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KHARKIV NATIONAL MEDICAL UNIVERSITY

SURGERY

Textbook for Vth year students of medical faculties

Thoracic, cardiovascular, endocrine surgery

Kharkov 2017

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In connection with the transition of teaching at universities in Ukraine on the principles of credit-modular system of learning from ESTS IV year medical students learn surgery on the new model curriculum «Surgery», which includes 5 modules.

Under the new program, students study surgery V courses on topics of Module 2 «Thoracic, cardiovascular, endocrine surgery».

The textbook combines educational materials of department, designed for classroom study of 7 themes in practical classes and extracurricular self-study in 5 subjects according to the thematic plans of discipline «Surgery» on the topics of Module 2.

Each topic is outlined in the textbook, contains sections that correspond to professionally-oriented tasks of medical practice, approved by industry standards of Higher Education — «Educational and professional program» and «Educational qualifying characteristic» (Kiev, 2003) as amended and updated in 2010 year.

Prepared by the teaching staff of the Department of Surgery № 1 KNMU tutorial is designed to provide students the necessary teaching materials comprehensively on all topics of practical training and for self extracurricular work that will improve the training of future doctors surgery according to the requirements of national industry standard for higher education.

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Contents

Foreword

Ukraine's integration into the European community dictates the need to restructure higher education in the country in this direction and teaching at universities in Ukraine according to the principles of the Bologna Declaration, which will prepare young Ukrainian professionals compete with graduates of European and other institutions of higher education.

In Kharkiv National Medical University is over-passage teachers of all disciplines, including surgery, for all courses on the principles of credit-modular system of training ESTS.

Starting from the IV course, students learn surgery on the new model curriculum "Surgery", which includes 5 modules, for students in higher educational institutions III-IV of the levels of accreditation in the field of 7.110101 "Medicine", 7.110104 "Pediatrics" and 7.110105 "Medical-prophylactic case" (Kiev, 2008).

Under the new program, students of Vth year learn surgery on the subject of the module 2, "Thoracic, cardiovascular, endocrine surgery", consisting of two informative modules 3 and 4. The textbook combines educational materials of department, dedicated for practical training (classroom studying 7 themes № 16-22) and for self extracurricular study of 5 subjects according to the thematic plans of discipline "surgery" on the subject of Module 2.

Each topic is outlined in the textbook, contains sections that correspond to professionally-oriented tasks of medical practice, approved by industry standards of Higher Education – "Educational and professional program" and "Educational qualifying characteristic" (Kiev, 2003). Highlights issues of etiology and pathogenesis, classification of disease according to ICD-10 (2001), clinical symptoms, standard regimens of additional laboratory and instrumental examination of the characteristic features of evidence-based medicine for each disease, emergency care at the prehospital stage under emergency conditions in surgical patients, justifies the choice of treatment tactics , is pathogenetically grounded conservative therapy, indications and types of surgical interventions.

Considerable attention is paid to modern minimally invasive interventions — video endoscope, VATS, X-ray endovascular.

For each topic listed recommended modern literature.

Prepared by the teaching staff of the Department of Surgery № 1 textbook is designed to provide students the necessary teaching materials integrated in all themes of workshops and self extracurricular work that will improve the training of future doctors surgery according to the requirements of national industry standard for higher education.

LIST OF ABBREVIATIONS

AA — aortic aneurysm
AAO — acute arterial obstruction
AAT — acute arterial thrombosis
ABB — acid—base balance
ABI — ankle—brachial index
ACA – aminocapronic acid
ACD — acute coronary death
ACE – angiotensin converting enzyme
ACED — anticholinesterase drugs
ADVT — acute deep vein thrombosis
AE — acute empyema
AE — arterial embolism
AIS — abdominal ischemic syndrome
AIVB — anterior interventricular branch
ALA – acute lung abscess
AMI — acute myocardial infarction
API — associated with proteins iodine
APTT — active partial thromboplastin time
APV – artificial pulmonary ventilation
ARD – acute respiratory disease
AS — aortic stenosis
AVB — atrioventricular block
AVI — aortic valve insufficiency
AVRI – acute viral respiratory infection
BAP — bone alkaline phosphatase
BC — breast cancer
BE — bronchiectasis
BEI — butanol extractable iodine
BMD — bone mineral density
BNUP — brain natriuretic peptide
BP — blood pressure
BT – breathing tube
CA — coronary artery
CAB — coronary artery bypass
CAG — coronary angiography
CB — circumflex branch
CCT — quantitative computed tomography
CE — chronic empyema
CHD — coronary heart disease
CI – calcium index
CIC – circulating immune complex
CLA — chronic lung abscess
CODALE — chronic obliterative diseases of arteries of lower extremities
CPK — creatine phosphokinase
CPK—MB — creatine kinase MB fraction.
CPR — cardiopulmonary resuscitation
CT — computed tomography
CVILE — chronic venous insufficiency of lower extremities
CVP — central venous pressure
DBD — dishormonal disease of the breast
DCM — diffuse cystic mastitis
DFA — diffuse fibroadenomatosis
DFS — diabetic foot syndrome
DLF — destructive lesions of the feet
DM — diabetes mellitus
DVT — deep vein thrombosis
E – emergency
ECG — echocardiogram
ECG — electrocardiogram
EF — ejection fraction
EPT – electropulse therapy
ESR – erythrocyte sedimentation rate
GRH — gonadotropin—releasing hormone
HBO — hyperbaric oxygenation
HF — heart failure
HH — hiatal hernia
HR — heart rate
Ht – hematocrit
HT – hemothorax
HT — hormone therapy
INR — international normalized ratio
ITA — internal thoracic artery
LA — left atrium
LCA — left coronary artery
LCB – lack of circulating blood
LDL — low density lipoproteins
LITA — left internal thoracic artery
LM — lactational mastitis
LMWH — low molecular weight heparins
LV — left ventricle
MASD — Morgagni-Adams-Stokes disease
MCT — multispiral computed tomography
MI — mitral insufficiency
MIMR — minimally invasive myocardial revascularization
MOF — multiple organ failure
MRI — magnetic resonance imaging
MVS — mitral valve stenosis
NAID – nonspecific anti—inflammatory drugs
NFA — neuroleptanalgesia
NYHA — New York Heart Association
OI — oscillographic index
PCG — phonocardiogram
PCT — polychemotherapy
PE — pulmonary embolism
PH — pulmonary hemorrhage
PM — pacemaker
PO — pump oxygenator
PT – pneumothorax

PTFE — polytetrafluoroethylene
PTS — postthrombophlebitic syndrome
RGEA — right gastroepiploic artery
RI — rheographic index
RITA — right internal thoracic artery
RTA – road traffic accident
RV — right ventricle
SCLC — small cell lung cancer
SSS — sick sinus syndrome
SVCS — superior vena cava syndrome
TBA — transluminal balloon angioplasty
TG — thyroid gland
TMLR — transmyocardial laser revascularization of myocard
TNAB — thin needle aspiration biopsy needle
TS — tricuspid stenosis
TSH — thyroid stimulating hormone
TSI — thyroid stimulating immunoglobulin
TV — tricuspid valve
UA — upper airways
UG — unfractionated heparin
US — ultrasound
VC — vital capacity
VTE — venous thromboembolism
VV — varicose veins
WHO – World Health Organization
WPWS — Wolff-Parkinson-White syndrome

Chapter I. Surgical pathology of the respiratory system

I.1. Purulent diseases of lungs and pleura

Acute pulmonary suppurations more frequent arises up in mature age, is more common at men which are undergoing to this pathology in 3-4 times more frequent, than women, that is explained by abuse by an alcohol, by smoking, by greater susceptibility to supercooling, and also professional harmful.

A right lung is affected in 60%, in 34% is left and in a 6% affectation may be bilateral. Big frequency of affectation of right lung is conditioned by the anatomical features of it structure: wide right main bronchi which as prolongation of trachea may an instrument for passage in the right lung of infected material.

Acute abscess of lung

The acute abscess (simple, generalized) of lung regard to the group of purulent-destructive affectation of this organ and primary are the cause of necrosis of pulmonary parenchyma. Than depending on resistant of organism of patient, type of microbial flora and correlation of alterative-proliferate processes, evolution or sequestration with separation of necrotic focuses or making progress of purulent-putrid lysis of surrounding tissues.

A pathological process in lungs is here characterized by dynamism and one form of development of disease can pass to other.

The acute (simple) abscess of lung (AAL) is the purulent or putrid lysis of necrotic focuses of pulmonary tissues, more frequent only within the limits of one segment with forming of one or a few cavities filled by pus and surrounded by peryfocal inflammatory infiltration of pulmonary tissues. A purulent cavity in a lung here more frequent than all is delimited from the unaffected areas by a pyogenic capsule.

A gangrenous abscess is purulent-putrid disintegration of area of necrosis of pulmonary tissues (lobe, segment) is characterized by propensity to sequestration and poor separation from the unaffected areas. A gangrenous abscess is yet named the delimited gangrene.

The following classification of purulent diseases of lungs by A. A. Shalimov is accepted in a clinic and is used:

I. Acute purulent diseases of lungs:

1) acute abscessing pneumonia;
2) acute single abscesses;
3) acute plural abscesses;
4) acute gangrenous abscesses;
5) widespread gangrene.

II. Chronic purulent diseases of lungs:

1) chronic pneumonia;
2) chronic single abscesses;
3) chronic plural abscesses;
4) purulent-inflammatory bronchiectases on etiology:
   a) acquired;

b) congenital;
c) cylindrical;
d) saccular;
e) mixed.

5) purulent-inflammatory cysts of lungs;
6) purulent-inflammatory parasite cysts of lungs (echinococcus);
7) pneumosclerosis;
8) purulent- inflammatory polycystosis of lungs;
9) mycosis purulent-inflammatory processes (actinomycosis, aspergillosis)

Complaints from the other organs and systems: cardio-vascular, organs of gastro-intestinal tract, genitourinary system, lymphatic and nervous system.

Anamnesis of disease: at collection of anamnesis it is necessary to find out when and as a patient became ill; did not have flu the day before, ARD, by pneumonia; where treated and what kinds of preparations; used antibiotics and what; what is the cause of disease according to opinion of patient (supercooling, stress and other).

At AAL beginning of disease can be both acute on background prosperity and on a background the improvement of the state (after carried ARD, pneumonias), a high temperature, chill appears and etc.

Anamnesis of life: it is necessary to find out the terms of work (work with harmfuls, dusty, toxic gases); how often has the cold diseases; use of alcohol, smoking, narcotics; peculiarities of life of patient; is he under observation due to TB or has permanent contact with TB patient. All these information can specify on the fact of decline of immunity as factors promote to the AAL development. At women it is necessary to find out obstetric-gynecological anamnesis (amount of pregnancies and births, carried inflammatory diseases, tumors of appendages, uteruses and etc).

Clinic-physical inspection (characteristic features at this disease).

1) Estimation of the common state of patient (state of consciousness, constitution, fatness).

At patients with AAL the state of middle heavity or heavy, is special at patients with the gangrenous abscess of lung or at patients with the plural abscesses of lungs. Consciousness is clear, but at the heavy condition of gangrenous abscess at the elderly persons it can be entangled due to a high temperature and intoxication. Constitutional peculiarities are not present, but some authors mark that AAL often meets at the asthenic persons with the lowered mass of body.

2) Collection of information about original appearance of patient (examination of skin, hypodermic-fatty layer, palpation of lymphatic nodes, thyroid and mammary glands).

At examination the pallor of skin covers pays attention, up to a grey-earthy color, cyanosis of mucous lips, general sweating is typical. Palpation of lymphatic nodes, thyroid and milk glands - without features.

3) Inspection of the cardio-vascular system: examination and palpation of area of heart and superficial vessels, palpation of main arteries of extremities and neck in projection points, determination of percutory border of heart, auscultation of heart and vessels, determination of the special symptoms at patients with AAL at examination of area of heart and superficial vessels, at palpation of main arteries of extremities and neck - without peculiarities. The percussion of borders of heart - within the

limits of normal. By auscultation marked tachycardia, A/D can be slightly raised. It is explained by the increase of temperature of body and intoxication.

4) Inspection of the state of organs of abdominal region (examination of abdomen, palpation and sucussion of stomach, intestine, livers, spleen, pancreas, kidneys, organs of small pelvis). At examination, as a rule, deviations from a norm do not detected. The lowering of appetite is marked. The characteristic changes are not present.

5) Inspection of the state of the bones-muscles system (examination and palpation). At patients with the AAL characteristic changes are not present.

6) «Locus morbi»: At examination the affected half of thorax delays in the act of breathing. By percussion is marked dullness of pulmonary sound over the place of location of abscess (in the phase of the purulent-necrotic lysis of lung), and after the opening of abscess to the bronchi may appears tympanic percutory sound. By auscultation in the first phase of disease detected weakening vesicular breathing, in the second phase may detected «amphoryc" breathing. Frequency of respiratory excursion depends on the volume of affectation of pulmonary tissues - different degree of expression tachypnea.

7) Leading clinical syndromes- acute respiratory insufficiency and intoxication.

8) On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose the following clinical diagnosis: ACUTE ABSCESS OF (UPPER, MIDDLE, LOWER) LOBE OF RIGHT/LEFT LUNG.

According to standard charts the plan of additional inspection (laboratory and instrumental) includes:

1) Clinical blood test.
2) Clinical analysis of urine.
3) Biochemical blood test.
4) Coagulogramm.
5) Immunological tests.
6) Bacteriological research of sputum.
7) Roentgenologic research of organs of thorax in two projections, tomography, on indication – bronchography.
8) Spyrography.
9) Fibrobronchoscopy diagnostic and treatment.

1. Clinical blood test: moderate anemia, leucocytosis with a deviation to the left, high ESR.

2. Clinical analysis of urine: the changes are unspecific.

3. Biochemical blood test: decline of level of general albumen, dysproteinemia.

4. Coagulogramm: violations of coagulation of blood to side of hypercoagulation.

5. Immunological tests: the decline of indexes of cellular immunity, decline of the A/G coefficient.

6. Bacteriological research of sputum, content of cavity of abscess: gives possibility detect microbe flora, which are the cause of destructive purulent process and define the sensitiveness of the last to antibacterial preparations.

7. Roentgenologic research of organs of chest in two projections: In the phase of the purulent – necrotic lysis of lung (phase of acute infiltration) on X-ray films determined infiltration of pulmonary tissues as focal (rounded) shade. In the

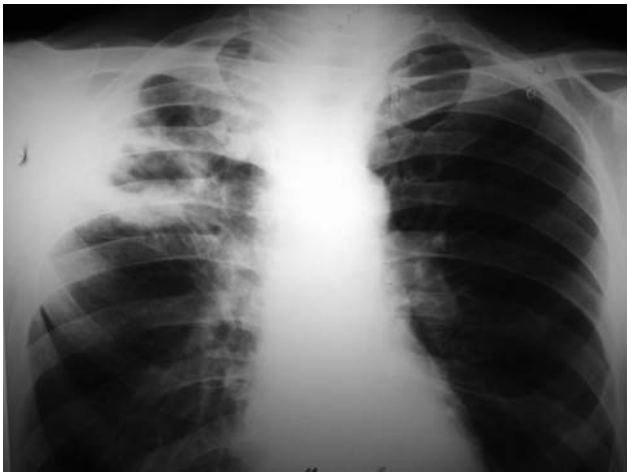


Fig. 1. Acute abscess of the upper lobe of right lung in draining stage

phase of draining of abscess through bronchi on X-ray film determined one or a few cavities of destruction, more frequent with the horizontal level of liquid and peryfocal inflammatory infiltration of pulmonary tissues around a cavity (Fig. 1). The superexhibited pictures or tomograms help to discover the cavities of disintegration in lungs. By tomography pulmonary sequesters can be diagnosed.

8. Spyrography — lowering the vital capacity (VC) and maximal ventilation of lungs (MVL) diminishment on 25-30%, the decline saturation by oxygen of the arterial blood less than 90%.

9. Fibrobronchoscopy: in the phase of draining of abscess through bronchi the phenomena of purulent tracheobronchitis is determined. It is possible to define localization of abscess by the mouth of draining bronchi.

The differentiated diagnosis at acute abscesses is necessary to be performed with cavernous tuberculosis, actinomycosis, echinococcus, suppuration of cyst of lung, interlobar encapsulated pleurisy, focal pneumonia, and also tumors and primary bronchiectases in the phase of abscessing. Cavernous tuberculosis is usually eliminated at the study of anamnesis, absence of tubercular sticks and roentgenologic changes of lungs characteristic for tuberculosis.

For actinomycosis the presence in sputum of druses is characteristic, finding out which is uneasy, the repeated careful researches are required in this case. For actinomycosis involving in the process of surrounding organs, pectoral wall is characteristic.

At the purulent parasite (echinococcus) and acquired cysts of lung the state of patient is not such heavy, as in case of acute abscess, is not marked preceding inflammation of lung; at roentgenologic research determined round, clear shades with absence of peryfocal inflammation.

Diagnostics at the interlobar pleurisies opened in bronchi and at encapsulated pleurisies are especially difficult. In such cases very useful repeated multiaxial roentgenoscopy by which it is possible to specify a diagnosis.

It is also difficult to distinguish from focal pneumonia the acute abscess before emptying of it through bronchi. The dynamic supervision of the patients, with repeated roentgenoscopy is helpful in differentiation

Abscesses at primary bronchiectases are the late phase of development of bronchiectatic disease and it is easily to distinguish according of anamnesis from the acute abscesses of lung.

It is necessary to remember that a acute abscess from the gangrene of lung can be distinguished on the clinical development, because all signs of acute abscess are expressed more considerably, intoxication is more expressed. Roentgenologic research at the gangrene of lung exposes the presence of the continuous darkening of part of lung on the side of affectation with gradual transition to the normal pulmonary picture on periphery. With development of putrid pyopneumothorax darkening occupies all half of thorax.

Ground and formulation of clinical diagnosis (according classification of disease, presence of complications and concomitant pathology):

1) basic — acute abscess of (upper, middle, lower) lobe of right/left lung;

2) complications (of basic disease if they are);

3) concomitant pathology (if it is).

Treatment of patient with the sharp abscess of lung

Choice of medical tactic: the treatment of patients with the acute abscesses of lungs mast be done only in hospital. Tactic of treatment of patients with the acute abscesses of lungs depends on the stage of purulent-destructive process. In stages of acute infiltration - conservative therapy in combination with bronchilogic methods is preferable.

In the stage of draining of abscess through bronchi — active administration of bronchilogic methods of sanation (including most effective — microtracheostomy) on a background conservative therapy. In the case of insufficient drainage of abscess through bronchi or it complete absence (blocked abscess) are used punctions and drainings surgical methods.

The pathogenetic grounded conservative therapy is directed on the straggle against infection which caused a purulent-destructive process in a lung.

1) The regime is a semi bed.

2) Diet — with a high power value of albumins in food ration. Food must contain the promoted amount of vitamins.

3) Medicinal therapy:

- antibiotics for empiric therapy (before the get of results of inoculation and determination of sensitivity of antibiotics to microbial flora) more frequent than all are used synthetic penicillins, macrolides (sumamed), ftorhinolons of III-IV generations, cephalosporins of III-IV generations;
- unspecific anti-inflammatory drags — movalis, ketoprofen and his derivates (oruvel, ketonal), which are used as injections and tablets forms; - immunocorrectors therapy (levamizol/decaris for 0,15 in days — 3 days with interruptions for 14 days, during 4-6 months);
- direct anticoagulants — heparin, fragmin, fraksyparin;
- preparations making better the escalator function of lights — lasolvan, fluimuzil and other;
- desintoxic therapy — sorbilact, reosorbilact, reombirin;
- infusion therapy — Ringer solutions, 5% of glucose, normal saline.

It is necessary to know that depending on the phase of development of acute abscesses their treatment can be conservative and surgical. At the use of antibiotics of wide spectrum of action and at beginning of treatment in an early phase (to 6-8 weeks from the moment of formation of abscess) it is succeeded to achieve

success by conservative methods at 65-70 % patients with a acute abscess. It allows recommending conservative treatment of patients with the acute abscess of lungs during 6-8 weeks from the moment of it appearance, if there are no special indications to operation. This treatment includes introduction of antibiotics of wide spectrum of action in according to information of antibioticogramm, which are used separately and in combination and is usually introduce intramuscularly or intravenously. At the connection of abscess with bronchi it is necessary to provide the regular deleting of pus from the cavity of abscess through bronchoscope or through "postural drainage" which is executed as follows. According of the location of abscess in a lung and place of localization of drained bronchi, a patient is laid in such position, that drained bronchi was the lowest point of abscess, and compel a patient expectorate the sputum. After such emptying of abscess from the pus of intratracheally antibiotics are insufflated, plasma is repeatedly transfused for raising of protective forces of organism, correction of albumins, electrolyte balance; for the increase of efficiency of sanation of bronchial tree use proteolytic enzymes (tripsin, chemotripsin, ribonucleasa) which introduced by the method of inhalation, through microtracheostomy or in time of bronchoscopy (curative bronchoscopy).

In the last time in connection with stability of microflore to the antibiotics and development of dysbacteriosis, and also allergic reactions some authors recommend to conduct treatment by antibiotics jointly with dimexide. Mixture 15-20 ml dimexide 20 % and 200000-300000 UN of antibiotics introduces in bronchial tree with intervals 4-5 days. AT 50 % patients after two-five procedures microbe flora disappears, are special pneumococcy and hemolytic streptococci. At the acute abscess of lung after punction aspiration of pus, a cavity washes by 50 % solution of dimexide up to getting of transparent content. In subsequent in the cavity of abscess insufflating 20-30 ml solution of preparation, this is joint with antibiotics.

If conservative treatment did not give a result, operative treatment is indicated.

Indications to operative interference:

1). Insufficient of draining of AAL through a bronchial tree.
2). Peripheral undrained abscess of lung.
3). Peripheral abscess of lung, the diameter of cavity of which exceeds 5 sm.
4). Appearance of complications (pyopneumothorax).
5). Distribution of purulent-destructive process (gangrene of lung).
6). Chronic abscess.

Possible postoperative complications:

– postoperative hemoptysis and pulmonary bleeding;
– pleurisies;
– empyema of pleura;
– insufficiency of stump of bronchi after resection methods and formation of bronchial fistulae.

Diagnostics and treatment of the possible complications AAL.

Are the most frequent complications of pulmonary suppurations pyopneumothorax, affectation of opposite lung and pulmonary bleeding. Frequency of pyopneumothorax after AAL makes from 10,6 to 38,5%.

Acute pyopneumothorax more frequent than all comes at patients after a strong cough and characteristic by several pains in a chest, dyspnoea, by the pallor of skin

covers, sweating, cyanosis of skin and mucous membranes. Arterial pressure goes down to 50-70 mm of Hg column, a pulse becomes threadlike.

Most heavily pyopneumothorax develops in combination with tense pneumothorax. From the increasing shortness of breath position of patient becomes forced – he is usually in sitting position. The affected part of thorax detain in the act of breathing, intercostal intervals are smoothed out, is loosened the vocal fremitus, by percussion is determined tympanitis, at auscultation bronchial breathing with an amphoryc tint. The state of patient is so heavy sometimes, that rescued he can be by urgent operative interference only. At patients acute respiratory insufficiency is marked and they need the urgent help. If this complication happened before hospitalization, it is necessary quickly to hospitalize patient in the thoracal department in sitting position, but here necessarily give urgent medical help: to administrate cardiac preparations (solution of corglycon 1,0 or solution 0,05% 1,0 ml strophantin, and if allows B/P, 2,4 % solution of euphylline 5,0-10 ml) intravenously, anesthetic (solution of the analgin 25 % — 5 ml or ketanov 1,0), to give the moistened oxygen to the patient. In hospital to such patient urgently need performs X-ray films of the chest in 2 projections or ULTRASONIC examination of the chest organs. Punction of pleura cavity with the receipt of contents mast be done. Contents from pleura cavity mast be send on cytology and bacteriological research. Pleura cavity is draining by Bulau (in VII ribs space) and takes a tube in a sterile small bottle with solution of furacilline 1:5000 or normal saline. On an end tubes attach a finger from a glove and cut it end. On this drainage the outflow of pus from a pleura cavity will be carried out.

The second drainage inserted in II ribs interspaces on a middle clavicular line (for aspiration of air).

Micro invasive technologies are widely used presently – execute vision thoracoscopy with coagulation of bronchopleural fistulae and sanation of cavity of abscess and pleura cavity. Operation is ended by separate draining of cavity of abscess and pleura cavity (Fig. 2, 3, 4).

Fig. 2. Percutaneous microtracheostomia

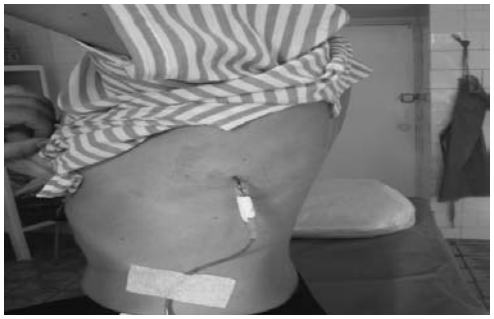


Fig. 3. Catheterization of peripheral abscess of lower left lung by Seldinger for sanation its cavity

Hemoptysis – it is expectoration of sputum with admixture of blood. The loss of blood here makes up to 50 ml.

Discharge of blood with sputum more than 50 ml is already the pulmonary bleeding.

It is necessary to take into account at hemoptysis: it is single or frequent hemoptysis. The common state of patient here in most supervision is satisfactory. Com-

plaints about a cough with the moderate expectoration of sputum up to 50-250 ml in days with the admixture of blood, pain in a chest. The pallor of skin covers is marked, here breathing frequency on the average 18 in 1 minute, frequency of pulse up to 60-80 per min., B/P 120/70 mm of Hg column. At frequent hemoptysis moderate anemia is marked, Hb – 110-100 g/l.

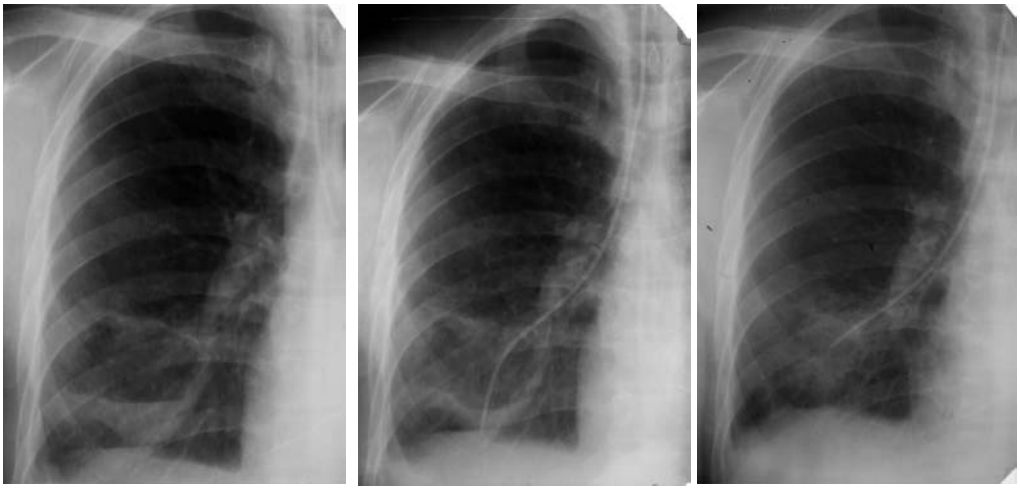


Fig. 4. Catheterization of peripheral abscess of lower right lung for sanation its cavity

Pulmonary bleeding is discharge of blood with sputum over 50 ml. The clinic of this complication depends from DCB (deficit of circulated blood — volume of the blood loss).

At patients with AAL pulmonary bleeding is common in the second period of disease – period of draining, when a plenty of sputum departs with a cough, and at the chronic purulent diseases of lungs this complication is observed in the period of intensification of chronic abscess of lung.

The condition of patients is usually heavy or very heavy, if blood loss makes over 500 ml. As a rule, the pulmonary bleeding arises up suddenly at a cough or physical loading. Sometimes LB arises up at night. Patients mark burning or pains in a thorax on the side of affectation.

The LB is characteristic by discharge of scarlet blood with the bubbles of air, here, as a rule, the vomitive reflex is absent. At abundant LB a blood can swallow by the patients and be poured out by vomitive motion outside and be had the appearance of «coffee-grounds». All patients at LB feel sense of fear, intensive weakness and dizziness; at some patients the swoon state is marked.

The big value in the LB diagnostics has the collected anamnesis. Almost always LB is accompanied by a cough, pain in a thorax and shortness of breath. Acute LB is accompanied by the intensive pallor of skin covers, by rise number of breathing up to 25-35 in 1 minute, a pulse in time and after LB becomes more frequent to 100-140 blows in 1 minute. At massive the LB pulse is threadlike and a collapse is even marked. B/P as a rule, up to 70-50 mm of Hg column The pulmonary bleeding at AAL is the erosive bleeding. The direct reason LB is the changes of histological structure of blood vessels in a pathological region; it results in violation of integrity

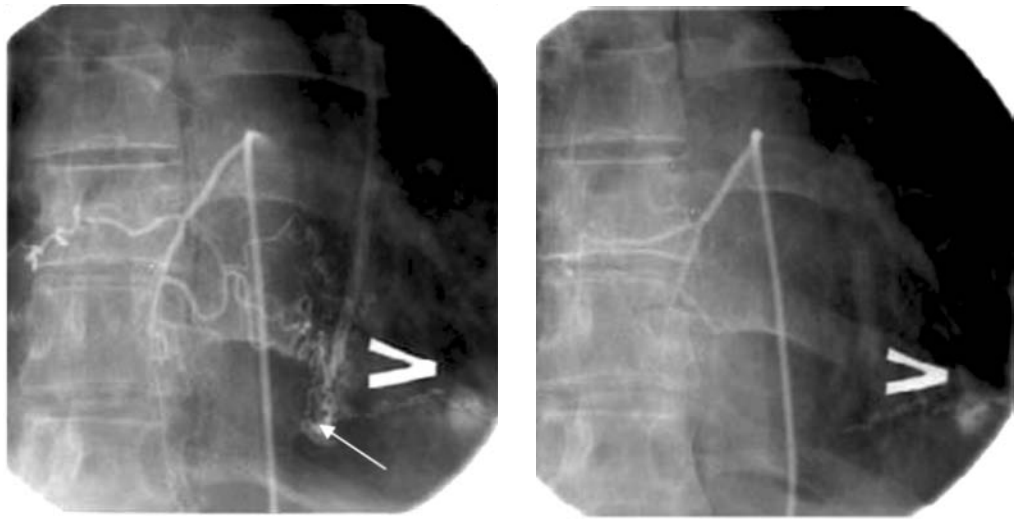
of wall of vessel. The danger of LB is possibility for a blood spreads in bronchial tree (aspiration) and be the cause of asphyxia.

The carefully collected anamnesis, clinical, roentgenologic, laboratory and endoscope results of investigation are very helpful in the LB diagnostics.

Taking into account that LB is the urgent state of patients, it is necessary to detect the side of affectation of thorax and source of bleeding. Such methods help it, as:

1) multiaxial x-raying of chest;
2) roentgenography of thorax in direct and lateral projections;
3) tomography; and also additional methods of research:
   — ULTRASONIC investigation of thorax;
   — bronchoscopy;
   — angiography of bronchial arteries (Fig. 5);
4) angiopulmonography.

Informing of roentgenologic methods is still high. They enable to detect the side of affectation, that presence of pathological process in a left or right lung, similarly as well as ULTRASONIC. Bronchoscopy enables to see what bronchi a blood acts from. Angiography of bronchial arteries on an affected side enables to expose the presence of extravasat and execute embolization of this artery.



a b

Fig. 5. Bronchial arteriograms before (a) and after embolization of left bronchial artery in patient with lung bleeding 5 days after lower lobectomy

The affectation of opposite lung by a acute purulent process arises up enough often is complication of chronic sick patients, lying and weakened patients. It is related to aspiration of purulent sputum from the lung affected by a destructive process in a healthy lung. This complication is observed up to 8% of cases.

The LB treatment (conservative and operative).

Foremost, it is necessary to quiet a patient and instill him a confidence in a favorable end.

Medical measures must promote:

1) to the lowering of pressure in the small circle of circulation of blood;

2) to the increase of coagulation of blood;

3) to closing of lumen of bleeding vessel.

From medicinal drags use: 1% solution of chloride calcium i/v; the EACA 5% solution — 200,0 i/v; solution of dicenone; solution of vicasoly i/m. It is obligatory urgent hemotransfusion. Necessarily enter of 1-3 ml 10% camphore butters + solution of the atropine 0,1% - 1,0-2,0 s\c.

From the instrumental methods of the LB stop apply bronchoscopy with occlusion of bronchi by haemostatic sponge or porolon, by the Fogarty catheter. It is the temporal stop LB. Presently, in connection with introduction of micro invasive technologies, at LB execute angiography of bronchial arteries with a subsequent embolization bleeding artery by the special spirals. This method may be independent and as preparatory to the resection methods of the LB treatment depending on basic pathology.

To the resection methods regards: segmentectomy (deleting of segment), lobectomy (deleting of lob), bylobectomy (deleting of 2 lobes), in heavy case – pulmonectomy (deleting of lung).

Rules of conduct of postoperative period, measures of prophylaxis, diagnostics and treatment of possible postoperative complications at patients with AAL.

One of threatening postoperative complication is the intrapleural bleeding. It can be one from two defects of hemostasis: surgical or biochemical. This complication meets in 7% of supervisions.

At insufficient surgical hemostasis as usually a big vessel bleeds. The clinic of LB depends on the caliber of bleeding vessel. A patient complains on a weakness, dyspnoea, thirst, a collapse comes in heavy case.

In basis of the biochemical intrapleural bleeding lying one or a few reasons from which most attention are the deficit of factors of coagulation of blood (in plasma and thrombocytes) , to surplus of anticoagulants, high fibrinolytic activity.

The window to pleural cavity is drainage by Bulau, through which in the case of the intrapleural bleeding a blood is discharged out. If a discharged blood forming the clot, it means that the bleeding is continuing, and, if on a background intensive haemostatic therapy the state of patient does not get better, rethoracotomy is needed - as a result it is necessary to find a bleeding vessel, stitch and tie it.

At the «biochemical» intrapleural bleeding the medical help consists in intensive hemotransfusion therapy - transfusion of plasma, fresh blood, fibrinogen, 1% chloride of calcium, hydrocarbonate of sodium, inhibitors of proteases (contrical, gordox and other), salt solutions, and also 5% EACA. In heavy case is possible to the reinfusion of drainage blood. The control of coagulogramm is necessary.

Thromboemboly of lung artery (TELA).

It is heavy complication of postoperative period the cause of which is blockade of trunk or branch of pulmonary artery. Sources of TELA may be system of lower vena cava or the stump of lung artery from which clot can transmits in the vessels of unoperated lung. Presently the use of fibrinolytic is the most effective method of treatment: striptease, streptokinase, farmakinase, actilise and other. These preparations can quickly dissolve a blood clot. Angiopulmonography is the most effective method of the TELA diagnostics.

Myocardial infarction after operations is not uncommon complication. As a rule this complication develops without a pain attack, a patient is on narcotic anesthetic. It is diagnosed on the basis of these ECG and clinic. Treatment of patient consists administration of coronarolytics, analgesics, antiarrhythmic preparations, oxygen therapy.

Atelectasis of the operated lung can develop gradually. It is characterized by the fever, dyspnoea, tachycardia. By percussion is marked dullness of percutory sound in the area of atelectasis, by auscultation is intensively weakened breathing above the area of atelectasis. A final diagnosis performs after roentgenologic research. Treatment of atelectasis includes immediate restoration of bronchial passage by bronchoscopy and washing of bronchial tree, administration of mucolytics, stimulations of cough.

Pneumonia of the operated lung — the most frequent reason is the presence of shallow atelectasis or low resistance of organism, due to the edema of lungs, aspirated pneumonia. The increase of temperature, shortness of breath, tachycardia, cyanosis is marked.

At auscultation there is moist rales, is special in the lower part of lung. Final diagnosis is put at roentgenologic research.

Treatment of pneumonia is powerful antibacterial therapy, introduction of fresh-frozen plasma, heparin therapy and mucolytics.

Contrlateral pneumothorax, yet name him spontaneous. It can be and iatrogenic during catheterization of subclavia vein. The state of patient gets worse sharply - the shortness of breath, cyanosis appears. At auscultation of the lung on the side of defeat breathing is not detected. It is necessary quickly to execute punction of pleural cavity and drainage of pleural cavity in II ribs interspace on a middle clavicular line.

Patients with carried AAL are subject for the supervision at pulmonologist and surgeon in regional policlinic.

Gangrene of lung

The gangrene of lung *is necrosis of pulmonary tissues under act of toxins and violation of feed, which does not have clear borders. Between the acute abscess of lung and gangrene of lung a lot in common, but, nevertheless, most authors consider these diseases independent. At AAL the inflammatory reaction and purulent focus has the limited character, and at the gangrene of lung is necrosis of pulmonary tissues, have not clear borders. At the gangrene of lung a necrotic process spreads in pulmonary tissues diffusely. The areas of normal tissues without noticeable borders pass to changed, losing a clear structure pulmonary tissues which also without clear borders.. Thus pulmonary tissues have the appearance of grey-green mass with foul smell. Usually is affected lobe, two lobes or all lung. Polymicrobe floras are the origin affectation of lung: staphylococci, gram-negative bacterias and different anaerobes. The supporting factors in appearance of gangrene are disorder of passage in bronchus with evolution of atelectasis, disorder of blood circulation; creation of the reserved space in the area of atelectasis and stopping of clearing of bronchial tubes from an infection by expectoration; and, especially, influence of a plenty of toxins of developing microorganisms on tissues of lung*

Clinic of gangrene of lung. All above mentioned, what are regard to the acute abscess of lung, belongs and to the gangrene of lung with that only a difference, that acute intoxication of organism of patient comes on the first place. The disease is accompanied constantly by a high temperature, which does not lowering long time, or has vibrations, in the morning and in the evening. A painful cough with especially foul sputum is characteristic.

Sputum has the appearance of foamy liquid, dirtily-grayish color, sometimes with the raspberry or chocolate coloring which is explained by the parenchimatose bleeding from disintegrating tissues. At precipitation the sputum divides on 3 layers: upper is liquid, middle is serous, lower is dense, consisting of granulated mass and fragments of pulmonary tissues. A plenty of sputum is usually expectorated at mornings and is accompanied by a excruciating cough.

Patients complain on several pains in the affected half of thorax. It is related to affectation of pleura, which is rich by the nervous endings.

The state of patients at the gangrene of lung is always heavy. They weaken quickly, is exhausted, sweating, absence of appetite, making progress anemia is marked.

At examination of patient lag of affected half of thorax is marked.

At percussion dullness of percutory sound with the unclear spreader borders is marked.

At auscultation there is a plenty of different calibers rales. A frequent and small pulse, deaf tone of hearts, lowering B/P is marked. At the beginning of disease leucocytosis with the change of leucocytes formula to the left side registers in a blood. As far as making progress of gangrene the lowering of leucocytosis is possible.

At roentgenologic research usually detected the intensive darkening of part of lung with gradual transition to the normal pulmonary picture on periphery..

If gangrene makes progress, and spreads on peripheral regions of lung, as result, parenchyma of lung disintegrates as sequesters and gets in a pleura cavity - develops the putrid pyopneumothorax and illness acquires the septic form.

Treatment of patients with the gangrene of lung

Treatment at the gangrene of lungs is exact the same, as well as at the acute abscess of lung with that only a difference, that it must be more intensive.

Conservative treatment at GL is produced:

1) at the neglected forms of gangrene of lungs and expressed pulmonary-cardiac insufficiency and bilateral affectation;

2) if there is place of transformation of gangrenous abscess to ordinary acute abscess with adequate draining;

3) uncomplicated gangrene of lights with the favorable clinic-roentgenologic dynamics of on a background producible treatment.

In all other case conservative treatment at the acute gangrene of lungs must be examined as preoperative period.

Intensive therapy at GL includes infusion therapy with the purpose of parenteral feeding, corrections of water-electrolyte disorders, improvements of reologic properties of blood, supportion of energetic balance and desintoxication. Maintenance of energetic balance is here provided by introduction of the concentrated solutions of glucose 25-40% to 1 liter.

Restoration of albumin losses more frequent than all is performed by introduction of solutions of aminoacids, fresh-frozen plasma, solutions of albumin and other.

For desintoxication and improvements of reologic properties of blood, improvement of capillary circulation of blood use infusion of gemodese, reopolyglukine, reamberine.

For correction of anemia use transfusion of erythrocyte mass, best of all washed red corpuscles.

To the extremely heavy patients with the gangrene of lungs with the clinic of septic shock with the appearance of polyorgan insufficiency the combined therapy with the use of preparations influencing on the cellular regulation of antioxidant immune answer is indicated: cytoflavin for 10 mgs 2 times per days on of a 200 or 900 ml 5% or 10% solution of glucose with the subsequent joining of cycloferone 4 ml 2 times per days after stabilization of the state of patient. In heavy case indicated introduction of pentaglobine (USA) i/v 10 or 20 ml on a solvent 50 ml or 100 ml (contains valuable and biologically intact immunoglobulins in a stable form.

In the case of unsuccessful of conservative therapy patients with the gangrene of lungs are subject to operative treatment. The methods of surgical treatment divide by resections and draining. Draining operations are less traumatical. Draining can be executed by thoracocentesis and drain tube. Presently draining is executed with help thoracoscopy. It is possible drainage at the gangrene only cavity with liquid pus and small sequesters. During thoracoscopy pus is deleted, all sequesters, complete sanation is conducted. Nevertheless, if allows the common state of patient, ***the resection of lobe of lung, two lobes or deleting of lung – pulmonectomy*** are more radical.

Bronchiectatic disease

> ***Bronchiectasis*** *are the irreversible morphological changes (dilatation, deformation) and functional inferiority of bronchial tree, resulting in the chronic purulent disease of lungs.*

Among other diseases of lungs bronchiectatic disease makes from 10 to 30%, and at fluorography this disease detects approximately at 1-2 from 1000 inspected. More than at the half of patients it is diagnosed under age 5 years and at one third of all patients - on the first year of life. Men are ill in 1,3-1,9 times more frequent, than women. Among the adult population (from sectional information) frequency of bronchiectatic disease makes from 2 to 4%.

Two theories of development of bronchiectasis are most known. According to one of them, they are examined as disease of innate, and on other - acquired character. Most authors adhere to the theory of the acquired origin of this pathology, considering a basic etiologic factor the genetically determined inferiority of bronchial tree (immature of elements of bronchial wall - muscles structures, elastic and cartilaginous tissues, insufficiency of mechanisms of defense and etc), which in combination with disorder of bronchial passage and appearance of inflammation results deformation of bronchial tree.

A left lung is affected in 2-3 times more frequent, than right. In child's age predominant left-side bronchiectasis. Since 20 years, frequency of affectation of right and left lung becomes equal, and after 30 years right-side processes prevail. The bilateral affectation marked identically often in all ages. Lower lobe localization of process is characteristic mainly. Lower lobes bronchiectasis often combines with

the impair of middle lobe on the right and uvular segments on the left. The generalize forms of disease with the total affectation of both lungs meet approximately at 6% of patients.

The following classification of bronchiectatic disease is accepted in a clinic and is used:

Originally:
– primary (innate);
– secondary (acquired).

By kinds of dilatation of bronchi:
– cylindrical;
– saccular;
– cyst like form;
– mixed.

By distribution:
– limited;
– widespread;
– one-sided;
– bilateral (with pointing of exact localization according segmental structure).

Depending on severity of clinical manifestation, separates next classification:
– mild form;
– middle expressed;
– heavy;
– heavy complicated form.

By the clinical feature:
– phase of remission;
– phase of attack.

Peculiarities of inspection of patient with suspicion on bronchiectatic disease. Bronchiectatic disease is characterized by the protracted flow with periodic intensification (more common in spring and in autumn by intensifications). Most patients with the starting point of origin of disease have pneumonia or chronic bronchitis.

At questioning of patient:

1). Complaints. Usually the disease flows with alternation of intensifications and remission, therefore a clinical picture directly depends from the period of disease. The moist cough with expectoration of mucous-serous sputum, especially expressed at mornings, evening subfebrile temperature, anorexia, gradually increase pallor of skin covers, asthenia, general weakness are the permanent symptoms in period of intensification. Discomfort or dull, increasing in the period of intensification of inflammatory process pains in a thorax is connected, mainly, with the affectation of mucous bronchial tree and reactive pleurisy. A pain syndrome is almost marked at every second patient. Dyspnoea is present at 40 % of patients and grows as far as making progress of disease. After such intensifications long time is saved cough with sputum, shortness of breath, indisposition. It is multiplied the amount of sputum gradually, the expressed intensifications in the first years of disease are not observed.

2). Complaints from the side of other organs and systems: on a general weakness, poor appetite, insomnia and other.

3). Anamnesis of disease: patients mark frequent appearance of bronchitis or pneumonia.

4). Anamnesis of life: in anamnesis there can be the frequent cold diseases which can be the cause of lowering of reactivity of organism.

Clinical physical inspection (characteristic features at this disease):

1). Common state of patient in the period of intensification more frequent of middle degree of heavity. Consciousness, as a rule, is clear. Constitutional features - asthenic is more frequent.

2). Collection of information about original appearance of patient. Skin covers are pale. During intensification is unpleasant smell from mouth, edema of face is marked.

3). Inspection of the state of the cardio-vascular system. Tachycardia is related to the increase of temperature of body. The tones of heart are muffled.

4). Inspection of the state of abdominal organs. Lowering of appetite. Some lowering of mass of body. More frequent than all characteristic changes is not marked.

5). Inspection of the state of bones-muscles systems: at the protracted presence of disease fingers as drumsticks, deformation of nail plates - «watch glasses». Chronic purulent intoxication can be result of affectation of long tubular bones with development of sclerosis of bone tissues and origin of inflammatory changes in joints.

6). «Locus morbi»: Symptomatic detected at the physical inspection of thorax is very various and is determined by localization of affectation, phase of disease, expressivity of anatomic changes, presence or absence of concomitant changes in surrounding pulmonary tissues. At examination the affected half of thorax delays in the act of breathing (at the massive affectation). The percutory changes are not characteristic. By auscultation: at centrally located or, especially, «dry» bronchiectasis the changes can be absent, but at filling by sputum of saccular bronchiectasis quite often possible listen above affectation the rales which have different calibers, at times of big sonority, sometimes with a «metallic» tint. On the whole an auscultative picture can be described as pied.

7). Leading clinical symptoms: - obstructive – intoxication

8). On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose the following clinical diagnosis: BRONCHIECTATYC DISEASE OF (UPPER, MIDDLE, LOWER) LOBE OF RIGHT/LEFT LUNG.

According to standard charts the plan of additional inspection (laboratory and instrumental) includes:

1). Clinical blood test.

2). Clinical analysis of urine.

3). Biochemical blood test.

4). Coagulogramm.

5). Clinical analysis of sputum.

6). Immunological tests.

7). Bacteriological research of sputum, washing waters of bronchial tubes, content of cavity of abscess.

8). Roentgenologic research of organs of thorax.

9). Spyrography.

10). Fibrobronchoscopy.

11). Bronchography after sanation bronchoscopy.

1. Clinical blood test: in the phase of intensification appears anemia, high leucocytosis of peripheral blood with drumstick deviation, the ESR increase. In a period of remission these changes are expressed indistinctly.

2. Clinical analysis of urine: the changes are unspecific, meet albumin urea, cylinder urea.

3. Biochemical blood test: hypoproteinemia, dysproteinemia.

4. Coagulogramm: violations of coagulation of blood in side hypercoagulation.

5. Clinical analysis of sputum: the presence of a plenty of leucocytes, elastic fibers is marked.

6. Immunological tests: the decline of indexes of reactivity of organism is characteristic, in particular cellular immunity.

7. Bacteriological research of sputum, washing waters of bronchial tubes, content of cavity of abscess: allows detecting the microbial flora and defining the sensitiveness of the last to antibiotics preparations.

8. Roentgenologic research of organs of thorax in two projections: diminishment of volume and compression of shade of the affected regions of lung is marked, segmental and lobar atelectasis, presence of pleura adhesions, hyperplasia and compression of lymphatic nodes of root of lung, increase of airiness of his unaffected departments due to vicar emphysema, high standing and limitation of excursion of diaphragm, on the side of affectation. By a basic roentgenologic method confirmative a presence and specifying localization of bronchiectasis are bronchography with the obligatory and complete contrasting of bronchial tree of both lights. For good filled of bronchial tubes by the roentgen contrast matter, and also for the removal of unfavorable consequences of this research conducting of preliminary careful sanation of tracheobronchial tree with the maximal release from it content (Fig. 6). On bronchogramms in the affected section of lung register one or another type of dilatation of bronchial tubes of 4-6th orders.

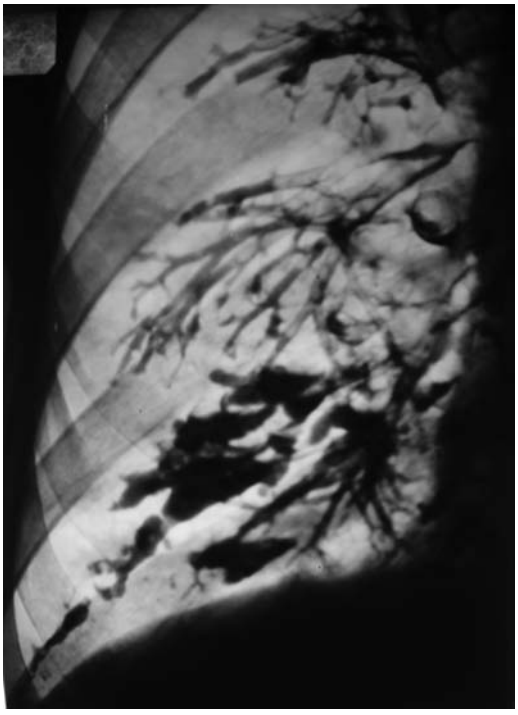


Fig. 6. Bronchiectasis of the lower lobe of right lung

9. Spyrography are the low indexes LVL, decline of compensate possibilities of lungs in combination with hyperventilation, decline of satiation of arterial blood by oxygen.

10. Fibrobronchoscopy: gives information about the degree of expressivity and localization of inflammatory process in a bronchial tree.

Differential diagnostics: bronchiectasis affectation is necessary to differentiate with TB, chronic pneumonia, chronic bronchitis, chronic abscesses, cancer and cysts of lungs.

Ground and formulation of clinical diagnosis (taking into account classification of disease, presence of complications and concomitant pathology):

1) basic — bronchiectatic disease of (upper, middle, lower) lobe of right/left lung;
2) complications of basic disease — (if they are);
3) concomitant pathology — (if it is).

Treatment of patient with bronchiectatic disease

Choice of treatment tactic

Treatment of patients with bronchiectatic disease is complex, directed on the stragl against an already present infection, on its warning, and also on maintenance of bronchial drainage and restoration of protective forces of organism; the surgical methods of treatment are used if necessary.

Pathogenetic grounded conservative therapy.

The base directions of conservative treatment are: sanation of tracheobronchial tree, antibacterial, desintoxic, desensibilization and general health improving therapy, physiotherapy, high-calorie diet.

1) The regime is general. Stimulation of motive activity of patients, respiratory gymnastics and physical culture. Treatment by position is used - postural drainage, when chouse such position of trunk which is optimum for expectoration of bronchial content.

2) Diet — with high energy content with high level of albumens and vitamins.

3) Medicaments therapy:
   - antibiotics for empiric therapy (before the receipt of results of inoculation and sensitivity of bacterial flora) more frequent than all are used synthetic penicillins, macrolids (sumamed), ftorhinolons of III-IV generations, cephalosporins III-IV generations;
   - unspecific anti-inflammatory drags - movalis, ketoprofen and its derivative (oruvel, ketonal);
   - immunocorrector therapy (levamisol/decaris for 0,15 in days 3 days with interruptions for 14 days, during 4-6 months, polyoxyzonium, imunofan).
   - preparations making better the escalator function of lungs - lasolvan, fluimuzil, ACC and other;
   - desintoxic therapy — sorbilact, reosorbilact, reombirin;
   - infusion therapy is the Ringer solutions, 5% of glucose, normal saline;
   - inhalations — antibacterial preparations (in accordance with the sensitiveness of microflore), muco- and photolytic preparations (tripsin, ribonuclease, desoxyribonuclease, terrytilin), stimulations of cough by daily insufflations of different solutions through a catheter, entered through microtracheostomy;
   - bronchoscopy sanation with performing bronchial lavage with solutions of antiseptics.

Existent types of operative interferences and indication to them

Operative treatment needs of about 40% patients with bronchiectatic disease. It performing is most optimum in age from 7 to 14 years (at presence of innate bronchiectasis).

Resection methods are used: the volume of operative interference in such cases is the resection of the affected area of lung is lobectomy, bylobectomy.

Indications to the resection of lung are determined on the basis of estimation of spreadness and features of disease, common state of patients and their functional possibility. The basic indications for operative treatment of patients with bronchiectasis:

– one-sided affectation with abscess formation, hemoptysis or bleeding, uncomplying to conservative treatment;
– one-sided processes with the big volume of sputum and expressed intoxication;
– one-sided making progress processes with frequent attack.

Operative treatment is contra-indicated at the bilateral widespread affectation, decompensated pulmonary-cardiac or kidney-hepatic insufficiency.

At bilateral limited bronchiectasis through 6-12 months after the first operation, the resection of lung is possible on an opposite side.

Tactics in postoperative period, possible postoperative complications.

In a postoperative period, as a rule, the before begun conservative therapy continues to be performed. The base tasks of treatment in a postoperative period are renewal and support of the disordered main systems of life-support - breathing and circulation of blood. After stabilization of the cardio respiratory systems basic direction of intensive therapy is prophylaxis of infectious complications. In late postoperative period in mostly cases is necessary only symptomatic therapy.

Complications after operations concerning to bronchiectatic disease meet in 15-20% of cases. The most frequent postoperative complications are:

– atelectasis;
– pneumonia;
– bronchial fistula;
– empyema of pleura;
– postoperative hemoptysis and pulmonary bleeding.

Diagnostics and treatment of possible complications of bronchiectatic disease.

Bronchiectatic disease can be complicated by the pulmonary bleeding, abscesses and gangrene of lung, by forming of extra lung abscesses and sepsis, by development on a background pneumofibrosis and emphysema of lungs of the expressed pulmonary-cardiac insufficiency and pulmonary heart, sometimes - cancer of lung and amyloidosis of internal organs. Quite often the flow of this disease is burdened by bronchial asthma and TB.

Detection of disability and prophylactic medical examination of patients with bronchiectatic disease.

After passing of course of treatment in hospital patients with bronchiectatic disease mast be under observation of pulmonologist in a regional policlinic. The operated patients parallel are observed at a doctor-surgeon.

Acute and chronic empyema of pleura

The problem of diagnostics and treatment of acute and chronic empyema of pleura is actual and on a modern time, as frequency of acute empyema of pleura after radical interferences on lungs hesitates from 6,7% to 15% . And at the patients operated due to purulent diseases of lungs, disintegrating tumors this number achieves at 23-24%. In spite of successes in treatment of acute empyema of pleura from 20 to 30%, and according to information of same authors up to 50% supervisions, acute forms transform to chronic.

Acute empyema of pleura

> ***Acute empyema of pleura*** *is the limited or diffuse inflammation of visceral and parietal pleura, characteristic by accumulation of pus in a pleura cavity and accompanying by the signs of purulent intoxication and quite often respiratory insufficiency.*

Unspecific empyema of pleura is caused different purulent or putrid microorganisms. From a pleura cavity more frequent than all staphylococci is revealed - to 77 %. It is explained the expressed their virulence and stability to most antibacterial medicines. In 30-45% of cases at inoculation of pus from pleura cavity gramnegative microorganisms get growth, which are different cultures of intestinal, blue pus bacillus, Bacillus proteus. Up to 80% of cases an anaerobic nonclostridial flora is detected (bacteroids, fuzobacterias, peptococcy, peptostreptococcy and others).

According to pathogenesis distinguished primary and secondary empyema of pleura. At primary empyema of pleura from the beginning of the disease the inflammatory focus is localized in a pleura cavity, at the secondary - it is complication of some other purulent-inflammatory disease.

Primary empyema of pleura arises up on a background unchanged, healthy pleura due to disorder of their barrier function with bringing of microbial flora. It is at the trauma of the chest, after manipulations in a pleura cavity and operations on a lung.

According to information of many authors in 85-90% of cases secondary empyema of pleura was complication of pneumonia, acute and chronic purulent diseases of lungs. Pneumonia can from the beginning flow with development of festering pleurisy (Para pneumonic empyema of pleura or empyema of pleura develops in period of ending of pneumonia and evolution to independent disease (meta pneumonic empyema).

At the abscesses of lungs empyema of pleura develops at 8-11% of patients, and at the gangrene of lung — at 55-90%.

In single case empyema can develop, as complication of purulent or parasite cyst, disintegrating cancer, spontaneous pneumothorax.

Secondary empyema of pleura can develops by a contact way, at suppuration of wounds of chest, osteomyelitis of ribs, spine, breastbones, chondritis, lymphadenitis, mediastinitis, pericarditis.

The acute inflammatory diseases of abdominal region can be the source of infection of pleura in rare case (subdiaphragmal abscess, purulent cholecystitis, pancreatitis and others). Penetration of microbes from an abdominal region to pleura takes place through lymphatic vessels and fissures in a diaphragm, or through hematogenic way.

***The following classification of empyema of pleura is accepted in a clinic and is used*:**

I. By etiology:

1. Unspecific:
   – purulent;
   – putrid;
   – anaerobic.
2. Specific:
   – tuberculosis;
   – mycotic;
   – syphilitic.
3. Mixed

II. By pathogenesis:

1. Primary
   – traumatic;
   – postoperative.
2. Secondary
   – para- and meta pneumonic;
   – contact;
   – metastatic.

III. By the clinical feature:

1. Acute (to 3 months)
2. Chronic (over 3 months)

IV. By the presence of destruction of lung:

1. Empyema of pleura without destruction of lung (simple)
2. Empyema of pleura with destruction of lung.
3. Pyopneumothorax.

V. According to connection with an external medium:

1. Closed.
2. Opened:
   – with bronchopleural fistula;
   – with pleurodermal fistula;
   – with bronchopleurodermal fistula;
   – by the latticed lung;
   – with other hollow organs.

VI. By spreads:

1. Delimited:
   – apical
   – paramediastinal
   – supradiaphragmal
   – interlobar
   – parietal
2. Widespread
   – total
   – subtotal

Features of inspection of patient with suspicion on acute empyema of pleura.

At questioning of patient:

1). Complaints on the basic disease. Usually the disease begins acutely. The permanent symptoms are: increase of temperature up to 38-39 ° C, pain in the chest and shortness of breathing. The pain syndrome – arises up, as a rule on the side of affectation and has permanent character, increasing at the deep breathing, cough, at change position of body. Sometimes may be present pain in upper part of abdomen due to irritation of diaphragm. Cough – quite often with expectoration plenty of sputum, the volume depends from intensivety affectation of lung's parenchyma, presence of bronchopleural fistula.

