Ministry of Health of Belarus
Gomel State Medical University

Department of Orthopedic, Trauma and military field surgery
with the course of Anesthesiology and Critical Care Medicine

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SUBJECT: «Intensive care of diseases accompanied by acute circulatory disorders» (IT is complications of myocardial infarction, acute lung injury, Paroxysmal tachycardia, atrial fibrillation, extrasystolia, Syndrom Morgagni-Adams-Stokes equations. Hypertensive crisis. Thromboembolism in the pulmonary artery) "

Educational and methodical development for students
4th year medical faculty

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Methodical development is designed for self-study. It provides:

1. Background.
2. The purpose of training (skills and knowledge).
3. Basic sections.
4. Suggested Reading.
5. Questions for self-preparation.
6. Topics UIRS.
7. Teaching tools for organization of independent work of students
8. Training Material
9. Self-study.
10. Clinical problems and test control.

Relevance of the topic

The problem of cardiovascular disease is extremely important. Of all the causes of death from cardiovascular disease accounts for 53% of coronary heart disease (CHD). Sufficiently high mortality from myocardial infarction (MI). On the treatment and prevention of myocardial infarction by scientists all over the world due to the high social and economic significance of the nosology. Myocardial infarction has a high risk of life-threatening complications, the outcome of which is largely dependent on the timeliness and accuracy of providing patient care. Near and long-term prognosis of patients with MI may be significantly improved if the organization diagnostic and medical help meet modern standards. Acute pulmonary embolism is also one of the most common causes of sudden death. However, even a massive pulmonary embolism diagnosed during life only in 30% of patients. Thus problem diagnosis and intensive therapy of pulmonary embolism is very important. Relevance of the topic of hypertension and hypertensive crisis for health care due to the prevalence of disease and significant economic losses due to disability and mortality from complications that developed as a result of complications of hypertension and hypertensive emergencies. The practitioner must own methods of treatment and prevention of hypertension, hypertensive crises including risk stratification and prognosis of complications.

Purpose of the lesson Teach students the general principles of diagnosis and pathogenesis-based intensive therapy of critical states with the most common cardiovascular disease.

The student should know:

• etiology and pathogenesis of acute myocardial infarction and its complications
• clinical and diagnostic of myocardial infarction and its complications
• Principles and methods of intensive therapy of myocardial infarction and its complications
• methods of drug and electro-correction of cardiac arrhythmias
• pathophysiology, classification, clinic and diagnostics of pulmonary embolism (PE)
• Principles and methods of intensive therapy for pulmonary embolism
• the risk factors leading to the development of hypertensive crises, the classification of hypertensive crises
• major complications arising from hypertensive crisis
• main groups of drugs used in hypertensive crisis
• Principles of emergency treatment of complicated and uncomplicated hypertensive crisis

The student should be able to:

• assign differentiated, individualized treatment of patients with complicated and uncomplicated myocardial infarction, control the efficiency and safety of treatment
• select a remedy for the correction of cardiac arrhythmias
• Identify and provide evidence of the non-drug method of treatment of arrhythmias
• diagnose a hypertensive crisis and its complications
• justify the tactics and choose wisely drugs for relief of hypertensive crisis
• interpret the results of instrumental and laboratory investigation of suspected pulmonary embolism
• Identify patient management with pulmonary embolism
Sections studied before and needed for the session
• basic pharmacology of drugs
• Human Physiology
• physiopathology
• Internal Medicine
• surgical disease

Recommended Reading

Textbooks on pharmacology, normal and abnormal physiology, surgery, internal medicine for medical students.

Suggested Reading on lessons

Main Reading

1. Radiology for Anaesthesia and Intensive Care, Second Edition (Cambridge Medicine) Richard Hopkins, Carol Peden, Sanjay Gandhi
2. Handbook of Drugs in Intensive Care: An A - Z Guide, Fourth Edition.Henry Paw, Rob Shulman
3.Morgan Jr.., Edward J. Clinical Anesthesiology / G. Edward Morgan, Jr., Michael S. Magid / / M., 2015. - V.1, № 3. - p3-375.
4. Kattsung, Bertram G. Basic and Clinical Pharmacology / Bertram G.Kattsung / / 2017. - P.5-577.
5. Marini, John J. critical care medicine / John J. Marini, Arthur P. Wheeler. - M., 2012. - 992s.
6. Hams, AN Internal Medicine: Prakt. Guide: V3t. T3.Kn.2. / AN Hams. 2017. - 480p.
7. Intensive Care Medicine: Annual Update 2010/Jean-Louis Vincent
8. Recent Advances in Anaesthesia and Intensive Care 022 (Recent Advances)/A. P. Adams, J. N. Cashman, R. M. Grounds (Editors), 2000. - 464.
9. Chest Physiotherapy in the Intensive Care Unit, 2nd Edition/Colin F. MacKenzie, P. Cristina Imle, Nancy Ciesla
10.Handbook of Drugs in Intensive Care: An A - Z Guide 3rd Edition/Henry G. W. Paw, Gilbert R. Park
11.Online Resources

Questions for self-Questions on basic knowledge

1. Anatomy of the cardiovascular system. Anatomy of the heart, vascular system, blood supply to the myocardium.
2. Classification of coronary artery disease. Characteristics of different forms of coronary artery disease.
3. Survey methods in CHD.

4. Pathogenesis, clinical features and diagnosis of myocardial infarction, the wording of the diagnosis.
5. Pathogenesis, clinical features and diagnosis of cardiogenic shock, pulmonary edema.
6. Formation mechanism of action potential causes of the violation of automatism, the mechanism of «re-entry».
7. Clinic tachy-and bradyarrhythmias.
8. Physiology of coagulation, anticoagulation and fibrinolytic system of blood.
9. Types of impaired patency of the vessel. Thrombosis, embolism.
10. Hypertension. Pathological changes in the target organs.

Questions about the topic studied

1. Intensive therapy in myocardial infarction.
2. Intensive therapy in myocardial infarction complicated by pulmonary edema.
3. Intensive therapy in myocardial infarction complicated by cardiogenic shock. Auxiliary circulation.
4. Intensive therapy in myocardial infarction complicated by heart rhythm disturbances.
5. Intensive therapy for cardiac arrhythmias: paroxysmal tachycardia, atrial fibrillation, arrhythmia, syndrome of Morgagni-Adams-Stokes equations. Countershock myocardial infarction and arrhythmias.
6. Hypertensive crisis, pathophysiology, intensive care.
7. Thromboembolism in the pulmonary artery. Pathogenesis, clinical features, diagnosis, resuscitation and intensive care.

Topics UIRS

1. Anesthetic management of a patient with acute myocardial infarction.
2. Intraaortic balloon pump. Capabilities of the method, indications for use, effectiveness.

Teaching tools for organization of independent work of students

1. Computer database.
2. Objectives, test control.
3. Thematic sick.
4. Patient records.
5. Bank jobs for self-study.

TrainingMaterial

Treatment of uncomplicated myocardial infarction (MI) Current approaches to the treatment of high demands on the organization of care for acute myocardial infarction (MI). Ambulance to arrive to the patient with suspected myocardial infarction after 15 minutes. after the call and must be equipped with a 12-lead electrocardiograph, defibrillator, and preferably be able to thrombolysis.

Treatment of MI include:

1. Management of pain and excitement
2. Reperfusion
3. Prevention of further blood clots
4. Hemodynamic unloading of the myocardium and decrease periinfarktnoy zone.

