

Ministry of Healthcare of Ukraine
Poltava State Medical University

**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

Academic discipline

Internal medicine

Module

Emergency conditions in clinic of Internal Medicine

Content module

Emergency conditions in clinic of Internal Medicine

Study subject

Curation of the patient with hypertensive crisis

Curation of the patient with acute heart failure

Course

VI

Faculty

of foreign students training

I. The urgency of the problem

Acute heart failure (AHF) - a condition that is characterized by the rapid appearance of clinical signs, due to decreased contractility of the heart that results in severe hemodynamic disturbances. Knowledge of the main etiological factors, clinical manifestations and the development of mechanisms in each case allows the doctor not only to quickly evaluate the clinical situation, diagnosis, but also time to take emergency measures for the relief of this life-threatening condition.

II. Learning Objectives.

- To analyze the etiological factors and pathogenetic mechanisms of development of SNS.
- Categorize DOS and analyze the clinical manifestations.
- to acquaint students with the survey methods that help determine etiological factors of DOS, the degree of severity.
- To teach students to interpret the results of their own studies.
- Be an individual scheme of diagnostic search, identify and provide the necessary volume and consistency of the methods of examination of patients with AHF.
- conduct differential diagnostics and substantiate the clinical diagnosis.
- To know the principles of first aid, treatment regimen, rehabilitation and prevention.

III. Content of the topic.

DOS, to determine the UCC in 2005 p., Is characterized as a syndrome which includes rapid appearance of symptoms and signs of violation of the functional state of the heart develops in the preceding his illness, or without it (de novo) and manifests as systolic and diastolic dysfunction, heart rhythm disturbances heart, life-threatening and in need of emergency treatment.

There are acute cardiac insufficiency, develop for the first time in patients without prior cardiac dysfunction and decompensation of chronic heart failure.

AHF is very difficult to myocardial infarction, mortality at which up to 80 - 90%.

Etiology. The main factors have GOS (UCC, 2005):

1. decompensation of chronic heart failure due to a variety of diseases of the cardiovascular system.
2. Acute coronary syndromes:
 - Myocardial infarction or unstable angina
 - Complications of myocardial infarction
 - Right ventricular infarction.
3. Hypertensive crisis.
4. Acute cardiac arrhythmias (ventricular tachycardia, ventricular fibrillation, atrial fibrillation, and atrial flutter, supraventricular tachycardia).
5. The valve regurgitation, endocarditis, chord gap, strengthening existing valvular regurgitation.
6. Severe aortic valve stenosis.
7. Acute myocarditis with severe.
8. Cardiac tamponade.
9. Aortic dissection.
10. Postpartum Cardiomyopathy
11. noncardiac factors:
 - Inadequate treatment of patients with cardiovascular disease and chronic heart failure,
 - Overload capacity;

- Infection, especially pneumonia and septicemia,
- Severe cerebral stroke
- Heavy surgery;
- Renal failure
- Asthma
- The use of drugs;
- Alcohol consumption;
- Pheochromocytoma.

12. syndromes that occur with a high cardiac output:

- Septicaemia
- Tirotoksichny crisis;
- Anemia.

Acute and chronic heart failure are identical in pathogenetic mechanisms, clinical manifestations are largely determined by the speed of progression of the pathological process. The probability of acute heart failure is the highest in the case of acute development, for example, a massive myocardial infarction, paroxysmal tachyarrhythmias, with the delay of the inclusion of compensatory mechanisms. OCH occurs when reducing myocardial contractility of the left or right ventricle.

Distinguish following clinical forms GOS:

1. Acute left heart failure: cardiac asthma, pulmonary edema.

To the left of the GOS type can cause a lot of reasons, among which are the most important are: 1) left ventricular dysfunction in ischemic heart disease, hypertension, aortic defects, mitral valve insufficiency, paroxysmal tachycardia (over and ventricular shape), a disease of the heart muscle; 2) dysfunction of the left atrium with a sharp mitral stenosis, myxoma; 3) extracardiac factors to a certain extent conditional (neurogenic pulmonary edema in traumatic brain injury, convulsions, cerebral circulatory disorders iatrogenic complications of infusion therapy, which is carried out without due regard to the water balance, acute renal failure at the stage of hydration).

These reasons cause pulmonary edema, which is defined by a pathological increase in fluid volume which extravasation. The basic mechanism - increasing gradient values of hydrostatic pressure in the pulmonary vessels and interstitial tissue.

2. Acute right heart failure (acute pulmonary heart).

Acute right ventricular ejection is characterized by a decrease in right ventricle with blood stasis and increased pressure on the paths of blood flow to it, that is in the veins of the systemic circulation. Isolated acute right ventricular failure develops in the case of sudden overload of the right ventricle pressure at thromboembolic pulmonary artery branches, and the vast - with hemodynamically significant right ventricular myocardial infarction in patients with myocardial infarction the back (bottom) of the left ventricular wall. The reason for failure can be pravoporedserdnoy right atrial myxoma.

3. Cardiogenic shock (low cardiac output syndrome).

4. Acute total heart failure.

Occurs when the simultaneous destruction of both ventricles (e.g. myocarditis) or left heart, which leads to a stable increase in left atrial pressure, pulmonary hypertension and right ventricular overload resistance and, as a consequence, right ventricular failure.

Decompensation of chronic heart failure is characterized by a sudden onset of symptoms of chronic heart failure, higher functional class, or left ventricular (usually) or right ventricular acute heart failure.

