Ministry of Education and Science of Ukraine
V.N. Karazin Kharkiv National University

ARTERIAL THROMBOSIS AND EMBOLISM OF THE EXTREMITIES

Methodical recommendations
for students of higher medical education in the discipline of «Surgery»
Faculty of Medicine

Electronic edition

Kharkiv – 2023

UDK 616.14-005.6
A 81

Reviewers:

Prasol V. O. – Doctor of Medicine, professor of the Department of Surgery № 1, Kharkiv National Medical University;
Ghnidenko J. P.– Candidate of Medicine, Associate Professor of the Department of Surgical Diseases, V. N. Karazin Kharkiv National University.

Approved for distribution in the Internet by the decision of the Scientific and Methodical Council of V. N. Karazin Kharkiv National University (Protocol № 1 of October 20, 2023)

Arterial thrombosis and embolism of the extremities: methodical recommendations for
A 81 students of higher medical education in the discipline of «Surgery» Faculty of Medicine [Electronic edition] / ed. by D. V. Oklei, S. O. Bychkov, V. O. Pronin. – Kharkiv : V. N. Karazin Kharkiv National University, 2023. – (PDF 26 p.)

Methodical recommendations are worked out on the basis of the Programs of disciplines of «Surgery» for students of higher medical educational institutions of III–IV levels of accreditation, authorized by Ministry of Health of Ukraine. These recommendations are intended for the 5 th year English-speaking medical students.

UDK 616.14-005.6

Електронне навчальне видання комбінованого використання
Можна використовувати в локальному та мережному режимі

Оклей Денис Вікторович
Бичков Сергій Олександрович
Пронін Василь Олександрович

АРТЕРІАЛЬНІ ТРОМБОЗИ І ЕМБОЛІЇ КІНЦІВОК

Методичні рекомендації
для здобувачів вищої медичної освіти з дисципліни «Хірургія»

(Англ. мовою)

Комп'ютерне верстання *В. В. Савінкова*

Підписано до розміщення 20.10.2023. Гарнітура Times New Roman.
Ум. друк. арк. 1,59. Обсяг 0,388 Мб. Зам. 173/23.

Харківський національний університет імені В. Н. Каразіна,
61022, м. Харків, майдан Свободи, 4.
Свідоцтво суб'єкта видавничої справи ДК № 3367 від 13.01.2009
Видавництво ХНУ імені В. Н. Каразіна

CONTENTS

List of abbreviations

AAI – acute arterial impassability
AAT – acute arterial thrombosis
AE – arterial embolism
ESR – erythrocyte sedimentation rate
LMH – lower molecular heparins
OI – oscillatory index
RI – reographic index
WHO – World Health Organization

Introduction

The statistical information of WHO of the last decades notes that there is growth of acute thromboemboly affectation of main arteries of extremities and bifurcation of aorta, which are the heaviest complications of row of diseases of the cardiovascular system. The results of treatment of this heavy pathology presently can not be yet considered satisfactory. So, a death rate at this group of patients, by information of different authors, makes from 25 to 35 %, and at every fifth patient surviving, almost at 20 % of them, the gangrene of extremity, requiring high amputation and resulting lead to invalidism of patient.

On Ukraine frequency of amputations at acute arterial impassability (AAI) of extremities makes 12–28 %, and lethality achieves at 25 %.

In the USA annually performs about 62000 amputations of extremities due to gangrene, which makes an annual loss in a sum about 9 billion of dollars.

In addition, at this pathology are common relapses.

Taking into account that with this urgent pathology may meet the doctors of all specialities, they must be well acquainted with the clinic of syndrome of AAI of extremities, to be able to diagnose it and timely send such kind of patients in the specialized surgical vascular department, only in the conditions of which to the patient can be given skilled effective help, moreover the results depend on the terms of beginning of treatment of patient.

Determination of concepts: "Syndrome of acute arterial impassability of extremities", «arterial embolism» and «acute arterial thrombosis» of extremities.

Acute arterial impassability of extremities (AAI) is a syndrome, acute violation of arterial circulation of blood in extremity, being due to stopping of blood stream for 1 or more main arteries and resulting in development in tissues of extremity of acute ischemic disorders causing development of irreversible necrobiotic changes in tissues of distal parts of extremity in an eventual result (in default of treatment) – development *of gangrene.*

Arterial embolism of extremity (AE) – is the sudden stopping of blood stream in a main healthy artery, caused by blockade, as a rule, by thrombus-embolus, by the torn off and brought current of blood from the cavities of heart or large arterial trunks, with evolution of acute violation of circulation of blood in extremity and development of heavy acute ischemic disorders of tissues.

The acute arterial thrombosis of extremity (AAT) – is the sudden stopping of blood stream in a main artery, caused by local formation of intravascular blood clot, with completely obstruction of the arterial lumen, which, in mostly cases, affected by stenotic occlusive process, and as result – acute violation of circulation of blood in extremity and development of acute ischemic disorders of tissues, to it being in a state *of chronic ischemia.*

Etiopathogenesis of emboli and acute thromboses, acute ischemia of tissues of extremities.

Arterial embolism (AE) of extremities is complication of different *embologenic diseases*. AT 90–95 % patients with the reason of embolism of arterial vessels of big circle there are the diseases of heart, at which thrombotic masses appearin its cavities.

Last years the changes happened in the structure of embologenic diseases: it was considerably multiplied specific gravity of atherosclerotic affectation of heart – socalled «atherosclerotic cardiopaty» (acute myocardial infarction, diffuse and post infarction cardiosclerosis, postinfarction aneurism of heart and other), which gained the lead on frequency and approximately in 55 % of cases are the reason of AAI of extremities; the rheumatic affectation of valves of heart (and among them mainly mitral stenosis) take second place and make 43 %; a septic endocarditis and congenital heart-diseases serve as a cause to embolism only in 1–2 % of cases. Thus, at patients with the atherosclerotic defeats of heart of embolic thrombotic masses more frequent is localized in the cavity of left ventricle, and at patients with rheumatic vices – in a left auricle; rarely blood clots appear on valves – at a septic endocarditis, prosthetic valve.

Among the extracardial embologenic diseases first place is taken by aneurism of aorta and its big branches – 3–4 % of cases, atherosclerosis of aorta in a thrombonecrotic stage (formation of parietal blood clots); extremely rarely peripheral AE can arise up at pneumonias, tumors of lungs (a source is the blood clots of pulmonary veins), at the acute thrombosis of peripheral veins (so-called «paradoxical embolism», when through the septum defects of the heart and opened arterial canal (Bottalo's duct) there are the cases of the anomalous moving of blood clot from right in the left cavities of heart).

More frequent than all (it is approximate in 75 % of cases)AE is observed at patients with violation *of cardial rhythm*, is special with different types of ciliary arrhythmia (cardiac fibrillation) which at patients with atherosclerotic cardiopaty and rheumatic heart-diseases is support formation of blood clots in the cavities of heart because of considerable violation of intracardial hemodynamics.

Tearing off of intracardial blood clot more frequent than all takes place at strengthening of cardiac activity – because of emotional and motion excitation, application of cardiac glycosides, after defibrillation with the purpose of removal of arrhythmia (so-called post-conversion embolism); the increase of fibrinolytic activity of blood can be support in fragmentation and tearing off of blood clot (application of powerful thrombolitic for the AE treatment is considered inadvisable in this connection – V. S. Saveliev and al., 1974).

