the study in 99%. However, its increase does not increase the accuracy of diagnosis of the disease. 4. **Pleural fluid**, often haemorrhagic, has signs of inflammation. 5. **ECG** criteria for the study: • a sudden shift of electrical axis of heart right, signs hypertrophy PSH; • negative prong T and displacement segment ST, and sometimes unstable prong Q in III and aVF-leads to a dynamically; • sinus tachycardia, extrasystoles, right bundle branch block bundle; • P-pulmonale: high gabled prong R in III and aVF-leads. 6. **Radiographic** criteria: • depletion of pulmonary vascular pattern (pathognomonic symptom), increasing the transparency of the lungs (symptom Westermarck); • deformation or increase in one of the roots of the lungs; • protrusion cone pulmonary artery; • enlargement of the heart by PSH; • myocardial lungs - conical shadow, which is directed to the root tip of the lungs; • high standing of the diaphragm on the affected side; • possible presence of fluid in the pleural cavity 7. Criteria-**echocardiography** study: • increased end-diastolic size of the PSH and reduced contractile ability of the latter; • asymmetric thickening of the interventricular membrane and its prolabuvannya in left ventricular cavity, which may impede the flow of blood from the left atrium in the LV, which is a risk factor for pulmonary edema. 8. The **computer tomography** (CT)criteria pulmonary angiography or selective: presence of thrombus, obturation vascular filling defect (olihemiya - reduction of perfusion in the periphery of the lungs). 9. Criteria for **selective angyopulmonography**: • increase the diameter of the pulmonary artery; • full or partial occlusion of the artery, no vascular staining of lung on the affected side; • «diffuse" or "patchy" nature of the staining of blood vessels; • defects of filling in the vessel in the presence of rare blood clots; • deformation of the pulmonary pattern as expansion; • changes in equity or segmental vascular lesions When multiple small branches.
Stratification of risk for cardiovascular complications	Key markers of stratification (Recommendation of the European Society of Cardiology, 2008) 1) Clinical markers: Shock. Hypertension.

	2) Markers dysfunction RV: Dilatation RV, hypokinesis and overload pressure on echocardiography. RV dilatation of spiral CT. The increase in blood levels of brain natriuretic peptide (BNP) or n-terminal brain natriuretic peptide (NT-proBNP). Increased pressure in RV for his catheterization. 3) Markers of myocardial damage: Positive cardiac troponin T or I.
Determining patient prognosis	Prognosis depends on what branch pulmonary arteries affected. Supermassive (lightning) form continues several minutes, accompanied by a rapid increase in dyspnea (respiratory rate [RR] - 45-50/pm), tachycardia (heart rate - 140-150/pm), warm cyanosis, fall in blood pressure to zero and the development of death. Massive form (main branches) is acute for ten minutes, characterized by wheezing (RR - 40-45/pm), palpitations (heart rate - 130-140/pm), diffuse cyanosis, pain in the chest and the sternum, fear of death. Nonmassive (secondary branches) is characterized by wheezing (RR - 25-35/pm), palpitations (heart rate - 100-130/pm), lower blood pressure to 80/60 mm Hg. century., cough, hemoptysis and of heart attack, pneumonia 48 hours after the start of PE. PE small branches of pulmonary artery accompanied by sudden shortness of breath (RR - 30-35/pm), palpitations (heart rate - 90-100/pm), sometimes short-term arterial hypotension and loss of consciousness, which can lead to chronic pulmonary heart.
Formulating a diagnosis	Example diagnosis: PE, submassive form, acute pulmonary compensated heart, pulmonary insufficiency II degree, left leg vein thrombophlebitis in the acute phase.
Plan of treatment	Make a plan of treatment, which should include non-drug and drug therapy
Treatment. Standards of care for patients with acute pulmonary embolism	
Selecting tactics of drug therapy	Treatment of patients with pulmonary embolism include removal of pain, reduction of pressure in the pulmonary artery, restoring blood circulation, prevent recurrence of thromboembolism
Emergency Prehospital treatment	1. Step 1. Haute anesthesia: • talamonal (fentanyl 1-2 ml of 0.005% solution of droperidol + 1.2 ml of 0.25% solution) in/v, in/m systolic blood pressure> 100 mm Hg. century.; • promedol - 1 ml of 1% solution, morphine - 0.5 ml of 1% solution i/m 2. Step 2. Relief of collapse: • Dopamine - 1 ml of 0.5% solution / drip; • Prednisolone - 60-90 mg in\v, in/m; • reopolyglukine - 400 ml of 10% solution, Neogemodez. 3. Step 3. Lowering the pressure in the pulmonary circulation: • Theophylline - 10 ml 2.4% solution in/v; • papaverine, drotaverine - 2 ml of 2% solution i/v, i/m 4. Step 4. Conducting anticoagulant therapy: • Heparin - 10000-15000 IU / v, then to 60 U/kg p/w; • fraxyparine - 0.6 ml s/c.
Treatment in a hospital	**Step 1**. Thrombolytic therapy: • In the first 4-6 hours after onset of pulmonary embolism with massive and submassive form to dissolve a blood clot injected: - Thrombolytics and generation streptokinase, streptase, streptoliase, kabikinase, avelizyn to 1.5 million units in/or streptodecase - 3 million units, or urokinase - 2 million units in / for 1-2 hours; - Second-generation thrombolytic - aktylize, tissue plasminogen activator 100 mg: 15 mg bolus, 50 mg over 30 minutes, 35 mg over 1 hour (the effect of the introduction of thrombolytics in / or directly to the pulmonary artery same). Thrombolytics convert inactive plasminogen into active plasmin, which is a natural thrombolytics. They better put into the pulmonary artery through a catheter. **Step 2.** Anticoagulation for prevention of thrombosis with pulmonary embolism nemasyvniy form: • Heparin - 5000 units to 4 times a day p / w (heparin injected w / a dose of 5000-10000 IU bolus and then in / infusion at a rate of 1000-1500 IU / h for 7 days under the control of activated partial thromboplastin time (APTT), which normally is 50-70 seconds, or fraksyparyn, nadroparin, kleksan in 0.6 ml of n / w for 7 days; • nadroparin (fraksyparyn Na) - to 86 IU / kg / per bolus, then - to 86 IU / kg every 12 hours or 190 IU / kg (forte) once a day p / w; • Enoxaparin (kleksan) and 1 mg / kg p / w every 12 hours; • warfarin - 5-6 mg 1-2 times daily or fenilin - 0.2 g / day for 2-3 months under the supervision of INR (international normalized ratio, which should be 2.0-3.0) for 3 months and more;

	• antiagrigant aspirin less effective than anticoagulants. **Step 3.** Surgery. Emergency embolectomy absolutely indicated for zverhmasyvniy and massive pulmonary embolism, accompanied by sustained systemic arterial and severe pulmonary hypertension. An alternative to surgery in some cases it may be an extension (bouginage) thromboembolism in the pulmonary artery via catheter Fogarty (controlled fluoroscopy injected probe with a balloon on the end and made thrombus fragmentation followed by administration of thrombolytics). **Step 4.** Prevention of recurrent pulmonary embolism: • Drug: anticoagulant warfarin within 3-6 months or more; • antiplatelet: aspirin, clopidogrel, abciximab administered in optimal doses (less effective than anticoagulants); • Surgical prophylaxis implantable umbrella coffee filters in infrarenalnyy department inferior vena cava or put "traps emboli" through skin punctures jugular or femoral veins, ligation of main veins (femoral vein below the mouth of a deep thigh veins).
Recommendations of the British Society regarding the therapeutic management of patients with pulmonary embolism assumptions (1997)	1. Therapy of choice for **massive pulmonary embolism** is the thrombolysis (level B). It can be used for clinical indications under the threat of cardiac arrest (level B). Recommended bolus alteplase 50 mg (Level C). However, thrombolysis is not a therapy of choice for nemasyvniy pulmonary embolism (level B). 2. Heparin should be administered to patients with moderate or high clinical probability of PE to image diagnosis (Level C). Unfractionated heparin bolus is used for the first dose with massive pulmonary embolism if necessary rapid termination of anticoagulation (Level C). 3. Low molecular weight heparins have the same efficacy as unfractionated heparin, but they are easier to use (level of evidence A). 4. Oral anticoagulants administered immediately after confirmation of the diagnosis of venous thromboembolism. INR should be maintained at 2.0-3.0. After reaching this level of heparin can cancel (level of evidence A). 5. The standard duration of therapy with oral anticoagulants is: 4-6 weeks in the presence of transient risk factors (level of evidence A), 3 months in primary idiopathic venous thromboembolism (level A) and a minimum of 6 months for other forms of the disease. The risk of bleeding must be balanced with the risk of recurrent venous thromboembolism (Level C). However, in a randomized controlled trial PREVENT after a standard course of anticoagulation patients consistently received low-dose warfarin (INR 1.5-2.0). Under warfarin recurrent thrombosis were observed less frequently because of bleeding less evolved.
Clinical management of patients with massive pulmonary embolism	**Step 1.** Cardiac arrest Cardiorespiratory resuscitation. 50 mg alteplase / in. Reevaluation of 30 minutes. **Step 2.** The deterioration of the patient with pulmonary embolism Specialist consultant. 50 mg alteplase / in. Broad-spectrum antibiotics if indicated. Urgent echocardiography. **Step 3.** Relapses of the disease 80 U / kg heparin / for 5-7 days and then warfarin 10 mg / day. Urgent echocardiography and ECG study. In case of deterioration perform Step 2. **Important**! In case of thrombocytopenia in the application of heparin platelet transfusion may complicate matters, the appointment of warfarin in large doses can cause gangrene of the limb.
Prevention	Prevention of pulmonary embolism is aimed at preventing the causes that contribute to disease development. In order to prevent phlebemphraxis in preoperative period prescribed: 1. Low molecular weight heparins: • Enoxaparin (kleksan, lavenoks) 40 mg (or 4000 IU) 1 per day or 30 mg (3000 IU) 2 times a day; • fraksyparyn (nadroparin) in 0.3 mL (or 3075 IU) for three days and on the 4th day - 0.4 ml (or 4100 IU) 1 per day; • dalteparin (FRAGMIN) of 5000 IU 1 per day or 2500 IU 2 times daily; • revyparyn (klivaryn) in 0.25-0.5 ml (or 1750-3500 IU) 1 time per day. 2. Timely expansion bed regime after the operation, mobilization of limbs, physiotherapy, use of antiplatelet drugs, and in the postoperative period - the use of elastic or pneumatic compression of legs, elastic stockings.