1. Management of pain and excitement. The first choice is morphine, which has not only analgesic, but pronounced hemodynamic effect and reduces the feeling of fear, anxiety, psycho-emotional stress. Recommended fractional intravenous morphine 10 mg (1,0 ml of 1% solution)

diluted in 10 ml of saline and injected slowly at first 5 mg, followed if necessary - additional 2-4 mg at intervals of not less than 5 minutes to complete elimination of pain or to occurrence of side effects. In the case of significant excitation can assign tranquilizers (diazepam 0.5% 2,0 ml), although often in pain management excitation removed independently.

Very effective method of anesthesia for anginal status is leptoanalgesia (NLA). Use the combined administration of a narcotic analgesic fentanyl and neuroleptic droperidol. The initial dose of fentanyl is 0.1 mg (2,0 ml), and for those over 60 years old, weighing 50 kg or less chronic lung disease - 0.05 mg (1,0 ml). Dose droperidol depends on the initial level of blood pressure and the range of 2.5 mg (1 ml of 0.25% solution), systolic blood pressure to 100 mmHg and 10 mg (4,0 ml) with blood pressure above 160 mm Hg. The mixture is injected, slowly, after prior dilution in 10 ml of normal saline control of blood pressure (BP) and respiratory rate. Potent analgesic and sedative effect has Hydrochloride - 1 ml of 0.01% solution intravenously, slowly. Analgesia occurs within 4-5 minutes, accompanied by the elimination of emotional and motor responses. Avoid subcutaneous or intramuscular administration of narcotic analgesics, since in these cases the analgesic effect occurs later and less pronounced than intravenous administration. Besides injection sites can be a source of bleeding during thrombolysis. In the case of refractory pain or intolerances NLA tools are used for anesthesia (nitrous oxide, sodium hydroxybutyrate, etc.)

2**. Myocardial reperfusion - a key objective in the treatment of patients with myocardial infarction with elevation ST (STEMI).** Thrombolytic therapy (TLT) - shown in all patients with suspected acute myocardial infarction in the presence of the following criteria:

• chest pain ischemic at least 30 minutes, not cropped again nitroglycerin;

• ST-segment elevation of 1 mm or more in at least two adjacent precordial (for suspected anterior MI) or in two of the three "lower" limb leads, ie II, III, and a VF (for suspected lower IM);

• the emergence of one of the legs of the blockade bundle branch block or idioventikulyarnogo rhythm;

• opportunity to start thrombolysis within 12 hours of onset. Contraindications to thrombolytic therapy:

Absolute:

1. Hemorrhagic stroke in history.
2. Ischemic stroke within the last 6 months.
3. Bleeding, hemorrhagic diathesis.
4. Brain injury or neurosurgical intervention on the brain or spinal cord in the last 3 weeks.
5. Recent (up to 1 month.) Bleeding from the gastrointestinal tract, or urinary tract.
6. Suspicion of dissecting aortic aneurysm.
7. Malignancies.
8. Allergic reactions to the TLT in history (with the introduction of streptokinase).

Relative:

1. Transient ischemic attack in the last 6 months.
2. Hypertension 180/110 mm Hg and above.
3. Stomach ulcer or duodenal ulcer exacerbation.
4. Surgery in the past 3 weeks.
5. Traumatic or long (over 10 minutes) resuscitation.

6. Puncture neperezhimaemyh vessels.
7. Deep vein thrombosis of the lower limbs.
8. Acute pericarditis.
9. Infective endocarditis.
10. Severe liver disease.
11. Pregnancy.
12. Streptococcal infection in the last 3 months.
13. Previous treatment with streptokinase barred from 6 days to 6 months (other drugs are not contraindicated);
14. Acceptance of indirect anticoagulants.

For thrombolysis is recommended to use one of the following drugs: **Streptokinase** -1.5 million IU is diluted in 100 ml of normal saline or 5% glucose solution and administered intravenously over 30 minutes. **Alteplase.** Tissue plasminogen activator (tPA). Standard: 15 mg bolus followed by infusion of the drug 50 mg over 30 minutes, and 35 mg for the next hour. Perhaps the introduction of alteplase two boluses of 50 mg every 30 minutes. **Tenecteplase.** Bolus regimen: 30-50 mg (0.53 mg / kg) for 10 seconds / intravenously. Thrombolysis using tPA necessarily accompanied by intravenous heparin or subcutaneous low molecular weight heparin for 48-72 hours or fondaparinux.

Effectiveness criteria of coronary reperfusion.
• Fast dynamic segment ST: ST-segment depression in lead with the highest rise of 50% and more than 1.5 hours from the start of thrombolytic therapy.
• reperfusion arrhythmias. Most information is considered to be an accelerated idioventricular rhythm and late ventricular premature within 2-3 hours from the start of thrombolytic therapy.
• Fast dynamics of biochemical markers of necrosis. Biochemical criteria of reperfusion is a manifold increase in blood markers of necrosis in 90-120 minutes after thrombolysis (a phenomenon of "washout") to achieve maximum total CPK to 12 hours, CK-MB - up to 6 hours, myoglobin - up to 3 hours . the beginning of TLT.
• Rapid reduction in intensity or complete relief of pain for the 60 minutes from the start of thrombolytic therapy.
• These angiographic study
Side effects of TLT:
• bleeding
• intracranial hemorrhage
• allergic and pyrogenic reactions
• hypotension
• reperfusion arrhythmias

3. Prevention of further blood clots

1. Antiplatelet therapy: acetylsalicylic acid (aspirin) is administered immediately after diagnosis in a dose of 160-325 mg. Tablets should be chewed to accelerate the drug. In the future, aspirin should be taken continuously for 75-160 mg per day. With contraindications to the use of aspirin and the development of side effects assigned clopidogrel 300 mg at day 1, then 75

mg 1 time per day.Addition of 75 mg of clopidogrel to aspirin in acute myocardial infarction prevents about 10 major vascular events per 1000 treated patients without increasing the risk of bleeding (cerebral, fatal or requiring transfusion of blood) - even in elderly patients and in patients undergoing thrombolysis. Co-administration of aspirin and clopidogrel was shown in all patients regardless of MI reperfusion therapy.Several studies have shown the beneficial effect of combined use of aspirin and inhibitors IIb / IIIa glycoprotein (GP), platelets (abciximab, tirofiban, eptifiban). However, no evidence that this combination is superior to the combination of aspirin and clopidogrel.

2. Anticoagulant therapy: heparin is necessary for tissue plasminogen activator thrombolysis and not necessarily the use of streptokinase, except in situations associated with an increased risk of thromboembolic complications.

Indications for heparin therapy:
• extensive anterior wall myocardial infarction of the left ventricle, especially when echocardiography revealed a blood clot in the cavity
• cardiac aneurysm
• recurrent MI
• presence of systemic or pulmonary embolism or indications they had a history
• heart failure
• atrial fibrillation
• presence of complications or comorbidities requiring prolonged bed rest
• elderly patients
• patients with uncomplicated myocardial infarction in the absence of convincing evidence of myocardial reperfusion
• reduction of APTT
• If not done TLT

If the patient of the risk groups received streptokinase, heparin can be started 4-6 hours after thrombolysis, provided that the aPTT increased no more than twice. Heparin bolus injected at the rate of 60-100 U / kg (with a patient weighing more than 70 kg the dose should not exceed 4000 IU), then infusion 12 U / kg per hour. The infusion rate is adjusted according to aPTT, which should be increased by 1.5-2 times. Duration of typically 48 hours, after which you can go to the subcutaneous injection of fondaparinux or low molecular weight heparins.