Important mark, that the all embologenic diseases may be the origin of repeated emboli (if radical treatment of basic disease is not undertaken.

By the current of blood the torn blood clot off can be brought to any artery of big circle of blood circulation. It is marked that most often thrombus-

emboli is brought in the branch of arc of aorta (including in cerebral arteries) — 36 %, on the second place are bifurcation of aorta and main arteries of lower extremities – 24 %,in visceral arteries – 22 % (including in kidney – 18 %), rarer – in the artery of upper extremities.

Usually the embolus is blocking the main vessels in area of their branching, where the diameter of vessel diminishes. Most typical places or so-called «surgical floors» of embolus occlusion are: bifurcation of aorta, bifurcation of arteria iliaca communis, arteria femoralis, arteria poplitea, humeral artery (Fig. 1). Embolism of arteries of shin and forearm meet more frequent, than is diagnosed, and is flowed as hidden to embolism. Embolus can migrate in distal direction from one «floor» to other (after introduction of anesthetics, spasmolitics, at transporting), that explains the cases of independent some improvement of the state of extremity.

Quite often is observed «floors» embolism – AE is simultaneous at different level of main vessels of one extremity, «combined» embolism – AE of different extremities, «associated» embolism - when AE of extremities combine from the AE of visceral or cerebral vessels.

Thus, in pathogenesis of AAI of extremities due to AE plays the following moments:

1) as embolus is localized in area of bifurcation or branching of big trunks, it, as a rule, results of excluding from circulation of blood of all distal main arteries;

2) after fixation of the embolus into arterial lumen of arterial embolisms “Surgical floors” with block of it, arises reflectory spasm of the peripheral vessels and collaterals as result of irritation of the nerves constrictors into adventitia. The support factor, which increases the disorder of blood circulation, is growth of prolonged thrombosis from the body of embolus to distally and less proximally, it increases ischemia of tissues;

3) at AE local violations of circulation of blood are aggravated by disorders of central homodynamic because of presence of heavy pathology of heart or big vessels. All of it stipulates development at the AE heavy, acute expressed ischemia of tissues of extremity and quick evolution of gangrene.

The principal causes of acute arterial thromboses (AAT) of extremities are the chronic occlusive diseases such as obliterated atherosclerosis (most often), obliterated endarteritis, obliterated thrombangitis, which are making progressing stenotic process of main arteries, which is in 75 % and more the cause of acute thrombosis, fully blocking the vessel.

In pathogenesis of pathological intravascular thrombi formation a leading role belongs to ***three basic factors*** known as the pathogenetic triad Virchov:

1) narrowing of vessel’s lumen with diminishing the blood stream,

2) changes of vascular wall (violation of smoothness of internal wall as a result of mechanical, thermal, chemical, bacterial influences, violation of microcirculation), increase of thromboplastic activity of the affected arterial

wall, including and lowering into it synthesis ***of prostocycline*** – powerful inhibitors of aggregation of thrombocytes and increase synthesis ***of thromboxane*** – powerful inductor of aggregation activity of thrombocytes,

3) violation of the functional state of the system of hemocoagulation with the increase of coagulative properties (foremost, growth of aggregation activity of thrombocytes) and suppression of antycoagulative and fibrinolytic activity of blood.

In pathogesis of AAI of extremities on the AAT there are differences from AE, influencing on the degree of intensivety of developing acute ischemia:

1) at AAT, as a rule, to acute ischemiain beginning undergoing limited arterial segment, which is more affected by stenotic process mostly due to chronic disease of artery, while on other artery a blood stream proceeds;

2) complete occlusion of artery as usually take place on a background protractedly existent chronic ischemia of tissues and developed collateral circulation of blood in extremity, that partly compensates the halted magisterial blood stream in one of arteries, it means that in case of AAT distally blood feeding of the tissues is not fully halted.

3) such patients relatively rare have heavy violations of central homodynamic. Due this peculiarity in case of AAT acute ischemic process in tissues of extremity not so expressed as in case of AE, gangrene is rarer and considerably more lately develops.

Thus, the AE and AAT of main arteries of extremity lead to *development of acute ischemia of tissues* distally than occlusion, to violation of function of extremity and to neurology disorders in extremity. In pathogenesis of acute ischemia the following factors are significant:

1. Development *of acute hypoxia*– anoxaemia of tissues, leading:

a) violation of all types of metabolism in tissues and, foremost, to development *of metabolic acidosis* as result of transition of aerobic oxidization in anaerobic with accumulation in tissues of nonoxide products of exchange – lactic and pyruvic acids;

b) appearance *of active enzymes (kinynes, kreatin-phosfotasa and others), that results in violation of permeability of cellular membranes and death of the muscles cells, and accumulation* intracellular potassium and myoglobin in an extra cellular fluid;

c) evolution of hyperpatassimia *and «myoglobinuritic nephrosis»*; violation of cellular permeability and ionic equilibrium results in development of

d) *subfascial edema of muscles*, aggravating violation of blood stream in tissues;

e) formation of the macroaggregates from elements of blood.

2. Due to quick evolution of the acute occlusion of main artery distally occurs *expressed artery spasm* spreading and on collaterals and aggravating violation of blood stream.

3. Violation of hemodynamics (stasis of blood), artery spasm, pathological changes of vascular wall as a result of hypoxia, the presence of macro aggregates from elements of blood and change in coagulative system is creating the conditions for further intravascular thrombus formation – evolution *the prolonged thrombosis* in proximal (ascending) and in distal (descending) directions from the place of primary occlusion. In the beginning period a blood clot flattering into the vessels lumen,unconnected with an arterial wall and may be easily removed in time of operation, and than develops adhesions between clot and arterial wall and became spreads on lateral branches, muscles arteries, capillaries and even on veins, that aggravates an ischemia and leads to unrestored changes in tissues of extremity, doing operative interference ineffective.

4. Violations of local circulation of blood cause *the changes in central hemodynamic* – develops lowering of B/P, worsening of indicators of cardiac function (decline of shock volume, cardiac index).

A sensitiveness to hypoxia of tissues is different*:* first suffer nervous, and then muscles cells – in them the irreversible changes appears after 10–12 hours; a skin is more steady to the ischemia – in it the irreversible changes come in 24 hours. Disorders of circulation of blood and ischemic damages of tissues are most visible in the distal parts of extremity, diminishing in intensity in proximal direction.

Classification of degrees (stages) of acute ischemia of tissues of extremities at *the AAI syndrome* **of** developed **V.S. Savelev** and **is accepted in a clinic. (**1987). It includes the «ischemia of tension», and also ischemia the I, II and III degrees, which, in same queue, divide by 2 stages, – A and B. Every from them is characterized by certain subjective objective signs.

Ischemia of tension is absence of signs of ischemia at rest and appearance of them at loading.

At the ischemia of the I degree violations of sensitiveness and motions in the affected extremity are absent: the IA degree is characterized by the presence of sense of numbness, coldness, by paresthesia, at the IB degree appear the pain in the distal parts of extremity.

For the ischemia of the II degree *is* characteristic by the disorder of sensitivity, and also active motions in joints from paresis (IIA degree) up to plegia (IIB degree).

Ischemia of the III degree *is* characterized by the beginning necrobiotic phenomena, that is expressed clinically in appearance of subfascial edema of muscles (IIIA degree), and afterwards – muscles contracture: partial (IIIA degree) or total (IIIC degree).

Depending on the clinical feature authors select an ischemia:

1) making progress,

2) moderate stable and

3) regressing (a gangrene is the end of the first form,second and third is chronic arterial insufficiency).