	3. When recurrent pulmonary embolism in the inferior vena cava insert temporary or permanent coffee filters through a catheter through the subclavian, jugular or femoral vein. After implantation of a decrease in the frequency of pulmonary embolism on the 12th day and after 2 years of observation, but coffee filters do not affect mortality. Coffee filters are implanted only in the case when anticoagulants are contraindicated or pulmonary embolism on their background recurs.

Sudden cardiac death (SCD) is an unexpected death due to cardiac causes occurring in a short time period (generally within 1 h of symptom onset) in a person with known or unknown cardiac disease. Most cases of SCD are related to cardiac arrhythmias. Approximately half of all cardiac deaths can be classified as SCDs. SCD represents the first expression of cardiac disease in many individuals presenting with out-of-hospital cardiac arrest. This article explores the epidemiology, pathophysiology, diagnostic approach, and treatment of patients who experience SCD.

Frequency

International

The frequency of SCD in Western industrialized nations is similar to that in the United States. The incidence of SCD in other countries varies as a reflection of the prevalence of coronary artery disease or other high-frequency cardiomyopathies in those populations. The trend toward increasing SCD events in developing nations of the world is thought to reflect a change in dietary and lifestyle habits in these nations. It has been estimated that SCD claims more than 7,000,000 lives per year worldwide.

History

Obtaining a thorough history from the patient, family members, or other witnesses is necessary to obtain insight into the events surrounding the sudden death. Patients at risk for SCD may have prodromes of chest pain, fatigue, palpitations, and other nonspecific complaints. History and associated symptoms, to some degree depend on the underlying etiology of SCD. For example, SCD in an elderly patient with significant coronary artery disease may be associated with preceding chest pain due to a myocardial infarction, while SCD in a young patient may be associated with history of prior syncopal episodes and/or a family history of syncope and SCD and due to inherited arrhythmia syndromes. As many as 45% of persons who have SCD were seen by a physician within 4 weeks before death, although as many as 75% of these complaints were not related to the cardiovascular system. A prior history of LV impairment (ejection fraction < 30-35%) is the most potent common risk factor for sudden death.

Risk factors that relate to coronary artery disease and subsequent myocardial infarction and ischemic cardiomyopathy also are important and include a family history of premature coronary artery disease, smoking, dyslipidemia, hypertension, diabetes, obesity, and a sedentary lifestyle. Specific considerations include the following:

Coronary artery disease

- Previous cardiac arrest
- Syncope
- Prior myocardial infarction, especially within 6 months
- Ejection fraction less than 30-35%
- History of frequent ventricular ectopy (more than 10 PVCs per h or nonsustained VT)

Dilated cardiomyopathy

- Previous cardiac arrest
- Syncope
- Ejection fraction less than 30-35%
- Use of inotropic medications

Hypertrophic cardiomyopathy

- Previous cardiac arrest
- Syncope
- Family history of SCD
- Symptoms of heart failure
- Drop in SBP or ventricular ectopy upon stress testing
- Palpitations
- Most are asymptomatic

Valvular disease

- Valve replacement within 6 months
- Syncope
- History of frequent ventricular ectopy
- Symptoms associated with severe uncorrected aortic stenosis or mitral stenosis

Long QT syndrome

- Family history of long QT and SCD
- Medications that prolong the QT interval
- Bilateral deafness

Wolff-Parkinson-White (WPW) syndrome (with atrial fibrillation or atrial flutter with extremely rapid ventricular rates)

With extremely rapid conduction over an accessory pathway, degeneration to VF can occur.

Treatment approach

The main goal of treatment in sudden cardiac arrest is to achieve a return of circulation. The algorithm of basic (BLS) and advanced cardiac life support (ACLS) provided by the International Liaison Committee on Resuscitation (ILCOR) gives a systematic approach to the treatment of sudden cardiac arrest. The methods vary slightly based on the underlying rhythm and cause of sudden cardiac arrest, but all rely on immediate attention to stabilising the patient's respiratory status, addressing airway management as needed, and providing medicines and other life-saving treatments aimed at correcting the unstable rhythm, as well treating the underlying cause.

After activation of emergency medical services, the first approach to sudden cardiac arrest is BLS, providing compressions (first priority), assessing the airway, and giving breaths. This C-A-B priority recommendation in adults is a change in the guidelines from the A-B-C priority taught historically in order to emphasise the importance of providing timely chest compressions. Patients who require further treatment are then given ACLS by trained providers.

At any given point in the provision of ACLS, the rhythm may change from ventricular tachycardia (VT)/ventricular fibrillation (VF) to pulseless electrical activity (PEA)/asystole or vice versa. In such an event, the appropriate ACLS algorithm for the new rhythm should be followed.

Shockable rhythms (VT and VF)

In the setting of pulseless VT/VF, the initial management is of BLS as described above (C-A-B method). Early provision of CPR by bystanders in out-of-hospital arrest increases the rate of survival from sudden cardiac arrest. Unfortunately, studies have shown that less than one third of patients with sudden cardiac arrest may receive the CPR they require, possibly due to fear on the part of bystanders that mouth-to-mouth ventilation may result in the transmission of communicable disease(s). Work has, however, shown that cardiac-only resuscitation by bystanders for out-of-hospital sudden cardiac arrest is equally, if not more, efficient in providing life-saving therapy.

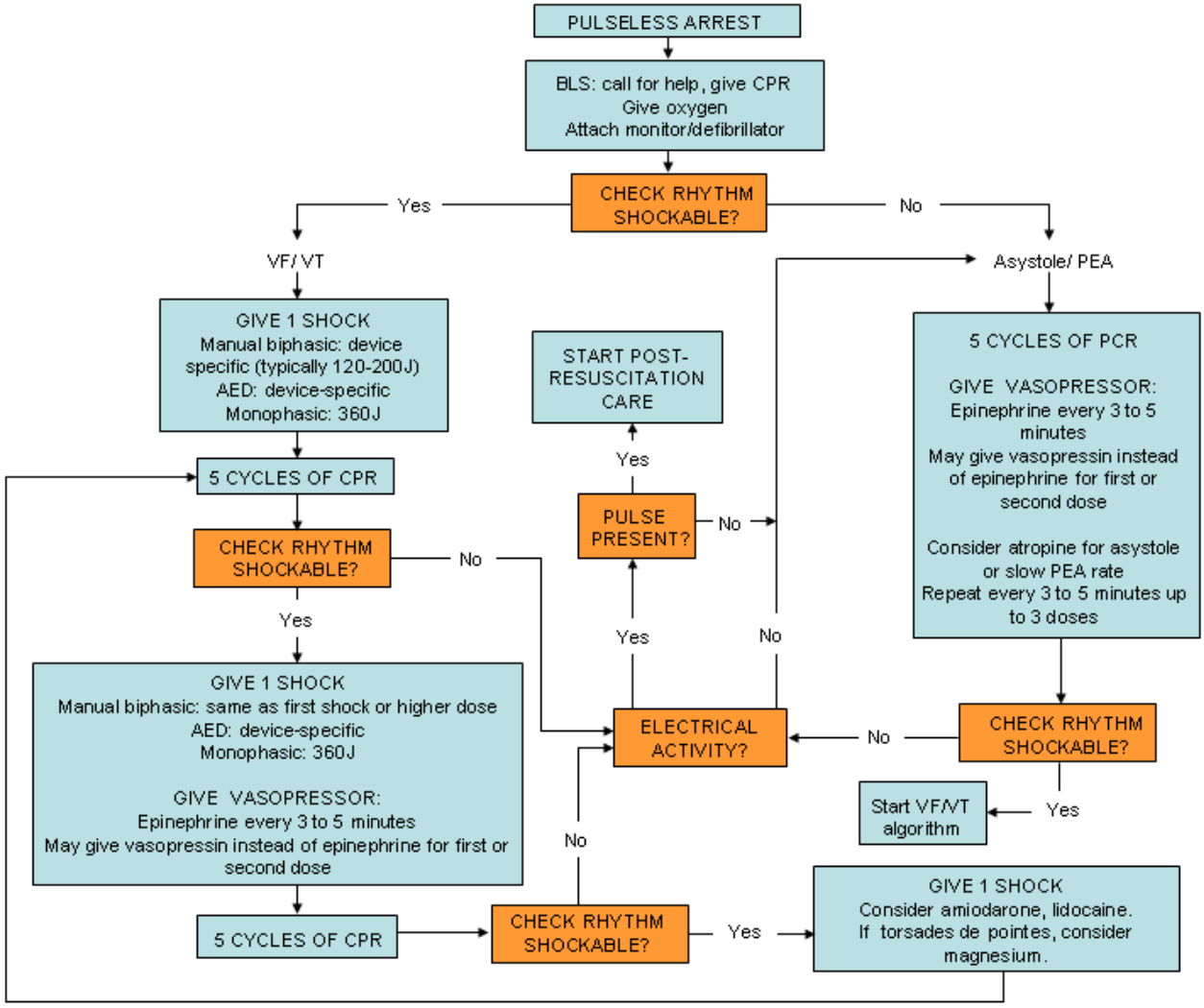
If circulation is not restored, 1 shock should be delivered (200 J for biphasic and 360 J for monophasic), followed by 5 cycles of CPR. The rhythm is again assessed and if the patient remains in VT/VF another shock is delivered and epinephrine (adrenaline) or vasopressin is given, followed by 5 cycles of CPR. If the rhythm is still VT/VF, another shock is delivered along with amiodarone or lidocaine, and CPR is continued for 5 cycles. If the patient remains in a shockable rhythm, the algorithm restarts at the stage of epinephrine (adrenaline)/vasopressin administration. This cycle continues until circulation is achieved or resuscitative measures are abandoned.

It should be mentioned that in situations of witnessed arrest, electrical defibrillation should be attempted as soon as possible, not necessarily after 5 cycles of CPR. http://bestpractice.bmj.com/best-practice/monograph/283/resources/references.html - ref-51Due to the importance of prompt defibrillation, the use of 'public access defibrillation' by lay rescuers using automatic external defibrillators has gained favour and been found to increase the rate of sudden cardiac arrest patients surviving to hospital discharge.

In patients with sudden cardiac arrest due to torsades de pointes, giving magnesium may restore a perfusing cardiac rhythm.