2). Complaints from the side of other organs and systems: on a general weakness, bad appetite, insomnia and other, evidences of intoxication.

3). Anamnesis of disease: the disease begins suddenly: on a background a trauma or preceding acute inflammation of pulmonary tissues.

4). Anamnesis of life: in anamnesis there can be the frequent cold diseases which can be result of lowering the reactivity of organism. It is necessary to pay the special attention to social status of patient.

Clinical physical inspection (characteristic peculiarities at this disease):

1). The common state of patient more frequent middle or heavy gravity. Consciousness as a rule is clear. Constitutional features – asthenic is more frequent.

2). Collection of information about original appearance of patient. Skin covers are pale. A bad breath from mouth is marked. A patient prefers to be in the forced position - sitting or lying on healthy side.

3). Inspection of the state of the cardio-vascular system. Tachycardia is characteristic and in mostly cases is related to the increase of temperature of body. The tones of heart are weak. There is tendention to hypotony. Development of pulmonary-cardiac insufficiency with increasing decompesation of circulation of blood and hypertension in a small circle is possible, to what indicated in an accent of the 2 tones on a pulmonary artery.

4). Inspection of the state of organs of abdomen. Decline of appetite. Some lowering of mass of body. More frequent than all characteristic changes is not marked.

5). Inspection of the state of bones – muscles system — the characteristic changes are not present.

6). «Locus morbi»: Limitation of respiratory excursion of the affected half of thorax, smoothed out of intercostal intervals, local edema of skin and hypodermic cellulose above the region of accumulation of pus in a pleura cavity is marked. In future tissues of pectoral wall in this area become dense, painfulness increases, hyperemia of skin appears. At percussion dullness above the area of accumulation of liquid is determined. In absence of air and adhesions in a pleura cavity the upper border corresponds to the Ellys- Damuazo line. At auscultation weakening of the vesicular breathing up to complete it absence over big accumulation of liquid is marked. Above the area of the compressed lung is bronchial breathing, is sometimes there are moist rales of different calibers, sometimes sound of friction of pleura due to a fibrin's pleurisy around the cavity of empyema. If there is bronchopleural fistula and cavity it is good draining through bronchi, can be listens the amphoryc

breathing. Increase of bronchophony sound above the region of accumulation of liquid is very characteristic.

7). Leading clinical symptoms: - intoxication - respiratory insufficiency.

8). On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose the following clinical diagnosis: ACUTE RIGHT-SIDE/LEFT-SIDE EMPYEMA OF PLEURA.

In accordance to standard charts there is the plan of additional inspection (laboratory and instrumental) of patient with acute empyema pleura:

1). Clinical blood test.

2). Clinical analysis of urine.

3). Biochemical blood test.

4). Coagulogramm.

5). Research of electrolytes of blood.

6). Immunological tests.

7). Bacteriological research of sputum, washing waters of bronchial tree, content of cavity of abscess.

8). Roentgenologic research of organs of thorax.

9). Spyrography.

10). Fibrobronchoscopy.

1). Clinical blood test: moderate anemia, leucocytosis with neutrophilia, by the change of leukocyte formula to the left, the ESR increase.

2). Clinical analysis of urine: the changes are unspecific is presence of signs of toxic nephropathy: albuminuria, cylindruria.

3). Biochemical blood test: it is sharply expressed hypoproteinemia, dysproteinemia.

4).Coagulogramm: disorder of coagulative function of blood to side hypercoagulation with diminishment of time of coagulation of blood, considerable increase of level of fibrinogen.

5). Research of electrolytes of blood: hyperpatassemia explainable by disintegration of tissues and elements of blood is marked.

6). Immunological tests: the decline of indexes of reactivity of organism is characteristic.

7). Bacteriological research of sputum, washing waters of bronchial tree, content of pleura cavity: allows to detect the microbial flora, which are the cause of inflammatory process and define the sensitiveness of the last to antibacterial preparations.

8). Roentgenologic research of organs of thorax: roentgenologic research at acute empyema of pleura and pyopneumothorax has most value, allows performs exact verification of diagnosis and detect the nearest tactic of treatment of patient (Fig. 7). Polypositional roentgenoscopy is more informing allowing to localize the region of affectation, exactly to define the degree of collapse of lung and displacement of mediastinum, amount of liquid, to expose the pathological changes in pulmonary parenchyma, to choose a point for adequate draining of pleura cavity, especially at limited empyema.

At rarely meeting interlobar empyema is possible punction of affect under the control roentgenoscopy. It should be noted that roentgenoscopy quite often is diagnostically sufficient research for the decision about implementations of urgent

medical measures - punction or draining of pleura cavity for its decompression at tense pyopneumothorax. In the case of its absence, if allows the state of patient, implementation of lateroscopy allowing exactly to define the vertical sizes of cavity is possible, to estimate the state of the basal sections of lung, «covered» by the level of liquid. If implementation of lateroscopy on the healthy side is risky due to the danger of aspiration of content of abscess (at the loosened patients), for determination of lower point of cavity there is enough roentgenoscopy in a lateral projection at inclination of trunk ahead or in a direct projection with inclination of trunk in a healthy side.

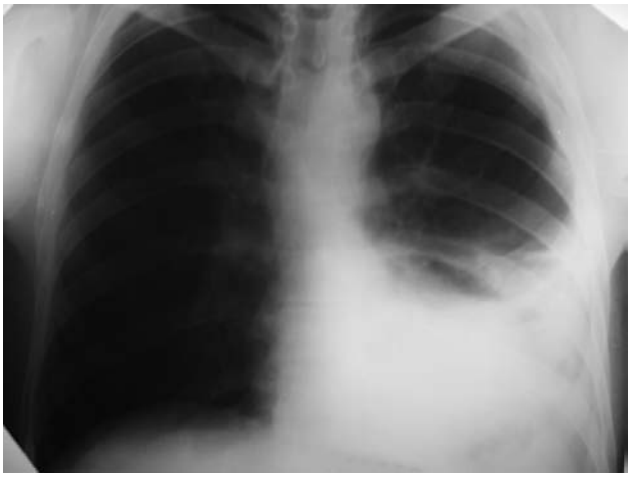


Fig. 7. Leftsided acute empyema of pleura (Demauso line)

Tomography allows to answer on foregoing questions; however, this research is not so informative if there is collapse of lung or presence considerable quantity of liquid in a pleura cavity. It is therefore expedient to execute it after draining of pleura cavity and liberation of it from pus. If the lung of collapsed more than on a 1/4 volume, interpretation of tomography information is difficult.

Pleurography in 3 projections is the very informing method of research. It allows to estimate the sizes of cavity, character of its walls, presence of sequesters and fibrins stratifications.

9). Spyrography are the low indexes VVL, decline of compensate possibilities of lungs in combination with hyperventilation, decline of satiation of arterial blood by oxygen.

10). Fibrobronchoscopy: gives information about the degree of expressed of inflammatory process in a tracheobronchial tree, allows defining the mouth of draining bronchi (at presence of destruction in pulmonary tissues). Ability of bronchoscopy rises at introduction of painting substance (water solution of methylen dark blue) to the pleura cavity in position on healthy side. It allows to define, what bronchial tubes participate in draining of area of destruction of pulmonary tissues, that is very important for planning of level of temporal endobronchial occlusion.

Differential diagnostics: it is conducted with pneumonia, obturated atelectasis of lung, hydrothorax, by the abscesses of lung, subdiaphragmal abscess, with the cancer of lung in the stage of disintegration and presence of cancer pleurisy, tumors of pleura, purulent cysts, echinococcus, diaphragmal hernia.

Ground and formulation of clinical diagnosis (taking into account classification of disease, presences complication and concomitant pathology):

1) basic — pneumonia /abscess/ of (upper, middle, lower) lobe of right/left lung;

2) complications — acute empyema of pleura;

3) concomitant pathology — (if it is).

Treatment of patient with acute empyema of pleura

Choice of medical tactic: presently in treatment of acute empyema of pleura is used combination of the surgical methods of treatment, directed on evacuation of purulent content of pleura cavity, and conservative therapy which is observed as basic part of preoperative preparation and postoperative conduct of patients.

The pathogenetic grounded conservative therapy is directed on the straggle against three basic pathological factors — suppurations, losses and resorption. Only by local treatment (surgical methods of sanation) - without influence on all organism, without normalization of the disordered functions of organs and liquidation of remaining complications, without therapy of primary and concomitant diseases - it is impossible to treat patients with empyema of pleura.

Such patients indicated complex therapy including etiologic, pathogenetic, symptomatic and local treatment. Intensity of treatment is depending on the common state of patient, presence of concomitant diseases and complications, from the stage of purulent process and character of empyema.

Application of antibiotics, sulfanilamides and antiseptic facilities is the basic method of therapy. Antibiotic therapy must include 2-3 preparations, which operate on pathogenic aerobic and anaerobic microorganisms.

Pathogenetic therapy consists in application of anti-inflammatory facilities, broncholitics (lasolvan, bronchobru, fluimuzil), in conducting of the measures directed on correction of violations of proteins (transfusions of fresh-frozen plasma, solutions of aminoacids), mineral (infusion of crystalloid solutions), carbohydrate exchanges (infusion of solution of the glucose 5%); oxygen therapy (5-7 litres per minute), desintoxic treatment (sorbilact, reosorbilact, reombirin).

In complex treatment of patients with empyema of pleura mast be included diet with rich content of proteins and vitamins. For the improvement of appetite at the exhausted patients anabolic hormones are used (retabolil for 50 mgs, superanabolon for 1-2 ml 1 times per a week, nerobol for 0,002 three times per a day and other).

Existent methods of operative treatment and indications to them.

Basic local treatments are surgical manipulations (punction of pleura cavity) and different methods of draining of empyema cavity.

Pleural punction, as independent method of treatment, has enough limited positions and can be used only in case without the presence of bronchopleural fistula (at primary empyema of pleura). During conducting of punction evacuation of purulent content is performs, lavage and sanation of cavity of empyemy by solutions of antiseptics and antibiotics, abandonment of the last in the cavity of empyema, In absence of effect after the punction treatment during 5-7 days need to change the treatment on the drainage methods (Fig. 8).

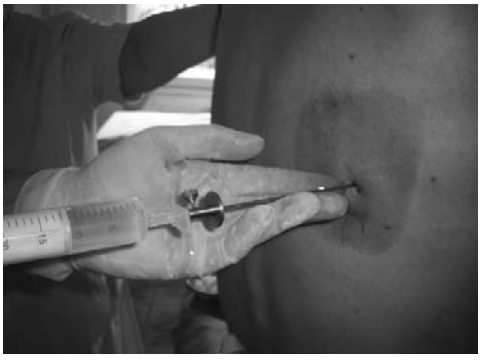


Fig. 8. Punction of the right pleural cavity

Drainage of pleural cavity with the permanent sucking of exudate and air (at presence of bronchopleural fistula) consider as more effective method of treatment. Methodic of draining: after conducting of

roentgenologic research a point for introduction of drainage is detected, on possibility at the bottom of empyema cavity. If there is a necessity in the second tube, it is entered under the apical region of cavity. Under local anesthesia conduct thoracocentesis of cavity and enter a drain tube. The internal channel of drainage must not be less than 0,6-0,8 cm. For the improvement of quality of procedure recommend to apply roentgencontrast drainages and draining under the ULTRASONIC control.

If the defect of bronchi (at presence of bronchopleural fistula) is considerable and a plenty of air passed through it, active aspiration from a pleura cavity does not make sense, because formation of vacuum is impossible, and the intensive sucking of air conduces only to strengthening of disorders of the external breathing. In similar cases uses valvular siphon drainage but Bulau (Fig. 9).

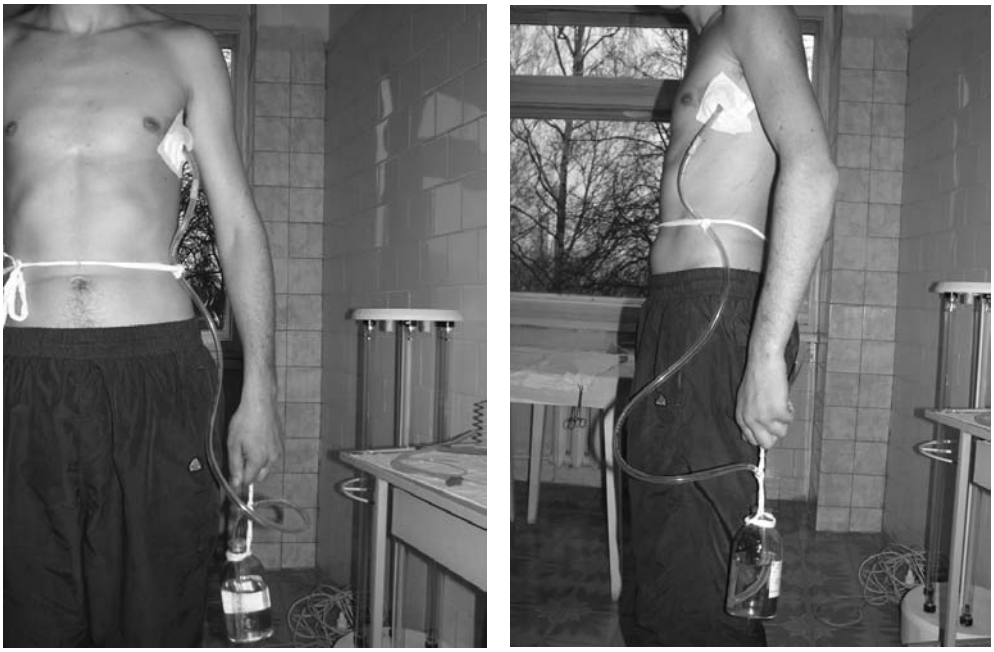


Fig. 9. Draining of the pleural cavity after Bulau

After implementation of draining daily 3-4 times per days the fraction lavage of empyemy cavity executes.

Considerably improve the results of aspirating methods of treatment at the use of methods of temporal occlusion of bronchial fistula by porolon (foam robber) or collagen obturators. Selective hermetization of the bronchial system by temporal occlusion of bronchial tubes allows differentiating a purulent process in a lung and pleura. Indications for application of this method are acute empyema of pleura with destruction of pulmonary tissues and big outflow of air through drainages, acute incompetence of bronchial stump or pulmonary tissues in the conditions of acute empyema after operation on lungs.

Lately wide application got thoracoscopy methods, which allow execute aspiration of pus, washing of empyema cavity, destruction of intrapleural encapsulations, deleting of fibrinous membranes from a visceral pleura, destruction of adhesions

between pleura membranes, directed draining of empyema cavity. To the patients with bronchopleural fistulas electro-coagulation of fistula or (at a big their diameter) temporal occlusion of bronchial tubes is executed.

At ineffectivety of micro invasive methods of treatment thoracotomy with plevrectomy is executed and possible resection of area of lung affected by fistula.

Rules of conduct of postoperative period, possible postoperative complications.

In a postoperative period, as a rule, continues the previous conservative treatment. The basic tasks of treatment in a postoperative period are: providing of valuable ventilation of lungs and prophylaxis of hypoxia; correction of water-electrolyte disorders and restoration of adequate hemodynamic; prophylaxis of hypercoagulation and infectious complications.

Possible postoperative complications:

– postoperative bleeding;
– development of remaining cavity;
– suppuration of postoperative wound;
– insufficiency of bronchial stump after resection methods;
– development of stump region abscess.

Medical diagnostic and medical manipulations.

Puncture of pleura cavity

Principles detection of disability and prophylactic medical examination of patient's with acute empyema of pleura.

After passing of course of treatment in hospital the patients with acute empyema of pleura mast be under observation of pulmonologist in a regional policlinic. The operated patients parallel are observed at a doctor-surgeon.

Chronic empyema of pleura

The generally accepted presently *clinical term "chronic empyema of pleura" designates the purulent-destructive process in a remaining pleura cavity with the rough and stable morphological changes, characterized by the protracted flow with periodic intensifications.*

Frequency appearance of chronic empyema is marked at 4-20% of patients with acute empyema of pleura. Big distinctions in frequency of chronic empyema of pleura of such origin are conditioned, foremost, by absence of single picture of criteria of transition of acute form of this disease in chronic. By histological researches of the material, got during pleurectomy, decortications and resections of lung, it is proved, that the stable and irreversible morphological changes of pleura and underlying tissues develop only to the end 2-3 months from the beginning of disease. The signs of disorder of regenerative mechanisms and intensification of purulent process appear in the same terms.

Unlike from acute empyemy of pleura considerably more frequent detects the mixed flora with predominance of Gram-negative bacterias (intestinal, blue pus bacillus).

Forming of remaining cavity can be conditioned by a few reasons:

1. Does not spreading fully lung due to a presence in the pleural cavity of exudate.
2. Presence of adhesions, which prevent spreadness of lung.

3. Considerable compression and sclerosis of pulmonary tissues.

4. Disparity of volumes of resected lung and pleura cavity.

5. Atelectasis of part of lung due to the obstruction of bronchial tree.

If at sharp empyema a lung did not fall out fully, between pleura covers there is a cavity the walls of which are covered by granulation tissues. In course of time this tissues transform to fibred connective tissues, which is denser. A lung in the initial stage of disease is mobile and at the release of cavity of pleura from exudate spreading, and at accumulation of exudate again collapsed. At the protracted flow of exudate inflammation a lung is covered by connective tissues, as by an armour, and loss possibility to fall out. These fibrose impositions on pleura carry the name of shvart. At the protracted flow of illness they achieve a considerable thickness (2-3 cm and more) and density. Consequently, the protracted inflammation is one of reasons of chronic empyemy of pleura.

In the clinic accepted and is used a next classification of empyema of pleura

Features of inspection of patient with suspicion on chronic empyema of pleura.

The clinical picture of chronic empyema of pleura develops gradually. There is no clear border between the clinical demonstration of acute and chronic empyema of pleura, as acute inflammation transforms to chronic. The expressed different of clinical features is characteristic only for the extreme forms of acute and chronic empyema of pleura.

At questioning of patient:

1). Complaints on the basic disease. Usually the disease begins gradually. The permanent symptoms are: increase of temperature of body to 37-38°C, moderate pains in a chest, shortness of breath, cough, presence of discharge from pleura fistula. Pain syndrome - arises up on the side of affectation, aching character, increasing at the deep breathing, cough, at the change of position of body. Cough - quite often with the expectoration of a plenty of sputum, that depends on the degree of affectation of lung, presence of bronchopleural fistula.

2). Complaints from the side of other organs and systems: on a general weakness, poor appetite, insomnia, display of intoxication and other.

3). Anamnesis of disease: the disease begins gradually, in anamnesis there is information about acute empyema of pleura.

4). Anamnesis of life: in anamnesis there can be the frequent cold diseases which can lower the reactivity of organism. It is necessary to pay the special attention on social status of patient.

Clinical physical inspection (characteristic features at this disease):

1). Common state of patient more frequent of middle gravity. Gravity of the state is related to the size of remaining cavity, in which pus detained, and is determined, by the degree of intoxication. Consciousness is clear. Constitutional features are asthenic, possibly expressed exhaustion.

2). Collection of information about original appearance of patient. Skin covers is pale, puffiness, cyanotic'.

3). Inspection of the state of the cardio-vascular system. Palpitation is characteristic. Tachycardia is related to the increase of temperature of body. The tones of hearts are weakened. There is tendention to hypotony. Displacement of heart is possible.

4). Inspection of the state of organs of abdominal cavity. Lowering of appetite. Lowering of mass of the body.

5). Inspection of the state of bones–muscles system — fingers as drumsticks, deformation of nail plates is «watch glasses». Purulent intoxication quite often results of affectation of long tubular bones with development of sclerosis of bone tissues and origin of inflammatory changes in joints.

6). «***Locus morbi***» — inspection of organs of the respiratory system

Limitation of respiratory motions of the affected half of thorax is marked, diminishment of the affected half in a volume, intercostal intervals are narrowed. There is pleural fistula with a purulent discharge.

At percussion above the area of accumulation of liquid dullness is determined, above the area of accumulation of air - tympanitis.

At auscultation weakening of the vesicular breathing up to complete it absence above the big accumulation of liquid is marked. Above the area of the suppressed lung is bronchial breathing, sometimes possible detecting moist rales of different caliber. Over region of the accumulated fluid possible listened bronchophony.

7). Leading clinical symptoms: - intoxication; - respiratory insufficiency

8). On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose the following clinical diagnosis:CHRONIC RIGHT-SIDE /LEFT SIDE/ EMPYEMA OF PLEURA

According to standard charts the plan of additional inspection (laboratory and instrumental) of patient with chronic empyema of pleura:

1). Clinical blood test.

2). Clinical analysis of urine.

3). Biochemical blood test.

4). Coagulogramm.

5). Research of electrolytes of blood.

6). Immunological tests.

7). Bacteriological research of sputum, washing waters of bronchial tree, content of cavity of abscess.

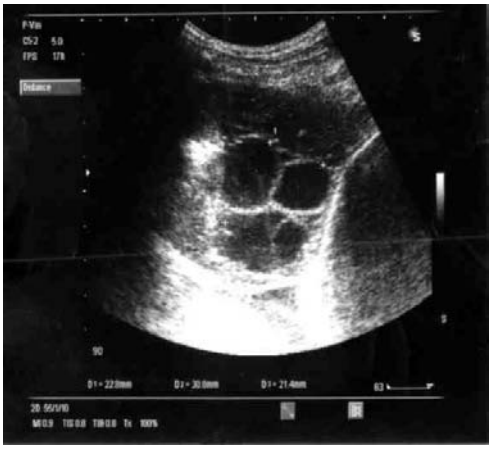


Fig. 10. Ultrasound right pleural cavity — multisacculation empyema

8). Roentgenologic research of organs of thorax (Fig. 10).

9). Spyrography.

10). Fibrobronchoscopy.

1). Clinical blood test: moderate anemia, leucocytosis with neutrophilia, by the change of leucocytes formula to the left, the ESR increase.

2). Clinical analysis of urine: the changes are unspecific is presence of signs of toxic nephropathy: albumynuria, cylyndruria.

3). Biochemical blood test: it is sharply expressed hypoproteinemia, dysproteinemia.

4). Coagulogramm: violations of coagulation to side of hypercoagulation with diminishment of time of clot formation increase of level of fibrinogen.

5). Research of electrolytes of blood: hyperpatassemia explainable by disintegration of tissues and elements of blood is marked.

6). Immunological tests: the decline of indexes of reactivity of organism is characteristic.

7). Bacteriological research of sputum and content of remaining cavity allows detecting the exciters of inflammatory process and defining the sensitiveness of the last to antibacterial preparations.

8). Roentgenologic research of organs of thorax: roentgenologic research at chronic empyema of pleura has big value, allows exactly make of diagnosis and define the nearest tactic of treatment of patient. Polyposition roentgenoscopy allowing localizing of the affectation is most informing, exactly to define the degree of collapse of lung and displacement of mediastinum, amount of liquid, to expose the pathological changes in pulmonary parenchyma, to detect a point for adequate draining of remaining cavity.

Tomography allows to answer on foregoing questions. Pleurography in 3 projections is the very informing method of research. It allows to estimate the sizes of remaining cavity, character of its walls, presence of sequesters and fibrinous stratifications. At pleurography in position on healthy side the areas of bronchial tree are contrasted quite often, that matters very much for further medical measures (implementation of temporal endobronchial occlusion, optimization of the regime of washing of pleural cavity).

Bronchography allows to estimate the state of bronchial tree, to detect localization and character of bronchopleural fistula, to found the reason of chronic flow of process (bronchiectasis, chronic abscess, etc.). The bronchography signs of heavy changes in lungs are: 1) presence of «empty area» from the uncontrasted bronchial tree in the collapsed segments of lung; 2) convergence of bronchial tubes with diminishment of angles of their branching; 3) different types of deformations, bends of bronchial tree, quite often with formation of bronchiectasis.

9). Spyrography are the low indexes VVL, decline of compensate possibilities of lungs, decline of satiation of arterial blood by oxygen.

10). Fibrobronchoscopy gives information about the degree of expressed of inflammatory process in a tracheobronchial tree, allows to define the mouth of draining bronchi (at presence of destruction in pulmonary tissues). Ability of bronchoscopy rises at introduction of dye-stuff material (water solution of methylen dark blue) to the pleura cavity in position on healthy side. It allows to define, what bronchial tubes participate in draining of area of destruction of pulmonary tissues, that is very important for planning of level of temporal endobronchial occlusion.

Differential diagnostics: it is conducted with osteomyelitis of ribs, breastbone and spine.

Ground and formulation of clinical diagnosis (taking into account classification of disease, presence of complications and concomitant pathology):

1) basic — Chronic empyema of pleura;

2) complications — (if it is);

3) concomitant: pathology — (if it is).

Treatment of patient with chronic empyema of pleura

Choice of medical tactic: presently combination of the surgical methods of treatment, directed on evacuation of purulent content of pleura cavity, and conservative therapy which is examined as basic part of preoperative preparation and postoperative conduct of patients, is used in treatment of chronic empyema of pleura. At the choice of methods of treatment at chronic empyema of pleura it should be remembered that conservative treatment rarely conduces to convalescence. The special difficulties arise up in that case, when bronchial fistula is opened in a remaining cavity. Only operation in such cases can gives success of treatment.

The pathogenetic grounded conservative therapy is directed on the straggle against three basic pathological factors — suppurations, losses and resorption. Only by local treatment (surgical methods of sanation) — without influence on all organism, without normalization of the disordered functions of organs and liquidation of remaining complications, without therapy of primary and concomitant diseases — it is impossible to treat patients with chronic empyema of pleura.

To such patients indicated complex therapy including etiologic, pathogenetic, symptomatic and local treatment. Intensity of treatment changes depending on the common state of patient, presence of concomitant diseases and complications, from the stage of purulent process and character of empyema.

Application of antibiotics is the basic method of therapy, sulfanilamide and antiseptic facilities. Antibiotic therapy must include 2-3 preparations, which operate on pathogenic aerobic and anaerobic microorganisms.

Pathogenetic therapy consists in application of anti-inflammatory facilities broncholitics (lasolvan, bronchobru, flumuzil), in conducting of the measures directed on correction of violations of proteins (transfusions of fresh-frozen plasma, solutions of amino acids), mineral (infusion of crystalloid solutions), carbohydrate exchanges (infusion of solution of the glucose 5%); oxygen therapy (5-7 litres in a minute), desintoxic treatment (sorbilact, reosorbylact, reombirin).

In complex treatment of patients with empyema of pleura the big value has the valuable feed with the rich content proteins and vitamins. At the exhausted patients anabolic hormones are used (retabolil for 50 mgs, superanabolon for 1-2 ml in 1 times per a week, nerobol for 0,002 three times per a day and others).

Existent methods of operative treatment and indications to them

To operative treatment usually used, when the conservative treatment does not give success due to rigidity pectoral wall, in case fixation of collapsed lung by adhesions, at presence of big bronchial fistula opened in a pleura cavity, and at with the decline of regenerative capabilities.

The basic task of operative treatment is liquidation of remaining cavity and closing of pleurobronchial fistula.

The methods of deleting of pus from a pleura cavity and its sanation are the basic methods of local preparation to radical operations. Depending on the sizes of cavity deleting of pus can be produced by punction and by draining. Draining of cavity of empyema is more preferable and if necessary fraction continuous irrigation of purulent cavity by solutions of antiseptics.

Lately wide application got thoracoscopy methods, which allow executing aspiration of pus, washing of empyema cavity, destruction of intrapleural encapsulations, deleting of fibrinous adhesions from a visceral pleura, destruction of joints between pleural

membranes, directed draining of empyema cavity. To the patients with bronchopleural fistulas electro-coagulation of fistula or (at big their diameter) temporal occlusion of bronchial tubes is executed.

At ineffective of micro invasive methods of treatment the operative interferences are indicated. There are a few groups of operative methods of treatment:

1. Restorative operations (pleurectomy and decortications of lung) (Fig. 11).

2. Resection-restorative operations (pleurectomy and decortications of lung with lobectomy, segmentectomy, wedge form-resection, flat resection).

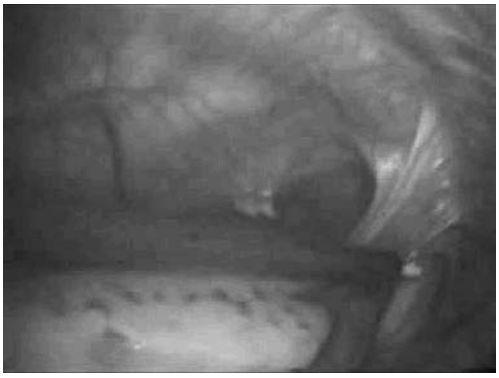


Fig. 11. Videothoracoscopic destruction of pleural joints

3. Resections (pleuropneumoectomy).

4. Reconstructive- plastic operations (thoracoplastyc and muscles plastic with the resection of lung or with plastic operations on stump of bronchi).

5. Occlusion and reamputation of stump of bronchi from transpleural, transsternal, contra lateral or transmediastinal access.

6. Thoracotomy with sanation and draining of empyema cavity.

Rules of conduct of postoperative period, possible postoperative complications

In a postoperative period, as a rule, the before begun conservative therapy continues to be conducted. By the basic tasks of treatment in a postoperative period are: providing of valuable ventilation of lights and prophylaxis of; correction of water – electrolyte disorders and restoration of adequate hemodynamic; prophylaxis of hypercoagulation and infectious complications.

Possible postoperative complications:

– hypoxia,
– postoperative bleeding;
– pneumonia;
– suppuration of postoperative wound;
– relapse of bronchial fistula;
– development of sharp postoperative empyema.

Medical diagnostic and medical manipulations. Punction of pleura cavity

Principles detection of disability and prophylactic medical examination of patients with chronic empyema of pleura.

After passing of course of treatment in hospital patients with chronic empyema of pleura must be observed at pulmonologist in a regional policlinic. The operated patients parallel are observed at a doctor-surgeon.

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I.2. Chest injuries

The amount of patients with the trauma of thorax steadily grows in connection with technical progress and increased motorization of the country. In the economically developed countries trauma, as a reason of death, occupies the third place after cardio-vascular and oncologic diseases. More than one hundred thousand persons annually die in the USA from accidents, and trauma remains the leading reason of death of people of able to work age. Among reasons of traumatic injuries of thorax a road traffic accident (RTA) occupies the special place on the stake of which, according to the data of numerous authors, there are 60-90% cases of the closed injuries of thorax.

Trauma of thorax is one of the heaviest types of injury. Even at modern achievements of surgery and anesthesiology lethality at the heavy trauma of thorax remains high and makes from 10 to 35%, and at the concomitant injuries of other organs, met in 80% cases, it is increased to 50-60%. Among reasons of lethality from a trauma the trauma of thorax occupies the second places after the craniocerebral trauma. Principal reasons of death of victims with the trauma of thorax are the injury of lung, wounds of heart, fat embolism, bleeding, traumatic pneumonia. Consequently, injuries of thorax are of special danger and conditioned, above all things, by location of vital organs in it.

For the last years in connection with growth of traffic traumatism interest to the closed traumas of thorax both for domestic and foreign surgeons has risen considerably.

The row of fundamental researches, which engulf the circle of questions, touching the analysis of mechanogenesis of the injuries of thorax and its organs, diagnostics, clinical picture and treatment of heavy injuries of thorax, appeared. D. D. Trunkey (1982) marks that according to the number of the taken away lives accidents, arising up only on roads as a result of road traffic accidents, on the amount and severity are tantamount to permanent war of middle scale. Probability of fatal outcomes for victims with the heavy injuries of thorax depends on age, amount and severity of concomitant injuries, and also on before existing changes in vital organs (lungs, heart, liver, kidneys etc.). Thus, the further increase of frequency, severity and lethality of the closed trauma of thorax due to growth of road traffic accidents has been marked within the last decades. Growth of amount of traumas, financial expenses, expended on warning of traumatism, treatment and rehabilitation of victims, and also on payment of manuals give to the traumatism tone of the social phenomenon of special meaningfulness.

In a clinical picture accepted and next **classification of trauma of thorax** is used (E.A. Wagner, 1981). The injuries of thorax, as well as all traumas, are divided on:

– *isolated,*
– *plural,*
– *associated,*
– *combined.*

Isolated is a trauma of one organ within the limits of one anatomic area. Plural is a trauma of a few organs within the limits of one anatomic area. Multitrauma or polytrauma is an injury of a few organs in different anatomic areas. The combined trauma is injuries, arising up at affecting organism etiologic different injuring factors.

All traumas of thorax are divided by 2 large groups: opened and closed.

I. **Classification of the opened injuries of thorax (wounds) of thorax**.

1. On localization of injury: one-sided and two-sided.
2. According to type scotching weapon : stab-cutting and gunshot [missile] wound.
3. In grain wound channel: blind and through.
4. In grain wounds: penetrating and nonpenetraiting. The injury of parietal sheet of pleura serves as a criterion.
5. Penetrating wounds are divided by 2 groups: with the injury of organs and without a injury.

A separate group is select thoracoabdominal wounds at which a diaphragm and wound channel passes through two cavity: pleura and abdominal.

II. Classification of the closed trauma of thorax.

1. Without the injury of bone sceleton of thorax are injuries, haematomas, tear of tissues.
2. With the injury of bone sceleton of thorax are fractures of ribs, breastbone, collar-bone, shoulder-blade.
3. Without the injury of viscera.

III. **Thoracoabdominal wounds** are divided by the followings groups:

1. Without the injury of organs of abdominal and thoracic regions.
2. With the injury of organs of thoracic cavity.
3. With the injury of organs of stomach and retroperitoneum space.
4. With the injury of organs of thorax, stomach and retroperitoneum space.
5. With the injury of viscera (lung, heart and large vessels, trachea and bronchus, esophagus and organs of posterior mediastinum).

Classification of the closed injuries of thorax (on And. E. Romanenko, 1982).

I. On the presence of injuries of other organs:

1. Isolated trauma.
2. Combined trauma.

II. On the mechanism of trauma:

1. Hurt.
2. Compression.
3. Concussion.
4. Fracture.

III. In grain injuries of thorax:

1. Without violation of integrity.
2. With violation of integrity of ribs, breastbone, spine.

IV. In grain injuries of organs of thoracic cavity:

1. Without the injury of viscera.
2. With the injury of viscera (lung, heart and large vessels, trachea and bronchus, esophagus).

V. On the presence of complications:

1. Uncomplicated.
2. Complicated:

– *early complications* (pneumothorax, hemothorax, subcutaneous emphysema, pneumomediastinum, floated fracture of ribs, traumatic shock, asphyxia);

– *late complications* (traumatic pneumonia, pleurisy, festering diseases of lungs and pleura).

VI. On the state the cardiovascular and respiratory systems:

1. Without the phenomena respiratory and cardiovascular insufficiency.
2. Sharp respiratory insufficiency (I, II, III degrees).
3. Acute respiratory failure (I, II, III degrees).

VII. On the degree of severity of trauma:

1. Easy.
2. Middle.
3. Heavy.

Injury of bones of the thorax

The direct action on the chest wall of injuring factor results in the fracture of ribs and breastbone. The fracture of breastbone often arises up as a result of trauma at the helm of car, localized in most cases in the upper and middle third.

From data of literature, in 41–48 % cases fractures of ribs recognized too late. It is related to that at the plural trauma of thorax basic attention is spared the injury of intrathoracic organ, requiring urgent treatment, and diagnostics of fractures of ribs is moved aside on the second plans. Depending on the mechanism of trauma the fractures of ribs are subdivided into direct, arising up in the place of blow, undirected, arising up in the distance from the place of appendix of injuring force, and combined.

The so-called "fenestrated" fractures of ribs flow especially heavily. There are fractures for to 2-3 anatomic lines, attended with pathological mobility of area of thorax. This phenomenon, observed at 17,8% patients, in literature described under the different names: "floated thorax", "float of thorax", " fenestrated fractures of ribs", "costal panels".

Classification of floated fracture of ribs:

1. A central floated segment is plural fractures of ribs on parasternal or middle-clavicular lines.

2. An anterior-lateral floated segment is plural fractures of ribs on parasternal and anterior-axillar lines.

3. A lateral floated segment is plural fractures of ribs on front and back axillar lines.

4. A back floated segment is plural fractures of ribs on back-axillar and paravertebral lines.

Physiopathology of this phenomenon consist in that at every inhalation in the moment of decline of intrapleural pressure the mobile segment of chest wall falls back, the considerable area of lung is squeezed as a result, and the air contained raised level of carbonic acid and lowered of oxygen is directed in an opposite lung and in the areas of that lung, not constrained by the floated segment of chest wall. The persons with the "floated" fractures of ribs require urgent medical measures.

Features of inspection of patient with the traumatic injury of bones of thorax. (Plan of practical preparation of student on practical lesson.) At questioning of patient:

1) Patients complaints on a sharp local painfullness in the site of injury, which increases at the deep breathing, cough and change of position of body of patient; shortness of breath at peace, general weakness.

2) Complaints from the side of other organs and systems: sense of interruptions in area of heart.

3) Anamnesis of illness: patients indicate on a trauma.

4) Anamnesis of life: the presence of harmful habits and chronic diseases is specified. 2.2.

Clinical inspection (characteristic features at this disease):

1). Estimation of the general state of patient. The general state of patient, as a rule, varies from the middle degree of severity to extremely heavy.

2). Collection of information about original appearance of patient: cyanosis of skin covers is possible. Examination of hypoderm, palpation of lymphatic nodes, thyroid and thoracic glands.

3). Inspection of the state of the cardio-vascular system: tachycardia is typifying heart sounds are muffled, arrhythmia is possible.

4). Inspection of the state of organs of abdominal region can be without features.

5). Inspection of the state of musculoskeletal apparatus: (examination and palpation).

6). «Locus morbi» is an inspection of the state of organs of the respiratory system (examination of thorax and upper respiratory tracts, palpation of thorax, percussion and auscultation of lungs).

7). Pain in a thorax is the leading clinical syndrome for the fracture of ribs and breastbone.

8). For example, preliminary clinical diagnosis: Closed trauma of thorax. Fracture of the V rib on the left.

The plan of additional inspection (laboratory and instrumental) of patient with the trauma of thorax includes:

1). CBC – as the result of possible bleeding (decline of Hb, red (blood) cells), increase of ESR.

2). Clinical uranalysis - changes can be not.

3) Determined the measure of bleeding.

4). Blood group and Rh-factor.

5). Coagulogram - changes can be not.

6). X-ray examination of thorax: the fractures of ribs, breastbones, pneumothrax, haemothorax).

7). ECG: (tachycardia, signs of myocardium hypoxia).

8). Bronchoscopy: receipt of scarlet blood from bronchus at the injury of lung.

9). Ultrasonic research of pleura cavity: hemothorax is determined in a pleura.

10). Ultrasonic research of heart is most informing at the trauma of heart: the decline of contractile function of myocardium, the external and internal fractures of heart are visualized.

11). Puncture of pleura cavity - serves as diagnostic and medical manipulation at suspicion on a hemopneumothorax.

12). An angiography enables to specify a diagnosis and exactly to define the place of defeat of heart, aorta and other large vessels of средостения.

13). Thoracoscopia is a high-informing method, allows to specify character, localization of injury.

Differential diagnostics. At presence of trauma in anamnesis, characteristic symptomatology, diagnostics of fracture of ribs and breastbone does not present difficulties.

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

1) basic: Closed trauma of the left half of thorax. A fracture of the V ribs is on the left with displacement;

2) complications: left-side haemothrax;

3) concomitant pathology (if it is).

Medical tactics of patient with the traumatic injury of thorax.

1 *Choice of medical tactics*. Treatment of fractures of bones of thorax depends on the size of trauma, presence of concomitant injuries of intrathoracic organs and complications. The basic method of treatment of the uncomplicated single fractures of ribs is conservative.

2. *Treatment of patient with the traumatic injury of lung.*

The traumas of lung, arising up at road traffic accidents, are subdivided i***nto fractures and contusions***.

Choice of medical tactics: Medical tactics at the traumatic injury of lung depends on the type of injury. Contusion of lung mainly subject conservative treatment. Treatment of fractures of lung and penetrable wounds of thorax with the injury of lung mainly surgical and depends on the degree of destructions of pulmonary tissue.

3. *Surgical treatment.*

Operative measures divided as ***reanimation***, urgent, deferred urgent and deferred.

Reanimation operations are indicated to suffering with the heavy trauma of intrathoracic organs and uncontrolled bleeder expressed acute respiratory failure or their combinations which conservative treatment having no prospects at. An operation is conducted regardless of severity of the state of patient, at once after determination threatening life of injury, during 20-30 mines from the moment of hospitalization. . Reanimation thoracotomy, reanimation tracheotomy, thoracocentesis and draining of mediastinum. refer to the reanimation operations at the isolated trauma of thorax.

Urgent operations (produced during 0,5–2 hours after hospitalization) are indicated to suffering with the expressed signs of shock and in less degree - bleeding and respiratory insufficiency.

The deferred urgent are performed through 2-6 h from the hospitalization. These operative interferences are produced to the patients with the vast injuries of soft tissure of thorax, plural fractures of ribs, fracture of lung, by a hemopneumothorax and with other complications of trauma, when intensive therapy directed on the removal of hypovolemia, anaemia, hypoxia, acidosis, appears uneffective.

Surgical interferences, executed after 6 h from the hospitalization, after the treatment from a grave condition and relative correction of haemodynamic indexes named as the deferred operations.

At suspicion on haemothrax with a diagnostic and medical purpose pleura puncture is used. Discovery at puncture more than 100 ml of blood and repeated its accumulation is an indication to the draining.

Indications to thoracotomy at the trauma of thorax:

1) continuing intrapleural bleeding, more than 150-200 ml of blood during an hour;

2) intrapericardial bleeding with development of cardiac tamponade;
3) extrapericardial cardiac tamponade;
4) hemorrhage in the mediastinum with the compression of respiratory tracts and main blood vessels;
5) increasing, in spite of draining, tense pneumothrax and mediastinal emphysema.

Rules of conduct of postoperative period, measures on a prophylaxis, diagnostics and treatment of possible postoperative complications at the trauma of thorax.

A primary value after operation has prophylaxis of atelectasiss, pneumonias, abscesses of lungs, empyemas of pleura, pericarditises, pulmonary embolism, suppurations.

At the trauma of thorax the origin of such urgent states, as tense pneumothorax, total hemothorax, mediastinal emphysema.

Puncture of pleura cavity. Puncture of pleura cavity serves as diagnostic and medical manipulation at suspicion on a hemopneumothorax.

Research of content of pleura cavity ***is the test of Ruvilua-Greguar*** – estimated as positive when clotting of the extracted blood and specifies on the continuing bleeding in pleura cavity.

The trauma of thorax is origin of complications. Early are pneumothrax, hypodermic and mediastinal emphysema, haemothrax, floating fracture of ribs, traumatic shock, asphyxia. Late are traumatic pneumonia, pleurisy, festerings diseases of lungs and pleura.

Classification of pneumothorax.

I. ***On prevalence of process:***

1. One-sided.
2. Bilateral.

II. On the degree of detelectasis:

1. Partial (a detelectasis is to 1/3 volume).
2. Subtotal (a detelectasis is to 2/3 volume).
3. Total (a detelectasis is a more than 2/3 volume).

III. On the mechanism of origin:

1. Closed.
2. Opened.
3. Valvular.

Closed pneumothorax is complication, which arises up at the injury of visceral sheet of pleura, results in entering of air pleura cavity and stipulates the collapse of lung. At the closed trauma of thorax reason of origin of closed pneumothorax is a perforation of visceral pleura and pulmonary tissue by injured fragment of rib.

Opened pneumothorax arises up because of formation of defect of chest wall at massive traumas and free receipt of air during inhalation in a pleura cavity, and at expiration - outside.

Valvular pneumothorax arises up at the injury of pulmonary tissue or chest wall with formation of valve, when air on inhalation enters pleura cavity, and on expiration, in connection with closing of valve, keeps indoors outside.

Subcutaneous emphysema. Reason of origin of this complication is an injury of parietal and visceral sheets of pleuraby fragment of rib within flow of air from pul-

monary tissue in pleura cavity and through the in jured chest wall (fracture of intercostal muscles) in hypoderm.

Mediastinal emphysema. It is complication is characterized by accumulation of air in the adipose tissue of mediastinum. Reason of mediastinum emphysema is partial or complete fractures of trachea, bronchus and esophagus.

Hemothorax. It is an accumulation of blood in a pleura cavity (Fig. 12). Reason of origin of this complication is an injury of vessels of chest wall, pleura, lung and mediastinum.

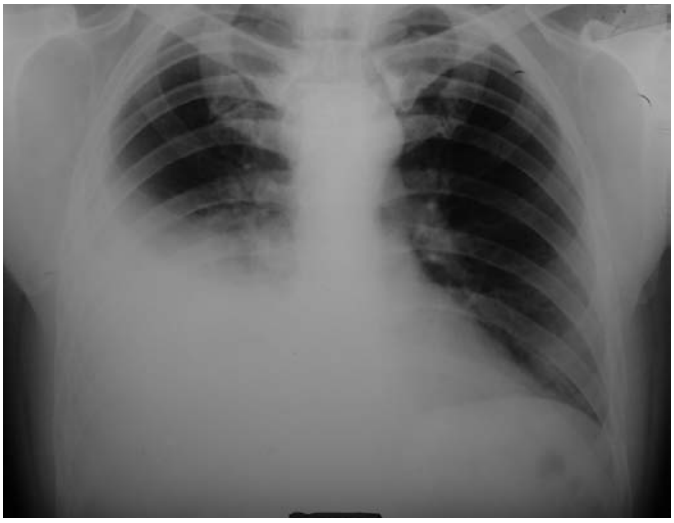

Fig. 12. Right medium posttraumatic hemothorax (after closed chest trauma)

Classification of hemothorax (E. And. Wagner, 1981)

I. On prevalence of process:

1. One-sided.
2. Bilateral.

II. On the size of blood loss:

1. Small (a loss is a to 10% volume of circulatory blood
2. Middle (loss to 10 - 20% circulating blood volume)
3. Large (loss to 20 - 40 % circulating blood volume) 4. Total (more than 40% circulating blood volume)

III. On continuation of bleeding:

1. With continueing bleeding.
2. With the stopping bleeding.

IV. On the presence of clots in a pleura cavity.

1. Coagulated.
2. Uncoagulated.

V. On the presence of infectious complications:

1. Uninfected (Germ-free).
2. Infected.

Literature 1. Hospital. surgery / Edited by L.J.Kovalchuk, J.P.Spizhenko, V.F.Sayenko and others. Ternopyl: Ukrmedkniga, 1999. – 560 p. 2. Bailey and Love's. Short practice of surgery. H.K.Levis and Co LTD, 1992

Chapter II. Surgical pathology of the heart

II.1. Acquired valvular disease

The acquired heart diseases, affecting people of different age, quite often lead to resistant disability. The social significance of this problem is determined by the fact that often young men suffer from acquired heart diseases. At the same time there are doubtless successes of the cardiosurgery in the developed countries that promoted significant improvement of the results of the treatment of this disease.

In the general structure of the acquired heart disease a lesion of the mitral valve, and also combination of the pathology of mitral and aortal valves occur more often. According to the data of the work of N.M. Amosov and J.A. Bendet (1983), founded on experience of 14 752 operations at the acquired heart diseases, the isolated lesion of the left atrioventricular valve makes 71,5 %, the valve of an aorta - 8,8 %, combined (associated) lesion of mitral and aortal valves - 12,3 %. The lesions of the tricuspid valve in combination with other malformations compound about 7,4 %.

At the present stage surgery of the acquired heart disease has various variants of the highly effective operative measures providing the favorable long-term results. Nevertheless, there are problems for reconstructive surgery of valves pathology, a prosthetic endocarditis, primary infective endocarditis, a pathology of an ascending aorta, a prosthetic repair at narrow root of aorta, multivalvular disease, and pathology of the valves combined with ischemic heart disease and arrhythmias of heart.

Classification of the acquired heart diseases.

Kiev 2000, VI National congress of cardiologists of Ukraine:

Mitral stenosis

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

On stages:

I stage — compensation;
II stage — pulmonary congestion;
III stage — right ventricular failure;
IV stage — dystrophic;
V stage — terminal.

Mitral insufficiency

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

On stages:

I stage — compensation;
II stage — subcompensation;
III stage — right ventricular decompensation;
IV stage — dystrophic;
V stage — terminal.

The combined rheumatic mitral disease (a rheumatic mitral stenosis with insufficiency).

– With predominance of a stenosis: stages and indications to surgical treatment, as at a mitral stenosis;
– With predominance of insufficiency: stages and indications to surgical treatment, as at mitral insufficiency;
– Without evident predominance: stages and indications to surgical treatment, as at mitral insufficiency;
– mitral valve prolapse.

Aortal stenosis.

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

On stages:

I stage — full compensation;
II stage — latent heart failure;
III stage — relative coronary insufficiency;
IV stage — expressed left ventricular failure;
V stage — terminal.

Aortal insufficiency.

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

On stages:

I stage — full compensation;
II stage — latent heart failure;
III stage – subcompensation;
IV stage — decompensation;
V stage — terminal.

The combined aortal disease.

On etiology:

– Rheumatic aortal stenosis with insufficiency;
– Not rheumatic aortal (valvular) stenosis with insufficiency (with specification of an etiology).
– With predominance of a stenosis: stages and indications to surgical treatment correspond to aortal stenosis;
– With predominance of insufficiency: stages and indications to surgical treatment correspond to aortal insufficiency;
– Without evident predominance: stages and indications to surgical treatment correspond to aortal stenosis.

Tricuspid stenosis.

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

Tricuspid insufficiency.

On etiology:

– Rheumatic;
– Not rheumatic (with specification of etiology).

Combined Tricuspid disease.

On etiology:

– Rheumatic tricuspid stenosis with insufficiency;
– Not rheumatic tricuspid stenosis with insufficiency (with specification of etiology).

Pulmonary artery stenosis. Pulmonary insufficiency.

Combined pulmonary disease (pulmonary artery stenosis with pulmonary insufficiency).

***Associated (multiple) heart diseases*:**

– associated lesion of mitral and aortal valves;
– associated lesion of mitral and tricuspid valves;
– associated lesion of aortal and tricuspid valves;
– associated lesion of mitral, aortal and tricuspid valves.

The evidence of "simple" heart disease is determined by three degrees:

I degree — slight;

II degree — moderate;

III degree – severe.

Considering that the calcification of the valve determines tactics of surgical intervention, there are 3 degrees of a calcification (Knyshov G.V., Bendet J.A., 1997):

I degree (+) — separate calcium lumps in strata of commissures or cusps;

II degree (++) — a significant calcification of cusps and commissures without valve ring involvement;

III degree (+++) — a massive calcification of the valve with transferring to the fibrous ring, and sometimes on wall of aorta and myocardium.

New York Heart Association (NYHA) Functional Classification

I) *ordinary* physical activity does not evoke symptoms (fatigue, palpitation, dyspnea, or angina).

II) *slight* limitation of physical activity; comfortable at rest; ordinary physical activity results in symptoms.

III) *marked* limitation of physical activity; less than ordinary physical activity results in symptoms.

IV) *inability* to carry out any physical activity without discomfort; symptoms may be present at rest.

Canadian Cardiovascular Society (CCS) Functional Classification

I) *ordinary* physical activity does not cause angina; angina only with strenuous or prolonged activity.

II) *slight* limitation of physical activity; angina brought on at > 2 blocks on leyel (and/or by emotional stress).

III) *marked* limitation of physical activity; angina brought on at ≤ 2 blocks on leyel.

IV) *inability* to carry out any physical activity without discomfort; angina may be present at rest.

Classification of heart failure by structural abnormality (ACC/AHA), or by symptoms relating to functional capacity (NYHA)

ACC/AHA stages of heart failure	NYHA functional classification
Stage of heart failure based on structure and damage to heart muscle	Severity based on symptoms and physical activity
Stage A. At high risk for developing heart failure. No identi ed structural or functional abnormality; no signs or symptoms.	Class I. No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnoea.
Stage B Developed structural heart disease that is strongly associated with the development of heart failure, but without signs or symptoms.	Class II Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
Stage C. Symptomatic heart failure associated with underlying structural heart disease.	Class III. Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnoea.
Stage D. Advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy.	Class IV. Unable to carry on any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased

The classification of chronic circulatory deficiency, offered by F. Lang, X. Vasilenko and X. Stragesko (1935).

There are three stages.

Stage I. The initial, latent circulatory deficiency is displayed by a dyspnea, palpitation and fatigability only at an exercise stress. In rest these signs peter. The hemodynamics is not disturbed.

Stage II. In this stage evolve two periods. The Stage II A - attributes of a circulatory deficiency are expressed moderately in rest , tolerance to an exercise stress is reduced; there are moderate disturbances of a hemodynamics in the big and small circle of the blood circulation. The Stage II **B** - in rest there are attributes of a heart failure; serious hemodynamic disturbances are marked both in big and small circle of a blood circulation.

Stage III. A final, dystrophic stage with the expressed disturbances of the hemodynamics and metabolism, irreversible changes in organs and tissues.

There are *three forms of a rheumatic carditis* - weak, moderate and expressed (A.I. Nesterov 1973).

I degree of activity (weakly expressed). Rheumatic carditis can be observed at any variant of primary or recurrent rheumatic disease. At acute and subacute flow of primary rheumatic disease clinico-laboratory attributes of high activity of rheumatic process are characterized by extracardiac syndromes.

II degree of activity (moderately expressed) rheumatic carditis educes at primary and recurrent rheumatic disease, with acute or subacute flow.

III degree of activity (expressed) rheumatic carditis is found out usually at acute and subacute flow of primary rheumatic disease. The clinic picture is defined by inflammation spreading on one, two and rarely three strata of heart (pancarditis).

Stenosis of the mitral valve

Clinical course of stenosis of the mitral valve.

I stage – full compensation. Hemodynamic disturbances are caused by insignificant {slight} narrowing of a mitral ostium (its area compounds 2- 2,5 sm^2). Pressure in the left auricle increased up to 10-15 mm hg Clinical onset is minimal:

– Complaints are absent;
– The working capacity is not limited.

II stage – pulmonary stagnation. It is characterized by narrowing of a mitral ostium up to 1,5-2 sm^2, pressure in the left auricle raises up to 20-30 mm hg, the pulmonary pressure also increses that enlarges the pressure on the right ventricle. In rest – normal minute volume of a blood which is not provided at an exercise stress. Clinical symptoms:

– A dyspnea at an exercise stress;
– Attributes of a hypertensia in a small circle with frequent development of complications: tussis, a pneumorrhagia, attacks of a dyspnea, more often at night, lungs edema;
– The working capacity is limited;
– no right ventricle decompensation

III stage – right ventricle failure. It is characterized by a resistant hypertensia in a small circle of a circulation of blood with formation of "the second barrier», intensifying of work of a right ventricle and development of its insufficiency. Sclerosis of the pulmonary vessels, decrease of a pulmonary blood flow causes rarer attacks of a cardiac asthma and lungs edema or their petering. Dystrophic changes in parenchymatous organs are expressed moderately. The area of a mitral ostium compound 1-1,5 cm^2. Clinical symptoms:

– A dyspnea in rest;
– Originating of attacks of a cardiac asthma;
– Hemoptysis occurs (it can be dry or with a sputum)
– The pneumorrhagia is possible;
– Periodical palpitation troubles patients;
– Pains in the region of heart;
– Sense of weakness and undue fatiguability;
– Peripheral edemas in the evenings;
– An intolerance of physical loads;
– Attributes of the right ventricle decompensation.

IV stage – dystrophic. The area of a mitral ostium is less than 1 sm^2. It is characterized by the expressed disturbance of a circulation of blood in the big and small circle. Patients are practically invalid. Following attributes are characteristic:

– The expressed dyspnea, strengthens in a prone position because of what the patient is forced to be in a semi-sitting position or sitting (orthopnea);
– Attacks of a cardiac asthma which are accompanied by tussis with excretion of a pink foamy sputum;

– A hemoptysis;
– Constant palpitation with development of the constant form of an atrial fibrillation; - Pains in the region of heart;
– Constant peripheric edemas;
– Feeling of gravity in epigastric area and hypochondrium;
– Weekness and undue fatiguability are severe.

V stage – terminal. The clinic corresponds to the III stage of N. D. Strazhesko, V. H. Vasilenko's classification. It is characterized by serious disturbances of a circulation of blood with irreversible degenerative changes of the internal organs (liver, kidneys) and an ascite, atrophy of the muscular system.

Complaints from other organs and systems. Disturbance of a swallowing because of sharp enlargement of the left auricle, and also pain and feeling of gravity in epigastric region and the right hypochondrium caused by the above-named causes are marked.

The anamnesis of disease. At assembly of the anamnesis it is necessary to specify the cause of originating of disease. In spite of the fact that the rheumatic genesis of the defect occurs at the majority of patients (practically at 100 % of cases), only at a part of them in the anamnesis it is possible to determine the attributes of the tolerated rheumatic attack, in these cases complaints, characteristic for a mitral stenosis, occur gradually, usually 5 – 20 years after "attack". The beginning of the disease is acute much rarer. At 30-40 % of patients the rheumatic attacks are not marked in the anamnesis.

The anamnesis of life (including working conditions, professional harmfulnesses, etc.; at women the obstetrical-gynecologic anamnesis).

Data of objective survey (characteristic features at the given disease):

1) The General state of the patient with the stenosis of the mitral valve depends on a stage of the stenosis. At the I, II stage of the disease the general state of the patient does not change.

2) The collecting of the information on a external state of the patient with the stenosis of the mitral valve (Survey of the skin, subcutis- fatty stratum, the palpation of the lymph nodes, the palpation of the thyroid and mammas). At the expressed mitral stenosis and pulmonary hypertensia at patients the typical mitral face (facies mitralis) is quite often observed – in the background of the pale facial skin there is a sharply outlined blush of cheeks with a cyanosis of labiums and the end of the nose («the mitral butterfly», or «the mitral blush»). «The mitral butterfly» is an attribute of the sharply expressed process. At patients with a high pulmonary hypertensia at an exercise stress the cyanosis strengthens, and also there is a grayish colouring of the skin («the ashy cyanosis»), that is caused by low minute volume of the heart.

3) The Survey of the state of respiratory organs of the patients with the mitral stenosis (the Survey of the thorax and the upper airway, palpation of the thorax, percussion and auscultation of lungs).

Survey of the state of the abdominal organs of patients with the mitral stenosis (survey of the abdomen, palpation and succussion of the stomach, palpation of the intestine, liver, spleen, pancreas, kidneys, organs of the small pelvis, auscultation of the abdomen).

5) Survey of state of the osteomuscular apparatus of patients with the acquired heart disease (Survey and palpation).

6) ***Local status*** (***Locus morbi***). Survey and palpation of the heart region and superficial vessels, palpation of the main vessels of the extremities and neck in the projective points, definition of the percutory borders of heart, auscultation of heart and vessels with obligatory definition of specific "pathognomonic" symptoms of the assumed acquired defect by means of additional technical manual methods, to describe the technique of their conduction.