The advantages of low molecular weight heparin (LMWH) to unfractionated heparin following:
• almost complete bioavailability after subcutaneous injection (90%)
• longer duration, which provides an opportunity subcutaneous injection 1-2 times a day
• a more predictable anticoagulant response
• lower risk of bleeding complications
• lower risk of heparin-induced thrombocytopenia
• less pronounced activation of platelets
• Lower risk of osteoporosis
• No monitoring of aPTT
• no withdrawal
• Ease of use and the reduction of hospitalization due to the possibility of out-

Patterns of use of LMWH:
Enoxaparin 30 mg IV bolus, then subcutaneously with 1 mg / kg, 2 times a day;
Nadroparin 86 IU / kg IV bolus, then sc 86 IU / kg 2 times a day;

Low molecular weight heparin is preferable in patients who had undergone TLT. The duration of treatment with LMWH is individual, but as a rule, at least two days. To a new class of selective inhibitors of factor Xa is a synthetic pentasaccharide fondaparinux. Fondaparinux has 100% bioavailability when s / c administration, antithrombotic activity for 24 hours, the predictable effect of a standard dose of 2.5 mg does not require monitoring of APTT and platelet count, and does not cause heparin-induced thrombocytopenia, significantly reduces mortality and recurrent MI without increasing the risk bleeding, stroke, and cardiac tamponade. Appointed as an adjunct to thrombolytic therapy, and also in cases where thrombolysis is not available in 2.5 mg intravenous bolus, followed by 2.5 mg subcutaneously once daily 1 to 8 days. For the prevention of venous thrombosis and pulmonary embolism - 2.5 mg subcutaneously one time per day. The use of bivalirudin in multicenter trials in patients with thrombolysis was not superior to heparin. But its use in patients with PCI resulted in a significant reduction of cardiac and hemorrhagic complications compared with a combination of heparin and IIb / IIIa inhibitors GP receptors.

4. Hemodynamic unloading of the myocardium and decrease near infarction zone.
Angiotensin-converting enzyme (ACE) inhibitors. Results of multicenter studies of ACE inhibitors can be considered as drugs whose use in macrofocal them mandatory.
Benefits of ACE inhibitors in acute myocardial infarction:
• Hemodynamic unloading of the myocardium;
• Improvement of the coronary circulation;
• Reduction of myocardial hypertrophy, the size of the cavities of the heart, myocardial remodeling of the left ventricle;
• Reduce the risk of left ventricular failure;
• Reducing the risk of dangerous arrhythmias;
• Improvement of diastolic function of the heart;
• Increased fibrinolytic potential of blood;
• Reduce the risk of recurrent MI;
• Increased life expectancy, reduced mortality;
• Improving the quality of life;
• Economic feasibility: cost reduction readmissions by 50%.

Treatment should begin on the first day of the disease with low doses and gradually increasing them with adequate tolerability and absence of hypotension

Nitrates. Nitroglycerin or isosorbide dinitrate administered intravenously, drip, with an initial rate of 10 mcg / min, followed by an increase of 5 mcg / min every 5-10 minutes. Care should be taken that during the administration of the drug in heart rate does not exceed 100 per minute, and systolic blood pressure did not fall below 100 mm Hg Preferably as a continuous infusion of nitrates in 48-72 hours. The daily dose is 80-120 mg. Further introduction of nitrates is justified only if the post-infarction angina or acute left ventricular failure. On the admission of nitrate tablets justified in cases where you can not use ACE inhibitors or β-blockers.

Nitrates are contraindicated in individual hypersensitivity to them in history, systolic blood pressure below 90-100 mm Hg, heart rate less than 50 per minute, acute myocardial infarction of the right ventricle, relatively contraindicated in severe sinus tachycardia. With a view to the appointment of appropriate metabolic cardioprotection by trimetazidine 35 mg two times a day. Since entering the patient with acute coronary syndrome in the hospital should be given a statin (simvastatin, atorvastatin, lovastatin, at least 20 mg per day), which have pleiotropic effects and contribute to the stabilization of the pathological process. Interventional and surgical methods for the treatment of acute myocardial infarction Emergency percutaneous coronary intervention (PCI) performed in patients with myocardial infarction with ST-segment elevation immediately after admission to the hospital, now is the most effective and fastest way to achieve complete reperfusion. In areas where there are opportunities to ensure rapid transportation of the patient with acute coronary syndrome with ST segment elevation in a specialized center that can carry out the procedure of emergency PCI (angioplasty and stenting, stenting metal or coated), interventional revascularization strategy should be considered as the preferred, as it allows have an impact not only on the quality of life, but also significantly improve the prognosis in these patients. Conducting PCI should be performed in the first 12 hours of onset.

Coronary artery bypass surgery is not recommended as an urgent reperfusion strategy, but often runs later field inefficient PCI.

Acute left ventricular failure in myocardial infarction Approximately 15% of patients with acute coronary syndrome defined symptoms of acute heart failure (AHF). AHF in MI is associated with both systolic (violation of contractility), and with diastolic myocardial dysfunction (increased stiffness and impaired relaxation process). Hemodynamic signs of left ventricular failure occur when seriously compromised muscle contractility 20-25% of the left ventricle. If it covers 40% of the myocardial tissue and more, there is a severe AHF and cardiogenic shock occurs. Classification of left ventricular failure in acute myocardial infarction (T. Killip):

I - there are no signs of heart failure;
II - moderate heart failure (rales of no more than 50% of the lung);
III - pulmonary edema (rales over more than 50% of light);
IV - cardiogenic shock.

Pulmonary edema in myocardial infarction - a clinical syndrome caused by hypervolemia pulmonary circulation as a result of left ventricular failure. Pulmonary edema refers to the urgent conditions with a high probability of death (40%) and usually develops in patients with significant left ventricular injury or acute mitral regurgitation due to ischemic dysfunction or papillary muscle rupture.

Clinic:
• choking, wheezing congestion in the lungs (50% of the lung fields), third heart sound (gallop);
• Severe arterial hypoxemia (SaO2 <80%);
• The radiological signs of pulmonary edema: venous engorgement, increased lung roots, round,

patchy shadows, scattered in all fields;
• cough with frothy pink sputum;
• Acrocyanosis, cold sweat, arrhythmic thready pulse, hypotension;
• Echocardiographic signs of global systolic (ejection fraction <40%) and / or diastolic dysfunction of the left ventricle
Treatment of pulmonary edema requires urgent intensive activities.
For maximum effect, should follow a certain sequence (and if possible simultaneity) urgent measures.
Recommendations for the treatment of pulmonary edema.
1. Oxygen 8-10 L / min (Level of evidence C).
2. Fast-acting diuretics intravenously (furosemide) (B).
3. Peripheral vasodilators intravenously (in the absence of hypotension) (B).
4. Inotropic support (dopamine and / or dobutamine) (with hypotension) (B).
5. Ventilation (ALV) (if applicable) (C)