Non-shockable rhythms (PEA and asystole)

In the setting of PEA/asystole, the initial provision is of BLS as described above (C-A-B method). If 5 cycles of CPR are unsuccessful to produce a circulatory rhythm, epinephrine (adrenaline) or vasopressin is given. Atropine may be given in the setting of asystole or slow rate of PEA. This cycle of giving CPR and medicine continues until a perfusing rhythm is attained or resuscitation is terminated. In addition, empirical treatment for likely reversible causes may be considered; for example, calcium bicarbonate for hyperkalaemia in patients with a history of renal failure. There is no evidence to suggest that transcutaneous pacing should be used in patients with asystolic arrest.



Post-resuscitation care

Postresuscitation care should be instigated immediately. This involves continued monitoring, organ support, correction of electrolyte imbalances and acidosis, and safe transfer to a critical care environment. A thorough search for potential aetiologies should be conducted and risk factors for sudden cardiac arrest modified or treated. Failure to maximise each of these elements will result in a spared heart with an unsalvaged brain.

Anoxic brain injury is a frequent complication of sudden cardiac arrest. Multiple studies of therapeutic hypothermia (TH; lowering the core body temperature to 32 to 34°C [89.6 to 93.2°F]) have demonstrated an improvement in neurological function. A systematic review of the literature confirms that hypothermia protocols improve survival and neurological outcome following resuscitation from sudden cardiac arrest, without a significant increase in the rate of adverse events. While the meta-analysis cited mostly includes studies of patients with VT/VF arrest, a recent comprehensive analysis including only patients with non-shockable arrest demonstrated similar benefit for TH hypothermia on mortality and neurological outcomes. While most studies of TH induce TH in the hospital setting, more recent preliminary work has demonstrated the safety and feasibility of TH induction by emergency providers in the field. Further study on whether this is beneficial compared with in-hospital induction of TH is necessary.

Long-term management focuses primarily on the attempted prevention of recurrence. Patients should abstain from toxic substances. Comparison of anti-arrhythmic therapy with the use of implantable cardioverter-defibrillators (ICD) has shown a significant reduction in mortality with the use of ICDs compared with drug therapy in the secondary prevention of sudden cardiac arrest.

Independent work

Studying of special issues:

Guidelines on the diagnosis and management of acute pulmonary embolism // European Heart Journal (2008) 29 , 2276–2315

- Mayo Clinic Cardiology – third edition (2007)
- Valentine Fuster – The HEART 11-edition (2007)
- Braunwald – Heart Diseases 8th edition (2008)
- Ragavendra R. Baliga - Practical Cardiology Evaluation and Treatment of Common Cardiovascular Disorders, 2nd Edition (2008)
- Swanton R.H. – Swanton's cardiology – 6th edition (2008)
- Mohammad Shenasa – Cardiac Mapping Third Edition (2009)

Preparing of summary due to the topic of the lesson by the content of magazines:

- American College of Cardiologists Journal
- American Heart Journal
- American Journal of Cardiology
- American Journal of Hypertension
- British Heart Journal
- The Canadian Journal of Cardiology
- Cardiology
- Circulation
- European Heart Journal
- Heart
- Hypertension
- International Journal of Cardiology

Answering of tests and situation tasks by STEP2

Writing of protocols of patient management.

Tests of initial level of knowledge

1. Is any of the signs does not comply with right ventricular failure?
A. increase in liver
B. * reduce venous pressure
C. slowing blood flow velocity
D. cyanosis
E. edema
2. Which of the following is the most effective antiplatelet drugs?
A. sustak-fort
B. * aspirin
C. fenilin
D. dihlotiazid
E. nifedipin
3. Intermittent dyspnea expiratory type is typical:
A. Cardiac asthma
B. pneumonia
C. * Bronchial Asthma
D. emphysema
E. pneumothorax
4. The term that defines the notion of shortness of breath are:
A. tachypnea
B. apnea
C. Polipnoe
D. * Dispnoe
E. All listed
5. Wheezing occurs suddenly with all the above diseases, except for:
A. Spontaneous pneumothorax
B. Pulmonary embolism
C. Paralysis of diaphragm
D. Psyhonevrotychnyy state
E. * Congestive Heart Failure
5. The most likely cause of sudden shortness of breath in bed sick after expansion mode are:
A. * Pulmonary Artery Embolism
B. Hypostatic pneumonia
C. Spontaneous pneumothorax
D. Paralysis of diaphragm
E. All listed
6. Acrocyanosis resulting from:
A. * Reducing the amount of oxygen through the vasospasm of small vessels
B. Increased oxygen via vasospasm of small vessels
C. Dilatation of small vessels
D. All listed
E. None of the above.
7. Characteristic signs of central cyanosis, which distinguish it from acrocyanosis is?
A. The widespread nature of the bluish color
V.. Ash-gray skin
C. Skin warm to the touch
D. Acrocyanosis tongue
E. * All listed.
8. The development of acrocyanosis with pulmonary embolism caused by?
A. left ventricular heart failure

V. * right ventricular heart failure
C. Change of vascular tone in violation of microcirculation
D. All listed
E. None of the above.
9. Female 64 years old with unstable angina during gait suddenly collapsed. Another doctor at the examination, the patient stated: fainting, lack of pulsations in A.carotis and heart tones, narrow pupil and liquid, shallow breathing. What is the most likely diagnosis?
A. Swoon
B. Collapse
C. PE
D. Asphyxia
E. * Sudden stop circulation

Tests to control the final level of knowledge

1. The patient was suddenly a sharp pain in the left half of the chest, there was an attack of breathlessness. Objective: the patient is excited, skin and mucous membranes pale, Crocq's disease. Varicose veins of lower extremities. Pulse 120 per min., Blood pressure 100/70 mm Hg Cardiac deaf, accent II tone of the pulmonary artery. Respiratory ratr 28/min., breathing weakened the left. What is preliminary diagnosis?
A. Myocardial infarction
B. asthmatic status
C. Dressler syndrome
D. pneumonia
E. * PE
2. Patient taken to hospital unconscious. Objectively: pale skin, Crocq's disease, swollen neck veins. Liquid breathing, noisy, pulse 120 per min., AT 130/70 mm Hg The ECG: heart rate 120 per min., Sinus rhythm, EMU is facing right, P-pulmonale, positive symptoms SIP, QII, TIII. Acute right bundle branch block bundle, signs of right ventricular overload. What is the pathological condition developed in this patient?
A. Myocardial infarction
B. Acute left ventricular failure
C. Dressler syndrome
D. Cardiogenic shock
E. * PE
3. The patient M., 70 years with acute shortness of breath, chest pain, post exercise, Crocq's disease, swelling of neck veins performed perfusion pulm scyntigraphy, according to which the accumulation of radiopharmaceuticals is defined only in the projection of the right lung. From history we know that sometimes there are attacks of atrial fibrillation. As evidenced by the survey data?
A. The central left lung cancer
B. Acute inflammation of the left lung
C. Emphysema left lung
D. * PE
E. Chronic bronchitis
4. When paroxysms of atrial fibrillation finished patient complaints of sudden chest pain, shortness of breath. Objectively: skin covered with sweat, neck veins edema. Pulse small, 140 per minute. Blood pressure is determined. The ECG: right axis deviation, presence of P-pulm., Positive symptoms S1, QII, TIII. What complications arose in the patient?
A. Cardiogenic shock
B. cardiac tamponade
C. * PE
D. Cardiac asthma
E. Dressler Syndrome
5. The patient complains of sudden shortness of breath, which appeared after physical exertion. Long time suffering from thrombophlebitis of the lower extremities. Objectively the patient's condition is difficult, cyanosis of mucous membranes, acrocyanosis. respiratory rate 38 per min., shallow breathing, the lungs - dramatically weakened vesicular breathing right. Heart rate 106 per min., Filling and low tension, blood pressure 90/60 mm Hg Cardiac muted. What method of diagnosis should be used?
A. Ultrasonography of the heart
V. spirography
S. * Electrocardiography
D. Bronchoscopy
E. X-ray of the chest
6. In patients 50 years of varicose veins of lower extremities after physical exertion appeared pronounced shortness of breath, pain in the chest on the right side, palpitations, dizziness, fainting brief states. Objectively: skin pale cyanotic. Tachypnea - 40 breath. for 1 min., dry whistling wheezing. Tachycardia - 130 bpm. at min. Blood pressure 80/50 mmHg. The ECG - electrical axis deviation to the right, right bundle branch block. What is the most likely diagnosis in a patient?
A. * Pulmonary Embolism
B. Myocardial Infarction
C. Spontaneous pneumothorax
D. Acute interstitial pneumonia
E. Pleurisy

7. The patient in '65 with thrombophlebitis of lower extremities suddenly appeared pain in the right half of the chest, marked dyspnea, hemoptysis, sudden weakness. OBJECTIVE: cyanosis of the face, neck, heart rate - 130 per min., Arrhythmic, BP - 80/60 mm Hg Atrial fibrillation, muted tones. Tachypnea - 30 breath. per min., small-and medium-vesicles wheezing on the right side. ECG - QSIII. Which drugs should be in the first place?
A. Heparin
B. Reopoliglyukin
C. *Streptokinase
D. Aspirin
E. Nitroglycerin
8. D. The patient is in the unconscious. In the history - atrial fibrillation. Objectively: Crocq's disease, swollen neck veins. Liquid breathing, noisy. Ps-130 per min., Blood pressure 110/70 mm Hg The ECG: heart rate 130 per min., Sinus rhythm, right axis deviation, P-pulmonale, positive symptoms SI, QII, TIII. Acute right bundle branch block. What should be the first?
A. Pentamin
B. Adrenaline
C. *Streptokinase
D. Digoxin
E. Atropine
9. Man in '55 was treated at the surgical department with acute venous thrombophlebitis of lower extremities. On the 7th day of treatment suddenly there were pains in the left half of the chest, choking, coughing. Temperature 36.1, frequency of respiratory movements 36 in 1 minute. Above the lungs weakened breathing, no wheezing. Pulse 140 in 1 minute, threadlike. Blood pressure 70/50 mm Hg. What is the most likely diagnosis?
A. * Pulmonary Artery Embolism
B. myocardial infarction.
C. Cardiac asthma.
D. Bronchial asthma.
E. Pneumothorax.
10. Sick '36, complains of severe shortness of breath ("shortage" of air), sharp pain in the lower right chest, coughing up blood in appearance of sputum, sharp weakness that appeared suddenly after physical stress (dug in the garden). The objective: the patient's condition is difficult, cyanosis of mucous membranes, acrocyanosis. respiratory rate 32 per 1 min. pulse-106 in 1 min. BP-100/60 mm Hg. The case in the lower chest sharply weakened vesicular breathing, percussion-sound is shortened. On the right shin in the lower third-varicosity of hyperemia of the surrounding skin. On that diagnosis should think?
A. right-sided focal pneumonia in the lower lot.
V. * pulmonary embolism, heart attack, right-sided pneumonia.
S. right-sided dry pleurisy.
D. Spontaneous pneumothorax.
E. Lower right-sided pneumonia holdings.
11. The patient was suddenly a sharp pain in the left half of the chest, there was an attack of breathlessness. Objective: the patient is excited, skin and mucous membranes pale, with cyanotic hue. Varicose veins of lower extremities. Pulse 120 per min., BP 100 and 70 mm. Hg. Cardiac deaf, the second tone accent over the pulmonary artery. Breathing weakened the left. Submit a preliminary diagnosis.
A. * Pulmonary Artery Embolism
B. Dressler's syndrome
C. Pneumonia
D. myocardial infarction
E. pleural effusion
12. The patient on the 4th day after surgery on the right ovary cyst suddenly appeared pain in the right half of the chest with a discharge pink sputum, fever to 37.7 C. An examination of the lungs revealed blunting of lung sounds in the lower divisions case, ibid listen isolated moist rales. What complication has developed in a patient?
A. Lung abscess
B. Pneumonia.
C. * Infarction of lung.
D. Pleural effusion.
E. Pneumothorax.
13. After elimination of paroxysms of atrial fibrillation in a patient suddenly have pain in the chest, shortness of breath. Objectively: skin covered with sweat, neck veins edema, eyes wide open. Pulse small, 140 per minute. BP is not defined. The ECG: heart axis deviation to the right. What complications arose in the patient?
A. Cardiogenic shock
B. * Pulmonary Embolism
C. Defects of IAS
D. Cardiac asthma
E. cardiac tamponade
14. In patient 60, who for 20 years suffers from hypertension, after a stressful situation arose suddenly an attack of breathlessness. OBJECTIVE: orthopnea position, pulse - 120/hv., BP - 210/120 mm Hg. And the tone of the apex of the