At the high pulmonary hypertensia the enhanced presystolic pulsation of cervical veins (waves "a") can be defined, which at the fibrillation and especially at the development of relative insufficiency of the tricuspid valve peters is replaced by the systolic one (a positive venous pulse, waves ("c + v"). Pulsus differens is characteristic owing to the appression of the left subclavian artery by the extended left auricle.

At the Survey and palpation of the thorax the apex beat is sharply weakened or not defined, as the left ventricle is pushed back by the hypertrophied right one. The pulsation of the right ventricle during the systole is well defined under the ensisternum (it is less often observed in the third –fourth intercostals at the left edge of the breast bone) and strengthens while breathing owing to the augmentation of its filling. At the exhausted patients, it is possible to observe some protrusion in the region of the lower third of the breast bone – « a cardiac hunch ». The closure of valves of the pulmonary artery (P2) in the second intercostals on the left can be defined by palpation. The diastolic tremor is characteristic– "the cat's purring" – in the region of the apex which is better defined on the left side.

The data of the heart auscultation have important diagnostic value:

a) the strengthened clapping 1st tone on the apex and in Botkin point;
b) flick of the opening of the mitral valve, this 3d tone appeares after the 2d (it is auscultated above the heart apex or in the fourth intercostals on the left at the edge of the breast bone);
c) the stress of the 2d tone above the pulmonary artery;
d) diastolic murmur.

7) The diagnosis of the mitral stenosis is defined on the basis of:

a) the characteristic melody of heart (see above);
b) hypertrophies LA and RV on the electrocardiogram;
c) mitral configuration of heart with the augmentation of the LA and the trunk of the pulmonary artery at the X-ray Survey. The diagnosis is confirmed by means of the echocardiography.

8) On the basis of the received data of the interrogation, of the anamnesis of disease and clinical physical Survey of the patient to define the *preliminary clinical diagnosis* to the patient with mitral stenosis: «Rheumatic carditis of the 1st degree of activity. The combined mitral defect with the predominance of the stenosis of the IVth stage, heart failure II, IIId functional class NYHA»

According to standard schemas the plan of additional Survey (laboratory and instrumental) includes:

Additional methods of research:

1) clinical blood test;
2) clinical urine test;
3) biochemical blood test;
4) a coagulogram;

5) immunologic blood tests;
6) an electrocardiogram;
7) a cardiophonography;
8) X-ray Survey;
9) an echocardiography;
10) catheterization of heart and angiocardiography.

At the latent flow of rheumatic disease the laboratory data does not change substantially. Changes of immunologic indexes in this case are more characteristic: the level of immunoglobulin, quantity of B-and T lymphocytes, reactions of the leucocytes' migration inhibition, etc.

For the expressed rheumatic carditis the following laboratory data is characteristic:

In clinical blood test: augmentation of ESR, leukocytosis, deviation of the leukogram to the left; in the biochemical blood test: rising of the level of a_2 and γ-GLOBULINS, of seromucoid, haptoglobin, fibrin, aspartic transaminase; in the clinical urine test: normal or small proteinuria, microhematuria; in the immunologic blood tests: the quantity of T-lymphocytes is reduced, the function of T-suppressors is reduced, the level of immunoglobulins and titres of antistreptococcal antibodies is raised.

On *an electrocardiogram* while the defect is progressing the following features appear: a) the attributes of the left auricle hypertrophy (P mitrale), b) hypertrophies of the right ventricle in the form of the enlarged amplitude of beaks of complex QRS in corresponding derivation in combination with the changed ending part of ventricular complex (applanation, inversion of the beak T, depression of segment ST) in the same derivations, disturbances of the heart rhythm are often recorded (fibrillation, atrial flutter).

On *phonocardiogram* at the mitral stenosis the change of intensity of the 1st tone is defined, the appearance of the additional tone (flick of the opening of the mitral valve), and also the appearance of murmur in diastole is discovered. Duration of an interval from the beginning of the 2d tone up to the flick of the mitral valve opening (the IId tone – QS) varies from 0,08 up to 0,12 s and it shortens up to 0,04 – 0,06 s at the progress of the stenosis. Various diastolic murmurs (pre-, meso- and protodiastolic) are recorded. The importance of the cardiophonography increases in conditions of tachysystolic forms of fibrillation of auricles when at the usual auscultation it is difficult to relate the auscultated murmur to this or that phase of the cardiac cycle.

The X-ray Survey ascertains the change of the size of heart due to augmentation of the left auricle and the right ventricle. All four arches on the left contour are differentiated clearly; however, the waist of heart peters out due to the protrusion of the second (the pulmonary artery) and the third (the left auricle) arches on the left contour, and also the hypertrophy and a dilatation of the right ventricle peter. Such pattern is characteristic for the mitral configuration of the cardiac silhouette. Absence of the characteristic for a mitral stenosis protrusion of the third arch of the left contour of the cardiac shade at an X-ray Survey allows assuming the presence of a thrombus in the left auricle. In lungs the congestive changes are defined. The clearest radiological attributes of augmentation of the left auricle are defined in the

first slanting projection with the contrasted esophagus. Thus the esophagus is deflected to the right and back. It is considered that the deflection of the contrasted esophagus on an arch of small radius (up to 6 sm) is characteristic for the mitral stenosis, and on an arch of the big radius (over 6-7 sm) – for mitral defect. In a lateral projection the augmentation of the right ventricle and protrusion of the pulmonary trunk are found.

The echocardiography can be a verifying method at a mitral stenosis at which the following changes are observed:

- The unidirectional (P-shaped) movement of the front and back cusps of the mitral valve forward (the back cusp during the diastole is displaced to the back normally;
- Depression of the speed of the early diastolic shelter of the front cusp of the mitral valve (up to 1 sm/sec);
- Decrease of the amplitude of the opening of the cusp of the mitral valve (up to 8 mm and less);
- Augmentation of the left atrial cavity (the anteroposterior size can be enlarged up to 70 mm);
- thickening of the valve (fibrosis and calcification).

Doppler echocardiography allows to measure the gradient of pressure on the mitral valve and to specify the area of the mitral ostium, to define the pulmonary pressure, the presence of subtend regurgitation of blood on the mitral, tricuspid valves and the valve of a pulmonary artery.

Differential diagnostics is conducted with the diseases, which are characterized by the similar changes from the direction of heart and development of pulmonary hypertensia at the little-changed heart size: a chronic pulmonary heart or a primary pulmonary hypertensia, a thyrotoxicosis, the secondary defect of an interatrial septum complicated by failure of the right ventricle, myxoma LV.

Substantiation and formulation of the clinical diagnosis at the patient (taking into account the classification of the disease, the presence of complications and subtend pathology).

«Rheumatic carditis of the 1st degree of activity. The combined mitral defect with predominance of stenosis of the IVth stage, calcification ++, HF II, the IIId functional class NYHA».

For the patients who have tolerated surgical correction of the acquired heart disease, in the diagnosis the name of the tolerated operation, its date, the diagnosis and a stage of defect in occasion of which the surgical intervention was conducted, the remote result of operation, its complication should find reflectance. For example: «the condition after a mitral commissurotomy in 1989 in occasion of a mitral stenosis of III stage. Moderate mitral restenosis. HF IIA, II class. NYHA», «a state after a prosthetic repair of the mitral valve ball-valve prosthesis МКЧ-25, in 1991, in occasion of the combined mitral defect with predominance of insufficiency of IV stage. The remote result of operation good. HF IIA, II class NYHA».

Treatment of the patient with mitral stenosis

A choice of medical tactics: conservative or operative treatment.

The 1st stage – surgical treatment is not indicated.

The 2d stage –to the patients a mitral commissurotomy is indicated.

The 3d stage –the failure of the right ventricle submits well to medicament therapy. The indications to the operation are absolute; moreover, the results are much better at the 2d and 3d stages.

The 4th stage is the last stage at which the conducting of the operation is still possible, but it prolongs life not for long. At the complicated forms of a mitral stenosis with calcification, and fibrosis of the valve and also of the undervalvular structures, an extensive clottage of the left auricle – the reconstitution of the valve by means of dissection of the adherent commissures (commissurotomy). At a coarse calcification and sharp deformation of the valve, and also after the preceded valvulotomy at its unsatisfactory result, the prosthetic repair of the valve is indicated.

The 5th stage – medicamental treatment is not effective. Operative treatment is not indicated.

Principles of pathogenetically proven conservative therapy.

There are no specific conservative methods of treatment of the mitral stenosis. Under indications the diuretic drugs, peripheric vasodilators, cardiac glycosides, anticoagulants are indicated.

The basic directions of treatment of patients with mitral stenosis in a stage of compensation:

- Improvement of the hemodynamics in a small circle of blood circulation,
- Augmentation of time of the diastole and building of conditions for filling of the left ventricle,
- Decrease of recurrence of blood to the right region of heart,
- improvement of microcirculatory and rheological blood qualities,
- Decrease of oxygen debt and retardation of development of chronic hypoxia.

The basic directions of treatment of patients with decompensate mitral stenosis complicated by fibrillation of auricles:

- Normalization of the heart rhythm,
- Optimization of frequency of cardiac contraction at patients with the constant form of fibrillation of auricles,
- Maintenance of the sufficient peripheric vasodilation and of the optimum level of arterial pressure,
- Correction of the homeostasis (electrolytic, proteinaceous, acid-base balance, etc.),
- The prevention of tromboembolic complications,
- Correction of the chronic hypoxia.

Methods of operative treatment and the indications to it.

Mitral stenosis is most successfully corrected by surgical methods. Thus the basic method of surgical correction of not complicated forms of a "pure" or predominating mitral stenosis is the mitral commissurotomy (valvulotomy). At the presence of the expressed fibrous changes of the cusps and subtend mitral insufficiency the question about valve preserving plastic operations or replacing of the valve by prosthesis is solved. Thus the prostheses of two kinds are used – mechanical among which barrier, petal and hinged (ball-valve of Edwards's type or disk of Byork-Shaley) and biological are defined by the principle of functioning.

Operation of a mitral commissurotomy is indicated to the patients with the expressed mitral stenosis at presence of the symptoms limiting physical activity and reducing working capacity. These operations are conducted both by "close" and "open" procedure in conditions of the artificial circulation.

Auditeurs of the American cardiologic college (1998) recommend to conduct a mitral balloon valvulotomy at the following categories of patients with the mitral stenosis:

1. Patients with II–IV functional class on NYHA, with the moderate or severe mitral stenosis (the area of the ostium not more than 1,5 sm^2) at which the state of the valve allows to conduct a transcutaneus mitral balloon valvulotomy (at the absence of thrombuses in the left auricle or presence of a mitral regurgitation).

2. Patients without characteristic clinical symptomatology with the moderate or severe mitral stenosis (the area of the ostium not more than 1,5 sm^2), a pulmonary hypertensia (systolic pressure in a pulmonary artery more than 50 mm hg in rest or 60 mm hg at an exercise) at which the state of the valve allows to conduct a transcutaneus mitral balloon valvulotomy (at absence of a thrombus in the left auricle or a mitral regurgitation).

3. Patients with II–IV functional class under NYHA, the moderate or severe mitral stenosis (the area of the ostium less than 1,5 sm^2), the calcinated mitral valve at which the high risk of a surgical intervention (at the absence of a thrombus in the left auricle or a mitral regurgitation) is marked.

Improvement of the state of patients after the mitral commissurotomy comes in 70- 89 % of cases. The inefficiency of the operation is usually caused by a late direction of patients to the surgeon, by the expressed changes of heart and other internals. Activity of rheumatic process is not a contraindication to the operation.

Rules of conducting the postoperative period, measures of prophylaxis, diagnostics and treatment of possible postoperative complications at patients with MS.

The operational trauma and the changes of the hemodynamics connected with the correction of the defect, result in deep pathophysiological alterations in the postoperative period. Postoperative complications develop acutely and progress quickly. Prophylaxis and treatment of these complications demand round-the-clock medical overseeing of the patients in the first days after the operation. Repeated careful surveys of the patients operated, hourly measurements of BP, pulse rate and respiration- are demanded, the strict count of diuresis and maintenance of the optimum level of water-electrolytic balance, and also the profound biochemical blood analysises and urine are demanded. It is necessary to carry out regular ECG-and the X-ray control. All this is carried out on a background of the medicamental therapy including cardiac glycosides, diuretics, antibiotics and the drugs that are improving function of the myocardium and liver, when indicated – hormonal drugs and anticoagulants.

Depending on specificity of possible complications resuscitation, early and late postoperative periods are defined.

The resuscitation period lasts within the first hours after the operation before final patients' recovering from anesthesia and resistant stabilization of the basic vital functions. Overseeing of patients is carried out at this time by the anaesthetist.

The early postoperative period (the first 4-5 days) is characterized by a strain of compensatory -adaptive forces of an organism. From complications of this pe-

riod it is necessary to define cardiac insufficiency, distresses of heart rhythm and bleeding. Patients at this time are in the special postoperative ward provided with all necessary for constant control over the state of health and carrying out of urgent resuscitation actions.

One of the most frequent postoperative complications is *the ciliary arrhythmia*. Appearance of a ciliary arrhythmia in the early postoperative period unfavorablely affects the state of the patient and promotes originating of the insufficiency of the right ventricle. Treatment of a ciliary arrhythmia is carried out by the drugs oppressing excitability and myocardial conduction (procainamide hydrochloride, quinidine, β-blockers). Special attention is given to reduction of electrolytic balance (correction of a hypokalemia), an acid-base balance. Electric-impulsive therapy is widely used for restoration of the sinus rhythm.

The mitral commissurotomy changes conditions of hemodynamics at once and sharply and considerably enlarges load by the weakened left ventricle. As a result within first hours or days of the postoperative period *the acute left ventricular failure* can originate that demands urgent, active therapy.

At a part of patients with a pulmonary hypertensia at the expressed "second barrier ", and also at nonradical failure correction operation does not result in significant decrease of load on the right ventricle. In these cases (more often at the IV stage of defect) in the postoperative period the failure of the right ventricle can increase that demands persevering therapy by cardiac glycosides, diuretics and the drugs improving function of myocardium.

In the postoperative period prophylaxis and treatment of various *pulmonary complications* (pneumonias, an atelectasis) is very important as deterioration of conditions of gas exchange result in these conditions in the extremely dangerous hypoxia, worsening the function of myocardium. The serious clinical pattern of pulmonary-cardiac failure educes. The Great value in prophylaxis of these complications is attached to physiotherapy exercises, in particular respiratory exercises with airing balls, rubber toys.

The late postoperative period comes with 5–6-th day and lasts up to the extract of the patient from hospital. It is characterized by adaptation to physical loads. The basic complications of this period – *failure of the right ventricle and an exacerbation of rheumatic disease*.

Treatment *of purulent complications* arising in the postoperative period puts a problem of a choice of effective antibiotics to which sensitivity of the inducer is still preserved.

The exacerbation of rheumatic process usually arises on the 2–3-d week after the operation. Frequency of postoperative exacerbations depends not only on activity of rheumatic disease before operation, but also on the effectivity of the surgical intervention itself. According to our data, at effective operations frequency of the exacerbation of rheumatic process in the postoperative period is 6 times less often, than after ineffective interventions. Treatment of these complications demands active medicamental therapy with application of not only antibacterial, but also corticosteroid drugs if necessary.

At uncomplicated disease of patients they are allowed to rise from bed starting with the 2–3-rd day and are discharged from hospital 2 weeks after the operation.

It is desirable to finish the postoperative treatment in specialized cardiologic sanatorium where patients are referred immediately from clinic of cardiac surgery.

It is necessary to note, that the results of operation worsen within some time, which is connected with originating of specific and nonspecific complications at a part of patients with the acquired heart diseases. The first are observed at patients with prostheses of valves of the heart, the second are common for all patients with AHD, who tolerated cardiac surgery.

Among specific complications of the remote postoperative period thromboembolisms are primarily important, the cause of which is forming of thromboses in the region of a foreign body (prosthesis of the valve) with their subsequent break off and drift with blood flow into vessels of various organs. More often vessels of brain, much less often – of other organs (kidneys, the lower extremities, etc.) are affected. As a rule, thromboembolisms arise for the first time within 3 years after the operation. The excessive inclination to them is marked at exacerbations of rheumatic process, of the left atriomegalia.

Prophylaxis of a thromboembolism is provided by anticoagulant therapy which promotes depression of its frequency in 5-6 times.

Therefore all the people having prostheses of valves of heart should undergo the therapy of anticoagulants of indirect action (Fenindionum) in individual doses, under the control of size of a prothrombin ratio which is desirable for sustaining within the limits of 40-50 %. According to the order of Minister of Health of Ukraine all patients with prostheses of valves of heart should receive anticoagulants free of charge, on the basis of the document about the tolerated operation.

At originating of tromboembolic episodes the patient should be hospitalized in a appropriate hospital – according to the localization of a lesion (neurologic, surgical, urological, etc.) where conservative or surgical treatment should be carried out.

The infective prosthetic endocarditis is referred to serious complications of the remote postoperative period. The only effective kind of treatment of this complication is urgent repeated operation with changing of the prosthesis of the valve. Considering gravity of complication, it is necessary to pay special attention to its prophylaxis.

Various disturbances of function of the prosthesis can also become a serious complication of the remote postoperative period. For example, paravalvular failure as a result of eruption of parts of the stitches fixing the prosthesis, because of a infective endocarditis or in aseptic conditions at changes of tissues of the fibrous ring. Its symptomatology corresponds to failure of the prosthetic valve. At expressed paravalvular failure the repeated operation is indicated, at moderate – careful observation, restrictions in a regimen, medicamental therapy of a heart failure.

The clottage of prosthesis of the valve is referred to other complications of this group that is caused by formation of thrombuses in the region of the prosthesis of the valve; it is connected with errors of the anticoagulant therapies, a infective endocarditis. All this result in a grave condition and demands urgent repeated surgical treatment.

Principles of prophylaxis of exacerbations of rheumatic process, and also renovating of a sinus rhythm at patients with a ciliary arrhythmia after a prosthetic repair of valves of heart are the same, as after the mitral commissurotomy.

Diagnostics, tactics and procedure of rendering of an emergency medical care of urgent states at patients with mitral stenosis.

To urgent states which can arise at a mitral stenosis, it is possible to refer: pulmonary edema, thromboembolism of the big circle of blood circulation.

For ***pulmonary edema*** are characteristic: dyspnea, the inspiratory dyspnea, strengthening in a prone position that forces patients to sit down; tachycardia, acrocyanosis, overhydratation of tissues, dry whistling rales, then bubbling rales in lungs, profuse foamy sputum, changes of the electrocardiogram (a hypertrophy or overload of the left auricle and a ventricle, block of the left crus of a His' bundle, etc.).

First aid

1. ***The general actions***:
- An oxygenotherapy;
- Heparin 10 000 units of activity intravenously streamly;
- At frequency of reduction of ventricles more than 150 beats per 1 min – the electric impulsive therapy and less than 50 beats per 1 min – electric cardiostimulation;
- At the profuse foaming – defoaming: inhalation of oxygen through 33 % solution of ethanol, in unusual cases – 2 ml 96 % of solution of ethanol is introduced into the trachea.

2. ***At normal arterial pressure***:
- To execute the 1st point;
- To set with the lower extremities pulled down;
- Nitroglycerin in pills (the aerosol is more favorable) 0,4-0.5 mg under the tongue repeatedly or once under the tongue and intravenously (up to 10 mg by intermittent introduction or drop-by-drop introduction in 100 ml of the isotonic solution enlarging the rate of injecting from 25 mkg/min up to the effect under the control over blood pressure);
- Furosemide (Lasix) 40-80 mg intravenously;
- Diazepam up to 10 mg or morphine 3 mg intravenously by intermittent introduction up to the effect or the general dose of 10 mg.

3. ***At arterial hypertension***:
- To execute the 1st point;
- To set with the lower extremities pulled down;
- Nitroglycerin in pills (the aerosol is better) 0,4-0,5 mg under the tongue once;
- Furosemide (Lasix) 40-80 mg intravenously;
- Nitroglycerin intravenously (point 2), or sodium nitroprussicum 30 mg in 300 ml of the isotonic solution intravenously drop-by-drop enlarging the rate of introduction from 0,1 mkg / (kg/min) up to the effect under the control over arterial pressure, or Pentamine up to 50 mg intravenously by intermittent introduction or drop-by-drop or Clonidine of 0,1 mg intravenously streamly;
- Intravenously up to 10 mg of Diazepam or up to 10 mg of morphine (point 2).

4. ***At moderate (systolic pressure of 75-90 mm hg) hypotension***:
- To execute the 1st point;
- To lay the patient, having raised the headboard;
- Dobutaminum (Dobutrex) 250 mg in 250 ml an isotonic solution enlarging the rate of introduction from 5 mkg/(kg/min) up to the stabilization of arterial pressure at minimum possible level;

– Furosemide (Lasix) 40 mg intravenously after stabilization of arterial pressure.

5. ***At the expressed arterial hypotension***:

– To execute the 1st point;
– To lay, having raised the headboard;
– Dopamin 200 mg in 400 ml 5 % of solution of glucose intravenously drop-by-drop enlarging the rate of introduction from 5 mkg / (kg/min) up to the stabilization of arterial pressure at minimum possible level;
– If rising of arterial pressure is accompanied by intensifying of pulmonary edema - in addition Nitroglycerin intravenously drop-by-drop (point 2),
– Furosemide (Lasix) 40 mg intravenously after stabilization of the arterial pressure.

6. ***Monitoring of the vital functions (cardiomonitor, a pulseoxymeter)***.

7. ***To hospitalize after possible stabilization of the state***.

Massive pulmonary embolism is displayed by a sudden stopping of blood circulation (electromechanical dissociation) or shock with the expressed dyspnea, tachycardia, paleness or sharp cyanosis of skin of the top half of the body, jugular venous distention, anginous-like pain, electrocardiographic manifestation of acute "cor pulmonale".

Non-massive pulmonary embolism is displayed by dyspnea, tachycardia, arterial hypotension, attributes of pulmonary infarction (pulmonary-pleural pain, tussis, at a part of patients – with sputum coloured by blood, a rise in temperature, crackling rales in lungs).

For diagnostics of pulmonary embolism except for the defect itself, it is important to consider the presence of such risk factors of development of a thromboembolism, as elderly age, long-term immobilization, recent surgical intervention, heart failure, ciliary arrhythmia, oncologic diseases, deep vein thrombosis.

The volume and the contents of first aid at pulmonary embolism are defined by acuteness and gravity of clinical course which depend on a degree of a lesion of pulmonary bloodstream. At very heavy massive pulmonary embolism thrombolytic therapy is indicated, and sometimes also embolectomy, at non- massive – the indication of straight anticoagulants (heparin or low molecular weight heparins). In all cases first aid should be given immediately as the majority of patients with massive pulmonary embolism die within the first hours after its development, and at patients with non-massive pulmonary embolism the danger of relapse of the thromboembolism is high.

At ***apparent death*** it is necessary to provide the immediate cardiopulmonary resuscitation. For reduction of adequate spontaneous respiration the intubation of the trachea and long-term pulmonary ventilation of 100 % oxygen is usually required.

At ***a shock*** for maintenance of arterial pressure it is necessary to carry out the infusion therapy (rheopolyglukin, 5 % solution of glucose, Haemodesum, etc.). If infusional therapy does not result in stabilization of hemodynamics, application of inotropic and vasoactive drugs is indicated (Dobutamine, dopamine, adrenaline), and at patients with massive pulmonary embolism – also thrombolytic drugs (streptokinase).

Dobutamine, Dopamine or adrenaline at a preserved arterial hypotension are indicated intravenously drop-by-drop, raising the rate of ijecting of drugs up to stabilization of arterial pressure.

Thrombolytic therapy is indicated at massive pulmonary embolism, running with the arterial hypotension, not corrected by means of infusion therapy. While deciding of the question on carrying out of thrombolytic therapy it is necessary to pay attention to such indirect attributes of massive pulmonary embolism, as serious anginous-like pain syndrome, the jugular venous distention, the expressed dyspnea and a tachycardia, accent of the II sound above thea pulmonary artery, attributes of acute "cor pulmonale" on the electrocardiogram.

The basic drug for carrying out of thrombolytic therapy is Streptokinasa. At the excessed risk of originating of allergic responses and in view of the initial arterial hypotension before the application of streptokinasa 30 mg of Prednisolone is injected intravenously streamly. Then 250 000 units of Streptokinasa (Streptasa, Awelysine), dissolved in 100 ml of the isotonic solution, is introduced intravenously within 20-30 min, then infusion of the drug is continued with the rate of 100000 units/h during 12 hours (in general 1500 000 units). In the subsequent 6-7 days hypodermic introductions of heparin are given.

Recently there were references about more active carrying out of thrombolytic therapy at pulmonary embolism, however the schema stated above is generally accepted.

At non-massive pulmonary embolism thrombolytic therapy is not indicated, for rendering the first aid heparin is used.

Heparin is the main drug for urgent treatment at the majority of patients with pulmonary embolism. Besides anticaegulant effect, the drug has anti-inflammatory, angiogenic and analgesic action.

At pulmonary embolism 10 000 units of heparin is introduced intravenously streamly, and then drop-by-drop with the rate of about 1000 units/h. For the first day 30 000-35 000 units of heparin is usually introduced. Treatment by heparin is continued during 6-7 days, 3-5 days prior to heparin withdrawal indirect anticoagulants are introduced (Syncumar), which should be taken not less than 3 months. Slight blood spitting (streaks of blood in sputum) is not a contraindication to anticoagulant therapy.

For treatment or prevention of pulmonary embolism low molecular weight heparins are successfully applied

Therapy by clexsan which at pulmonary embolism is prescribed 1 mg/kg twice a day is effective and safe.

At submassive pulmonary embolism the use of fraxiparin is less often accompanied by originating of hemorrhagic complications, than application of non-fractionated heparin. 3-5 days prior to the withdrawal of low molecular weight heparin indirect anticoagulants are prescribed (Syncumarum etc.).

For All patients with pulmonary embolism a long-term oxygenotherapy is indicated.

At the expressed pain syndrome a neuroleptanalgesia is carried out (Fentanyl and Droperidol are prescribed).

For depression of pulmonary hypertensia intravenous introduction of 10 ml of 2,4 % solution of aminophylline can be useful.

At thromboembolism of fine branches of the pulmonary artery and rather stable state of the patient the oxygenotherapy is carried out, nonfractioned or low molecu-

lar weight heparin is used. 3-5 days prior the withdrawal of heparin indirect anticoagulants are prescribed (Syncumar, etc.). For decrease of the pulmonary hypertensia the aminophylline is indicated.

Failure of the mitral valve

The clinical pattern depends on the stage of mitral failure.

I a stage – compensation. It is characterized by the minimum regurgitation of blood through the left atrioventricular foramen, disturbances of a hemodynamics are practically absent:

– Complaints are not present;
– The working capacity is not limited.

II stage – subcompensation. The reversed current of blood into the left auricle is enlarged. Disturbances of hemodynamics result in dilatations of the left auricle and a hypertrophy of the left ventricle. Compensation of disturbances of hemodynamics is effectively carried out by the left ventricle and is constantly conserved. Unlike mitral stenosis the pulmonary edema arises extremely rarely at patients. Physical activity of patients is limited slightly as the opportunity of augmentation of minute volume of blood is preserved. Clinical signs:

– A dyspnea at significant exercise load;
– Weakness and excessive fatiguability periodically arising ;
– Periodic pains in the heart region;
– The working capacity is reduced.

III stage –decompensation of the right ventricle. Comes at a significant regurgitation of blood into the left auricle. Overstretching of the left ventricle result in its dilatations. There is the decompensation of cardiac activity periodically arises, that can be eliminated by medicamental therapy. Clinical signs:

– A dyspnea at an exercise load;
– Originating of attacks of the cardiac asthma;
– Tussis is marked (it can be dry or with sputum);
– The blood spitting is possible;
– Patients are troubled by periodic palpitation;
– Pains in the heart region;
– Sense of weakness and excessive fatiguability;
– Peripheric edemas in the evenings;
– An intolerance of exercise loads;
– Attributes of the decompensation of the right ventricle.

IV stage – dystrophic. It is characterized by appearance of constant failure of the right ventricle. The working capacity is lost. Following attributes are characteristic:

– The expressed dyspnea;
– Attacks of the cardiac asthma which are accompanied by tussis with excretion of small amount of sputum;
– A blood spitting;
– Constant palpitation with development of the constant form of atrial fibrillation;
– boring, pressing pains in the heart region;
– Constant peripheric edemas;

– Sense of gravity in epigastric region and hypochondrium;
– Weakness and excessive fatiguability are sharply expressed.

V stage – terminal. The clinic signs correspond to the III d stage by N. D. Strazhesko, V. H. Vasilenko's classification. It is characterized by serious disturbances of blood circulation with irreciprocal degenerative changes of the internals (liver, kidneys) and an ascites.

Complaints from other organs and systems. The pain and sense of gravity in epigastric region and by the right hypochondrium caused by augmentation of the liver and a distention of its capsule is marked.

The anamnesis of the disease. Often enough failure of the mitral valve is combined with a stenosis of the mitral valve or with other defects. The basic etiological factors promoting organic mitral failure are:

1) rheumatic disease;
2) infective endocarditis;
3) congenital defect;
4) ischemic heart disease (chronic forms and an acute myocardial infarction);
5) trauma;
6) general diseases of connective tissue – a systemic lupus erythematosus, pseudorheumatism, systemic scleroderma, etc.;
7) degenerative lesions of the valve apparatus of an unknown etiology.

The anamnesis of life (including working conditions, professional harmfulnesses, etc.; at women the obstetric-gynecologic anamnesis).

Data of the objective Survey (characteristic features at the disease given):

1) The Evaluation of the general state of the patient with failure of the mitral valve (consciousness, the constitution, fatness).

2) The Congregating of the information on a external state of the patient with FMV (survey of the skin, subcutaneously futty stratum, palpation of lymph nodes, thyroid and mammas). At disease of the I, II stage the general state of the patient does not change. Acrocyanosis can be marked at the increase of development of stagnation in the small circle of blood circulation at later stages of disease.

3) Survey of the state of respiratory organs of patients with FMV (survey of the thorax and the upper respiratory tracts, palpation of the thorax, percussion and auscultation of lungs).

4) Survey of a state of organs of the abdominal cavity of patients with FMV (survey of the abdomen, palpation and sucussion of the stomach, palpation of the intestine, liver, lien, pancreas, kidneys, organs of a small pelvis, auscultation of the abdomen).

5) Survey of state of the osteomuscular apparatus of patients with MVF (survey and palpation).

6) ***The local status*** (***Locus Morbi***). Survey and palpation of the heart region and superficial vessels, palpation of the main vessels of extremities and neck in projective points, definition of percussion borders of heart, auscultation of heart and vessels with obligatory definition of special "pathognomonic" signs of the prospective acquired defect by means of additional technical manual techniques, to describe the techniques of their carrying out.

Arterial pulse is usually not changed or attributes of the ciliary arrhythmia are marked. At a high pulmonary hypertensia there will be defined pulsation of the right ventricle in the third-fourth intercostals at the left edge of the breast bone and in epigastric region. At development of dilatation of the right ventricle and relative failure of the tricuspid valve the positive venous pulse due to the systolic wave "v" is defined.

At survey and palpation of the thorax, owing to a significant regurgitation, the cardiac murmur caused by the expressed hypertrophy of the left ventricle, is observed especially if the defect educed in childhood. The strengthened and diffuse apex beat localized in the fifth intercostals to the outside from middle-clavicular line, testifying about hypertrophy and the enhanced work of the left ventricle is defined. Above the apex of heart the systolic thrill is often palpated.

At auscultation of heart:

Systolic murmur can occupy a part of systole or all systole (*pansystolic murmur*) – serves as the brightest manifestation of mitral failure. Arises owing to transit of the wave of regurgitation from the left ventricle into the left auricle through rather narrow foramen between leaky closed cusps of the mitral valve. Murmur is well auscultated on an apex of heart, conducted to the left axillary region and along the left edge of the breast bone; its intensity varies over a wide range, and is usually caused by the degree of manifestation of valve defect. The timbre of murmur can be various – soft, blowing or rasping, that can be combined with palpatory defined systolic thrill on the apex. The louder and longer the systolic murmur is, the harder the mitral failure is.

Weakening or full absence of the I cardiac sound, that is caused by disturbance of the mechanism of closing of the mitral valve (absence of " period of the closed valves »), and also a wave of regurgitation;

The accent of the II sound above the pulmonary artery is usually expressed moderately and arises at development of stagnation in a small circle of blood circulation;

Splitting of the II sound above the pulmonary artery, is connected with the delay of the aortal component of sound as the period of ejection of the enlarged amount of blood from the left ventricle becomes longer. Often muffled III sound, is defined on the apex of the heart, arising because the enlarged amount of blood from the left auricle strengthens the fluctuations of walls of the ventricle.

Diagnosis MS is put on the basis of:

- Pansystolic murmur with the epicenter above the apex which is conducted to the axillary region and is accompanied by weakening of the I sound and III sound, and also intensifying and shift of the apex beat to the outside and down;
- Attributes of hypertrophy of the left auricle – P-mitrale and left ventricle on the electrocardiogram;
- Dilatations of these regions at X-ray and echocardiac researches;
- A mitral regurgitation at Doppler echocardiogram;
- In obscure cases the diagnosis proves to be true by means of the angiocardiography which allows estimating quantitatively the manifestation of the regurgitation.

On the basis of the received data of interrogation, the anamnesis of disease and clinical physical Survey of the patient to put ***the preliminary clinical diagnosis*** to the patient with FMV.

«A rheumatic carditis of the II degree of activity. The combined mitral defect with predominance of failure of the mitral valve of the III stage, HF II, III functional class NYHA»

Additional methods of research:

1) clinical blood test;
2) clinical urine test;
3) biochemical blood test;
4) a coagulogram;
5) immunologic blood tests;
6) electrocardiogram;
7) cardiophonography;
8) X-ray Survey;
9) echocardiography;
10) catheterization of heart and angiocardiography.

Clinical blood test, clinical urine test, a biochemical blood test, a coagulogram, immunologic blood tests — see mitral stenosis.

On ***the electrocardiogram***:

a) attributes of hypertrophy of the left auricle (P mitrale),

b) hypertrophies of the left ventricle in the form of the enlarged range of waves of complex QRS in appropriate abductions in combination with the changed final part of the ventricular complex (flattening, inversion T-wave, depression of ST- segment) in the same abductions,

c) at 30-35 % disturbances of heart rhythm are recorded (atrial fibrillation, atrial flutter);

d) at the expressed pulmonary hypertension the hypertrophy of the right auricle and right ventricle is marked.

Phonocardiogram is of great importance for diagnostics of failure of the mitral valve, as it allows to characterize systolic murmur in more detail. At record the amplitude of the I sound from the apex of heart is considerably reduced. Interval Q – the I sound is enlarged up to 0,07-0,08 sec. as a result of hypertension in the left auricle and some delay of the closing of cusps of the mitral valve. Systolic murmur is recorded right after the I sound and occupies all systole or its greater part. The amplitude of murmur is as more, as the more expressed the failure of the valve is.

At ***a X-ray Survey*** in anteroposterior projection the heart is enlarged, more to the left. The waist of heart is absent due to substantial growth of the left auricle which can reach huge dimensions and project out of the right contour of heart in the form of the additional shade.

Echocardiographic research does not tap specific attributes of disturbance of locomotion of cusps of the valve. Nevertheless, there are some attributes on which it is possible to judge failure of the mitral valve:

– A dilatation of the left departments of heart;
– Excessive excursion of an interventricular septum;
– multidirected locomotion of mitral cusps during the diastole;
– Absence of diastolic clamping of the cusps of the mitral valve;

– Attributes of the fibrosis (calcification) of the front cusp;

– Augmentation of the right ventricular cavity.

At ***Doppler-echocardiography*** the evaluation of the degree of manifestation of a mitral regurgitation is possible. A direct attribute of defect –turbulent systolic stream of blood in the cavity of the left auricle, correlating with the manifestation of regurgitation.

The catheterization of cardiac cavities allows to tap series of valuable diagnostic attributes. The pulmonary pressure is usually raised. On the curve of pulmonary-capillary pressure it is possible to note the characteristic pattern of failure of the mitral valve in the form of augmentation of a wave "v" more than 15 mm hg with fast and abrupt falling after it, that is an attribute of regurgitation of blood through the foramen of the mitral valve. At ***a ventriculography*** it is possible to observe, how contrast agent fills a cavity of the left auricle during the systole of a left ventricle. Intensity of contrasting of the last depends on the degree of failure of the mitral valve.

Differential diagnostics is a difficult problem, especially if it is carried out between pure rheumatic MF and ***the relative***, caused by dilatation of the ventricle at patients with cardiomegaly, and also ***congestive biventricular heart failure***. The absence of the rheumatic anamnesis and dominance in clinical pattern of attributes of lesion of a myocardium testifies in favour of relative mitral failure. Sonority and duration of pansystolic murmur at such patients can variate and decrease at retrogress of the left ventricular failure under influence of treatment whereas murmur of the organic MF thus becomes louder day by day. At relative failure of the valve on EchoKG instead of hyperdynamic reduction of walls LV its diffuse hypokinesia is marked.

Intensive pansystolic murmur above the apex of heart and at the left edge of breast bone in the region of its bottom is characteristic for ventricular septal defect ***and failure of the tricuspid valve***. Unlike MF systolic murmur at defect of the interventricular septum is the loudest in the third –fifth intercostals to the left and is well conducted to the region of the right half of thorax, the I sound is preserved.

Except for this the differential diagnosticsof the MF should be conducted with *failure of the tricuspid valve*, and also *stenosis of the ostium of aorta*.

A substantiation and formulation of the clinical diagnosis at the patient (in view of classification of disease, presence of complications and a concomitant pathology).

«A rheumatic carditis of the II degree of activity. The combined mitral defect with predominance of failure of the mitral valve of the III stage, HF II, III functional class NYHA».

Treatment of the patient with MVF

A choice of medical tactics: treatment of the MVF can be conservative or operative.

I stage – surgical treatment is not indicated.

II stage – operative treatment is not indicated.

III stage – operative treatment is indicated.

IV stage – surgical treatment is indicated.

V stage – operative treatment is not conducted.

Principles of pathogenetically proved conservative therapy.

Under indications antibiotics are prescribed for prophylaxis of relapses of rheumatic disease and infective endocarditis, diuretic drugs, peripheral vasodilator, cardiac glycosides, anticoagulants etc.

The basic directions of treatment of patients with mitral failure at ***a stage of compensation***:

– Restriction of inflow of blood into the small circle of blood circulation,
– Decrease of intensity and volume of exhaust of blood from the left ventricle (restriction of the mechanism of Franc – Starling),
– Enriching of the metabolism of myocardium,
– Correction of the microcirculatory and rheological property of blood and the prevention of development of chronic hypoxia,
– The decision on surgical treatment.

The basic directions of treatment of patients with mitral failure at ***a stage of a decompensation***:

– Normalization of heart rhythm,
– Optimization of frequency of cardiac reductions at patients with constant form of fibrillation of auricles,
– Prescription of the peripheral vasodilator in combination with diuretic drugs, the control and correction of electrolytic, proteinaceous, acid-base balance,
– Correction of microcirculation and rheological properties of blood,
– Optimization of metabolism of myocardium, elimination of chronic hypoxia,
– Prophylaxis of tromboembolic complications,
– Indication to the surgical treatment.

Existing methods of operative treatment and the indications to it.

Surgical correction is a method of choice at the expressed mitral regurgitation. With a view of confirmation of the diagnosis, and also for the decision on the operation the catheterization of heart and angiocardiography are conducted.

Indications to reconstructive plastic saving valve operations (a plasty of the mitral valve with a resection of a region of cusp and application of Carpentier's ring) are defects without rasping changes of cusps, chordas, papillary muscles and at absence of calcification of valves. In other cases including valve infective endocarditis, postinfarction defects, calcification of the II-III degrees, operation of a choice is changing of the valve by a mechanical or biological prosthesis (Fig. 13).

The prosthetic repair of the mitral valve to the majority of patients provides prolongation of life, and also reduction of working capacity in the postoperative period.

Complications of the early postoperative period and complications of the remote postoperative period are distinguished

Complications of the early postoperative period:

1) *The Bleeding.* The causes of postoperative bleeding are: free heparin in blood, thrombocytopenia (less than $5\times\times0^{10}$/L), low Ht (less than 30 %), fibrinolysis, defect of surgical techniques, excessive introduction of the matters intensifying bent to hemorrhage. Tactics at a bleeding includes: an evaluation of size and rate of bleeding, an evaluation of coagulability of blood and restore of deficiency of its components, carrying out of the reoperation.

2) *The Syndrome of low minute volume and arterial hypotension (shock).* Treatment is carried out in the following sequence: correction of a residual pathology or the pathology which have arisen after operation demanding reoperation, stopping of the bleeding and restore of the hemorrhage, liquidation of tamponade by means of reoperation, correction of the acidosis and electrolytic imbalance, elimination of arrhythmias, elimination of a hypovolemia.

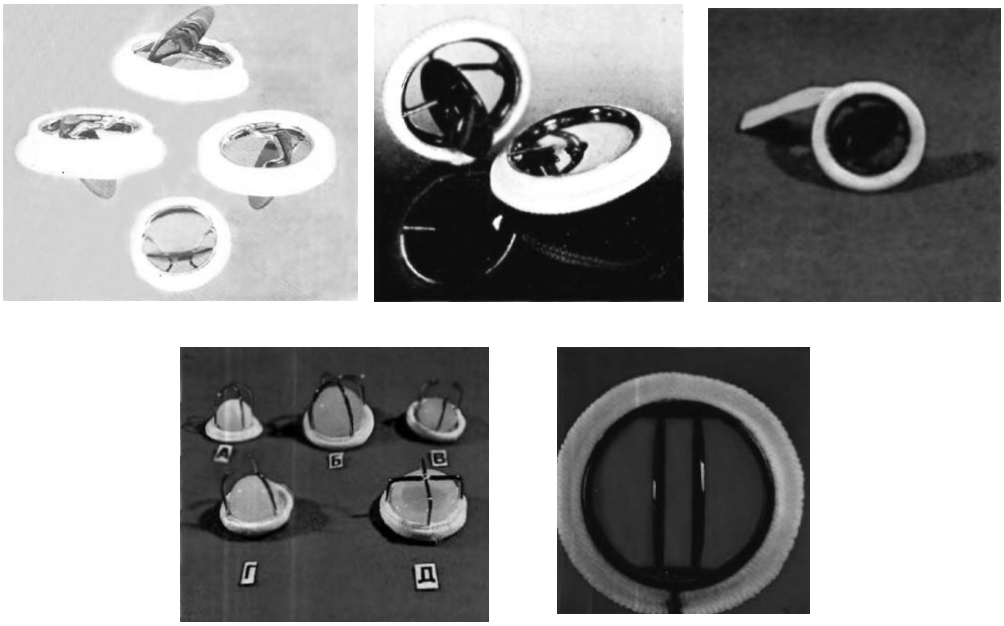

Fig. 13. Prosthetic heart valves

3) *Arrhythmias*: ventricular extrasystoly, an atrial tachycardia, a nodal (atrioventricular) rhythm, a ciliary arrhythmia, an atrial flutter, a ventricular tachycardia, transversal (atrioventricular) block, fibrillation of ventricles, cardiac arrest.

4) *Acute hemolysis.*

5) *Disturbance of diuresis.*

6) *Disturbances of acid-base balance*

7) *The Electrolyt imbalance.*

8) *Pulmonary complications:* traumatic larynacquiredracheitis, an atelectasis, a bronchopneumonia, tracheobronchiolitis, "a shock lung", a pleuropneumonia, chylothorax, a hydrothorax, a hemothorax, valvular pneumothorax, a hypodermic air emphysema, pleural empyema, an abscess of a lung.

9) *Hepatonephric failure.*

10) *Complications from the central nervous system*: hypoxic brain edema, air, trombotic or calcium embolism, cerebral hemorrhage or arachnoid membrane hemorrhage

11) *Necrosises of myocardium*: subendocardial necrosises of myocardium, focal myocardial infarction.

12) *Congestive failure of a myocardium*: residual shunt of blood, noncorrected regurgitation, a residual pressure gradient, paravalvular fistula, particulate clottage of a valvular prosthesis etc.

13) *Mental diseases.*

14) *Purulently – septic complications*: early septic endocarditis, septic shock, sepsis, a mediastinitis, a pericarditis, pleural empyema, abscess of a lung, a bronchopneumonia, tracheobronchiolitis, an osteomyelitis, suture sinus, thrombophlebitis, decubituses, wound dehiscence with secondary infection.

Complications of the remote postoperative period:

1) The Arterial thromboembolism: in basin of cerebral vessels, into coronary vessels, into the peripheral artery, into the renal artery.

2) Late septic endocarditis.

3) Paravalvular fistula.

4) Prosthetic valve dysfunction.

5) Recanalization of septal defect.

6) False aneurysm.

7) Distresses of rhythm: ciliary arrhythmia, extrasystole, bradycardia, paroxysmal tachycardia.

8) The circulatory insufficiency.

9) Neuropsychic distresses.

Aortal stenosis

The clinical pattern depends on athe stage of stenosis.

I stage — full compensation. Disturbances of hemodynamics are practically absent, defect is defined only at auscultation:

– Complaints are not present;

– The working capacity is not limited.

II stage — the latent heart failure. Clinical signs:

– A dyspnea at a physical load;

– Giddiness;

– Weakness and excessive fatigability arises periodically;

– The working capacity is reduced.

III stage - relative coronary failure. Clinical signs:

– A progressing dyspnea;

– Pains of stenocardiac character;

– Giddinesses and syncopes after physical loads;

– An intolerance of physical loads.

IV stage — the expressed left ventricular failure. The working capacity is lost. Following attributes are characteristic:

– Periodically there are attacks paroxysmal dyspneas at night;

– Attacks of cardiac asthma;

– Constant giddiness, syncopes;

– There are attributes of a pulmonary edema;

– Weakness and excessive fatigability are sharply expressed;

– A pain in right hypochondrium, due to the enlarged liver.

V stage — terminal. It is characterized by progressing failure of the left and right ventricles. All subjective and objective attributes of defect are sharply expressed. State of the patient is extremely grave.

Complaints from other organs and systems. The pain and sense of gravity in epigastric region and by the right hypochondrium caused by augmentation of liver and distention of its capsule is marked.

The anamnesis of disease. Quite often the AS is combined with failure of the aortal valve. Etiological factors are:

1) congenital pathology;

2) rheumatism;

3) calcification of cusps of the aortal valve of an unknown etiology.

The Anamnesis of life (including working conditions, professional harmfulnesses, etc.; at women the obstetric-gynecologic anamnesis).

Data of objective Survey (prominent features at the given disease):

1) The Evaluation of the general state of the patient with failure of the mitral valve (consciousness, the constitution, fatness).

2) The Congregating of the information on the external state of the patient with the AS (survey of the skin, subcutaneous fat stratum, palpation of lymph nodes, thyroid and mammas). At the I, II stage of the disease the general state of the patient does not change. At later stages of disease the expressed paleness of the skin, acrocyanosis is marked.

3) Survey of state of respiratory organs of patients with the AS (survey of thorax and the upper respiratory tracts, palpation of thorax, percussion and auscultation of lungs).

4) Survey of state of organs of the abdominal cavity of patients with the AS (survey of the abdomen, palpation and sucussion of the stomach, palpation of the intestine, liver, lien, pancreas, kidneys, organs of a small pelvis, auscultation of the abdomen).

5) Survey of a state of the osteomuscular apparatus of patients with the AS (survey and palpation).

6) ***The local status*** **(*Locus morbi*)**. Survey and palpation of the heart region and superficial vessels, palpation of the main vessels of extremities and neck in projective points, definition of percussion borders of heart, auscultation of heart and vessels with obligatory definition by means of additional manual techniques of special "pathognomonic" signs of the provisional acquired defect to describe technics of their carrying out.

Arterial pulse is slow, small and plateau (pulsus tardus, parvus et longus). Susceptibility to bradycardia promoting enriching of diastolic filling of the left ventricle and arterial hypotension is marked.

At survey and palpation of the thorax the slowly rising, high, resistant apex beat which is gradually displaced to the left and from top to bottom is defined. In position of the patient laying on the left side the jerk of the left auricle can precede it at its strengthened reduction and expulsion of blood into a tenacious left ventricle. In the region of the base of heart, jugular incisure and above carotid arteries the systolic thrill ("purring thrill") with an epicenter in the second intercostals on the right at the edge of the breast bone is defined. It is best defined in position of the patient sitting bending the body to the front breath- holding at a breath. At development of the left ventricular failure with depression of a stroke output of heart the thrill sharply weakens.

Auscultation of heart has great diagnostic importance:

Systolic murmur is rough and has the epicenter in the second intercostals on the right at the edge of the breast bone. It is well conducted through the blood flow to the region of jugular incisure and onto the carotid arteries, and also to the apex of heart. The murmur differs in characteristic scratching shade and it is best heard in position of the patient sitting with an inclination of the body to the front. At development of the heart failure with decrease of the stroke output and blood flow through the valve it becomes quiet and short.

Click of opening of the aortal valve. It is especially characteristic in childhood and youth at a congenital stenosis of the ostium of aorta, and at development of the calcification and low movement of the valvular cusps it peters.

Paradoxical splitting of II sound is defined at sharply expressed stenosis.

IV sound is connected with a hypertrophy of the left ventricle and rising of end-diastolic pressure.

7) The Diagnosis of the stenosis of ostium of aorta is put on the basis of:

- Systolic murmur with the epicentre in the second intercostals on the right which is conducted into the vessels of the neck and it is accompanied by thrill;
- The expressed hypertrophy of the left ventricle on the electrocardiogram;
- Retardations of rise and splitting of the ascending part of the curve of arterial pulse;
- Calcification of the aortal valve at X-ray Survey (at adults);
- Fibrosis and calcification of the aortal valve and expressed hypertrophy of the left ventricle according to the data of echocardiography. It proves to be true by means of Doppler echocardiografy (pressure gradient on the aortal valve) and at catheterization of heart.

8) On the basis of the received data of interrogation, the anamnesis of disease and clinical physical Survey of the patient to put the provisional clinical diagnosis to the patient with the AS: «Rheumatic carditis of II degree of activity, associated mitral-aortal defect of IV stage (aortal stenosis and mitral failure), HF IIA, III class NYHA».

Additional methods of research:

1) clinical blood test;
2) clinical urine test;
3) biochemical blood test;
4) coagulogram;
5) immunologic blood analysises;
6) electrocardiogram;
7) cardiophonography;
8) X-ray Survey;
9) echocardiography;
10) catheterization of heart and an angiocardiography.

Clinical blood test, clinical urine test, biochemical blood test, coagulogram, immunologic blood analysises see a mitral stenosis.

Changes on the *electrocardiogram* depend on the degree of changes in the muscle of the left ventricle. At early stages of development of defect the electrocardiogram data can be without changes. While progressing of the stenosis attributes of hypertrophy of the left ventricle in the form of the enlarged amplitude of waves of complex QRS in conforming abductions, more often in combination with the variated end part of the ventricular complex are defined. In late stages of defect on the electrocardiogram the changes specifying the overload of myocardium of the left auricle, — high split waves P I and P II and low waves P III can be marked. Disturbances of atrioventricular conduction of various degrees are quite often recorded: from elongation of interval P-Q up to full AV-blockade.

Cardiophonography. Systolic murmur has the characteristic rhomboidal or fusiform.

The X-ray Survey at the aortal stenosis has great value: at early stages the moderate enlargement to the left and elongation of the arch of the left ventricle with curving apexes occurs. At long flow of defect and expressed narrowing of the aortic ostium of heart has a typical aortal configuration. At development of relative failure of the mitral valve, that is at «mitralisation» of defect the enlargement of dimensions of the left auricle and appearance of radiological attributes of stagnation in the small circle of blood circulation is marked.

Echocardiographic research allows to discover the following:

- Incomplete systolic disclosing of the cusps of the aortal valve,
- Fibrosis and calcification of cusps,
- Presence of the systolic gradient on the aortal valve,
- Enlargement of thickness of myocardium of the left ventricle (up to 15 mm and more),
- Augmentation of the anteroposterior size of the left ventricle (systolic - more than 40 mm, diastolic - more than 60 mm).

The catheterization of heart and angiocardiography are indicated to all patients at whom according to clinical and non-invasive Survey the expressed stenosis of the ostium of aorta is diagnosed, for the final decision on the question on expediency of surgical treatment. Thus coronarography for the evaluation of permeability of coronary arteries is often conducted. The following is defined:

1) the gradient of the systolic pressure between the left ventricle and aorta which specifies data of the Doppler echocardiografy and it is a unique reliable method of the evaluation of the expression of stenosis;

2) functional state of the left ventricle;

3) exact localization of the obstacle to expulsion of blood, diameter of the ascending part of aorta, presence of concomitant defects and their expression.

Differential diagnostics. The symptomatology of aortal stenosis is similar to the clinical pattern of some cardiovascular diseases. It is necessary to differentiate systolic murmur *at aortal stenosis* and murmur *at congenital defects* (coarctation of aorta, stenosis of the ostium of the pulmonary artery), *an obstructive hypertrophic cardiomyopathy* and not rheumatic mitral failure (rupture of the tendinous chorda), and also the murmur arising at aortic dilatation at an idiopathic hypertension, atherosclerosis of aorta, a syphilitic aortitis. In similar cases short duration of murmur, absence of the thrill, characteristic for stenosis changes of the II sound, pulse and attributes of calcification of the valve testifies against the organic stenosis of the ostium of aorta. Quite often systolic murmur is observed at a hyperkinetic syndrome owing to the acceleration of blood flow. Careful auscultation in the region of the neck where it is louder and can peter at pressing of the subclavial artery to the first a rib in a supraclavicular fossa allows to discover its cause.

Treatment of the patient with a stenosis of the ostium of aorta

A choice of medical tactics: treatment of patients with the AS can be conservative or operative.

I stage — surgical treatment is not indicated.

II stage — at echocardiographic research the moderate gradient of the systolic pressure on the aortal valve (up to 50 mm hg) is defined. Operative treatment is indicated.

III stage — at echocardiography a gradient of the systolic pressure above 50 mm hg is defined. Operative treatment Is indicated.

IV stage — Surgical treatment in most cases is not possible, the question is solved individually.

V stage — state of the patient the is extremely serious, treatment is not effective. Operative treatment is not conducted.

Principles of pathogenetically proved conservative therapy

All patients accept ***prophylaxis of a infective endocarditis***. Therapy is symptomatic.

The basic directions of treatment of patients with the aortal stenosis at the stage of compensation:

- The control of pressure over the big circle of blood circulation and decrease of the pressure gradient between the left ventricle and aorta;
- Under indications - prescription of cardiotonic drugs;
- Correction of metabolism of the myocardium;
- The settle the question on surgical treatment.

The basic directions of treatment of patients with decompensated aortal stenosis:

- The control of pressure over the big circle of blood circulation and decrease of the afterload;
- Correction of metabolism of the myocardium;
- Optimization of the coronary circulation;
- Under indications - prescription of cardiotonic drugs;
- Correction of electrolytic disturbances;
- Normalization of the heart rhythm;
- Optimization of frequency of cardiac reductions at patients with the constant form of fibrillation of auricles;
- The prevention of tromboembolic complications;
- A direction to the surgical treatment.

Existing methods of operative treatment and the indication to it.

As well as at other defects, the unique effective method of treatment is surgical which includes:

1) operative valvulotomy;

2) transcutaneus balloon aortal valvuloplasty;

3) prosthetic repair of the aortal valve.

Choosing the surgical treatment of the aortal stenosis the degree of stenosis, a clinical pattern, a functional class is considered. At III and IV classes the operation is indicated absolutely. Presence even one of the triad of attributes (syncopes, cardiac asthma, anginous pains) serves as the direct indication to operative treatment. The cardiomegaly, the expressed electrocardiograms-attributes of the hypertrophy of the left ventricle, systolic aortal gradient more than 50 mm hg even at insignificant complaints and not expressed clinical pattern are indications to operation. Surgical treatment consists in a prosthetic repair of the aortal valve in conditions of cardiopulmonary bypass.

At patients with the AS and a congestive heart failure the risk of operation is extremely high, therefore they are considered inoperable. It is known, that at a total heart failure duration of their life makes 7-8 months At of some these patients as the unique variant of an intervention at the given stage it is possible to execute cath-

eter balloon valvuloplasty for decompression of the left ventricle. The procedure of "fractional" dilating of the aortal ostium with the use of catheters of «Meadox» or «Schnieder medintug», and also the procedure of single-stage dilating by balloon catheters in diameter of 23-28 mm. In cases of successful valvuloplasty the area of the aortal ostium is enlarged, the pressure gradient and the final diastolic pressure in the left ventricle lowers, the fraction of exhaust is enlarged. The main advantage of balloon valvuloplasty is that at some patients there is an expressed clinical enriching and the degree of heart failure that allows to execute the operation of the prosthetic repair of the valve lowers.

To urgent states which can arise at aortal stenosis, it is possible to refer: *myocardial infarction, thromboembolism of the big circle of blood circulation.*

For the acute myocardial infarction the pain which, as a rule, arises suddenly, more often behind the breast bone or to the left of the breast bone is characteristic. The pain is described by patients as compressing, pressing, tearing, etc. It irradiates into the left scapula, the left brachium, and arm. Unlike the attack of stenocardia: at the myocardial infarction the pain can spread to the right from the breast bone, epigastric region irradiates into both scapulas.

Intensity of pain at a myocardial infarction considerably exceeds those at a uncomplicated stenocardia. Duration of pain attack at a myocardial infarction is estimated in tens of minutes, hours, and sometimes days. During the attack there can be periods of some weakening of pain, however it does not peter completely and soon recommence with the former strengh.

In many cases the pains are so excruciating, that the patient has a fear of death

Repeated taking of Nitroglycerinum at a myocardial infarction does not take the pain out.

In the acute period of myocardial infarction the weakening of the 1 cardiac sound, quite often presystolic gallop rhythm, and sometimes arrhythmia is discovered. Appearance of more or less local pericardial friction (Kernig's symptom) is the important diagnostic attribute. Pulse, as a rule, becomes frequent up to 100-120 per minute, sometimes, on the contrary, is retarded. Arterial pressure, especially systolic, in overwhelming majority of cases falls, sometimes up to a critical level (a cardiogenic shock) though in some cases it initially, on the contrary, raises. Changes of the electrocardiogram occur and then undergo certain dynamics, that allow to solve questions of localization, extensiveness and revertive development of focal changes in cardiac muscle.

According to modern beliefs, at focal damage of a myocardium there first occurs a change of the initial part of ventricular complex QRS. So, at a subendocardial necrosis the wave Q enlarges and the wave R decreases; at the transmural myocardial infarction there is only the wave Q reversed downwards; at subepicardial – the wave Q is not expressed, the wave R is reduced, and at the intramural - the initial part of the ventricular complex remains normal. Changes of the terminal part of the ventricular complex (interval S-T and the wave T) are defined basically by the ischemia of the myocardium, the disturbances connected with it and morphological damages of the cardiac muscle.