It is mandatory that a permanent venous access using a catheter (preferably two catheters) into a peripheral vein. If it is impossible invasive monitoring of intracardiac and central hemodynamics in / infusion conducted under the close control of blood pressure, pulse, BH, auscultation and, if necessary, bedside X-ray of the lungs. In the absence of systemic hypotension is recommended semi-sitting position of the patient with a lower down, of course, which leads to a decrease in the hemodynamic load on the heart due to a partial deposit of venous blood in the veins of the lower extremities. Possible imposition of venous tourniquets on hips. Oxygen is appointed by nasal catheters or mask at a rate of 4.6 l / min with its rise as necessary to 8-10 l / min. Possible to use defoamers (70% solution of ethyl alcohol or 10% alcohol pp antifomsilana) wire monitoring of blood oxygen saturation pulse oximeter. All the patients in addition to oxygen shown noninvasive mechanical ventilation with positive end-expiratory pressure (PEEP / PEEP from 5-7 do10sm water column). Morphine and other drugs. The use of morphine is not necessary in the treatment of pulmonary edema. However, with the pain syndrome, severe tachycardia and excitement it can be successfully used as a I / boluses divided doses. Furosemide is administered in a dose of 20-40 mg as / in the bolus. Diuretic effect develops in 3-4 minutes after i / v administration, reaches a maximum after 30 minutes and lasts 1-2 hours re-introduction depends on the clinical situation. The total dose may be 160 mg or more. In the application of furosemide should be considered a possible reduction in systemic blood pressure. Nitroglycerin in / infusion in the absence of hypotension appointed immediately (before vein catheterization may use tablets, 0.5 mg sublingually every 7-10 minutes). Infusion (2 ml of a 1% solution of 200 ml 0,9% sodium chloride) start at a rate of 10-20 mg / min and increased by 5-10 mcg / min every 5 minutes until a decrease in Td 10-15 % or to 90 mm Hg. Art. When the desired effect of the infusion rate gradually. When failure of nitroglycerin infusion of sodium nitroprusside conduct, a split ganglion blockers. In case of severe hypotension shows the assignment of inotropic agents. If oliguria or anuria (signs honorable hypoperfusion), resistant to the action of diuretics, dopamine is shown in the form of in / infusion with an initial velocity of 2.5-5.0 mcg / kg / min. With the dominant pattern of pulmonary edema are preferred as dobutamine in / infusion with an initial rate of 2.5 mcg / kg / min, with a gradual increase in the rate of infusion every 5-10 min to 10 mg / kg / min or until the hemodynamic response. In some cases, perhaps, co-administration of nitroglycerin and inotropic agents and a long in / infusion in different veins (under the control

of blood pressure). If, despite the inhalation of 100% oxygen at a rate of 8-10 l / min it is possible to achieve a sufficient increase O2 saturation of the blood (PaO 2 <60 mm Hg. Cent.) Is shown intubation and mechanical ventilation. Cardiogenic shock (CS) (or true cardiogenic shock) applies to emergency states with a high probability of death (80-90%), and usually develops in patients with significant damage to the left ventricle (40-70% of the mass of the myocardium).
The cause of cardiogenic shock can be bradycardia, ventricular fibrillation, ventricular tachycardia.Choice of treatment depends on the KS pathogenesis of left ventricular failure and baseline clinical and hemodynamic situation in each case.

Recommendations for treatment of true cardiogenic shock:
1. Oxygen 8-10 L / min (Level of evidence C).
2. Inotropic support (dopamine, dobutamine, norepinephrine) (B).
3. Ventilation (if necessary) (C).
4. Adequate thrombolytic therapy (A).

The absence of convincing clinical effect within 1-2 hours of intensive medical therapy is the basis for the use of intra-aortic balloon counterpulsation. Hemodynamic unloading of the left ventricle leads to an increase in cardiac output, blood pressure, improved coronary perfusion, functional status and contractility area of ischemic damage and creates conditions for a significant increase in the effectiveness of medical treatment. Significant improvement in the results of treatment CABG may be the result of the opening of the infarct-related coronary artery, recovery of myocardial perfusion and limit the area of necrosis. In actual clinical conditions that may contribute to early and intense as thrombolytic therapy. Recommends the introduction of 3,000,000 IU of streptokinase for 30 minutes, followed by drip infusion of heparin for the correction of microcirculation. TLT CABG patients is justified, even for late admission (after 12-24 hours of onset). The most promising treatment for CABG in recent years is the opening thrombosed coronary artery by PCI. Restoring full patency of the infarct-related coronary artery is achieved with a 55-75% of patients, and the survival rate at 30 days - in 65-70% of patients with CABG. Coronary angioplasty significantly improves short-term and long-term prognosis in these patients. In the event of a rupture of CABG interventricular septum or separation (strain) papillary muscle shows an urgent surgical correction of the anatomic defect simultaneously with aorto-coronary bypass surgery. Intraaortic balloon pump (IABP) is a method of assisted circulation (along with extracorporeal membrane oxygenation, artificial ventricles, artificial heart).

Assisted circulation can be used short term in cardiogenic shock in the following situations:
• after heart surgery
• myocardial infarction
• fulminant myocarditis with
• If cardiac arrest during cardiac surgery (survival rate low in this case)

Mechanism of action of IABP. System for IABP consists of a console, tank and gas line. Plastic container for dibs from 2 to 50 mL injected into the descending aorta via the femoral artery, so that its tip was immediately distal left subclavian artery. The balloon is filled with helium. It

swells in early diastole, which leads to improved coronary perfusion, and deflates just before systole, which reduces afterload. This is accompanied by improvement in the ratio of delivery and myocardial oxygen demand. The increase in cardiac output may reach 40%.
Indications:

• disconnection from the heart-lung machine
• perioperative refractory ischemia
• in the postoperative period - low cardiac output refractory to inotropic support
• afterload reduction in cardiogenic shock.

Contraindications:
• aortic regurgitation (balloon inflation in diastole worsen aortic regurgitation)
• aortic dissection (may increase stratification, the risk of the introduction of the cylinder in the false lumen).

Intensive Care Unit of cardiac arrhythmias

From a practical point of view, allocate:
• life-threatening arrhythmias: ventricular fibrillation, sustained ventricular tachycardia, asystole, subnodal complete AV block;
• potentially life-threatening arrhythmias: ventricular premature high gradation of Lown and Wolf (frequent, early, politopic heart beats, couplets, triplets), unstable ventricular tachycardia, alternating block bundle-branch block, AV block grade 2 type 2 Mobittsa;
• hemodynamically unfavorable fibrillation: marked tachycardia or bradycardia (any location), paroxysmal supraventricular tachycardia, atrial fibrillation and atrial flutter;
• nonvitadangerous fibrillation: a moderate sinus tachycardia or bradycardia, rare supraventricular or ventricular extrasystoles, AV block grade 1 or grade 2 type 1 without intraventricular conduction disorders.

Features appointment antiarrhythmic drugs against acute myocardial infarction
Class I antiarrhythmic drugs increase the risk of death in patients with acute myocardial infarction despite the fact that they effectively eliminate the disruptions. Found that drugs IA and IC classes shortened action potential, which leads to increased likelihood of ventricular fibrillation. Therefore, against the background of acute myocardial infarction is contraindicated drugs IA and IC classes etatsizin, propafenone, procainamide, hinidine, flecainide. β-blockers in the absence of conduction disorders and severe heart failure, it is desirable to assign in the first days of MI. Determine the effect of shortening of the action potential to increase the risk of ventricular fibrillation explains antifibrillation action β-blockers, sotalol and amiodarone by their ability to prolong the action potential and effective refractory period. However, be aware that the drugs class III (amiodarone, sotalol) can cause excessive prolongation of the action potential (it is shown on the ECG QRS complex extension and prolongation QT), which leads to the risk of ventricular tachycardia type torsades de pointes. Data about the use of calcium antagonists verapamil contradictory - according to some, they may limit the zone of necrosis, but there is also information about the increased risk of death when used.
Proved by the low efficiency of cardiac glycosides and high risk of digitalis toxicity in patients with acute left ventricular failure. Currently, they are used only when tachysystolic atrial

fibrillation.