heart is weakened, in diastole additional audible tone, respiratory rate - 32/b.p.m. Breath of the lower regions of the lungs weakened, isolated noteless moist rales. What complications arose?
A. Dissection of the aorta
B. attack of hysteria
C. Nonmassive pulmonary artery embolism
D. pneumonia
E. * Acute left ventricular failure
15. Patient 62 years, joined the admissions department with an attack of breathlessness. Suffering from hypertension 16 years, 3 years ago suffered a myocardial infarction. OBJECTIVE: orthopnea, skin pale, cold sweat, acrocyanosis. BP - 230/130 mm Hg., pulse -108/b.p.m., respiratory rate - 36/b.p.m. Auscultatory: scattered dry rales over all areas of the lungs, in the lower divisions - Wet rales. What is the most likely complication?
A. Acute myocardial infarction
B. * Acute left ventricular failure
C. An attack of asthma
D. Pulmonary embolism
E. Acute right ventricular failure
16. Patient 56 years old, complained of severe pain behind the breastbone fiery nature, which lasted for an hour. Nitroglycerin has not given effect. OBJECTIVE: agitated patient. Pulse - 90/b.p.m. BP - 160/90 mm Hg. And the tone of the tip relaxed. The ECG: sinus rhythm, significant ST elevation in leads I and AVL. What is the most likely diagnosis?
A. Hypertensive disease management
B. * Unstable angina
C. Pulmonary embolism
D. myocardial infarction
E. Pericarditis
17. The patient in '42 suddenly appeared pain behind the breastbone, fainting, shortness of breath after 2 days, coughing up blood. OBJECTIVE: orthopnea, cyanosis pronounced diffusion, swollen neck veins. respiration rate - 42/b.p.m., heart rate - 120/b.p.m., BP - 90/60 mm Hg. Liver 5 cm below the edge of the costal arch, more action, painful varicose veins of legs. ECG: sinus tachycardia, deep QIII S1, ST segment elevation in lead III, R in V1 = 9mm. The clinical situation is regarded as:
A. Pneumothorax
B. Myocardial infarction
C. Lobar pneumonia
D. * Pulmonary Artery Embolism
E. Paroxysm of atrial fibrillation
18. Patients in '46 after removal of plaster from his feet suddenly felt a pain behind the breastbone, shortness of breath. On the 3rd day of the disease was coughing up blood. OBJECTIVE: orthopnea, cyanosis pronounced diffusion, swollen neck veins. RR - 40/b.p.m., Heart rate - 120/hv. BP - 90/60 mm Hg. Liver 4 cm, painful. Swelling of the left tibia. ECG: sinus tachycardia. R in VI -9 mm. Which additional methods gives the most reliable data to confirm the diagnosis?
A. Bronchoscopy
B. Holter monitoring of ECG
C. Radiography of the lungs
D. Determination of MB CK
E. * Pulmonary angiography
19. Patient 58 years old, after abdominal surgery complains of pain in the left half of the thorax. Infrastructural facilities, favored maintaining status quo: heart rate 102/b.p.m., Weakened heart tones. The ECG pathological Q in I, aVL; QS in V1 V2, V3 leads and ST elevation with negative T. What is the most likely diagnosis?
A. Pulmonary embolism
B. Dissection of the aorta
C. Variant angina
D. exudative pericarditis
E. * Myocardial infarction
20. Patient 54 years turned complaining of sudden chest pain, shortness of breath, palpitations, cough. OBJECTIVE: jugular venous distension, the second tone accent on pulmonary artery systolic sound, beat, enlarged liver. In the lungs - blunting of pulmonary tone, pleural rub. The ECG: right bundle branch block, SI, QIII. Which department should hospitalize the patient?
A. pulmonological
B. * Vascular
C. Surgical
D. infarct
E. Therapeutic

Topic 5.
Management of patients with paroxysmal arrhythmias and conduction.

Background. Strategy for management of patients with cardiac arrhythmias has undergone over the past 10-15 years, dramatic changes. Priority examination of patients was the assessment of opportunities and mechanisms of radical removal of arrhythmias. Significantly decreased values antiarrhythmics determined by the absence of convincing evidence of their effectiveness to improve the prediction of survival of patients with the most common cardiac arrhythmias. In many clinical situations, the appointment of medicines no longer be justified at all, due to an unfavorable ratio of expected benefits and potential risks of antiarrhythmic therapy. On the other hand, the use of non-invasive methods of examination of patients allowed in many clinical situations to identify patients at increased risk of cardiac arrhythmias and thus determine the reason for the primary prevention of arrhythmias and sudden cardiac death. Leading role in the treatment of cardiac arrhythmias were playing catheter intervention and implantation of devices such as pacemakers and internal cardioverter-defibrillator.

Despite the development of improved methods of treating cardiac arrhythmias, the principles of primary diagnosis in clinical practice has not significantly changed. The basis of examination of patients remaining algorithms electrocardiography and other methods that include ECG.

Objective lessons: teach students modern tactics of the patient with arrhythmia and conduction of the heart, to practice modern standards for diagnosis, differential diagnosis and prevention of Tsikh conditions, determine the tactics in many ways, including, complicated course, under the Supervision of patients with arrhythmia and conduction of the heart in the hospital.

The student should know:

1. Definition of arrhythmias and conduction of the heart
2. Etiology and pathogenesis of arrhythmias and conduction of the heart
3. Risk stratification of cardiovascular complications, prognosis and performance of patients with arrhythmia and conduction of the heart
4. Clinical management (examination, treatment) patients with impaired cardiac rhythm and conduction disturbances depending on the type and risk
5. The principles of non-pharmacological and pharmacological treatment of patients with arrhythmia and conduction of the heart
6. Drugs in the treatment of arrhythmias and conduction of the heart, indications and contraindications for ix destination
7. Current recommendations on the choice of tactics therapy (drug or electrocardiostimulation)
8. Types of permanent pacemakers and selection criteria
9. Primary and secondary prevention of arrhythmias and conduction of the heart

The student should be able to:

1. Conduct survey and physical examination of patients with arrhythmias and conduction of the heart
2. Interpret ECG
3. Identify various options for arrhythmias and conduction of the heart
4. Draw up a plan of survey of patients with arrhythmias and conduction of the heart, justify application of basic invasive and non-invasive methods of examination, to determine the indications and contraindications of ix and possible complications
5. Based on the analysis of laboratory and instrumental examination conduct differential diagnosis, formulate and justify the diagnosis of major diseases and conditions that are occurring with arrhythmia and conduction of the heart
6. Prescribe treatment to conduct primary and secondary prevention of disorders of heart rhythm and conduction
7. Diagnose and assist in neuskladnenomyh arrhythmias and conduction disturbances of the heart rhythm and conduction of the heart
8. To demonstrate the moral and ethical principles of medical specialist and principles of professional subordination
9. Classes are held in the form of student work within small teams to the bedside of arrhythmia and conduction of the heart. According to the curriculum "Internal Medicine" for higher medical institutions III-IV accreditation levels, the organization of the curriculum is to provide students participated in the conduct of 4 patients with impaired cardiac rhythm and conduction. When a student is sick Supervision relevant protocol and fills a patient (see Annex)

Practical skills

- Be able to carry out surveys, physical examination of the patient with arrhythmia
- Be able to analyze the data of laboratory inspection
- Be able to assign basic therapy in patients with arrhythmia

Emergency Conditions

- Be able to assist with paroxysmal rhythm disorders

Cardiac arrhythmia - disturbances of frequency, a regularity and sequence of muscle contraction of the heart. Arrhythmias are a result of change of the basic functions of heart: automatism, excitability and conductivity. They develop at disturbance of formation of potential duration of a cell and change of speed of his conducting as a result of change of sodium, potassium and calcium channels.

The Basic Etiological Causes of Heart Rhythm Disorders

I. The functional causes (at healthy heart):
   a Psychogenic (corticovisceral);
   b Reflex (viscerocardial);
   c At exercises (heart of the sportsman).
II. Organic:
   a myocardial ischemia (Ischemic heart disease);

b hemodynamic (the congenital and got valve diseases, mitral valve prolapse, hypertension, acute and chronic cor pulmonale, heart failure, shock);
c infective - toxic (diffusive disease of a connective tissue of immune genesis); viral and bacterial infective lesion of a myocardium; nonspecific toxoallergic myocarditis);
d the pathological located changes in a myocardium (aneurysms, cicatrixes, tumours, sarcoidosis of myocardium);
e myocardial dystrophies (alcoholic, peripartum, uraemic, anemic, at disease endocrine glands);
f neuromuscular diseases (muscular dystrophy, etc.);
g primary cardiomyopathy (idiopathic dilated, hypertrophic, restrictive).

