

**MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

"Approved"
at the meeting of internal
medicine №1 department
Head of Department
Prof. Skrypnyk I.M.

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

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|----------------------------|---|
| <i>Academic discipline</i> | Internal medicine |
| <i>Module</i> | Emergency conditions in clinic of Internal Medicine |
| <i>Content module</i> | Emergency conditions in clinic of Internal Medicine |
| <i>Study subject</i> | Curation of the patient with pulmonary embolism Curation of the patient with acute respiratory failure |
| <i>Course</i> | VI |
| <i>Faculty</i> | of foreign students training |

Poltava 2016.

Background of I

Thromboembolic complications are the actual problem of modern medicine, as a leader in the structure of mortality and lead to serious long-term consequences.

PE is the third most common cause of death of people from cardiovascular disease (CVD) after myocardial infarction (MI) and stroke. Every year from pulmonary embolism die 0.1% of the world population, and the mortality rate of patients Wednesday, did not receive antithrombotic therapy, up to 30-40%. Massive pulmonary embolism end lethal in 70% of cases, death usually occurs within the first two hours. In life, the diagnosis of pulmonary embolism is set less than 70% of cases, and overdiagnosis occurs in 65% of cases.

II Learning Objectives:

- [Learn how to identify and diagnose thrombotic pulmonary embolism \(PE\)](#)
- [Learn how to identify and prevent pulmonary embolism, etc. Ichin](#)
- Understand [Pathogenesis of pulmonary embolism](#)
- To learn the basic aspects of [the treatment I PE](#) , p assmotret medicines used during treatment
- [Prevention of pulmonary embolism](#)

III. Content of the topic.

Pulmonary embolism (PE) - a blockage (occlusion) of the arterial bed of light (the trunk, the right or left pulmonary artery and / or their branches) thrombotic masses of different caliber, formed in the veins of the systemic circulation (deep vein thrombosis (DVT) legs and ileo -kavalnogo segment of the pelvis, that is in the basin of the inferior vena cava, rarely - in the pool of the superior vena cava), at least - in the right atrium or the right ventricle of the heart. As a result, developing branches of the pulmonary artery spasm, acute pulmonary heart, reducing cardiac output, decreased blood oxygenation and bronchospasm.

The main risk factors for pulmonary embolism:

- DVT, as well as a great saphenous vein and the veins of the pelvis;
- Forced chained to a bed for various reasons: large surgeries, severe cardiovascular diseases, lung infections, paralysis due to stroke or other disabling diseases;
- Surgical operations on the pelvic organs, the abdomen and lower extremities. Obesity and age also increases the risk;
- Diseases of the lower limbs: plastered fractures, varicose veins. Thrombosis distal deep vein can cause even the tight bandaging of the knee;
- Malignant tumors of the organs of the abdomen, pelvis, and other sites during metastasis;
- Obesity;
- Pregnancy, early postpartum and surgical delivery;
- DVT and pulmonary embolism in history;
- Thrombophilia (the effect of oral contraceptives, hereditary forms of resistance to activated protein C, hyperhomocysteinemia, antiphospholipid syndrome);

Small risk factors:

- Long-term air travel;
- Oral contraceptives or hormone replacement therapy with female hormones.

Classification

In the classification of the following criteria are used PE: the level of embolic occlusion, degree of violation of lung perfusion, the flow speed of the process, the nature of hemodynamic disorders. M. Ferstrate, modifying the existing classification, identified four degrees of pulmonary embolism:

Grade 1 (mild) - thromboembolism of small branches;

Grade II (moderate severity) - the defeat of the segmental branches;

Grade III (severe) - blockage of the pulmonary artery branches equity;

IVstepen (extremely hard) - a massive, developed with obstruction of the pulmonary artery trunk and its bifurcation (saddle thrombus).

By the nature of hemodynamic disorder, when developing PE, there are three degrees of severity. This should be taken into account: the pressure in the right ventricle, systolic aortic pressure, end-diastolic pressure in the pulmonary arteries.

Clinical manifestations

The manifestations of PE are numerous (and therefore it is called "the great maskirovshitsy") and depend on the size of the embolus and the state of the cardiovascular and respiratory systems. Symptoms of pulmonary embolism are nonspecific, so to clarify the diagnosis in most cases necessary instrumental tests.

By excluding other diseases of massive pulmonary embolism say when a patient with relevant risk factors pronounced shortness of breath and tachycardia, hypotension (systolic blood pressure less than 90mm.rt.st.), cyanosis, increased venous pressure (expansion of the neck veins, enlarged liver). There is no single clinical sign that always have met with PE, but the most common *are the following symptoms:*

1. Bol in the chest;
2. Odyshka (20 / min);
3. Tahikardiya (more than 100 / min);
4. Aktsent D tone of the pulmonary artery;
5. Kashel;
6. Tsianoz;
7. Obmoroki;
8. Krovoharkane.