Emergency care for tachyarrhythmias. Sinus tachycardia require treatment, if a cause of post-MI angina, increase of heart failure, hypotension. Drugs of choice are β-blockers. In the absence of conduction disorders and severe heart failure, it is desirable to assign them in the first days of MI, as this is the only anti-arrhythmic drugs, which proved a significant increase in life expectancy of patients after myocardial infarction. When a paroxysm of supraventricular tachycardia treatment should begin with vagal techniques. If this is not enough, the drug of choice is verapamil. Adenosine (or ATP), which is widely used in supraventricular tachycardia, acute myocardial infarction is contraindicated. For relief of Hazards Introduction to \ to propranolol, in marked tachycardia and ineffectiveness of other drugs - amiodarone. The ineffectiveness of medication and growth of left ventricular failure, cardioversion (EIT). Flicker, flutter, atrial fibrillation is an independent risk factor for increasing mortality. Patients who have atrial fibrillation (AF) has evolved in acute myocardial infarction, have a higher risk of death within 1 year after MI compared with patients who have maintained sinus rhythm, or with those who developed AF month after MI. With worsening ischemia or hemodynamic instability shows an urgent restoration of rhythm by cardioversion (EIT). With a relatively stable hemodynamics for restoring sinus rhythm are used primarily amiodarone or cardiac glycosides that reduce ventricular tachycardia and restore sinus rhythm. For rate control when emergency recovery rate is not shown, β-blockers are used, provided preserved left ventricular contractility, AV block or absence of asthma. When the duration of AF for more than 48 hours is shown anticoagulant, especially when macrofocal anterior infarctions. In patients without MI and other organic heart disease for medical recovery rate can be used propafenone or flecainide. Ventricular extrasystoles (VES) - the most common heart rhythm disturbance in MI. Beats detected in more than 93% of patients with acute myocardial infarction. Traditionally divide graduation VES by Lown and Wolf, whereby VES 3-5 classes (early, politopical, bi-trigeminy, couplets, triplets) is considered potentially malignant, and its appearance is a poor prognostic sign. The drug of choice is lidocaine, amiodarone rarely, perhaps - β-blockers. Register paroxysms unstable ventricular tachycardia (duration of 4 consecutive complexes up to 1 minute) is essential to determine the prognosis of patients after myocardial infarction. The emergence of even unstable paroxysmal ventricular tachycardia worsens prognosis. In this case, the emergence of unstable ventricular tachycardia in the first 24 hours of AMI are not associated with an increased risk of mortality (both overall and sudden). The occurrence of this rhythm disturbance at a later date due to a doubling of mortality and requires appropriate treatment. Drugs of choice are lidocaine, β-blockers, amiodarone. Possible use of magnesium sulfate (for the type of ventricular tachycardia torsades de pointe). When spasm sustained ventricular tachycardia (VT) and ventricular fibrillation (VF), the need for urgent restoration of rhythm in the first place by the EIT. Emergency care for bradyarrhythmias. Intensive therapy bradycardia when a syncope, seizures Morgagni-Adams-Stokes (MAS), hypotension, chest pain appears, congestive heart failure, increase ventricular arrhythmias. Temporary pacing (pacing) is indicated in all patients with symptomatic bradycardia. In case of impossibility of ECS: atropine, ortsiprenalin, in the absence of other agonists - adrenaline (2-10 mg / min / drip timely controlled blood pressure, heart rate and ECG). The issue of a permanent pacemaker should be made no earlier than 7-10 days from myocardial infarction. It should be remembered that the two-chamber atrial-ventricular pacing, keeping the contribution of atrial systole and the physiological sequence of atrio-ventricular contraction, significantly reduces the severity of heart failure and improve the prognosis of patients after myocardial infarction.

Technique of cardioversion (EIT):
1. Adjust oxygen therapy.
2. Premedication: fentanyl 0.05 mg / in, or trimeperidin 10 mg / in, pulmonary edema - morphine 1 mg / in, with initial respiratory depression - analgin 2-2.5 g / in.
3. While maintaining consciousness in an outpatient setting - drug dream diazepam 5 mg intravenously and 2 mg every 1-2 minutes to fall asleep, in the event of an emergency at the hospital - sodium thiopental 2% solution of 20-30 ml / slow (under the control of blood pressure).
4. ECG monitoring of cardiac rhythm.
5. During the EIT:
• Use well-moistened pads or gel;
• at the time of application of the discharge with the power to press the electrodes to the chest;
• Apply the discharge at the time of expiration, observing safety regulations;
• Interval between repeated discharges of at least 1 minute.
• the absence of effect repeated cardioversion, doubling the energy of the discharge;
• the absence of effect repeated cardioversion maximum discharge energy;
• The maximum allowable discharge energy - 360 J (5 J / kg);
• If there is no effect to introduce the drug intravenously, shown in this arrhythmia and repeated cardioversion maximum discharge energy;

When atrial fibrillation, supraventricular tachycardia - start with 50 J for atrial fibrillation - start with 100 J, with monoformic VT - starting with 100 J. With the ineffectiveness of these parameters, the energy level is increased by 2 times. When poliformic VT, VF - start with 200 J, the ineffectiveness of increasing energy level to a maximum.
Hypertensive crisis. Hypertensive crisis (HC) - clinical syndrome characterized by a sudden and rapid worsening of hypertension and symptomatic hypertension. a sharp increase in blood pressure to high values individually, subjective and objective manifestations of cerebral, cardiovascular and vaguscommon violations. The main factors leading to the development of hypertensive crises:

• psycho-emotional stress;
• excessive use of salt;
• changes in weather and variations in atmospheric pressure; gipertonicheskiekrizy often recorded in the spring and autumn months, at least - in the winter and summer, are more sensitive to meteovliyaniyam patients with neurosis, asthenic-neurotic reactions suffering from osteochondrosis of the cervical spine;
• recurrent episodes of cerebral ischemia (predominantly in the elderly due to lack of blood flow in the vertebral-basilar basin, for example, the morning hypertensive crises in an awkward position of the head during sleep);
• the impact of infectious diseases (during influenza epidemics is increasing the frequency of hypertensive crises);
• discontinuation of clonidine after treatment with the optimal dose for 3 months or longer;
• sudden cancellation Duration Allowed sympatholytics (dopegita, izobarina, etc.), resulting in a sharp increase in the sensitivity of the OC-adrenoceptor resistance vessels to catecholamines;

• the introduction of diuretics patient pheochromocytoma.
The most important pathogenetic factors of GC are:
• hyperactivation sympathoadrenal system;
• acute or gradually increasing the delay of sodium and water;
• activation of the calcium mechanism of smooth muscle cells of arteries and arterioles;
• activation of the renin-angiotensin II-aldosterone.
The main diagnostic criteria for hypertensive crisis:
• relatively sudden onset (from several minutes to several hours);
• individually high blood pressure, and as a rule, diastolic blood pressure greater than 120 mm Hg. Art.
There are different classifications of hypertensive crises, but from a clinical point of view, and in order to provide effective assistance to the most appropriate hypertensive crises divided into two groups (GifFord et al., 1991).
Crease I - for conditions requiring immediate reduction of blood pressure (within 1 hour):
• hypertensive encephalopathy;
• acute left ventricular failure;
• acute aortic dissection;
• eclampsia;
• postkoronarny arterial bypass;
• Some cases of hypertension, combined with increased levels of circulating catecholamines (pheochromocytoma, hypertension syndrome clonidine, food and drugs that interact with monoamine oxidase inhibitors, injections or oral sympathomimetics, cocaine);
• hypertension in intracerebral hemorrhage;
• acute subarachnoid hemorrhage;
• acute infarcts (strokes) in the brain;
• unstable angina or acute myocardial infarction.
Crease II - with conditions requiring reduction of blood pressure within 12-24 hours:
• high diastolic hypertension (140 mm Hg. Cent.) Without complications;
• malignant hypertension without complications;
• hypertension in the postoperative period.