V. S. Savelev and coauthors offer *classification of stages of development of acute ischemia of tissues of extremity:*

The I stage – the stage of acute ischemic disorders – develops in the early time after embolism or acute thrombosis and is characterized by the expressed signs of ischemic affectation of extremity, clinically it shows up by all above mentioned symptoms.

The II stage – the stage of relative collateral compensation of blood circulation of extremity – develops after a few hours (6–12); coming compensation of collateral blood circulation results diminishing of pains in extremity and of pallor of skin, extremity gets warm, motions and skin sensitiveness appear in it (it is marked, mainly, at acute thromboses, rarer – at emboli). *The III stage – the stage of decompesation of circulation of blood* – develops in 8–14 hours approximately, but can follow from the I stage directly, is characterized by appearance of signs of beginning necrobiotic changes of muscles, that shows up as the subfascial edema, painfulness of muscles at palpation, rigidity and limitation of passive motions in joints up to muscles contracture. *The IV stage – the stage of irreversible changes in tissues of extremity, stage of gangrene* – after of 24 hours and more from the moment of development of acute occlusion, is characterized by total contracture of big joints, by the edema of muscles, by a several painfulness at palpation and passive motions, by appearance of necrotic changes of skin on peripheries or zone of demarcation, general intoxication of organism. In practical work it is comfortably to use *classification of degrees of the acute ischemia by* **A. A. Shalimov** and coauthors (1979) which are divided on 4 degrees of ischemic affectation of tissues of extremity:

I – easy,

II – middle,

III – heavy and

IV – stage of gangrene or irreversible changes of fabrics of extremity.

I an easy degree – includes the cases of the effaced and easy clinical evidences as pain at loading or slightly expressed pain at rest, paresthesia, coldness of extremity,which arise up after blockade but without the expressed violations of sensitiveness and motive function of extremity. Timely executed operation at AE results in complete restoration of function of extremity.

II middle degree – is characterized by the signs of ischemic affectation, mainly, *nervous system*, by violation of pain and tactile sensitiveness, by limitation of motions in fingers or absence of active motions up to a complete paralysis, however, rigidity, contracture and edema *of muscles are absence.* Restoration of blood stream in this stage usually restores the function of extremity, but can be observed ischemic neuritis requiring long treatment.

III heavy degree (or «complete ischemic syndrome») – is characterized by the signs *of beginning necrobiotic changes of muscles*: rigidity, painfulness of muscles at palpation, limitation of passive motions in the distal joints of extremity because of contracture of separate muscles groups; there can be subfascial edema of separate groups of muscles. A «complete ischemic syndrome» is clinically characterized byappearance of prognostic threatening combination of three «A» – akinesia, areflexia and anesthesia. Restoration of blood circulation in extremity usually leads to development of post-ischemic disorders, which have local and general character. May be present necrosis of separate muscles and muscles groups with late their replacement by fibrotic tissues is possible, complete restoration of function of extremity usually is not observed (there are the consequences as Folkman's contracture, muscles weakness, ischemic neuritis, the gangrene of separate fingers and foot can develop).

IV stage of gangrene – is characterized *by total contracture of big joints* (ankle, knee, wrist, elbow), by the several painfulness of muscles at palpation and passive motions, by their edema, by the irreversible changes of tissues of distal segments of extremity with appearance of zone of demarcation and necrotic changes of skinon periphery, general intoxication of organism. Possible treatment is amputation of extremity.

Degree of expressed of acute ischemia of tissues of extremity at development of the AAI syndrome, as well as a clinical picture which they are associate, mainly depend from the reason of sharp occlusion, from the degree of development of collaterals, concomitant artery spasm, development of the continued thrombosis, state of central hemodynamics.

***At questioning of patient*:**

1) Complaints characteristic for the syndrome of AAI of extremities.

The most characteristic symptom is *suddenly appearing several pain in extremity,* so intensive, that patients compare it to the «blow of whip». *Complaints* about feeling of coldness, numbness, paleness of leg, «crawl of small ants»; the weakness of extremity, depriving the patient of possibility to walk and even stand, can appear after paresis or paralysis of extremity.

2) The additional question about complaints on the systems allows to suspect or detect the reason of arising acute arterial impassability: either the presence of one of embologenic diseases (atherosclerotic cardiopaty, heart-diseases, violation of cardiac rhythm and etc) or signs of chronic occlusion affectation of arteries of extremity is the "symptoms of intermittent claudication", trophic disorders and other.

3) Anamnesis of disease (date and time of disease, possible reason, got or not got medical help and where, what medicines were adopted by and what effect from their reception, information of preliminary additional methods of research – laboratory, instrumental). Information about anamnesis helps to get the reason of acute pathology.

4) Anamnesis of life (including terms of work, professional harmfulness and etc, at women obstetric-gynecological anamnesis).

Clinical physical inspection (characteristic of peculiarities at this disease):

1) Estimation of the common state of patient with the syndrome of AAI of extremity: consciousness as usually safed, general condition is middle degree or heavy; position is forced (lying position, due to impossibility to walk).

2) Collecting of information about external view of patient with the syndrome of AAI of extremity (examination of skin, subcutaneous fat, palpation of lymphatic nodes, thyroid and mammary glands).

3) State of the cardio-vascular system of patient with the syndrome of AAI extremity including (examination and palpation of region of heart and main vessels of neck in projection points, determination of percutory borders of heart, auscultation of heart and vessels; determination of the special symptoms.

4) State of organs of breathing (examination of thorax and respiratory tracts, palpation of thorax, percussion and auscultation of lungs; determination of the special symptoms).

5) State of organs of abdominal region (examination of stomach, palpation and percussion of stomach, palpation of kidneys, livers, spleen, pancreas, organs of small pelvis).

6) Inspection of bones-muscles systems (examination and palpation).

7) ***Local status***:

a) discoloration skin covers (from a pallor to cyanosis and «marble» of skin);

b) expressed lowering of skin temperature (as compared with healthy extremity);

c) violation of sensitiveness (in the beginning lowering of sensitivity on touch and pain, after – deep and up to complete anesthesia);

d) violation of motive function of extremity (from the lowering of muscles force and limitation of active motions in fingers to absence of active motions the in beginning in distal joints, and then and in more proximal up to the complete paralysis of extremity – *akinesia* and, *areflexia*;

e) development of subfascial edema of muscles, limitation of passive motions in connection with development of rigidity and contracture of separate groups of muscles, painfulness at palpation of muscles of shin (signs of beginning necrobiotic changes in tissues;

f) development in future of total contracture of big joints, edema of extremity, necrotic changes on periphery and as the eventual result of ischemia – the gangrene of extremity, appearance of zone of demarcation; *Basic objective symptom is absence of pulsation of arteries distally than place of occlusion*, allowing to detect its level.

Basic clinical symptomatic at AAI of extremities is variable. The acuteness of development, expressivity and dynamic of development of clinical feature depends, foremost, from a reason (AE or AAT), and also from the caliber of

vessel (places of occlusion), character of block (complete or partial), expressivity of artery spasm and from *the degree of acute ischemia of extremity, which, in same queue, depends from all above mentioned factors*.

For AE is characteristic the acutest beginning, expressed of clinical symptoms, proximal border of ischemic disorders clear and more frequent than all corresponds to the level of embolism, as a rule, heavy degree of acute ischemia and quick its progressing lead to development of gangrene.