First aid at the acute myocardial infarction is reduced basically to the following:

1. Full rest.

2. Relief of the pain syndrome.

At systolic BP not below 90 mm hg the patient is offered a pill of nitroglycerin (0,5 mg) if he did not do it yet, then the second one.

In many cases first aid at the myocardial infarction begins with intravenous introduction of an mixture of Analginum (from 2 up to 5 ml 50 % of solution) and diphenhydramine hydrochloride (1 ml 1 % of solution) in the isotonic solution of Sodium chloride (in one syringe), or Analginum (the same dose) and 1-2 ml 0,5 % of solution of Seduxen (in a separate syringe, slowly, in 20 ml 40 % of solution of glucose).

Now the most active method of anesthesia at a myocardial infarction is the neuroleptanalgesia (NLA) — a combination of an analgetic Fentanyl and a neuroleptic Droperidol. Doses of the drugs should be differentiated. The dose of Fentanylum 1 ml 0,005 % of solution (0,05 mg) is recommended for the sick people who weighs less than 50 kg, older than 60 years or with concomitant diseases of lungs at a stage of pulmonary failure. For the others the pristine dose makes 2 ml.

The dose of Droperidol depends on the emotional state and the initial BP: at a systolic BP up to 100 mm hg — 1 ml 0,25 % of solution (2,5 mg); up to 120 mm hg — 2 ml (5 mg); up to 160 mm hg — 3 ml (7,5 mg), above 160 mm hg - 4 ml (10 mg). It is recommended to apply NLA with predominant neurolepcy, that is in a volume ratio more than Droperidol, than Fentanyl, accordingly 2-3 ml and 1 ml; 3-4 ml and 2 ml. Drugs are dissolved in 10-20 ml of the isotonic solution of glucose or Sodium chloride and are introduced slowly, on the basis of 1 ml of Fentanyl during 2 minutes

At the significant respiratory depression produced by NLA, and also by morphine or Omnoponum, a specific antidote is introduced- Nalorfine (1 ml 0,5 % of solution from 15-20 ml of the isotonic solution of Sodium chloride intravenously).

It is falsely to begin anesthesia at a myocardial infarction with morphine and its analogues and only after their poor efficiency to go on to the neuroleptanalgesia as to an extreme drug: action of morphine and Fentanyl on the respiratory center is summarized and danger of respiratory distresses increases.

Morphine in a dose of 5-10 mg (0,5-1 ml 1 % of solution) can appear useful at a persistent pain syndrome at the young, physically strong men accustomed to alcohol, and also at the cardiac asthma, but without a hypertension.

Dipidolor in a dose of 7,5-15 mg (1-2 ml 0,75 % of solution) can be used at bad survivability of other morphine-like drugs if arterial pressure is not lowered.

Fortral in a dose of 30-60 mg (1-2 ml) can be applied 3 % of solution at a pain syndrome with hypotension; it is counter-indicated at the hypertension in the big and small circle of blood circulation.

In case of persistent, resistant to medicamental treatment of a pain syndrome they use a nitrous oxide narcosis in the ratio 80 % of nitrous oxide and 20 % of oxygen. 20 % solution is introduced from initial calculation of 50-70 mg/kg with speed 1-2 ml/min. Introduction of a drug is stopped at the onset of sleep.

3. Application of the drugs that warn originating or limit the infarction region, and prophylaxis of tromboembolic complications is necessary.

With this purpose application of anticoagulants of direct action is indicated: Fibrinolysinum 40–60 thousand units (either Streptokinase 500000-1000000 units) with heparin 10–15 thousand units it is intravenously drop-by-drop during 6 hours. Further heparin 5000–10000 units intramuscularly under the control of at least blood clotting time. To the end of course of treatment by heparin they add, and then go on without it with anticoagulants of indirect action (Syncumar, Phenilinum, etc.) under the control of a prothrombin index.

4. Faster introduction of kontrikalum 20-50 thousand units intravenously drop-by-drop can be rather useful.

5. Actions on prophylaxis and struggle against reactive syndromes of the myocardial infarction, especially with arrhythmia, heart failure and a cardiogenic shock.

Ventricular extrasystole (polytopic, polymorphic, etc.). Clinically the given state is displayed by filling of "strokes of heart»,by its freezing behavior, intermittence. If ventricular *extrasystole* starts being frequent character (5-6 and more extrasystoles per I minutes), or becomes «R on T» even of rare character, it is the indication to their immediate reduction, otherwise the development of fibrillation of ventricles with a fatal outcome is possible. A drug of choice for treatment of the given pathology is Lidocainum. Recommended doses of Lidocainum: 80-120 mg i.v., then after 30 minutes 80-100 mg more, after that, if necessary, each hour the dose can be repeated. After reducing arrhythmia 200 mg of this drug is introduced intramuscularly.

At absence of Lidocainum or at its inefficiency it is possible to use other medicines:

1. Procainamide hydrochloride. 10 % solution-10,0 desirably in combination with 0,5 ml 0,2 % of solution of noradrenaline, is introduced slowly, i.v., under the control of BP (taking 1 tab. 0,5 g 4 times per day).

2. Ethmosinum - 100 mg 4 times per day.

3. Aethacizinum - 50 mg 3 times per day.

4. Obsidanum - 5 mg is introduced i.v. streamly, under the control of a BP (tab. on 20 mg 4 times into day).

At absence of effect from medicamental treatment the electrical defibrillation is made. Indications to carrying out of a countershock are:

1. Appearance of attributes of the acute left ventricular failure (pulmonary edema).

2. Development of clinic of the arrhythmic shock.

3. Absence of effect from conservative treatment within 3 hours.

After conducting of the countershock the maintenance therapy Obsidanum (20 mg 2 times per day), or an Ethmosinum (100 mg 2 times per day), or Ethacizinum (50 mg 2 times per day) is prescribed.

First aid at fibrillation of ventricles and paroxysmal tachycardia

Fibrillation of ventricles is subdivided into primary and secondary. *Primary develops* suddenly at patients without or with the minimum attributes of the circulatory insufficiency and can arise as complication of frequent ventricular extrasystole, and without clinical harbingers. *The secondary fibrillation* is a terminal stage of a progressing acute left ventricular failure or decompensated forms of the chronic heart failure. Actually fibrillation of ventricles is a state of apparent death. The patient suddenly loses consciousness, pulse and BP are absent, the respiration is preagonal or it

stops completely. The unique way of saving the patient — early defibrillation. Transfer of the patient into the ALV, medicamental therapy, is of secondary importance.

The ventricular paroxysmal tachycardia is clinically displayed by a heart consciousness, sense of shortage of air, a dyspnea, and ischemic pains in the region of heart. At absence of necessary treatment attributes of an acute left ventricular failure quickly educe at patient.

First aid is carried out by use of Lidocainum, procainamide hydrochloride, Obsidanum. Doses of used drugs and indications to a countershock are similar to used at treatment ventricular extrasystole.

Paroxysmal supraventricular tachycardia is clinically displayed by the expressed palpitation, sense of shortage of air, sometimes boring pains in the region of heart.

First aid. Introduction of Lidocainum is counter-indicative because of an inefficiency and risk of an acceleration of ventricular contractions. Treatment same as at the ciliary arrhythmia, is begun with i.v. drop-by-drop introduction of a polarizing mixture and Riboxinum (200-400 mg). On a background of the polarizing mixture Isoptinum (5-10 mg) is introduced i.v. streamly; at absence of effect the given dose is recommended to be repeated in 30 minutes It is possible to try to use intravenous drop-by-drop introduction of cordarone, dissolved in the polarizing mixture at the rate of 5 mg/kg (on average, 300 mg). At absence of effect - countershock.

Paroxysmal ciliary arrhythmia is clinically displayed by attacks of palpitation, feelings of defects in its work, shortage of air. The patient, as a rule, can specify exact time of the beginning of the attack.

First aid. At tachysystolic form of paroxysmal ciliary arrhythmia it is necessary to conduct emergency restoration of rhythm, otherwise during short time at the patient attributes of an acute heart failure will educe.

Disturbances of conduction at the myocardial infarction. First aid at an atrioventricular heart block.

The most frequent variant of disturbance of conduction at an AMI is an appearance of atrioventricular heart blocks (A-V block) of various degrees. The given kind of pathology is mostly typical for back-diaphragmatic AMI, especially at its spread onto the right parts of heart.

First aid is required at full A-V block. Its presage can be represented by the appearance at the patient of A-V block of the I-II degree, also sudden development of the given terrible complication however is possible. Clinically full A-V block is displayed by giddiness, loss of consciousness (attack Morgagni-Adams-Stokes syndrome (MAS). Skin gets cyanotic shade, sharply expressed bradycardia is marked - the heart rate decreases up to 30 and less, on the electrocardiogram there are marked attributes of full A-V block which, in its turn, can provoke fibrillation of ventricles. The most effective method of treatment at attack of MAS is urgent transthoracic pacing, then transvenous and transesophageal and the least effective — epicutaneous. Widely used before introductions of atropine shoud be recognized inefficient, the insignificant positive clinical effect can be given by use of Isadrinum (tab. 5 mg under tongue) or alupent (1 ml of 0,05% solution i.m or i.v.).

The WPW-syndrome (W-P-W), or a syndrome of premature excitation of ventricles, is characterized by presence on the electrocardiogram of shortening of interval PQ up to 0,08-0,11 sec. Waves P of a normal form. Shortening of interval PQ

is accompanied by the widening of complex QRS more than 0,10 sec. The given complex reminds of block of bundle of His in form. On the background of syndrome W-P-W there can be various disturbances of rhythm: supraventricular tachycardias, paroxysms of fibrillation or an atrial flutter. On an electrocardiogram it is displayed by appearance of frequent (up to 200-300 contractions per 1 minute) and irregular heart rhythm. Because of presence of delta-waves the width and amplitude of complexes QRS is deformed. Clinically the given syndrome is displayed by appearance of weakness, giddiness, and unpleasant feelings in the region of heart.

First aid. Depending on the initial heart rate, two methods of treatment are used.

If the heart rate is not more than 200 per 1 minute, it is necessary to introduce the drugs blocking accessory paths: solution of Ajmalinum 2,5 % - 2,0 per 10-20 ml of physiologic saline , or 100-150 mg of disopyramide per 10-20 ml физраствора. The given drugs are introduced i.v., slowly.

If the heart rate is more than 200 per1 minute, carrying out of the urgent electrical defibrillation is indicated.

The note. At treatment of the given syndrome introduction of the drugs retarding transit of electrical impulses through conductive system of heart is contra-indicative: cardiac glycosides, beta-adrenergic blocking agents, calcium channel blocking agent.

Acute cardiovascular failure on the background of the AMI can be displayed as left-ventricular, right-ventricular and mixed (total) forms.

Acute left-ventricular heart failure cardiac asthma.

The given pathological syndrome in clinic of internal diseases traditionally refers to as a cardiac asthma. The most typical sign is appearance of the increasing dyspnea passing into suffocation. The pain syndrome for the given state is not characteristic. If the cardiac asthma has arisen on the background of postinfarction cardiosclerosis then the given variant of the attack can run in painless form, or pains in the region of heart will have "ischemic" character. Forced position typically occupied by patients: semisitting, with the lowered legs. Anxiety, excitetion are marked; skin is pale, acrocyanosis. Cardiac sounds, as a rule, are muffled and badly listened because of plenty of rales in lungs. Appearance of accent of the II sound above the pulmonary artery is possible. BP at initial stages of disease is raised (influence of sympathoadrenal reactions), loweres later. The tachycardia is marked, disturbances of rhythm of cardiac activity are possible. Breathing is complicated. In lungs rales are defined. Dynamics of their appearance begins with back basal parts of lungs, is symmetric from two sides. The quantity and character of rales depends on the intensity of heart failure. In the most serious cases there appears foam from the respiratory tracts sometimes having pink shade. First aid. All medical actions should be directed to depression of the preload on heart, rising contractive abilities of myocardium, "unloading" of the small circle of blood circulation and to include the following directions:

1. *Decrease of inflow of blood from periphery into the small circle* is achieved by using peripheral vasodilators (first of all - nitrates) or ganglionic blockers . With this purpose it is possible to use a simple method - to prescribe pills of nitroglycerin under tongue, 1 pill with an interval of 5-10 minutes, or to introduce nitroglycerin agents intravenously . At use of the ampule form of 1 % nitroglycerin solution ("Concentrate"), its contents, right before the application, it is dissolved in

the isotonic solution until reception of 0,01 % solution (0,1 mg = 100 mkg in 1 ml). The given solution is introduced i.v. drop-by-drop. Initial rate of introduction — 25 mkg per minute (1 ml of 0,01 % solution during 4 minutes). A rate of introducing is adjusted individually, achieving lowering of BP for 10-25 % from the initial (the systolic pressure should be not lower than 90 mm hg) . At insufficient effect the rate of administering is enlarged by 25 mkg per minute every 15-20 minutes. Usually the amount of nitroglycerin needed for achieving effect, does not exceed 100 mkg (1 ml 0,01 % solution per minute). On occassion doses can be higher (up to 2-4 ml in per minute).

2. An effective drug for treatment *of an alveolar pulmonary edema* is i.v. introduction of 1-2 ml 1 % solution of morphine. In the given situation other than analgesic properties of the given drug are used: inhibitory action on overexcited respiratory center lead to bradipnoe with its simultaneous recess, owing to vagotropic effect, as a result of it the respiratory minute volume is enlarged; Ill-defined ganglionic blocker action reduces the inflow of blood into the small circle of blood circulation.

3. *Rising of contractive abilities of myocardium.* Ssympathomimetic agents have clinically proved positive effect. A drug of choice is dopmine (Dofaminum, Dopamine).

4. *For decrease of volume of circulating fluid* intravenous introduction of diuretic drugs from the saluretic group, for example, Furosemidum, in a dose of 60-90 mg is indicated. Use osmodiuretics at the given pathology is counter-indicative, as they in the first phase of the action enlarge volume of circulating fluid in the bloodstream due to the attraction of water from interstitial space that finally can enhance the pulmonary edema.

5. *If the alveolar pulmonary edema* proceeds with the expressed bronchospasmodic component, that is specified by the expiratory dyspnea and rough breathing, slow introduction of aminophylline , in a dose 5-10 ml of 2,4 % solution is indicated i.v.

6. *For reduction of hypoxia and carrying out of defoaming in alveoles* from the moment of the beginning of treatment inhalation of oxygen in volume of 3-5 l/min passed through the defoamer is indicated. As a defoamer it is recommended to use Antifomsilanum; at its absence - ethanol. It is necessary to know, that ethanol possesses the most expressed defoaming effect at high concentrations (96 %), however in such concentration it can produce burn of the upper respiratory tracts, therefore ethanol is recommended to be dissolved into 70-80 %.

7. *The volume of infusional therapy should* be minimum (200 300 ml of 5 % glucose) and, basically, is directed to avoidance of repeated punctures of peripheric veins.

Acute right-ventricular heart failure most often educes at an AMI of the right ventricle, tear of the interventricular septum and at pulmonary embolism. On the first place at the given kind of pathology there are signs of overload of the small circle of blood circulation, dyspneas are clinically displayed, jugular venous distention , high CVP. If within 1-2 day the patient does not die, attributes of stagnation in the big circle join - the liver is enlarged in dimensions, becomes painful at palpation, pastose of feet and shins with the tendency of diffusion upwards.

Failure of the aortal valve

The clinical pattern depends on thea stage of stenosis.

I stage – compensation:

– Complaints are not present;
– The working capacity is not limited.

II stage – the latent heart failure. Clinical signs:

– Stenocardiac pain at a physical load;
– Unpleasant sensation of heart contractions in the form of strokes or pulsations on the head, especially in the position on the left side;
– Dyspnea at a physical load;
– Giddiness;
– Periodically arising sense of weakness and excessive fatiguability;- The working capacity is moderately reduced.

III stage – relative coronary failure. Clinical signs:

– The dyspnea strengthens in process of increase of the left ventricular failure – in rest and reminds of cardiac asthma;
– Attacks of anginous pain, especially at night, difficultly stopped by nitroglycerin;
– Sharp weakness and sweating;
– Giddinesses and syncopes especially arising at transferring into vertical position;
– Significant depression of physical activity is marked.

IV stage – decompensation. The working capacity is lost. Following attributes are characteristic:

– A dyspnea at night;
– Attacks of cardiac asthma;
– Frequent stenocardiac pains at night;
– Constant giddiness and syncopes;
– Peripheric edemas;
– There are attributes of pulmonary edema;
– Weakness and excessive fatiguability are sharply expressed.

V stage – terminal. It is characterized by progressing failure of the left and right ventricles, deep degenerative changes of the internals (trophic disturbances, ascites, augmentation of liver). State of the patient the extremely serious.

Complaints from other organs and systems. The pain and sense of gravity in epigastric region and right hypochondrium caused by augmentation of liver and distention of its capsule is marked.

The anamnesis of disease. Quite often AVF (failure of the aortal valve) is combined with the aortal stenosis. At assembly of the anamnesis it is necessary to consider factors which result in development of AVF:

1) rheumatic disease and rheumatic diseases, (ankylosing spondylitis, Reiter's syndrome);
2) systemic diseases of connective tissue;
3) congenital defect (the two-cuspidate aortal valve);
4) idiopathic myxedema degeneration;
5) infective endocarditis;

6) trauma;
7) lues;
8) atherosclerosis of the aorta;
9) a dissecting aortic aneurysm;
10) Morphan's syndrome;
11) aortitis (an aortic arch syndrome, a temporal arteritis); 11) nonspecific ulcerous colitis.

The anamnesis of life (including workingconditions, professional harmfulnesses, etc.; at women the obstetric-gynecologic anamnesis).

Data of objective Survey (prominent features at the given disease):

1) The Evaluation of the general state of the patient with failure of the aortal valve (consciousness, the constitution, fatness).

2) The Congregating of the information on the internal state of the patient with AVF (survey of skin, subcutaneous fatty stratum, palpation of the lymph nodes, thyroid and mammas). At disease of the I, II stage the general state of the patient does not change. At later stages of disease the expressed paleness of skin, acrocyanosis is marked.

3) Survey of the state of a respiratory organs of patients with AVF (survey of the thorax and the upper respiratory tracts, palpation of thorax, percussion and auscultation of lungs).

4) Survey of the state of organs of the abdominal cavity of patients with AVF (survey of abdomen, palpation and succussionof the stomach, palpation of the intestine, liver, lien, pancreas, kidneys, organs of the small pelvis, auscultation of the abdomen).

5) Survey of the state of the osteomuscular apparatus of patients with AVF (survey and palpation).

6) ***The local status*** **(*Locus morbi*)**. Survey and palpation of the heart region and superficial vessels, palpation of the main vessels of extremities and neck in the projective points, definition of percussion borders of heart, auscultation of heart and vessels with obligatory definition by means of additional manual techniques of special "pathognomonic" signs of the provisional acquired defect, to describe the technique of their carrying out.

For the expressed failure of the aortal valve the peripheric signs caused by great pressure drops in the bloodstream are characteristic:

- Paleness of the skin;
- A distinct carotid pulsation ("carotid shudder");
- A pulsation of temporal and brachial, subclavian and axillary arteries;
- Concussion of the head (Musset's sign) synchronous with a carotid pulsation;
- Pulsing narrowing of pupils (sign Landolfi);
- The enhanced pulsation of the aortic arch in the region of the jugular incisure and the abdominal part of aorta in the epigastric region;
- Capillary pulse;
- Rhythmical change of intensity of colouring of the uvula and tonsils (Muller's sign), alternating hyperemia and blanching of the skin at the base of the nail bed at pressing on its end (Quincke's symptom)

At survey high (big), fast and short pulse (Corrigan's pulse) is defined. Fluctuations of all left half of thorax, (cardiac thrust), enhanced and diffuse apex beat,

displaced to the left and down to the sixth –seventh intercostals are defined by palpation. Borders of heart are dilated to the left and downwards. Above large vessels double Trube sound is auscultated, at pressing by a stethoscope on the iliac artery in the region of inguinal [Poupart's] ligament there is a double murmur of Duroziez. Systolic arterial pressure is more often raised up to 160-180 mm hg, and diastolic – is sharply lowered – up to 50-30 mm hg At the expressed aortal failure it is characteristic the augmentation of pulse BP due to depression of diastolic (practically up to 0) and to a lesser degree – rising of systolic which can reach 200 mm hg and more. The Systolic pressure on the popliteal artery exceeds the brachial pressure in 80–100 mm Hg. (Hill's attribute).

Auscultation of heart:

The protodiastolic murmur "decrescendo" caused by a reversed stream of blood from an aorta into the left ventricle. Murmur begins at once behind the II sound, quite often muffling it, gradually weakens to the end of the diastole. At perforation of cusps murmur gets ringing, "musical" shade. With development of the heart failure attributes of defect become less expressed, intensity and duration of diastolic murmur decreases.

Other cardiac murmurs:

- midsystolic murmur of the ejection above the aorta with conducting onto the carotid arteries. It is caused by its relative stenosis at sharp augmentation of blood flow through the valve;
- Short midsystolic or presystolic murmur of Flint above the apex. It is formed at stroke of the reversed current of blood at the wall of the ventricle and a cusp of the mitral valve;
- Pansystolic murmur of relative mitral failure testifies about a so-called mitralisation of the aortal valve.

Change of cardiac sounds:

- Weakening of the II second sound above the aorta (A_2);
- Appearance of the III sound. IV sound is sometimes defined also;
- Changeable additional systolic sound of expulsion of blood into the aorta at its dilatation.

At auscultation above the femoral artery it is auscultated:

- Loud clapping systolic sound ("a sound of a pistol shot");
- Systolo-diastolic Duroziez murmur at a slight compression of the artery by a stethoscope.

The Diagnosis of aortal failure is put on the basis of:

- The expressed arterial pulsation and augmentation of pulse BP;
- Protodiastolic murmur along the left edge of the breast bone especially in combination with a sharp weakening of the II sound;
- Attributes of hypertrophy of the left ventricle on the electrocardiogram;
- The aortal form of heart with a dilatation of actively contracting left ventricle and ascending part of the aorta at the X-ray Survey and echocardiography;
- A tremor of the front cusp of the mitral valve. The diagnosis proves to be true by means of Doppler echocardio-aortographies.

On the basis of the received data of interrogation, the anamnesis of disease and clinical physical survey of the patient to deliver the preliminary clinical diagnosis to

the patient with AVF. «The infective endocarditis in a phase of exacerbation, aortal failure of the III stage, HF IIA, III class NYHA»

Additional methods of research:

1) clinical blood test,
2) clinical urine test,
3) biochemical blood test,
4) coagulogram,
5) immunologic blood tests,
6) electrocardiogram,
7) cardiophonography,
8) X-ray Survey,
9) echocardiography,
10) catheterization of heart and angiocardiography.

Clinical blood test, clinical urine test, a biochemical blood test, coagulogram, immunologic blood tests- see mitral stenosis.

The electrocardiogram at not sharply expressed defect can be normal. At the expressed aortal failure attributes of the hypertrophy of the left ventricle in the form of the enlarged amplitude of waves of complex QRS in conforming abductions, more often in combination with the variated terminal part of the ventricular complex (flattening, inversion of wave T, depression of segment ST) in the same abductions and a deflection of the electrical axis of heart to the left are marked. The elongation of interval P-Q specifying the disturbance of AV-conductivity is sometimes marked.

On *phonocardiogram thre* is diastolic murmur which follows the II sound is recorded and can occupy all diastole.

The X-ray Survey defines augmentation of heart: the left ventricle is massive, extended, with the rounded apex. The ascending part of the aorta is dilated, outlines the right contour of the shade of heart, on the left contour the expanded aortic arch is defined. Heart gets an aortal configuration.

The echocardiography is the important method in diagnostics of aortal failure. Following changes are most often found out:

- Dilatation and hypertrophy of the left ventricle;
- Augmentation of amplitude of movement of the interventricular septum and back wall of the left ventricle;
- Augmentation of the anteroposterior dimension of the left auricle (into the systole – up to 55 mm, into the diastole – up to 70 mm);
- Absence of diastolic closure of cusps of the aortal valve, small amplitude diastolic flutter of the front cusp of the mitral valve, invoked by the regurgitation of blood.

The catheterization of right departments allows to define the level of pulmonary-capillary pressure and a wave of regurgitation. The catheterization of the left parts of heart discovers augmentation of amplitude of pulse pressure. At *an aortography* the expressed regurgitation from the aorta into the left ventricle is defined.

Differential diagnostics. At detection of protodiastolic murmur on the base of heart and along the left edge of the breast bone differential diagnostics with Graham Steell's murmur is conducted first of all. In favour of failure of valves of the pulmo-

nary artery the detection of attributes of the arterial pulmonary hypertensia testifies: accent P2, protrusion of the trunk of the pulmonary artery at the X-ray Survey, respective alterations on the phlebogram, the pulmonic rheogram and features of movement of the valve of the pulmonary artery at the echocardiography.

Presence of murmur of Flint at aortal defect demands differential diagnostics with concomitant organic mitral stenosis. At auscultation of pansystolic murmur relative MF it is necessary to exclude its organic origin. Distinguishing characters of such rarely met associated lesions are: sharp dilatation of the LA which is not characteristic for "pure" AF; conservation of contractive abilities dilatated LV (at mitralisation it will be always reduced, and expressed HF is marked), intensifying of systolic murmur of the mitral regurgitation at decrease of the expression of cardiac weakness under ithe nfluence of the treatment (at relative MF it weakens).

Peripheric attributes of fast outflow of blood from the aorta inherent for the AF are characteristic also for the open arterial duct and tear of the aneurysm of sinus of Valsalva in the RV. At these diseases the augmentation of pulse BP and continuous murmur at the left edge of the breast bone and at the base of heart which should be distinguished from the pattern of mitralisation of the aortal defect is marked. Specific features of the anamnesis help to put the diagnosis, registration of continuous murmur unlike two continuous murmurs) at cardiophonography, expression of the pulmonary hypertensia and intensifying of a vascular drawing at the X-ray Survey.

Treatment of the patient with a stenosis of an ostium of the aorta

A choice of medical tactics: conservative or operative treatment.

I stage – surgical treatment is not indicated.

II stage –surgical treatment is not indicated.

III stage – operative treatment is indicated.

IV stage – prescription of medicamental therapy and a bed rest promote only temporary improvement of state of patients. Operative treatment is indicated.

V stage – medicamental treatment is not effective. Surgical treatment is not conducted.

To define principles of pathogenetically proved conservative therapy.

Medicamental treatment is conducted basically in occasion of congestive CH by the general principles, and in the beginning it is effective enough.

Under indications diuretic drugs, peripheral vasodilators, blockers of β-adrenoreceptors, cardiac glycosides, anticoagulants, metabolic drugs.

The basic directions of treatment of patients with aortal failure in the stage of compensation:

- Decrease of intensity and volume of exhaust from the left ventricle (restriction of the mechanism of Franc – Starling);
- Correction of metabolism of the myocardium;
- The decision on the question about the surgical treatment.

The basic directions of treatment of patients with aortal failure in a stage of decompensation:

- The control of pressure over the big circle of blood circulation;
- Decrease of the preload;
- Correction of metabolism of the myocardium;

– Optimization of the coronary circulation;
– Correction of electrolytic disturbances;
– Normalization of the heart rhythm; - direction to the surgical treatment.

To list the existing methods of operative treatment and the indication to it.

Surgical treatment is the basic method. At acute arterial failure it is recommended early surgical treatment. At a lesion of cusps of the aortal valve its prosthetic repair by mechanical or biological prosthesis is made. In case of the aortal failure caused by aneurismal dilating of the root of the aorta, its plasty with conservation of autovalve, or excising and stitching in of the vascular prosthesis with a simultaneous prosthetic repair of the aortal valve is made.

The left ventricular failure is the basic complication of defect; it proceeds at the preserved sinus rhythm and badly yields to treatment in the stage of the developed clinical presentations.

Diagnostics, and also tactics and procedure of rendering of the emergency medical care at originating of the acute left ventricular failure are presented in septum aortal stenosis -item 5.5.

At **AVF** it is necessary to be able to carry out the electrocardiography and to estimate it.

The basic complications of aortal failure except for the left ventricular failure are:

1) infective endocarditis;

2) atrioventricular block meets rarely and at the inflammatory genesis of defect.

Tricuspid stenosis

In clinical pattern attributes of mitral (mitral-aortal) defect dominates. Addition of the stenosis of the right atrioventricular foramen can be suspected at decrease of a dyspnea and the appearance of sharply expressed peripheric edemas, an ascites, augmentation of the liver, accompanied by the icterus and development of fibrosis. Except for it sharp general weakness is marked.

Complaints from other organs and systems. The pulsation of the liver is marked.

The Anamnesis of disease. The TS is a rarely met defect and usually has a rheumatic origin. As a rule, it is combined with mitral or mitral-aortal stenosis. Occasionally the lesion of the tricuspid valve is connected with a carcinoid syndrome.

The Anamnesis of life (including working conditions, professional harmfulnesses, etc.; at women the obstetric-gynecologic anamnesis).

Data of objective Survey (prominent features at the given disease):

1). The Evaluation of the general state of the patient with tricuspid stenosis (consciousness, the constitution, fatness).

2). The Congregating of the information on the internal state of the patient with TS (survey of skin, subcutaneous fatty stratum, palpation of the lymph nodes, thyroid and mammas).

3). Survey of state of the respiratory organs of patients with TS (survey of the thorax and upper respiratory tracts, palpation of the thorax, percussion and auscultation of lungs).

4). Survey of the state of organs of the abdominal cavity of patients with TS (survey of the abdomen, palpation and sucussion of the stomach, palpation of the intes-

tine, liver, lien, pancreas, kidneys, members of the small pelvis, auscultation of the abdomen).

5). Survey of the state of the osteomuscular apparatus of patients with TS (survey and palpation).

6). ***The local status* (*Locus morbi*)**. Survey and palpation of region of heart and superficial vessels, palpation of the main vessels of extremities and neck in the projective points, definition of percussion borders of heart, auscultation of heart and vessels with obligatory definition by means of additional manual techniques of special "pathognomonic" signs of the provisional acquired defect, to describe the technique of their carrying out. The basic clinical diagnostic attribute is significant jugular venous distention with their expressed presystolic pulsation (wave "a") and simultaneous pulsation of the liver. Collapse of veins (a negative wave "Y") is considerably slowed down. At auscultation accent P_2 is absent. Middiastolic murmur with presystolic intensifying at a sinus rhythm with an epicentre in the fourth intercostals on the left or above the xiphoid process. Unlike the murmur of a concomitant mitral stenosis this murmur strengthens on the breath in and weakens on expiration and at Valsalva test. Sometimes it is possible to auscultate the click of opening sometimes. On the basis of the received data of interrogation, the anamnesis of disease and clinical physical Survey of the patient to deliver the preliminary clinical diagnosis to the patient with the TS.

«Rheumatic carditis of the II degree of activity, associated mitral-tricuspid defect of the IV stage (mitral stenosis and tricuspid stenosis), HF IIA, III class NYHA».

Additional methods of research:

1) electrocardiogram,
2) cardiophonography,
3) X-ray Survey,
4) echocardiography,
5) catheterization of heart and angiocardiography.

On the electrocardiogram characteristic attributes of defect are hypertrophy of the right auricle (high wave P in abductions II, III and aVF) with not sharply expressed hypertrophy of the right ventricle, nonspecific changes of the terminal part of the ventricular complex (flattening, inversion of wave T, depression of segment ST) in many thoracal abductions are possible.

On the phonocardiogram the greatest diagnostic importance is given to the characteristic diastolic murmur strengthening in first half of breathing in, decrease of the interval of the II sound - opening sound of the tricuspid valve, splitting of the I sound with the enhanced high-pitched components.

At the X-ray Survey the augmentation of the dimensions of the right auricle is defined.

At echocardiographic research the typical pattern of the stenosis of the foramen without the expressed fibrosis of cusps is defined.

The catheterization of the right parts of heart is considered one of the main methods of diagnostics of rheumatic defects of the tricuspid valve in surgical clinic. The basic hemodynamic attribute of narrowing of the right atrioventricular foramen is the gradient of the diastolic pressure between the right ventricle and the right auricle.

Differential diagnostics. Differential diagnostics is more often conducted with the mitral stenosis. Unlike the last at tricuspid stenosis developments of stagnation in the small circle are not expressed. Auscultatory attributes of defect are better auscultated at the breast bone and strengthen at height of breath in. The diagnosis of stenosis of the tricuspid foramen proves to be true at the angiocardiography.

It is necessary to note, that stricture formation of the atrioventricular foramen can probably be made by the intracardiac tumour, more often a myxoma of the right auricle. Except for this various variants of the tricuspid defect can be a component of complex congenital cardiac anomalies (the atrioventricular communications, Ebstein's anomaly, etc.), characterized by specific symptomatology.

Treatment of the patient with tricuspid stenosis

A choice of medical tactics: surgical treatment is indicated at the expressed stenosis with pressure gradient not less than 4 mm Hg.

Specific conservative methods of treatment of tricuspid stenosis do not exist, an educing heart failure is treated, using the standard methods. Under indications there should be prescribed diuretic drugs, peripheric vasodilators, blockers of β-adrenoreceptors, cardiac glycosides, anticoagulants, metabolic drugs. Treatment is directed to decrease of stagnation in the big circle of blood circulation, augmentation of filling of the right ventricle.

Surgical treatment is conducted in conditions of an artificial circulation, using plasty and prosthetic repair of the valve. The "open" valvulotomy is more often made, or in cases of the earlier conducted correction of mitral defect, balloon valvuloplasty, at impossibility of their performance, and also at rough changes of cusps and subvalvular structures causing serious stenosis, the prosthetic repair of the tricuspid valve is indicated.

Failure of the tricuspid valve

The Most typical complaints at failure of the tricuspid valve.

Complaints on dyspnea, weakness, palpitation, gravity in right hypochondrium are typical. If defect is formed at the patient with the mitral stenosis got earlier, developments of stagnation in a small circle of blood circulation decrease, the dyspnea weakens, the patient tolerates horizontal position easier.

Complaints from other organs and systems. Sense of gravity in the right hypochondrium.

The Anamnesis of disease. Failure of the tricuspid valve can be organic or relative (functional). Principal causes of organic defect are:

1) rheumatic disease;
2) Ebstein's anomaly;
3) carcinoid syndrome;
4) idiopathic myxedema degenaration of the valve with prolapse its cusp during the systole;
5) infective endocarditis.

The Anamnesis of life (including working conditions, professional harmfulnesses, etc.; at women the obstetric-gynecologic anamnesis).

Data of objective Survey (prominent features at the given disease):

1) The Evaluation of the general state of the patient with failure of the aortal valve (consciousness, the constitution, fatness).

2) The Congregating of the information on the intarnal state of the patient with TVF (tricuspid valve failure) (survey of skin, subcutaneous fatty stratum, palpation of the lymph nodes, thyroid and mammas).

3) Survey of the state of the respiratory organs of patients with TVF (survey of the thorax and the upper respiratory tracts, palpation of the thorax, percussion and auscultation of lungs).

4) Survey of the state of organs of the abdominal cavity of patients with TVF (survey of the abdomen, palpation and sucussion of the stomach, palpation of the intestine, liver, lien, pancreas, kidneys, organs of the small pelvis, auscultation of the abdomen).

5) Survey of the state of the osteomuscular apparatus of patients with TVF (survey and a palpation).

6) ***The local status* (*Locus morbi*).** Survey and palpation of the heart region and superficial vessels, palpation of the main vessels of extremities and neck in the projective points, definition of percussion borders of heart, auscultation of heart and vessels with obligatory definition by means of additional manual techniques of special "pathognomonic" signs of the provisional acquired defect, to describe the technique of their carrying out. At survey the systolic pulsation of the cervical veins caused by the reversed current of blood from the right ventricle, jugular venous distention in a prone position, the expressed pulsation in epigastric region, in the region of the liver attract attention. The pansystolic murmur of medium intensity strengthening to the II sound is Auscultatory defined and at the base of the xiphoid process, at the left at the inferior edge of breast bone is defined, murmur strengthens on breath in (sign Rivero-Corvallo).

7) the Diagnosis of failure of the tricuspid valve is put on the basis of: Attributes of the venous hyperemia, positive venous pulse, detection of systolic murmur with characteristic topography, and also on data of the X-ray survey of heart.

8) On the basis of the received data of interrogation, the anamnesis of disease and clinical physical Survey of the patient to deliver the preliminary clinical diagnosis to the patient with TVF. «The infective endocarditis in a phase of the exacerbation, the combined defect of the tricuspid valve with predominance of failure of the III stage, HF IIA, III class NYHA»

Additional methods of research:

1) electrocardiogram,

2) cardiophonography,

3) X-ray Survey,

4) echocardiography,

5) catheterization of heart and angiocardiography.

On the electrocardiogram attributes of a hypertrophy of the right ventricle in the form of the enlarged amplitude of waves of complex QRS in conforming abduc-

tions in combination with the changed terminal part of the ventricular complex (flattening, inversion of wave T, depression of segment ST) in the same abductions, augmentation of wave P in abductions II, III and aVF are defined.

On X-ray dilating of the superior vena cava, prevailing augmentation of the right parts of heart, rounding of their contours in all projections, augmentation of the right auricle and ventricle in the second slanting projection are defined.

At echocardiographic research dilating of the cavity of the right ventricle, attributes of regurgitation on the tricuspid valve is defined.

At a cardiophonography tricuspid failure is displayed by systolic murmur (occupies all systole) with the epicentre at the base of the xiphoid process, with augmentation of amplitude at breath in and in position on the right side.

The catheterization ofthe right parts of heart is the most informative in diagnostics of the tricuspid defects. At TVF increase of the pressure in the right auricle and "ventriculisation" of the pressure curve due to augmentation of positive wave "V", invoked by increase of the pressure during filling of the auricle with blood during its diastole is marked.

Differential diagnostics. If tricuspid failure is combined with a mitral stenosis without hypertension in the small circle, with the greatest probability it is organic. In cases of revealing of this defect in combination with mitral and (or) aortal defect in serious stages its functional character connected with dilatation of the fibrous ring is most probable.

Relative tricuspid failure can be observed at patients in far late stages of dilated cardiomyopathy. The echocardiography allows define the dilatation of cardiac cavities, absence of organic lesions of valves and a significant depression of function of the myocardium.

At mitral failure systolic murmur unlike tricuspid, irradiates in axillary region and does not strengthen on the breath in.

Jugular venous distention, substantial growth of the liver, ascites, high venous pressure demands differential diagnostics between tricuspid defect and an adhesive pericarditis. At a pericarditis sound presentations of the multivalvular lesions are not observed, on the X-ray the cardiac shade is not enlarged and in many cases regions of calcification of the pericardium are defined.

A choice of medical tactics: conservative or operative treatment.

To define principles of pathogenetically proved conservative therapy

Specific conservative methods of treatment tricuspid do not exist failure, the educing heart failure is treated by using standard methods. Under indications diuretic drugs, peripheral vasodilator, blockers of β-adrenoreceptors, cardiac glycosides, anticoagulants, metabolic drugs. Treatment is directed to decrease of stagnation in the big circle of blood circulation,, decrease of volume of exhaust from the right ventricle, correction of metabolism of the myocardium and homeostasis.

To list existing methods of operative treatment and the indication to it.

The semicircular annuloplasty offered H. M. Amosov in 1972 became widespread as surgical treatment. It provides narrowing of the extended fibrous ring correspondingthe front and back cusps of the valve by means of semipouch suture.

From other plastic operations it is necessary to note ring annuloplasty with use of basic ring Carpentier. It provides strengthening of the fibrous ring by the metal ring selected by size covered by synthetic tissue, for decrease of the dimensions of the tricuspid foramens and maintenance of closure of cusps of the valve.

The basic complications of the tricuspid defects are defined by the early and expressed venous stagnation with development of cirrhosis of the liver, ascites, other disturbances of the major organs and systems. Tricuspid defects cannot be isolated and, being combined with defects of other valves, burden their process substantionally.

Literature: 1. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. European Heart Journal (2008) 29, 2388–2442; 2. Hurst, J. Willis, et al. The Heart. 7th ed. New York: McGraw-Hill, 1990.

II.2. Coronary heart disease and its complications

On a background of considerable successes of medicinal treatment of ischemic heart disease (IHD) and its complications surgical methods did not lose their value, but began to be wider used in everyday clinical practice. After wide introduction in clinical practice of coronarography, that allows to conduct exact diagnostics of lesions of coronary arteries, the methods of direct revascularization of myocardium began to develop extraordinarily widely. In some countries the number of operations of direct revascularization of myocardium reaches more than 600 on a 1 million population. World Health Organization set that requirement in such operations taking into account frequency of death rate from IHD must make no less than 400 on 1 million population per year. Today already there is not a necessity to prove efficiency of surgical treatment of IHD by the methods of direct revascularization of myocardium. Presently operations are accompanied with low mortality (0,8-3,5%), result in an improvement of quality of life, prevent the development of heart attack, increase life-span for many severe patients.

Ischemic heart diseaseis the disease of heart, caused diminishing or arrest of blood delivery tomyocardium associated with a pathological stenotic process in coronary vessels (Fig. 14).

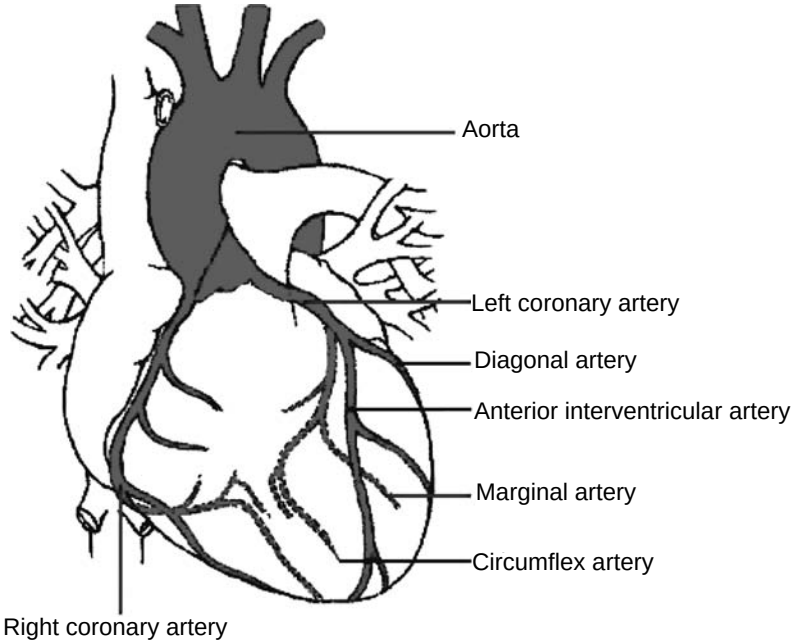

Fig. 14. Anatomy of coronary arteries

However not every pathology of coronary vessels of heart which gives the typical clinical presentation of coronary insufficiency (stenocardia, heart attack), is defined as IHD. IHD is violation of delivery blood to myocardium due to atherosclerosis of coronary vessels of heart.

IHD has the following forms: Stenocardia; Heart attack; Acute coronary insufficiency, one of typical signs of which is sudden death on a background of coronary atherosclerosis; Painless form which shows up insufficiency of blood circulation or violation of heart rhythm.

Etiology. The number of factors is instrumental in the development of IHD (risk factors). Among them on the first place it is necessary to put arterial hypertension which it is found in 70% of patients with IHD. Arterial hypertension is instrumental in more rapid development of atherosclerosis and spasm of coronary arteries of heart. Risk facrot of development of IHD is also diabetes mellitus which is instrumental in development of atherosclerosis as a result of violation of exchange of proteins and lipids. Smoking also acts in development of IHD. The spasm of coronary vessels develops at smoking, the hypercoagulation of blood is instrumental in the development of thrombosis of the changed coronary vessels. Genetic factors have the defined value. It is found that if parents suffer IHD, in their children it develops in 4 times more often, than in persons the parents of which are healthy. Hypercholesterolaemia promotes probability of development of IHD to a great extent, as is one of important factors, that contributes development of atherosclerosis in general and coronary vessels in particular. At obesity IHD meets twice more frequent, than in persons with normal body mass. In patients with obesity the level of cholesterol is increased in blood, in addition these patients conduct not mobile regimen of life, which also assists development of atherosclerosis and IHD.

IHD is one of the most widespread diseases in the industrially developed countries. For the last 30 years frequency of development of IHD was increased in 2 times, that is associated with psychical overexertion. IHD develops approximately on 10 years earlier in men, than in women.

Pathoanatomical changes depend on the degree of atherosclerosis lesion of coronary vessels. At stenocardia and absence of heart attack there are only single foci of cardiosclerosis. At least 50% stenosis of coronary vessels is needed for development of stenocardia. Course of stenocardia is especially severe, if two or three coronary vessels are affected simultaneously. At the heart attack already in the first 5-6 hours after a pain attack there is necrosis of muscular fibers. In 8-10 days after the heart attack plenty of new capillaries appear. Thereafter in the areas of necrosis connective tissue develops rapidly. After this time scarring begins in the areas of necrosis. Through 3-4 months the area of myocardium infarction corrugates and is fully substituted with fibrotic tissue. Parietal thrombosis develops often in the lesion of endocardium.

A myocardial ischemia arises up as a result of violation of balance between providing of cardiac muscle with oxygen and its requirement in it. For a concrete patient can take place changes of one or both of these parameters. Although in majority of patients the permanent blockage of arteries develops as a result of their atherosclerotic lesion, modern information shows frequency of spasm of coronary arteries to be far more often than was considered before. There are three basic determinants of the myocardium providing with oxygen and there are three determinants of oxygen demand of myocardium.

Causes of myocardial ischemia:oxygen delivery of myocardium is decreased, obstruction of coronary arteries, permanent blockage, arteriosclerosis, other causes,

spasm of arteries, system hypotension, severe anemia, the oxygen demand of myocardium is increased, hypertrophy of myocardium, tachycardia, basic determinants of oxygen supply of myocardium and its requirement in oxygen, supply, pressure of diastole in an aorta, resistance of coronary vessels, duration of diastole, necessity, rate of heart contractions, tension of walls of heart cavities, pre-loading is eventual pressure of diastole in the left ventricle, post-loading is middlepressure in an aorta, contractility.

An ischemia causes the basic changes of two important functions of myocardium cells - their electric activity and contractility. In ischemic cell transmembrane potential of action changes acutely. So, potential of action in the ischemic myocardium cell of ventricle is characterized by the increase of potential of rest, deceleration of speed of intensification and shortening of phase a plateau. Between normal and ischemic tissue of myocardium there is an electric difference of potentials, which generates different arrhythmias, occurred in stenocardia or acute myocardium infarction (AMI). The most essential consequence of violation of contractility of myocardium is weakening of function of the left ventricle. At first its capacity is lost for the normal weakening in a diastole which results in the decline of contractility of ventricle and is presented by 4^{th} sound clinically. At deeper ischemia a capacity is lost for contraction of systole, and the affected area becomes hypokinetic or akinetic. In the case of development of myocardium infarction affected area loses inflexibility quickly (within few minutes or hour), becomes dyskinetic with paradoxical movement during a systole. All of it results in diminishing of ejection fraction. Minute volume in such conditions the cardiovascular system often compensates by the increase of pressure of filling for support of adequate stroke volume according to the Frank - Sterling mechanism.

The natural course of IHD is determined mainly by two physiopathology factors:

1) by the degree of obstruction of arteries (blockage of one, two or three vessels);

2) by the condition of function of the left ventricle.

Association of symptoms and clinical information with natural course of IHD can be evaluated only taking into account these two factors, some researchers consider although, that complex ventricular ectopia (gradual "downshift" ventricular ectopic contractions or "splashes" of ventricular tachycardia) is the factor of early mortality. In different studies it is revealed also, that in few of cases of IHD presents without clinical symptoms; such "mute" IHD occurs at 2,5-10% persons of middle age, which belong to the different groups of population. Mortality among them, presumably, is lower, than among patients with the symptomatic forms of IHD.

Clinical classification of IHD (1979, WHO).

1. Primary arrest of circulation of blood
2. Stenocardia
   - 2.1. Exertional stenocardia
     - 2.1.1. First developed stenocardia
     - 2.1.2. Stable
     - 2.1.3. Making progress stenocardia
   - 2.2. Stenocardia of rest (spontaneous stenocardia)
     - 2.2.1. Special form of stenocardia

3. Myocardium infarction
3.1. Acute myocardium infarction
3.1.1. Definite
3.1.2. Possible
3.2. Old myocardium infarction
4. Cardiac insufficiency
5. Arrhythmias.

Clinical classification of IHD (SKRC AMS, 1983)

1. *Acute coronary death*
2. *Stenocardia.*
2.1.1. First arising up stenocardia of tension;
2.1.2. Stable exertional stenocardia (with pointing of functional class);
2.1.3. Making progress exertional stenocardia;
2.2. Spontaneous stenocardia.
3. *Myocardium infarction*
3.1. Large-focal myocardium infarction;
3.2. Small-focal myocardium infarction.
4. *Post-infarction cardiosclerosis.*
5. *Disturbance of cardiac rhythm.*
6. *Cardiac insufficiency.*

Classification of post-infarction aneurysm:

1) True aneurysm:
a) diffuse;
b) saccular (with narrow basis);
c) dissecting.

2. False aneurysm — walls of myocardium, which are formed at rupture, and limited by pericardiac adhesions.

3. Functional aneurysm — areas of viable (so-called hybernation) myocardium which lost contractive ability and protrude at the systole of ventricles.

Many authors subdivide aneurysm into three basic types (modification of classification of W. Stoney, 1994):

I — aneurysm with normokinesis of part of the left ventricle which is contracted, EF – 50%.

II — aneurysm with hypokinesis of segments of part of the left ventricle (on Coltharp, 1994: normokinesis of anterior wall and hypokinesis of posterior wall of the left ventricle) which is contracted, EF more than 30%.

III — aneurysm with expressed hypokinesis of walls of the left ventricle (on Coltharp, 1994: normokinesis of anterior wall and akinesia of posterior wall), EF less than 30%.

Disturbance of cardiac rhythm and cardiac insufficiency, if they are not consequence of acute myocardial ischemia and post-infarction cardiosclerosis, must be attributed to complications of atherosclerotic cardiosclerosis and never are the independent forms of IHD.

Classes of stenocardia (classification of Canadian society of heart and vessels study)

Functional class of stenocardia	Description of stenocardia as reaction on the physical loading
I	The everyday physical loading (walking, lifting upwards on a stair) does not cause a stenocardia. A stenocardia arises up only at the short or protracted tense physical loadings during work or sport exercises
II	Small limitation of the everyday physical loadings A stenocardia is caused: a) walking on a horizontal surface, and also lifting on a stair b) after meals, in cold weather outdoors, under the action of wind; c) in a condition of negative emotional stress; d) during 4 hours after awakening of patient; e) walking more than 2 quarters on distance, on a horizontal surface; f) lifting upwards on two floors on an ordinary stair in the comfort terms of environment
III	Considerable decrease of tolerance of the ordinary physical loading. A stenocardia is caused|: a) walking less than, than two quarters on distance; б) walking on the horizontal surface of any duration and intensity; в) lifting on an ordinary stair in a middle rate in the comfort terms of environment
IV	Impossibility of the everyday physical loadings of any intensity and duration without a stenocardia, a stenocardia can arise up in the conditions of rest

Clinical presentation. A stable stenocardia is characterized by the episodic retrosternal pain which lasts during a few minutes (5-15 minutes), which is provoked by the physical loading or stress and disappear in a condition of rest or by the reception of nitroglycerine (under a tongue). Pain is almost always localized behind breastbone and often irradiates into the neck, lower jaw and shoulders (or below — to left or both hands). Pain can be accompanied by the secondary symptoms — dizziness, accelerated palpitation, sweating, shortness of breath, nausea or vomiting. At auscultation transitory S-sound can be determined or systolic noise on apex. On ECG during acute attack are changes (registered approximately during the half of time), often decline of segment ST or (rarer) its increase, are observed. The level of serum creatininkinase does not increase.

An unstable (increasing or pre-infarction) stenocardia is the clinical ocndition between stable stenocardia and acute myocardium infarction (AMI). Categories of unstable stenocardia:

1) exertional stenocardia, which developed recently, usually within the limits of 4-8 weeks;

2) stenocardia with a making progress course, increase of degree of severity which is characterized by increased duration of attacks or increase of requirement in nitroglycerine;

3) rest stenocardia.

An unstable stenocardia is presently considered as a result of increase of severity and increase of degree of atherosclerosis of coronary arteries, spasms of these arteries or hemorrhage in non-thrombosed plaques with subsequent thrombotic occlusion which develops during a few hours or days.

A variant stenocardia (***stenocardia of Princmetal***) is observed mainly in a condition of rest and without provocations. Attacks have a tendency to repeat at one and the same time of day. Pain is accompanied by the increase of segment ST, which represents the presence of transmural myocardial ischemia. At the increase of segment ST can take a place painless episodes. Attacks can be associated with tachyarrhythmia, by the blockade of Gis crura or with atrioventricular blockade. Presently this variant of stenocardia is considered as consequence of spasm of epicardial coronary arteries. At a coronary angiography such patients approximately in third of cases have absence or little expression of atherosclerosis, and in other cases, except for spasm, IHD is revealed. For the patients of the last group, except for a variant stenocardia, can take a place stenocardia of tension. A spasm is characteristic not only for a variant stenocardia; it is observed in patients with a typical stenocardia or AMI.

Acute myocardium infarction

Although majority of patients with AMI suffer ischemic heart disease, unique point of view on exact nature of process, which provokes acute myocardium infarction, absents. The modern pictures of direct reason of AMI assume co-operation of great number of factors:

– progress of atherosclerotic process till complete occlusion of vessel; hemorrhage under vessel intima on its narrowed area;
– embolism of coronary artery; spasm of coronary artery;
– thrombosis on the area of atherosclerotic plaque.

Recent studies confirm the important role of acute intracoronary thrombosis and (in less degree) of arterial spasms. Both processes are potential convertible, that again make interesting early aggressive intervention in AMI. Main determinant of success is time which passed from appearance of symptoms to beginning of treatment. The doctors of first-aid, prognosing the result of major trauma, talk usually about the “gold first hour”; at AMI such determination of time also is true in relation to the first 2 hours. The former treatment approaches of AMI, providing of rest of the cardiovascular system and treatment only of its complications, gradually are replaced by interventions able to remove the provoking factor of myocardium infarction.

Like an ischemia, a myocardium infarction results in the serious changes of two major functions of myocardium cells: electric depolarization and contractility. Disturbance of one or both functions causes complications of AMI. In a few first hours a myocardium infarction is a process which was not yet completed; areas of myocardium infarction are surrounded by the ischemia or damages foci. A main factor at prognostication of result of disease and mortality is an amount of myocardium

infarction tissue. These ischemic areas around myocardium infarction serve as the potential object of medicinal and surgical treatment.

At AMI often there is arrhythmia. Electric heterogeneity of surrounding areas of normal and ischemic myocardium causes tachyarrhythmia and ventricular ectopia usually. Bradyarrhythmia and atrioventricular blockade is caused by the increase of tone of vagus or direct influence of myocardium infarction on the conducting system.

The basic result of disturbance of contractility is insufficiency of pumping function of the left ventricle (LV) of heart. At worsening of work of myocardium of LV on 25% cardiac insufficiency develops usually; if 40% of tissues is affected, quite often there is cardiogenic shock. Recent studies pay more attention to the influence of AMI on the pumping function of right ventricle (RV). Acute mitral regurgitation which results in acute pulmonary edema and hypotension can develop at worsening of work of papillary muscles of mitral valve.

A myocardium infarction area can be subjected to autolysis with the development of expressive clinical syndromes, related to the rupture of wall of ventricles, interventricular septum or papillary muscles.

Blood congestion can result in the thrombosis of veins and embolism of branches of pulmonary artery. Blood congestion in the ventricle cavity and exposure of collagen layer on the area of myocardium infarction is able to result in a parietal thrombosis and system embolization of arteries

A classic symptom is severe anginal pain which lasts more than 15-30 min. As well as in stenocardia, pain can be atypical, but it is accompanied by such symptoms, as dizziness, shortness of breath, sweating, accelerated palpitation, nausea or vomiting. On development of AMI in not young persons (unlike junior patients) pain is more often localized not behind breastbone or quite absent. In addition, in not young patients it is higher probability of presence of nonspecific symptoms. As studies of population show, the myocardium infarction remains clinically not recognized almost in 25% cases. Although it is not the generally accepted point of view, some doctors consider that patients with diabetes mellitus more often have such "mute" myocardium infarctions. Untruthfully normal information can be got at the inspection of patient. As a rule, the mild or moderate tachycardia is determined, although in the myocardium infarctions of posterior wall quite often there is bradycardia. The increase of arterial pressure depends on severity of pain and degree of activation of the sympathic nervous system. An mild fever takes place often, but the body temperature rarely exceeds 39 °C. Palpating apex can be diffuse or limited. As worsening of contractions of LV develops S-sound can weaken. At the increase of time of banishment of blood from LV the paradoxical splitting of S2-sound is sometimes listened. The decline of compliance of ventricle walls very often causes appearance of S4-sound, and sometimes audible and soft S3-sound. New noises of systoles require a careful study. They can specify on:

1) mitral regurgitation as a result of dysfunction or rupture of papillary muscle;
2) rupture of interventricular septum;
3) noise of pericardium friction.

Q-positive and Q-negative myocardium infarction

Terms "Q-positive" and "Q-negative" myocardium infarction are often used for differentiating of transmural and nontransmural (or subendocardial) myocardium

infarction, as they are characterized of presence or absence of Q wave. Appearance of Q wave very badly correlates with the presence of transmural myocardium infarction, visible at pathanatomical exam. Rate of complications and mortality at AMI depend on the degree of lesion of myocardium, but not from appearance of Q wave. Q-positive myocardium infarctions are more extensive (higher maximal level of serum creatininephosphokinase and decrease of EF, determined at radioisotope exam), that shows that more myocardium tissue is affected, than at "Q-negative" myocardium infarctions. It is marked at group analysis, that Q-negative myocardium infarctions are accompanied with less in-hospital mortality, but more often recur or abandon after itself stenocardia. As a result mortality of these two types of myocardium infarction by the time becomes equal.

Disturbance of conducting system

AMI can injure conducting system and sometimes causes complete (III degree) atrioventricular blockade (AVB). The risk of development of complete AVB in patients with AMI depends mainly on two factors:

1) location of myocardium infarction and

2) presence of new disturbances of conducting system.

If the location of myocardium infarction is usually known, the duration of existence of disturbances of conducting system very often remains not clear. In many studies term "fresh or block of conducting system of indefinite duration" is used. In addition, in number of studies it is talked about "AVB of high degree", that implies equal secerity of AVB of the third and second degree. The risk of development of complete blockade is especially large in two groups of patients. At first, it is increased in those patients at which the certain form of disturbance of conducting system in AV-node (AVB of the first or second degree) is superimposed on the lower (infranodal) block of conducting system. Secondly, patients with the myocardium infarction of anterior wall in the case of development of complete AVB have a risk of development of deep, as "slip" of ventricular driver of rhythm often is slow and unreliable.