Treatment of hypertensive crises
1. With drug treatment of hypertensive crises is necessary to solve the following problems:
2. Relief of blood pressure increase. It should determine the degree of urgency of treatment, medication and choose the method of its administration, to set the desired speed reduction of blood pressure, determine the level of acceptable reduction in blood pressure.
3. Ensure adequate monitoring of the patient during blood pressure reduction. The need for timely diagnosis of complications or excessive blood pressure reduction.
4. Securing of impact. This usually prescribe the same medicine with which reduce blood pressure, if you can not - other antihypertensives. Time is determined by the mechanism and for the selected products.
5. Management of complications and co-morbidities.
6. Selection of the optimal dosage of drugs for maintenance treatment.
7. Preventive measures to prevent crises

Emergency relief of hypertensive crisis is in the states, as the crisis marked Giflford I, ie situations, which threaten the patient's life.
The most common - is:
• convulsive form of hypertensive crisis (acute dire hypertensive encephalopathy);
• hypertensive crisis in pheochromocytoma;
• hypertensive crisis, myocardial infarction, hemorrhagic stroke, pulmonary edema, dissecting aortic aneurysm.

Program emergency relief of hypertensive crisis assumes a reduction in blood pressure for 1 h at 25-30% from baseline. This reduces the risk of irreversible damage to the brain and internal organs and death. Patients should be hospitalized in the intensive care unit. For emergency relief of hypertensive crisis is usually applied, intravenous active antihypertensive drugs with the transition in the future to ingestion of effective medicines. Aids for the emergency relief of hypertensive crisis: Dibazol - imidazole derivative, has vasodilating, antispasmodic, hypotensive properties, improves regional blood flow in the brain, heart and kidneys. Hypotensive effect by dibazol expressed moderately. Dibazol administered intravenously at a dose of 4.3 ml of a 1% solution (6.8 ml of 0.5% solution), and the hypotensive effect seen in 10-15 minutes and lasts for about 1-2 hours may be applied dibazol intramuscular injection of the same dose, but hypotensive effect occurs later (30-40 minutes) and less pronounced than intravenous administration. Side effects are rare dibazol: perhaps paradoxical transient increase in blood pressure when administered intravenously, in elderly patients may decrease cardiac output. Contraindicated of dibazol injection for severe heart failure. Magnesium sulfate - reduces CNS excitability, has anticonvulsant, antispasmodic, dehydration effect. In addition, magnesium sulfate is effective in cardiac arrhythmias, particularly those related to hypokalaemia, overdose of antiarrhythmics and cardiac glycosides, cardiac failure. Shows the use of magnesium sulfate in hypertensive crisis flowing with seizures and ventricular tachycardia. Magnesium sulfate is administered intravenously slowly (over 5-7 minutes) in 10 ml of 25% solution in 10 ml of isotonic sodium chloride solution or intramuscularly. By intravenous administration of magnesium sulfate may stop breathing. Diazepam (seduksen, Relanium) - tranquilizer takes excitement, fear, anxiety, increases the effects of antihypertensive drugs. Slowly injected 2 ml of 0.5% solution

Pulmonary embolism by clot. Etiology. The most common causes of pulmonary embolism (PE) are the separation of venous emboli and plugging them or all of the pulmonary artery bed. In 85-90% of cases, the source of embolus is in the inferior vena cava or in the veins of the legs and pelvis, at least - in the right heart and the veins of the upper extremities. Predispose to thrombosis various injuries (including postoperative), chronic heart failure, bed rest and the resulting slowing of blood flow. In patients with myocardial infarction, stroke, cardiac decompensation, chronic insufficiency of the lower limbs venochnoy PE develops most often. Acute venous thrombosis can develop on the background of different cancers. Cancer intoxication alters hemostasis, inhibiting fibrinolysis system and strengthening giperkoagulyatsionnye properties of blood. PE is the "scourge" of the postoperative period. Most often it develops after surgery on the prostate, bladder, Macka, colon and stomach. This complication often occurs in trauma patients, particularly the elderly. Predisposing factors for pulmonary embolism are also considered obesity, pregnancy, use of oral

contraceptives, hormonal, hereditary thrombophilia (deficiency of antithrombin III, protein C). PE occurs as a consequence of subclavian vein, and in children as a complication of umbilical sepsis.

Depending on the size of the arterial bed is off a small distinction, subtotal, massive and fatal acute embolism to the volume of the channel is off the pulmonary artery by 25, 50, 50 and 75%. In 10-25% of cases after embolism develops pulmonary infarction. If vascularization located distal to the obstruction of lung tissue through bronchopulmonary anastomoses sufficient, pulmonary infarction does not develop. European Society of Cardiology asked to classify in terms of pulmonary embolism pulmonary vascular lesions (solid and nonmassive on severity of the pathological process (acute, subacute, and chronic recurrent). Regarded as a massive pulmonary embolism, if the patient has the phenomenon of cardiogenic shock or hypotension (not associated with hypovolemia, sepsis, cardiac arrhythmias). Nonmassive diagnosed pulmonary embolism in patients with a relatively stable hemodynamics without obvious signs of right heart failure. Pathophysiological changes in the PE exercise increased resistance pulmonary arterial and pulmonary arterial hypertension, which leads to increased load on the right ventricle, and in some cases, to its acute deficiency. In patients without concomitant heart and lung disease, pulmonary hypertension occurs when a threshold value is exceeded embolic obstruction - 50% occlusion of the pulmonary circulation. A further increase of this index leads to a parallel increase in the total pulmonary vascular resistance, the pressure in the pulmonary trunk and right chambers of the heart, decreased cardiac output and oxygen tension in the arterial blood. In the acute stages of a massive pulmonary embolism may lead to a rise in pressure in the pulmonary circulation of 70 mmHg Excess of the level of this parameter indicates the long-term nature of the embolic occlusion or the presence of chronic pulmonary heart disease.

Depending on the size of the arterial bed is off a small distinction, submassive, massive and fatal acute embolism to the volume of the channel is off the pulmonary artery by 25, 50, 50 and 75%. In 10-25% of cases after embolism develops pulmonary infarction. If vascularization located distal to the obstruction of lung tissue through bronchopulmonary anastomoses sufficient, pulmonary infarction does not develop. European Society of Cardiology asked to classify in terms of pulmonary embolism pulmonary vascular lesions (solid and nonmassive) on severity of the pathological process (acute, subacute, and chronic recurrent). PE is regarded as a solid, if patients develop cardiogenic shock effects or hypotension (not related to hypovolemia, sepsis, cardiac arrhythmias). Nonmassive PE diagnosed in patients with a relatively stable hemodynamics without obvious signs of right heart failure.

Clinic PEC. The clinical picture of pulmonary embolism vary considerably from almost complete absence of symptoms to rapidly developing state of acute pulmonary heart disease. Usually there are three main options for pulmonary embolism, acute, subacute and relapsing. During acute occurs in 30-35% of patients, it is characterized by the sudden development of dyspnea, collapse, agitation, cyanosis and fraught with massive thromboembolism or major. This variant of PE accumulate lightning legal outcome. Subacute (45-50%) is, as a rule, with increasing pulmonary thrombosis, superimposed on the original small or large emboli. This option appears progressive respiratory and right heart failure, symptoms of pleuropneumonia, often hemoptysis. Recurrent in 15-25% of patients characterized by repeated, 3 to 5 times or more acute attacks because of small vessel thrombosis, occurring half mask transient syncope, seizures tiles, unexplained fever, atypical angina, pneumonia, dry pleurisy.