Pain syndrome most often occurs in PE (58 -88%), and may have several options for display. In most patients, the pain comes on suddenly. Chest pain tearing character develops in the main trunk embolism and pulmonary artery arises from the increase in right heart and pulmonary artery enlargement, as well as, possibly due to reflex spasm or compression of the coronary arteries. Often the pain is localized behind the breastbone, it is constricting in nature and resembles angina. If pulmonary infarction chest pain may be due to reactive pleurisy in the affected area (usually 2 - 3 hours). Such pain may be aggravated by coughing and breathing. Pleural pain with shortness of breath, or without it is the most common clinical symptom of pulmonary embolism. When recurrent pulmonary embolism pain occurs less frequently, it is uncertain and is described by patients as "discomfort in the chest." Pain at the PE may be localized in the right upper quadrant, which is associated with acute swelling of the liver in right heart failure.

The second sign of the frequency of occurrence of pulmonary embolism is a sudden shortness of breath occurs, which is the inspiration in nature. Very arising dyspnea is usually the result of thromboembolism trunk and major branches of the pulmonary artery and evolving with acute oxygen deficiency. It can further be amplified after the occlusion of small pulmonary artery branches. Sometimes the shortness of breath may develop gradually over several weeks.

The third most constant feature of PE is to tachycardia (increased heart rate over 100 beats / min), which often occurs suddenly and is progressive in nature.

As a result, reduce the minute ejection of blood from the right ventricle and the right atrium function disorders is an increase in central venous pressure should be oriented to the jugular vein and accent II tone of the pulmonary artery.

In the case of forming a syndrome of acute pulmonary heart disease is usually observed swelling of the neck veins, their pathological pulsation. On the right in the second intercostal space auscultated accent II tone and systolic murmur over the xiphoid process or in the fourth intercostal space. Often there gallop. Perhaps a sharp increase in the liver. When pulmonary embolism is common and discoloration of the skin - is characterized by pale skin, which gets ashy color. Pronounced cyanosis occurs only when a massive pulmonary embolism. He appears suddenly and is distributed on the upper half of the torso and neck. When the occlusion of small pulmonary artery branches bed may be noticeable only cyanosis of the lips and nose wings. Many patients with embolic blockage of pulmonary blood flow leads to the development of arterial hypotension - persistent or transient (the latter is more common). The presence of hypotension leading to a sharp decrease in blood flow to the left side of the heart and reduce heart ejection fraction. With a sharp decrease in blood pressure may be a violation of the formation of urine, until the development of oligo / anuria. In persons suffering from hypertension, pulmonary embolism in blood pressure can remain within normal values.

When a massive pulmonary embolism develops cerebral hypoxia, which causes brain damage (disturbance of consciousness of varying depth, convulsions, vomiting, drowsiness, anxiety and fear of death). One of the earliest signs of pulmonary embolism is a cough, which may increase the intensity within a few days. Initially, the cough is usually dry, scant mucous or bloody sputum appear later. Hemoptysis usually attaches great importance in the diagnosis of pulmonary embolism, but this feature is not early. It arises on 2 - 3 hours of PE in patients who develop pulmonary infarction. Hemoptysis rarely massive, and often there is only blood in the form of clots. Lung infarction develops mainly with thromboembolism equity and segmental branches of the pulmonary artery. Its development depends on the caliber of the vessel affected, the status of collateral blood flow and the state of bronchopulmonary apparatus. Formation of pulmonary infarction begins at 2 - 3 days after embolization and complete its formation typically occurs at the end of 1-3 weeks. In addition to hemoptysis, clinical signs of pulmonary infarction are chest pain, shortness of breath, tachycardia, crackles and rales over the corresponding portion of the lung fever. In 50% of cases with pleural effusion developed a small amount of serous or hemorrhagic exudates. In patients with severe heart failure, pleural effusion may be a large amount. Sometimes pulmonary infarction complicated by the formation of cavities. The process of decay contribute previous lung, bronchopulmonary infection, large infarct size. Thus vast cavity may be formed in a few days. Sometimes lung infarction can be complicated by lung abscess, pleural empyema, spontaneous pneumothorax. In patients with pulmonary embolism usually occurs weakness, which is a manifestation of cardiovascular disease, as well as a consequence of intoxication in the development of infarction pneumonia and pleurisy. When the occlusion of small pulmonary artery branches main clinical symptom can be general weakness and slight shortness of breath.