Embolism of bifurcation of aorta. Heavy disorders of circulation of blood in lower extremities and pelvic organs come at it, that shows up by general and local symptoms: beginning acute – suddenly there are so intensive pains in both lower extremities and in the lower half of abdomen, that quite often restores patients to a state of shock; pains of radiates in a sacrum, small of the back, perineum; the rapid drop of the temperature of feet with feeling of their numbness and muscles weakness is marked; skin covers (up to inguinal folds and buttocks) become pale, after – «marmorate»; violation of all types of sensitiveness comes quickly; lower extremities are in extended position, active motions in them are absent, feet hang down, the fingers as claw distort; a pulsation is absent on all arterial segments of extremity; because of ischemia of pelvic organs false urges are possible or involuntary urination and defecation.

The picture of ischemia in beginning can be more extensive in one extremity (as embolus has the appearance of «horseman» and can not completely blocking one of general iliac arteries). In case of absence possibility for skilled urgent help the disease makes progress quickly, as a result ascending continued thrombosis blocking the mouths of kidney arteries with development of acute kidney insufficiency, quickly the gangrene of both extremities develops and patient perishes from endogenous intoxication and polyorgan insufficiency.

Embolism of iliac arteries. Embolus more frequent than all is localized in area of bifurcation of general iliac artery or above an inguinal ligament, a clinical picture reminds embolism of bifurcation of aorta, but from one side (the pulsation of aorta and main arteries of other extremity is determined).

Embolism of femoral arteries. Most frequent localization on extremities. More frequent than all embolus it is localized in area of bifurcation of general femoral artery (in the place of branching deep artery of thigh) or above the entrance in a Gunter's channel. A clinical picture shows up the symptoms of acute ischemia of shin and foot (or foot and distal half of shin).

Embolism of popliteal artery in area of it bifurcation in upper third of shin or *arteries of shin* meets rarer, not always gives a clear clinical picture – the expressed pains in a foot and in the lower part of shin, but insignificant violations of skin sensitiveness and motive function in ankle joint and foot; although the circulatory ischemic necrosis of muscles (pretibial groups), related to the increasing edema of muscles, causing compression of tissues and vessels in a close fascial bed, can develop sometimes.

At AAT, unlike AE, beginning of disease is more frequent more gradual and the clinical evidences are less expressed – during walking or at other physical exertion (and sometimes and at peace) at patients may have moderate intensivety pain in the muscles of shin, in a foot, unpleasant feelings of drop in a temperature, numbnesses of extremity, intensive weakness, convulsive contraction of muscles; cases are possible, when pains can develop during a few hours gradually, and sometimes and days, slowly making progress; the degree of violation of sensitiveness (superficial and deep) varies in wide limits – from insignificant hyposensitivity to complete anesthesia; possible appearance prognostic threatening combination of three «A» – akinesy, areflexy, anesthesia (such cases, as a rule, end with a gangrene).

The phenomena of decompesation of circulation of blood and development ofgangrene of extremity usually take place during 5-10 days from the moment of the AAT and come relatively rarer, than at embolism (due to the peculiarities of pathogenesis – presence to the moment of acute thrombosis of network of collaterals and occlusion of only one vessel at patients with chronic diseases of arteries).

At AAT the proximal border of acute ischemia is not so clear, as usually is absent accordance of localization of blood clot and level of ischemic disorders, the evidences of acute ischemia are less expressed, rarer develops the heavy degree of acute ischemia, compare with AE.

8) On the basis of findings of questioning, anamnesis of disease and clinical physical inspection of patient to put a preliminary clinical diagnosis.

Diagnostics of syndrome of AAI extremities at most patients does not represent difficulties, as for it a bright clinical picture is characteristic. Some difficulty can cause the question of establishment of etiologic diagnosis, reasons of origin of this syndrome – arterial embolism or acute arterial thrombosis. In this plan considerable help is given by anamnesis information – has the patient of embologenic diseases or chronic occlusion diseases of arteries of extremity. The important value has establishment *of level of acute occlusion*, which can be determined clinically by palpation of extremity in the projection points of main arteries (determination of presence or absences of pulsation).

The main clinical syndromes –pain in extremity, paresis or paralysis of extremity.

Examples of preliminary clinical diagnosis:

I – variant: post infarction cardiosclerosis; cardial fibrillation; arterial embolism of right (left) lower (upper) extremity (supposed localization – for example, bifurcation of general femoral artery or bifurcation of humeral artery), acute ischemia of the III degree.

II – variant: obliterated atherosclerosis of the right (left) lower extremity; the chronic ischemia of the III-й degree, acute arterial thrombosis (the supposed localization – superficial femoral artery), acute ischemia of the II-й degree.

In accordance with standard charts the plan of additional inspection (laboratory and instrumental) of patient with the syndrome of AAI of extremity in the conditions of the specialized vascular surgical department includes:

1) Clinical blood test – possible rise of leucocytosis and the ESR.

2) Clinical analysis of urine – possible signs of nephropathy.

3) Biochemical blood test: at the atherosclerotic affectation of heart and main arteries the characteristically rise of level of cholesterol, lipoproteins.

4) Coagulogramm – as usually present hypercoagulation (lowering the time of coagulation, rise of prothrombin index, fibrinogen A, fibrinogen B, fibrin and other).

5) Oscillography (in the conditions of OPD) is acute lowering of oscillatory index (OI).

6) Reovasography longitudinal segmental (in the conditions of policlinic) is acute lowering of reographic index (RI), intensive decline of form of reographic curvature.

7) Ultrasonic *investigation of vessels* (including ultrasonic dopplerography), allowing to define even the terms of formation of blood clots,

8) Angiography, allowing most exactly to define not only a level and extent of acute occlusion and chronic stenotic process but also to get the clear picture of its extent, expressed of concomitant arteriospasm, intensity of collateral circulation of blood; punction femoral arteriography by Seldinger, puncture translumbal abdominal aortography (by Dos Santos), punction arteriography (by Seldinger) of arteries of upper extremity.

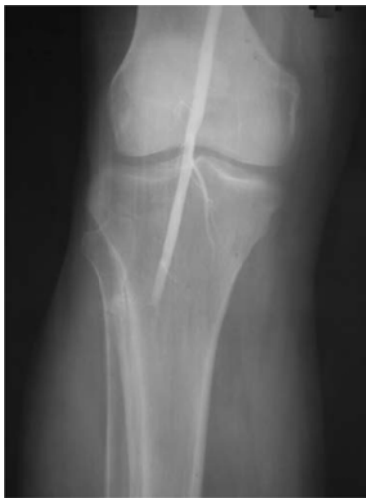

Fig. 1. Popliteal artery embolism In its bifurcation

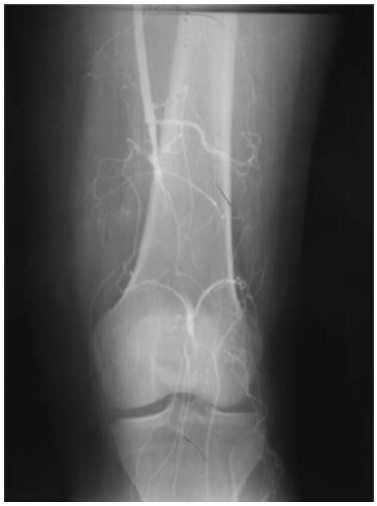

Fig. 2. Acute thrombosis of the superficial femoral artery in the lower third in its atherosclerotic lesions

Angiographic signs: at AE at partial obturation of artery – the embolus is good bypassing by contrast and on arteriogram has the appearance of oval or round formation; at complete occlusion there is sharp «precipice» of shade of vessel with the well visible protuberant high bound of embolus (as the symptom of the «inverted cup» – V. S. Savelev) (Fig. 1), the contours of leading segment of obturated artery are even, smooth, collaterals, as a rule, is absent; *at AAT* the internal contours of artery are uneven, filling by the contrasted blood is unequal, sometimes there is the phenomena of fadeaway of contrasting material, the level of thrombosis has the form of uneven line with coulisses, a collateral network often is well developed (Fig. 2).