By the degree of decreasing the frequency of symptoms of pulmonary embolism can be

represented as follows: shortness of breath, tachycardia, fever, cyanosis, pain, accent II tone in the pulmonary artery, cough, wheezing in the lungs collapse, allergic reactions, swelling of the neck veins, pleural friction rub, hemoptysis , fear of death, liver swelling, irregular heartbeat, brain damage, systolic murmur at the pulmonary artery, vomiting, bronchospasm, involuntary urination and defecation, pulmonary edema, bradypnea.

Depending on the prevalence and the combination of these symptoms are following syndromes:
1. Lung and pleural syndrome - shortness of breath, chest pain, coughing, sometimes with sputum. This syndrome occurs in small and submassivnoy embolism, ie with occlusion of one common artery or peripheral pulmonary artery branches.
2. Cardiac syndrome: pain and discomfort in the chest, tachycardia and hypotension until the collapse or fainting. There may be swelling of the neck veins, positive venous pulse, accent II rut of the pulmonary artery, increased central venous pressure. This option is typical for massive pulmonary embolism.
3. Cerebral syndrome: loss of consciousness, hemiplegia, seizures. This variant is characteristic of lime elderly.

These syndromes can be combined in various combinations. Dyspnea and tachycardia are the most common manifestations of pulmonary embolism, they are observed in 70-100% of patients. Shortness of breath, usually appears suddenly, combined with the fear of death, varies widely, with an average of 30-40 breaths per minute. In most cases, shortness of breath is a "quiet" nature, is not accompanied by noisy breathing and the forced movement of the chest. Heart rate varies from 90 to 160, with an average of 110 per minute. Possible disruptions in the form of arrhythmia and atrial fibrillation. Discoloration of the skin and mucous membranes may depend on the caliber of pulmonary embolism in the thrombosed vessel, the degree of hypotension, concomitant vasoconstriction, etc. Only when stem massive thromboembolism develops cyanosis of the skin, the rest of the patients have "Ash" pale in conjunction with acrocyanosis, in some patients, the combination of cyanosis and icterus. Raising the temperature to low-grade, less febrile digits starting from the first day of PE and stored up to 10-12 days. Fever is associated with inflammatory changes in the lung and pleura often with lesions of medium and small branches of the pulmonary artery. Hemoptysis, considered a classic manifestation of pulmonary embolism, in fact, occurs not more than 30% of patients. Chalice observed cough with scanty mucous expectoration in combination with non-specific physical changes in the saw deadened sound, relaxed breathing and finely wheezing in a limited area, some patients auscultated pleural friction. Pain syndrome occurs in 40-70% of patients, it can be varied: 1) anginozopodobny often with massive obstruction pulmonary trunk, and 2) lung and pleural and 3) mixed, combining the first two options, and 4) abdominal characterized picture mistaken of acute abdomen due to lesions more often right diaphragmatic pleura. At the same time there may be dysphagia, hiccups, belching, stool disorders etc. Hypotension and collapse - frequent and regular manifestations of pulmonary embolism. Observed in about half of patients, characterized by a combination of hypotension with venous hypertension, hemodynamic changes in the systemic circulation may be accompanied by cerebral disorders, mesenteric and renal blood flow. More common in the collapse of a massive embolism, duration is a poor prognostic criteria.
Syndrome of acute pulmonary heart is with PE distinct clinical and electrocardiographic

manifestations. Observed enhancement of cardiac shock, swelling jugular veins, expanding the right border of the heart, pulsation of the left intercostal space, II, II accent colors and its breakdown on pulmonary artery systolic murmur over it, similar to the noise may listens projected tricuspid valve, possible arrhythmias, signs of stress Right heart on an electrocardiogram.
Physical symptoms of pulmonary infarction and its complications like hospital pleuropneumonia. For mild heart attack (usually the right) seroplastic or hemorrhagic pleurisy arising after 1-2 weeks of illness. In the subacute phase of myocardial lung (2-5 weeks) may experience allergic syndrome due to absorption of the products of necrosis of lung tissue. It is manifested by skin rash, eosinophilia, a second wave of inflammatory changes in the lung and pleura. Methods of diagnosis of pulmonary embolism. Diagnosis of all forms of pulmonary embolism remains unsatisfactory, diagnostic errors are common. The key to the diathesis of PE is to remain aware of the possibility of its occurrence in the respective categories of patients. Correct and timely diagnosis of pulmonary embolism - this is at least half the success of treatment. ECG reveals a number of characteristic for pulmonary embolism syndrome:

1) negative T waves in leads V1-3 and their broadening;
2) ST-segment elevation in leads III, AVF, V1-3 and its decline in leads I, II, AVL, V5-6;
3) development of right bundle branch block, and cardiac arrhythmias;
4) evidence of an overload of the right heart.

Echocardiography to judge the development of acute, subacute or chronic pulmonary heart disease, valvular pathology excludes and left ventricle, the method is also used to assess the patency of the central pulmonary arteries and detection of patent foramen ovale. Without contrast lung radiography is not a specific way of diagnosing venous thromboembolism and, at best, can be suspected pulmonary embolism. Radiographic signs: acute dilatation of the right heart, the expansion of the inflow tract by hypertension, high standing of the diaphragm and a symptom of Westermarck (weakening pulmonary pattern in the area of embolic occlusion) showed a massive pulmonary embolism. The classic tapered shadow pulmonary infarction is rare. Currently, X-ray data are important not for the diagnosis of pulmonary embolism, and to exclude other similar symptoms to pathology. Perfusion lung scan performed after intravenous administration makrosfer albumin labeled with 99mTc. The absence of pulmonary perfusion scintigram, performed in at least two different views (front and back) completely exclude the diagnosis. Ideally, with this method, the study should begin evaluation of patients with suspected pulmonary embolism. Criteria of high-segmental emboli are "off" pulmonary blood flow. Ultrasound scanning of deep veins can reliably judge the presence of thrombosis. Angiography plays a crucial role in the diagnosis and selection of treatment for pulmonary embolism. Oka is shown in all cases where not excluded massive thromboembolism and need to decide on the need for surgical intervention - embolectomy. Unfortunately, the conduct of this important research possible only in specialized centers where angiography is performed in conjunction with right heart catheterization and retrograde ileocavagrafy. Laboratory Methods. A retrospective analysis of parameters of coagulation and anticoagulation system suggests that the majority of patients to the development of pulmonary embolism tendency to hypercoagulability in acute thromboembolism is compensatory activation of anticoagulation system, and in subacute tendency to hypercoagulability increases, which creates

conditions for recurrent pulmonary embolism. In the acute phase marked the early appearance of leukocytosis (often with stab shift), which is stored between 2 and 5 weeks, in parallel with the increase of leukocytosis characteristic ESR (approximately 80% of patients). In the acute phase can be observed eosinopenia, lymphopenia and relative monocytosis in the subacute stage against allergic reactions can eosinophilia within 12-23%. In 40-46% of patients have hypochromic anemia, with increased indirect bilirubinemia faction. For Talley characterized by increased LDH (especially LDG3), aldolase, ALT, alkaline phosphatase with normal AST and CPK.

In the last decade in clinical practice for diagnosis of deep venous thrombosis and pulmonary embolism apply methods based on the detection of markers of activation of coagulation and fibrinolysis, as the definition of fibrinopeptide A and D-dimer. These methods are highly sensitive in thrombosis, but not specific enough for the diagnosis of DVT and PE. Thus, the sensitivity of the methods for the determination of D-dimer was 99%, specificity (compared with venography) - 53%. If in the case of a negative reaction to the presence of D-dimer test can confidently talk about the absence of venous thrombosis, then the positive reaction to the D-dimer thrombosis diagnosis should be confirmed by other methods.