Sometimes there is an allergic syndrome with pulmonary embolism. It is caused by absorption of necrosis products in myocardial and lightweight generation of antibodies in the body to the damaged lung tissue, the release of histamine and serotonin. He develops in the second week of the onset of the disease. It is characterized by urtikariopodobnaya skin rash, sometimes accompanied by pruritus and eosinophilia.

Recurrent pulmonary embolism occurs in 9 - 35% of patients with pulmonary embolism. The most common recurrent embolism occurs against a background of cardiovascular diseases occurring with cardiac arrhythmias and heart failure, malignant tumors, after operations on the abdominal cavity. PE recurrent flow often occurs under the guise of other diseases. Its clinical picture may show recurrent episodes of feeling of constriction in the chest with unmotivated episodes of shortness of breath. There may be repeated "pseudopleuropneumonii" dry or exudative pleurisy, especially hemorrhagic, effusion, bouts of shortness of breath and tachycardia. It occurs and the increase of heart failure, atelectasis formation of discoid detected X-ray examination. Elderly

patients with chronic cardiac or pulmonary disorders, symptoms of pulmonary embolism can simulate a complication of the underlying disease (circulatory failure decompensation, angina or cognitive impairment due to brain ischemia).

In elderly patients with severe cardio-pulmonary pathology can quickly develop decompensation even with thromboembolism of small branches of the pulmonary artery. At the same time signs of pulmonary embolism is often mistaken for worsening of the underlying disease and the correct diagnosis is made too late. PE Often asymptomatic. Weighting of the underlying cardiopulmonary disease may be the only manifestation of pulmonary embolism. In this case, the correct diagnosis is difficult to establish. The presence of comorbidities not only contributes to the appearance of the clinical picture of pulmonary embolism, but also worsens the prognosis. If you have chronic heart failure, the risk of death increases by 2.7 times during the first year, and in the presence of lung pathology - 2.2 times

Laboratory and instrumental diagnostics

Absolutely reliable clinical manifestations of pulmonary embolism is not, so it is very difficult diagnosis. It is necessary to take into account the presence of predisposing factors, clinical symptoms, physical examination findings, x-ray picture of the changes of electrocardiogram (ECG), echocardiography indices (echocardiography), laboratory tests, lung scan data, spiral computed tomography and angiography.

Electrocardiographic signs of right ventricular overload

- QIIISI symptom, ie, the appearance of pathological Q wave in lead III and deep S wave in I;
- increase in R-wave amplitude in the right chest leads - $RV1 > SV1$;
- increasing the depth of wave S in the left chest leads - $SV5 > RV5$;
- the appearance of negative T wave in leads V1-V3;
- appearance of high (over 2 mm) in tapered prongs P III and aVF leads (p-pulmonale);
- the appearance of the blockade right bundle branch block.

ECG changes are observed in approximately 80% of patients with massive PE, with obstruction of the pulmonary artery branches - 65%. By decreasing the caliber of the vessel obturated incidence of ECG changes decreases. No changes on the ECG can not serve as a pretext for excluding the diagnosis of pulmonary embolism.

X-ray of the lungs. Pathological changes on chest radiography detected in 84% of patients with pulmonary embolism, but they malospetsifichny. The most typical are: the expansion of the shadow of the superior vena cava, the expansion of the shadow of the heart to the right, protrusion of the cone of the pulmonary trunk, high standing dome of the diaphragm on the affected side. There may be signs of the expansion of the root of the lung, it docked and strain on the affected side, as well as the depletion of lung pattern (Westermarck's sign).

The classic wedge-shaped shade with the top facing the light gate ("Hampton hump"), is rare, but it has important diagnostic value. A similar pattern is found not earlier than 2nd day of the disease, and often later, which further reduces the diagnostic value of radiographic examination. Sometimes finding accumulation of fluid in the pleural cavity, subpleural infiltrate. X-ray examination is carried out, basically, with differential diagnostic purposes (except banal pneumonia, pneumothorax, rib fractures, tumors). The combination of severe dyspnea and hypoxemia in the absence of bronchospasm and pathological changes on chest radiograph allows suspected pulmonary embolism.

Chest X-ray is needed to interpret the results of lung scintigraphy, which is carried out in the next stage of diagnostic search.

Visualization of the lower extremities - an important study, which is required for all patients with suspected pulmonary embolism and is held regardless of whether the patient's symptoms of DVT or

not. Detection of DVT speaks in favor of PE. However, 30% of patients with PE at venography changes are detected. Venography is considered the "gold standard" diagnosis of venous thrombosis. The sensitivity and specificity of nearly 100%. Compression ultrasonography to evaluate accurately femoropopliteal segment of NC, but often does not detect asymptomatic clots in the veins of the lower leg. Since the method is non-invasive, it can be carried out repeatedly.