Differential diagnostics

Differential diagnostics is conducted between the syndrome of AAI of extremity and dissecting aneurism of aorta, with ”white pain phlegmasia”, and then – between arterial embolism and acute arterial thrombosis of extremity.

Ground and formulation of clinical diagnosis at a patient (taking into account classification of disease, presence of complications and concomitant pathology are possible variants):

I variant:

1) basic – post infarction cardiosclerosis, post-infarction aneurism of the left ventricle of heart, fibrillation of heart.

2) complications (of basic disease): arterial embolism of left lower extremity (bifurcation of the general femoral artery), acute ischemia of the III degree.

3) concomitant pathology (if there is).

II variant:

1) basic - obliterated atherosclerosis of the right lower extremity, chronic ischemia of the

III degree.

2) complications (of basic disease) – acute arterial thrombosis (right superficial femoral artery), acute ischemia of the II degree.

3) concomitant pathology (if there is): GSD, I stages. Chronic relapsing cholecystitis.

Table 1

Differential diagnostics of different types of OAN extremities

№	Differential-diagnostic sign	Embolism	Thrombosis
1.	Basic disease	Atherosclerotic and rheumatic affectation of heart, fibrillation of heart	Chronic occlusive diseases of arteries of extremity
2.	Beginning of disease	Sudden	Relatively slow
3.	Pain syndrome	Very intensive	More frequent moderate
4.	Acute ischemia	It is sharply expressed, is expressly limited	It is moderately expressed, is not sharply outlined
5.	Colouring of skin of extremity	Pale, almost white or «marble»	Pale
6.	Skin temperature	It is sharply reduced	It is reduced moderately
7.	Motive function	Ischemic paralysis	Lowering of muscles force
8.	Skin sensitiveness	Anaesthesia	It is reduced, but stored
9.	Angyography	Contrasted main vessel with the unchanged wall, with the clear level of precipice with a protuberant and smooth high bound as the «inverted cup», collaterals is absent	Main vessel with the uneven «eaten» contours, the line of precipice is unsmooth, expressed network of collaterals.

Treatment of patient with the syndrome of AAI of extremity

Choice of medical tactic:

Treatment of patients with the syndrome of AAI extremities must begin after establishment of diagnosis. *Providing of the first medical aid in period before hospitalization* consists in administration of the medicine directed *on warning of making progress of acute ischemia* and extension the same of viability of tissues of extremity. *For the removal of pain* isintravenous introduction of promedoly with dimedroly or even morphine; *for the fight against arteriospasm and with the purpose of opening of collaterals* is intravenous introduction of big doses of spasmolitics (no-shpa, papavering, complamyn, galidor and other.); *for prevention of further growth of the continued thrombosis* is intravenous introduction a 10–15 thousand of UN heparin. *For the improvement of central hemodynamic* – intravenously cardiac glycosides (corglycon, strophantin), in case of indication antiarhythmic drags (novocainamyd and other.). It is necessary *to forbid*performing of massage, warming of distal parts of extremity and *quickly as posible to hospitalize in the specialized vascular department.*

In the vascular department *the choice of method of treatment* depends, above all things, *from etiology* of syndrome of AAI of extremities.

Establishment of diagnosis *of «arterial embolism» with the acute ischemia of the I–III degrees is a indication to urgent operative interference – implementation of operation of embolectomy*, it means, that **AE must be treated by a surgical method.**

The exception is made by patients which have absolute contra-indications to this operation:

1) extremely grave condition of patient, eliminating implementation of any on volume operative interference,

2) at emboli peripheral arteries of small diameter (shin and forearm), when conservative therapy is used,

3) waiver of patient and relatives from operation. All the other possible contraindications are relative and is considered individually in every special case.

Choice of method of treatment of patients *with acute arterial thromboses* – more intricate problem and depends, mainly, *from the degree of sharp ischemia:* at the I easy degree of ischemia conservative therapy is indicated; at the II degree of ischemia (middle) is a trial conservative therapy, in case ineffectivety the urgent deferred operative treatment, at the III heavy degree of acute ischemia and at the IV degree in the stage of gangrene is urgent operative treatment after a necessity, minimum on volume, inspection and short preoperative preparation.

Surgical treatment of arterial embolisms

For treatment of AE of extremities and bifurcation of aorta in present time proposed operation *of embolectomy*, which can be considered the radical method of treatment. Proposed 2 methods of embolectomy:

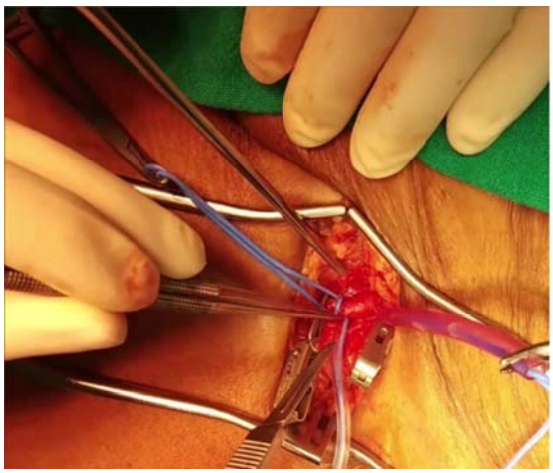

Fig. 3. Direct embolectomy

1) *direct embolectomy* – the artery is opening directly over the place of embolus localization – embolus removing (bifurcation of general femoral, humeral arteries, rarer – popliteal arteries) (Fig. 3);

2) *indirect embolectomy* – performs in cases if the affected section of the artery is very difficult to access (bifurcation of general iliac artery, aortas, at localization of embolus in a superficial femoral artery above the entrance in a Gunter's channel, in a humeral artery on a shoulder). In this case performing arteriotomy of the superficial artery and than the thrombus removes by catheter of Fogarty (thin by a diameter to 2–2,5 mm elastic probe with a rubber inflateble bulb on an end) (Fig. 4).