Based on clinical and hemodynamic data release (UCC, 2005):

- Decompensated congestive heart failure (de novo or decompensation of chronic heart failure), which has mild symptoms of this syndrome, but do not meet the criteria for cardiogenic shock, pulmonary edema or hypertensive crisis;
- Hypertensive acute heart failure
- pulmonary edema;
- Cardiogenic shock
- Acute heart failure with a high cardiac output;
- Right ventricular acute heart failure.

Grading of patients with acute heart failure

Killip by T. (1997):

I - shortness of breath, dry inspiratory crackles; stagnant wheezing no, there is no additional IRS tone over the apex of the heart (mortality rate is 2 - 4%).

II - breathlessness, congestion crackles who listen to the minor (less than 50%), lung area, but determined protodiastolic gallop (case fatality rate - 10-15%).

III - choking, wheezing stagnant over much of the lung (50%), signs of pulmonary edema, protodiastolic gallop (case fatality rate - 20-60%).

IV - cardiogenic shock (case fatality rate - 80 - 90%).

Classification according to clinical severity.

Based on the assessment of the peripheral circulation (perfusion) and lung auscultation (stagnation). It provides for the allocation of classes or groups for the characteristics of the patients: Class I (Group A) - "warm and dry", class II (Group B) - "warm and wet", class III (group of L) - "cold and dry" class IV (group C) - "cold and wet". The prognostic value of this classification is proved in patients with cardiomyopathy, it is used to assess the condition of patients with decompensated heart failure in the hospital and on an outpatient basis.

Alveolar pulmonary edema may develop very quickly, but usually it is preceded by a long stage of interstitial edema.

The clinical course distinguish between instant (death occurs after a few minutes), acute (lasting up to 1 hour), prolonged (lasting up to 2 days), and recurrent variants. The latter has a wavy character, most often found in myocardial infarction. Typical clinical signs are severe respiratory distress, coughing up pink frothy sputum, orthopnea, pale, covered with cold sweat skin. There are also peripheral cyanosis. Auscultation determine wet wheezing different caliber of most of the lungs, tachycardia, gallop rhythm protodiastolic and relative systolic murmur of mitral insufficiency: SA may rise or fall sharply (shock). During X-ray of the chest confirmed the presence of wet lung syndrome. In the analysis of blood gas find heavy or moderate hypoxemia, hypercapnia, decreased arterial blood pH (respiratory acidosis).

Diagnostics. Acute heart failure is a life-threatening condition and requires urgent treatment, so the diagnostic measures should be carried out quickly.

1. Evaluation of the clinical condition

Filling RV with decompensated heart failure is usually assessed by measuring the central venous pressure (CVP) in the subclavian, jugular veins and the right atrium. If the internal jugular veins are

difficult to use for this purpose (for example, due to the presence of venous valves) can be carried out external jugular venous catheterization. You must be wary of interpreting the increase in CVP for the corresponding parameters of the Doppler study of the aorta and pulmonary artery. Furthermore, with this study can detect the pressure in the pulmonary artery (in degree tricuspid regurgitation) and thus to monitor left ventricular preload.

2. Electrocardiogram

ECG is quite rare intact. The identification of the etiology can help the study of rhythm and signs of cardiac overload. If there is suspicion of acute coronary syndrome, ECG-registration is required. The ECG can detect signs of increased stress on the LV or RV, atria, symptoms of pericarditis, ventricular hypertrophy. Arrhythmias can also be found during continuous ECG monitoring.

3. Chest X-ray and other imaging techniques

Chest X-ray and other visualization techniques should be performed as soon as possible to all patients with AHF to assess how the heart condition (heart size and contours of shadows), and to address the issue of the presence of congestion in the lungs. It is also used to confirm the diagnosis, monitoring or improving the contrary, refractory to therapy. Radiography may be needed, provided the differential diagnosis of left ventricular failure and bronchopulmonary infections, and other conditions. Computed tomography of the chest using both contrast angiography, and without it is necessary to identify the pulmonary pathology and diagnosis of pulmonary embolism. If there is suspicion of an aortic aneurysm rasslayuvayuschuyu require computed tomography and transesophageal echocardiography.

4. Laboratory examination

Research arterial blood gas analysis (Astrup on) provides a measure of oxygenation (pO_2), respiratory function (pCO_2), and acid-base balance (pH) and its violations. Thus, this study is recommended for all patients with severe heart failure. Not invasive research methods: pulse oximetry and determination of CO_2 in exhaled air may replace the study by Astrup, but not for the state, accompanied by a small release of vasoconstrictor or shock.

Determination of brain natriuretic peptide (BNP) in the blood plasma (a substance that is produced in the ventricles of the heart by mechanical stretching of its walls and volume overload) is used to exclude or confirm the presence of heart failure patients admitted to hospital with complaints of shortness of breath. Limit values up to 300 pg / mL for NT-proBNP and 100 pg / mL for BNP, however, the determination of these limits to research almost did not include elderly patients. During the development of pulmonary edema value BNP until hospitalization may be normal. On the other hand, BNP has a high negative predictive value for the deletion of CH. Various clinical conditions may affect the BNP concentration, such as renal failure and septicemia. When detected elevated concentrations, requires further diagnostic tests. If confirmed by the GOS, elevated plasma BNP and NT-proBNP provides important prognostic information.

5. Echocardiography

Echocardiography is indispensable to identify the structural and functional changes caused by DOS, as well as the presence of acute coronary syndrome. Echocardiography with Doppler study should be used to explore the monitoring of systolic and diastolic left ventricular function, and PN, the structure and function of the heart valves, pericardium identify possible pathology, mechanical complications of acute myocardial infarction and injury. Cardiac output is determined using the relevant parameters of the Doppler study on the aorta and pulmonary artery. Furthermore, with this

study can detect the pressure in the pulmonary artery (in degree tricuspid regurgitation) and thus to monitor left ventricular preload.