Echocardiography can confirm the diagnosis of massive PE, as well as rule out other diseases. Echocardiography reveals signs of increased pressure in the pulmonary artery.

Typical echocardiography-signs of a massive pulmonary embolism are: expansion and hypokinesis of the right ventricle, the change in the ratio of the volume of the right and left ventricles, due to bulging of the interventricular septum in the left ventricular cavity, the expansion of the proximal part of the pulmonary artery, increase the speed of tricuspid regurgitation, a violation of the spectral characteristics of the flow in the outlet tract of the right ventricular enlargement of the inferior vena cava and its collapsibility inspiratory less than 50%. The negative result of echocardiography in no way does not exclude the diagnosis of pulmonary embolism. Interpretation of echocardiography during PE is difficult in patients with chronic obstructive pulmonary disease and congestive heart failure.

In most cases, *the blood picture* no pathological changes. Inflammatory changes (leukocytosis, increased erythrocyte sedimentation rate) appear during the development of infarction pneumonia. Sometimes there is an increase of LDH activity and ACT serum bilirubin is at a normal level.

Determining the level of D-dimer is used to exclude venous thrombosis and pulmonary embolism. The content of D-dimer should theoretically match the fibrinolytic activity. Pathology reflects D-dimer level above 500 ng / ml (ELISA method) or 200 ng / ml (of whole blood agglutination method). In the absence of D-dimer increase the probability of pulmonary embolism is very low (1 - 3%). Small or fresh blood clot does not give a significant increase in the concentration of fibrinolytic products, so the results of the study are sometimes false-negative. False-positive results occur much more frequently, and their causes may be malignant tumors, recent (within 3 months.) Surgery or injury, liver disease.

The content of troponin (T or I) in the blood increased from 30 - 40% with a massive pulmonary embolism, which is associated with acute overload of the right heart.

The gas composition of the blood. The majority of patients with confirmed pulmonary embolism show a decrease in the partial pressure of oxygen in arterial blood (PaO₂ less than 80 mm Hg) or an increase in the alveolar-arterial oxygen gradient (more than 20 mm Hg), but most of PaO₂ values or alveolar arterial gradient does not differ from the norm, especially in patients with minimal embolism, compensation due to tachypnea, and in the absence of background diseases of the heart and lungs, so the normal values of these parameters do not exclude pulmonary embolism. Diagnostic value also has a simultaneous decrease in PaO₂ and the decrease in PaCO₂.

Other methods of examination. In a hospital environment can be taken further examination: lung perfusion scintigraphy, ventillyatsionnaya pulmonary scintigraphy, spiral CT angiography of the pulmonary artery, which is the "gold standard" for diagnosis of pulmonary embolism, and others.

Treatment of pulmonary embolism

- Provide rest - physical activity should be minimal;
- Normalization of hemodynamics;
- Relief of pain;
- Restoration of adequate perfusion of the lungs;
- Prevention of relapse and death;
- Treatment of venous thrombosis / thrombophlebitis.

The basis of medical treatment - the use of anticoagulants. Antithrombotic therapy - highly effective treatment method to reduce the mortality rate from 30% to 2 - 8%. The standard antithrombotic

therapy for venous thromboembolism is the appointment of heparin (unfractionated or low molecular weight) and oral anticoagulants, provided a stable condition of the patients. The exception is unstable patients who require thrombolysis or immediate formulation of cava filter.

Heparin. Heparin is a direct anticoagulant and has no direct effect on an already formed thrombus. The objective of the heparin therapy is to prevent the occurrence of blood clots, and if any - in the prevention of a further increase in size and reduction of secondary thromboembolic complications. The mechanism of anticoagulant action of heparin is due to its interaction with the cofactor antithrombin III, On which accounts for about 75 - 90% of the spontaneous blood anticoagulant activity and in combination with heparin, which catalyzes the inactivation of factors Pa and Ha 1Ha.

Unfractionated heparin (UFH) is administered in an initial dose of standard 5000 ME intravenous bolus followed by intravenous infusion at the rate of 1680 IU / h. Monitoring the severity hypocoagulation effect is generally carried out by determining the activated partial thromboplastin time (aPTT) which is to 1.5-2.5 times higher than the normal range, which corresponds to plasma concentration of heparin in the range 0,2-0, 4 IU / mL. The first determination of APTT is performed after 4-6 hours after the start of therapy, after the therapeutic range - 1 time per day. The main drawbacks of unfractionated heparin include: the need for individual adjustment of the dosage controlled by APTT and related practical difficulties, the need for long-term (a few days), intravenous administration, "reactivation" of the disease after cessation of the infusion, the possibility of development of immune thrombocytopenia with a paradoxical increase in the frequency of blood clots.