AVB of the first degree or AVB Mobitz I (Venkebakh) usually is conditioned by disturbances of conducting system in AV-node as a result of increase of vagus tone and it id observed, as a rule, at an ischemia or myocardium infarction of posterior wall. Passing to complete AVB meets rarely and rarely is sudden; if it occurs, a stable infranodal rhythm is usually supported with the narrow complexes of QRS and moderate rate — about 50 impulses per minute. In those cases, when treatment is needed, such blockade usually is stoped by atropine.

AVB (Mobitz II) of the second degree more often develops as a result of structural damages of infranodal conducting tissue and is observed, as a rule, at an ischemia or myocardium infarction of anterior wall. Complete AVB can develop suddenly, here cardiac activity is determined only by slow and unstable ventricular slip of pacemaker. The presence of block of Mobitz II serves as indication to the prophylactic use of artificial rhythm driver. Myocardium infarctions, which cause such blocks, usually extensive, and even at pacemaker treatment many patients die from insufficiency of pumping function of heart.

Mechanical defects

Rupture of heart is a catastrophe, which is characterized by sudden renewal of retrosternal pain, hypotension, pericardiac tamponade and heart arrest, with dissociation of electric and mechanical processes, which leads to death. The group of increased risk is consists of patients with the first myocardium infarction, patients with arterial hypertension, which is preserved after myocardium infarction, and in elderly; in 50 % it occurs in the first 5 days, and in 90 % cases — in the first 14 days after myocardium infarction. Mortality is 95 %; not many patients survive during the resuscitation, immediate surgical intervention: pericardiotomy removal of tamponade.

The rupture of interventricular septum shows up acute development of pulmonary edema and development of rough noise of systole on the left margin of breastbone. The rupture of interventricular septum meets approximately with equal rate in the myocardium infarctions of anterior and posterior wall of myocardium and is localized in muscular part of interventricular septum. Treatment is begun with diminishing of post-loading by nitroprusside or (if it appears ineffective) by an intraaortic balloon counterpulsation.

Quite often, especially at the myocardium infarctions of inferior part of posterior wall, dysfunction of papillary muscle develops. Clinical symptoms usually are very moderate, transitory systole noise is marked only, however they can become more serious at development of pulmonary edema. Treatment of ischemia and decline of post-loading in such cases is usually effective. A prognosis at moderate dysfunction is good enough.

More severe complication is tearing off of papillary muscle; its prognosis depends on the extension of tearing off (all muscle or only its head). Tearing off of all of muscle is characterized by high mortality – up to 50% in the first days. It arises up usually at posterior diaphragmatic myocardium infarction and involves a posterior papillary muscle. Clinical diagnostics of dysfunction of papillarry muscle or its tearing off always is not easy and there can be necessity for introduction of catheter of Svan-Ganz.

Thromboembolic complications

Patient with AMI are subjected to the prolonged bed regimen and generalized circular stasis - the risk factor of development of venous thrombosis and embolism of pulmonary artery. In addition, parietal blood clots can be formed in the place of myocardium infarction. Such blood clots are observed less than in 5% of patients with a Q-negative myocardium infarction or myocardium infarction of inferior part of posterior wall and in 30-40% patients with the Q-positive myocardium infarction of anterior wall. In some patients a blood clot is formed already in the first two days; in these patients extensive myocardium infarction is usually revealed in hemodynamic complications and are is characterized by high in-hospital mortality. After forming blood clot can regress in LV, remain asymptomatic or embolized peripheral vessels. According to data of most studies, rate of systemic embolization in the patients with AMI ranges from 1 to 6%, although in patients with parietal blood clots the risk of this complication is about 30%. The good tool of sxreening is cardiac ECHO; at the exposure of parietal blood clot and in absence of contra-indications anticoagulative therapy begins (intravenous introduction of large doses of heparin).

Pericarditis

The acute form of pericarditis which is presented by pain and noise of friction of pericardium can develop in the first 7 days after myocardium infarction. According to studies frequency of this complication in the patients with AMI is 6-10%, it is higher in Q-positive myocardium infarction. Cause of pericarditis is inflammation, related to area of necrosis of myocardium, adjoining to the pericardium. The syndrome of postmyocardium infarction (Dressler syndrome) develops, as a rule, later and is characterized by retrosternal pain, fever, pleuropericarditis and effusion in pleura cavity. Its cause is an immunological reaction on myocardium antigens which appear in AMI.

Myocardium infarction of right ventricle

It is presently found that from 19 to 43% cases of myocardium infarctions of posterior wall are the myocardium infarctions of RV. Myocardium infarction of RV is the result of occlusion of right coronary artery; it almost always is transmural and related to the lesions of LV. It was considered before, that a right ventricle has mainly capacity function and dysfunction of its pumping activity does not have clinical value. However severe insufficiency of pumping function of RV is accompanied by hypotension, usually with the increase of pressure in right auricle and normal or decreased pressure in the left auricle. Therefore clinical symptoms include hypotension, swelling of jugular veins and increase of transparency of pulmonary tissue. In addition, in most patients with the hemodynamically significant myocardium infarction of RV pressure increases in jugular veins and the noisy breathing appears (Kussmaul symptom). Disturbance of pumping function of RV can mask the hidden left ventricular insufficiency, constricitve pericarditis, pericardiac tamponade and restrictive cardiomiopathy. For exact diagnostics of violation of pumping function of RV the simultaneous measuring of pressure of blocking of pulmonary artery and pressure in right auricle or visualization of insufficient contractions of RV by a radioisotope scan-out are needed. Support of minute volume in patients with the myocardium infarction of RV is possible only at increased pressure of filling of RV, that is why it is necessary to avoid its decline by diuretics or nitrates. Some increase of minute volume can be obtained by volume infusion, but far more effective is application of inotrope support by preparation of type of Dobutaminum.

Features of examination of patient with suspicion on IHD

At questioning of patient:

1) Complaints about pain behind the breastbone in a condition of rest or at the physical loading with an irradiation to the left arm, shoulder-blade, shortness of breath.

2) Complaints from the side of other organs and systems, as a rule, caused by concomitant pathology or complications of IHD.

3) In anamnesis of disease to pay a regard to duration of disease, loadings, which cause the attacks of stenocardia, its duration, what stops it; information about previous methods of examination.

4) Anamnesis of life

Clinical physical examination:

1) The general condition of patient depends on the form of IHD and presences of its complications.

2) Appearance of patient (the color of skin, as a rule pale, acrocyanosis is possible).

3) Inspection of the condition of pulmonary system (weakening of the vesicular breathing is possible, in the case of cardiac insufficiency with development of hydrothorax respective physical information).

4) Inspection of the condition of organs of abdominal region (at insufficiency of major circle of blood circulation - enlarged liver, ascites is possible).

5) Inspection of the condition of bone-muscular system.

6) Locus morbi (cardio-vascular system): an inspection and palpation of area of heart can be within the limits of norm; as a rule, an heart apex is displaced to the left; accordingly the left border of heart can be also displaced to the left, tones of heart are absorbed, at presence of arrhythmia the respective changes of pulse rate and heart beat rate).

7) Leading clinical syndrome – pain (retrosternal pain at physical exrcise or in a condition of rest).

8) Formulation of previous clinical diagnosis: IHD, stable stenocardia, II functional class, atherosclerotic cardiosclerosis, insufficiency of circulation of blood of 0 stage.

***Plan of additional examination of patient with IHD includes*:**

1) *Cardiomarkers*

Historically for diagnostics of acute coronary death determination of activity of one of enzymes, which is synthesized in our organism, was used (and continues to be used) – creatininephosphokinase (CFK). More precisely, one of its constituents — MB-fraction of creatininekinase (CFK-MB). Increase of activity of CFK-MB is the most specific for AMI. The increase of activity of CFK-MB begins already in 4-8 hours after the onset of attack and reaches maximum in 12-24 hours; on the third day activity of this fraction goes back to normal. At expansion of area of myocardial ischemia, activity of CFK-MB remains high far longer, that allows to diagnose AMI in the further terms of supervision.

However an increase of fraction of CFK-MB is determined also in unsurgical diagnostic manipulations on heart. Radio-therapy of thorax can cause the insignificant increase of level of enzyme also. However arrhythmia, tachycardia and cardiac insufficiency, practically does not influence the level of CFK-MB.

Valuable contribution to the decision of all of afore-mentioned questions was introduction to clinical practice the determination of serum myocardium markers — troponins I and T – absolutely specific and highly sensitive for diagnostics of acute coronary death (OCD). The presently leading cardiologic societies of Europe and USA accepted recommendations in which determination of concentration of troponin T (in serun, plasma) of blood is examined as a major diagnostic criterion of the AMI.

Reliable cardiomarker is cerebral natriuretic peptid (NTproBNP). Reliably and evidential grounded, that the exposure of its increased concentrations confirms the presence in the patient of cardiac insufficiency (CI). Therefore determination of level of this peptid already became a standard in a diagnostics of CI in the whole world. It is impossible not to mark that it was rotined in a number of large international researches, that application in clinical practice of tests of "NT-proBNP" and

"BNP" allows to reduce the cost of inspection of patients and exactly enough to foresee a presence or absence of SN, even to the leadthrough of echocardiography. A fact is yet important: presently a test found a wideuse not only at cardiovascular pathology but also at some clinical conditions which are accompanied the decline of contractive ability of cardiac muscle.

2) *Electrocardiography*

Electrocardiography is used for:

a) diagnostics of hypertrophy of myocardium;

b) diagnosticians of disturbance of rhythm of heart;

c) diagnostics of violations of coronary circulation of blood;

d) conduction of ECG at implementation of stress-tests.

12 leads – 3 standard, 3 intensified unipolar and 6 thoracic are used in practice. Precardial cartography (35 electrodes are set in 5 horizontal rows) is used for clarification of location of heart changes, size of post-infarction and necrotic area, study of their dynamics1 In addition, ECG can be registered by the esophageal lead.

Daily (Holter) monitoring of ECG is conducted in patients in which stress-tests are contra-indicated (physical loading, pharmacological tests, test of loading with a transesophageal electrostimulation).

3) *Echocardiography*

Echocardiography is a method, which is based on registration of reflected ultrasonic signals from heart structures, which move. It is used for:

a) diagnosticians of violations of morphology and mechanical activity of heart;

b) monitoring of activity of heart;

c) control of minimally invasive endocardiac interventions (fibrillation ablation).

Echocardiography allows:

1. To identify the valves of heart, their relative position.

2. To recognize interatrial and interventricular septa, to analyze their continuity, assess the type of motion.

3. To estimate anatomic relative position of valves of heart and interventricular septum.

4. To evaluate motion of leaves of valves.

5. To conduct measurings and define the changes of thickness of walls and sizes of chambers of heart for determination of presence and severity of dilatation of cavities of heart and hypertrophy of myocardium of the left and right ventricles.

6. To conduct Doppler echocardiography together with two-dimensional echocardiography, for the exposure of symptoms of valvular regurgitation, narrowing on the way of blood flow and endocardiac shunts. Stress-echocardiography is used for replacement of ECG stress-tests, more often with the physical loading. Contrast echocardiography is also used (contrast is entered intravenously). Except transthoracic echocardiography, transesophageal echocardiography (including three-dimensional) and endocardiac echocardiography are used.

4) *Electron beam computer tomography*

Electron beam computer tomography is new technology of CT-scan, which allowed considerably to shorten examination time by comparison to ordinary CT. Mainly it is used for diagnostics of proximal occlusions of coronary arteries, anomalies of origin of coronary vessels, and also their aneurysm. Disadvantage – difficulties with visualization of distal stenosises of coronary arteries.

5) *Spiral tomography*

Multispiral computer tomography (MSCT) for diagnostics of atherosclerotic lesion of vessels began to be used from 90th of the last century. Visualization of coronary arteries was impossible at the beginning, because of low resolution and high percentage of artifacts of motion, that is why the atherosclerotic lesions of arteries were evaluated by the count of intravascular calcium content. The quantitative estimation of coronary calcinosis is based on the coefficient of roentgen absorption and area of calcinates. Calcium index (CI) on the Agatston method is determined as product of area of the calcinated lesion on the factor of density. It was marked that CI represents the prognosis of lesion of the cardio-vascular system and directly correlates with the rate of development of atherosclerosis: the higher index, the higher risk of atherosclerotic lesion. For example, at low CI from 10 units and below - probability of atherosclerosis of coronary arteries makes no more than 5-10%. From 11 to 100 units (moderate CI), possibility of presence of 50% narrowing - no more than 20%, at CI 101-400 units – 75%, that is moderately high risk of atherosclerosis. And at high CI, more than 400 units – probability of atherosclerotic lesions of coronary arteries is about 90%. CI is predictor of development of future cardio-vascular catastrophes.

High ability of 64-MSCT allows:

a) definite visualization of coronary arteries, with clarification of location of atherosclerotic lesions, exposures of anomalies of development of coronary vessels of heart;

b) to determine the condition of aorto-coronary shunts and intravascular endoprosthesiss (stents);

c) to conduct the count of calcium index with the purpose of clarification of prognosis of disease;

d) to mark violation of perfusion and viability of myocardium for patients in the early and late terms of myocardium infarction;

e) to assess contractive ability of heart;

f) to study the condition of pericardium, valves of heart.

Visualization of atherosclerotic lesion of coronary arteries by MSCT is the alternative of invasive coronaroangiography (CAG) and used for both well-proven IHD, at suspicion on IHD and asymptomatic patients with the purpose of diagnostics, exposures of risk groups and determination of AMI, subsequent prognosis.

In particular indications for the MSCT are:

1) atypical pain in thorax;

2) presence of risk factors:
   - arterial hypertension,
   - lipidemia,
   - obesity,
   - diabetes mellitus,
   - smoking,
   - high CI,
   - family anamnesis of coronary arteries, sudden death, lesion of peripheral arteries;

3) acute coronary syndromes, myocardium infarctions without elevation of ST for the estimation of lesion of coronary arteries of heart. MSCT allows to diagnose possible complications at the myocardium infarction, for example, rupture of interventricular septum, and also development of aneurysm of the left ventricle.

6) *Isotope methods*

Isotope methods are based on introduction intravenously of albumin or red corpuscles with a radio-active mark (thallium-201 or technecium-99m). These methods allow to specify contractive function and blood supply of myocardium, find out an ischemia and foci of necrosis of myocardium, cicatrical areas.

7) *Angiography*

A coronary angiography is a method, which allows to reveal presence, severity, location and prevalence of coronary stenosis, and also to find out their cause (atherosclerosis, blood clot, spasm) (Fig. 15). Coronary angiography is considered "golden standard" in diagnostics of coronary atherosclerosis. It belongs to the selective angiography, conducted by the special catheters on S. Seldinger (1953), as a rule, by retrograde access through a femoral artery. Indications: severe stable stenocardia (class 3 on classification of Canadian caedio-vascular society); stable stenocardia (class 1 or 2), if a patient have suffered the myocardium infarction or there are ischemic episodes at the little physical loading; at a stable stenocardia for patients with the blockade of His bundle crus, if the areas of ischemia are found at scintigraphy; in patients which have indications to the operations on coronary vessels; at severe ventricular arrhythmias; at the recurrence of moderate or severe stenocardia in patients in which revascularization of myocardium wasconducted (cathether angioplasty or aorto-coronary by-pass).

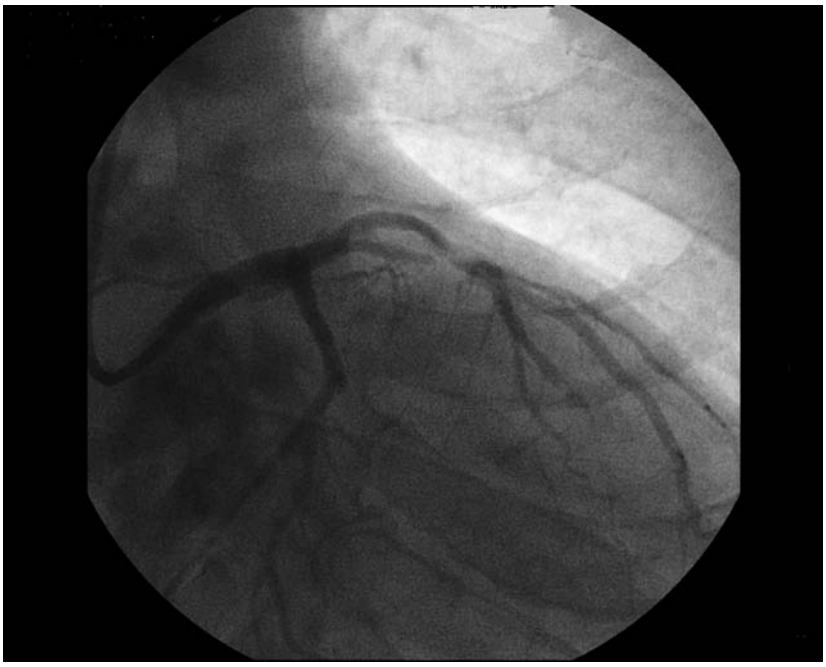

Fig. 15. Coronarography – the occlusion of the left anterior descending artery

Coronarography allow to define a volume and tactic of intervention. Thus, without coronarography it is impossible to conduct any operative intervention, coronary angioplasty with a stenting or aorto-coronary by-pass.

Coronarography is invasive procedure, related to the certain risk! During the Coronarography complications are possible, but the risk of development of these complications is very small. However bleeding in the place of puncture, allergic reaction on contrast, disturbance of heart rhythm, myocardium infarction can develop. Before coronarography, a patient must pass some examinations: general analysis of blood, blood group, Rh-factor, tests on the viruses of hepatitis C and B, HIV, RW, ECG in a 12 leads, echocardiography, and also to purchase an elastic bandage.

Technique: under local anesthesia (during procedure a patient is in consciousness) puncture of artery is performed (thigh, axillar, radial). In an artery a small hollow tube (introducer) of diameter less than 2 mm is entered. Through a tube a catheter is introduced to the coronary arteries of heart. Through catheter contrast is entered and under X-ray control of the special angiograph an exam record is performed. After implementation of coronarography control of bleeding in the place of puncture is carried out, for this purpose an elastic bandage is used. Presently procedure lasts from 10 to 20 minutes. At a necessity it is possible simultaneous balloon dilatation and/or introduction of vascular endoprosthesiss – stents, not «taking off a patient from a table». After the exam specialist demonstrates a patient the record of his coronarography, explains the degree of lesion of coronary vessels and recommends subsequent tactic of treatment.

Differential diagnostics. It is necessary to differentiate IHD with other diseases of heart: cardiomyopathy, acquired heart-diseases, myocarditis, pericarditis, disturbances of rhythm, tumor of heart; and also with the diseases of other organs and systems which can cause pain in area of heart: cholecystitis, hernia of the esophageal opening of diaphragm, acute pancreatitis; intercostal neuralgia, herpes zoster, osteochondrosis of thoracic department of spine; pleuropneumonia, and others.

Substantiation and formulation of clinical diagnosis of patient:

1) Basic: IHD, stable stenocardia, II functional class, atherosclerotic cardiosclerosis, insufficiency of circulation of blood of 0 stage.

2) Complication: atrial fibrillation, paroxism, tachysystolic form.

3) Concomitant: arterial hypertension of II stage

Treatments of patient with IHD. The choice of medical tactic in the patients with IHD depends on the form of disease, results of additional methods of examination, risk of operative treatment. The medical program in IHD has to provide:

1. Correction of risk factors.

2. Medicinal treatment.

3. Transcutaneous invasive interventions.

4. Surgical treatment. Indications to operative treatment (both minimally invasive and aorto-coronary by-pass) are based on information of coronography. Principles of the pathogenic substantiated conservative therapy

Treatment by antianginal medicines. Antianginal medicines are medical preparations which result in relative accordance of blood supply of myocardium to its demand, and stop or warn the attacks of stenocardia. There are following groups of antianginal medicines:

– Nitrates and close to them group of sidnonims;
– Blockers of β-adrenergic receptors and cordarone;
– Antagonists of calcium;
– Activators of the potassium channels.

Treatment of disaggregants. Preparations which inhibit specific receptors of platelet membrane to chemical mediators: ketanserin, nonapeptide, tripeptide, β-adrenoblockers. Blockers of freeing of intracellular calcium: verapamilum, nifedipin, diltiazem. Inhibitors of phospholipase: glucocorticoids, some anaesthetics, β-adrenoblockers (propranolol, atenolol). Activators of adenylatecyclase: prostaglandin E1, prostacyclin, nafazotrom, nicotine acid. Inhibitors of cyclooxygenase: aspirin and other nonsteroid preparations, Sulfinpyrazonum. Inhibitors of tromboxansynthase: dazoxiben, pimagrel. Inhibitors of tromboxansynthase and receptors of thromboxane: ridogrel, picotamide. Inhibitors of phosphodiestherase: papverinum, theophyllinum, dipiridamole, pentoxiphyllinum, trapedil. Preparations with the combines mechanism of action: ticlopidin. Preparations which influence lipid composition of platelets: essentiale, lipostabil. Polyunsaturated fat acids: eykozopentaenic acid (eykonol), dokozopentaenic acid. Correction of lipid composition of plasma of blood by antiatherosclerotic diet, hypolipidemic medicines, regimen of physical activity.

Psychopharmacologic influence: extract of valerian radix. Calming collection. Valocordinum

Corvalolum. Tranquilizers. Neuroleptics.

Physical therapy treatment:

1) electrotherapy (electrosleep, electromagnetic field of of 460 Mhz, low frequency variable magnetic field, sinus modulated currents (amplipulsetherapy), electrophoresis of medical preparations);

2) balneotherapy (general carbonate baths, bisulphid baths, radon baths, chloride-natrium iodum-brom baths, oxygen baths);

3) laser therapy;

4) ultrasonic therapy.

Surgical treatment of IHD and its complications

The primary purpose of surgical treatment of IHD is renewal of blood supply of ischemic myocardium (revascularization) in the case of resistance to medicinal therapy. The revascularization of myocardium is performed by the following operations (open with the use of pump oxygenator and without it, and minimally invasive interventions):

– Transluminal balloon angioplasty of coronary arteries;
– Mammaro-coronary by-pass;
– Aorto-coronary autovein by-pass;
– Laser angioplasty of coronary arteries;
– Intraluminal coronary atherectomy;
– Indirect revascularization of myocardium.

Transluminal balloon angioplasty and stenting

Endovascular methods of treatment are separate enormous section of treatment of IHD. In 1977 Grunzig offered a balloon catheter which was entered by puncture of general femoral artery in coronary vessels and at inflating dilates the lumen of the

narrowed areas of coronary arteries. This method, named TLBA, quickly got wide distribution in treatment of chronic IHD, unstable stenocardia, acute disturbance of coronary circulation of blood. The methods of endovascular treatment and surgery of IHD do not compete, but complement each other. The number of angioplasty with the use of stent in the economic developed countries increases steadily. Each of these methods has the indications and contra-indications. Coronaroangioplasty means balloon angioplasty and stenting of coronary arteries. Catheter with a balloon on its end is entered in the affected coronary artery at a balloon angioplasty in cath-lab, under X-ray control it is positioned in the location of narrowing of coronary artery and is inflated for 1–2 minutes. Thus, an atherosclerotic plaque is squashed, and the lumen of vessel is increased. At stenting after balloon inflation the metallic wireframe cylinder opens up with the special medical covering (stent) which supports the form of vessel lumen. Medical covering on the walls of stent serves as the prophylaxis of thrombosis in it. After insertion of stent the control coronaroangiography is needed in 4–6 months. This method of treatment is acceptable at the lesion of one or two arteries, at small atherosclerotic plaques. In the case of multiple and severe lesion of coronary arteries it is necessary to perform operative treatment. Advantages of TLBA:

1) more high efficiency by comparison to conservative treatment;

2) does not need general anaesthetizing and thoracotomy (for patients, at which by-pass is intolerable);

3) does not require the prolonged rehabilitation period. Disadvantages: the risk of development of dangerous for life complications during the balloon angioplasty and stenting makes about 2%. Mainly it is related to the damage of atherosclerotic plaque and development of acute myocardium infarction (AMI) as a result of blockage of coronary artery. In such complication the operation of aorto-coronary bypass is performed urgently on vital indications.

Indications to the choice of heart revascularization are determined by cardiosurgeon together with cardiologist and interventionalist. The basic problem of angioplasty is the recurrent narrowing of coronary artery – restenosis which can develop in a few weeks or months. Rate of restenosis is about 10%.

Stenting was developed to decrease the disadvantages of TLBA – mainly risk of development of AMI. A coronary stenting is a method of intravascular prosthesis of coronary arteries at various pathological changes of structure of their wall. For the reconstruction of coronary arteries stents are used. Stent is metallic wireframe which is a little metallic tube from a wire. A stent is inserted into the artery after its balloon expansion and positioned in the place of the lesion of artery with the purpose of prevention of restenosis. A stent supports the walls of artery. More than 60 different constructions of coronary stents are presently offered. Depending on a design stents are divided on:

– wire (made from one wire);
– tubular (made from cylindrical tube);
– rings (made from separate links);
– reticulated (as the wattled net).

Depending on a technique of insertion the self-expandable stents and stents which are expanded by a balloon-catheter are distinguished. This intervention is

performed, as angiography, by puncture of femoral artery. An operation is conducted under the local anesthesia. Through a puncture of femoral artery balloon with stent on the special guiding catheter is positioned in the location of narrowing of coronary artery. At inflating of balloon the lumen of artery is dilated and restored by stent. Heart obtains the necessary volume of blood through the restored artery which results in diminishing or disappearance of pains behind breastbone at the physical loadings.

Advantages of stenting: lasts a few longer than TLBA; the use of stent decrease by the necessity of the repeated operations; duration of hospitalization is shorter – 3-4 days; a stenting can be performed in patients, to which aorto-coronary bypass cannot be performed, but TLBA is not indicated; by comparison to TLBA diminishes the necessity of implementation of urgent aorto-coronary bypass; finally, less traumatic, than aorto-coronary bypass. Disadvantages of stenting: thrombosis of stent; restenosis in stent. There are three types of stenting:

1) bailout (saving, urgent) stenting – conducted at the acute blockage of coronary artery by the fragments of atherosclerotic plaque after TLBA in place of urgent aorto-coronary bypass;

2) planned (elective, primary) stenting – a stenting is conducted initially regardless of results of TLBA (at the various forms of IHD);

3) stenting on indications (provisional) – used on results an angiography after TLBA, when its results are not optimal, with the purpose of prevention of the acute blockage of coronary artery or development of its restenosis.

Mammarocoronary and aorto-coronary bypass (ACB)

Indications to the operation of ACB in asymptomatic patients or patients with the exertional stenocardia of I-II functional class are:

1. Reliable stenosis (> 50%) of trunk of the left coronary artery (LCA).

2. Equivalent of stenosis of trunk of LCA - > 70% stenosises of proximal part of anterior interventricular branch (AIVB) and circumflex branch (CB) of LCA.

3. Three-vascular lesion - indications are more increased at EF < 50%.

Indications for the operation of ACB in patients with the stable exertional stenocardia of III-IV functional class are:

1. Reliable stenosis (> 50%) of trunk of the left coronary artery.

2. Equivalent of stenosis of trunk of LCA - > 70%, lesion of proximal departments of AIVB and CB.

3. Three-vascular lesion (effect of operation is better for patients with EF < 50%).

4. Twovascular lesion with reliable proximal stenosis of AIVB and EF < 50% or with an obvious myocardial ischemia at noninvasive tests.

5. One- or twovascular lesion without proximal stenosis of AIVB, but with the large area of ischemic myocardium and symptoms of high risk of fatal complications, revealed at noninvasive tests.

6. Severe stenocardia which is preserved, despite of maximal therapy. If the symptoms of stenocardia are not fully typical, it is necessary to obtain other confirmations of severe myocardial ischemia.

Indications for ACB in patients with an unstable stenocardia and nonpenetraiting myocardium infarction are:

1. Reliable stenosis of trunk of LCA.
2. Equivalent of stenosis of trunk of LCA.
3. Presence of myocardial ischemia, in spite of maximal therapy.

Essence of operation of the aorto-coronary bypass consists of creation of shunts (by veins or arteries taken from patient) between intact parts of coronary arteries and aorta in the bypass of the affected areas (Fig. 16).

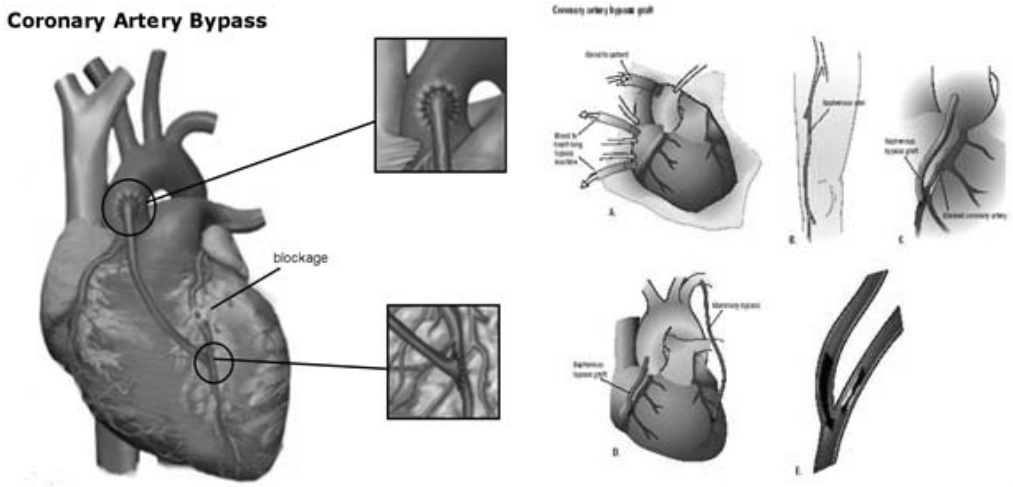

Fig. 16. Coronary artery bypass surgery

More often vein of patient as shunts is used. In a number of cases the internal thoracic artery as shunt is used. Such operations are named the mammaro-coronary by-pass. In some cases both methods are combined, such operation is named the mammaro-aorto-coronary bypass. Radial artery as a shunt also can be used, more often from the left arm. In the future, after taking of shunts from a leg or hand, a patient has no problems, as vessels which do not play principle role in life of man are taken as shunts. For diminishing of trauma of extremities during the dissection of saphenas, endoscopic technique is used presently, in most cases 3 small scars (about three centimeters) remain. The operation of the coronary by-pass is performed under general anesthesia. For access to the heart a thorax is opened through a breastbone. During the operation heart and breathing is stopped and pumping oxygenator is applied. It means that during an operation work of heart and lungs is performed by pumping oxygenator, which provides support of blood flow in an organism and oxygenation of blood and taking away of carbon dioxide. Sometimes during this stage the general cooling of organism can be used to 30 degrees (hypothermia), and also blood transfusion can be required. Simultaneously with access to the heart the dissection of saphena magna or the internal thoracic artery, sometimes there radial artery are performed. The next stage is putting on shunts between healthy part of vessel of heart and aorta. Shunts can be put on many vessels. Upon termination of imposition of shunts of heart activity is restored, pumping oxygenator is disconnected, an operation is finished. If before the operation, there was suspicion on the presence of heart aneurysm and it was confirmed during an operation, except the imposition of

shunts surgeons will delete aneurysm and will conduct the plasty of cardiac muscle. Patient in a condition of anesthesia from an operating-room is transported intensive care unit, where the experienced doctors and trained nurses observe him. In intensive care unit control after all of the systems of organism is performed by monitoring systems (arterial blood pressure, central venous pressure, ECG, pulse rate, oxygen saturation of blood and other indexes).

It should be noted that there is an alternative variant of operative treatment – the coronary by-pass without the cardiac arrest. In order to stabilize the certain area of heart and impose a shunt on this area, the special stabilizing systems are used. Such operations are more physiological, period of rehabilitation after such operations, as compared to operations with cardiac arrest, is shorter. However such operations require higher qualification of surgeon. Wide distribution of operations without the cardiac arrest hinders the fact that technically to perform such operation at the multiple lesions of vessels, especially if there is a lesion of vessels on the posterior surface of heart, is very difficult. Clearly, that at small incision, mini-incision it is more difficult to work with an internal thoracic artery on a large area. The special endoscopic instruments which are inserted into thoracic cavity successfully allow to solve this problem. 11 different methods of minimally invasive endoscopic coronary surgery are today developed with the use of mini-incision, that allows to shunt one, two and even three coronary arteries, and then from small incision to suture the end of this artery directly to the affected coronary vessel, restoring the circulation of blood in heart. Moreover, the method of endoscopic coronary surgery is developed that replaces mini-incisions by few small punctures. It is a new section of coronary surgery. In its basis is implementation of operations on a working heart without application pumping oxygenator and use of minimum access. Limited, 5 cm long, thoracotomy or partial sternotomy is performed in order to preserve stability of breastbone.

These operations are indicated for not young, hyposthenic patients which can not tolerate pumping oxygenator due to the presence of disease of kidneys or other paernchymatous organs. Minimally invasive operation can be performed on right coronary artery or two branches of the left coronary artery from the left or right access.

Transmyocardial laser revascularization

Transmyocardial laser revascularization is performed from thoracotomy access - lateral thoracotomy without connecting to by-pass machine. In area of myocardium with the low level of blood supply the great number of the point ducts is done through which blood enters to ischemic area of myocardium. These operations can be performed both independently and in combination with by-pass of coronary arteries. Positive results obtained in the large group of the operated patients allow to consider a method close to the direct revascularization of myocardium.

Postoperative care.

Transluminal balloon angioplasty and stenting in the uncomplicated flow does not need the prolonged hospitalization. As a rule, a patient is discharged already on the third day after intervention with the proper recommendations regarding the prophylaxis of thrombotic complications. It is necessary in a nearest period to pay attention to possibility of development of AMI and in more late - to restenosis of coronary artery (Fig. 19, 20).

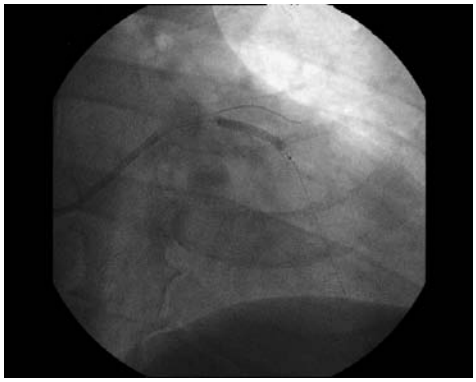

Fig. 17. Balloon dilation of the anterior interventricular branch of the left coronary artery (LCA)

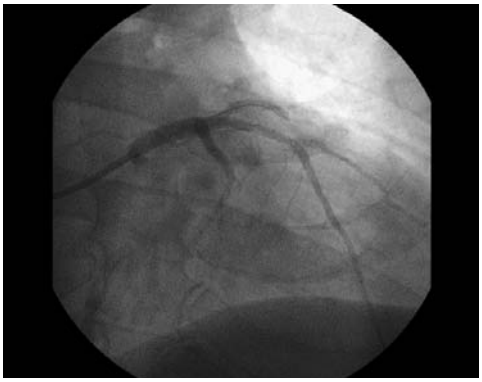

Fig. 18. Standing stent in the anterior interventricular branch dilated LCA

Bypass operations on coronary arteries need all general principles of postoperative care of patients operated on a heart: monitoring of the vitally important systems and correction of their violations, infusion therapy, antibiotic prophylaxis, anticoagulant therapy, anaesthetizing, symptomatic treatment.

Treatment of AMI

For all of patients with confirmed or supposed AMI central venous catheter is set; solution of D5W is usually used; it is better not to apply salt solutions in order to avoid an overload and sodium congestion in lungs. Central venous pressure at AMI can not serve as the reliable index of volume status or cardiac insufficiency. The cardiac monitoring is performed by skilled personnel. All of patients are provided with additional oxygen. To the patients with an increased sensitivity to oxygen in anamnesis or with the chronic obstructive disease of lungs it is necessary to give low concentrations (2 L/min, or 24%), and for others – higher concentrations (4-6 L/min, or 40%). A severe hypoxia or hypercapnia often requires the intratracheal intubation and artificial ventilation; it is better to apply a volume-cyclic respirator, as a change of pulmonary tissue compliance can hinder functioning of respirator, managed by pressure. It is necessary to liquidate present violations of acid-base balance, as they play a certain role in the development of arrhythmia.

Because of danger of diagnosis of AMI many doctors make hyperdiagnostics of myocardial ischemia and "pulmonary edema" in hospital. Practice of many clinics foresees hospitalization in the intensive care unit of all patients with retrosternal pain (for the "exclusion of myocardium infarction"). Recent studies showed that some groups of patients rarely needed (or not need) measures, performed in intensive care unit; an in-hospital mortality in these patients is very small. Patients with AMI or unstable stenocardia, with permanent pain, changes on ECG, arrhythmias or hypodynamic complications, often require intensive care, and their stay in the proper department is fully justified. Patients with an unstable stenocardia and normal ECG without arrhythmias and hypodynamic complications can be hospitalized in cardiologic department; it is possible to carry out monitoring in them, being not afraid of increase in-hospital mortality. Patients with normal ECG, admitted for the "exclusion of heart myocardium infarction", can be hospitalized in ordinary depart-

ment. Patients with an unstable stenocardia after stabilizing of hemodynamics and relief of ischemic pain during 24 hours also can be transferred from the department of intensive care to an ordinary chamber. The same for patients with relatively non-severe myocardium infarction, who during 24 hours no longer feel ischemic pain.

It is considered that optimum heart rate on the early stage of AMI must be within the limits of 60-90 beats per minute. Many doctors recommend prehospital (or in an ambulance) prophylactic injection of lidocaine to all patients with suspicion on AMI. However expedience of this prophylactic injection remains unproven.

It is necessary in admission department as early as possible to relieve pain. Traditionally intravenous introduction of morphine sulfate is used. It is needed consistently to enter its small doses (4-6 milligrams each 10-15 minutes), however for the complete pain relief it is needed full dose of morphine (15-20 milligrams). For patients with the severe hypotension (an arteriotony less than 80 mm of Hg) or with the chronic obstructive disease of lungs it is necessary to apply morphine with a carefulness. At AMI the favorable effect of morphine is conditioned mainly by its sedative and analgesic action which results in the decline of oxygen demand of myocardium. Morphine does not render permanent influence on pre-loading and capable even to reduce the minute volume of heart. An analogical effect has meperidine hydrochloride, which also can be used. Pentazocinum increases pre- and post-loading, reduces resistance and increases the oxygen demand of myocardium.

It was considered that in the patients with AMI it is not necessary to apply nitroglycerine in order to avoid a hypotension and tachycardia. However recent studies showed efficiency and safety of sublingual or intravenous application of nitroglycerine in AMI. In addition, nitroglycerine diminishes the degree of ischemia and, probably, size of area of myocardium infarction, reducing the in-hospital death rate in AMI. It is necessary repeatedly to give small doses (0,4-1,6 milligrams) under a tongue with 3-5-minute intervals all the time while pressure is preserved at sufficient level (for example, more than 100 mm of Hg at most patients or more than 120 mm of Hg in patients with arterial hypertension). A reaction on preparation in different patients varies considerably; sometimes for complete relief of pains the full dose of nitroglycerine is needed (20-30 milligrams). Initial rate of intravenous infusion of nitroglycerine can be 10 mcg/min; then it is increased until pain will not disappear or while an arterial pressure will not go down approximately on 10%; for most patients rate of infusion is from 50 to 100 mcg/min. Sometimes the patients with AMI have the so-called vasovagal reaction on nitroglycerine which is presented by bradycardia and hypotension. Usually it is transitory and stopped after the simple raising of leg edge of bed and, if necessary, atropine.

Presently there is a number of methods of treatment, directed on the removal of causal factor of acute myocardium infarction or on limitation of area of myocardium infarction. Majority of them were developed only recently and passes clinical tests with the purpose of more exact determination of indications, and also assessment of their advantages and possible risks.

Blood clot which appeared acutely in coronary arteries it is possible to dissolve by thrombolytic preparation, entered within 2-4 hours after onset of symptoms. Indexes of perfusion renewal (that confirms dissolution of blood clot) varies depending on used preparation, its dose and way of introduction. All conducted studies showed

that at intracoronary introduction of thrombolytic preparation rate of perfusion renewal is 60-90%, and at its intravenous introduction - 35-83% depending on a dose and used preparation (streptokinase or activator of tissue plasminogen). Analysis of all information, obtained in early extensive studies with intravenous introduction of streptokinase, showed 10-20% decline of the nearest death rate during the thrombolytic therapy. The information in relation to intracoronary introduction of streptokinase shows that successful renewal of perfusion reduces the nearest death rate more than on 80%. However severe complication of intravenous thrombolytic therapy is bleeding. Contra-indications for thrombolytic therapy are: recent operation; recent stroke; peptic ulcer or bleeding from GIT; pregnancy or post-delivery condition; coagulopathy; condition after pneumocardial resuscitation; trauma. In patients with cardiogenic shock such therapy cannot give an effect.

Disappearance of both pains and ECG-signs of ischemia confirms the successful renewal of perfusion. Quite often temporal renewal of perfusion is accompanied with ventricular arrhythmia (extrasystole, acceleration of idioventricular rhythm) or AV-block. After acute thrombolytic therapy intravenous introduction of heparin (for prevention of thrombosis) and, possibly, angiographic exam is performed (for complete evaluation and treatment of the initial atherosclerotic occlusions).

In some centers at AMI urgent angioplasty is used. If treatment is conducted within the limits of 4 hours after onset of symptoms, an urgent angioplasty in experienced hands gives the same rate of renewal of perfusion as intracoronary introduction of streptokinase (more than 80%). But, as recent studies show, an angioplasty is more effective in the removal of stenosis of coronary artery and able to result in the greater maintainance of function of LV. In addition, in some centers urgent by-pass of coronary arteries is performed successfully in AMI.

Treatment of complete atrioventricular blockade

It is necessary:

1) To enter preparations which diminish vagal influences (cholinolytics): atropine 0,1%-1,0 i/v; Platyphyllinum 0,2%-1,0 s/c; if i/v, on 500 ml of 5% glucose.

2) To increase sympathic influences on the conducting system: Noradrenalinum 0,2%-1,0 i/v on glucose; ephedrine 5%-1,0 i/m, s/c, i/v; alupent 0,05%-0,5-1,0 i/m or i/v; isadrine of 0,1%-1 ml.

3) Glucocorticoids: hydrocortizone 200 mg/day. Relieve inflammation, edema. Diminishes contents of potassium in the area of transmission of impulse through the damaged area. Potentiate sympathic influences. Entered repeatedly through a few hours.

4) To decrease maintenance of potassium: Lasixum 1%-1,0 i/v.

5) If above measures appear ineffective or there is complete block or Mobitz II in combination with the blockade of the left crus of His bundle, temporal pacing is indicated (an electrode is entered by a probe or catheter in a right ventricle). If patient has anterior myocardium infarction, a complete block can develop quickly, so it is also indication for the pacing. If the flow of disease was complicated by the syndrome of Morgan'I-Adams-Stox — a prompt medical assistance is needed: few shots on breastbone by fist as mechanical irritation of heart, indirect massage of heart (60 per minute) + "mouth-to-mouth" ventilation – 14 per minute. ECG is needed for determination of character of disturbance of cardiac activity. If it is in-

effective intracardiac adrenalin or noradrenalinum injection is indicated, the discharge of electric current is repeatedly performed then; it is sometimes necessary 10 and more discharges. If there is an asystole of ventricles, intracardiac injection of calcium chloride 10%-5,0 and Noradrenalinum is recommended and on this background to conduct an electrostimulation. A needle is used as an electrode, which is entered in myocardium; impulses are conducted through it.

Complications of IHD

Sick sinus syndrome

Sick sinus syndrome *(SSS) is dysfunction of sinus node which is presented by bradycardia and arrhythmias.*

Syndrome consists in diminishing of heart rate due to disturbance of forming of impulse in sinus node, or disturbance of its conduction to the atrium. Thus, a syndrome includes sinus bradycardia both actually and sinoatrial blockade of the II degree. Symptoms and terms of development of these conditions are similar, that is why they usually are not divided. Bradycardia at SSS is often accompanied by arrhythmias, which develop as a result of activating of sublaying departments of the conducting system of heart which stop to be controlled by the impulses of sinus node.

Reasons of disturbance of rhythm activity of sinus node can be divided into primary, conditioned by the organic lesion of node, and secondary, caused by extracardial processes.

Primary lesion of SN is more frequent in IHD, arterial hypertension, heart defects, myocarditis, hemochromatosis, heart operations, especially with the use of by-pass machine. SSS develops at the myocardium infarction in 5% cases, more frequent at the myocardium infarction of posterior wall, that is supplied, as well as SN, by right coronary artery.

To the secondary causes of disturbance of activity of SN attribute:

Increase of activity of vagus or sensitivity to its influence, which can develop in sportsmen, and also at the diseases of larynx, esophagus, increased intracranial pressure, emotions such as fear, pain.

Disturbance of hemostasis: increase of level of K, Ca in blood, mechanical icterus, hypothyreosis, anaemia.

Application of medications which decrease heart rate: β-blockers, blockers of the calcium ducts, cardiac glycosides.

The clinical symptoms of SSS develop as a result of decrease of minute volume of heart which develops at severe bradycardia. As a cerebrum is the most sensible to the hypoxia organ and reacts first on it, the clinic of acutely developed bradycardia and asystole is close to the symptoms of cerebral ischemia as dizzinesses, faint, up to development of syndrome of Morgani - Adams - Stox (unconscious condition with cramps), that often leads to death. The severity of these symptoms depends on the initial level of cerebral blood supply. In some patients any symptoms were absent at complete asystole for 15 seconds and light dizziness appeared only at asystole that lasted 30 seconds. At prolonged permanent bradycardia it is possible development of coronary insufficiency, oligouria, activation of ectopic foci of rhythm.

Additional exams:

Daily monitoring of ECG (especially when revealed both clinical signs and signs of sinus bradycardia, but there is no connection with AMI).

Rithmography with an orthostatic test (graphic visualization of intervals of RR as touches of different length).

Massage of carotid sinus, test of Valsalv.

Test with the physical loading (at SSS heart rate rises moderately, up to 60-70 beats per minute)

Measuring of time of renewal of the function of sinus node, which shows time, necessary for renewal of its own rhythm after stopping of frequent stimulation of aurium. At healthy this time is 1,2-1,49 seconds, and at SSS it can be increased to 3-5 seconds.

Treatment in absence of clinical symptoms is possible to limit to therapy of basic disease which gives good results sometimes, for example antiinflammatory therapy at myocarditis.

At the well-proven association of clinical symptoms with bradycardia, lengthening of time of renewal of the function of sinus node to 3-5 seconds, development of chronic cardiac insufficiency, refractory supraventricular tachycardias setting of cardiostimulator which works in the mode "on demand", that generates impulses only at falling of heart rate to the critical level, is indicated.

Medical therapy is ineffective. At the hyperactivity of vagus and refuse of patient from a cardiostimulation euphyllinum is applied 0,45-0,9 g/day, apressin 50-150 mg/day. At complication of bradycardia with atrium fibrillation, intravenously enter cardiac glycosieds which control the attack, or, control even heart rate. In this case to apply verapamilum and obsidane is impossible, as they result in further suppressing of sinus node.

Atrioventricular blockade

Atrioventricular blockade is the disturbance of transmission of impulse from an atrium to the ventricles, including atrioventricular bundle.

There is a transversal blockade (conductivity is disturbed due to the lesion of Ashof-Tavar node and general part of His bundle) and longitudinal blockade (consequence of blockage of one of the crura of His bundle). Also partial (incomplete) and complete atrioventricular blockades are distinguished.

Other name is atrioventricular dissociation. AV node consists of three departments:

1) actually AV node;

2) His bundle;

3) crura of His bundle.

Delay or stopping of conduction of impulses from an atrium to the ventricles as a result of lesion of one of three of afore-named levels causes AVB. Thus, the lower lesion, the more severe clinical symptoms, the more unfavorable prognosis. If a damage takes place at the level of dividing of crura of His bundle, a complex QRS does not change on ECG; if below — distension or change of complex takes place, as in the blockade of crus.

3 degrees of blockade are distinguished:

1 degree. Delay of atrioventricular conductivity. All of impulses come to the ventricles, but rate of their conduction is decreased. Subjective symptoms are not present, diagnostics only on ECG: a rhythm is correct, but the interval PQ is enlarged (normally no more than 0,2 seconds). Duration of interval is different. At the very long interval PQ it is sometimes possible to hear the separate rhythm of atrium.

Etiology:

a) quite often functional disturbances (vagotony of sportsmen);

b) organic – inflammatory processes in myocardium, cicatrical changes of atrioventricular node;

c) electrolyte disturbances.

More often than other are "a" and "b". Vagotony of sportsmen it is difficult to differentiate, for this purpose a test with an atropine is applied: at vagotony after its application characteristic changes disappear on ECG.

2 degree. Not all impulses reach ventricles, ventricles are contracted under act of separate impulses (unlike 3 degrees). Distinguish 2 types of blockade of 2nd degree:

1) Periods of Venkenbakh-Samoylov (Mobitz I) — during the conduction of impulses the interval PQ is gradually prolonged to the complete falling of pulse wave. Usually at this type damage is comparatively high, that is why QRS is not changed. Prognosis of this type is comparatively favorable.

2) Type of Mobitz II with the permanent interval of PQ, here not all of impulses come to the ventricles - every second impulse is conducted in one case, in other - every third et cetera. The more lower impulsive conductivity, the more severe clinic. There is low lesion in this pathology, that's why a complex QRS changes. Quite often Mobitz II is the precursor of complete transversal blockade. Clinical symptoms: slow pulse, slow rhythm of ventricles. Prognosis unfavorable. Often meets at the anterior myocardium infarction (Fig. 19).

3 degree: complete transversal blockade. Thus the conduction of impulses is fully absent to the ventricles, the heterotopic focus of idioventricular rhythm appears in ventricles, thus the lower automatism, the more severe clinic. There is complete dissociation: the rhythm of atriums is normal, and ventricles contracts at frequency 40 per minute and less. The last depends on the level of damage: if AV node is involved - 40-50 per minute, if crus of His bundle - 20 per minute and less. Pprognosis depends on basic disease and level of damage. More often complete transversal block occurs at severe organic lesion (luis and other). Stroke volume of heart is increased, high systolic pressure, diastolic pressure is low or normal, pulse pressure increases. Ventricles have a large pause of diastole, they are strongly overfilled with blood in a diastole, that is why there is their dilatation and hypertrophy. A pulse is slow. The sizes of heart are increased, mainly - to the left, sometimes systolic noise of relative insufficiency of mitral valve (due to dilatation) is found. Heart sounds are weakened, periodically the "gun" sound appears during I tone - when the systoles of atrium and ventricles almost synchronize. There can be III additional tone. Systole noise of banishment can appear on the basis of heart. The pulsation of veins, related to reduction of atria, especially expressed in Strazhesko gun tone, is often revealed. There can be severe complications:

а) making progress cardiac insufficiency, especially at the physical loading, related to low heart rate;

б) syndrome of Morgani-Adams-Stox — often arises up in transition of incomplete blockade in complete and at progress of disturbances of AV-conductivity. Thus own automatism did not yet have time to be formed, blood does not goes to periphery, and a sensible cerebrum answers by the loss of consciousness. In basis of syndrome is of stopping of blood delivery, ischemia. In one cases it is related to the asystole of ventricles on background of complete blockade, in other with fibrillation of ventricles. Regardless of reason, the end-point is the same - loss of consciousness.

Clinic. Sudden pallor, loss of consciousness, a pulse is not determined, heart sounds are not audible. A patient is cyanotic, cramps appear. There can be involuntary urination and defecation. Death is possible in 3-4 minutes, but often attack finishes in 1-2 minutes: the idioventricular pacemaker of ventricles is activated.

There are several phases of AV blockade of 3degree:

A — permanent form;

B — episodic (intermittent), more often causes the syndrome of Morgani-Adams-Stox. Prognosis is the most favorable in this form. A blockade is both complete or incomplete.

Diagnostics.

Clinically: correct slow pulse (rhythm). There is complete dissociation on ECG: there is own rhythm in atria, and more slow own rhythm in ventricles. More lower block, the deformation of QRS is more expressed.

Treatment.

At the occurrence of blockade, especially in not young man, obligatory hospitalization, especially at the syndrome of Morgani-Adams-Stox and its equivalents.

At the chronic flow of blockade treatment of basic disease is important. During medication intoxication their abolition is needed. At inflammatory diseases the special treatment is also needed.

Electrostimulation — the artificial heterotopic pacemaker is created (Fig. 19).

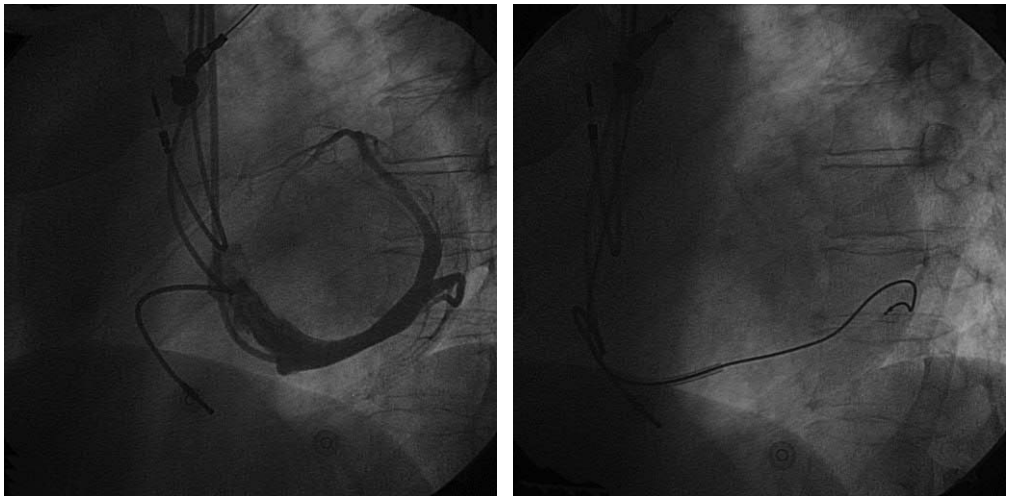

Fig. 19. The left ventricular electrode implantation via the coronary sinus

Indications to the electrostimulation:

a) all of blockades which are accompanied with the syndrome of Morgani-Adams-Stox;

b) insufficiency of blood circulation due to blockade;

c) heart rate less than 40 in minute

d) expressed SSS with severe paroxysms, rather than just bradycardia.

There are different types of electrostimulation - external and internal, permanent and temporal. There are two types of stimulators:

1) Pace-make: cardiostimulator of permanent action, works regardless of own heart rhythm.

2) De-kampe - physiologically more advantageous, as gives impulses only in case that the interval of R-R becomes longer than was set.

In the patients in which a slow pulse alternates with tachycardia, in addition, the reception of medicinal preparations can be needed.

Often an optimum effect is obtained at combination of cardiostimulator and reception of medications which slow the rhythm of heart, for example β-adrenoblockers or verapamilum.

7. Principles of examination of disability and follow-up of patients with IHD depend on the form of IHD, functional class, presence of complications of IHD and character of treatment. The patients with IHD need permanent follow-up, conduction of prophylactic measures, permanent reception of number of medicinal preparations, especially disaggregates.

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II.3. Modern methods of diagnosis, mini-invasive methods of surgical treatment of heart diseases

Actuality is dictated by high rate of diseases of heart, their progressive increase during the last decades, from one side, and also by appearance of new possibilities of treatment of such patients. To date diseases of the cardio-vascular system is principal reason of death rate in population. A timely ervealing, diagnostics of diseases of heart is the extraordinarily important factor of successful treatment of patients. From other side, introduction of minimally invasive treatment of diseases of heart improves the results of treatment considerably, accelerates the rehabilitation period of patients.

Modern methods of diagnostics of diseases of heart

To date in diagnostics of diseases of heart are widely used non-invasive methods, including electro-physiology, and invasive methods which directly provide the complete picture of lesion of heart. They are used from more simple screenung methods to the complex difficult and invasive methods of examination.

Noninvasive methods of diagnostics of diseases of heart

Cardiomarkers

Historically for diagnostics of acute coronary death determination of activity of one of enzymes, which is synthesized in our organism, was used (and continues to be used) – creatininephosphokinase (CFK). More precisely, one of its constituents — MB-fraction of creatininekinase (CFK-MB). Increase of activity of CFK-MB is the most specific for AMI. The increase of activity of CFK-MB begins already in 4-8 hours after the onset of attack and reaches maximum in 12-24 hours; on the third day activity of this fraction goes back to normal. At expansion of area of myocardial ischemia, activity of CFK-MB remains high far longer, that allows to diagnose AMI in the further terms of supervision.

However an increase of fraction of CFK-MB is determined also in unsurgical diagnostic manipulations on heart. Radio-therapy of thorax can cause the insignificant increase of level of enzyme also. However arrhythmia, tachycardia and cardiac insufficiency, practically does not influence the level of CFK-MB.

Valuable contribution to the decision of all of afore-mentioned questions was introduction to clinical practice the determination of serum myocardium markers — troponins I and T – absolutely specific and highly sensitive for diagnostics of acute coronary death (OCD). The presently leading cardiologic societies of Europe and USA accepted recommendations in which determination of concentration of troponin T (in serun, plasma) of blood is examined as a major diagnostic criterion of the AMI.

Reliable cardiomarker is cerebral natriuretic peptid (NTproBNP). Reliably and evidential grounded, that the exposure of its increased concentrations confirms the presence in the patient of cardiac insufficiency (CI). Therefore determination of level of this peptid already became a standard in a diagnostics of CI in the whole world. It is impossible not to mark that it was rotined in a number of large international researches, that application in clinical practice of tests of "NT-proBNP" and "BNP" allows to reduce the cost of inspection of patients and exactly enough to foresee a presence or absence of SN, even to the leadthrough of echocardiographies. A fact is yet important: presently a test found a wideuse not only at kardiovaskulyar pathology but also at some clinical conditions which are accompanied the decline of contractive ability of cardiac muscle.

Electrocardiography

Electrocardiography is used for:

a) diagnostics of hypertrophy of myocardium;
b) diagnosticians of disturbance of rhythm of heart;
c) diagnostics of violations of coronary circulation of blood;
d) conduction of ECG at implementation of stress-tests.

12 leads – 3 standard, 3 intensified unipolar and 6 thoracic are used in practice. Precardial cartography (35 electrodes are set in 5 horizontal rows) is used for clarification of location of heart changes, size of post-infarction and necrotic area, study of their dynamics. In addition, ECG can be registered by the esophageal lead.

Daily (Holter) monitoring of ECG is conducted in patients in which stress-tests are contra-indicated (physical loading, pharmacological tests, test of loading with a transesophageal electrostimulation).

Phonocardiography

Phonocardiography is a method of graphic registration of sounds and noises of heart. Most often the method is used in diagnostics of congenital and acquired heart-defects. Normal PCG consists of I and II sounds, between which there is straight line which corresponds to systole and diastole pauses. During a diastole pause III and IV sounds are sometimes registered.

Echocardiography

Echocardiography is a method, which is based on registration of reflected ultrasonic signals from heart structures, which move. It is used for:

a) diagnosticians of violations of morphology and mechanical activity of heart;

b) monitoring of activity of heart;

c) control of minimally invasive endocardiac interventions (fibrillation ablation).