III. Toxic (medical drugs: sympathomimetic agent, cardiac glycoside, caffeine,nystatin, some antiarrhythmic agents, *corticosteroids).*

IV. Hormonal factors and diseases (a thyroid gland, pregnancy, menstrual disturbances, the period of dishormonal disturbances).

V. Electrolytic disturbances (K, Na, P).

VI. Mechanical (during operative interventions on heart, bodies of a thorax, invasive procedures).

The mechanism of occurrence of an arrhythmias:

1. Disturbance of formation of an impulse – abnormalities automatism and triggered activity (early and late afterdepolarizations).
2. Reentry circuit (an electrical current is caught in a reentrant loop, excessively stimulating the heart).
3. Disturbance of conductivity of an impulse.
4. Combinations of these disturbances.

REENTRY. Disorders of impulse propagation (reentry) are generally considered to be the most common mechanism of sustained paroxysmal tachyarrhythmia. The requirements for initiating reentry include (1) electrophysiologic inhomogeneity (i.e., differences in conduction and/or refractoriness) in two or more regions of the heart connected with each other to form a potentially closed loop; (2) unidirectional block in one pathway; (3) slow conduction over an alternative pathway, allowing time for the initially blocked pathway to recover excitability; and (4) reexcitation of the initially blocked pathway to complete a loop of activation. Repetitive circulation of the impulse over this loop can produce a sustained tachyarrhythmia. While anatomic obstacles may underlie reentry and provide an inexcitable center around which the impulse can circulate, they are not essential. Reentrant arrhythmias can be reproducibly initiated and terminated by premature complexes and rapid stimulation. The response of these arrhythmias to stimulation can help distinguish them from arrhythmias caused by triggered activity.

ENHANCED AUTOMATICITY. Disorders of impulse formation can be subdivided into tachyarrhythmias caused by enhanced automaticity and those caused by triggered activity. In addition to the sinus node, automatic pacemaker activity can be observed in specialized atrial fibers, fibers of the atrioventricular junction, and Purkinje fibers. Myocardial cells do not normally possess pacemaker activity. Enhancement of normal automaticity in latent pacemaker fibers or the development of abnormal automaticity due to partial depolarization of the resting membrane occurs as a consequence of a variety of pathophysiologic states, which include (1) increased endogenous or exogenous catecholamines, (2) electrolyte disturbances (e.g., hypokalemia), (3) hypoxia or ischemia, (4) mechanical effects (e.g., stretch), and (5) drugs (e.g., digitalis). Tachycardia caused by automaticity cannot be started or stopped by pacing.

TRIGGERED ACTIVITY. Rhythms due to triggered activity are events that do not occur spontaneously but require a change in cardiac electrical frequency as a trigger. Triggered activity may be caused by early afterdepolarizations, which occur during phases 2 and 3 of the action potential, or delayed afterdepolarizations, which occur following completion of phase 3 of the action potential. Triggered activity has been observed in atrial, ventricular, and His-Purkinje tissue under conditions such as increased local catecholamine concentration, hypercalcemia, and digitalis intoxication (delayed afterdepolarizations) or during bradycardia, hypokalemia, or other situations prolonging action potential duration (early afterdepolarizations). All of these conditions produce an accumulation of intracellular calcium. With increasing amplitude of the afterdepolarizations, threshold can be reached and repetitive activity produced. The exact role of triggered activity in spontaneous clinical arrhythmias is unknown, but tachyarrhythmias associated with digitalis intoxication, accelerated idioventricular rhythm in acute infarction and/or reperfusion, and exercise-induced ventricular tachycardia (VT) are believed to be caused by triggered activity due to delayed afterdepolarizations.

CLASSIFICATION OF CARDIAC ARRHYTHMIA AND CONDUCTIVITY

I. Disturbance of formation of an impulse

A. Disturbance of automatism of sinus node:

1. Sinus tachycardia
2. Sinus bradycardia
3. Sinus arrhythmia

Sick sinus syndrome

B. Ectopic (heterotopic) rhythms caused by prevalence of automatism of the ectopic centers.

1. Slow (alternated) slipping out complexes and rhythms:

a) atrial;

b) AV junctional;

C) ventricular.

2. Migration of the supraventricular driver of a rhythm.
3. Accelerated ectopic rhythms (non-paroxysmal tachycardias):

a) atrial;

b) AV junctional;
C) ventricular.

V. Ectopic (heterotopic) rhythms, mainly not connected to disturbances of automatism (the re-entry mechanism, etc.):

1. Premature complexes (atrial, AV junctional, ventricular).
2. Paroxysmal tachycardia (atrial, AV junctional, ventricular).
3. Atrial flutter
4. Atrial fibrillation
5. Ventricular flutter and fibrillation

II. Disturbances of conductivity:

1. Sinuatrial block.
2. Introatrial block.
3. Atrioventricular block (I, II, III degrees).
4. Intraventricular blocks (branches block of bundle of His):

a) One branch;
b) Two branches;
C) Three branches.

5. Asystole of ventricles.
6. Preexcitation syndrome

a) Wolff-Parkinson-White syndrome (WPW);
b) short P-Q syndrome (LGL).

III. The combined heart rhythm disorders

1. Parasystole
2. Ectopic rhythms with block of an output.
3. AV dissociation

ROUGH CARD of WORK of STUDENTS (Stages of diagnostic search and a choice of treatment)

I stage. To assume presence of heart rhythm disorders on the basis of the analysis of complaints and the anamnesis.

1. Complaints on: palpitation, feeling "skipped beats", thumping or fluttering in the chest, sensation of the heart racing, feeling faint or tired, light headedness or passing out (syncope), shortness of breath, chest pain or discomfort. Occurrence or progressing of heart failure at arrhythmias isprognostically adverse.
2. The anamnesis: the first occurrence of unpleasant sensations in the field of heart and the phenomena accompanying them; diagnosing (if it was carried out) objective infringements on the part of cardiovascular system and other organs and systems which could cause development of infringements of an cardiac rhythm; treatment carried out earlier and his efficiency; changes of development of symptoms; risk factors - alcohol, smoking, stresses, physical loading, etc.

II stage. With the help of methods of physical examination to find out: increase or reduction of a pulse rate and frequency of heart rate, deficiency of pulse, a rhythm of gallop. Objective acknowledgement of disease on which background there was heart rhythm disorder arises.

III stage. Drawing up of the program of examination for diagnosing an arrhythmias.

1. Electrocardiogram in rest.
2. Holter monitoring.
3. Transtelephonic monitoring.
4. Super-high resolution electrocardiograph
5. Using of vagul tests.
6. Using of pharmacological tests (with atropine, propranololi, isadrine, ajmaline).
7. Treadmill testing
8. Definition of a vegetative tone.
9. Definition of the biochemical status.
10. Esophageal electrophysiologic procedure.
11. Intracardiac electrophysiologic procedure (record electrical signals from the normal electrical system and the heart is stimulated).
12. Electrophysiologic testing and mapping
13. Tilt table studies

CRITERIA of ARRHYTHMIAS

Sinus tachycardia. Increase of heart rate owing to strengthening of automatism pacemaker structures of sinoatrial node. ECG: rate is greater than 100 (maximal heart rate (beats/min) = 220 - age), rhythm - regular, P waves - upright, consistent, and normal in morphology (if no atrial disease), P-R interval - between 0.12-0.20 seconds and shortens with increasing heart rate, QRS complex - less than 0.12 seconds, consistent, and normal in morphology.

Sinus bradycardia is defined as a resting heart rate of under 60 beats per minute, though it is seldom symptomatic until the rate drops below 50 beat/min. The mechanism - depression in automatism of sinoatrial node. ECG: heart rate is from 59 up to 40 though at the trained sportsmen it happens smaller 40 per minute, P wave is sinoatrial an origin, lengthening of P-Q interval (up to 0,20-0,21 seconds).

Sinus arrhythmia is wrong sinus rhythm with the periods of retardation and acceleration of a heart rhythm owing to change of function of automatism of sinoatrial node.

Ⅰ - respiratory

Ⅱ - not-respiratory

a) periodic - gradual retardation or acceleration of heart rate irrespective of breath;

b) not-periodic - retardation or acceleration of heart rate passes non-uniformly, without their gradual transitions and irrespective of breath.

ECG: P wave is sinoatrial an origin, P-Q interval is constant and normal length (0,11-0,21 seconds), heart rate is 45-100 per minute, a wrong rhythm with a difference between the longest and shortest P-P (R-R) interval 0,16 seconds and more.

Premature complexes (PC). Premature in relation to the basic rhythm excitation of all heart or its departments. The mechanisms exist for PC: reentry circuit or triggered activity. Depending on a source it can be supraventricular (sinoatrial, atrial, AV junctional) and ventricular.

Premature supraventricular contractions:

1. **Premature sinoatrial contraction** is a type of premature heart beat which start in the sinus node. An electrocardiogram: premature the P waves is same forms as the P waves of sinus origins, complex QRS is not changed. Absence of compensatory pause.
2. **Premature atrial contraction** is a type of premature heart beat which start in the atria. An electrocardiogram: an abnormally shaped P wave that is premature, and may be "buried" or superimposed on the preceding T wave. The QRS complex that follows the abnormal P wave may be premature and slightly narrowed. Occasionally after very early premature atrial contractions, QRS configuration is abnormal, due to refractoriness of part of the normal conduction pathway. Usually absence of full compensatory pause.
3. **Premature AV junctional contraction** occur due to abnormal impulse formation at or near the AV junction. The impulse may spread both retrograde and antegrade from its source. On electrocardiogram: P waves are typically early and may have an abnormal configuration (negative P waves may be seen in lead II). One may observe a prolonged or shortened P-R interval depending on the distance from the origin of the impulse to the AV node. If the ectopic beat originates in high nodal tissue, the QRS complex can be narrow. Compensatory pause can be full or incomplete.
4. **Ventricular premature complexes (VPCs)**

   Ventricular premature complexes are ectopic impulses originating from an area distal to the His Purkinje system. ECG: wide (duration exceeding the dominant QRS complexes) and bizarre QRS complexes are present. No preceding premature P waves occur, and, rarely, a sinus P wave is conducted. The T wave usually is in the opposite direction from the R wave. Full compensatory pause is common. VPCs originating from the left ventricle typically produce a right bundle-branch block (BBB) pattern on QRS. VPCs originating from right ventricle typically produce left BBB-like pattern on QRS.

   VPCs can be classified in different ways.

The Lown classification (1971) was introduced to gauge effects of antiarrhythmic drugs.