In this regard, while venous thrombosis is increasingly used low molecular weight heparins (LMWH) :: Dalteparin, nadroparin, enoxaparin, etc. Advantages of LMWH due to their improved pharmacokinetic properties as a result of structural differences between UFH. Therapeutic doses of LMWH are appointed on the basis of:

- dalteparin - 100ME / kg every 12 h subcutaneously or 200 IU / kg subcutaneously once per day;
- nadroparin - 86 IU / kg bolus, undertaking 86 IU / kg subcutaneously every 12 hours;
- enoxaparin - 1 mg / kg (100 IU / kg) via subcutaneous 12h.

The duration of heparin therapy with venous thrombosis - not less than 4-5 days, while the appointment of heparin anticoagulants of indirect action (IDA). Cancel heparin is possible after the selection of adequate doses of AEDs (MHO achievements in the therapeutic range for two consecutive days). Longer periods of initial heparin therapy may be considered in the case of massive pulmonary embolism or thrombosis ileofemoralnogo.

Contraindications to the appointment of heparin :

- hemorrhagic syndrome of any etiology;
- severe uncontrolled hypertension;
- peptic ulcer disease or a tumor of the gastrointestinal tract with a high risk of bleeding;
- infective endocarditis;
- retinoangiopatiya;
- thrombocytopenia (less than 100,000 l);
- diseases accompanied by impaired blood clotting;
- brain surgery and spine;
- known hypersensitivity to heparin.

Anticoagulants of indirect action

AED (warfarin fenilin) are highly effective drugs for the treatment of venous thrombosis. Patients with proximal thrombosis prolonged AED therapy reduces the incidence of objectively confirmed

venous thrombosis recurrence from 47% to 2%. IDA does not have a direct impact on an already formed thrombus. The destination of the IDA - the prevention of blood clots and further increase their size, as well as reducing the risk of thromboembolism recurrence. IDA action mechanism associated with the inhibition of hepatic four vitamin K-dependent coagulation factors - II, VII, IX, X, which reduces the formation of thrombin and hypocoagulation effect. Clinically significant changes are determined not earlier than 8-12 hours in blood clotting after the first dose of the IDA, the maximum effect is seen after 72-96 hours and the duration of action once the dose can be from 2 to 5 days. MHO therapeutic range for the treatment of IDA venous thromboembolism and the prevention of their recurrence corresponds to 2.0-3.0. In identifying the antiphospholipid syndrome MHO values should be increased to 2.5-3.5. During the period of selection of a therapeutic dose of AEDs recommended daily definition MHO. The dose is considered to be selected when receiving MHO in the "therapeutic range" for 2 consecutive days. Typically, at 12 the therapeutic range is recommended following MHO control algorithm:

- the first definition of MHO-in 5-10 days
- second - after 2 weeks;
- the third - in 3 weeks;
- fourth and all subsequent - 4 weeks.

The duration of anticoagulation therapy for venous thromboembolism depending on the clinical situation. The first episode of idiopathic venous thromboembolism or venous thromboembolism with the presence of avoidable risk factors (immobilization, surgery, trauma, use of estrogen) AED therapy is carried out for 3-6 months.

The first episode of venous thromboembolism and the remaining risk factors therapy AEDs continue until the elimination of risk factors.

At repeated episode of idiopathic venous thromboembolism, or during the first episode of thromboembolism in patients with thrombophilia, treatment duration of the IDA to date not specified, but it should last at least 12 months.

The main serious complication during the treatment of IDA is bleeding, that risk increases significantly and exceeds the benefits of therapy with an INR > 5.0-6.0.

Anticoagulation is not intended to dissolve embolism, but prevents the progression of the process, thereby reducing mortality to 5%.

thrombolytic drugs

Thrombolytic therapy is indicated for the purpose of the most rapid restoration of occluded pulmonary artery, reducing pulmonary hypertension and right ventricular afterload.

Relationship to TLB ambiguous. It is believed that the systemic effects TL- drugs may contribute to the destruction of a thrombus embolism -source and lead to a recurrence of pulmonary embolism. Convincing data on the positive impact of thrombolytic therapy on outcomes of PE no. Thrombolytic therapy is associated with an increased risk of bleeding.