Echocardiography allows:

1. To identify the valves of heart, their relative position.

2. To recognize interatrial and interventricular septa, to analyze their continuity, assess the type of motion.

3. To estimate anatomic relative position of valves of heart and interventricular septum.

4. To evaluate motion of leaves of valves.

5. To conduct measurings and define the changes of thickness of walls and sizes of chambers of heart for determination of presence and severity of dilatation of cavities of heart and hypertrophy of myocardium of the left and right ventricles.

6. To conduct Doppler echocardiography together with two-dimensional echocardiography, for the exposure of symptoms of valvular regurgitation, narrowing on the way of blood flow and endocardiac shunts. Stress-echocardiography is used for replacement of ECG stress-tests, more often with the physical loading. Contrast echocardiography is also used (contrast is entered intravenously). Except transthoracic echocardiography, transesophageal echocardiography (including three-dimensional) and endocardiac echocardiography are used.

X-ray of heart

X-ray of thorax – allows to evaluate shade of heart, reveal its increase or increase of separate chambers of heart, evaluate the lesser circle of blood circulation. With appearance of the echocardiography value of X-ray diminished.

X-ray ventriculography – allows to evaluate the volume of ventricles, ejection fraction, systole and diastole functions.

Computer tomography

Computer tomography is method with the use of x-ray for visualization of chambers of heart, pericardium, presence of blood clots in the cavity of heart, tumors. Lately ordinary CT was fully replaced by spiral CT or MRI.

Electron beam computer tomography

Electron beam computer tomography is new technology of CT-scan, which allowed considerably to shorten examination time by comparison to ordinary CT. Mainly it is used for diagnostics of proximal occlusions of coronary arteries, anomalies of origin of coronary vessels, and also their aneurysm. Disadvantage – difficulties with visualization of distal stenosises of coronary arteries.

Spiral tomography

Multispiral computer tomography (MSCT) for diagnostics of atherosclerotic lesion of vessels began to be used from 90th of the last century. Visualization of coronary arteries was impossible at the beginning, because of low resolution and high percentage of artifacts of motion, that is why the atherosclerotic lesions of arteries were evaluated by the count of intravascular calcium content. The quantitative estimation of coronary calcinosis is based on the coefficient of roentgen absorption and area of calcinates. Calcium index (CI) on the Agatston method is determined as product of area of the calcinated lesion on the factor of density. It was marked that CI represents the prognosis of lesion of the cardio-vascular system and directly correlates with the rate of development of atherosclerosis: the higher index, the higher risk of atherosclerotic lesion. For example, at low CI from 10 units and below - probability of atherosclerosis of coronary arteries makes no more than 5-10%. From 11 to 100 units (moderate CI), possibility of presence of 50% narrowing - no more than 20%, at CI 101-400 units – 75%, that is moderately high risk of atherosclerosis. And at high CI, more than 400 units – probability of atherosclerotic lesions of coronary arteries is about 90%. CI is predictor of development of future cardio-vascular catastrophes.

With development of 4th in 1999, and then 8th spiral computer tomographs possibility of diagnostics of not only static objects with the assessment of indirect signs of atherosclerotic lesion but also direct visualization of the condition of coronary flow appeared in 2001. However only on the threshold of 2005, non-invasive 64-MSCT appeared, that allow sufficiently quickly to obtain an image, with the reconstruction of volume image less than 0,5x0,5x0,6 mm.

MSCT is used in diagnostics of the cardio-vascular system at:

1. Ischemic heart disease (IHD).
2. Diseases of aorta (coarctations, aneurysm, dissection and others).
3. Lesions of peripheral arteries (obliterative atherosclerosis of arteries of lower extremities, atherosclerosis of carotids and others).
4. Myocarditis.
5. Pericarditis.
6. Infectious endocarditis.
7. Pulmonary embolism.
8. Congenital anomalies of development of the cardio-vascular system.
9. Acquired heart defects (for example, calcinosis of aortic valve with development of stenosis or insufficiency and others).
10. Arrhythmias.

Coronary arteries of heart

High ability of 64-MSCT allows:

a) definite visualization of coronary arteries, with clarification of location of atherosclerotic lesions, exposures of anomalies of development of coronary vessels of heart;

b) to determine the condition of aorto-coronary shunts and intravascular endoprosthesiss (stents); c) to conduct the count of calcium index with the purpose of clarification of prognosis of disease;

d) to mark violation of perfusion and viability of myocardium for patients in the early and late terms of myocardium infarction;

e) to assess contractive ability of heart;

f) to study the condition of pericardium, valves of heart.

Visualization of atherosclerotic lesion of coronary arteries by MSCT is the alternative of invasive coronaroangiography (CAG) and used for both well-proven IHD, at suspicion on IHD and asymptomatic patients with the purpose of diagnostics, exposures of risk groups and determination of AMI, subsequent prognosis.

In particular indications for the MSCT are:

1) atypical pain in thorax;

2) presence of risk factors:

– arterial hypertension;
– lipidemia;
– obesity;
– diabetes mellitus;
– smoking;
– high CI;
– family anamnesis of coronary arteries, sudden death, lesion of peripheral arteries;

3) acute coronary syndromes, myocardium infarctions without elevation of ST for the estimation of lesion of coronary arteries of heart. MSCT allows to diagnose possible complications at the myocardium infarction, for example, rupture of interventricular septum, and also development of aneurysm of the left ventricle.

MSCT angiography is based on the technique of scan during an arterial phase during the rapid passage of contrasting matter.

The special preparation of patient to exam is not required. MSCT is performed in an out-patient order. A patient is in supine position. After preliminary scan necessary for exact determination of location of organs in the inspected area, intravenously the bolus of iodinated contrast is injected, then with a small delay a scan begins under control of ECG during 10 seconds, than findings are analyzed by computer. Complete time of exam of patient occupies about 15-20 minutes. Final information than is available for an analysis and construction of the 4-dimensional images.

The method has: high diagnostic value, relative simplicity of implementation, speed of receipt of information, comfort for a patient (absence of necessity for premedication), and also that MSCT is the first non-invasive technique of visualization of coronary arteries, which does not require hospitalization, without the risk of intra- and postprocedure complications. The last results confirmed the identity of the obtained results of MSCT and coronarography in diagnostics of atherosclerosis of coronary arteries. Thus, modern 64-MSCT is not inferior to the invasive methods which are a «gold standard»: angiography, having before selective angiography the number of advantages: relative simplicity of implementation of diagnostic procedure, absence of possible intra- and postprocedure complications; speed of exam and receipt of information; absence of necessity for hospitalization; no premedication.

Besides mentioned advantages MSCT gives additional description of atherosclerotic plaques (exposure of “soft” plaques, degrees of calcinosis and others), determines the function of systole of heart (according to the indexes of volumes of dias-

tole and systole of the left and right ventricle, the exact count of ejection fraction), reveals the areas of dyskinesia of myocardium of the left ventricle (at the construction of the 4-dimensional volume image), with the additional evaluation of anatomy of heart and vessels. And, that is important for a prognosis, MSCT has possibility to determine perfusional ability and assess viability of myocardium, it is especially important in patients with old myocardium infarction and patients with cardiac insufficiency.

Magnetically-resonance imaging (tomography)

Magnetically-resonance imaging is considered a "gold standard" in diagnostics of global and regional function of the left ventricle. MRI-coronaroangiography has number of limitations: it diagnoses only hemodynamically significant stenosises of coronary arteries (more than 50%) and visualizes only proximal and middle departments of coronary arteries. New direction of MRI is catheter-associated MRI-visualization. It combines catheter angiography and MRI. It allows to evaluate the wall of vessel on the whole, visualize an unstable atherosclerotic plaque, define speed of blood flow, assess the functional condition of surrounding tissues.

Isotope methods

Isotope methods are based on introduction intravenously of albumin or red corpuscles with a radio-active mark (thallium-201 or technecium-99m). These methods allow to specify contractive function and blood supply of myocardium, find out an ischemia and foci of necrosis of myocardium, cicatrical areas.

Invasive methods of research

Cannulation of heart and main vessels

Cannulation of heart and main vessels – a method allows to define the basic indexes of hemodynamics: pressure and saturation of blood oxygen in the different departments of heart and vessels, stroke volume and cardiac ejections, vascular resistance in the lesser and great circles of blood circulation, localization, size and direction of pathological blood flow, presence and degree of valvular regurgitation, size of the valvular openings, pressure in the cavities of heart and vessels (Fig. 20).

Angiography

A coronary angiography is a method, which allows to reveal presence, severity, location and prevalence of coronary stenosis, and also to find out their cause (atherosclerosis, blood clot, spasm). Coronary angiography is considered "golden standard" in diagnostics of coronary atherosclerosis. It belongs to the selective angiography, conducted by the special catheters on S. Seldinger (1953), as a rule, by retrograde access through a femoral artery. Indications: severe stable stenocardia (class 3 on classification of Canadian caedio-vascular society); stable stenocardia (class 1 or 2), if a patient have suffered the myocardium infarction or there are ischemic episodes at the little physical loading; at a stable stenocardia for patients with the blockade of His bundle crus, if the areas of ischemia are found at scintigraphy; in patients which have indications to the operations on

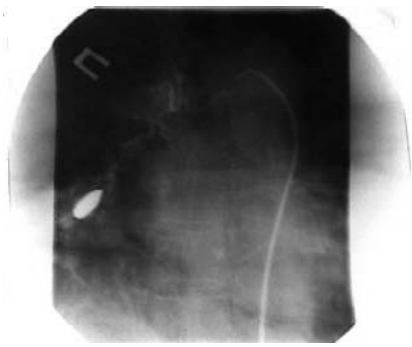


Fig. 22

coronary vessels; at severe ventricular arrhythmias; at the recurrence of moderate or severe stenocardia in patients in which revascularization of myocardium wasconducted (cathether angioplasty or aorto-coronary by-pass).

Coronarography allow to define a volume and tactic of intervention. Thus, without coronarography it is impossible to conduct any operative intervention, coronary angioplasty with a stenting or aorto-coronary by-pass.

Coronarography is invasive procedure, related to the certain risk! During the Coronarography complications are possible, but the risk of development of these complications is very small. However bleeding in the place of puncture, allergic reaction on contrast, disturbance of heart rhythm, myocardium infarction can develop. Before coronarography, a patient must pass some examinations: general analysis of blood, blood group, Rh-factor, tests on the viruses of hepatitis C and B, HIV, RW, ECG in a 12 leads, echocardiography, and also to purchase an elastic bandage.

Technique: under local anesthesia (during procedure a patient is in consciousness) puncture of artery is performed (thigh, axillar, radial). In an artery a small hollow tube (introducer) of diameter less than 2 mm is entered. Through a tube a catheter is introduced to the coronary arteries of heart. Through catheter contrast is entered and under X-ray control of the special angiograph an exam record is performed. After implementation of coronarography control of bleeding in the place of puncture is carried out, for this purpose an elastic bandage is used. Presently procedure lasts from 10 to 20 minutes. At a necessity it is possible simultaneous balloon dilatation and/or introduction of vascular endoprosthesiss – stents, not «taking off a patient from a table». After the exam specialist demonstrates a patient the record of his coronarography, explains the degree of lesion of coronary vessels and recommends subsequent tactic of treatment.

Minimally invasive methods of treatment of diseases of heart

To the minimally invasive methods of treatment of diseases of heart belong:

- transluminal balloon angioplasty (TLBA) and stenting of coronary arteries; thoracoscopic mammarocoronary by-pass;
- aortocoronary by-pass on a working heart – at IHD; ligation of patent ductus arteriosus by thoracoscopic approach;
- implantation of cardiostimulator and ablation of arrhythmogenic foci at arrhythmias.

A basic indication for operative treatment of IHD is stenosis of coronary arteries on 70% and more. The methods of myocardium revascularization are used:

- TLBA and stenting of coronary arteries;
- mammarocoronary by-pass (MCBP);
- aortocoronary by-pass (ACBP) and their combinations.

Transluminal balloon angioplasty and stenting

Endovascular methods of treatment are separate enormous section of treatment of IHD. In 1977 Grunzig offered a balloon catheter which was entered by puncture of general femoral artery in coronary vessels and at inflating dilates the lumen of the narrowed areas of coronary arteries. This method, named TLBA, quickly got wide distribution in treatment of chronic IHD, unstable stenocardia, acute disturbance of coronary circulation of blood (Fig. 21). The methods of endovascular treatment and surgery of IHD do not compete, but complement each other. The number of

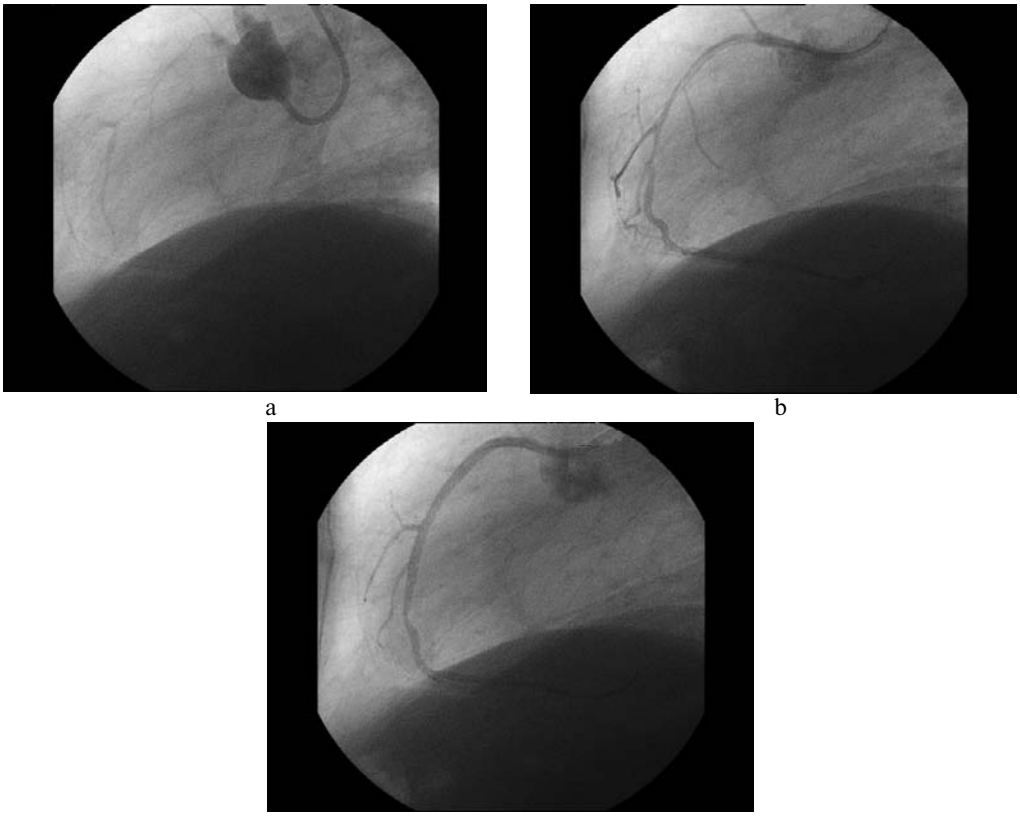


Fig. 21. a – occlusion of the right coronary artery, b – balloon dilatation of the right coronary artery, c – stenting of the right coronary artery

angioplasty with the use of stent in the economic developed countries increases steadily. Each of these methods has the indications and contra-indications. Coronaroangioplasty means balloon angioplasty and stenting of coronary arteries. Catheter with a balloon on its end is entered in the affected coronary artery at a balloon angioplasty in cath-lab, under X-ray control it is positioned in the location of narrowing of coronary artery and is inflated for 1–2 minutes. Thus, an atherosclerotic plaque is squashed, and the lumen of vessel is increased. At stenting after balloon inflation the metallic wireframe cylinder opens up with the special medical covering (stent) which supports the form of vessel lumen. Medical covering on the walls of stent serves as the prophylaxis of thrombosis in it. After insertion of stent the control coronaroangiography is needed in 4–6 months. This method of treatment is acceptable at the lesion of one or two arteries, at small atherosclerotic plaques. In the case of multiple and severe lesion of coronary arteries it is necessary to perform operative treatment. Advantages of TLBA:

1) more high efficiency by comparison to conservative treatment;

2) does not need general anaesthetizing and thoracotomy (for patients, at which by-pass is intolerable);

3) does not require the prolonged rehabilitation period. Disadvantages: the risk of development of dangerous for life complications during the balloon angioplasty

and stenting makes about 2%. Mainly it is related to the damage of atherosclerotic plaque and development of acute myocardium infarction (AMI) as a result of blockage of coronary artery. In such complication the operation of aorto-coronary bypass is performed urgently on vital indications.

Indications to the choice of heart revascularization are determined by cardiosurgeon together with cardiologist and interventionalist. The basic problem of angioplasty is the recurrent narrowing of coronary artery – restenosis which can develop in a few weeks or months. Rate of restenosis is about 10%.

Stenting was developed to decrease the disadvantages of TLBA – mainly risk of development of AMI. A coronary stenting is a method of intravascular prosthesis of coronary arteries at various pathological changes of structure of their wall. For the reconstruction of coronary arteries stents are used. Stent is metallic wireframe which is a little metallic tube from a wire. A stent is inserted into the artery after its balloon expansion and positioned in the place of the lesion of artery with the purpose of prevention of restenosis. A stent supports the walls of artery. More than 60 different constructions of coronary stents are presently offered. Depending on a design stents are divided on:

– wire (made from one wire);
– tubular (made from cylindrical tube);
– rings (made from separate links);
– reticulated (as the wattled net).

Depending on a technique of insertion the self-expandable stents and stents which are expanded by a balloon-catheter are distinguished. This intervention is performed, as angiography, by puncture of femoral artery. An operation is conducted under the local anesthesia. Through a puncture of femoral artery balloon with stent on the special guiding catheter is positioned in the location of narrowing of coronary artery. At inflating of balloon the lumen of artery is dilated and restored by stent. Heart obtains the necessary volume of blood through the restored artery which results in diminishing or disappearance of pains behind breastbone at the physical loadings.

Advantages of stenting:

– lasts a few longer than TLBA;
– the use of stent decrease by the necessity of the repeated operations;
– duration of hospitalization is shorter – 3-4 days;
– a stenting can be performed in patients, to which aorto-coronary bypass cannot be performed, but TLBA is not indicated;
– by comparison to TLBA diminishes the necessity of implementation of urgent aorto-coronary bypass; finally, less traumatic, than aorto-coronary bypass.

Disadvantages of stenting:

– thrombosis of stent;
– restenosis in stent.

There are three types of stenting:

1) bailout (saving, urgent) stenting – conducted at the acute blockage of coronary artery by the fragments of atherosclerotic plaque after TLBA in place of urgent aorto-coronary bypass;

2) planned (elective, primary) stenting – a stenting is conducted initially regardless of results of TLBA (at the various forms of IHD);

3) stenting on indications (provisional) – used on results an angiography after TLBA, when its results are not optimal, with the purpose of prevention of the acute blockage of coronary artery or development of its restenosis.

Mammarocoronary and aortocoronary by-passing without the cardiac arrest

It should be noted that there is an alternative variant of operative treatment – the coronary by-pass without the cardiac arrest. In order to stabilize the certain area of heart and impose a shunt on this area, the special stabilizing systems are used. Such operations are more physiological, period of rehabilitation after such operations, as compared to operations with cardiac arrest, is shorter. However such operations require higher qualification of surgeon. Wide distribution of operations without the cardiac arrest hinders the fact that technically to perform such operation at the multiple lesions of vessels, especially if there is a lesion of vessels on the posterior surface of heart, is very difficult. Clearly, that at small incision, mini-incision it is more difficult to work with an internal thoracic artery on a large area. The special endoscopic instruments which are inserted into thoracic cavity successfully allow to solve this problem. 11 different methods of minimally invasive endoscopic coronary surgery are today developed with the use of mini-incision, that allows to shunt one, two and even three coronary arteries, and then from small incision to suture the end of this artery directly to the affected coronary vessel, restoring the circulation of blood in heart. Moreover, the method of endoscopic coronary surgery is developed that replaces mini-incisions by few small punctures. It is a new section of coronary surgery. In its basis is implementation of operations on a working heart without application pumping oxygenator and use of minimum access. Limited, 5 cm long, thoracotomy or partial sternotomy is performed in order to preserve stability of breastbone.

These operations are indicated for not young, hyposthenic patients which can not tolerate pumping oxygenator due to the presence of disease of kidneys or other paernchymatous organs. Minimally invasive operation can be performed on right coronary artery or two branches of the left coronary artery from the left or right access.

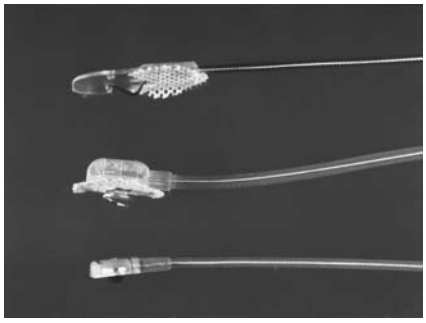
Transmyocardial laser revascularization

Transmyocardial laser revascularization is performed from thoracotomy access - lateral thoracotomy without connecting to by-pass machine. In area of myocardium with the low level of blood supply the great number of the point ducts is done through which blood enters to ischemic area of myocardium. These operations can be performed both independently and in combination with by-pass of coronary arteries. Positive results obtained in the large group of the operated patients allow to consider a method close to the direct revascularization of myocardium.

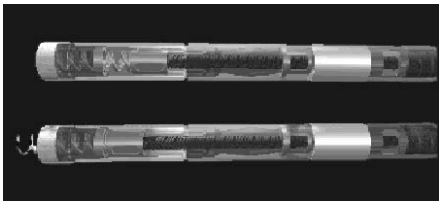
Implantation of electro-cardiostimulator and ablation of arrhythmogenic foci

Implantation of electro-cardiostimulator is a basic method of treatment of patients with the sick sine syndrome (SSS), the syndrome of carotid sine, atrioventricular blockade (Fig. 22).

Passive fixation



Miocardial



Active fixation

Electrocardiostimulator

Fig. 22. Endocardial electrodes

Method of permanent endocardial electrostimulation

Access. Most widespread is access through venesection of vena cephalica, that allows to introduce electrode to the vein under visual control. Incision 6-8 cm long, after the local anaesthetizing, is performed on the line of sulcus of Morenheim, or parallell to the clavicle on 4-5 cm below its middle third (Fig. 23).

The practical value of the use of jugular veins is preserved in case of occurring of difficulties of electrode introduction to vena cephalica. Incision from 2 to 3,5 cm long on 2-3 cm higher and parallell to the clavicle in the its middle third after the section of platisma allows to dissect and conduct venesection of external jugular vein (v. jugularis ext.). In the most exceptional cases, for example, at the expressed anomaly of venous vessels, the use of internal jugular vein is possible (v. jugularis int.). For this purpose incision on a neck proceeds parallell to the clavicle in medial direction to the level of lateral margin of the sternocleidomastoideus muscle, which is taken medially. An internal jugular vein is mobilized on 1-2 cm by silicon tubes. On the anterior-lateral wall of vein by non-traumatic monofilament non-absorbable thread 4/0-5/0, purse-string suture is put 5-7 mm in a diameter. Venesection is performed in a center. The introduced electrode is fixed by tightening of purse-string suture and necessarily by additional sutures to the back surface of sternocleidomastoideus muscle. An electrode under X-ray control is introduced into right auriculum or apex of right ventricle, and at bilocular stimulation in right auriculum and right ventricle. Visual control with the use of two projections allows to specify localization of distal end of electrode and form a loop for an electrode in the cavity of heart.

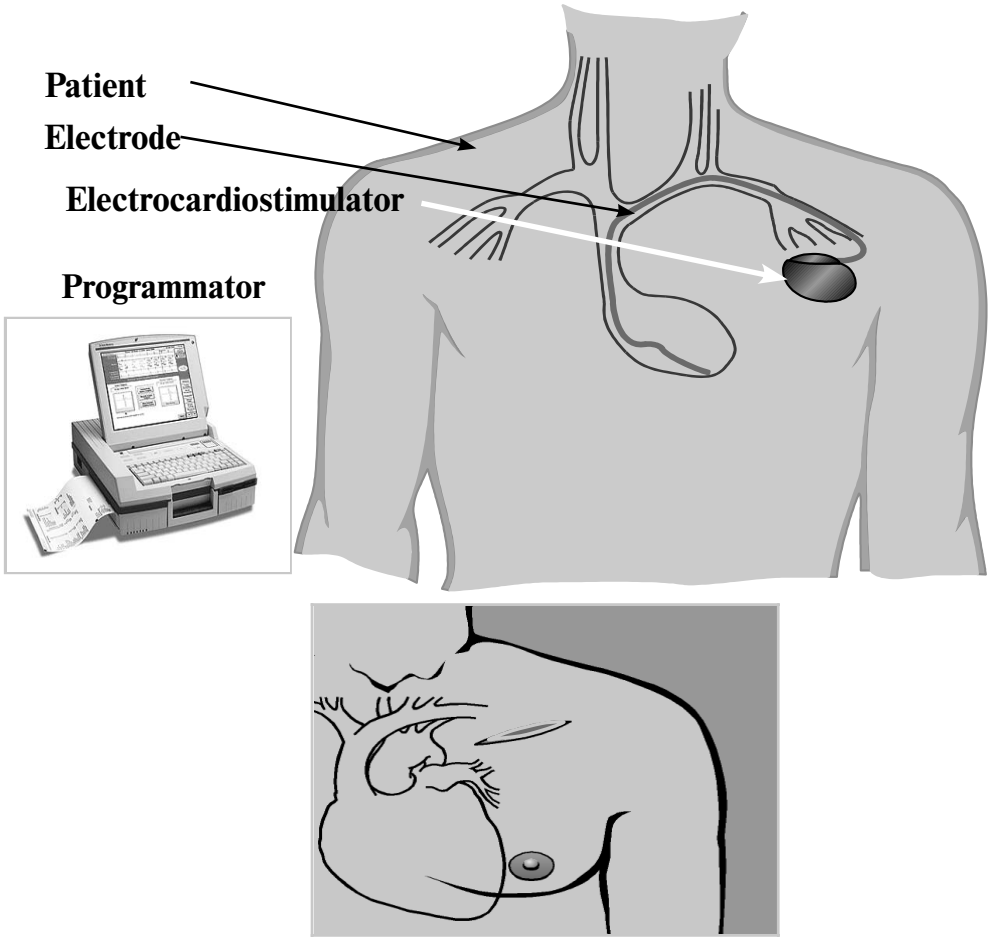


Fig. 23. Electrostimulation system

The proximal end of electrode is joined to the electro-cardiostimulator, which is placed in a bed, formed hypodermic in subclavian area (Fig. 24). Wound is closed.

Complications.

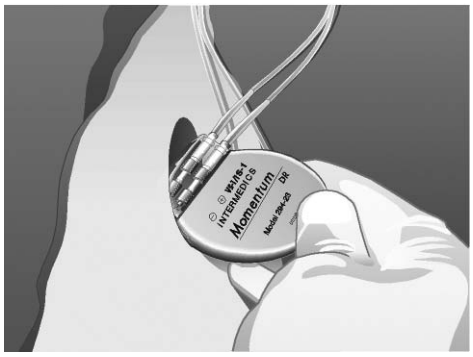


Fig. 26

1) The most threatening complication at implementation of permanent endocardial stimulation is perforation of heart with development of hemopericardium. The measures of prophylaxis of perforation is a steady observance of technology of implantation of electrode.

2) Dislocation of electrode is possible both in an early postoperative period and in more late terms. Diagnostics of this complication: the exposure of disturbance of permanent electrostimulation. In all

cases the repeated operative intervention is needed with the purpose of electrode replacement.

3) Haematoma of area of bed of electro-cardiostimulator, more often develops as a result of venous and arterial hypertension, presence of spontaneous or medicinal coagulopathy, imperfect technique of surgical intervention. In all of cases of uncertainty in hemostasis it is necessary to conduct the medicinal prophylaxis of haematoma, impose a compressing bandage on the area of bed of ECS. In the case of forming of haematoma it is necessary to conduct its evacuation by puncture by thick needle under local anesthesia or removal of sutures from the part of wound with the obligatory repeated imposition of sutures.

4) Infection of bed of electro-cardiostimulator develops to great extent from the unrecognized and unliquidated haematomas. In other cases in suppuration of bed of ECS changes of the immune system of patient results together with not enough careful following of asepsis during implantation. After confirmation of diagnosis by puncture reimplantation of ECS, sanation of inflammation is indicated, during which cardiostimulation must be performed from external ECS through the proximal contact of endocardial electrode. After completion of sanation the repeated intervention is indicated during which an endocardial electrode is removed and the new stimulant system is simultaneously implanted by other access.

5) Bedsores of electro-cardiostimulator. Principal reasons: mistakes at forming of bed of electro-cardiostimulator, positioning close to skin covers, violation of principle of connection of homogeneous tissues, ignoring of the constitutional features of patient.

Ablation of arrythmogenic foci

The modern methods of surgical intervention in tachyarrythmias include: classic methods of cutting or delete of some areas of the conducting system, transvenous cathether electro-destruction, cryoablation, radio frequency ablation of arrythmogenic foci. Presently advantage is given to radiofrequency ablation of arrythmogenic foci. Advantages of this method consist in absence of arrythmogenic and cardiodepressive actions and possibilities of conduction of operation without general anaesthesia. A radio frequency current is a current with variable polarity at frequency from 30 KHz to 300 MHz. The leading mechanism of coagulation of tissues under act of current is transformation of electricenergy to thermal. At the high density of current and low conductivity due to excitation of ions "friction" heat appears. Thus, tissue, adjoining to the electrode, but not an electrode is the source of heat. The method of radiofrequency ablation lately is widely used at fibrillation arrhythmia (as a rule, the isolation of inflow of pulmonary veins is conducted in the left atrium under control of angiographic, echoendocardiac and electro-physiology exams) and flutter (in this case more often ablation of isthmo-tricuspid tract is performed). Access for ablation of atrial fibrillation and its control includes the cannulation of both femoral veins and right subclavian vein. Lately robotic ablation of arrythmogenic foci is used.

Regardless of the chosen method, cutting or delete of areas of conducting system of heart or ablation of arrythmogenic foci, operative treatment is directed on the complete removal of focus of tachycardia or on creation of partial or complete atrioventricular blockade.

Electro-physiological methods reveal the exact location of ectopic focus at the complete removal of focus of tachycardia. Its cutting is than conducted. At impossibility to find the focus of tachycardia partial or complete destruction of atrioventricular node with subsequent establishment of ECS is performed.

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Chapter III. Diseases of the mediastinum

III.1. Diseases and injuries of the esophagus

The diseases of oesophagus make an enough big group among a different pathology of gastro-intestinal tract. To most often meeting concern such diseases as cancer, cardiospasm or achalasia of oesophagus, diverticulas of oesophagus, chemical burns of oesophagus or oesophagus corrosive, oesophageal strictures and , iatrogenic damages of oesophagus. Cardiospasm meets in 16-25 % cases among all diseases of oesophagus and takes 3 places after a cancer and scar strictures. Diverticulas of oesophagus is made from 5 to 29 % cases, often their existence have not clinical evidences and they are a roentgenologic find, but sometimes bring the severe sufferings to the patient, leading, in default of treatment, to disability, and at joining of some complications to death.

With the above mentioned diseases of oesophagus may meet all doctors of basic specialities and each of them must be able, to recognize this pathology, to appoint the necessary investigation and in the case of necessity administrate the conservative treatment or to point him at operation.

Achalasia of oesophagus (cardiospasm)

By the most widespread classification of cardiospasm, accepted and in our clinic, there is the B. V. Petrovsky (1957) classification subdividing the disease on the clinical evidences on 4 stages:

I stage (inconstant spasm of cardia) – is characterized by short timing dysphagia arising up due to ill-timed opening of cardia. By roentgenologic investigation the normal sizes of length and width of oesophagus are saved, the contours of oesophagus and esophagi-gastric section are even, tone of walls is normal, a peristalsis is active, deep, passes along all oesophagus and is finished at cardia, often not opening of it; periodically cardia opens and part of barium by a «throw» enters to a stomach; evacuation from a oesophagus takes place in good time.

II stage (stabile spasm of cardia) – makes progress disorders functions of cardia, dysphagia is more expressed therefore. Evacuation from an oesophagus begins to be slowed. Roentgenologic investigation detects insignificant even dilatation of the distal part of oesophagus with the conical narrowing in a terminal part. A peristalsis a speed-up, opening of cardia is not regular, between openings big intervals, which lead to disorder of evacuation from an oesophagus.

III stage (scar changes in cardia and dilatation of oesophagus) – is characterized by appearance of organic changes in cardial part of oesophagus due to fibrosis - scars processes. It leads to sharply expressed dysphagia, pressing pain in retrosternal region and regurgitation during a meal and at nights. Roentgenologic investigation of oesophagus is considerably extended, the_distal part of it has the conic form , «mouse tail»; the contours of oesophagus and cardia are even, the complete opening of cardia is not present, a peristalsis have disorderly character, is sharply weakened, segmental spasms are marked.

IV stage (severe scary changes of cardia with excessive dilatation and deviation of the oesophagus) – cardial channel severely narrowed and looks like scary tube/ Oesophagus maximally dilated, lengthening, curved. The wall is atonic. The ingest-

ed food may be delayed in oesophageal lumen more than 24, in it lumen there is big volume of liquid.

At questioning of patient:

1) All subjective symptoms (complaints) of cardiospasm are divided on primary and secondary.

To primary symptoms are regard: dysphagia, regurgitation, retrosternal pain, abundant selection of oesophageal mucus and hypersalivation; to the secondary – aerophagia, gydrophagia, falling of mass of body, loss of ability to work.

Dysphagia registers in 100% of patients and is the main symptom of cardiospasm. As a rule, at the beginning of illness it carries brief intermittent character, and then becomes permanent. .The expressivity of it may be different. Strong, long timing dysphagia can be during a few hours and days.

Regurgitation (oesophageal vomiting) meets in 88-99% of patients. Regurgitation may occurs shortly or several hours after meals or at night. Unlike the gastric vomiting the oesophageal vomiting does not have a sour taste, does not violate an appetite, an appetite is good, because satiation comes rarely. In the initial stages of the disease regurgitation occurs due to segmental spasm of the oesophageal wall. In the letter stages of the disease, when the oesophageal wall becomes atonic and its lumen is overfilled with residual fluid, vomiting occurred during sleeping in the night. In the horizontal position (e.g. at night) oesophagus tries to eliminate its contents onto pillow through a mouth – symptom of "wet pillow". Sometimes the patient may aspirate the oesophageal contents and usually awakes up from sleep due to cough.

Pain is observed at 78 % patients and is localized mainly in an epigastric region or retrosternaly. By character it can be: burning, related to concomitant oesophagitis, pressing- due to overtention of the oesophageal wall by ingested food, spastic - as result of peristaltic activity in early period of disease.

Hypersalivation and abundant secretion of oesophageal mucus discharge is the evidences of the permanent accumulation of the fluid in the oesophagus.

Aerophagia, as secondary symptom, which is using by patients for improve passage of food through cardia. Patients swallow air for the increase of pressure in an oesophageal lumen that improves passing of food. With the same purpose patients use a liquid in time or after a meal – hydrophagia.

For the best mastering of complaints and their estimation use next table:

	Complaints
Primary	Dysphagia, regurgitation, pain, hypersalivation.
Secondary	Aerophagia, hydrophagia, loss of mass of body, loss of ability to work.

2) It is necessarily to gather complains about other organs and systems of patient for the exposure of concomitant pathology.

3) Anamnesis of disease. It is necessary to find out, when the first signs of disease appeared at a patient, important to trace the dynamics of development of clinical symptoms, to find out the reaction of patient on different types of food, to define character of dysphagia, time of appearance of the oesophageal vomiting, presence

of symptom of the "wet pillow". It is necessary to pay attention to the dynamics of development of pains from the moment of disease.

4) At collection of anamnesis of life of patient it is necessary to pay attention on the possible reason of origin of disease; character of work of patient, connection of it with the psychical overstrains, previous diseases, presence of chronic diseases.

Results of clinical physical investigation.

1) The common state of patient depends on the stage of disease.

At the disease I, II and III stages the common state of patient do not change. The external signs of disease are possible in the IV stage: lowering of weight, dehydration. In mostly cases there are change in the behavior of patient (nervousness, estrangement), expressivity of which depend from stage of disease and from duration of disease.

2) At the general investigation of patient it is necessary to control thoroughly other organs and systems for the exception of heavy concomitant diseases and possible complications of basic disease, especially pulmonary system in the late stages of disease (bronchiectatic disease, chronic pneumonia and all).

3) Local status (Locus morbi). Because objective pathognomic symptoms at cardiospasm is not determined, «Locus morbi» at this pathology is not distinguished, the reliable diagnosis of cardiospasm only on the basis of objective methods of investigation is put can not be.

4) A basic clinical symptom at achalasia of oesophagus is dysphagia.

5) On the basis results of findings of questioning, anamnesis of disease and clinical physical investigation of patient it is possible to put a preliminary clinical diagnosis: Achalasia of oesophagus.

In accordance with standard charts the plan of additional investigation (laboratory and instrumental) includes:

Additional methods of research:

1) roentgenoscopy of thorax,

2) contrasting roentgenography of the oesophagus,

3) roentgenokinematography,

4) oesophagoscopy,

5) oesophageal manometry,

6) PH-metry have important role in raising of diagnosis.

Roentgenologic methods, researches allow not only correctly to diagnose but also determined stage of disease (Fig. 25). Already at survey roentgenoscopy of thorax it is possible to discover pathology in lights, being complication of cardiospasm. Yet to the reception of contrasting mass at 20% of patients the contours of the extended oesophagus are determined, that sometimes results in an error, when is diagnosed tumor of mediastinum or paramediastinal pleurisy. Sometimes the horizontal level of liquid is determined in an oesophagus. The gas bubble of stomach at cardiospasm practically is always absent.

By contrasting research of oesophagus with barium meal - a form, position and contours of oesophagus, tone of walls, relief of mucous membrane, motor function of oesophagus and cardia is determined.

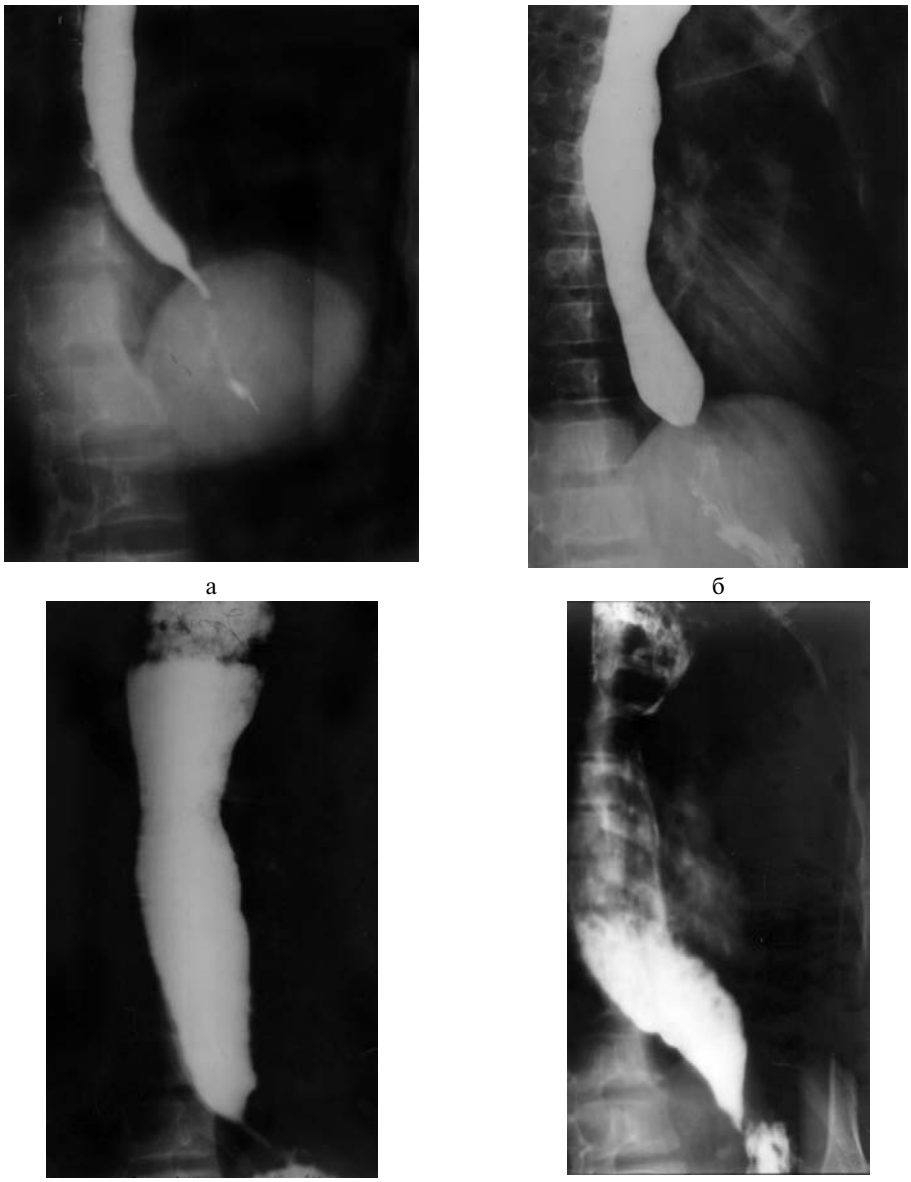


Fig. 25. Roentgencontrast investigation of the oesophagus achalasia: a – I stage, b – II stage, c – III stage, d – IV stage

Roentgenokinematography allows on the basis of study of motor activity a oesophagus and cardia to specify a diagnosis.

Oesophagoscopy is valuable additional, by a research method at cardiospasm which confirms a diagnosis, is special in late stages, allows studying the condition of mucous membrane, perform differentiation between cancer and achalasia. In the early stages of disease usually is very problematic to expose changes in an oesophagus except for some difficulty of passing of end of gastroscop through cardial sphincter. The oesophagoscopy picture of cardiospasm is very characteristic: extended

oesophagus, hyperemia of the mucosal membrane, which is covered by mucus; a cardial channel is closed and is had the appearance of point or crack.

Differential diagnostic. It is necessary to differentiate achalasia of cardia with the benign and malignant tumors of oesophagus, diverticulas, strictures of oesophagus. All of them have general symptoms: dysphagia, pains, regurgitation.

The benign tumors of oesophagus meet rarer than cardiospasm, at them slowly making progress dysphagia is observed only at the half of patients. For diagnostics, conducting of roentgenologic research, exposing the «defect of filling» with clear smooth contours, is important.

Malignant tumors meet considerably more frequent than cardiospasm. A cancer develops with the displays of quickly making progress dysphagia during a few months, with the simultaneous sharp worsening of the general condition, while cardiospasm is the protractedly flowing disease: with slow development, saved good general condition. A diagnosis is specified roentgenologic: at a cancer formation of "defect of filling" is marked, deformation of contours of oesophagus, oesophagoscopy helps diagnostics in doubtful case.

The scar narrowing of cardia more frequent than all arises up as a result of reflux-oesophagitis and chemical burns of oesophagus. To differentiate with cardiospasm sometimes is difficult, information of anamnesis, indication on protractedly existent reflux-oesophagitis, helpful and also oesophagoscopy, oesophagomanometry, PH-metry.

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

1) Basic - Achalasia of cardia (cardiospasm) ... stages.
2) Complications (basic disease) (if it is present).
3) Concomitant pathology (if it is present).

Treatment of patient with achalasia of cardia

Choice of medical tactic

Presently at treatment of cardiospasm 3 basic methods of treatment are proposed: conservative, method of cardyodylatation and operative.

1. Conservative treatment as independent method presently does not find application from short duration and unstableness the achieved direct results, even if it is begun in the I stage of disease.

2. Cardiodilatation is used at patients with I-Ш-stages, and at contra-indication to operative treatment and at the patients of the IV stage.

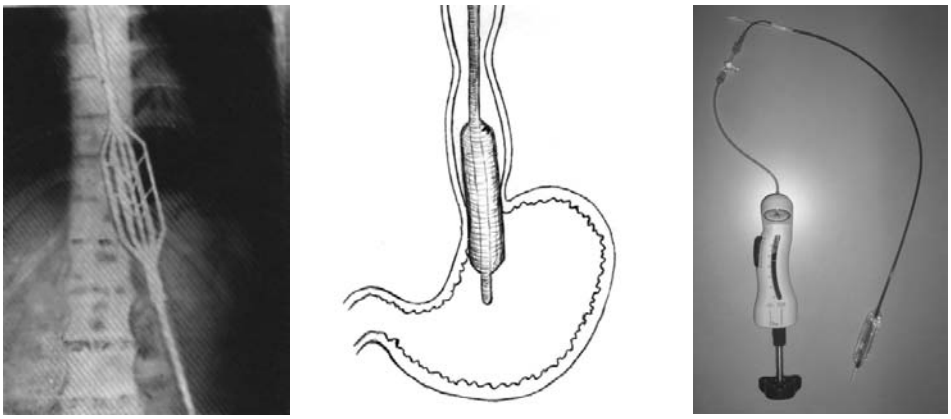
3. Indications to operative treatment of cardiospasm are following:
   1) IV stage of cardiospasm (contra-indication is heavy concomitant pathology);
   2) III stage is only selective: patients, at which the 3-4-multiple course of treatment by cardiodilatation did not give a satisfactory effect, and also patients at which was not succeeded attempt to conduct cardyodilatator through cardia;
   3) child's age in any stage of cardiospasm;
   4) relapse of disease, inadequate result of treatment after repeated cardiodilatations.

Conservative treatment

It is used for preparation of patients to cardiodilatation or operation is diet, spasmolitics, sedative drags, and physiotherapy. In early stages of disease is possible use and botulinum toxin.

Cardiodilatation

It is carried out by different types of cardiodilatators (Fig. 26), among which is most widespread metallic cardyodilatator of Shtark, pneumatic or hydrostatic dilatators of different constructions. Our department proposed modification of pneumodilatators, which has certain advantages after comparison with present (Fig. 27).



a b c

Fig. 26. a – Stark, b – pneumatic cardiodilatation, c – Wilson Cook dilatator

Fig. 28. Cardiodilatators

Technique of cardiodilatation. It is carried out on empty a stomach usually after premedication and anesthesia of pharynx by a 1% solution of dicaine. It performs under X-ray control, the empty balloon of cardiodilatator inserted up to gastro-oesophageal sphincter and inflated. Usually 3-4 dilatations are performed with the interval 3-4 days. Effect of cardiodilatation is controlled with help of manometry of cardial pressure. It mast be 11-12 mmHg.

Complications of cardiodilatation are: rapture of the cardial part of oesophagus, incompetence of gastro-oesophageal sphincter with formation of reflux-oesophagitis.

Operative treatment

Among the great number of proposed operations most widespread are:

I) operation of Heller – longitudinal oesophagocardiomyotomy. A single anterior incision is made through the longitudinal and circular muscular layers of the oesophagus down to mucosa. The mucosa is then separated from the muscles up to 2/3 of diameter. Allowing the mucosa to herniated through the gap. To the lacks of this operation is regards: the frequent relapses and appearance of reflux-oesophagitis because of insufficiency of cardial sphincter. In an early postoperative period, because of unnoticed perforation during the selection of mucous, may develop sharp empyema of pleura or peritonitis;

2) B. V. Petrovsky operation — longitudinal oesophagocardiomyotomy with its covering by graft of diaphragm on crus. Failings are the frequent relapses because of scar regeneration of graft;

3) V. I. Kolesov operation – longitudinal oesophagocardiomyotomy with omentoplastic; failings are those, that at the B.V.Petrovsky operation;

4) Gotschtein-Shalimov operation - longitudinal oesophagocardiomyotomy with the selection on this area of oesophageal mucosa on 2/3 its diameter with transversal stitches of dissected cardial muscles and fundophrenopexy; failings of this operation is insufficiency of cardia with development of reflux-oesophagitis;

5) Gotshteyna-Shalymova operation in modification KRIGUS-previous operation with adds fundoplication by Nissen, that prevents development of reflux-oesophagitis;

6) laparoscopic longitudinal cardiomyotomy. Possible complications are the same as during operation of Heller.

Among complications registering in an early postoperative period, it should be noted development: mediastinitis, pleurisy, peritonitis (it is special often after operation of Heller).

Peculiarity of treatment in postoperative period are included:

1) permanent presence in the stomach of the transnasal entered probe (to 6 days) which gives possibility to evacuated of gastric content and perform early feeding from the third days after operation;

2) restoration of water-electrolyte balance;

3) antibiotic therapy for the prophylaxis of purulent-inflammatory complications;

4) prophylaxis of cardio- respiratory complications;

5) symptomatic therapy.

Urgent conditions. At achalasia of oesophagus possible develops the urgent states as spontaneous rapture of oesophagus, bleeding.

Manipulations:

– PH-metry of the oesophagus for control of incompetence of the cardia;
– Oesophagomanometry after cardiodilatation or operations;
– Cardiodilatation.

Possible complications of achalasia of cardia:

a) oesophageal complications: oesophagitis**,** spontaneous rapture of oesophagus, cancer, bleeding;

b) pulmonary complications: aspirated pneumonia, chronic bronchitis, atelectasis, bronchiectatic disease, bronchial asthma, abscesses of lungs.

Principles detection of disability and prophylactic medical examination. Patients with achalasia of cardia of I-Ш-stages (without indication to operative treatment) must be under the clinical supervision at a surgeon. One-two times per a year the patient must pass roentgenologic research for the control dynamics of development of disease and early detection such complication, as cancer and chouse necessary kind of treatment.

Diverticulosis of the oesophagus

In clinic uses classification of diverticulosis of oesophagus proposed by A. A. Shalimov (1985):

I. ***By localization***:

1) diverticulas of neck;

2) diverticulas of thoracal segment of oesophagus: diverticulas of the upper third, bifurcational diverticulas, supradiaphragmal diverticulas;

3) diverticulas of the abdominal part of oesophagus;

4) multiple diverticulas.

II. ***By the mechanism of appearance***:

– pulsing; traction;
– combined (pulsing and traction);
– functional (relaxation)

III. ***By clinical course***:

– compensated (asymptomatic);
– decompensated:
  a) with predominant oesophageal symptoms;
  b) with predominant gastric symptoms;
  c) combined form of clinical presentation.

IV. ***By complications***:

– inflammatory processes:
– acute, chronic and recurrent diverticulitis, peridiverticulitis, tracheitis, bronchitis, oesophagitis, mediastinitis;
– perforation of diverticula:
– cervical phlegmona, mediastinitis, tracheo-oesophageal fistula, empyemy of pleura, abscess of lungs; perforation of diverticula in a pleura cavity, cavity of pericardium and peritoneal cavity; bleeding from diverticula; stenosis of the oesophagus;
– tumors of diverticula: benign and malignant.

At questioning of patient.

1) It is necessary to pay attention that character of complaints depends from localization of diverticula.

a) Diverticulas of cervical part (pharyngeal pouch or Zenker's diverticula) located above the upper sphincter at the border between pharynx and oesophagus. The diverticulum penetrates dorsally between the circular and oblique part of the cricopharyngeal muscles (triangle of Lane –Hakerman). The cause of diverticula is pulsing factor. To symptoms registering in the early stages of appearance of diverticula regards: the symptoms of irritation of throat - sensation of burning, dryness, cough, feeling of foreign body in a throat, disorder of swallowing, abundant saliva-

tion, unpleasant smell from a mouth, sensation of nausea. As far as diverticula sack has tendention to increase became disordering swallow act – sensation of arrest of a boll of meals upper part of oesophagus, that compels patients to eat slowly, and during swallowing the patient bend down the head to improve the food passage. Sometimes during a meal "phenomenon of blockade" may appear (symptom of Bensaud - Gregoire): after swallow act, a person blushes, develops dyspnoea. Facilitation comes after vomiting. Regurgitation may arise up at certain position of patient. Aspiriontion of content of diverticula in respiratory tracts is the reason of aspirated pneumonias. Hoarseness of voice is sometimes marked.

b) Diverticulas of middle part of oesophagus often asymptomatic and may be found incidentally. Sometimes the patient complains on transitory dysphagia, retrosternal pain, bleeding.

c) Diverticulas of lower third of oesophagus tends to be asymptomatic and may be found incidentally during radiography examination. They do not always and become larger. Dysphagia is a common symptom, which occurs, when the diverticulum pouch becomes full and hence compresses the oesophagus. Dysphagia usually disappears after drinking water. Sometimes there may be pain under lover part of sternum, regurgitation, aerophagia, cough, anorexia. The big pouch may lead to tachycardia and dyspnoea.

2) Anamnesis of disease can carry the protracted character with gradual growth of symptomatic.

Clinical physical investigation.

1) The general condition of the patients in most cases satisfactory. The condition becomes poor, if there are complications (mediastinitis, lung abscesses, perforation of diverticula, fistula formation, bleeding, and evolution of malignant tumor).

2) Local status (Locus morbi). Most informing local status is in case of cervical diverticulum. Depending on the size of the pouch (diverticulum), there may be bulking more often from the left side of soft consistence. After meals, percussion of the bulking may found splashing sound (symptom of Cooper). Sometimes it is possible to detect symptom of Claude- Bernar- Horner (enophtalm, myosis blepharoroptosis). In the localization of diverticulum in the middle-third or lover-third of oesophagus, local changes are absent.

3) The main clinical syndrome in beginning period of disease is syndrome of irritated oesophagus, at development of disease dysphagia is more frequent symptom.

4) Preliminary diagnosis at formed cervical diverticula, and also at diverticula of middle and lower parts of oesophagus putting on foundation of only physical examination is very difficult, because the similar symptomatic and anamnesis of disease can be characteristic for many diseases of oesophagus such as: cancer of oesophagus, cardiospasm, strictures of oesophagus and etc

According to standard charts the plan of additional investigation (laboratory and instrumental) at a patient with diverticulas of oesophagus includes: roentgenoscopy, contrasting roentgenography, roentgenokinematography and oesophagoscopy.

At roentgenologic investigation contrast fills diverticula, and then passes to the oesophagus. Filled diverticulum becomes visible on the back or lateral surface of oesophagus (Fig. 28).

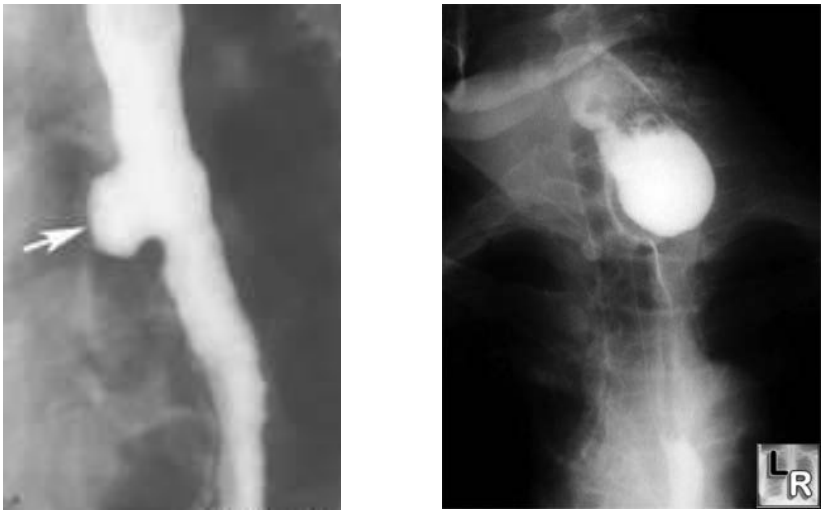


Fig. 28. Diverticulas of middle part of oesophagus and cervical part

The method of oesophagoscopy gives possibility not only to confirm roentgenologic diagnosis, but also to detect the changes which present into diverticula. The mucus membrane may be hyperemic, sometimes there are erosions.

At localization of diverticulas in middle and lower third of thoracal part of oesophagus oesophagoscopy can find out some indentation of mucous membrane on the wall of gullet with the signs of inflammation of mucous around.

Differential diagnostics of the patient with diverticula of oesophagus mast be performed with the diseases having a similar clinical picture, such as: benign and malignant tumors, parsoesophageal hernia, cardiospasm, scar stricture of oesophagus, ulcerous oesophagitis, ulcerous disease of stomach.

Sometimes the cavity ***of disintegrating cancer*** is taken for diverticulum, but for a cancer is characteristic roentgenologic ulceration of the outline of mucous membrane, distention of oesophageal lumen above place of narrowing. In doubtful cases a diagnosis very helpful oesophagoscopy with a biopsy.

At ***diaphragmal hernia of oesophagus*** compare with diverticula barium meal gets to hernia sack after passage through cardia, in case of diverticula – directly from oesophagus.

Epiphrenal diverticulum mostly of the small size need differentiates with peptic ulcer of oesophagus. X-ray examination detects presence of "niche" with abruption contours of mucosa near it. In doubtful cases oesophagoscopy with biopsy is indicated

At ***cardiospasm*** there is precardial dilatation of the oesophagus with smooth clear outline of the walls and delay passage of contrast through cardia.

At ***ulcerous illness of stomach*** roentgenologic and the oesophagoscopy changes are determined in a stomach and is absent in an oesophagus.

Ground and formulation of clinical diagnosis at a patient (according classification of disease, presence of complications and concomitant pathology):

– basic – diverticula of neck (с/3, н/3, epiphrenal) part of oesophagus; compensated or decompensated;

– complication – diverticulitis (perforation of diverticula, bleeding from diverticula and etc);
– concomitant pathology – at presence of.

Treatment of patients with diverticula of oesophagus

Choice of treatment tactic

Presently for treatment of diverticula of oesophagus uses conservative and operative methods.

Indications to conservative therapy:

At the moderately expressed clinical picture.

At presence of contra-indications to operative treatment (age, presence of heavy concomitant pathologies).

Indications to operative treatment of diverticula of oesophagus (B. V. Petrovsky, E. N. Vanzan, 1966): large diverticula (more than 2 cm) and even with the brief delay of contrast in a diverticula sack; small diverticula (less than 2 cm), but with the protracted delay of contrast in a sack; presence of diverticulitis; expressed clinical picture of disease independently from sizes of diverticula; complications of diverticula (oesophagotracheal fistula, ulceration of diverticula, bleeding, polyposis, cancer).

Conservative treatment

It is directed on prevention of food delay in the diverticulum pouch and avoidance of inflammatory process (diverticulitis). It includes: soft diet, taking much fluid after meals for irrigation of diverticula, using adopt special position of the body after meal for quick empting of the diverticula pouch. Acute dishes and hot food is forbidden. Administration of spasmolitics sometimes is necessary.

Operative treatment

Presently operation of choice at diverticula of oesophagus is diverticulectomy or invagination of diverticula to oesophageal lumen, the last is used at small diverticula only.

***The peculiarities of postoperative period are*:**

1) transnasal insertion of probe to the stomach (up to 6 days) for it emptying and for early feeding of patient since from 3 day from the moment of operation;

2) restoration of the water-electrolyte disbalances;

3) antibiotic therapy for the prophylaxis of inflammatory complications;

4) prophylaxis of cardio respiratory complications;

5) symptomatic therapy.