6. Other methods of investigation

In the presence of coronary artery disease, such as unstable angina or myocardial infarction, angiography is important and determine the need for revascularization procedures, conduct which significantly improves the prognosis.

Common approaches to the treatment of patients with AHF

Addressing the causes of acute heart failure in individual patients is the most important in its treatment. Consideration should be given the opportunity to address such diseases and conditions:

- Tachy or bradycardia if they are the causes of congestive heart failure, or reinforce it;
- Coronary artery occlusion with the development of acute coronary syndrome,
- Violation of intracardiac hemodynamics due to valvular defects, defects of the atrial or ventricular septal, etc.,...
- Hypertensive crisis;
- Cardiac tamponade.

Drug treatment should be comprehensive and aimed at:

- 1) reducing the hydrostatic pressure in the vessels of the pulmonary circulation and decrease venous return to the right ventricle;
- 2) a decrease in BCC and dehydration of the lungs;
- 3) decrease in the permeability of the alveolar-capillary membrane;
- 4) enhancement of myocardial contractility
- 5) elimination of pain and acute cardiac arrhythmias and conduction
- 6) the fight against hypoxia and disorders of acid-base balance and water-electrolyte metabolism;
- 7) elimination of bronchospasm and improve alveolar ventilation.

DOS leads to progressive deterioration of oxygenation of the blood in the lungs, arterial hypoxemia and hypoxia peripheral tissues. The simplest method of combating this disease is a manifestation of **breath 100% oxygen at a high speed of its feed (8-15 liters / min)** to maintain the arterial oxygen saturation greater than 90%.

An effective means of increasing myocardial contractility and cardiac output except sympathomimetic amines is **intraaortic balloon pump**, which, in contrast to inotropic agents, does not increase myocardial oxygen demand without suppresses myocardial contractility and decreases AT (some drugs used for the removal of ischemia infarction or reduction in afterload). Aortic balloon counterpulsation is contraindicated in aortic regurgitation, aortic dissection and severe peripheral atherosclerosis.

Positive inotropic agents used temporarily to increase myocardial contractility. It should be borne in mind that their action is often accompanied by increased myocardial oxygen demand.

Pressor (sympathomimetic) amines (noradrenaline, dopamine and dobutamine). Treatment is usually started with low doses that are gradually increased if necessary (titrated) to optimum effect. Mainly for the selection of a dose it is advisable to carry out invasive monitoring of hemodynamic parameters with determination of cardiac output and pulmonary artery wedge pressure. A common drawback of the drugs in this group is the ability to induce or increase tachycardia (bradycardia or using norepinephrine), cardiac arrhythmias, myocardial ischemia.

Norepinephrine is peripheral vasoconstriction (including abdominal and arteriolar vessels in the kidneys) due to stimulation of α -adrenoceptors. It is indicated for patients with severe hypotension

(systolic AT lower than 70 mm Hg. Art.), In the case of a low peripheral vascular resistance. The initial dose of norepinephrine is 0.5-1 g / min; it further titrated to achieve an effect in the case of refractory shock may be 8-30 g / min.

Dopamine stimulates α - and (3-adrenergic receptors, and receptors dopaminergichnye contained in the kidneys and mesenteric vessels. When infusion at a dose of 2-4 mg / kg in 1 min produces predominantly dopaminergiyne effect on receptors that leads to the expansion of abdominal vessels and arterioles kidney. This increases the rate of diuresis and diuretic overcome refractoriness caused by the reduced renal perfusion. in doses of 5-10 mg / kg in 1 min predominantly stimulates dopamine β adrenergic receptors, helps to increase cardiac output, and in doses of 10-20 mg / kg 1 min predominant stimulation of α -adrenergic receptors with peripheral vasoconstriction Dopamine is used to increase myocardial contractility, the relief of arterial hypotension, and (rarely) -. to increase the heart rate in patients with bradycardia that requires correction.

Dobutamine - a synthetic catecholamine, stimulates predominantly (and-addresses noretseptory This leads to improved myocardial contractility and decreased peripheral vascular resistance, and the drug is contraindicated in hypotension usually used doses of 5-20 mg / kg in 1 minute Dobutamine can be combined... with dopamine. Dobutamine is able to reduce pulmonary vascular resistance and is the drug of choice in the treatment of right ventricular failure.

Appointment of **digoxin** for infusion is indicated only for patients with severe decompensated heart failure in the presence of atrial fibrillation tahisistolicheskoy. Strofantin and Korglikon, which are widely used previously, are not recommended in clinical practice.

Means of enhancing the sensitivity of contractile proteins to calcium cardiomyocytes. The only representative of this class, the safety and efficacy has been proven in a number of multicenter studies is **levosimendan**. Its positive inotropic effect is not accompanied by an increase in myocardial oxygen demand and increased sympathetic effects on the myocardium. In addition, levosimendan produces a vazodilativnomu and antiischemic effects due to activation of potassium channels. 24-hour infusion of the drug (loading dose of 24-36 mg / kg followed by infusion at a dose of 0.4-0.6 mg / kg in 1 min) leads to hemodynamic and symptomatic improvement in acute heart failure and prevents repeated episodes in chronic decompensated heart failure.

Peripheral vasodilators rapidly decrease pre- and afterload due veins and arterioles, leading to a decrease in pulmonary capillary pressure, decreased peripheral vascular resistance and AT. they can not be used in acute heart failure occurs with hypotension.