Indications for use of thrombolytic therapy - the development of a massive pulmonary embolism with symptoms of hypotension (systemic blood pressure less than 90 mm Hg or a reduction of blood pressure by 40 mm Hg over 15 minutes, not caused new problems or heart rhythm disorders, hypovolemia or sepsis) or shock.

With the development of submassive PE, accompanied by the phenomenon of right heart failure issue of thrombolytic therapy is decided on an individual basis. The presence of perfusion deficit of more than 30% according to scintigraphy, or an increase in systolic pulmonary artery pressure more than 60 mm Hg are additional arguments in favor of the thrombolytic therapy. Patients without evidence of overload of the right heart Thrombolytic therapy is not indicated.

There is no generally accepted scheme of thrombolytic therapy for pulmonary embolism. It is more effective in the early stages of occurrence of pulmonary embolism - in the first 3-7 days of the disease. Thrombolysis is recommended for 48-72 hours under the supervision of angiopul-

monograph. In the absence of thrombolysis treatment should cease. Upon reaching thrombolysis, thrombolytic therapy should be continued for another 24-48 hours.

Thrombolytics administered in a peripheral vein, providing the same efficiency as when administered directly into the pulmonary artery.

TLT is preferably carried out with the use of alteplase. The drug is administered at a dose of 100 mg during 2 hours / a preliminary bolus of 10 mg to 13 for 1-2 minutes, but is very expensive because the drug in the absence of contraindications allowed the introduction of other members of this group. For example, strepto kinase is administered at a dose of 250,000 IU bolus of 50 ml of 5% glucose solution for 30 min, then conduct a constant infusion of 100,000 IU per hour .techenie 24 hours; recombinant tissue plasminogen activator is administered at 100 mg for 120 minutes.

Absolute contraindications to thrombolytic therapy are ongoing internal bleeding, intracranial bleeding within the last month. Relative contraindications: big surgery within the past 10 days, internal bleeding in the preceding 10 days, a biopsy body or needle biopsy of an artery that can not be mechanically squeeze in the last 10 days, a serious injury in the last 15 days, neurosurgical and ophthalmological surgery within 1 month., ischemic stroke within 2 months., hemorrhagic stroke in history, uncontrolled hypertension (over 180/110 mmHg), severe diabetic retinopathy, coagulopathy (platelet count less than 100,000 / mm³, prothrombin index of less than 50 %), pregnancy, menstruation, infective endocarditis, hemorrhagic diathesis, a brain tumor.

At the time of thrombolysis heparin should be discontinued.

When low cardiac index and blood pressure in the normal / injected at a rate dobutamine 2.5-10 mg / (kg min) or dopamine - 2.10 mg / (kg min) with a gradual increase of the intensity of administration (depending on effect) every 2 -5 min 50 to 20 mg / (kg min).

Oxygen Inhalation shown anyway.

With a low blood pressure / in infusion fluid administered and used *vasopressors* : in / adrenaline at a speed of 4.2 g / min if no signs of shock or dopamine - 2.10 mg / (kg min) with a gradual increase of the intensity of administration (in depending on the effect) every 20-50 min to 5.2 g / (kg min).

The amount of fluid injected should not exceed 1000 ml.

When the pain in the chest, associated with breathing, prescribed *NSAIDs* (indomethacin 25 mg 3 times a day), or *narcotic analgesics* (if there is no oppression of the respiratory center, for example, against a background of severe hypercapnia).

Surgical embolectomy can be used for the treatment of high risk of pulmonary embolism in the presence of absolute contraindications to thrombolytic therapy or its ineffectiveness. An alternative to surgical treatment are methods catheter embolectomy or fragmentation of proximal clots in the pulmonary arteries.

In specialized surgical departments sometimes spend embolectomy. If thrombolytic therapy is ineffective or if there are contraindications to it, in the acute period of possible *implantation of vena cava filter* in the inferior vena cava.

prevention

Prevention of venous thrombosis and pulmonary embolism is based on the determination of the risk incurred for each particular patient and assigning it to one of three risk categories: low, moderate or high.

Category risk of venous thrombosis is determined by the availability of each patient's risk factors for venous thrombosis, which include: cancer, heart failure, myocardial infarction, sepsis, dilated cardiomyopathy, atrial fibrillation, stroke, broncho-obstructive disease erythremia, inflammatory bowel disease, obesity, nephrotic syndrome, surgery, trauma, age over 40 years, estrogen, prolonged immobility, pregnancy, varicose veins, venous thrombosis history, confinement to bed for more than 4 days., thrombophilia.