Class	Arrhythmia
0	None
I	Unifocal; <30/h
II	Unifocal; > 30/h
III	Multiform
IV A	2 consecutive
IV B	>3 consecutive
V	R-on-T phenomenon

Clinical classification is as follows:

- Benign
- Potentially malignant
- Malignant

Classification according to frequency is as follows:

- Frequent - 10 or more VPCs per hour (by Holter monitoring) or 6 or more per minute
- Occasional - Fewer than 10 VPCs per hour or fewer than 6 per minute

Classification according to relationship to normal beats is as follows:

- Bigeminy - Paired complexes, VPC alternating with a normal beat
- Trigeminy - VPC occurring every third beat (2 sinus beats followed by VPC)
- Quadrigeminy - VPC occurring every fourth beat (VPC following 3 normal beats)
- Couplet - 2 consecutive VPCs
- Nonsustained VT - 3 or more consecutive VPCs (<30 s)

Classification according to origin is as follows:

- Number of foci

a. Unifocal/unimorphic - Beats originate from 1 focus, ie, all VPCs have the same morphology

b. Multifocal/multimorphic - VPCs have more than 1 morphology and may originate from more than 1 site

- Site of origin

a Left ventricular

b Right ventricular

- Associated heart disease

a None (idiopathic)

b Structural heart disease present

Paroxysmal tachycardia is attacks of palpitation with heart rate 140-240 bpm per minute and a correct rhythm. At paroxysmal tachycardia it is registered not less than three consecutive ectopic impulses. Depending on a source can be supraventricular (sinoatrial, atrial, from AV junctional, at accessory pathway for conduction), ventricular, and on the mechanism of occurrence - reciprocal, focal, trigger.

Supraventricular tachycardia (SVT), a common clinical condition, is any tachyarrhythmia that requires only atrial and/or atrioventricular (AV) nodal tissue for its initiation and maintenance.

Sinus nodal reentrant tachycardia is due to a reentry circuit, either in or near the sinus node. Therefore, it has an abrupt onset and offset. The heart rate is usually 100-150 bpm, and ECG tracings usually demonstrate normal sinus P-wave morphology .

Atrial tachycardia is an arrhythmia originating **in** the atrial myocardium. Enhanced automaticity, triggered activity, or reentry may result in this rare tachycardia. The heart rate is regular and is usually 120-250 bpm. The P-wave morphology is different from the sinus P waves and is dependent on the site of origin of the tachycardia. Because the arrhythmia does not involve the AV node, nodal blocking agents such as adenosine and verapamil are usually unsuccessful in terminating this arrhythmia.

Atrioventricular nodal tachycardia is the most common type of reentrant supraventricular tachycardia. An ECG: Evaluation usually reveals a supraventricular origin of QRS complexes at rates of 150-250 bpm and a regular rhythm. The QRS complex usually narrows unless a conduction abnormality is present or is functionally induced from the rapid heart rate. A P wave may be absent, negative, or buried in the following QRS complex. A pseudo R prime may be seen in V-,, or pseudo S waves may be seen in leads II, III, or aVF. The onset is abrupt with an atrial premature complex, which conducts with a prolonged PR interval. The PR interval may shorten over the first few beats at onset, or it may lengthen during last few beats preceding termination of the tachycardia. Abrupt termination occurs with **a** retrograde P wave, sometimes followed by a brief period of asystole or bradycardia.

Ventricular tachycardia is a general term that includes any rapid rhythm, faster than 100-120 beats per minute, arising in the ventricle. It is caused by a reentry. An ECG: QRS complexes (duration greater than 120 milliseconds) are usually aberrant in shape and P waves are not usually present. A P wave may be present if retrograde conduction occurs and may cause a capture beat (if the ventricles are not refractory), ventriculoatrial dissociation. When the ventricular activation sequence is constant, the electrocardiographic pattern remains the same, and the rhythm is called monomorphic ventricular tachycardia. Alternatively, polymorphic ventricular tachycardia occurs when the ventricular activation sequence varies.

Polymorphous ventricular tachycardia (Torsade de pointes). It is important to recognise this pattern as there are a number of reversible causes: heart block, hypokalemia or hypomagnesaemia, drugs (e.g. tricyclic antidepressant overdose), congenital long QT syndromes, other causes of long QT (e.g. IHD). This is a form of VT where there is usually no difficulty in recognising its ventricular origin. ECG: wide QRS complexes with multiple morphologies, changing R - R intervals, the axis seems to twist about the isoelectric line.

Atrial fibrillation is an abnormal heart rhythm which involves the two the atria. Atrial fibrillation is caused by an abnormalities automaticity and reentry circuit. On an electrocardiogram: discrete P waves are absent; instead, undulating fibrillatory (f) waves are present. The ventricular rate typically is irregular (irregularity of R-R interval), complex QRS is constant; the amplitude of R waves can be different.

Atrial flutter is a regular, rhythmic tachycardia originating in the atria. The rate in the atria is over 220 beats/minute, and typically about 300 beats/minute. In the case of atrial flutter, there is a very particular block pattern at the AV node level. In atrial flutter, the AV node typically will block every other electrical impulse, or three out of four impulses. Atrial flutter is caused by a reentrant rhythm in either the right or left atrium. On an electrocardiogram: the common form of type I atrial flutter has sawtooth flutter (F) waves, best seen in leads II, III, and aVF, with atrial rates of 240-340 bpm and without an isoelectric interval between these F waves. The ventricular response may be regular or irregular. The ventricular rate is a fixed mathematical relationship of F waves and the resulting QRS complexes. Variable AV conduction can also be seen (commonly present with 2:1 or 3:1 AV conduction).

Clinical forms of atrial flutter and fibrillation:

1. Paroxysmal atrial fibrillation (atrial flutter) is when terminates spontaneously within 7 days, most commonly within 24 hours.
2. Persistent atrial fibrillation (atrial flutter) is AF established for more than seven days (irrespective of whether AF is terminated with pharmacologic therapy or electrical cardioversion).
3. Permanent atrial fibrillation (atrial flutter) - any patients with long-standing AF in whom cardioversion has not been indicated or attempted.

Ventricular fibrillation is a cardiac condition that consists of a lack of coordination of the contraction of the muscle tissue of the large chambers of the heart that eventually leads to the heart stopping altogether. The mechanisms of development are re-entry, triggered activity and automaticity. On an electrocardiogram: frequency (from 200-500 1 minute), bizarre, irregular, random waveform , the different form and amplitude that no clearly identifiable QRS complexes or P waves wandering baseline.

Ventricular flutter is a tacharrhythmia characterized by a high ventricular rate with a regular rhythm. An electrocardiogram: shows large sine wave-like complexes that oscillate in a regular pattern with often (150-300 1 minute). P waves are not present, and the QRS complex is indistinguishable from the T wave. Ventricular flutter, if untreated, may precede ventricular fibrillation.

IV stage. A justification of the diagnosis and his detailed elaboration.

On the basis of complaints on palpitation which is accompanied by weakness, dizziness, **a** short wind, polyuria, chest pain, and also faults, pauses in work of heart, faints, edemas; the anamnesis (the data on the transferred acute rheumatic fever, myocarditis, ischemic heart disease. Sometimes communication of arrhythmia with any disease will establish is impossible; factors which promote arrhythmia - alcohol, smoking, stress, overwork), the data of physical examination (abaissement of pulse wave, premature contractions, tachycardia, bradycardia, increased or diminished loudness of sounds of heart, enlargement of the heart), the data of imaging studies which confirm presence at the patient of arrhythmia. The DIAGNOSIS is formulated proceeding from the basic disease, and at the end of the diagnosis specify heart rhythm disorders.

V stage. Complications.

Ventricular fibrillation at ventricular premature complexes and paroxysmal tachycardias, heart failure, thromboembolisms.

VI stage. The differential diagnosis.

THE DIFFERENTIAL DIAGNOSIS OF ATRIAL FIBRILLATION AND PREMATURE COMPLEXES

Signs	Atrial fibrillation	Often multifocal couplet extrasystoles
Causes	Ischemic heart disease, myocardial infarction, atherosclerosis, mitral stenosis, hyperthyroidism, endomyocarditis, cardiomyopathy, pericarditis.	Rheumatic carditis, ischemic heart disease, heart diseases, reflex genesis at extracardial pathologies, functional disturbances.
Pulse	Inordinate, chaotic alternation of pulse waves of various sizes through equal intervals can be deficiency of pulse.	There is a law: after the short interval the pulse wave is weaker usual, the compensatory pause with the subsequent increase of pulse wave further follows.
Auscultation	Absence of consecutive rhythmical heart rate, unequal diastolic pauses, and sounds of unequal sonority at each heart reduction.	Between usual extrasystoles the group of several consecutive rhythmical heart reductions with identical pauses is found out. Long pauses follow premature complexes. On a background extrasystoles can be loud and split second heart sound.
Reaction to exercises	Significant strengthening of arrhythmia	Can disappear or become more infrequent at functional extrasystoles
Venous pulse	Absence of a "a" wave which reflect atrial system on phlebogram.	There can be a positive pulsation of veins that coincides with premature complexes.
ECG	See above	See above

VII stage. A formulation of the clinical diagnosis. For example:

- Ischemic heart disease: acute anterior wall transmural myocardial infarction of left ventricle (15.08.2004). Multifocal ventricular premature complexes, attacks of paroxysmal ventricular tachycardia, ventricular fibrillation, condition after reanimation actions. HF II A st., systolic dysfunction, Class III.
- Dilated cardiomyopathy, permanent atrial fibrillation. HF II B st., systolic dysfunction, Class IV.
- Hypertrophic cardiomyopathy, the obstructive form. Paroxysm of supraventricular tachycardia (12.11.2004). HF II A st., diastolic dysfunction, Class III

VIII stage. To administer correct individual treatment.

Regimen I, II, III

Diet N° 10

Methods of treatment of arrhythmias:

1. Exception of provoking factors;
2. Treatment of the basic disease;
3. Sedative therapy, psychotherapy;
4. Antiarrhytmic drugs;

5. Not medicamental treatment of arrhythmias.

Classification of *antiarrhythmics* drugs (on *Vaughan* Williams).

Class I agents interfere with the sodium (Na+) channel.

Class la agent increasing action potential duration (quinidine, procainamide and disopyramide).

Class Ib agents shorten the action potential duration and reduce refractoriness (lidocaine, mexiletine, tocainide, phenytoin).

Class Ic agents decrease conductivity, but have a minimal effect on the action potential duration (encainide, flecainide, moricizine, propafenone).