Nitroglycerin effect develops after 1-2 minutes and can last up to 30 minutes. In cardiac pulmonary edema is the fastest and most affordable way to reduce acute symptoms syndrome - if AT higher than 100 mm Hg. v., nitroglycerin infusion usually begin with 10-20 mg / m and increased by 5.10 mg / min every 5-10 minutes to obtain the desired hemodynamic and / or clinical effect. Nitrates are effective in myocardial ischemia, urgent situations that have arisen in connection with hypertension or decompensated heart failure (such as mitral and aortic regurgitation).

A new class of vasodilators is **naseretid** - recombinant human brain peptide identical to an endogenous hormone produced in response to increased wall tension or volume overload hypertrophy. Naseretid has properties venous, arterial and coronary vasodilator, reduces pre- and afterload of the left ventricle and increases cardiac output and without directly positive inotropic effect.Reduced hydrostatic pressure in the pulmonary vessels and decrease venous return to the heart is carried by some drugs. In particular, they include **narcotic analgesics, and antipsychotics**

that are α -adrenoceptor blocking properties - morphine hydrochloride, droperidol, fentanyl, haloperidol.

Morphine hydrochloride - by vials 1 ml of 1% solution. Gives an analgesic effect, it depresses the respiratory center, reduces shortness of breath, stimulates the vagus nerve center with the development of bradycardia. It reduces fear of death, eliminating anxiety. It lowers blood pressure and systemic venous return to the heart. Apply a slow intravenous administration of 1 ml of a 1% solution of morphine hydrochloride. In less extreme cases the same dose administered intramuscularly. Contraindications to the appointment of the drug: severe airway obstruction, chronic pulmonary heart, pregnancy. It is better to enter opioids intravenously at 1:20 in isotonic sodium chloride solution - for 2 to 3 ml every 30-60 minutes. Except Morphine analgesic, sedative, and an increase in vagal tone has properties peripheral venodilatators. It is the drug of choice for the relief of pulmonary edema and removal of chest pain associated with myocardial ischemia. Intravenously administered small doses (3-5 mg every 5 minutes to achieve the effect).

Promedol - 1 ml ampoule of 1% or 2% solution. Also administered as morphine hydrochloride and its effect is much weaker than morphine hydrochloride.

Fentanyl - ampules of 2 ml and 10 ml of 0.005% solution. It gives a strong, quick, but short-term analgesic effect (duration 15 - 30 minutes). It inhibits the respiratory center, causing bradycardia (atropine sulfate eliminate it, though, the latest in pulmonary edema should be carefully). Inject 1 - 2 ml of 0.005% solution of fentanyl intravenously or intramuscularly in combination with neuroleptics - droperidol (2 - 4 ml of 0.25% solution) or haloperidol (1 - 2 ml of 0.5% solution), or using a combined preparation Talamonal (in 1 ml containing 2.5 mg of droperidol and fentanyl 0.05 mg).

Droperidol - 10 ml ampoules of 0.25% solution. It belongs to the group of neuroleptics. The action is fast, strong, but short. It has anti-shock and protiblyuvotpi properties, lowers blood pressure, has an antiarrhythmic effect.

Ganglioplegic used to expand the peripheral blood vessels and the deposition on the periphery, resulting in reduced venous return to the right ventricle. Used under elevated or normal blood pressure is not used in the case of arterial hypotension. Pentamin 1 ml of 5% solution diluted in 20 ml isotonic sodium chloride solution, and titration is administered intravenously, that is 2 to 3 ml of the diluted formulation every 2 - 3 minutes under continuous blood pressure control. After reaching the desired effect introduction kentalmina stop.

Nitrates - nitroglycerin perlinganit, izoket - increase the capacity of the veins, reduce venous pressure, reducing venous return to the heart. Nitrates directly relax vascular smooth muscle. Vazodilagatsiya occur 2 minutes after the start of infusion.

ACE inhibitors provide vasodilation by inhibiting angiotensin II-induced vasoconstriction, aldosterone production inhibitory reduce left ventricular filling pressure, right atrial pressure, cardiac output increased with little effect on HR.

Typically used captopril or enalapril or fozinolril. Captopril action begins 30 min after oral administration of 12.5 mg. After 4 - 6 hours into the patient give 25 mg every 6 - 8 hours. Enalapril is first administered orally at a dose of 2.5 mg maintenance dose is 10 - 20 mg per day. These drugs are limited to use for the treatment of emergency conditions due to lack of injectables, they are used in the further treatment

Calcium antagonists of the dihydropyridine group (nifedinin) are contraindicated in myocardial infarction through activation of the sympathetic-adrenal system with increased rates of mortality.

Reduction and elimination of dehydration BCC lung mainly engage **diuretics**. At high or normal blood pressure use potent loop diuretics - furosemide (Lasix), ethacrynic acid (Uregei). their effect is due to inhibition of reabsorption number + and water in the loop of Henle, a decrease in extracellular fluid volume, cardiac output, a decrease in response to the impact of presynthetic angiotensin II and norepinephrine. Furosemide (Lasix) is administered intravenously at a dose of 20 - 60 - 120 mg. The clinical effect was observed after 3 - 5 minutes. Re-drug is administered, if necessary, after 2 - 4 hours, Furosemide is contraindicated in cases of hypotension, hypovolemia, anemia, acute or chronic renal insufficiency with sharp decrease in glomerular filtration rate.