Among possible ***early postoperative complications can be marked*:**

1) due to incompetence of stitches oesophagus: mediastinitis, acute purulent pleurisy, peritonitis; pleurisy;

2) pneumonia.

Possible complications diverticulas of oesophagus:

1) bleeding;

2) perforation with development of peritonitis, pleurisy, mediastinitis;

3) diverticulitis;

4) oesophagotracheal and oesophagobronchial fistulas;

5) evolution of malignant;

6) development of different pulmonary complications (relapsing pneumonias, abscesses of lungs, chronic bronchitis).

Principles detection of disability and prophylactic medical examination. Patients with diverticulas of oesophagus must be under the medical supervision. At the waiver of operation or presence of contra-indications to it the patient mast undergoing one-two times per a year to roentgenologic examination for the control the dynamics of development of diverticula for detection such complication, as cancer of oesophagus.

Esophagus corrosive

It is occurs after accidental or suicidal ingestion of strong acids or alkalis.

There are: coagulative (burn by acid) and coliquation (burn by an alkali).

Classification of burns of oesophagus, which proposed by A.A.Shalimov (1975) is used in our clinic:

1. Pathomorphology changes (depending on the depth of lesion):
   The I stage – lesion of superficial layers of epithelium;
   The II stage – necrosis involving mucosal membrane;
   The III stage – necrosis involving all membranes of the oesophageal wall;
   The IV stage – necrosis, which is extents on paraoesophageal fat, pleura, sometimes pericardium, behind wall of trachea and other surrounding organs.
2. By clinical presentation: slight, moderate and severe.

At questioning of patient

Complaints. The clinical presentation depends from severity of lesion.

The slight form (initial stage) is characterized by reflectory vomiting, often blood tinged. The patients complain on pain in throat, which increasing during the swallowing, hypersalivation, general weakness, increase body temperature during the first 3 days, thirst, decrease of urine output. Usually all above mentioned symptoms are decrease or disappear after 3-6 days.

Moderate form is characterized by severe pain in the mouth, retrosternaly, epigastric region, repeated vomiting blood tinged. All the above mentioned arise immediately after ingestion of the chemical substance. The patient usually becomes anxious. They are very pale. There are hypersalivation, hoarseness of the voice, severe thirst; body temperature increases up to 38-39 °C. The volume of urine is decreased. After 3-4 days condition of the patient has tendency to improve. They may start to eat after 12-15 days, symptoms of acute inflammation is diminishing.

The severe cases the patient may be unconscious. Patient complains in very several pains in the oral cavity, throat, retrosternaly, epigastric region, dyspnoea, repeated vomiting with blood and fragments of the mucosal membrane. Swallowing is impossible due to pain (odinophagia).

As far as calming down of the sharp period after the burn of oesophagus and became evolution stricture of oesophagus, which usually begins to be formed after 3 weeks from a moment a burn, appears complaints on dysphagia: there is difficulty at swallowing of hard food, and than due to progressivety of process - and liquid. The patients complain on hypersalivation, sometimes on obturation of the oesophagus after ingestion of the hard food. An obstruction may be present a few hours. Sometimes vomiting appears and the swallowed obstructed piece together with vomiting masses goes out. On occasion the obstructed piece of food may be removed only

with help oesophagoscopy. During a meal patients usually feel pressure and pain retrosternaly. Regurgitation is often marked.

Anamnesis of life. To find out anamnesis usually does not difficult, considerably more difficult sometimes to detect chemical nature of the ingested liquid by the patients.

Clinical physical examination

1) At patients with the burn of oesophagus objective information depend from the stage of burn. More frequent patients are excited. The skin and visible mucus are pale. There are oedema and hyperemia of the lips and surrounding skin, tongue, cavity mouth and throat mucus are edematic with necrosis, which are covered by fibrin, sometimes bleed. In severe form of burn the blood pressure is very low, Ps is very rapid (tachycardia) and sometimes cannot be determined due to shock, dyspnea, a temperature rises, intoxication takes place, excitation often replaced on adynamic, appears the entangled consciousness, quite often delirium.

The state of patients gets worse at joining of early complications of burns of oesophagus - acute mediastinitis, pericarditis, pleurisy, empyema of pleura, bleeding, oesophago-tracheal and oesophago-bronchial fistulas.

2) ***Local status***. At examination of the cavity of mouth at patients with the burn of oesophagus there are the edema of lips and mucous cavity of mouth, which hyperemic, sometimes bleed, covered by fibrin. During palpation of the anterior surface of the neck possible detect different by intensivety painfulness and edema. There is painfulness in epigastric region.

3) *The mine clinical symptom* of this disease is retrosternal pain.

4) On the basis of complaints, information of anamnesis (important to specify character of the ingested liquid), gotten objective findings it is possible to put a preliminary diagnosis – chemical burn of gullet (by an alkali, acid), degree of severity...

In accordance with a standard chart the plan of additional investigation of patient with the burn of oesophagus includes:

1) clinical blood test;
2) clinical analysis of urine;
3) Ht;
4) electrolytes of blood;
5) biochemical research of blood;
6) coagulogramm;
7) daily diuresis;
8) ABB (acid-base balance);
9) EKG;
10) roentgenoscopy of the chest;
11) contrasting roentgenologic research of oesophagus in fresh case of burn is performing rarely due to impossibility to swallow barium;
12) oesophagoscopy in the acute period of burn is not performing due to possibility to get perforation of oesophageal wall.

In late period after burn contrasting roentgenography of oesophagus gives possibility to detect place, extent, degree of narrowing and deformations of oesophagus..

Differential diagnostics. In the acute period of chemical burn of oesophagus in mostly cases differential diagnostic is not needed.

Ground and formulation of clinical diagnosis of patient (taking with account classification of disease, presence of complications and concomitant pathology):

1) basic – chemical burn of oesophagus by solution of alkali or acid of the II degree, middle or other degree of severity;

2) complication – the erosive bleeding of the I degree, (or right-side bronchopneumonia;...);

3) concomitant pathology – (if it is) hypertonic disease or other.

Treatment of patients with the chemical burn of oesophagus

Choice of treatment tactic. Chemical burn of oesophagus is the urgent state requiring the quick medical aid on a period before admition to hospital and after hospitalization to surgery department.

Principles of the pathogenetic conservative therapy

First medical aid at the chemical burns of oesophagus.

The first medical aid mast be given as early as possible. First aid outside of clinic: lavage of stomach with ingestion of water or milk to dilute the corrosive and induction of artificial vomiting. The patient mast urgently admitted to hospital, where the stomach and oesophagus mast be flush off by slight concentrated vinegar (1-2%) or bicarbonates of soda (3%) depending on ingested chemical liquid with help nasogastric tube. Before introduction of probe to the patient need gives to him a few mouthfuls 5-10% of solution of novocaine with the purpose of anaesthetizing of mucous throat and anesthesia of oesophageal mucosa for the removal of pain and vomiting reflex, and then a probe is introduces into a stomach. Not extracting a probe from a stomach, the patient mast drink neutralizing solution or warm water. An ingested liquid washes off the oesophageal and stomach walls and than passes out through tube outside. Procedure is repeated.

Along with deleting and neutralization of chemical substance, the patient mast gets the antishock treatment.

Treatment in permanent establishment. After providing of the first aid outside of clinic, a patient must be urgently admitted to hospital for complex intensive therapy which mast includes: parenteral feeding, desintoxic therapy, antibiotics therapy (for prevention of purulent complications), symptomatic therapy directed on normalization of function of different organs and systems. For the prophylaxis of strictures of the oesophagus in an early period after burn steroid hormones are administrated in combination with antibiotics for 6 week, which considerably minimize fibrous and scar formation, reduce expressivity of inflammatory processes and due to it prevent development in the oesophageal lumen of rough fibrose-scars changes

In 2 days after a burn it is possible to enter through a nose into a stomach a thin duodenal probe for feeding of patient. If a burn of oesophagus very excessive and the patient can't ingest the liquid during 5-7 days through a mouth, appears indication to perform gastrostomy.

In 2 weeks after a burn, if there is no fever or some other complications, condition of the patient becomes stable, need perform early bougienage dilatation of oesophageal strictures, which mast be done 1 time per week after burn and than 1 time per month during 1 year.

Technique of early bougienage. In beginning very carefully the soft bougie covered by vaseline or glycerin with diameter near 15 mm inserts to the oesophagus, where it

mast be situated up to 30 minutes. Introduction of probe is repeated during 3 weeks daily. The caliber of probe every time is slowly increasing up to 20 mm. If there are no signs of stenosis, bougienage performed one time in a week by approximately 2 months.

Very serious complication during the bougienage is perforation of oesophagus.

Formation of scar on place of burn affectation of oesophagus is proceeds from 2 to 6 months. If prophylactic bougienage is not prolonged or treatment is not completed, in the near time after the receipt of chemical burn evolves scar stenosis, which characteristic by appearance of progressing dysphagia.

Possible complications of chemical burns of eosophagus: erosive bleeding, perforation of oesophagus with formation of mediastinitis, peritonitis, pleurisy, pneumonia, oesophago-tracheal and oesophago- bronchial fistulas, pericarditis, edema of larynx.

Principles detection of disability and prophylactic medical examination. Patients with the burn of oesophagus after gotten treatment must be constantly under the medical control at a surgeon. They mast one-two time per year undergo to roentgenologic research for timely diagnostic of possibility formation of postcorrosive strictures of oesophagus.

Poscorrosive scar stryktures of eosophagus

***In a clinic the V. H. Vasilenko (1971) classification is accepted, which distribute strictures of oesophagus depending from the extent of it*:** membrane (soft thin membranes with a thickness up to 2-3 mm); circular – length of them not more 2-3 cm; tubular – length more 5-10 cm; subtotal; total; solitary or multiple.

For clinical estimation of postcorrosive impassability of oesophagus uses and ***the clinic-roentgenology classification of G.L.Ratner and V.I.Belokonev (1982)***, subdividing expressivity of strictures of oesophagus on 5 degrees:

I degree – selective. Impassability of oesophagus arises only at the ingestion of some types of food. Diameter of lumen of oesophagus in area of narrowing 1,0-1,5 sm.

II degree – compensated. The oesophagus is passable for semi-fluid or carefully mechanically treated food. Diameter of lumen in area of narrowing 0,3-0,5 sm. There is small dilatation of oesophagus above narrowing.

III degree – subcompensated. The oesophagus is passable for liquids and butter. Diameter of narrowing less than 0,3 sm. Suprastenotic dilatation of oesophagus is determined. Thick barium contrast stays too long at the level of narrowing. Oesophagus is passable for the water-soluble contrasting matters.

IV degree – decompensated. The possibility of oesophagus is completely disordered. Suprastenotic dilatation of oesophagus is expressed. Under action of anti-inflammatory therapy possibility of oesophagus may be partly restored.

V degree – unrestored. It is characterized by obliteration of the oesophageal lumen.

At questioning of patient

1) *Complaints* of patient on the basic disease.

Dysphagia of organic type is a basic symptom, which may be variable in severity – from the indefinite unpleasant feelings during swallowing of food, to complete impossibility to swallow ingested food include and water). More dry, dense

and rough food passes worse and more frequent causes the complete obstruction of oesophagus with feeling of retrosternal pressure, pain, urges on vomiting. Acute obstruction can be present up to few hours and frequently may disappear by the artificial vomiting. At the considerable narrowing of oesophagus patients may ingest liquid food only.

Frequent symptoms are: hypersalivation, retrosternal pain, regurgitation, exhaustion.

1) *Anamnesis of disease* is very important for detection history of swallowing of chemical liquid.

2) *Anamnesis of life* usually does not give information in performing of diagnosis.

Clinical physical examination

1) Estimation of the common state of patient depends on the degree of expression of stenosis of oesophagus and can be from satisfactory to heavy.

2) The objective examination of patient at the stages subcompensation and decompesation gives possibility to found asthenisation of patient, with considerable loss of mass of body, signs of anemia, and sometimes lowering of turgor of skin due to water-electrolyte violations.

3) At the clinical inspection of patient with this pathology "Locus morbi" is not possible expressly to select.

4) Basic leading symptom for this disease is dysphagia of a different degree of expression.

5) On the basis of questioning, anamnesis of disease, clinical physical investigation of patient it is possible to perform a preliminary diagnosis: postcorrosive scar's stricture of oesophagus (the chemical burn of oesophagus at 2004 year), degree... (as example).

***In accordance with standard charts the plan of additional investigation (laboratory and instrumental) of patient with stricture of oesophagus includes*:**

1) clinical blood test – anemia is possible;

2) clinical analysis of urine – the increase of specific gravity is possible;

3) daily diuresis – lowering of volume of urine;

4) Ht has tendention to increase;

5) the general albumen of blood is lowering;

6) electrolytes of blood – lowering of the concentration K^+ and Na^+ is possible;

7) roentgenologic research: the scar narrowing of oesophagus has usually a tube form; relief of mucous membrane in area of narrowing is absent, a peristalsis is not determining, proximal part are extended; sometimes even liquid contrast can long time stay above of narrowing; narrowing of oesophagus after the burn of oesophagus can be multiple;

8) oesophagoscopy: gives possibility to detect the more or less expressed concentric narrow of oesophagus; mucous membrane within the limits of narrowing usually is hyperemic; it is possible to detect erosions, fresh granulations, grayish tapes of necrosis and fibrinous impositions; the length of stenosis with help oesophagoscopy in mostly cases is impossible.

Differential diagnostics at patients with postcorrosive strictures of oesophagus must be performed: with the syndrome of Plammer-Vilson, benign and malignant tumors, ulcerous oesophagitis, cardiospasm. Differential diagnostics at patients with

scar stenosis of oesophagus usually does not represent large difficulties. In doubtful cases additional methods of investigation are helpful in diagnostic.

Ground and formulation of clinical diagnosis at a patient with postcorrosive scar stenosis of oesophagus according adapted classification: (as example)

1) basic – postcorrosive annular scar stricture of lower third of oesophagus, stage of subcompensation (or other);

2) complications of basic disease – proximal erosive esophagitis;

3) concomitant pathology.

Treatment of patients with postcorrosive scar strictures of oesophagus

Choice of treatment tactic. Treatment of postcorrosive scar strictures of oesophagus can be performed conservatively and operatively. The choice of method of treatment depends on an extension and expressed of stricture, effectivety of gotten conservative therapy.

Conservative therapy is indicted in case of limited by extension strictures mostly cases segmental of the of the I-III degree.

Operative treatment is indicated if conservative treatment (bougienage) is not effective or if stenosis is very expressed and extended.

Principles of the pathogenetic grounded conservative therapy

Conservative treatment (minimally invasive treatment) begins after 7 weeks after corrosive agent ingestion, the patient may undergo late bougienage dilatation of formed postcorrosive stricture of oesophagus, which is directed on destruction of granulations and young connecting fibrotic tissues and which participates in formation rough scar stenotic changes (Fig. 29). The perforations of oesophageal wall are most often complication possible in this period.

There are various kinds of bougienage: Blind method – the bougie is gently inserted into the oesophagus without visual control (if stricture is very short); Under oesophagoscopy guidance – the bougie is passed through the stricture under oesophagoscopy guidance; Bougienage without end – may be performed if gastrostomy is present. The patient swallows a thread with small fixed metallic ball at the end. The thread is withdrawn through gastrostomy and used for bougie passage. It (thread) is used as a guide; Bougienage by thread (in patients without gastrostomy). By metallic string – the metallic string introducing through the mouth to stomach is used as guide

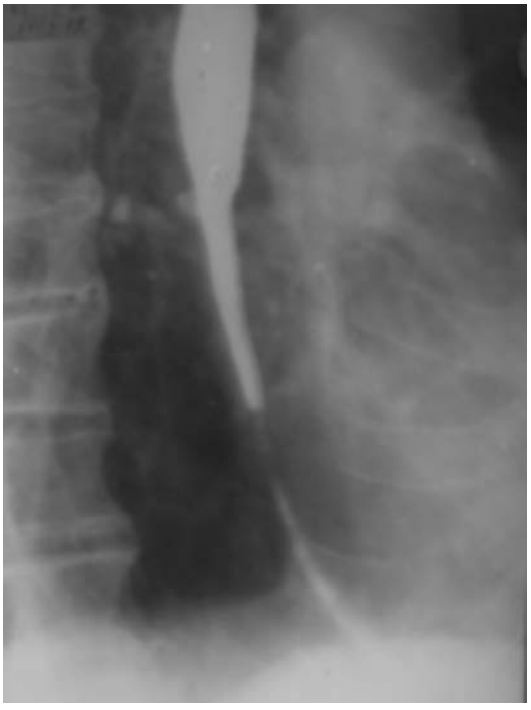


Fig. 29. Postcorrosive scar strictures of oesophagus

for bougie; Vibrobugienage – the proximal end of bougie connects with special vibroset and then the bougie under vibration slowly and gently dilates the stricture.

Fig. 30. Oesophageal stent

Methods of operative treatment.

1. Circular resection of the affected segment of oesophagus if the stricture not extend.

2. Plastic replacement of the oesophagus by stomach, segment of small intestine or colon in cases extended multiple affectations.

3. Stenting of the oesophageal stricture (Fig. 30).

4. In case of extreme exhaustion of patient before plastic oesophagus for same period need apply gastrostomy (by Kader or Vitzel). Sometimes gastrostomy may be as final operation for the enteral feeding of patient (at presence contra-indications to it or if patient reduces of plastic operation).

The peculiarities of postoperative period include:

1) administration of anesthetic;

2) antibiotics;

3) restoration of water-electrolyte and proteins;

4) symptomatic therapy.

***Possible complications of oesophageal strictures*:** acute obstruction of oesophagus in phase of affectation, repeated aspirated pneumonias due to regurgitation of oesophageal content, appearance of cancer of oesophagus, perforation of oesophagus.

Principles of detection disability and prophylactic medical examination the patients with postcorrosive scar strictures of oesophagus. A patient must be under the medical supervision at a surgeon. The patient mast one-two time per a year performs roentgenologic research and if present indication oesophagoscopy too, it necessary for detection worsening of disease and in time to begin adequate treatment, and also for recognition some complications, such as malignant tumors of oesophagus.

Iatrogenic damages of oesophagus

Classification of damages of oesophagus which is accepted in a clinic, proposed by Paulson et al. (1960):

I. Traumatic perforations.

A. Direct.

Instrumental:

a) oesophagoscopy and gastroscopy;

b) intubation of trachea;

c) cardiodilatation;

d) gastric intubation;

e) bougienage of oesophagus;

f) surgical.

Foreign bodies:

a) swallowed;

b) performing penetrable wounds.

Burn. B

– Indirect.
– Spontaneous rapture;
– Rapture as a result closed trauma;
– Rapture combining with narrowing.

II. Inflammation:

a) oesophagitis;
b) peptic ulcer;
c) TB lymphadenitis;
d) diverticulitis;
e) abscess or aneurism;
f) neurogenic ulceration;

III. Tumors:

a) benign;
b) malignant.

At questioning of patient

1) *Complaints of patient on the basic disease.* Usually at once after the damage of oesophagus patients begin complain on pressure or pain in a throat, retrosternaly, subxiphoidal region, in epigastria, which increase at swallowing, deep inspiration and cough. Pain often irradiated in the back, sub scapular region and has a tendency to quick increase.

Second by frequency symptom is dysphagia, difficulty of breathing can come together with it. The accompanying spasm of oesophagus and inflammatory edema of mucous is the case of dysphagia. A patient can not ingest a food and even water. Regurgitation can be marked, often with the admixture of blood, and sometimes bleeding. As usually there is hypersalivation. A temperature rises at development of inflammatory process.

2) *Anamnesis of disease.* Appearance of several pains of above mentioned localization in time or after performed of surgical manipulations on a oesophagus allows to suspect their reason. Important to know real time, which is passed from the moment of appearance of complaints.

Clinical physical investigation

1) *The general condition of patient* is heavy has a tendency to the progressive worsening and at absence of adequate help due to toxemia and shock the patient dies.

The patients frequently behave uneasily; trying to decrease pain, as usual uses forced position (bend forward position). The skin is pale, often covered by sweat. At the damage of pleura there is the several shortness of breath and the phenomena of respiratory insufficiency quickly grow.

2) Cardio-vascular system - expressed tachycardia, the B/P decrease, the borders of heart have tendention to increase due to developing mediastinitis.

3) Lung system – at the damage of pleura due to rapture of oesophagus pneumothorax and hydrothorax can be diagnosed.

4) Organs of abdominal cavity – at the damage of lower third or abdominal part of oesophagus may be present several pains in an epigastric region. Symptomatic of acute peritonitis develops.

6) ***Local status «Locus morbi»***. At the damage of cervical part of oesophagus it is possible to detect painfulness at pressing on a larynx, palpation can find out inflam-

matory infiltration of neck tissues. It is very often possible to found hypodermic emphysema which appears in a few hours after a trauma. At the damage of the lower third or abdominal parts of oesophagus as usual there are acute strong pains in epigastric region, developing symptomatic of acute peritonitis.

7) The main clinical symptom is progressing permanent pain, which located retrosternaly and in area of neck.

8) On the basis of the gotten complaints, anamnesis of disease, objective examination it is possible to put a preliminary diagnosis: Iatrogenic damage (perforation) of oesophagus.

***In accordance with standard charts the plan of additional investigation (laboratory and instrumental) of patient with the damage of oesophagus includes*:**

1) the clinical blood test - leucocytosis with the change of formula to the left;
2) clinical analysis of urine;
3) biochemistry of blood;
4) coagulogramm;
5) electrolytes of blood;
6) Ht;
7) EKG. All foregoing researches have auxiliary character for estimation of the general condition of patient. By the important methods of research, necessary for rising of diagnosis, are:
8) examination of pharynx (pharyngoscopy);
9) contrasting roentgenologic research;
10) oesophagoscopy,
11) computer tomography (CT).

Examination of oesophagus must be begun with examination of pharynx, because it is here succeeded to find out the perforation in area of sinus. Then proceed to roentgenologic research of oesophagus, which is the most exact and simple method of diagnostics.

At the damage of the upper third oesophagus roentgenography research is performed in two projections, the layer of gas is here determined in is soft tissues of neck back from a oesophagus or horizontal level of liquid. At research in lateral projection possible detect the increase of volume of prevertebral space and displacement oesophagus and trachea to anteriory. After giving to the patient of water or liposoluble contrast possible found it deviation through the rapture to soft tissues of neck.

At the damage of thoracal section of oesophagus can be found out the following roentgenology signs:

1) emphysema of mediastinum;
2) subcutaneous emphysema;
3) hydropneumothorax;
4) deviation of the contrast material out of oesophageal lumen.

***Computer tomography* (*CT*)** – gives possibility to estimate condition cellulose of mediastinum, lungs and pleural cavities. May detects insignificant presentation of air in the cellulose of mediastinum and infiltration of it.

At doubtful roentgenologic information need perform oesophagoscopy.

Differential diagnostics. According information of anamnesis and clinical symptomatic usually differential diagnostic does not cause of difficulties.

***Ground and formulation of clinical diagnosis at patients with the iatrogenic damage of oesophagus (account classification, presence of complications and concomitant pathology)*:**

1) basic – iatrogenic damage of upper third of oesophagus;
2) complications – mediastinitis, left-side hydrothorax;
3) concomitant pathology – arthritis of right knee-joint.

Treatment of the patients with the damage of oesophagus.

***Choice of treatment tactic*:** The main kind of treatment in case of damage of oesophagus is surgical, but conservative method in early period of beginning of disease is not excluded too if there are small perforation and satisfactory condition of the patient.

***Principles of the pathogenic grounded conservative therapy*:**

1) bed regime;
2) feeding through a nasogastral thin probe;
3) antibiotic therapy;
4) desintoxic therapies;
5) restoration of levels of electrolytes and albumens;
6) symptomatic therapy.

Existent methods of operative interferences and indications to it

At presence of iatrogenic damage in the neck part of oesophagus operation include stitching the wound of the oesophagus with drainage the place of stitching and surrounding tissues.

The damages of pectoral thoracal part of oesophagus require operative treatment mainly. Three groups of operations are applied:

1) operations for exclude of oesophagus from digestion (gastrostomy, crossing of oesophagus in neck part with formation oesophagostomy;

2) draining operation (mediastinotomy – neck, dorsal extraplevral by Nasilov, transdiaphragmal, transpleural);

3) operations on the perforated oesophagus (stitching the place of perforation, resection of the perforated part of oesophagus).

Peculiarities of the postoperative period.

1) feeding through a nasogastric probe or gastrostomy on 4-5 days from the moment of operation;
2) correction of water-electrolyte and protein disbalances;
3) antibiotic therapy;
4) desintoxic therapy;
5) symptomatic treatment.

Possible postoperative complications:

a) incompetence of stitches;
b) pneumonia;
c) proceeding mediastinitis with development of sepsis and polyorganic insufficiency (PON);
d) thromboemboly of pulmonary artery;
e) bleeding.

***Possible complications of iatrogenic perforation of oesophagus*:**

1) mediastinitis;
2) pneumothorax;
3) hydrothorax;

4) peritonitis;
5) bleeding;
6) sepsis.

Principles of detection disability and prophylactic medical examination the patients with iatrogenic damage of oesophagus.

A patient mast be under medical supervision of the surgeon. He mast one-two time per year performs roentgenology research for recognition late complications such as formation of strictures of oesophagus or evolution of malignant tumor.

III.2. Diseases of the mediastinum and diaphragm

Diseases of the mediastinum - group of diseases related to the involvement into the pathological process of organs located in the mediastinum. They are often found both in outpatient and hospital practice. Benign tumors and tumor diseases of mediastinum occur much more frequently than malignant tumors (in the ratio 4:1). Among the most frequent benign tumors there are teratomatous and neurogenic, and among malignant - tumors of lymphoid tissue. In the anterior mediastinum tumors are 2 times more frequently than in the poterior. Patients are mostly young and middle-age and both sexes equally often.

Congenital cysts are found in 22.3%, neurogenic tumors — in 15,8%, mediastinal goitre — in 5,2%, thymoma — in 2,1%, pericardial cyst — in 2,8% of cases. Malignant tumors are found in 23.6% (including lymphogranulomatosis and lymphosarcoma). Metastasis of various tumors is quite often observed in the mediastinum, which can also give mediastinal syndrome.

Blunt injuries of diaphragm occur in car accidents, falls from height, air contusion, compression of the abdomen. Rupture of diaphragm in these cases occurs due to the sudden increase in intraabdominal pressure. Injuries are more frequent in the tendinous center or at the point of transition into the muscular part of the diaphragm. In 90-95% of cases there is rupture of left dome of the diaphragm. Often at the same time there are injuries of the chest, pelvic bones, organs of the abdominal cavity. When the diaphragm ruptures as a result of negative intrathoracic pressure the stomach, small or large intestine, omentum, spleen, part of the liver begin moving into the pleural cavity. Moving organs may occur either directly after the injury, and through any period of time.

Open injuries happen when the diaphragm is damaged due to stab and gunshot thoracicoabdominal wounds. They are in most cases combined with damage to the chest and abdominal cavities.

Diaphragmatic hernia – moving of the abdominal organs into the chest cavity through defects or weak zone of the diaphragm. True hernia is characterized by the presence of hernial ring, hernial sac and the hernia contents. In the absence of hernial sac hernia is false. In true hernia hernial sac is formed by parietal peritoneum, the top of which iscovered with parietal pleura.

They are divided into traumatic and nontraumatic by origin of hernia gates.

Among isolated nontraumatic hernias there are: false congenital hernias (defects) of diaphragm, true hernia of weak zones of diaphragm, true hernia of atypical localization, hernia of openings of diaphragm (esophageal opening, rare hernia orifices).

False congenital hernias (defects) are formed due to non-closure of the diaphragm junctions that exist in the embryonic period between the thoracic and abdominal cavity.

True rupture of weak zones of the diaphragm occur with increased intra-abdominal pressure and protrusion of the abdominal organs through sternocostal space (Larrey-Morgagni hernia - retrocostosternal hernia), lumbocostal space (Bochdalek hernia), directly in the underdeveloped part of the sternal diaphragm (retrosternal hernia). Contents of hernial sac can be omentum, transverse colon, preperitoneal fatty tissue (parasternal lipoma).

True hernia of atypical localization are rare and differ from the relaxation of the diaphragm by presence of hernial ring, and, consequently, the possibility of incarceration.

Hiatal hernia is separate group, since they occur most frequently, has particularly anatomy, clinical manifestations, and require certain principles of treatment.

Special cases of the rare hernias of other natural orifices are described (slit of the sympathetic nerve, the openings of the inferior vena cava).

A. Anatomy of the mediastinum and diaphragm.

Mediastinum - part of the chest cavity with borders: top – the upper thoracic aperture, bottom - the diaphragm, in front - the sternum, posterior – the spine, on the sides – mediastinal pleura.

Mediastinum is divided into: anterior, middle and posterior mediastinum.

The border between the anterior and middle mediastinum is the frontal plane, carried out on the front wall of the trachea, the boundary between the middle and posterior mediastinum is at the back of the trachea and the root of the lungs in a plane close to the front.

Content of the anterior and middle mediastinum: the heart and pericardium, ascending aorta and arch with its branches, the pulmonary trunk and its branches, the vena cava superior and brachiocephalic trunk, trachea, bronchi with surrounding lymph nodes, bronchial arteries and veins, pulmonary veins, thoracic portion of the vagus nerves lying above the level of the root; diaphragmatic nerves, lymph nodes, thymus gland in children and adults – fatty tissue, which replaces it.

In the posterior mediastinum are: esophagus, descending aorta, the vena cava inferior, azygos and hemiazygos veins, thoracic lymph duct and lymph nodes, thoracic portion of the vagus nerve, which lies below the root of the lungs; sympathetic trunk with the splanchnic nerves, nerve plexus.

In addition, by horizontal plane, which is held at the bifurcation of the trachea, the mediastinum is divided into upper and lower.

Roentgenanatomic analysis.

Direct projection. In direct projection the mediastinal organs form intensive, so-called, the median shade, which is mainly made by the heart and major vessels, which cover the rest of the projection organs.

The external contours of the shadow of the mediastinum are clearly distanced from the lungs, they are more convex at contours of the heart and straighten up some of the vascular bundle in particular at the right location at superior vena cava.

Upper part of mediastinum seems less intense and uniform, since trachea is projected in midline.

Mediastinal lymph nodes normally do not give a differentiated picture and visible only in enlargement, calcification or staining.

The shape and size of the median shadow is variable, and depend on age, constitution, phase of respiration and the position of patient.

When breathing median shade, changing its transverse size, does not perform significant lateral shifts. Side shifts of the median shadow with the rapid and deep breath is one of the signs of violation of bronchial patency.

Lateral projection. Anterior mediastinum in the X-ray image is projected between the posterior surface of the sternum and the vertical, held on the anterior wall of the trachea. In the upper part of it in adult the shadow of ascending aorta, anterior contour of which extends slightly anteriorly, well defined, directed upward and backward passes into the shadow of the aortic arch is seen. In children, the thymus gland is anterior to the ascending aorta. The area of light triangular form limited in front by sternum, below by the heart, in the back – by the ascending aorta, is called the retrosternal space. The high transparency of the retrosternal space should be considered in the recognition of pathological processes in the anterior mediastinum, as even massive pathological formation (prevascular enlarged

lymph nodes, tumors and cysts of the mediastinum) may give the shadow of low intensity as a result of the projection effect "weakened" by the air of the lung tissue.

Lower part of anterior mediastinum is filled with shadows of the heart, against which the vessels in the middle part and lingular segments are projected.

The middle mediastinum in the upper part is conglomerate, resulting in a clear image of the air column of the trachea, down from a projected shadow of the mediastinum to the root of the lungs. Lower part of middle mediastinum is also occupied by heart. In the back of cardio-diaphragmatic angle the shadow of the inferior vena cava is seen.

Posterior mediastinum is projected between the posterior wall of the trachea and the anterior surface of the bodies of the thoracic vertebrae. In the X-ray image, it takes the form of longitudinal strips of located enlightenment, against which in elderly people it is possible to see the shadow of a vertically descending aorta located a width of about 2,5-3 cm. Upper part of posterior mediastinum is blocked by muscles and scapulas, because of what it has low transparency. Lower part of posterior mediastinum, limited by the heart, diaphragm and vertebrae, has great clarity and is called retrocardial space. Against its background of vessels of key segments of lungs are projected.

Normally, the transparency of the retrosternal and retrocardial spaces in the bottom of its department is almost identical.

Anatomy of the diaphragm

Diaphragm is tendon-muscle formation, which separates the chest and abdominal cavities. Muscular part of the diaphragm begins in the lower aperture of the chest from the sternum, the inner surface of the cartilage of VII-XII of ribs and lumbar vertebrae (sternum, rib and lumbar diaphragm). Muscle fibres run upward and radially and terminate at the center of the tendon, forming the right and left dome-shaped protuberance. Between the sternum and rib division is sternocostal space (the triangle of Morgagni-Larrey) filled with fatty tissue. Lumbar and rib sections are separated by lumbocostal space (triangle Bochdalek). Lumbar diaphragm on each side consists of three legs: the outer (lateral), intermediate and inner (medial). The tendon edges of both the internal (medial) crura of diaphragm at the level of the I lumbar vertebra to the left of the median line restrict the opening for the aorta and thoracic lymph duct. Esophageal opening is formed in most cases by the right internal (medial) diaphragm leg, left leg is involved in its formation only in 10% of cases. Through esophageal opening the vagus nerves also pass. Through lumbar intermuscular openings of diaphragm sympathetic trunks and splanchnic nerves, and azygos and hemiazygos veins also pass. Opening for the inferior vena cava is located in the center of the diaphragm tendon.

Above the diaphragm is covered by intrathoracic fascia, pleura and pericardium; bottom - peritoneal fascia and peritoneum. The retroperitoneal part of the diaphragm is in contact with the pancreas, duodenum, kidney and adrenal glands, surrounded by the adipose capsule. To the right dome of the diaphragm the liver adjoins to the left - the spleen, the fundus of the stomach, the left lobe of the liver. Between these organs and the diaphragm are the appropriate ligaments. The right dome of the diaphragm is located above (fourth intercostal space) than the left (the fifth intercostal space). The height of standing of diaphragm depends on the constitution, age, presence of different pathological processes in the thoracic and abdominal cavities.

Blood supply to the diaphragm is provided by the upper and lower diaphragmatic artery, which depart from the aorta, muscular-diaphragmatic and pericardial-diaphragmatic artery, which depart from the internal thoracic artery, and six lower intercostal arteries.

The outflow of venous blood occurs in veins of the same name, by azygos and hemiazygos veins, as well as through the veins of the esophagus.

Lymph vessels of the diaphragm form several networks: subpleural, pleural, intrapleural, subperitoneal, peritoneal. For lymphatic vessels along the esophagus, aorta, inferior vena cava and other vessels and nerves that pass through the diaphragm, the inflammatory process may spread from the abdominal cavity into the pleural and vice versa. Lymphatic vessels divert lymph through the upper and posterior prelateroretropericardial mediastinal nodes from below - through the para-aortal and periesophageal. Innervation of the diaphragm is performed by diaphragmatic and intercostal nerves.

Static and dynamic function of the diaphragm are distinguished. Static function of the diaphragm is to support the pressure difference in the chest and abdominal cavities and normal relations between their organs. It depends on the tone of the diaphragm. Dynamic function of the diaphragm is caused by the action of moving of diaphragm during breathing in the lungs, heart and abdominal organs. Movement of the diaphragm provides ventilation, facilitate the flow of venous blood into the right atrium, contribute to an outflow of venous blood from the liver, spleen and abdominal organs, the movement of gases in the gastrointestinal tract, the act of defecation, lymph circulation.

In the clinic, the following **classification of diseases of the mediastinum and diaphragm** *is used***:**

Injury of mediastinum:

– Open;

– Blunt.

Mediastinitis:

– Acute;

– Chronic.

***Classification of tumors of the mediastinum according to the source of origin*:**

1. Primary mediastinal tumors.

2. Secondary malignant tumors of the mediastinum (metastases of malignant tumors of organs located outside the mediastinum, the mediastinal lymph nodes).

3. Tumors of the mediastinal organs (esophagus, trachea, pericardium, thoracic lymphatic duct).

4. Tumors of the tissues, which limit the mediastinum (pleura, sternum, diaphragm).

5. Pseudotumor disease (lymph nodes in tuberculosis, a disease of Besnier-Beck-Shaumian, parasitic cysts, aneurysms and malformations of major vessels, limited inflammation and some others).

Classification of tumors of the mediastinum, proposed by Schlumberger (1951)

It is based on morphological principle. All tumors Schlumberger divides according to the initial tissue of development: neurogenic tumors, mesenchymal, hematopoietic tissue and mixed tumor. However, this classification is not very useful for clinicians, because often in the mediastinum various kinds of tumor diseases develop, which require complicated differential diagnosis.

From these positions ***the classification of tumors and tumor diseases of mediastinum, proposed by Z. V. Golbert***, is of great practical value and clearly delineates the disease of this area.

1. Tumors, which are formed from the organs of the mediastinum (esophagus, trachea, major bronchi, heart, thymus, etc.).

2. Tumors, which are formed from the walls of the mediastinum (tumor of chest wall, diaphragm, esophagus).

3. Tumors, which are formed from the tissues of the mediastinum and located between the organs (extraorgan tumor).

Tumors of the third group are true mediastinal tumors. They in turn are divided on the histogenesis on tumors of the nervous tissue, connective tissue, blood vessels, smooth muscle tissue, lymphoid tissue and mesenchyme.

In addition, Z.V.Golbert allocates mediastinal cysts (from the embryonic germ foregut, coelomic and lymph) and tumors of tissue shifted to the mediastinum due to defects of embryonic development (rudiments of the thyroid gland, parathyroid gland, multipotent cells).

Mediastinal tumors are divided on benign and malignant. It should be noted, however, that in some cases the clinical course of morphologically benign mediastinal tumors, which originate from mesenchymal tissue, should be classified as malignant tumors because they tend to have infiltrative growth, recurrence and often result in the death of patients (some forms of lipomas, chondroma, mesenchoma).

Classification of tumors of the mediastinum, proposed by academician Petrovsky B.V.:

1. Cysts (epidermal, bronchiogenic, echinococcus)

2. Malignant neoplasms: a) primary — lymphogranulomatosis, sarcoma, melanoma, and b) metastases - lung cancer, melanomas, etc.

3. Benign (fibromas, lipomas, thymoma, tumors of vascular, neurogenic nature).
4. Goitre (diving, retrosternal, intrathoracic).

***Congenital cysts of the mediastinum*:**

Thymic cyst - usually localized in the upper anterior mediastinum;

- Cysts of the pericardium and their variety - diverticula, usually located in the inferior part of the anterior mediastinum - in cardiodiafragmatic corners;
- Bronchiogenic and duplication cyst - usually in the posterior and middle mediastinum;
- Dermoid cysts - usually in the anterior mediastinum;
- Teratomatous cyst - a kind of teratoma;

Cysts of the thyroid gland - usually in the middle mediastinum, upper portions of the anterior and posterior mediastinum.

Histo-genetic classification of primary mediastinal tumors:

I. ***Tumors arising from tissues that are normally found in the mediastinum***.

1. Tumors of the nervous tissue:
   a) of the nerve cells — ganglioneuroma, sympathogonioma;
   b) from nerve membranes — neurinomas, neurofibroma, neurogenic sarcoma.
2. Tumors derived from mesenchyme.
   a) of the fibrous connective tissue — fibroma, fibrosarcoma.
   b) from fat — lipoma, hybernoma, liposarcoma.
   c) tumors of vessels — hemangioma, chylangioma, hemangiopericytoma.
   d) from muscle — leyomyoma, leyomiosarcoma.
   e) of the lymph-reticular tissue — lymphoma, reticulosarcoma.

II. ***Neurogenic tumors of mediastinum***:

1. Benign (ganglioneuroma, neurolemmoma, neurofibroma);
2. Malignant (neurofibrosarcoma, neuroblastoma, ganglioneuroblastoma).
3. Neurogenic tumors of the type “sand-glass”, which are formed from the spinal roots and consist of two components: mediastinal and spinal, is localized in the spinal canal.

***Classification of vascular tumors of the mediastinum*:**

a) homologous (hemangiomas lymphangioma, glomus, angioleyomioma, hemangiopericytoma);

b) heterologous (angioendotelioma, peritelioma - malignant periendothelioma, angiosarcoma).

***Morphological classification of thymoma (O. Husselmann, 1978)*:**

1) epithelial;
2) lymphoid;
3) lymphoepitelial thymoma.

Histological types marked by letter are developed in the WHO classification: type A - medular-cell thymoma, type AB - mixed-cell thymoma, type B1 - predominantly cortical type of thymoma, B2 – cortical-cell thymoma, B3 - well differentiated carcinoma, the type C - undifferentiated carcinoma.

Classification based on the nature of growth and invasiveness criteria (proposed by N. Bergh et al. (1978) and modified by A. Masaoka et al. (1981)):

Stage 1. Macroscopically the tumor is completely encapsulated and microscopically no invasion of the capsule.

Step 2. Macroscopically invasion into surrounding fatty tissue or mediastinal pleura or microscopic invasion of the capsule.

Stage 3. Macroscopic invasion into neighbouring organs (pericardium, major vessels or the lungs).

Stage 4. Pleural or pericardial dissemination, lymphogenous or hematogenous metastases.

Myasthenia

Classification of myasthenia gravis of Osserman is the most common in the world (adopted at an international congress in 1959 in Los Angeles, and then it made some changes).

- Generalized myasthenia gravis; ·
- Myasthenia of newborns; ·
- Congenital myasthenia gravis; ·
- Benign with ophthalmoparesis or ophthalmoplegia; ·
- Family Children; ·
- Juvenile myasthenia gravis; ·
- Generalized myasthenia adults; ·
- Generalized myasthenia gravis (II type): mild (II a) - without bulbar disorders and severe (II b) - with bulbar disorders (in some variants of the classification there is an intermediate form - of moderate severity); ·
- Acute fulminant (III type); ·
- Late severe (IV type) with the early development of muscle atrophy; ·
- Ocular myasthenia gravis (type I).

Lobzin B. C. et al. proposed two classifications of myasthenia.

The **first** (in 1960) - on flow:

1. Acute onset with rapid development of symptom and further slow progress;
2. Acute onset, a longer (from 3 months to 1 year) development of a syndrome, with over remissions, but steady progress;
3. Gradual start, slow development over several years and the subsequent slowly progressive course;
4. Start of limited group of muscles and slow progress.

The **second** (in 1965) — on the localization of the pathological process with regard to violations of vital functions:

1. Generalized:
   a) without visceral disorders,
   b) violations of respiratory and cardiac activity;
2. Local:
   a) facial form (eye, pharyngeal-facial),
   b) the skeletal muscle form (without respiratory failure and respiratory disorders).

The most convenient for the practitioners is the clinical classification proposed in 1965 by B.M.Hecht.

It takes into account the nature of the disease, the degree of generalization of the myasthenic process, the severity of motor disorders and degree of their compensation on anticholinesterase medications that can sufficiently and accurately formulate diagnosis.

By the nature of flow:

1. Myasthenic episodes (single or relapsing-remitting course);
2. Myasthenic condition (i.e., constant flow);
3. Progressive course;
4. Malignant form.

Localization:

1. Local (limited) processes: ocular, bulbar, facial, cranial, trunk.
2. Generalized processes:

a) generalized without bulbar disorders,

b) generalized violations of breath.

On the severity of motor disorders: mild, moderate, severe.

On the degree of compensation of motor disorders on the background of anticholinesterase drugs: complete, sufficient, insufficient (bad).

Classification of hiatal hernias of esophagus, based on anatomical features of hernias:

I. Sliding (axial) hernia. Characterized by the fact that the abdominal part of esophagus, cardia and fundus portions of the stomach can herniate through the esophageal opening to the chest cavity, and return back to the abdominal cavity (changing position of the patient).

II. Paraesophageal hernia. In this case the distal part of the esophagus and cardia remain under the diaphragm, but the fundus part of the stomach gets into the chest cavity and is located next to the thoracic esophagus (paraesophageal).

III. Mixed version hernia. In the mixed form of hernia observed combination of axial and paraesophageal hernias.

There is also classification of hiatal hernia (HH) depending on the amount of penetration of the stomach into the chest cavity (I.L.Tegher, A.A.Lipko, 1965). The basis of this classification are X-ray manifestations of the disease.

There are three degrees of hernia:

- HH I degree - in the chest cavity (above the diaphragm) is abdominal esophagus, and the cardia - at the level of the diaphragm, stomach directly adjoins to the diaphragm;
- HH II degree - in the chest cavity is abdominal esophagus, and directly in the area of esophageal opening - is already part of the stomach;
- HH III degree - over the diaphragm are abdominal esophagus, cardia and part of the stomach (fundus and body, and in severe cases, antrum).

Clinical classification of V.H.Vasilenko, and A.L.Grebenev (in 1978)

1. Type of hernia: ·

- Fixed or unfixed (for axial and paraesophageal hernia);
- Axial - esophageal, cardiofundal, subtotal - and total gastric;
- Paraesophageal (fundus, antral);
- Congenital short esophagus with thoracic stomach (an anomaly of development);
- Hernia of different type (intestinal, omental, etc.).

2. Complications: ·

- Reflux esophagitis (morphological characteristics - catarrhal, erosive, ulcerative); ·
- Peptic ulcer of the esophagus;

– Inflammatory cicatricial stenosis and / or shortening of the esophagus (acquired shortening of the esophagus), the degree of severity;
– Acute or chronic oesophageal (esophageal-gastric) bleeding;
– Retrograde prolapse of mucous membrane of the stomach into the esophagus;
· Invagination of the esophagus into hernial part;
– Perforation of the esophagus;
– Reflex angina;
– Hernia strangulation (with paraesophageal hernia).

3. Generating reason: ·
– Dyskinesia of the digestive tract;
– Increase of intraabdominal pressure;
– Age-related weakening of the connective tissue structures, etc.

4. The mechanism of hernia: Pulsion; · Traction; · Mixed.

5. Associated diseases.

6. Degrees of severity of reflux esophagitis:
– Mild (mild symptoms, sometimes its absence (in this case the presence of esophagitis is noted on the basis of X-ray of the esophagus, esophagoscopy and biopsy));
– Moderate severity (symptoms of the disease is expressed, there is a deterioration of general condition and reduced efficiency);
– Severe degree (expressed as symptoms of esophagitis, and associated complications - primarily peptic structures and cicatricial shortening of the esophagus).

Relaxation of the diaphragm *- the thinning of the diaphragm and shift it along with the adjoining organs of the abdominal cavity into the chest. Line of fixation of diaphragm remains at the usual place.*

Relaxation is congenital (on the basis of complete aplasia or hypoplasia of the diaphragm muscle) and acquired (usually as a result of damage of phrenic nerve). Relaxation can be complete (total), when the entire dome of the diaphragm (usually left) moves to the chest and partial (limited) with thinning of any of its departments (usually the right anterior-medial).

In relaxation of diaphragm compression of lung on the affected side and mediastinal shift to the opposite side occurs, there may be transverse and longitudinal volvulus of stomach (cardiac and antral are on the same level), the torsion of splenic flexure of the colon.

Examination of patients

Damage of the mediastinum. Clinical manifestations of injury depend on which organ of mediastinum is damaged, the intensity of internal or external bleeding. In blunt injuries of mediastinum almost always an internal hemorrhage with hematoma formation is present, which can lead to compression of the vital organs (especially thin-walled veins of mediastinum). In compression of recurrent nerve dry cough, hoarseness of voice develop; in compression of sympathetic trunk - Horner's syndrome (ptosis, miosis, endophthalm).

Diagnosis is based on the data of history (investigation of the nature of injury), the dynamics of disease symptoms (development of subcutaneous emphysema,

respiratory disorders, etc.). On X-ray mediastinal shift in one direction or another, its expansion due to hemorrhage are found. Significant clearing of mediastinum shadow - symptom of mediastinal emphysema.

Treatment aims at normalizing the functions of vital organs (heart, lungs). It includes antishock therapy; in violation of the skeleton function of the chest (due to the impossibility of active respiratory movements) apply mechanical ventilation. Indications for surgical treatment: compression of vital organs with a severe disturbance of their function, rupture of the esophagus, trachea, main bronchi, the major blood vessels, the ongoing bleeding.

Open injuries are usually associated with damage of the organs of the mediastinum, which is accompanied by appropriate symptoms: bleeding, the development of mediastinal emphysema. In open injuries of the mediastinum the surgical treatment is indicated. The chosen method depends on the nature of the damage, the degree of infection in the wound, the general condition of the patient.

Inflammatory diseases of the mediastinum

Acute mediastinitis *— acute purulent inflammation of the mediastinal fat, which occurs mostly in the form of a phlegmon and much less in the form of limited abscess. The most common acute mediastinitis develops as a result of open mediastinal lesions, perforation of esophageal foreign bodies, instrumental examination of the trachea and main bronchi, dehiscence of sutures after operations on the esophagus, the spread of deep phlegmon of the neck to the mediastinum fat.*

Clinic and diagnosis: acute purulent mediastinitis develops rapidly, leading to severe condition of patients. Chills, high fever, tachycardia, shock, shortness of breath, thrusting and arching, chest pain and neck are characteristic. Localization of pain depends on the localization of the inflammatory process. Pain increase with extension of neck and averting of head back, pressing on the sternum (at the anterior mediastinitis), pressing the back part of the ribs (at the posterior mediastinitis). Because of the pain patients take a forced position (semisitting with the forward flexion of head), which reduces pain. In perforation of the esophagus, trachea or bronchus mediastinal, and then the subcutaneous emphysema develops. In general, the blood analysis reveals high leukocytosis with shift of formula to the left, the increase in ESR. In X-ray the expansion of shadow of the mediastinum is determined, in perforation of hollow organs - gas on the background of the shadow of the mediastinum.

Treatment: surgical, aimed primarily at addressing the causes that induce acute mediastinitis. In open injuries with the presence of foreign body in the mediastinum, rupture of the esophagus, trachea or main bronchi, dehiscence of sutures of esophageal anastomosis emergency surgery to remove the source of infection is performed. Operative intervention is finished by drainage of the mediastinum. Some surgeons recommend active aspiration of the purulent focus through the double-lumen drains imposed in the mediastinum for treatment of acute mediastinitisis. Narrow channel of the drainage tube is for antiseptic solution (dioxidine, chlorhexidine) in combination with broad-spectrum antibiotics, proteolytic enzymes. Wider channel is for fluid aspiration. Prolonged washing of purulent cavity with active aspiration allows to

remove nonviable tissue, pus, to suppress the growth of microbial flora. Depending on the localization of abscess drainage can be inserted through the cervical, transthoracic, transsternal or laparotomy approach. Important role in the treatment of patients with mediastinitisis belongs to massive antibiotic, desintoxication, infusion therapy, parenteral and enteral ("tube") feeding. "Tube" feeding – drop infusion of fluid with nutrients and food through thin tube, inserted into duodenum or jejunum using an endoscope.

Chronic mediastinitis (and mediastinal fibrosis) may be the result of acute mediastinitisis, as well as tuberculosis, actinomycosis, syphilis. Sclerosis (idiopathic) mediastinitis is characterized by enlargement of connective tissue formation in the mediastinum. Chronic mediastinitis observed much less than acute.

Clinic and diagnosis: symptoms usually are little - the organ temperature is normal or low-grade, pain in the chest little, although the general condition of patients gradually is deteriorated. The consequence of long-term chronic mediastinitisis may be fibrosis of mediastinal fat, enlarged intrathoracic lymph nodes, followed by compression of vital organs - superior vena cava, esophagus, trachea, major bronchi. The diagnosis is established according to history, according to X-ray studies (extension of the shadow of the mediastinum), serological blood tests for TB, actinomycosis, syphilis. X-ray examinations with contrast of the esophagus, superior vena cava are also used.

Treatment. Most patients are treated with medications aimed at suppressing of the inflammatory process that causes the development of chronic mediastinitisis (TB therapy, treatment of actinomycosis, and antibiotics, etc.). In idiopathic sclerosing mediastinitis radiation therapy and corticosteroids are used. Compression of vital mediastinal structures is indication to surgical treatment - removal of mediastinal lymph nodes, the elimination of cords and adhesions, which caused compression of the organs.

Tumors and cysts of the mediastinum

Primary tumors and cysts of the mediastinum. Their origin may be neurogenic (ganglio-neuroma, neuroblastoma, sympathycoblastoma, pheochromocytoma, neurinomas, and neurogenic sarcomas); mesenchymal origin (fibromas, fibrosarcoma, lipoma, liposarcoma, lymphangioma, hemangioma, angiosarcoma, etc.). There are also tumors that develop from the reticulum mediastinal lymph nodes (lymphosarcoma, reticulosarcoma, lymphogranulomatosis), thymus gland - thymoma (lymphoid, epithelial, mixed). In addition, mediastinal tumors develop as a result of violations of embryogenesis (dermoid cysts and teratoma, mediastinal seminoma, chorionepithelioma, intrathoracic goiter). Cysts of the mediastinum is divided into true (coelomic cysts and pericardial diverticula, bronchiogenic and duplication cyst), and such that develop as a result of violations of embryogenesis, parasitic (echinococcus).

A large variety of tumors and cysts of the mediastinum, resembling the clinical picture of disease, determine the complexity of diagnosis and differential diagnosis of these tumors. To simplify the diagnostic search must take into account the most frequent localization of various tumors and cysts of the mediastinum.

Clinic: in the early stages tumors and cysts of mediastinum can be asymptomatic and detected only on preventive X-ray examinations of the chest. Up to 40% of me-

diastinal tumors at the time of detection do not have symptoms. In 60% of patients the chest pain, shortness of breath, respiratory infections is present. In compression of the sympathetic nerve trunk appears Horner syndrom; tumor compression of the recurrent nerve causes hoarseness of voice; compression of the heart is manifested by pains in the left half of the chest, heart rhythm abnormalities. Neurogenic tumors, which grow as an "sound-glass" (when part of the tumor is located in the lumen of the spinal canal), causing compression of the spinal cord, are manifested by disturbances of various kinds of sensitivity, paresis, paralysis, dysfunction of pelvic organs.

In the compression of lumen of superior vena cava by mediastinal tumors characteristic syndrome "Syndrome of superior vena cava" is developed. In 93% of cases the cause of syndrome are malignant tumors of the lungs and mediastinum, in 7% - benign. Edema and cyanosis of the face, neck, arms, upper half of the body are clinically observed. As a result of increased pressure and rupture of the delicate walls of the small veins often develop nasal, esophageal, tracheobronchial bleeding. Characteristic also are headaches, confusion, hallucinations. The main method of diagnosis - the upper cavography. To elucidate the etiology of superior vena cava syndrome using polypositional chest x-ray, bronchoscopy, mediastinoscopy, CT are used (Fig. 31).

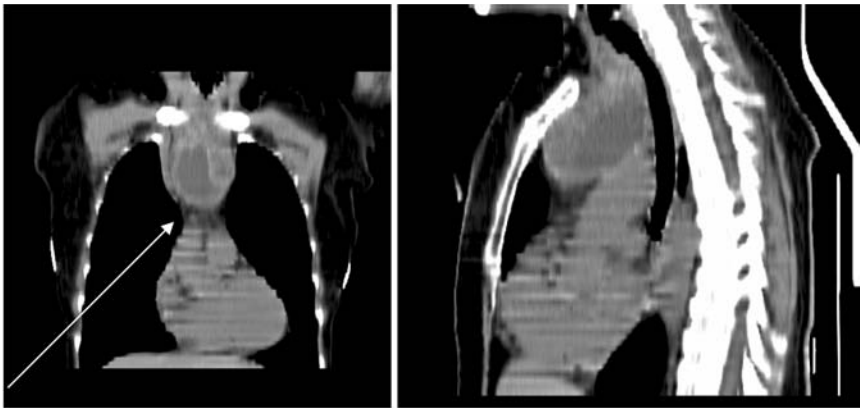


Fig. 31. CT: Tumor of the mediastinum

In addition to the compression symptoms caused by the tumor or its penetration into surrounding organs, symptoms associated with the structure of the neoplasm can be present. In tumors of the thymus gland the first place often takes the symptoms of myasthenia. Some groups of tumors cause various hormonal disorders. So for ganglioneuroma is characteristic arterial hypertension, with thymoma may develop Cushing syndrome. Malignant tumors of the mediastinum have a short asymptomatic period of development, rapidly increasing in size, metastasize, give the symptoms of compression of mediastinal organs, often effusion appears in the pleural cavity, characterized by fever.

Methods of examining of tumors and cysts of the mediastinum

The main method of diagnosis is complex of X-ray study (X-ray, polypositional radiography, tomography). Sometimes localization, shape, size of the tumor, taking into account sex, age and patient history are characteristic features that allow the correct diagnosis.

Mediastinal localization of tumor, its relationship with surrounding organs computed tomography helps in most cases. It provides an opportunity to obtain an image cross-section of the chest at any given level.

With difficulty in assessing the data of computed tomography, lack of capacity for its implementation, the need for biopsy material to clarify the nature of the pathological process special methods of investigation are indicated. These methods can be divided into two groups depending on the goals, what they pursue.

To define the localization, size, contours of tumor, its connection with neighbouring mediastinal organs the following methods may be used:

Pneumomediastinography — X-ray study of the mediastinum after the introduction into it of gas. Depending on the location of the tumor gas is injected through a puncture of the skin over the jugular fossa of the sternum, under the xiphoid process or parasternally so that the end of the needle does not touch the tumor. First gas is distributed into tissue of anterior mediastinum, through 45-60 minutes penetrates into the posterior. Against the background of the injected gas tumor is well-defined, can be detected its adherence with vessels or deformation as a result of the penetration of the tumor.

Artificial pneumothorax — the introduction of gas into the pleural cavity - perfromed on the affected side. In atelectasis it is possible for radiographs to distinguish tumor from mediastinal cysts.

Angiography — a contrast study of the heart, major arterial and venous trunks. Angiography gives the possibility to exclude an aneurysm of the heart and major vessels, to detect compression of the superior vena cava.

To obtain biopsy material and establish final diagnosis the following methods of research, which are performed in the operating room, can be used.

Transbronchial puncture is used most often for biopsy of lymph nodes, which cause compression of the bronchus. Under the supervision of bronchoscopy determine where is the compression of the bronchus, puncture of pathological tumor is performed.

Thoracoscopy allows to view pleural cavity, biopsy of mediastinal lymph nodes or tumor, located directly beneath the mediastinal pleura.

Transthoracic aspiration biopsy is used for tumors of the mediastinum, located in close proximity to the chest wall.

Mediastinoscopy — through a small skin incision above the sternum exposing the trachea in its course by finger makes the blunt channel in the anterior mediastinum for the introduction of a rigid optical system. Mediastinoscopy can take a biopsy of mediastinal lymph nodes. The effectiveness of this method reaches 80%.

Parasternal mediastinotomy — parallel to the edge of the sternum is made incision of 5-7 cm in length, cross one rib cartilage, tumor or lymph node of anterior mediastinum is isolated for morphological studies. Parasternal mediastinotomy is indicated in the presence of tumor or lymph nodes in anterior mediastinum.