Class II agents are anti-sympathetic nervous system agents. All agents in this class are beta-blockers.

I Group - nonselective beta-adrenergic receptor blockers:

Ⅱ Group - selective beta-adrenergic receptor blockers (acebutolol, atenolol, metoprolol).

Ⅲ Group - nonselective beta-adrenergic receptor blockers with arreceptor antagonism (carvedilol, celiprolol, labetolol).

Class III agents affect potassium (K+) efflux (amiodarone, bretylium, sotalol, ibutilide).

Class IV agents are slow calcium channel blockers. They decrease conduction through the AV node (verapamil, diltiazem).

Class V agents work by other or unknown mechanisms (adenozine, atropine, digoxin).

Not medicamental treatment of arrhythmias:

1. Defibrillation / Cardioversion (external and Internal).
2. Cardiac pacing (temporary and permanent; ventricular-based pacing (Wl mode), atrial-based pacing (AAI, DDD modes); frequency - adaptive and is not- adaptive; one and bipolar).
3. Implantable cardioverter-defibrillator (ICD) (ventricular or atrial).
4. Radiofrequency catheter ablation (intervention destruction of various conducting structures of heart: AV-node, accessory pathway, channels of AV-node, a loop re-entry, the focus of a tachycardia).
5. Cardiothoracic surgery. Application for treatment of infringements of a rhythm of operations open-heart surgical procedure, is justified only at presence of other pathology demanding similar intervention (an aneurysm left ventricle, critical defect of valves of heart, etc.).

Sinus tachycardia.

1. To remove the cause of ascending of heart rate (thyrotoxicosis, anemia, heart failure, fever, hypovolemia, psychogenic factors).
2. Exception of smoking, alcohol, abusing of coffee, tea, spices.
3. Sedative drugs (Corvalolum, Tinctura Leonuri, Tinctura Crataegi), small tranquilizers, beta-adrenergic blockers (atenolol, metoprolol, bisoprolol) at neurogenic form of a tachycardia;
4. Medicamental therapy at patients without heart failure: atenolol, verapamil, amiodarone.
5. Medicamental therapy at patients with heart failure: digoxin, digoxin in a combination with beta-adrenergic blockers or calcium channel blockers, or amiodarone.

Sinus bradycardia

1. Treatment of the basic disease.
2. Treatment of sinus bradycardia is carried out in those cases if it causes an angina pectoris, arterial hypotension, syncope, heart failure, and ventricular arrhythmias.
3. Use of drugs that increase heart rate: drugs of belladonna (Belladonae extract, Guttae Zelenini, Bellaspon), atropine, isoproterenol hydrochloride, adrenaline, nifedipine, and various combinations of these drugs.
4. Implantable of cardiostimulator.

Sinus arrhythmia

At people of young age at presence respiratory arrhythmia medicamental therapy is not shown. Other kinds of sinus arrhythmia if they do not lead to infringement of blood circulation, also in treatment do not require. Is subject to supervision.

Premature complexes

To obligatory treatment are subject:

1. ventricular premature complexes Ill-V class;
2. allorhythmia;
3. often premature supraventricular contractions (more than 5 per minute);
4. occurrence or increase in amount extrasystoles during an attack of a stenocardia, at **a** myocardial infarction;
5. ventricular premature complexes after an attack of paroxysmal ventricular tachycardias or ventricular fibrillation;
6. extrasystoles which is accompanied by unpleasant subjective sensations;
7. extrasystoles at diseases which promote occurrence antiarrhytmic-induced arrhythmias (mitral valve prolapse, long QT syndrome, Wolff-Parkinson-White syndrome).

Premature supraventricular contractions

Monotherapy of calcium channel blockers (verapamil, diltiazem), not-selective and selective beta 1-adrenergic receptor blockers (propranolol, metoprolol, bisoprolol), agents affect potassium (K^+) efflux (amiodarone, sotalol), class la agents (ajmaline, procainamide, disopyramide) and class Ic antiarrhythmic agents (flecainide, propafenone), in average therapeutic doses.

Ventricular premature complexes (VPCs)

1 .Absence of significant structural heart disease (eg, normal ventricular function, no coronary or valvular heart disease):

- Asymptomatic VPCs require no therapy.
- For symptomatic VPCs, recommended treatment usually involves patient education and reassurance,

avoidance of aggravating factors (eg, stress, caffeine-containing products), and anxiolytic drugs if education and avoidance of aggravating factors are ineffective. Beta-blockers and nondihydropyridine calcium channel blockers (eg, verapamil, diltiazem) can be used to treat symptomatic patients. Beta-blockers with intrinsic sympathomimetic activity may be particularly helpful. The use of antiarrhythmic therapy is not generally recommended and is only used to prevent symptoms. The risk of the drug (including the risk of arrhythmic death from proarrhythmia) must be weighed against the benefits of VPC suppression. In patients who are symptomatic on Beta-blockers and/or calcium channel blockers, consider cautious use of Amiodarone.

- 2. Presence of underlying heart disease (eg, VPCs in patients post-MI)
- Various strategies, both invasive and noninvasive, predict prognosis in patients with VPCs post-MI.
- The most powerful combination of noninvasive prognostic variables that identify patients in whom invasive strategies are suitable includes the presence of 2 or more of the following variables, (1) LV EF less than 0.40, (2) ventricular late potentials (on signal-averaged ECG), and (3) repetitive VPCs.

Class IA drugs (eg, procainamide, quinidine, disopyramide) are moderately effective but have proarrhythmic effects. Class IB drugs (eg, mexiletine) may have less proarrhythmic effect (although one post-MI trial showed higher mortality for mexiletine than placebo) than class I antiarrhythmic drugs. These drugs may show reasonable efficacy in some patients. Class IC drugs (eg, flecainide, propafenone) are effective for reducing ventricular ectopy and are relatively well tolerated in patients with normal or minimally reduced LV function and no ischemic heart disease. They are not recommended in patients with ischemic heart disease. Class II drugs (beta-blockers) are the drugs of choice in patients who are symptomatic but do not have structural heart disease. Also, class II drugs are considered the first choice of therapy for patients with underlying heart disease, even if their EF is reduced. Class III drugs (eg, amiodarone, sotalol) are approved for use only in life-threatening arrhythmia. Recent data suggest that amiodarone is safe post-MI for patients with VPCs, even though they do not reduce the risk of death. Class IV drugs (calcium channel blockers), in general, have no role in the treatment of VPCs. However, occasionally these drugs may suppress triggered automaticity or idiopathic VPCs. Currently, no evidence supports treatment of asymptomatic VPCs after Ml with medication other than beta-blockers. Clinical trials have suggested that type I antiarrhythmic agents and racemic sotalol increase mortality in patients post-MI. Amiodarone may have no adverse effect on mortality in this setting.

Patients deemed to be at high risk of sudden cardiac death may benefit from implantable cardioverter defibrillator (ICD) implantation.

Paroxysmal tachycardias. The choice of a method of treatment depends on a condition of hemodynamic. At unstable hemodynamic is shown synchronized cardioversion.

Signs of hypoperfusion (unstable): an attack of a stenocardia, breathlessness, arterial hypotension (the blood pressure is less than 90 mm Hg), cardiac asthma, pulmonary edema, cardiogenic shock.

Supraventricular tachycardia

I. Unstable:

1. Oxygen.
2. Valium (Diazepamum) 2,5mg - 5mg slow IVP (or Thiopentalum-natrium up to achievement of **a** subnarcotic condition).
3. Synchronized cardioversion (100/200/300/360 joules).

II. Stable:

1. Increasing vagul tone (such as coratid sinus massage, valsalva maneuver or induced retching).
2. Adenosine 1 % -2,0 ml IV over 5-10 seconds.
3. At inefficiency - isoptine 0,25 % - 2-4 ml IV over 2-3 minutes, at an inefficiency introduction can be repeated of 5 mg through 30 minutes (except for WPW syndrome with wide complexes), then IV drip.
4. Or procainamide 10 ml IV.
5. Or amiodarone 150-300 mg IVP, then IV drip (a daily doze up to 1200 mg).
6. Synchronized cardioversion carry out the patient with a heavy attack that does not give in to medicamental treatment or often relapses.
7. It is possible to usealso ajmaline, aethacizine, propafenone, digoxin, dizopyramide.

Ventricular tachycardia

I. Unstable:

1. Oxygen.
2. Valium (Diazepamum) 2,5mg - 5mg slow IVP (or Thiopentalum-natrium up to achievement of **a** subnarcotic condition).
3. Synchronized cardioversion (100/200/300/360 joules).
4. At relapse of ventricular tachycardias - lidocaine 1mg/kg IVP, synchronized cardioversion with rank which was effective, then - novocainamidum IVP.

II. Stable:

1. Oxygen.
2. Lidocaine 70-150 mg bolus. If necessary the doze can be increased, but no more than 4-5 mg / kg over 15-20 minutes.
3. At inefficiency - synchronized cardioversion (100/200/300/360 joules).

It is possible to use also amiodarone, novocainamide, ajmaline, aethacizine, propafenone, mexiletine.

Ventricular fibrillation and flutter

Treatment - carrying out cardiopulmonary reanimations, at success - actions *on* prevention of repeated occurrence of ventricular fibrillation_and sudden death.

Atrial fibrillation and atrial flutter I. Paroxysmal atrial fibrillation (atrial flutter)

1. At stable hemodynamic treatment does not demand, at unstable hemodynamic - electrical cardioversion.
2. At presence of risk factors of thromboembolism - anticoagulant treatment (warfarin, aspirin, clopidogrel).

II. Persistent atrial fibrillation (atrial flutter):

1. Conversion to sinus rhythm:

-at unstable hemodynamic may influence the decision to cardiovert immediately (electrical cardioversion), -at stable hemodynamic:

A) If the arrhythmia is present for less than 48 hours, cardioversion can be accomplished safely without further need for anticoagulation. If uncertain, a transesophageal echocardiogram could be performed to exclude left atrial thrombus. In any case, acute conversion must be accompanied by anticoagulation.

B) In patients with AF longer than 48 hours in duration *or* of unknown duration, cardioversion is not recommended until sufficient anticoagulation is achieved. The most conservative route is to anticoagulate with warfarin for 3-4 weeks prior to any attempt to restore sinus rhythm. Because embolic events can occur following cardioversion as atrial mechanical function returns, continue anticoagulation for an additional 4-6 weeks. Alternatively, initially perform a TEE, and if *no* thrombus is present, cardiovert and therapeutically anticoagulate for 4-6 weeks after sinus rhythm is restored

-chemical cardioversion: class la agent (quinidine, procainamide and disopyramide), class Ic agents (flecainide, propafenone), class **III** agents (amiodarone, sotalol).