By thrombophilic states, which is essential to determine the risk of venous thrombosis and tactics pursued by antithrombotic therapy include the following defects in the hemostatic system: anomaly

factor V (Leiden mutation), hyperhomocysteinemia, an anomaly of prothrombin (G20210), the presence of antiphospholipid antibodies, antithrombin deficiency W, deficiency of protein C or S. At surgery the risk of venous thromboembolism is determined by the assessment of the severity of the surgery and the patient's condition. The basis for the prevention of venous thrombosis in these patients is their early activation, elastic compression of the lower extremities and heparin. For purposes of heparin in preventing venous thromboses in surgical patients are usually used in the following doses of LMWH:

- 5 000ME dalteparin (0.2 mL) 1 time per day subcutaneously;
- nadroparin 0.4 ml (3 800ME) the first three days, then on. 5700 ME (0,6 ml) per day subcutaneously;
- enoxaparin 40 mg (4000 IU / 0.4 ml) per day subcutaneously for high-risk group and 20 mg (2,000 IU / 0,2ml) per day subcutaneously for a group of moderate and low risk.

Heparin begins 2 hours before surgery and continues for at least 5-7 days after surgery to the patient activation. In urgent surgery and at high risk of bleeding interoperative heparin therapy can be started within 12 hours after the operation is complete. In such situations, it is advisable to use higher doses of LMWH.

The use of direct anticoagulants (LMWH, UFH) for the prevention of pulmonary embolism in surgical and other invasive procedures included in the industry standard with the highest level of evidence criteria (level A on the scale of credibility, "convincing evidence: there is strong evidence of the proposed approval").

Secondary prevention of pulmonary embolism For the prevention of recurrence of pulmonary embolism using oral anticoagulants, the reception of which is continued for 3 months. In patients with unavoidable risk factors, when some unknown reason, pulmonary embolism and in case of recurrence of anticoagulation may be appointed for an indefinite period. In patients with cancer for long-term anticoagulation therapy instead of oral anticoagulation is recommended to use LMWH.

Warfarin - derivative monokumarina vast synthesis in hepatic vitamin K-dependent coagulation factors, namely - II, VII, IX and X. Anticoagulant effect fully manifested at 3-5 days after the onset of the drug, and is terminated after 3-5 days after the last dose.

Intensity of the anticoagulant effect is evaluated by the value of international normalized ratio (INR). In the treatment of pulmonary embolism shows a mean level of anticoagulation with a target INR of approximately equal to 2.5 (2.0-3.0). it takes several days to reach the target INR, so warfarin with PE should start from the first days of the disease is still on the background of heparin. Before the start of treatment should be to assess the risk of bleeding on a scale of HAS-BLED. If the total score of 3 or more the risk of bleeding is high. High risk of bleeding on the HAS-BLED scale is not a ground for refusal of antithrombotic therapy, since the risk of bleeding associated with warfarin increases only with an increase of more than INR 3.5-4.0, and the therapeutic range in patients with pulmonary embolism is 2.0 -3.0. Thus, a high index of scale HAS-BLED (3 or more) indicates the need for careful monitoring of INR levels and eliminate potentially reversible risk factors for bleeding, such as hypertension, receiving antiplatelet agents, and nonsteroidal anti-inflammatory drugs.

Contraindications to warfarin purpose are :

1. Allergy drug
2. Pregnancy
3. Active bleeding at any site
4. Diseases and conditions, fraught with the development of bleeding
 - recent or planned at shortly time surgery , recent trauma
 - thrombocytopenia and other infringement clotting of blood
 - hemorrhagic complications at history (a hemorrhagic stroke , gastro -

intestinal bleeding , gross hematuria , uterine bleeding)

• disease , associated from high risk of bleeding (intracerebral aneurysm and vascular malformation , dissecting aneurysm of the aorta , pericarditis or effusion at pericardium , infective endocarditis , malignant hypertension , peptic ulcer disease stomach or 12 duodenal ulcer at stage of exacerbation, portal hypertension with esophageal varices, severe kidney or liver failure, cancer)

5. Dementia, alcoholism, psychosis, social exclusion

6. High risk of falling (epilepsy and other seizure disorders)

7. The inability to control the INR.

Absolute contraindications include active bleeding, allergies to medication, hemorrhagic stroke in history, and thrombocytopenia (platelet count less than $100 \times 10^9 / L$). In other cases, the possibility of warfarin is decided on the basis of an individual assessment of potential risk of thromboembolic and hemorrhagic complications. Warfarin is taken 1 time a day, after meals, in the evening, and the MNO determines the morning. Titration is carried out in accordance with an algorithm