Treatment of pulmonary embolism. Inhalation of oxygen, inadequate ventilation - endotracheal intubation and mechanical ventilation (ALV). In the application of mechanical ventilation have to use antibacterial filters. Infusion therapy, to ensure an adequate flow of blood to the right heart, with care as a bolus of 250-500 ml with subsequent evaluation of hemodynamic effect. Use drugs improve blood rheology (reopolyglucin in total 400-800 ml / in cap.). If necessary, the support means inotropic (dopamine, dobutamine) for a critical fall in blood pressure - norepinefrine.

With massive pulmonary embolism, accompanied by severe pain, opioids are used (promedol of 10 mg). Avoid drugs that cause reduction of central venous pressure due to venous vasodilation (morphine, nitroglycerin, diuretics). Specific therapy. The main method of conservative treatment of pulmonary embolism is anticoagulation therapy and in combination with, activate their own crowns and fibrinolytic activity improves the rheological parameters. Restore patency of the pulmonary artery can be through fibrinolytic therapy. To this end, use of endogenous fibrinolysis activators: streptokinase, urokinase, alteplase. The "classic" dose of streptokinase is 250,000 units for 25-30 min., And then 100,000 units / hour infusion over 12-24 hours. According to some authors, if necessary infusion of fibrinolytics may be extended to 1-3 days. In severe cases, the initial dose may be increased to 1000000 units and put in a few minutes. If an hour after intensive treatment systolic blood pressure is below 90 mm Hg, urine output of less than 20 ml / h, the oxygen pressure less than 60 mm Hg, shown embolectomy surgery in specially equipped for this clinic. Anticoagulation direct action, according to most authors should begin after fibrinolytic therapy, although some clinicians prescribe both heparin and fibrinolytic There is also a third option in the treatment strategy of PE when thrombolytic therapy is started after a single bolus injection of 10.5 thousand units of heparin. The duration of treatment with heparin is, according to different authors, from 5-7 to 10-14 days. Daily doses ranged from 30 to 60 thousand units. Treatment is carried out under the supervision of APTT (activated partial thrombin time) and blood clotting, which should be 1.5-2 times longer than normal.

Low molecular weight heparin - a possible alternative to unfractionated heparin: enoxaparin (1 mg / kg every 12 hours p / c), fraxiparine (0.1 ml per 10 kg, 2 times a day). LMWH is equally effective and safer, but there is a risk of insufficient dose selection. 2-3 days prior to the

incidents move to indirect anticoagulants (fenilin, Coumadin and others) at doses that support the INR (international normalized ratio) between 2.0-3.0. Treatment a long-term indirect anticoagulants for 1.5-6 months.

Self-study

Task number one

Analyze the history of a patient with cardiogenic pulmonary edema, is in the intensive care unit and intensive care. To do this:

• appreciate the severity of respiratory failure in a patient based on the diaries of follow-up;

• analyze the amount of emergency assistance.

Task number two

Examine and analyze the patient's history, with the jetlag. To do this:

• Determine the nature of the disruptions

• appreciate the correction method arrhythmias, offer alternative methods

• Determine the risk of thromboembolic complications.

Clinical problems

Objective number one

In the emergency room "ambulance" brought the young man unconscious in the street. In the car recovered consciousness. Patient tells about feeling interruption of the heart, "heart turned over in his chest and stopped" on questioning revealed that a similar incident took place one month ago, the medical care is not addressed. ECG signs of complete AV block III degree.

Explain the cause of established states. Assign a drug for relief of attacks.

Objective number two

Patient 56 years old, was admitted to the intensive care unit with complaints of severe, pressing chest pain that developed during the last two hours, headache, nausea, vomiting, a flicker in the eyes of flies. OBJECTIVE: BP 185/110 mm Hg. Art., HR-90 min. The patient exhibited diagnosed CHD macrofocal myocardial infarction, acute period, hypertensive crisis of the first order. What are the directions and methods of intensive care intensive care.

Task number 3

Patient 76 years brought to the emergency department with a diagnosis of acute myocardial infarction. Complains of shortness of breath for 6-7 hours. No pain. He suffers from diabetes, hyperglycemia at admission 16 mol / L, no acetonuria, BP 160/100 mm Hg. Art., excited over the light on both sides of dry rales are heard in all lung fields, expiratory wheezing, swelling in the legs moderately expressed. Your tactics?

Test control:

1. What or which of the signs on the ECG are the evidence for myocardial infarction?

1). Segment depression, ST;

2). Low voltage teeth;

3). Equilateral high T waves;

4). Barb QS combined segment elevation ST *.

2. Which of the following does not meet ECG signs conclusion that there is a patient Frontapical and lateral myocardial infarction?

1). QS in leads V4 - V6;

2). ST-segment elevation in leads V4 - V6 *;

3). T-wave inversion in these leads;

4). Ventricular complexes in leads II, III, aVF has the form: small - r, deep - Q *.
3. Which of the following drugs is impractical to use in cardiac asthma in patients with acute myocardial infarction:
1). Digoxin;
2). Strophanthin *;
3). Lasix;
4). Morphine;
5). Nitroglycerin.
4. Which of the following drugs should not be administered in cardiac asthma and pulmonary edema in patients with stenosis and subaortal isolated mitral stenosis:
1). Sodium nitroprusside *;
2). Strophanthin *;
3). Lasix;
4). Digoxin;
5). Morphine.
5. Cardiogenic shock is used to treat the following drugs except:
1). Dopamine;
2). Mezaton *;
3). Norepinephrine;
4). Strophanthin *;
5). Lasix.
6. Which of the following is not an indication for intra-aortic balloon counterpulsation:
1). Cardiogenic shock;
2). Severe unstable angina;
3). Severe circulatory failure;
4). Dissecting aneurysm of the aorta;
5). Acute left ventricular failure *.
7. Drug of choice for supraventricular arrhythmia:
1) lidocaine
2) verapamil *
3) nifedipine
4) atropine
8. Thyroid dysfunction, characteristic side-effect:
1) verapamil
2) ethmosine
3) Cordarone *
4) izadrina
9. Initial shock at elektrodefibrillyatsii in adults is:
1) 100 J
2) 200 kJ
3) 200 J *
4) 300 J
10. Hypertensive encephalopathy is characterized by:
1) Vomiting
2) decrease in visual acuity *
3) * seizures

4) hyponatremia
11. To reduce the swelling of the brain are used:
1) Lasix *
2) dexamethasone *
3) Magnesium sulphate
4) nitroglycerin

Answers:
Objective number one
Morgagni syndrome - Adams - Stokes equations. Can be used to relieve pp atropine 1 ml of 0.1% solution in 10 ml fizr-ra in / tively every 3-5 minutes to get the effect or a total dose of 0.04 mg / kg. Without waiting for the effect of temporary pacing is conducted to prevent - to solve the problem of permanent pacing
Objective number two
1.Obezbolivanie
2.Trombolitícheskaya therapy
3. Nitrates.
4.β-blockers
5.Ingibitory ACE
6.Geparin, aspirin
7.Statiny
Task number 3
You can think about having a patient atypical course of myocardial infarction (asthmatic variants), pain in patients with diabetes may be missing subindemnification diabetes and age are not contraindications to thrombolytic therapy. Carefully using beta-blockers (diabetes mellitus, bronchial obstruction).