Wiring impose on a limb for the deposit of the BCC at the periphery. When properly tourniquet to each leg may be delayed 600-800 ml of blood and more. After elimination of alveolar pulmonary edema harnesses should slowly weaken, to avoid rapid admission into the bloodstream at the same time a significant amount of blood.

In order to reduce the permeability of the alveolar-capillary membrane used antihistamines and corticosteroids. Diphenhydramine are administered in a dose of 10 - 20 mg (for polarizing the mixture) or by intramuscular injection.

Hydrocortisone 150-300 mg introduced into 200 ml of isotonic sodium chloride intravenous infusion, prednisolone at a dose of 60-120 mg is administered in 100 ml of isotonic sodium chloride solution or 5% glucose solution intravenously.

In resistant cases, a very low ejection fraction (<20%):

1. 3 order to enhance myocardial contractility with great caution should appoint cardiac glycosides (general condition monitoring, ECG, heart rate) and non-glycoside inotropic agents. With cardiac glycosides, preference is given strofantinu. Typically strofantin administered 0,250-0,375 mg dose (0.5 - 0.75 ml of a 0.05% solution) intravenously slowly over 5 min. It is best to introduce the dose infusion over 10 - 15 min. If necessary, may be added at intervals of 1 hour, 0.1 -0.125 mg (0.2 - 0.25 ml of 0.05% solution) of the drug until the clinical effect (heart rate slowing, reducing shortness of breath). The total daily dose for 4 hours should be not more than 0.5 - 0.625 mg (1 - 1.25 ml of a 0.05% solution). It should be remembered that in acute myocardial infarction tolerance of cardiac glycosides reduced. The drug is not recommended in patients receiving cardiac glycosides, calcium salts, as the accelerated development digitalis intoxication with the occurrence of a fatal ventricular fibrillation.

2. neglikozidnye inotropic agents may be used dobutamine (Korotrop), which is available in bottles of 250 mg. This amount is diluted in 250 ml of isotonic sodium chloride solution and injected at the rate of 0.5 - 20 mg (kg * min), starting from a speed of 1 drop for 1 min, gradually increase the frequency drops to 28 in 1 minute (risk of tachycardia, arrhythmia , blood pressure fluctuations, increased myocardial ischemia).

Eliminating pain and acute cardiac arrhythmias and conduction carried out individually, depending on the specific situation.

To combat hypoxia and disorders of acid-base balance and water-electrolyte balance before establishing oxygen therapy or, better, the introduction of oxygenated perflorana to correct severe hypoxemia, decrease the permeability of the lung membrane. The best method of oxygen therapy is inhalation of oxygen through the tubes installed in the nasal passage to a depth of 7 - 10 cm, with oxygen fed through them in a volume of 8-10 liters per 1 min. To ensure the access of oxygen to the lungs is an urgent need to restore patency of the upper airway. To conduct this foam aspiration of the upper respiratory tract and trachea by means of mechanical, electrical or other aspirators.

Aspiration of foam and liquid from the trachea is sometimes possible only after tracheal intubation or tracheostomy overlay. foam aspiration from bronchi medium caliber possible. In order to use special quenching surfactants - so-called antifoaming agents that are administered by inhalation (ethyl alcohol antifomsilan). Ethyl alcohol (70 - 96% solution) was poured into the water instead of a humectant, alcohol vapor contributes to the destruction of foam in the bronchi. Sometimes ethanol (5 ml of 100% alcohol + 15 ml of 5% glucose solution) 2 ml 1 is administered directly into the trachea or intravenously (20 - 30 ml).

Antifomsilan - 0.6-1 ml of 10% alcohol solution - applied in the form of inhalation solution after the preliminary dispersion (eg sprayers Gorski). The clinical effect is primarily a decrease in the number of wheezing. Considerable importance is the correction of acid-base status - 5% sodium bicarbonate solution is injected intravenously (100-150 ml).

Acute right heart failure (acute pulmonary heart). The cause of acute failure right heart in myocardial infarction can be a **pulmonary artery or its branches (PE, TEVLA)**, which in turn can be caused by atrial fibrillation, thrombophlebitis of the lower limbs, pelvis, increase blood clotting, circulatory insufficiency, especially in obese patients, the elderly.

In the case of pulmonary artery comes instant death.

Cardiogenic shock (low cardiac output syndrome).

Cardiogenic shock - this is one of the most common and serious complications of myocardial infarction. Mortality in this cardiogenic shock is 80-90%.

It develops necrosis more than 35-40% of left ventricular muscle mass. Cardiogenic shock occurs most often during the first days as a result of left ventricular systolic dysfunction as a result of extensive ischemia or necrosis. Enough is rarely the cause is a gap mezhzheludochnoy walls or mitral insufficiency. This clinical syndrome is characterized by hypoperfusion of organs and hypotension. Prolonged hypoperfusion violates the function of many organs. Renal insufficiency manifested a decrease in urine output (less than 20 ml / h), cerebral hypoperfusion - lethargy and drowsiness or psychomotor agitation.

Criteria for diagnosis of true cardiogenic shock:

- 1) systolic AT of less than 80 mm Hg with a significant reduction in pulse pressure. When pre-hypertension it may occur with the systolic AT 90-100 mm Hg.;
- 2) oliguria or anuria (urine output of less than 20 ml / h);
- 3) tachycardia, supraventricular or ventricular tachyarrhythmias;
- 4) cold clammy sweat, pale skin, confusion, dizziness.

There are four clinical forms of cardiogenic shock (for EI, Chazova):

- 1.reflektorny (early, pain)
2. This,
- 3.aritmichny (tahisistolicheskoy, bradisistolicheskoy)
- 4.areaktivny.