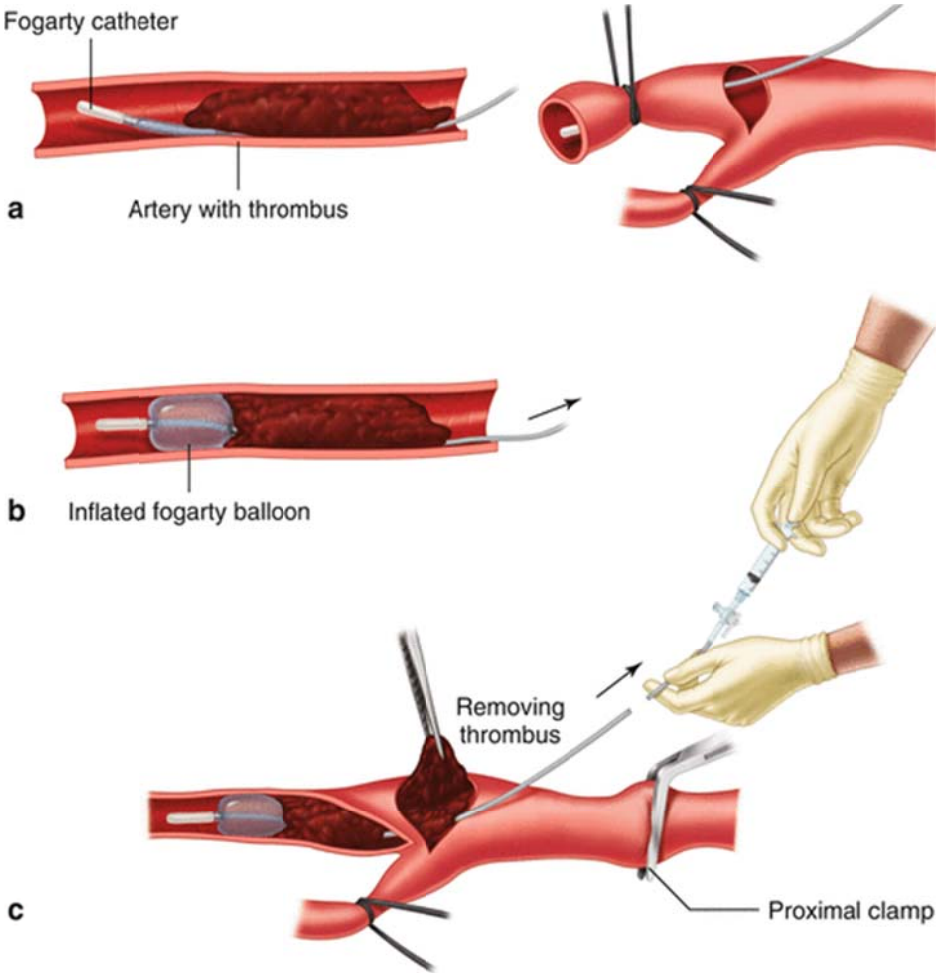

Fig. 4. Indirect embolectomy by catheter of Fogarty (a, b, c)

At production of both types of embolectomy more typical accesses are: in upper third of thigh below inguinal ligament with baring of bifurcation of general femoral artery, in upper third of forearm below than elbow bend for baring of bifurcation of humeral artery, in a u/3 shin on a back surface below than popliteal pit.

After implementation of direct or indirect embolectomy sutures artery (more frequent by Karrel) if transversal arteriotomy was produced, after

longitudinal arteriotomy in order to avoid stenosis and postoperative thrombosis is closing of the arteriotomy opening by sewing of «patch» from autovein (segment of gig hypodermic vein). In case of absence of bulb catheter for implementation of indirect embolectomy it is possible to use the polyethylene catheters of a different diameter for sucking of embolus. All types of embolectomy are produced under the local anaesthetizing, that allows them to execute without the special risk even at heavy patients (Fig. 5).

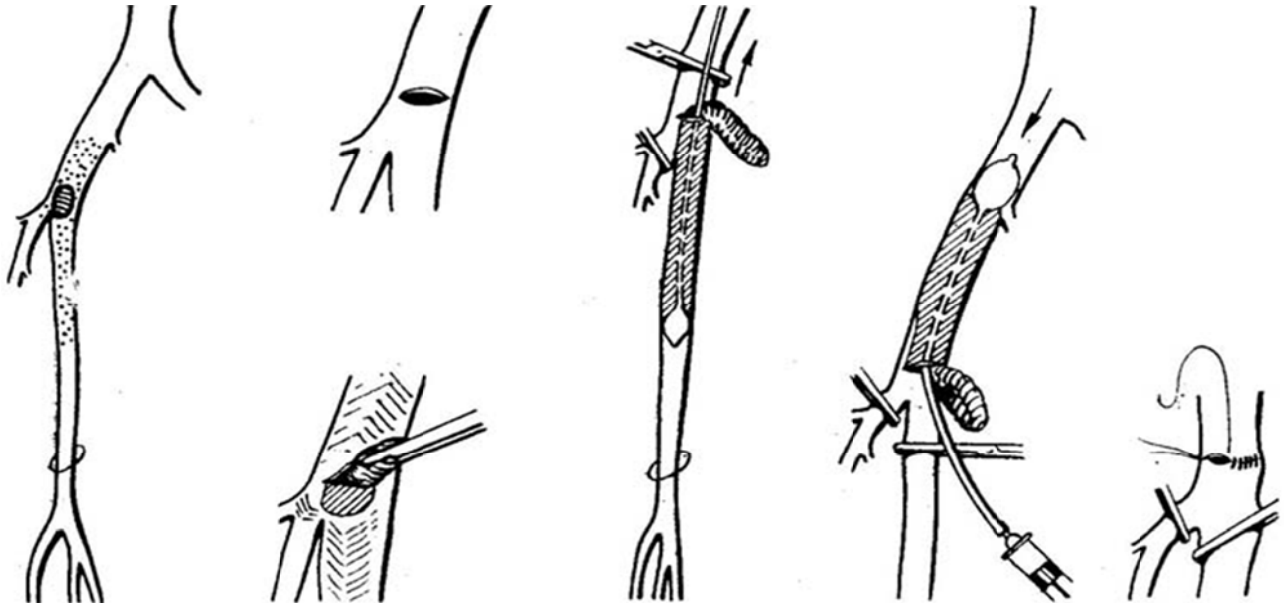

Fig. 5. Scheme stages of operations embolectomy,
a, b – direct embolectomy from the common femoral artery;
c- indirect embolectomy of the external iliac artery; d – artery suture

In a postoperative period – anticoagulant therapy (heparin, it is better – LMH), desagregants, hemocorrectors (for the prophylaxis of postoperative thrombosis), treatment of basic disease, decision of question about possibility of radical correction for the prophylaxis of the possible relapse of AE.

Treatment of acute arterial thromboses

Conservative therapy is indicated:

1) at the easy degree of acute ischemia of tissues of extremity,

2) in quality a trial therapy at the middle degree of ischemia,

3) when the heaviness condition of the patient does not allow to produce operation in spite of present indications to it,

4) at the waiver of patient of operation,

5) at the acute thromboses of peripheral arteries of small diameter (shin and forearm).

Tasks of conservative treatment: the lysis of blood clot with restoration of blood stream in a affected artery, prophylaxis of growth and distributions of blood clot, improvement of circulation of blood and tissue metabolism in the

area of acute ischemia, the improvement of function of vital important organs, prophylaxis of making progress of basic disease.

Thrombolitic and anticoagulant therapy is the pathogenetic grounded therapy at AAT, directed on renewal of ability of passage of blocked artery, on the fight against growth and distribution of the continued thrombosis, the same – on the improvement of circulation of blood and tissue metabolism in the affected extremity.

Thrombolitic preparations are possess a high lysing effect, either directly affecting a blood clot or activating the own fibrinolytic system of patient, being the activators of proactivators nonactive plasminogen and blocking action of inhibitors of fibrinolysis. Very effective thrombolitic is *«streptokinase» («streptase» – Federal Republic of German)* is high-cleared albumen producing by actively growing b-hemolytic streptococcus of group C, possessing properties of activator of proactivators of plasminogen and are the cause of lysis of blood clot ("indirect" thrombolitic).