IV. QUESTIONS FOR SELF

Select one or more correct answers

1. significant risk factor for PE APPLY

1) fracture of the femur or tibia

2) bed rest for more than 3 days,

3) cancer

4) prosthetic hip or knee replacement

2. Moderate risk factor for PE APPLY

1) a large abdominal surgery

2) spinal cord injury

3) a paralyzing stroke

4) chronic heart failure

3. minor predisposing factor in the development of PE APPLY

1) laparoscopic surgery

2) oral contraceptives

3) Pregnancy

4) the postnatal period

4. IN PE at high risk of death INDICATE

1) shock or hypotension

2) signs of right ventricular overload

3) positive D-dimer test

4) positive troponin test

5. PE at low risk of death indicated by the following combination of features

1) negative D-dimer test

2) the absence of shock or hypotension

3) negative troponin test

4) the absence of signs of right ventricular overload

6. If you suspect a pulmonary embolism With high risk of death and inaccessible computed tomography should immediately

1) ultrasound of the lower limbs

2) echocardiography

3) a highly sensitive D-dimer test

4) troponin test

7. SIGNS OVERLOAD echocardiographic right ventricular SERVE

1) reduction of the ejection fraction of the right ventricle

2) diastolic size of the right ventricle 30 mm

- 3) the ratio of right and left ventricular size greater than 1
- 4) smoothing systolic ventricular septal
8. If you suspect a pulmonary embolism with a low risk of death should first
 - 1) conduct echocardiography
 - 2) perform a D-dimer test
 - 3) assess the clinical probability of pulmonary embolism
 - 4) perform a troponin test
9. AT HIGH clinical probability of pulmonary embolism should first HOLD
 - 1) ultrasound of the lower limbs
 - 2) echocardiography
 - 3) a highly sensitive D-dimer test
 - 4) multislice computed tomography pulmonary artery
10. If HIGH clinical probability of pulmonary embolism and inaccessible computed tomography should be performed
 - 1) ultrasound of the lower limbs
 - 2) echocardiography
 - 3) a highly sensitive D-dimer test
 - 4) troponin test
11. When is not high clinical probability of pulmonary embolism should first HOLD
 - 1) ultrasound of the lower limbs
 - 2) echocardiography
 - 3) a highly sensitive D-dimer test
 - 4) troponin test
12. PRI give a low clinical probability of PE NEGATIVE TEST highly sensitive D- dimer
 - 1) confirms the diagnosis of pulmonary embolism
 - 2) exclude the diagnosis of pulmonary embolism
 - 3) has no diagnostic value
13. When is not high clinical probability of pulmonary embolism POSITIVE TEST highly sensitive D- dimer
 - 1) confirms the diagnosis of pulmonary embolism
 - 2) exclude the diagnosis of pulmonary embolism
 - 3) has no diagnostic value
14. Indications to thrombolysis or embolectomy SERVE WITH PATE
 - 1) shock or hypotension
 - 2) signs of right ventricular overload
 - 3) positive D-dimer test
 - 4) positive troponin test
15. Absolute contraindications to thrombolytic therapy SERVES
 - 1) ever transferred a hemorrhagic stroke
 - 2) ischemic stroke within the last 6 months,
 - 3) transient ischemic attack within the last 3 months,
 - 4) a stroke of unknown nature in history
16. Relative contraindications to thrombolytic therapy SERVES
 - 1) gastrointestinal bleeding within the last month
 - 2) trauma or surgery within the past 3 weeks,
 - 3) use of oral anticoagulants
 - 4) infective endocarditis
17. FOR TREATMENT WITH PATE high risk of death YOU CAN USE
 - 1) thrombolytic agents
 - 2) unfractionated heparin

3) low molecular weight heparins

4) dabigatran

18. To prevent a recurrence of pulmonary embolism CAN USE

1) warfarin

2) dabigatran

3) rivaroxaban

4) apixaban

19. RECEPTION oral anticoagulants AFTER PE SHOULD CONTINUE AT LEAST

1) 3 weeks

2) 1 month

3) 3 months

4) 6 months

20. WHEN USING Warfarin to prevent a recurrence of PE target INR EQUAL

1) 1.5-2.5

2) 2.0-3.0

3) 2.5-3.5

4) 3.0-4.0

ANSWERS TO QUESTIONS FOR SELF

1 - 1, 4; 2 - 3, 4; 3 - 1 and 3; 4 - 1; 5 - 2, 3, 4; 6 - 2; 7 - 2, 3, 4; 8 - 3; 9 - 4; 10 - 1; 11 - 3; 12 - 2; 13 - 3; 14 - 1; 15 - 1, 2, 4; 16 - 3, 4; 17 - 2, 3; 18 - 1, 3; 19 - 3; 20 - 2.

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A. Main:

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Additional literature:

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Composed by

N.P. Prikhodko