When the reflex form of cardiogenic shock decline in cardiac output due to less extensive necrosis as reflex influences of the lesion on the heart and vascular tone (typical sinus bradycardia and decreased blood pressure). Arrhythmic form of cardiogenic shock due to a sharp decrease in cardiac output due tachy or bradyarrhythmias.

This cardiogenic shock is the result of weight reduction function of the left ventricle. Sometimes the present cardiogenic shock may develop even with small focal myocardial infarction. In such cases, there is a functional deficiency of the intact myocardium (diffuse cardio).

Due to reduction of myocardial contractility is sharply reduced cardiac output (stroke volume and cardiac output), in response to that the increased total peripheral resistance (vasoconstriction is less pronounced than the decrease in cardiac output). The decrease in cardiac output and blood pressure lowering with peripheral vasoconstriction adversely affect organ and tissue microcirculation. Reduced blood circulation in vital organs, which can result in necrosis in the liver, stomach and intestinal ulcers (sometimes with fatal bleeding). The sharp decrease in the filtration of kidney function leading to azotemia. Increased blood viscosity, increased aggregation of blood cells are formed multiple microthrombuses. Coronary blood flow is not sufficient to meet the metabolic needs of even the preserved areas of the myocardium, causing further deterioration of contractile ability of the heart. As a result of acidosis, hypoxia develops generalizovanaya vasodilation, increases the capacity of the vascular bed, develops sludge syndrome. As a result, circulation of centralization (for a satisfactory function of the right ventricle) wet lung syndrome occurs and then alveolar pulmonary edema. Sometimes, however, it decreases venous return (blood deposition pas periphery) and then on a background of dry lung progresses low cardiac output syndrome.

There are three degrees of the cardiogenic shock (C. Blizzard, 1987):

I degree - a relatively light, shock duration is 3 - 5 hours, BP - 90/50 - 60/40 mm Hg. Art., fast and stable response to the administration of pressor amines (kordiamin).

II degree - moderate severity, duration of 5 - 10 hours, the indicators AB -80/50 - 40/20 mm Hg. Art., signs of acute left ventricular failure (cardiac asthma), oliguria, the pressor response to the introduction of drugs (dopamine) slow and unstable.

Grade III - a very hard, more than 10 hours duration, systolic blood pressure less than 20 mm Hg. Art., pulse pressure less than 15 mm Hg. Art., is the alveolar pulmonary edema, anuria, pressor responses to medications (dopamine, glucocorticoids, angibtensinamid) short-term and unstable.

Emergency assistance in cardiogenic shock include:

- 1) improvement of central and peripheral hemodynamics
- 2) conducting antianginal therapy;
- 3) correction of cardiac arrhythmias and conduction
- 4) the use of intra-aortic balloon counterpulsation.

In cases of low CVP is expedient to hold such events.

Adrenomimetickie means (intravenously)

- Dopamine - 0.5 - 20 mg / (kg • min), or 5 ml of a 4% solution (200 mg) in 250 ml of isotonic sodium chloride solution 36 droplets at a frequency of 1 min, and 40 ml of a 0.5% solution of (200 mg) in 210 ml of isotonic sodium chloride solution 36 droplets at a frequency of 1 min; adrenaline hydrochloride - 4 ml of a 0.1% solution in 250 ml of isotonic sodium chloride solution at a frequency of 2 to 9 drops of 1 min;

- Dobutamine (Korotrop) - 250 mg per 250 ml of isotonic sodium chloride solution at a frequency of 1 to 28 drops per 1 min, isoproterenol (izadrin, alupent) - 5 ml of a 0.05% solution in 500 ml of 5% glucose solution at a frequency of 2 to 40 drops per 1 min;

- Norepinephrine tartrate - 2 - 4 ml (4 - 8 mg), 0.2% solution in 1000 ml of 5% glucose solution at 20 - 30 drops of 1 min.

A low CVP is recommended to introduce the low molecular weight dextran: reopoligljukin, reoglyuman, reomakrodeks, reotran - 250-500 ml intravenously at a frequency of 20-30 drops in 1 min and glucocorticosteroids (iv): prednisolone - 60-120 mg hydrocortisone - 250 mg every 2 4 hours to stabilize blood pressure indicators.

Applied antiplatelet agents (inside) aspirin (325 mg daily) or tiklid (500 mg per day) or clopidogrel (150 mg daily).

Sodium bicarbonate is introduced to improve hemostasis (intravenously) at the rate of:

8.4% solution (mmol / L) - $VE \cdot 0.3 \cdot \text{body weight}$;

5% solution (mmol / l) = $BE \cdot \text{body weight} \cdot 0.5$;

4.2% solution (mmol / l) = $BE \cdot \text{Body weight} \cdot 0.2$ 2 wherein EE - base deficit or excess acids.

In case of cardiogenic shock with increased CVP used: Droperidol (2 - 5 ml of 0.25% solution in 250 ml of 5% glucose solution for infusion days), nitroglycerin, izoket.

Among other methods of treatment of cardiogenic shock is worthy way of intra-aortic balloon counterpulsation. This method consists in introducing the balloon into the abdominal aorta through the skin by hydrochloric femoral artery puncture. Electronic device inflates the balloon during diastole of the heart, thus increasing diastolic pressure, which in turn maintains the pressure required for adequate coronary perfusion. The balloon deflates rapidly in late diastole, which leads to a decrease afterload and improves cardiac systole of the left ventricle (increased stroke volume of the heart). However, when using this method, every third patient develops complications, ischemia of the lower limbs, internal organs, dissection, hemolysis, infectious processes, balloon rupture. Develop methods of foreign pnevmokontropulsatsii.