In connection with present practically at all people in a blood the antistreptococcus antibodies, thrombolitic therapy by these preparations begin with intravenous introduction of initial «inactivated» dose 250 000 UN on a 300 ml of physiological solution or 5 % of solution of glucose during 30–40 minutes, and then continuous intravenous infusion a medical dose of preparation, which can achieve a 1500000–3000000 UN (speed of infusion – 750 000 UN for 8 hours), is conducted under the angiography control, with next transition on heparin therapy.

To avoid of basic complication of thrombolitic therapy – a different *bleeding*, the careful laboratory control for the state of hemocoagulation system is needed: research *every 8 o'clock* of indexes of coagulogramm: time of coagulation of blood, concentrations *of fibrinogen A*, thrombin time, *fibrinolytic activity of blood*.

In case of occurring of hemorrhagic complications (bleeding sickness of wounds, micro- and macrohematuria, indexes of coagulogramm) – introduction of thrombolitic is temporally halted, as antidote therapy introduction of fibrinogen is used, 5 % of solution e-amynocapronic acids, 1 % of solution of chloride calcium, nativeor fresh-frozen plasma, 1 % of solution of ambena, direct blood transfusion is used in especially heavy case. After stopping of manifestation hemorrhage is continues thrombolitic therapy for the getting of therapeutic effect, but slower introduction of preparation.

For diminishment of toxic and antigen influences of thrombolitic administration hormonal (prednisolon and other) and desensitizing (suprastine, tavegile, pipolfeneand other) drags is indicated.

To other preparations of this group is regard: cabicinase (Sweden), *streptodecase, streptoliase, celiase, urocinase*.

The increase of efficiency of thrombolisis is related to creation in 90x years a new *specific thrombolitic* preparation – synthetic *tissue activator of plasminogen*

actilise (operating matter – alteplase), after *metalyse, reteplase, tenecteplse* (group of "direct" thrombolitics).

Anticoagulant therapy on which pass after ending of thrombolitic, possessing by *anti thrombotic action,* lowering the coagulative potential of blood, prevent the formation and growth of the continued blood clot, blocking of collaterals, possesses spasmolitics action and reduces viscidity of blood, but does not dissolve an appearing blood clot and does not liquidate embolus.

Anticoagulant therapy, as a rule, begin by administration *of direct anticoagulant – heparin,* which is entered only parenteraly, it is optimum intravenously, for 5000–10000 UN (1–2 ml) in every 3–4 hours (as for this time it removing from an organism), day's medical dose makes from 40 000 to 80 000 UN; course of treatment by heparin – to 5 days with the gradual decline of dose and subsequent transition on indirect anticoagulants.

Heparin, as well as *thrombolitic*, can cause *hemorrhagic complications* (although it is marked relatively rarely in connection with that he not accumulating in a liver). Therefore at it uses the careful laboratory control is also required: *daily research of* basic indexes of coagulogramm (basic control index – *time of coagulation of blood by Ly-Uayt, which for effective therapy lengthens up to 20–30 mines), and microscopy* of urine for detection *of microhematuria.*

At appearance *of signs of overdose* (bleeding sickness of wounds, micro- and macrohematuria, indexes of coagulogramm) – 1–2 introductions of heparin are stopped, *solution of protamine-chloride or protamine-sulfate* (intravenously 5–10 ml on normal saline) is applied as *antidote*, after continue the heparin therapy by reduced doses. *It is impermissible sharply to halt heparin therapy*, due to possibility of development of opposite complication – so-called «ricochet effect», when after sudden abolition of heparin therapy coagulation of blood sharply increases and rethrombosis can develops.

In present time heparin is successfully replaced lower molecular *heparins* – fraxiparin (for 30 mg – 0,3 ml in a syringe – 2–3 times per days), *clexan (enoxaparin* – for 20–40–80 mgs – 0,2–0,4–0,8 ml in a syringe – 2–3 times per days); these preparations are entered only hypodermic in area of anterior abdominal wall.

After ending of course of heparin therapy pass to *the indirect anticoagulants – neodicumarin (pelentan), fenylyn, sinkumar, omefin, varfarin*, which possess, besides the used antythrombotic property, by negative effects: extremely variably sensitiveness to them of different patients (at some is increased, when even from ordinary doses and bleeding develops at brief application, at other is the expressed tolerance, when the even increased doses have an insignificant effect), by ability of accumulation in a liver and at long application to cause the massive bleeding. Due to it for every patient the medical dose of indirect anticoagulant must be picked up *individually*, since the standard dosage (for to a 1 pill 3 times per a day), and then, daily checking up the level of prothrombin index, to pick up such dose

which will support this index of coagulogramm at level necessary for a medical effect – 35–40 %. To avoid dangerous accumulation of preparation, the course of treatment must make no more than 7–10 days.

Rules of therapy by indirect anticoagulants the same, as well as direct: they can give similar complications – hemorrhages (at an overdose or protracted application), «ricochet effect» (sharp abolition). *The very careful laboratory control* is therefore *needed:* after the selection of dose *every 3 days t*he basic control index of coagulogramm for indirect anticoagulants is explored of *prothrombin index* (which must not lowering down below than the therapeutic level 35–40 %) and the microscopy of urine for the detection of *microhematuria is produced*, and by the end of course is gradual decline of dose to minimum one (0,5 tabl. 1–2 times per a day).

It is necessary to remember *a transition rule from direct to the indirect anticoagulant*: indirect anticoagulants have another feature related to the mechanism of action – at introduction to the organism they reduce coagulation not at once, and through same period of time (it is more frequent 12–24 hours), in this connection there is the rule, which consists that in the day of beginning of therapy by an indirect anticoagulant yet during days introduction of direct anticoagulant is continued in a minimum dose, abolishing it on next days from the beginning of application of indirect anticoagulant.

At appearance *of signs of overdose of indirect anticoagulant* (bleeding sickness of wounds, micro- and macrohematuria, indexes of coagulogramm) – 1–2 introductions of preparation are skipped, *a 1 % solution of vitamin K* (vicasolum – intravenously 5–10 ml) is applied in quality of *antidote*, treatment by the reduced doses is after continued. *It is impermissible sharply to halt introduction of indirect anticoagulant,* as well as at heparin therapy, development of opposite complication is possible – so-called *«ricochet effect»*, when after sudden abolition of preparation sharply rises coagulative activity of blood and rethrombosis can develop.

New standards of thrombolitic and anticoagulant therapy are presently offered allowing to improve the nearest and remote results of “system” thrombolisis by perfection so-called the adjuvant anticoagulant and antiaggregation therapy applied *simultaneously with thrombolitics*.

For the increase of clinical efficiency of thrombolitic therapy advantage of application of lower molecular heparins (LMH) is proved, mainly enoxiparyn (clecsan), as compared to usual heparin thus not only at the use of fibrinspecific thrombolitics but also at application of streptokinase.

In complex conservative therapy includes hemocorrectors, desagregants (acetylsalicylic acid, clopydogrel (plavix, ticlopidin), spasmolitics, cardiac glycosides, antiarrhythmic preparations (on indications), conducts correction of the acid-alkaline state, measure on normalization of function of kidneys (at its violation).

Conservative therapy is used as independent method of treatment and is obligatory addition to operative treatment.

The following *restoration* operations are used *in surgical treatment of AAT of extremities*: «ideal» thrombectomy (rarely) or combination of it's with endarterectomy, endarterectomy with auto venous plastic, resection of artery with prosthetics and permanent bypass shunting (in area of bifurcation of aorta and iliac arteries – synthetic prosthetic appliances, on extremity – autovein). Operations are executed under the common anaesthetizing.