-electrical cardioversion: atrial defibrillators.

2.Preventive maintenance of relapses of atrial fibrillation (atrial flutter):

- medicamental: class la agent (quinidine, procainamide and disopyramide), class Ic agents (flecainide, propafenone), class

III agents (amiodarone, sotalol).

- not-medicamental: AV nodal ablation or updating, atrial-based pacing (AAI, DDD modes) (biatrial pacing and dual-site atrial pacing), surgical treatment.

III. Permanent atrial fibrillation.

1.Control of heart rate (in rest of 60-80 bpm per minute, at moderate physical loading of 90-115 bpm per minute):

- medicamental: cardiac glycoside (digoxin), calcium channel blockers (verapamil, diltiazem), beta-adrenergic blocking agents (metoprolol, atenolol, propranolol), other drugs (propafenone, amiodarone, sotalol).
- not-medicamental: radiofrequency ablation (destroy abnormal electrical pathways in heart tissue), AV nodal ablation and to implant a pacemaker instead, surgical procedures: "traditional maze" (an open-heart surgical procedure), minimaze surgical procedures (these procedures use microwave, radiofrequency, or acoustic energy to ablate atrial tissue near the pulmonary veins).

2.Preventive maintenance of thromboembolisms - long-term anticoagulation: warfarin, aspirin, clopidogrel.

Treatment of AF patients over age 60, who also have one or more of: previous strokes (or warning strokes), hypertension, diabetes, or congestive heart failure, with warfarin who have any structural heart disease (i.e. valvular heart disease, ejection fraction <= 35%, history of heart attack) may also benefit from warfarin.

Patients under age 65 who do not have structural heart disease do not require warfarin, and can be treated with aspirin or clopidogrel. There is evidence that aspirin and clopidogrel are effective when used together, but the combination is still inferior to warfarin.

Physiotherapy

Difficult heart rhythm disorders are contraindication for purpose of physiotherapeutic procedures and directions on sanatorium-health-resort treatment and carrying of medical physical training out.

IX stage. The prognosis and prevention.

Dangerous to life arrhythmias: group supraventricular and ventricular extrasystoles, paroxysmal supraventricular and ventricular tachycardia, atrial and ventricular fibrillation, atrial and ventricular flutter. Such patients determine usually invalids II, III groups. Accompanying diseases, the basic disease and complications determine the forecast for a life and work capacity.

Secondary prevention maintenance will consist from treatment of the basic disease, the complications, accompanying diseases, elimination of risk factors of arrhythmias (smoking, the use of alcohol, etc.).

Independent work

1. Study of the special literature:

Cardiac Pacing and Cardiac Resynchronization Therapy // European Heart Journal doi:10.1093/eurheartj/eht150

Atrial Fibrillation (Management of) 2010 and Focused Update (2012) // European Heart Journal (2012) 33, 2719–2747

ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death // Europace (2006) 8 ,746–837

- Mayo Clinic Cardiology – third edition (2007)
- Valentine Fuster – The HEART 11-edition (2007)
- Braunwald – Heart Diseases 8th edition (2008)
- Ragavendra R. Baliga - Practical Cardiology Evaluation and Treatment of Common Cardiovascular Disorders, 2nd Edition (2008)
- Swanton R.H. – Swanton's cardiology – 6th edition (2008)
- Mohammad Shenasa – Cardiac Mapping Third Edition (2009)

Preparing of summary due to the topic of the lesson by the content of magazines:

- American College of Cardiologists Journal
- American Heart Journal
- American Journal of Cardiology
- American Journal of Hypertension
- British Heart Journal
- The Canadian Journal of Cardiology
- Cardiology
- Circulation
- European Heart Journal
- Heart
- Hypertension
- International Journal of Cardiology

Decision of tests and situatioonal tasks of KROK 2.
Writing of protocols of clinical analysis of patients.

Tests initial level of knowledge

1. Patient 50 years old, was admitted to clinic at 14 o'clock with complaining of chest pain and palpitations .Her past medical history show : the disease began acutely at 12 a.m., She had . tightness, chest pain, palpitations. After the doctor's emergency aid slightly decreased, but the situation is not improved. Patient was hospitalized. On examination: the state of the patient was heavy, pale skin, cold arms in the lung vesicular breathing. Cardiac sound is quite, rhythmic, pulse 186 beat per min.., blood pressure 85/60 mmHg ECG: P is absent, f-wave, R-R intervals are different. What diagnosis is right?.

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation, paroxysmal form
E. Atrial fibrillation, persistent form

2. The patient in '59 with acute myocardial infarction (first day) developed heart attack, accompanied by severe weakness, low blood pressure. The ECG: P is not defined, QRS - 0.12sec, abberant , the number of ventricular rate- 150 beat per minute. What is the diagnosis in this patient?

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation, paroxysmal form
E. Atrial fibrillation, persistent form

3. The patient, who is in the cardiology department at the CHD: progressive angina pectoris, was sudden deterioration: there was a pronounced chest pain sit up. At examination: inspiratory shortness of breath , orthopnea, agitated, distant wheezing. He has cough with a pink form sputum , 25 breathing per minute. He has cyanosis of face and neck . Pulse=100 beat per minute, arrhythmic, BP 180\110 mm Hg. The ECG - R prong missing, f-wave, R-R intervals are different, and ST depression (-) T in I, avL V4-V6. What complications developed in the patient?

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation, paroxysmal form
E. Atrial fibrillation, persistent form

4. In patients with acute myocardial infarction at 25 days of the disease suddenly appeared dizziness, sudden general weakness of the transition in syncope. Pulse and blood prassure in peripheral vessels were not defined. The ECG: RR - 0,30 with, heart rate-200 beats / min, P is not defined , deformed ventricular complexes, dilated, no baseline, QS-type ventricular complex in V6. What complications arose in this patient?

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation, paroxysmal form
E. Atrial fibrillation, persistent form

5. The ECG - R-wave recorded before QRS-complex, PP interval = RR = 0,35 seconds, the QRS complex is normal (<0.12sek) interval QT> 50% RR. Estimate the changes on the ECG.

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation, paroxysmal form
E. Atrial fibrillation, persistent form

6. The ECG show: QRS complex extended (> 0.12sek), P-wave are not determined , heart rate above 200 per minute. Estimate the changes on the ECG.

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias

D. Atrial fibrillation
E. Ventricular fibrillation

7. The ECG - no P waves, recorded waves f, ventricular rhythm is wrong, the QRS complex is normal (<0.12sek). Estimate the changes on the ECG.

A. Sinus tachycardia
B. Supraventricular paroxysmal arrhythmias
C. Paroxysm ventricular arrhythmias
D. Atrial fibrillation
E. Ventricular fibrillation

8. When sudden death occurred in patient outside of hospital chance of survival does not exceed 10 %. Therefore, great importance is its primary prevention. Purpose is to bring the drug class most effective in reducing the risk sudden death in patients with heart failure?

A. Antagonists of Ca
B. Diuretics
C. ß-blockers
D. Digoxine
E. Nitrates

9. In patients 70 years of atrial fibrillation after nervous strain was a sharp chest pain , shortness of breath 36 per min., pallor, acrocyanosis. He had dry whistling in the lungs. Second sound of the pulmonary vessel was loud .Rs – 100 beat per 1 min .,BP - 90/60 mmHg In blood: MB-CK fraction is normal. The ECG-overload right heart, elevation ST v1-v2. What caused the deterioration of the patient?

A. Asthma attacks
B. Rupture of the aortic aneurysm
C. Cardiogenic shock
D. Myocardial infarction
E. Pulmonary embolism

10. The patient 56 years old with CHD, heart failure II A periodically (2-3 times per week) have episode of atrial fibrillation, who disappear without treatments, accompanied by typical chest pain . At examination: good condition .Pulse-82 for 1 min., normal, BP -130/80 mm Hg What is the treatment most appropriate in this case to prevention attacks of arrhythmia?

A. Amiodarone
B. Quinidine.
C. Novocainamid.
D. Digoxine.
E. Rytmilen.

11. The patient 55 years old, with complains of palpitations, shortness of breath on exertion, oedema of the legs in the evening. Ill 2-3 years, the symptoms grew gradually. Arrhythmic pulse, 90 per min., The deficit - 20. BP - 130/70 mmHg Heart slightly enlarged in the left side, the heart sound is silent , irregular. Liver - + 2 cm, smooth. On ECG - atrial fibrillation, heart with ventricular rate - 110 per min. Chose the drug for treatment.

A. Propranolol.
B. Digoxine.
C. Enalapril
D. Furosemide.
E. Novocainamid.

12. The patient 28 years old, complains of palpitations, cardiac arrhythmia, fatigue, tearfulness, decreasing of the weight. She is ill 5-6 years, the symptoms grew gradually. Condition of the patient is not good, appals of yese extended, positive symptoms Gref. The thyroid gland Is increased to 3 degrees, smooth, painless, moving. Arrhythmic pulse, 95 min onedge smooth. The deficit - 20. BP - 140/90 mmHg Cardiac sound is normal , arrhythmic. Liver - 2 cm, The ECG: heart rate - 115 per minute. Atrial fibrillation. Select the drug for the treatment of arrhythmias.

A. Quinidine.
B. Novocainamid.
C. Propaphenon.
D. Digoxin.
E. Propranolol.

Correct answers: 1D, 2C, 3D, 4C, 5B, 6C, 7D, 8C, 9E, 10A, 11B, 12E.

Appendix 1

Protocol of patient clinical analysis

Name of patient _____
Clinical diagnosis
Main pathology
_____
_____
_____
Complications
_____
_____
Concomitant diseases
_____
_____
Complaints of patient
_____
_____
Anamnesis morbi _____
_____
_____
Physical examination data: _____
_____
_____
_____
_____
What diseases need perform differential diagnosis with?
1. _____
2. _____
3. _____

Plan of examination: _____
_____
_____
Treatment (concrete drug, dose, duration of usage, regimen, rules of drug cancel if it's need):
_____
_____
_____
_____
Recommendations for prophylaxis of relapses and exacerbations
_____
_____

URGENT CONDITIONS IN CARDIOLOGY

Textbook for 6th course students of medical university

Sent to the printer's 17.01.2015 Signed for printing 25.01.2015
Offset paper. Rizograph printing
Edition 300. Order № 2501
ZSMU (Zaporozhya State Medical University) Press
26 Mayakovskyi Street, Zaporizhya, 69035.