Treatment of patients with cardiogenic shock reflex form consists of adequate analgesia, the use of short-pressor amines (kordiamin, mezaton) and elimination of sinus bradycardia (small doses of atropine sulfate).

Treatment arrhythmic forms of cardiogenic shock is carried out on the principles of antiarrhythmic therapy.

The diagnosis of cardiogenic shock unresponsiveness retrospectively adjusted with no effect on the administration of norepinephrine tartrate, dopamine, GCS.

Diseases of the heart, which is the cause of AHF and require surgical intervention.

1. cardiogenic shock after acute myocardial infarction, with the defeat of several coronary vessels.
2. Postinfarction ventricular septal defect
3. ventricular wall rupture.
4. Acute decompensation of existing valvular pathology.
5. Lack of or an artificial heart valve thrombosis.
6. Aortic aneurysm, or dissecting aortic aneurysm in the pericardial cavity.
7. Acute mitral regurgitation due to:
 - Ischemic papillary muscle rupture;
 - Coronary dysfunction papillary muscle;
 - Myxomatous chordae rupture of the tendon;
 - Endocarditis;
 - Injury.
8. Acute aortic regurgitation due to:
 - Endocarditis;
 - Dissecting aortic aneurysm
 - Closed chest injuries;
 - Burst Aneurysm sinus of Valsalva.
9. Acute decompensation of chronic cardiomyopathy.
10. Mechanical assistive devices and heart transplantation

Heart transplantation can be considered as a therapeutic activity in the presence of severe AHF with a known poor prognosis. An example is the severe acute myocarditis, postpartum cardiomyopathy or myocardial infarction with poor prognosis after revascularization. However, a heart transplant is not possible as long as the state is not achieved stabilization - in a natural way or circulatory support.

Conclusions:

The clinical AHF syndrome may be presented as DOS, which developed de novo or as decompensated CHF with predominantly left ventricular or right ventricular insufficiency. A patient with AHF requires urgent diagnosis and treatment, and sometimes resuscitation.

Initial examination should include a medical history, ECG, X-ray of the chest cavity, the determination of plasma BNP and NT-proBNP (if possible), and other laboratory tests. If possible, all patients need to spend an echocardiography.

Clinically important is to define the parameters of preload, afterload, the presence of mitral regurgitation and other complications (valvular disease, arrhythmias), concomitant diseases such as infections, diabetes, diseases of the respiratory or urinary. The most frequent cause of AHF is an acute coronary syndrome, in which you need to coronary angiography.

After the initial examination, it is necessary to establish reliable access to the vein and start monitoring of physiological parameters, ECG parameters, oxygen saturation. If necessary, set the arterial catheter.

The initial treatment of AHF

- Oxygen therapy using a mask or POS mode (target oxygen saturation of 94-96%).
- Vasodilatation with nitrate or sodium nitroprusside.
- Diuretic therapy furosemide or other loop diuretic (intravenous bolus followed by continuous infusion).
- Morphine for relief of physical and mental stress and arousal, as well as to improve hemodynamics.

Intravenous infusion volume is needed to increase the preload or filling in the presence of low pressure if necessary. Pre sometimes requires water load test.

- The treatment of metabolic and organ damage.
- In the case of acute coronary syndrome and other complications of heart disease is necessary to cardiac catheterization and angiography to study the question of surgical intervention.
- Relevant pharmacotherapy using b-blockers and other preparations based on these recommendations in these statements. Other specific therapy based on clinical and hemodynamic characteristics of the patient in the absence of clinical response to initial treatment. This use of inotropic agents or a calcium sensitizatorov in severe decompensated heart failure, or inotropic agents in the case of cardiogenic shock.

The goal of therapy is to correct hypoxia, DOS, and an increase in cardiac output, renal perfusion, sodium excretion, increased diuresis. May recommend other drugs, such as infusion aminophylline or b2-agonists for bronchodilation. Ultrafiltration or hemodialysis can be used for refractory heart failure. Patients with refractory AHF or end-stage heart failure require further support: intraaortic balloon counterpulsation, mechanical ventilation, circulatory support devices as temporary measure and as a "bridge" to heart transplantation.

IV. Means of self-control:

Tests:

1. Patient V., 64r., Complaints constricting pain in the chest that lasts more than 1 hour, feeling short of breath, wheezing. On examination revealed: Dribnpuhirtsevi moist rales over 2/3 of the lung, 3 heart sound, heart rate 90 / min, blood pressure 150 / 90mm Hg. Art. ECG: sinus rhythm, right, a sharp rise in the ST segment of the contour line in leads V1-V5. Acute condition occurs in a patient?

- A. Acute myocardial infarction, cardiogenic shock
- B. Acute myocardial infarction, myocardial free wall rupture
- C. Acute myocardial infarction, pulmonary edema
- D. PATE
- E. E. Pulmonary edema on the background of mitral stenosis.

2. Patient B., 64r., Complaints constricting pain in the chest that lasts more than 1 hour, feeling short of breath, wheezing. On examination revealed: Dribnpuhirtsevi moist rales over 2/3 of the lung, 3 heart sound, heart rate 90 / min, blood pressure 150 / 90mm Hg. Art. ECG: sinus rhythm, right, a sharp rise on the contour line ST in leads V1-V5.

What measures of emergency medical care to the patient must perform?

- A. The situation with raised lower limbs, administration of cardiac glycosides, furosemide.
- B. Oxygen therapy, morphine, furosemide, nitroglycerin.
- C. The situation of half-sitting, morphine, aminophylline, cardiac glycosides.
- D. Provisions half-sitting, administration of furosemide, aminophylline, dexamethasone.
- E. E. Oxygen therapy, infusion of pressor amines.