In the case of *contracture of muscles of shin* at acute arterial violations of blood circulation due to emboli or thromboses with the purpose of diminishment of danger of development of threatening post-ischemic disorders is recommended, along with deleting of thrombus-embolus and the continued blood clot, to perform phlebotomy of main concomitant vein with bloodletting for deleting of unoxidated toxic products, or conducting of regional perfusion of extremity.

At the developing *edema of muscles* with a purpose decompressions and improvements of blood stream wide fasciotomy is indicated. At development *of the IV degree of sharp ischemia* – stage of gangrene both at AE and at AAT *amputation of extremity* is indicated. It is necessary to remember at the choice of level of amputation, that the ischemic affectation of muscles is usually expressed in a greater degree, than skins.

Prophylaxis of emboli and acute thromboses of arteries of extremities consists in radical surgical correction of embologenic diseases (implementation of comissurotomy at miter stenosis, excision of post infarction aneurism of the heart, resection of aneurism of aorta) and chronic occlusion diseases of arteries of extremities (implementation of reconstructive operations). In the cases when gravity of the common state or age of patients does not allow to produce these interferences, lifelong anticoagulant therapy is indicated by indirect anticoagulants with the individual selection of dose at which prothrombin index would not exceed 60 % (more frequent than all varfarin is used).

Principles of examination disability and prophylactic medical examination. Patients with the syndrome of AAI of extremities must be under the medical supervision. Two times in a year and on indication and more must be examined by therapeutic and vascular surgeon. Very necessarily is to control the indexes of coagulogramm. If it necessary a surgeon appoints ultrasonic of main arteries of extremities.

CONTROL TESTS

1. For acute arterial obstruction is not characteristic:
A) lack of pulse
B) parasthesia
C) trophic ulcers
D) plegia

E) limb pain
ANSWER: C

2. Thrombosis develops as a result of all except
A) coagulation disorders
B) changes of an endothelium of the vessel
C) massive bleeding
D) stasis of blood flow
E) embolism
ANSWER: C

3. Which criterias can help to differentiate arterial embolism from thrombosis:
A) severity of pain
B) lack of pulse in the distal arteries
C) presence of source of embolism
D) based on all the listed criteria
E) all criteria are not informative
ANSWER: C

4. Which criterias can help to differentiate arterial embolism from thrombosis:
A) speed of development of ischemia
B) severity of pain
C) lack of pulse in the distal arteries
D) based on all the listed criteria
E) all criteria are not informative
ANSWER: A

5. Which criteria can help to differentiate arterial embolism from thrombosis:
A) "intermittent claudication" in the anamnesis
B) lack of pulse of the distal arteries
C) severity of pain
D) based on all the listed criteria
E) all criteria are not informative
ANSWER: A

LITERATURE

1. Aboyans V, Ricco JB, MEL B, Björck M, Brodmann M, Cohnert T, Collet JP, Czerny M, De Carlo M, Debus S, et al. 2017 ESC guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS): document covering

atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries. Endorsed by: the European stroke organization (ESO) the task force for the diagnosis and treatment of peripheral arterial diseases of the European Society of Cardiology (ESC) and of the European Society for Vascular Surgery (ESVS). Eur Heart J. 2018; 39:763–816.

2. Anand SS, Caron F, Eikelboom JW, Bosch J, Dyal L, Aboyans V, Abola MT, Branch KRH, Keltai K, Bhatt DL, et al. Major adverse limb events and mortality in patients with peripheral artery disease: the COMPASS trial. J Am Coll Cardiol.2018; 71:2306–2315. doi: 10.1016/j.jacc.2018.03.008

3. Bonaca MP, Bauersachs RM, Anand SS, Debus ES, Nehler MR, Patel MR, Fanelli F, Capell WH, Diao L, Jaeger N, et al. Rivaroxaban in peripheral artery disease after revascularization. N Engl J Med.2020; 382:1994–2004. doi: 10.1056/NEJMoa2000052

4. Bugaev N, Como JJ, Golani G, Freeman JJ, Sawhney JS, Vatsaas CJ. Thromboelastography and rotational thromboelastometry in bleeding patients with coagulopathy: practice management guideline from the eastern association for the surgery of trauma. J Trauma Acute Care Surg.2020; 89:999–1017. doi: 10.1097/TA.0000000000002944

5. Gerhard-Herman MD, Gornik HL, Barrett C, Barshes NR, Corriere MA, Drachman DE, Fleisher LA, Fowkes FGR, Hamburg NM, Kinlay S, et al. 2016 AHA/ACC guideline on the management of patients with lower extremity peripheral artery disease: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. Circulation.2017; 135:e726–e779. doi: 10.1161/CIR.0000000000000471

6. Guirgis M, Thompson P, Jansen S. Review of aspirin and clopidogrel resistance in peripheral arterial disease. J Vasc Surg.2017; 66:1576–1586. doi: 10.1016/j.jvs.2017.07.065

7. Gupta R, Majumdar M, Imran R, Yi J. A comprehensive review on antithrombotic therapy for peripheral artery disease. Semin Vasc Surg.2022; 35:124–131. doi: 10.1053/j.semvascsurg.2022.04.004

8. Hartinger J, Novotny R, Bilkova J, Kvasnicka T, Mitas P, Sima M, Hlubocky J, Kvasnicka J, Slanar O, Lindner J. Role of dipyrone in the high on-treatment platelet reactivity amongst acetylsalicylic acid-treated patients undergoing peripheral artery revascularisation. Med Princ Pract.2018; 27:356–361. doi: 10.1159/000489970

9. Hussain MA, Al-Omran M, Creager MA, Anand SS, Verma S, Bhatt DL. Antithrombotic therapy for peripheral artery disease: recent advances. J Am Coll Cardiol.2018; 71:2450–2467. doi: 10.1016/j.jacc.2018.03.483

a. Jay D. Coffman, Robert T. Eberhardt. Periferal arterial disease: diagnosis and treatment. Human Press, 2003, 356 p.

10. Kim Y, Patel SS, McElroy IE, DeCarlo C, Bellomo TR, Majumdar M, Lella SK, Mohebali J, Dua A. A systematic review of thromboelastography

utilization in vascular and endovascular surgery. J Vasc Surg.2022; 75:1107–1115. doi: 10.1016/j.jvs.2021.11.037

11. Majumdar M, Lella S, Hall RP, Sumetsky N, Waller HD, McElroy I, Sumpio B, Feldman ZM, Kim Y, DeCarlo C, et al. Utilization of Thromboelastography with platelet mapping to predict infection and poor wound healing in postoperative vascular patients. Ann Vasc Surg.2022. Online ahead of print. doi: 10.1016/j.avsg.2022.03.008

12. Markel KM, Avgerinos ED. Clopidogrel resistance in lower extremity arterial endovascular interventions. Curr Pharm Des.2018; 24:4554–4557. doi: 10.2174/1381612825666190101111123

13. Shu J, Santulli G. Update on peripheral artery disease: epidemiology and evidence-based facts. Atherosclerosis.2018; 275:379–381. doi: 10.1016/j.atherosclerosis.2018.05.033

14. Zierler RE, Jordan WD, Lal BK, Mussa F, Leers S, Fulton J, Pevec W, Hill A, Murad MH. The society for vascular surgery practice guidelines on follow-up after vascular surgery arterial procedures. J Vasc Surg.2018; 68:256–284. doi: 10.1016/j.jvs.2018.04.018