3. Patient C, 55p. with acute myocardial infarction during the physical examination revealed: the patient is braked, the skin moist and cold, "Gray cyanosis" pulse - 128 / min, rhythmic, thready, BP 70/40 mm, heart sounds dull, breath in the lungs vesicular on. catheter was allocated 30 ml of urine. What has become more complicated for myocardial infarction in a patient?

- A. cardiogenic shock
- B. Pulmonary Edema
- C. hypovolemic shock
- D. PATE
- E. E. Acute left ventricular aneurysm

4. Patient C, 55p. with acute myocardial infarction during the physical examination revealed: the patient is braked, the skin moist and cold, "Gray cyanosis" pulse - 128 / min., rhythmic, thready, BP 70/40 mm, heart sounds dull, breath in the lungs vesicular by catheter was allocated 30 ml of urine. Vvedennnya a drug shown to the patient?

- A. Dopamine
- B. prednisolone
- C. Furosemide
- D. atropine
- E. Caffeine

5. Patient C, 55p. with acute myocardial infarction during the physical examination revealed: the patient is braked, the skin moist and cold, "Gray cyanosis" pulse - 128 / min, rhythmic, thready, BP 70/40 mm, heart sounds dull, breath in the lungs vesicular on. catheter was allocated 30 ml of urine. Diagnosed cardiogenic shock. Invasive technique can be applied in this state?

- A. intraaortic balloon counterpulsation.
- B. Implantation of an artificial pacemaker.
- C. Implantation cardioverter defibrillator.
- D. Statement cava filter.
- E. Pericardial puncture.

Recommended literature:

I. Main:

1. Internal Medicine: in 2 books. Book 1. Diseases of the Cardiovascular and Respiratory Systems: textbook / N.M. Seredyuk, I.P. Vakaliuk, R.I. Yatsyshyn et al. Київ, Медицина., 2019. - 664 + 48 кольор. вкл.).
2. Internal medicine: Part 1 (cardiology, rheumatology, haematology): textbook for English-speaking students of higher medical schools / edited by Professor M.A. Stanislavchuk and Professor V.A. Serkova. - Vinnytsia: Nova Knyha, 2019. - 392 p.
3. Медицина за Девідсоном: принципи і практика / Навчальний посібник: пер. 23-го англ. вид.: у 3 т. Т.3 С. Ралстона, Я. Пенмана, М. Стрекена, Р. Гобсона; К.: ВСВ «Медицина», 2021. – 642 с.
4. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)/
5. Побічна дія ліків – Side Effects of Medications: навчальний посібник 2 т. / за заг. ред. В.М. Бобирьова, М.М. Потяженка. – Вінниця:
6. Cardiovascular diseases. Classification, standards of diagnosis and treatment / Edited by Academician Kovalenko V.M., Prof. Lutaia M.I., Prof. Sirenko Yu.M., Prof. Sychova O.S. – Kyiv. – 2020.
7. Perederii V.H., Tkach S.M. Principles of internal medicine. – Vol.2 / Textbook for students of higher educational institutions. – Vinnytsia: Nova knyha. – 2018.
8. Internal diseases. The textbook based on the principles of evidentiary medicine, 2018.

II. Additional literature:

1. Recommendations of the Association of Cardiologists of Ukraine for the diagnosis and treatment of chronic heart failure / Voronkov L.H. – moderator, working group of the Ukrainian Association of Heart Failure Specialists. – 2017.
2. Respiratory diseases / Ghanei M. - In Tech, 2012. - 242 p.
3. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
4. Principles and practice of interventional pulmonology / Ernst A., Herth F. -Springer, 2012. - 757 p.
5. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
6. Petrov Y. The chief symptoms and syndromes in patients with cardiovascular pathology : The practical handbook for medical students / Ye. Petrov, Yu. Goldenberg, N. Chekalina; UMSA. - Poltava : TexcepBic, 2010. - 143 .
7. Gastroenterology and Hepatology Board Review: Pearls of Wisdom, Third Edition (Pearls of Wisdom Medicine) by John K. DiBaise (May 11, 2012)

8. Clinical Pulmonology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Oct 30, 2011) - Kindle eBook
9. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Hematology: Clinical Principles and Applications, 4e by Bernadette F. Rodak MS MLS (Feb 18, 2017)
12. Rheumatology, 2-Volume Set: EXPERT CONSULT - ENHANCED ONLINE FEATURES AND PRINT, 5e by Marc C. Hochberg MD MPH, Alan J. Silman MD, Josef S. Smolen MD and Michael E. Weinblatt MD (Oct 19, 2019)
13. Endocrine Pathology: Differential Diagnosis and Molecular Advances by Ricardo V. Lloyd (Nov 5, 2018)
14. Clinical Endocrinology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2017) - Kindle eBook
15. Williams Textbook of Endocrinology: Expert Consult-Online and Print, 12e by Shlomo Melmed, Kenneth S. Polonsky MD, P. Reed MD Larsen and Henry M. Kronenberg MD (May 27, 2016)
16. Electrocardiography, 3e with Student CD (Booth, Electrocardiography for Health Care Personnel) by Kathryn A. Booth (Jan 27, 2017)
17. Echocardiography Review Guide: Companion to the Textbook of Clinical Echocardiography: Expert Consult: Online and Print, 2e (Expert Consult Title: Online + Print) by Catherine M. Otto (Mar 7, 2017).

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