The differential diagnosis of mediastinal tumors is needed with the non-tumor diseases of mediastinum - aortic aneurysm and heart, parasitic cysts, retrosternal goiter, tuberculous hypostatic abscess. Moreover, in the differential diagnosis should be borne in mind, some pulmonary diseases (lung cancer, adjacent to the mediasti-

num, paramediastinal encapsulated pleurisy, diaphragm hernia). All these diseases have their own distinct symptoms and with careful X-ray and endoscopy can be recognized.

Treatment. Main method of treatment of benign mediastinal tumors is surgical. Removal of mediastinal tumors is sometimes a difficult task because it requires intervention in the zone of vital organs. Intratracheal anesthesia is applied, which should be particularly careful due to shokogenic operation. The choice of approach depends mainly on the localization of the tumor. In tumors of the posterior mediastinum lateral thoracotomy is applied on the right or left in the intercostal space, corresponding location of the tumor. In tumors of the anterior mediastinum access can also be transpleural, anterior-lateral or side. Sometimes for large tumors of the anterior mediastinum, which occupy a central position, is useful the anterior access (longitudinal or transverse sternotomy). Such access provides great opportunities for operations in the anterior mediastinum.

Surgical treatment of malignant tumors of the mediastinum gives poor results.

Radiation therapy is well for systemic diseases — lymphogranulomatosis, reticulosarcoma and others with the localization of nodes in the mediastinum. In true mediastinal tumors — teratoblastoma, neurinomas and connective tissue tumors — radiation therapy is ineffective. Chemotherapy treatment of malignant tumors of the mediastinum is also true ineffective.

Prognosis. In benign tumors of the mediastinum after radical removal of the prognosis is good. In some cases, mesenchymal origin tumor (lipoma, chondroma, mezenhimoma), while morphologically benign, becoming malignant nature of the flow and prone to recurrence. The prognosis of malignant tumors of the mediastinum is bad.

The clinic, diagnosis and treatment of certain forms of tumors and cysts of the mediastinum.

Neurogenic tumors takes the first place (20% of all mediastinal tumors) on the frequency among all tumors and cysts of the mediastinum. They arise at any age and are more often benign. Their usual localization is posterior mediastinum. Neurofibromas develop from nerve fibres and their shells. Neurolemmoma develops from lemmocytoma (Schwann cells), ganglioneuroma - from the sympathetic trunk and contain both ganglion cells and nerve fibres. These tumors often develop in children. Mediastinal paraganglioma and pheochromocytoma derive from elements of the chemoreceptor apparatus of nerves and by the structure are similar to tumors of the carotid sinus area, contain chromaphynn cells and often possess hormonal activity, which causes arterial hypertension, manifested by frequent crises. Half of these tumors are malignant. Malignant neurogenic tumors are also neuroblastomas (sympathycogonioma, sympathoblastoma, ganglioneuroblastoma, neurogenic sarcoma).

Distinctive feature of neurogenic tumors are back pain, symptoms of compression of the spinal cord in the growing of part of the tumor into the spinal canal, with the development of paresis and paralysis. On X-ray neurogenic tumors are characterized by an intensive round shadow in the vertebral-costal sinuses with distinct outline, sometimes changes of vertebra, ribs are visible (uzuration). Hemorrhagic

effusion into the pleural cavity on the affected side is often in malignant tumors. To obtain the cellular elements of the tumor before operation is usually not possible because of the difficulties puncture posterior mediastinal tumor.

Treatment: Surgery - removal of the tumor through thoracotomy approach. With the growth of tumors in an "sand-glass" type resection of several arches of vertebrae and removal of part of tumor from the spinal canal is indicated.

Intrathoracic goitre - the most frequent type of tumor of the upper and anterior mediastinum. It is about 10-15% of all mediastinal tumors. In 15-20% of cases the node is located in the posterior mediastinum. Intrathoracic goitre occurs predominantly in women (75%) aged about 40 years. This type of goiter is attributed to aberrant goitre, which develops from dystopically located rudiment of the thyroid gland. The vast majority of patients has goiter located in the right parts of the mediastinum.

Clinically, the disease can manifest by compression of the trachea, major vessels, nerves; sometimes by symptoms of hyperthyroidism. In some patients the upper pole of intrathoracic goiter is available for palpation, especially when straining of the patient during swallowing. When X-ray define circular shadow with clear contours, the displacement of the trachea, esophagus in the opposite direction. The most informative is diagnostic scan with I131. Isotope accumulates in the thyroid gland, located retrosternal, which is seen on the scanning image. Due to the "compression syndrome", the possibility of malignancy operative treatment is indicated - removal of goiter by cervical or transsternal approach.

Thymomas are found in 10-12% of patients with tumors of the mediastinum, and among all tumors and cysts of the anterior mediastinum, they make up 20%. Thymomas are found mainly in middle-aged people. The term "thymoma" includes several different types of tumors - lymphoepithelioma, spindle and granulomatous thymoma, thymolipoma etc. Depending on the degree of differentiation of cells, they may be benign (in 50-65% of patients) or malignant.

However, the histological concept of "benign thymoma" does not always correspond to the nature of its growth, as reported cases of infiltrative growth of benign thymomas with penetration into adjacent organs. Men and women suffer equally often. In 1/3 of patients thymoma is asymptomatic. Disease, except for "compression syndrome", is presented by chest pain, dry cough, shortness of breath, aregenerative anemia, agammaglobulinemia, cushingoid syndrome. 10-50% of patients with thymoma exhibit symptoms of myasthenia gravis: acute weakness of skeletal muscles and the prevalence of violations of chewing, swallowing, respiration, development of muscle wasting. Myasthenia gravis is characterized by decrease of weakness after the introduction anticholinesterase drugs.

The most informative method of diagnosing is considered pneumomediastinography at which the tumor location, coming from the thymus gland, is revealed. In children the cysts of thymus can develop, which for a long time, are asymptomatic. Hyperplasia of the thymus gland is possible, which occurs mainly in childhood and young age and is often accompanied by myasthenia.

Etiology and pathogenesis of this disease has not been fully elucidated, but nevertheless clear relationship between hyperplasia of the thymus gland and myasthenia gravis is found. Removal of the thymus gland or the thymus gland with the tumor in 70-75% of patients leads to cure or significant improvement in the condition.

Pericardial cysts are developmental lesions (7% of mediastinal tumors). The most frequent localization of them - right or left (more rare) cardiodiaphragmatic corner. True cyst does not communicate with the lumen of the pericardium. If communication between the cavity of the cyst and cavity of the pericardium is present the diagnosis is pericardium diverticulum. Flow of disease is usually asymptomatic, and detected during preventive fluorographic examination. At large sizes cyst or compression of the heart in diverticulum may cause cardiac abnormalities - arrhythmia, cardialgia. Treatment - removal of the cyst.

Dermoid cysts and teratoma are observed in 5-8% of patients with tumors of the mediastinum. These tumors arise from violations of embryogenesis. Dermoid cyst develops from ectodermal elements, have thick walls of fibrous connective tissue. In the cavity of the cyst brown, viscous fluid, the elements of the skin, hair are often found. 10% of patients has calcinates. Teratoma arise from multiple germ layers and are composed of several different in the structure tissues, and the degree of differentiation of cells can be benign or malignant (10 - 20% of patients). Although these diseases are congenital, they are usually diagnosed in patients only in adulthood due to development of the pain or "compression syndrome". In 95% of patients these tumors are located in the anterior mediastinum. Treatment - removal of cysts or tumors.

Bronchiogenic and duplication cysts of mediastinum (7-8% of mediastinal tumors) appear during fetal development and are formed from dystopic intestinal or bronchial epithelium.

Bronchiogenic cysts may be localized in the mediastinum, and deep in the lung tissue. In the mediastinum, they often are very close to the trachea and major bronchi, usually behind the bifurcation of the trachea. Clinical symptoms occur in large cysts. The most characteristic symptoms of compression of the airways - a dry cough, shortness of breath, stridor breath. X-ray reveals round shadow, which is adjacent to the trachea. Especially good it is visible on pneumomediastinography. Rarely a cyst is connected with lumen of the respiratory tract and then on X-ray it can be seen the fluid level in the lumen, and in introduction of contrast agent into the bronchial tree it fills the cavity of the cyst. Surgical treatment - excision of the cyst. Indications are due to the danger of suppuration of the cyst, perforation of its wall with the development of mediastinitis, pneumoempyema, bleeding. From the walls of the cyst the growth of malignant tumor may start.

Duplication (enterogenic) cysts develop from the dorsal divisions of the primary intestinal tube, and are localized in the lower parts of the posterior mediastinum, close to the esophagus. On microscopic exam the structure of the cyst may resemble the wall of the stomach, esophagus, small intestine. Due to the fact that the inner layer of some cysts has gastric epithelium, which produces hydrochloric acid, there may be ulcers of the wall, bleeding from ulcers and their perforation. In addition, duplication cyst may become infected and then rupture into the pericardial cavity, esophagus, hollow viscus. The most informative method of diagnosis - pneumomediastinography. Connection between the pathological formation and trachea, bronchi, esophagus is revealed. Due to real possibility of complications operative treatment is indicated - removal of the cyst.

Mesenchymal tumors occur in 4-7% of patients with tumors of the mediastinum. They develops from fat, connective and muscular tissue, as well as from the endothelium of the vascular wall. In this group of tumors lipoma occurs most frequently. Favourite their localization - cardiodiaphragmatic right corner. On the x-ray homogeneous shadow that is adjacent to the shadow of the heart is determined. Artificial pneumothorax and pneumomediastinography are the most informative in the differential diagnosis of lung tumors, pericardial cysts. Using the latest it is seen that the pathological formation (lipoma) on all sides is surrounded by gas and has no connection with the pericardium and diaphragm. Benign tumors of mesenchymal origin are mostly asymptomatic, with the growth can reach very large sizes without the symptoms of compression of mediastinal organs. Surgical treatment - removal of the tumor.

Lymphoma

Occurs in 3-5% of patients with tumors of the mediastinum and in 20-25% of patients with all malignant tumors of mediastinum. Lymphomas develop from mediastinal lymph nodes. The most frequent localization of lymphoma — anterior mediastinum, although this type of tumor can damage the lymph nodes of any department of the mediastinum. All three types of lymphoma: lymphosarcoma, reticulocytoma and lymphogranulomatosis are characterized by malignant course. Symptoms at first are caused by intoxication — malaise, weakness, low-grade febrile temperature, loss of weight, itching of skin. In large packages of lymph nodes dry cough, chest pain, symptoms of compression of mediastinal organs are observed. The most informative radiological studies for the diagnosis are mediastinoscopy, mediastinotomy with parasternal lymph node biopsy. Surgical treatment is indicated only in the early stages of the disease, when the process locally affects a certain group of lymph nodes. At later stages of the disease radiation therapy and chemotherapy are used.

Secondary tumors of mediastinum - metastases of malignant tumors of chest or abdominal lymph nodes into the mediastinum. Mostly occur in lung, esophagus, proximal stomach cancer. Clinically frequently is asymptomatic. At first symptoms of the underlying disease and general signs of the cancer process are present. Only in large metastases different compression syndromes develops - often superior vena cava syndrome, Horner syndrome. The principles of diagnosis are similar in this case of primary mediastinal tumors. The presence of metastases in the mediastinum significantly reduces the effectiveness of surgical treatment and its usefulness. Prognosis is usually unfavourable.

B. Injuries of the diaphragm

Blunt injuries of the diaphragm occur in car accidents, falls from height, air contusion, compression of the abdomen. Rupture of diaphragm in these cases is due to the sudden increase of intraabdominal pressure. Injuries are more common in the tendinous center or at the point of transition of it into the muscle of the diaphragm. In 90-95% of cases there is rupture of the left dome of the diaphragm. Often at the same time there are injuries of the chest, pelvic bones, and organs of the abdominal cavity. Moving into the pleural cavity of the stomach, small or large intestine, omentum, spleen, part of the liver begins in the diaphragm ruptures and injuries as a result of negative intrathoracic pressure. Moving organs may occur either directly after the injury, and through any period of time.

Open injuries of the diaphragm happen in stab and gunshot thoracicoabdominal wounds. They are in most cases combined with damage to the chest and abdominal cavities.

Clinic presentation and diagnosis in the acute period is dominated by symptoms of related injuries (pleuropulmonary shock, cardio-vascular and respiratory failure, bleeding, peritonitis, hemopneumothorax, bone fractures). Symptoms of compression of the lungs and shift of mediastinal organs have the diagnostic value of. There may be incarceration of organs that have fallen into the pleural cavity.

Diaphragm injury may be suspected in the presence of tympanitis on percussion of the thorax, intestinal noises on auscultation with the development of symptoms of intestinal obstruction, hemo- or pneumothorax in abdominal injuries. X-ray study is the main method of diagnosis of diaphragm injuries. It begins with plain X-ray of chest and abdomen, and then if necessary in the later periods contrast X-ray is conducted during the study of the stomach, small and large bowel. In injuries of the right dome of the diaphragm the condition of the liver is judged according to X-ray, ultrasound and scanning.

Treatment: in ruptures and wounds of the diaphragm an emergency operation is indicated, which consists of suturing of the defect by the interrupted non-absorbable sutures after reposition of the abdominal organs. Depending on the prevalence of symptoms associated with injuries of the abdominal or thoracic cavity operation begins by laparo- or thoracotomy.

Relaxation of the diaphragm - the thinning of the diaphragm and shift it along with the adjoining organs from the abdominal cavity into the chest. Line attachment of diaphragm remains at the usual place.

Relaxation is congenital (on the basis of complete aplasia or hypoplasia of the diaphragm muscle) and acquired (usually as a result of damage to phrenic nerve).

Relaxation can be complete (total), when the the whole dome of the diaphragm (usually left) is affected and displaced into the chest and partial (limited) with thinning of any of its department (usually anteromedial right).

In relaxation of diaphragm compression of lung on the affected side and mediastinal shift to the opposite side occurs, there may be transverse and longitudinal volvulus of stomach (cardiac and antral parts on the same level), the volvulus of splenic flexure of the colon.

Clinic and diagnosis: limited relaxation of the right part is asymptomatic. In left-sided relaxation symptoms are the same as in diaphragmatic hernia. Due to the absence of hernial ring incarceration is impossible.

Diagnosis is based on the presence of symptoms of displacement of the abdominal organs into the corresponding half of the thorax, lung compression, shift of mediastinal organs. X-ray study is the main method of confirming of the diagnosis. On the diagnostic pneumoperitoneum displaced into the chest cavity organs are determine as the shadow of the diaphragm. Limited right-sided relaxation is differentiated with tumors and cysts of the lungs, pericardium, liver.

Treatment: in the presence of pronounced clinical symptoms surgical treatment is indicated. The operation is reposition of the displaced abdominal organs into normal position and the formation of duplicature of thinned diaphragm or strengthening by mesh, skin, muscle or musculo-periosteal-pleural graft (autotransplantation).

The paralysis of the diaphragm. Unilateral paralysis is usually caused by damage of the phrenic nerve as a result of trauma or tumor of mediastinum, but in half of the cases the etiology is unknown. Usually asymptomatic. The diagnosis is made by means of chest X-ray. Bilateral paralysis can be caused by damage of the upper cervical spinal cord, motor neuron disease, poliomyelitis, polyneuropathy, bilateral phrenic nerve injury after surgery of the mediastinum. The examination reveals suffocation. Paradoxical movement of the abdominal wall in supine position is characteristic.

Diaphragmatic hernia

Diaphragmatic hernia is movement of the abdominal organs into the chest cavity through defects or weak zone of the diaphragm. The actual hernia is characterized by the presence of hernial ring, hernial sac and the hernia contents. In the absence of hernial sac hernia is called false. In true hernia hernial sac is formed by parietal peritoneum, the top is covered with parietal pleura.

By origin hernia of diaphragm are divided into traumatic and nontraumatic.

Among isolated nontraumatic hernias are: false congenital hernias (defects) of diaphragm, true hernia of weak zones of diaphragm, true hernia of atypical localization, hernia of openings of diaphragm (esophageal opening, rare hernia openings).

False congenital hernias (defects) are formed by the diaphragm cleft compounds that exist in the embryonic period between the thoracic and abdominal cavity.

True rupture of weak zones of the diaphragm occur with increased intra-abdominal pressure and stepping through the organs of sternocostal space (Larrey's - Morgagni hernia - retrocostosternal hernia), lumbocostal space (Bochdalek hernia), directly into the underdeveloped part of the diaphragm sternum (retrosternal hernia). Contents of hernial sac can be omentum, transverse colon, paraperitoneal fatty tissue (lipoma parasternal).

True hernia of atypical localization are rare and differ from the relaxation of the diaphragm by presence of hernial ring, and, consequently, the possibility of incarceration.

Hiatal hernia is a separate group, since it occurs most frequently, has particular anatomy, clinical manifestations, and require certain principles of treatment.

Special cases of the rare hernias of other natural openings are described (opening of the sympathetic nerve, the openings of the inferior vena cava).

Clinic and diagnosis: occurrence and severity of symptoms of diaphragmatic hernias depend on the nature of the displaced abdominal organs into the pleural cavity, its volume, the degree of filling of displaced hollow organs, compression of them into the hernial ring, the degree of lung collapse and mediastinal shift, size and shape of hernial gate.

Symptoms: gastrointestinal, cardiopulmonary, and general. Factors that lead to increased intraabdominal pressure, increase the severity of symptoms due to the increased contents of hernial sac.

Patients complain of feeling of heaviness and pain in the epigastric region, the chest and hypochondrium, shortness of breath and palpitations that occur after meals. Often noted gurgling and rumbling in the chest on the side of herniation, strengthening of suffocation in horizontal position. There can be vomiting after meals by eaten food that brings relief. In volvulus of the stomach, which is accompanied by the bend of the esophagus, paradoxical dysphagia develops (passage of solid food is better than of liquid).

High diagnostic value has the direct dependence of severity of clinical symptoms and the degree of filling of the gastrointestinal tract.

Incarceration of diaphragmatic hernia is characterized by acute pain in the corresponding half of the chest or in the epigastric region, radiating to the back, shoul-

der blade. Incarceration of the hollow organ can lead to necrosis and perforation of its wall with the development of pyopneumothorax.

Diaphragmatic hernia may be suspected if a history of trauma, the above complaints, reducing of the mobility of the chest and smoothing of intercostal intervals on the affected side, diminishing of abdomen with large long-existing hernia, the tympanitis over the corresponding half of the chest, which change intensity depending on the degree filling of the stomach and intestines, intestinal peristalsis, or listening to the splashing noise in weakening or complete absence of respiratory noise, shifting dullness of the mediastinum in the unaffected side.

The final diagnosis is made by the X-ray examination. X-ray image depends on the nature and nunber of the abdominal organs, which moved into the chest, the degree of filling of the gastrointestinal tract. At the protrusion of the stomach a large horizontal level in the left half of the chest, which rises in fluid intake and eating, is seen. At the protrusion of loops of small intestine against background of pulmonary fields the specific areas of lightening and darkening are defined. Moving of the spleen or liver provides shadowg in the corresponding side of the pulmonary fields. In some patients dome of the diaphragm and abdominal organs, located above it, are clearly visible.

In contrast study of the gastrointestinal tract the nature of protruded organs (hollow or parenchymal) are determined, the location and size of hernial ring on the basis of the image of compression of protruded organs at the level of the hole in the diaphragm (a symptom of hernial ring) is specified. Some patients for further diagnosis need diagnostic pneumoperitoneum. In false hernia air can pass into the pleural cavity (pneumothorax on X-ray).

Treatment: Due to the possibility of incarceration of diaphragmatic hernia operation is indicated. In right-sided localization of hernia surgery is performed through a transthoracic access in the fourth intercostal space, for parasternal hernia upper midline laparotomy is better approach, in left-sided hernias transthoracic approach is indicated in the seventh - eighth intercostal space.

After dissection of adhesions, release of the margins of the defect in the diaphragm, displaced organs are reduced into the abdominal cavity and the defect is closed by interrupted sutures forming duplicature. For large defects alloplastic prosthetic material (nylon, Teflon, polyester, etc) is used.

In parasternal hernia (Larrey's hernia, retrosternal hernia) displaced organs are reduced, hernial sac is dissected and cut off at the hernia neck, and consistently U-shaped sutures on the margin of the defect of the diaphragm and the posterior leaf of sheath of the abdominal muscles, periosteum of the sternum and ribs are put.

In hernias of lumbocostal space defect of diaphragm is closed by transabdominal sutures with the formation of duplicature.

In incarcerated diaphragmatic hernia transthoracic access is recommended. After dissection of the ring the contents of hernial sac is examined. If protruded organ is viable, it is reduced into the abdominal cavity, with irreversible changes - resection. The defect in the diaphragm is closed with stitches.

Hiatal hernia

Hernias may be congenital or acquired, sliding (axial) or paraesophageal hiatal hernia (Fig. 32).

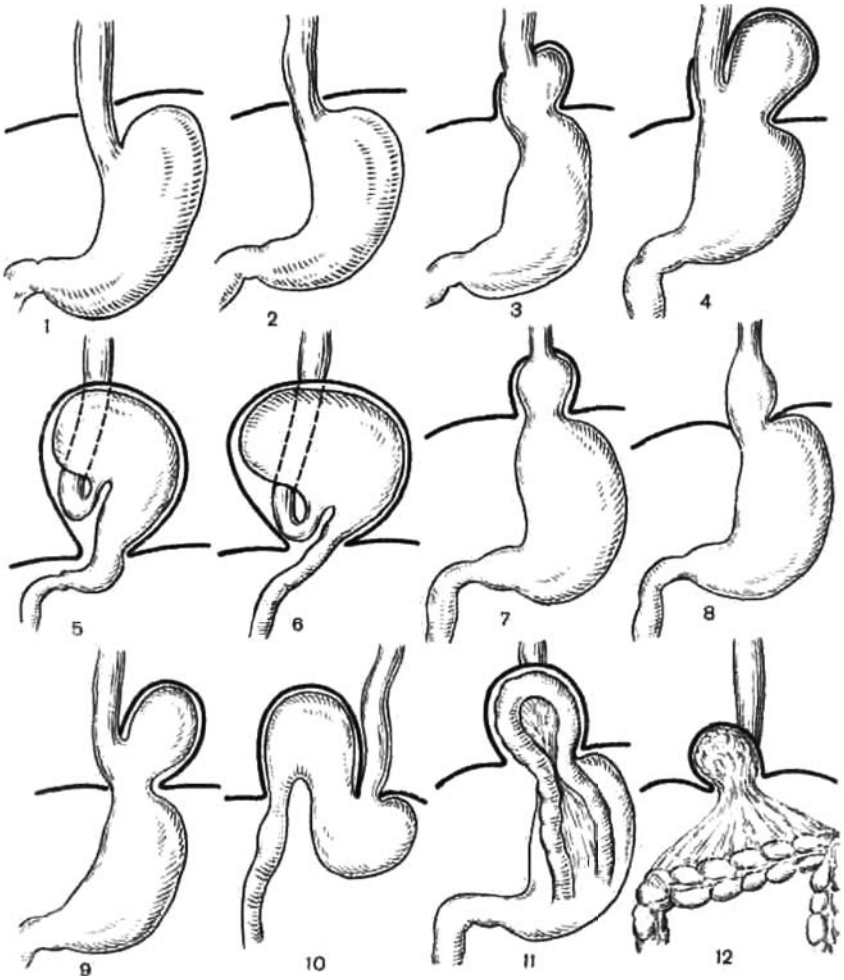


Fig. 32. Different types of hiatal hernias of esophagus (the scheme).
1 — location of the esophagus, diaphragm and stomach in normal onditions, 2 — esophageal, 3 — cardiac; 4 — cardiofundal 5 — subtotal gastric, 6 — total gastric; 7 — acquired short esophagus; 8 — congenital short esophagus; 9 — fundal, 10 — antral, 11 — intestinal; 12 — omental

Sliding hernia

Cardial part of stomach, located mesoperitoneally, moves above the diaphragm on the axis of the esophagus and participates in the formation of the wall of hernia sac. Sliding hernia classification of B.V.Petrovsky, N.N.Kanshin divides sliding hernia into esophageal, cardiac, and giant cardiofundal (subtotal and total gastric), which are accompanied by volvulus of the stomach in the chest cavity. Sliding hernias can be fixed and non-fixed. In addition, acquired short esophagus (cardia is located above the diaphragm at the level of 4 cm — I degree, higher than 4 cm —

II degree), and congenital short esophagus (thoracic stomach) are defined. By etiological factor sliding hernia may be traction, pulsion and mixed. Major importance in the development of acquired sliding hiatal hernias of esophagus has a traction mechanism, which occurs in the reduction of the longitudinal muscle of the esophagus as a result of vagal reflexes from the stomach and other organs in peptic ulcer, cholecystitis and other diseases. In development of pulsion hernias constitutional weakness of the connective tissue, involution due to age, obesity, pregnancy, factors contributing to increased intraabdominal pressure play key role. In sliding hiatal hernia insufficiency of the cardia, which leads to gastro-esophageal reflux, often develops.

Clinic and diagnosis: in sliding hiatal hernia symptoms are caused by reflux esophagitis. Patients complain of burning or dull pain behind the breastbone, at the level of xiphoid process, hypochondrium, the heart, which irradiates to the region of the scapula, the left shoulder. Often, patients are followed by therapeutists for angina pectoris. The pain increased in the horizontal position of the patient and during physical exertion, symptom of "shoe laces", that it is typical for gastro-esophageal reflux. The pain is accompanied by belching, regurgitation, heartburn. Over time, patients develop dysphagia, which usually is intermittent in nature and becomes constant in the development of peptic stricture of the esophagus. Frequent symptoms - bleeding, which is usually occult, blood or "coffee ground" vomiting, tarry stools is rarely seen. Anemia may be the only symptom of the disease. Bleeding occurs by diapedesis from erosions and ulcers in peptic esophagitis.

Decisive importance in making the diagnosis of hiatal hernia has X-ray examination (Fig. 33).

X-ray is conducted in the vertical and horizontal position of the patient and in the Trendelenburg position (with a lowered head end of the table).

In sliding hernias there is the continuation of folds of mucous membrane of the stomach and cardia above the diaphragm, presence or absence of shortening of the esophagus, the distortion of the Hiss angle, high confluence of the esophagus into the stomach, reducing of the gas bladder of stomach, reflux of contrast material from the stomach into the esophagus. Location of the cardia above the diaphragm is pathognomonic sign of cardiac hiatal hernia. In the accompanying reflux esophagitis esophagus can be extended and shortened. Esophagoscopy is indicated in peptic stricture of the esophagus, ulcers, suspicion on cancer, bleeding. It allows you to specify the length of the esophagus, to assess the severity of esophagitis, to determine the degree of insufficiency of the cardia, to exclude malignancy of the ulcer. The presence of gastro-esophageal reflux disease can be confirmed by intraesophageal pH metry (pH lower 4.0).

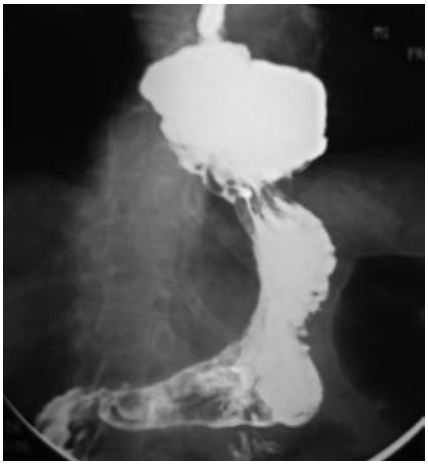


Fig. 33. Hiatal hernia of 2 degrees – cardiac part is 4 cm above the diaphragm

Paraesophageal hernias are divided into fundal, antral, intestinal, gastro-intestinal, omental. Cardia remains in place, but through the esophageal opening of diaphragm near the esophagus is a shift into the mediastinum of the stomach or intestine. Unlike sliding hernia in paraesophageal hernias may develop incarceration. Fundal hernia is often observed.

In paraesophageal hernias clinical picture depends on the type and content of hernia, degree of displacement of surrounding organs. Obturator function of the cardia is not impaired (no symptoms of gastro-esophageal reflux disease). The gastro-intestinal and cardio-pulmonary complaints may dominate. The most common is moving into the chest cavity of the stomach, manifested by pains in the epigastric region and behind the sternum arising after meals, dysphagia. In the incarceration there are acute pains, blood vomiting.

In paraesophageal hernias rounded enlightenment, sometimes with the level of liquid, is found in the posterior mediastinum on the background of heart shadow during X-ray of the chest. In contrast X-ray the location of gastric cardia in relation to the diaphragm is specified, the condition of protruded into the chest part of the stomach and its relationship with the esophagus and cardia is studied. Esophagogastroscopy is indicated in suspected ulcer, polyp or cancer of the stomach.

Treatment: in uncomplicated sliding hiatal hernia conservative treatment is indicated, which aims at reducing of gastro-esophageal reflux, reduction of the phenomena of esophagitis, prevention of increase of intra-abdominal pressure. Patients are recommended of sleeping with elevated head of the bed, to avoid postures that facilitate the occurrence of reflux, to follow regular bowel function. Meals should be small 5-6 times per day, the last meal before 3-4 hours to sleep. Depending on the severity of esophagitis mechanically and chemically sparing diet is appointed (Table 1a, 1b, 1 after Pevsner). Foods must contain a large number of proteins. Local anesthesing drugs, spasmolytics, antacids, prokinetics, sedatives and vitamins are prescribed. Surgical treatment in sliding hiatal hernia is indicated in bleeding, the development of peptic stricture of the esophagus, as well as the failure of long-term conservative therapy in patients with symptomatic reflux esophagitis.

In paraesophageal hernias, in all patients surgical treatment is indicated due to the possibility of its incarceration. The operation consists of reposition of the abdominal organs and suturing together of the edges of esophageal diaphragm (cruroraphy) behind the esophagus. In combination of paraesophageal hernia with complete failure of the cardia fundoplication after Nissen is indicated. In strangulated hernia patients are operated in the same way as in other diaphragmatic hernia.

> ***Syndrome of superior vena cava (SSVC) (syndromum venae cavae superioris)*** *— combination of dilation of veins of the chest, cyanosis and facial swelling with increased intracranial pressure caused by compression or thrombosis of superior vena cava.*

SSVC develops as a result of obstacles of blood flow or thrombosis, or in compression by the tumor from the outside. Often, both processes occur together. The blood from the head, neck, upper extremities and upper half of the chest comes into the vena cava superior (VCS). VCS passes through the narrow space surrounded by the middle mediastinal lymph nodes, trachea, aorta, right main bronchus, pulmo-

nary artery and the sternum. The anomalous increase of any of them can compress the vein and cause SSVC.

Prior to 1950, the most common "benign" causes of this syndrome were tuberculosis and syphilitic aortic aneurysm.

"Benign" causes account for about 20% of SSVC, and most due to thrombosis as a result of central venous catheterization or introduction of pacemakers (pacemakers). Other cases of the "benign" causes are fibrosis due to mediastinal infection, sarcoidosis, benign tumors and inflammatory diseases. Currently, 75-85% of cases of SSVC have a malignant etiology (lung cancer makes up to 80%). It is most common in small cell lung cancer. Other reasons are due to lymphoma and tumors of mediastinum primary, and their metastases.

SSVC was long considered as a life-threatening clinical condition that often justify empirical therapy (without providing reasons). However, this approach is challenged, although obstruction of the vena cava can lead to pronounced swelling of the upper half of body, causing headaches, dizziness, blurred vision. However, these symptoms are rarely progressive and lead to laryngeal edema, stroke, coma or death. In the absence of truly urgent situations it should take time to make the diagnosis. Diagnostic procedures, including bronchoscopy and mediastinoscopy not only safe, but also important for the purpose of the treatment.

Patients usually complain of shortness of breath, edematic face, feeling of heaviness in the head and often cough. Slow development of obstruction promotes the formation of collaterals and reduce the severity of symptoms.

Clinical manifestations of superior vena cava syndrome:

– Cough
– Feeling of lack of air (shortness of mixed character)
– Chest pain
– Swelling of the face and upper half of the body
– Dilation of the jugular veins
– Dilation of superficial veins of the chest, head, sublingual region.

The main serious complication - violation of the patency of upper breath ways at compression by tumor. It manifests by the development of dyspnea of inspiratory character (stridor breath), increasing in intensity until the stridor.

In 2/3 of patients stagnant blood filling of the veins of the neck and superficial veins of the chest, which shows collateral circulation through the intrathoracic veins, lateral veins of the chest wall, unpaired vein, paravertebral veins and veins of the esophagus is noted. In half of the patients swelling of the face and in 1/4 - pletora and peripheral cyanosis is noted.

In 15% of patients on chest X-ray there is no abnormalities, the expansion of the upper mediastinum is observed in 66% and pleural effusion – in 25% of cases. Tumor of the radix of the right lung was observed in 10-20% of patients. CT can help to determine the size of the tumor and often gives information about the presence and prevalence of thrombosis.

In diagnosis of cancer patients are offered treatment, depending on the histological type of tumor. Patients with small cell lung cancer, lymphoma or herminoma are given chemotherapy, since they are sensitive to it. In nonsmall cell lung cancer irradiation is usually applied. Surgical treatment is rarely indicated - only in retrosternal goiter and aneurysm. Anticoagulants are appointed in thrombosis.

Myasthenia crisis *— a dramatic increase in muscle weakness, which can cause paralysis of all extremities, respiratory failure, and various autonomic disorders.*

It may develop in patients with myasthenia after acute infection, trauma, physical and mental stress, surgical procedures, after receiving neuroleptics or tranquilizers with miorelaxants; there is the risk of development of crisis in the menstrual period. Myasthenic crisis may be a consequence of other diseases, infections, intake of some medicines.

Crisis can be provoked by exogenous (mostly influenza, acute respiratory infections, more rare intoxications, physical and mental strain) or endogenous reasons (endocrine changes, metabolic disturbances).

The basis of the crisis is block of neuromuscular conduction by the type of competition (curariform) block insensitivity as a result of addiction to anticholinesterase drugs during their chronic administration.

Clinically myasthenic crisis is presented by rapid generalizing myasthenic disorder with acute oculomotor and bulbar disorders. The latter can reach the degree of bulbar paralysis (myasthenic bulbar paralysis of Goldflam) with aphonia, dysarthria, dysphagia. Patients can not swallow, not only food, but also saliva. Appears difficulty in breathing, it is frequent, superficial. Paresis of the diaphragm, intercostal muscles, and paralysis of the limbs are developed. Psychomotor agitation, which will be replaced by lethargy, apathy, typical vegetative disorders: mydriasis, tachycardia, weak pulse, dry skin, or sweating, intestinal and sphincter paresis are characteristic. The intensity of symptoms increases rapidly, sometimes within 20-30 minutes. Severe cerebral hypoxia may develop, loss of consciousness and death occur.

Crisis varies in degrees of severity from mild to severe and fulminant.

Treatment. It begins with intravenous slow introduction of 0,5-1 ml of 0.05% solution of neostigmine methylsulfate, and then another 2 - 3 ml intramuscular, if necessary, neostigmine methylsulfate can be used again after 40-60 minutes. Combining with the introduction of 1 ml of 10% solution of ephedrine subcutaneously, with potassium drugs or potassium preserving diuretics (veroshpiron) is used for the best effect. Besides neostigmine methylsulfate, 0.01 g of oxazil per os or in the suppositorium and 0,06 g of mestinon (kalimin) per os, drugs with neostigmine methylsulfate and oxazil or oxazil with mestinon are used.

If, despite therapy by anticholinesterase drugs, the condition is not improving, emergency intubation or tracheostomy for mechanical ventilation is required. The constant suction of mucus from the nasopharynx and trachea is indicated, to reduce salivation and tracheobronchial secretions atropine is appointed: 0,5-1 ml of 0.1% solution subcutaneously.

Cardiac glycosides, caffeine, kordiamin, in vascular collapse - adrenomimetics (mezaton, adrenaline, dopamine), and infusion of reopolyglucin, polyglucin are used in the addition.

Chapter IV. Endocrine surgery

Euthyroid and toxic goiter

The endocrine system plays important part in the vital functions of organism and closely connected with all organs and systems. Many endocrine diseases develop because of violation of interrelation of the endocrine, nervous and immunological systems, at the change of genetic code, and also at violation of function of internal secretion and hormonreceptors influence, because of disorder of regulation of secretion, metabolism of hormones and defect in operations of hormones.

On prevalence of disease of thyroid gland has the first place between illnesses of the endocrine system. They are a heterogenous group, and the clinical picture of many of them is different depending on a stage and duration of disease.

It is most often widespread euthyroid and diffuse toxic goiter.

IV.1. Toxic goiter (TG)

A diffuse toxic goiter (Bazedov illness, the Greyvs illness) is the genetically determined autoimmune disease with the presence of diffuse goiter, ophthalmopathy and antibodies to tissues of thyroid gland. The disease is caused by the hyper secretion of thyroid hormones by a diffusely — enlarged thyroid gland and is accompanied by heavy disorder of function of different organs and systems, above all things cardio-vascular and nervous. The disease more frequent arises up at women at age 20-50 years (correlation of number of sick women and men makes 5:1, 7:1). City population is affected more often than country people.

The following classification of the thyroid diseases accepted.

Classification of goiter:

1. On distribution:
   1.1. Endemic;
   1.2. Sporadic (without pointing in a diagnosis);
2. By the form:
   2.1. Diffuse;
   2.2. Nodular (single or plural nodes);
   2.3. Mixed (diffusely-nodes).

Classification of diffuse toxic goiter:

1. By the degree of enlargement:

I — a gland is unnoticeable on an eye, an isthmus is palpable;
II – lateral lobes are well palpable, a gland is noticeable at swallowing;
III — the increase of thyroid gland is noticeable at examination («thick neck»);
IV — a goiter is clear visible, configuration of neck is changed;
V — goiter of big sizes.

2. By the degree of intensivety of thyrotoxicosis:

– Mild degree — pulse no more than 100 per/min., loss of mass of body on 3-5 kg, eye symptoms are absent or expressed insignificantly, increase of the absorption I 131 in 24 hours.

– Intermidiate degree — pulse 100–120 per/min, loss of mass of body on 8–10 kg, expressed tremor, increase of systolic and lowering of diastole pressure, eye symptoms are expressed, increase of the absorption I131 from the first hours, a workability is partly reduced.
– Severe grade — frequency of pulse exceeds 120–140 per/min., loss of mass of body achieves the degree of cachexy, adding disorder of function of liver, suprarenal glands, cardio-vascular system. Complete loss of workability.

Clinical classification of sizes of goiter, proposed by WOH (1994)

0 degree – a goiter is not present;

1 degree – sizes of lobes (lob) more than distal phalanx of thumb. A goiter is palpable, but is not visible.

2 degree — a goiter is palpable and visible by an eye.

Degrees of increase of thyroid (accepted in a clinic):

I degree – at examination a gland is not determined an isthmus palpable.

II degree — a gland is visible at swallowing, an isthmus and both lobes palpable.

III degree — a gland is clearly visible at examination, neck configuration is deformed, rounded, «thick neck», the lateral lobes of thyroid gland spreads up to border of m.sternoclaidomastoideus.

IV degree — deformation of neck, the lateral lobes of thyroid gland spreads beyond the borders of m.sternoclaidomastoideus.

V degree — the goiter of enormous sizes, disfiguring.

***Questioning of patient*:**

1). Complaints on the basic disease:
– increase of thyroid gland of a certain degree, exophtalmos (goggle-eyed), tachycardia increasing at the physicalexertion;
– feeling of pulsation of neck vessels, pulse beating in the neck area aching pain in the heart area; shortness of breath, which is the sign of cardiacinsuf- ficiency;
– nervousirritation,excitability,loweringofworkability,memory;
– sleepdisorders,theincreaseofbodytemperaturetosubfebrile;
– sexualdisordersofsphere(violationsofmenstrualcycleinwomen,lowering of libido and potency inmen).

2). Anamnesis of disease: in most cases development of diffuse toxic goiter takes place slowly, symptomatic grows gradually. The illness is slowly progressing; however there are the cases of sharp development of disease.

3). Anamnesis of life: the place of residence of patient (endemic areas) and domestic anamnesis is specified (due to genetic predisposition to this disease).

Clinical physical inspection (characteristic peculiarities at this disease):

1). General condition of patient is often moderately impaired. Consciousness as a rule is clear, the promoted irritating, lability of mood, verbosity, fussiness is marked. Constitutional features areasthenic.

2). Collection of information about original appearance of patient. Skin covers are tender, velvety. Wrinkles it is small, patients look younger than the years. General hyperhidrosis is typical; the skin is moist and warm. The hyperpigmentation of eyelids maybe present, as pigmentation in the places of friction with clothes, skin

itch is possible. Total hyperpigmentation may be seen sometimes. The hair is dry and thin,fragile, loss of hair is possible. Nails are fragile, thin, and transparent. There can be a pre- tibial myxedema - affection of skin of the front surface of shin, back of foot looks asdenseedemawiththeroughnessofskin.

3). Examination of the cardiovascular system.The tachycardia increasing at the physical exertion is characteristic. The tachycardia is permanent, does not change with the change of body position and ispresent in sleep. The pulseratevaries from 90 to 150 per/min, extrasystoles are possible. Patients older than 40 years may manifest with arrhythmia. The heart sounds of areloud, the reis functional systolic murmur. At the early stages of thyrotoxicosis the margins of heartarenot changed. The systolic arterial pressure is increased, but the diastolic is decreased.

4). Inspection of respiratory organs. The chest is normal. There can be a slight tachypnea, the reduction of inspiration volume, shortness of breath-the Bryson's symptom.

5). Inspection of abdominal region. Progressing loss of weight at the increase appetite. «Obesity Bazedov» is possible is increase of mass of body. There is increase of peristalsis of intestine at the expressed toxic goiter. Frequent diarrhea is possible. Sometimes spastic constipations. The attacks of stomach-aches, vomiting are possible.

6). Inspection of the state of bones–muscles systems.

a). There is widespread, moderately expressed osteoporosis at heavy and protracted thyrotoxicosis. Thyroid acropaty is possible — thickness of phalanges of hand's fingers.

b). One of the early and permanent signs of disease is a muscular weakness. It varies from mild fatigability to the expressed weakness up to the muscu- lar atrophy. The affection of lower limb girdle muscles is more expressed, rarer than upper limb girdle. Patients can not get up without help and to go up thestaircase.

Shereshevsky's symptom — a patient is unable to get up from squat position; Plummer's symptom — inability of patient tositinachairdueto thyrotoxic myopathy.

7). ***«Locus morbi»***. At examination of neck can be determined increase of thyroid gland, especially expressly it is possible to see at swallowing act. By palpation: a thyroid palpating on anterior and lateral surfaces of neck and may be found equal diffuse enlargement. Consistency of thyroid is more frequent soft- elastic, but can have a moderate density. Possible detect presence of one or a few nodes in tissues of diffusely increased gland – it is the mixed goiter. A thyroid gland is mobile at swallowing, is not adhesed with surrounding tissues. The degrees of increase of thyroid gland are indicated in the section of classifications.

There is no correlation between the size of goiter and expressed of thyrotoxicosis.

In diffuse toxic goiter the increase of neck lymph idnodesand pharyngeal tonsils is possible.

Sometimes it is possible hear a systolic murmur above the gland,accompanied with trembling detected by palpation. In another cases a Gutman's symptom is detected a high buzz tingnoi seabovea thyroid.

Ophthalmopathy changes. In the majority of patients we can see bulging eyes– thyrotoxic exophthalmos. As a rule, exophthalmos is bilateral and symmetric. The function of eyes is not affected, the diplopiaisabsent.

Some eye ***symptoms*** are observed at a diffuse toxic goiter:

– Abady's symptom — the spasms of m. levator of the upper eyelid so that the line of the sclera is seen over the cornea;
– Balle's symptom — complete disappearance of arbitrary motions of eyeballs with saving of reaction of pupil and automatic motions of eyes;
– Beysman's symptom — increasing of pulsation of arteries of retina;
– Brama's symptom – during the strong laughter of eye remain widely opened;
– Synger-Enroth's symptom — edema of the eyelids, especially of the upper eyelid near the supraorbital margin;
– Knys's symptom – anisocoria;
– Ellynek's symptom — pigmentation of skin of eyelids;
– Shtelvag's symptom — the rare and incomplete blinking;
– Koher's symptom — a upper eyelid moves up quick, than eyeball.

8). Leading clinical symptoms: the diffuse enlargement of thyroid gland — a goiter; tachycardia; thyrotoxic exophtalm.

9). On the basis of findings of questioning and clinical objective inspection of patient it is possible to propose the following clinical diagnosis: DIFFUSE TOXIC GOITRE of the I-V degree.

***According to standard the plan of addition investigations (laboratory and instrumental) of patient with diffuse toxic goiter includes*:**

1). Clinical blood test.
2). Clinical analysis of urine.
3). Biochemical blood test.
4). Level of TG.
5). Metabolism of iodine.
6). Immunological tests.
7). The TG radiometry.
8). Radioisotope scanning (scintigraphy) of TG.
9). Ultrasonic research of TG.

1. Clinical blood test: the changes are unspecific: it is possible anemia, leucopenia on a background lymphocytosis, insignificant increase ESR.

2. Clinical analysis of urine: lowering of secretion of creatinin.

3. Biochemical blood test: disorder of lipid exchange – lowering contents of cholesterol, lecithin, beta-lipoproteins, on a background the increase of level of free fat acids. Quite often increase of level of dextrose on an empty stomach.

4. Research of hormonal background: the increase of basal level of thyroid hormones is typical: T3 (norm 1,2-2,7 nmol/l), T4 (norm 70-120 nmol/l). General contains of thyroxin in the blood serum can make from 130 to 400 nmol/l and more depending on heaviness of disease. The level T3 is most informative, as the level T4 is normal sometimes. Also is marked decrease TTH.

5. Metabolism of iodine: a butanolextracted iodine (BEI) and iodine connected with proteins (CPI) are the basic markers of functional activity of thyroid. For a

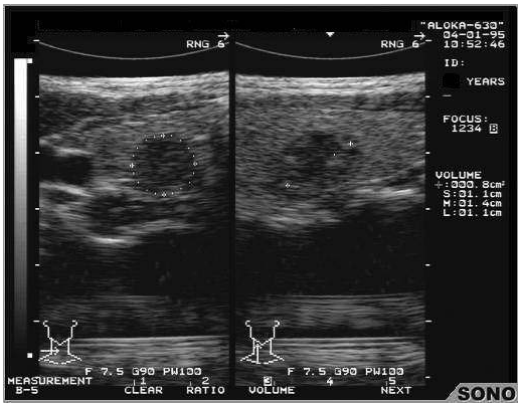


Fig. 34. Echographic picture of the right lobe node

diffuse toxic goiter the BEI increase is characteristic more than 440 nmol/l, CPI more than 670 nmol/l.

6. Immunological tests: the detection of thyroid stimulating immunoglobulins is characteristic (TSI). Criterion of treatment effectivety is lowering the level of the TSI on 35% and more from initial level.

7. Radiometry of thyroid: determination of degree of absorption of radio-active iodine by a thyroid. At a diffuse toxic goiter the increase of numbers of capture is marked: over 40 % in 24 hours.

8. Radioisotope scanning (scintigraphy): the increase of sizes of thyroid is characteristic; the capture of isotope of iodine at even its equal distribution is increased. This research allows found ectopic tissues of thyroid gland.

9. Ultrasonic research: the increase of sizes of thyroid gland is characteristic, hyperechogenic tissues, absence of nodes (Fig. 34).

***Differential diagnostics*:** at presence of characteristic symptomatology (The Merseburg's triad — a goiter, exophtalm and tachycardia) diagnostics of diffuse toxic goiter of difficulty does not represent. Difficulty during making of diagnosis may appear in case at presence of symptoms affectation of one system. As a rule differential diagnostics performs with: vascular dystonia, neuroses, rheumatic affectation of the heart, and atherosclerotic cardiosclerosis, toxic-allergic tonsillitis, encephalitis and other.

Formulation of clinical diagnosis (taking into according classification of disease, presence of complications and accompanying pathology):

1) basic - diffuse toxic goiter of the I-V degree;

2) complications (basic disease if they are);

3) concomitant pathology (if it is).

Management of patients with a diffuse toxic goiter

***Choice of medical tactic*:** it is necessary to begin treatment of patients with the diffuse toxic goiter of middle and heavy form in the hospital, if it is impossible, for a time, necessary appointed the bed regime up to achievement of compensation.

Treatment of patients with a diffuse toxic goiter can be performs by three methods:

1 – the protracted reception of thyroidstatic drags;

2 – administration of treatment dose of radio-active iodine in combination with thyroidstatic drags;

3 – by surgical operation with the preliminary reception of thyroidstatic drags.

The pathogenetically substantiated conservative the ray is directed on the lowering of the level of circulating thyroid hormones to normal.

1) The regime — a bed.

2) Diet – with a high energetic value to achievement of normal mass of body. Contents of albumens in the food ration 1-1,5 g/kg mass of body at normal content

of fats and carbohydrates. Food must contain the high amount of vitamins, mineral salts, is special calcium and iodine. It is necessary to limit acute dishes, tea, coffee, chocolate.

3) Medicinal therapy:

– thyrostatic preparations:
  a) derivative of thiouracil (methylurocil, propylthyouracil);
  b) derivative of imidasole (mercasolil, carbimasol);
– preparations of iodine;
– beta-adrenoblocers (anaprilin 160-240 mg/day);
– sympatolics (reserpin 0,1-0,25 мг/2-3 times per a day);
– corticosteroids (prednysolon 10-15 mgs/days, hydrocortison 30-75 mgs);
– preparations of potassium (panangin or asparcam for 1 tabl. 3 times daily);
– blocators callicrein-cinin systems (parmidin, prodectin for 0,25 g 3 times per a day);
– immunocorrector therapy (levamisol/decaris for 0,15 in days 3 days with interruptions for 14 days, during 4-6 months);
– preparations reducing are excitability of the nervous system (valerian, cardiovalen, tasepam).

Types of operative managerment and indications

At a diffuse toxic goiter the subfascial subtotal resection of thyroid gland by O. V. Nykolaev.

Indications to operative interference:

1. Uneffective medicinal therapy during 4-6 months.
2. Rapid increase of thyroid gland up to grade IV-V.
3. High density of thyroid gland with the symptoms of compression of the neck organs of.
4. Severe thyrotoxicosis.
5. Need to stop using mercasolil because of allergy, granulocytopeny etc.
6. Relapse of diffuse toxic goiter.
7. Complications of thyrotoxicosis (encephalopathy, ophthalmopathy).
8. Development of complications of diffuse toxic goiter with concomitant of diabetes mellitus.
9. In the nodular, mixed, atypical, aberrant forms of goiter.
10. In the period of pregnancy and lactation.
11. In the severe forms of diffuse toxic goiter, complicated with ciliary arrhythmia.

Possible postoperative complications:

– postoperative thyrotoxic crisis;
– damage, paresis of recurrent nerve (hoarseness or complete disappearance of voice);
– hypoparathyrosis, tetany;
– early hypothyrosis;
– late hypothyrosis;
– relapse of disease;
– postthyrotoxic encephalopathy.

Urgent conditions

In patients with diffuse toxic goiter thyrotoxic crisis may develop.

It happens rarely enough – in 1-12% of of patients. High lethality is characteristic (up to 25-30%) within the first 2 days.

Thyrotoxic crisis is an acute condition aggravating all symptoms of thyrotoxicosis.

It is necessary to begin treatment immediately, at the first clinical evidences. Specific therapy must be directed at the lowering of level of circulatory hormones, removal of nervous and cardio-vascular disorders, correction of suprarenal glands insufficiency, liquidation of hypoxia, hyperthermia and dehydratation.

Measures:

- 1% Lugol solution i/v prepared together with sodium iodide with 5% dextrose;
- mercasolile startingfrom 60-100 mg/per day;
- hydrocortisone — 100 mg every 6–8 hours or prednisolone — 200–300 mg/per day;
- cardial glycosides; strophanthin — 1–2 ml 0,05% with 5% solution of dextrose i/v;
- adrenoblockers: reserpin a 0,25–0,5 mg every 4 hours per os or i/m a 2,5 mg every 6–8 hours,
- sedative therapy by barbiturates, bromides.

List of medical diagnostic and treatment manipulations:

- palpation of thyroid gland.

Diagnostics and treatment of possible complications of diffuse toxic goiter.

4 types of complications of basic disease are selected:

Group 1: complications related directly to the medical management or operative intervention - thyrotoxic crisis, paresis of vocal cords, hypothyrosis and hypoparathyrosis.

Group 2: the remaining phenomena of thyrotoxicosis - a neurosis, encephalopathy, exophtalmos , changes in the internal organs.

Group 3: the consequences of the main disease - postthyrotoxic hypertonia, and postthyrotoxic encephalopathy with ophthalmopathy or without it, postthyrotoxic myocardiopathy, obesity, sexual disorders .

Group 4: relapse of thyrotoxicosis.

IV.2. Euthyroid goiter

The euthyroid (untoxic, sporadic) goiter - the diffuse or nodular enlargement of thyroid gland, may sporadically meet in regions with presence of deficit of iodine. The function of thyroid is not changed, from here and the name «untoxic goiter». In a clinical plan, to tactic of supervision and treatment big matter have cases of nodular euthyroid goiter. The term «nodular euthyroid goiter» unites all focal formation in a thyroid gland, different from each other by a morphological structure. Morphologically a nodular euthyroid goiter may have the following forms: follicle adenoma, colloid goiter, solitary cyst, cancer of thyroid, autoimmune thyroiditis with formation of false node. Distribution of disease has family character. More frequent it is fixed at women.

The classification of the TG diseases **accepted in a clinic is** described in a previous section.

At questioning of patient:

1). Complaints: on the increase of thyroid gland of a different degree. The local changes related directly to the goiter itself and forming complains such as sense of pressure in area of neck, suddenly attacks of coughing (due to pressure on laryngeal nerves), dizziness and headache caused by compression of big vessels in area of neck (there can be the edema of face). There can be the attacks of difficulty in breathing. Difficulties at swallowing because of compression of oesophagus are sometimes marked. May be present same vegetative disorder related to the irritation or suppression of sympathetic nervous trunk and other nervous trunks.

2). Anamnesis of disease: in most cases development of goiter takes place slowly, symptomatic grows gradually. The disease is making progress.

3). Anamnesis of life: the place of residence of patient (endemic areas) and family anamnesis is specified (due to genetic predisposition to the origin of this disease).

***Clinical physical inspection (characteristic features at this disease)*:**

1). Estimation of the general condition of patient. The general condition of patients depends on the form of euthyroid goiter and it sizes, and as a rule relatively satisfactory. Consciousness is clear, is adequate. Constitutional features are not present.

2). Collection of information about original appearance of patient. Skin covers are ordinary. There can be the edema of skin covers of face.

3). Inspection of the state of the cardio-vascular system. The characteristic changes are not present.

4). Inspection of organs of breathing. Thorax - without features. There can be difficulty of breathing, shortness of breath.

5). Inspection of the state of organs of abdominal cavity. The characteristic changes are not present.

6). Inspection of the state of bones- muscles system. The characteristic changes are not present.

7). «***Locus morbi***». At examination can be determined increase of thyroid gland, especially expressly it is possible to see at swallowing act. By palpation: the thyroid gland palpating on front and lateral surfaces of neck, and usually equally diffusely enlarged. Consistency of thyroid gland is more frequent soft and elastic, but can have a moderate density. At presence of one or a few nodes in tissue of gland possible say about a nodular (multinodular) goiter. A thyroid gland is mobile at swallowing, is not connected with surrounding tissues. The degrees of increase of thyroid are resulted higher.

8). Leading clinical symptoms. The diffuse increase of thyroid gland — *a goiter*, or the presence of nodes in tissues of thyroid gland — *a nodular goiter.* Combination of diffuse increase of thyroid with the presence of nodes is possible — *the mixed goiter.*

9). On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose a preliminary clinical diagnosis: Euthyroid (untoxic) diffuse/nodular/, mixed goiter of the I-V degree.

***In according to standard charts the plan of additional inspection (laboratory and instrumental) of patient with an euthyroid goiter includes*:**

1). Clinical blood test.
2). Clinical analysis of urine.
3). Biochemical blood test.
4). Research of the hormonal background TG.
5). Research of indexes of metabolism of iodine.
6). Immunological tests.
7). Radiometry of thyroid.
8). Radioisotope scanning (scintigraphy).
9). Ultrasonic research TG.

1. Clinical blood test: the changes are not usually present.
2. Clinical analysis of urine: the changes are not usually present.
3. Biochemical blood test: the changes are not usually present.
4. Research of hormonal background: it is typical normal level of thyroid hormones: T3 (norm 1,2-2,7 nmol/l), T4 (norm 70-120 nmol/l). The normal level TTH is also marked.
5. Research of indexes of metabolism of iodine: a butanolextracted iodine (BEI) and iodine connected with proteins (CPI) are the basic markers of functional activity of thyroid. For an euthyroid goiter is characteristic the normal values BEI and CPI.
6. Immunological tests: the changes are not usually present.
7. Radiometry of thyroid: determination of degree of absorption of radio-active iodine by a thyroid The changes are not usually present.
8. Radioisotope scanning (scintigraphy): the increase of sizes of thyroid at a diffuse form is characteristic; at nodular forms the presence of «cold nod» in tissues of gland is characteristic.
9. Ultrasonic research: the increase of sizes of thyroid and/or presence of nodes is characteristic.

***Differential diagnostics*:** As a rule need performs the differential diagnostic with: by chronic autoimmune thyroiditis (the Khashymoto goiter), fibroblastic goiter of Rydel, lateral and middle cysts of neck, tumors and cysts of mediastinum, malignant tumors of thyroid gland, metastases of malignant tumors in lymphatic nodes of neck.

Ground and formulation of clinical diagnosis (taking into account classification of disease, presence of complications and concomitant pathology): 1) basic: Diffuse (key/mixed) untoxic goiter of the I-V degree 2) complications (basic disease if they are) 3) concomitant pathology (if it is)

Treatment of patient with an euthyroid goiter

***Choice of medical tactic*:**

Treatment of patients with an euthyroid (untoxic) goiter is conducted by the following methods:

1). Conservative:
- protracted reception of microdoses of iodine
- application of medical dose of thyroid hormones

2). Surgical treatment with the preliminary reception of thyroid hormones.

The pathogenetic grounded conservative therapy is directed on prevention of enlargement of thyroid gland and maintenance of the euthyroid state (normal level of thyroid hormones).

1) The regime - general.

2) Diet – with sufficient maintenance in the food ration of albumins, fats and carbohydrates. Food must contain the increased amount of vitamins, mineral salts, and especially calcium and iodine. It is necessary to limit acute dishes, tea, coffee, chocolate.

3) Medicinal therapy.

At a simple untoxic goiter for the prophylaxis of development of nodular forms it is recommended to use thyroid hormones: thyroxin in a dose a 0,15-0,20 mg per days. Good results were got by a number of researchers at the use for these aims of indometacin on the following chart: during 2 weeks - 1 pill 3 times per a day, 2 weeks - 1 pill 2 times per a day, 4 weeks - 1 pill 1 times per a day. After above mentioned therapy compacting focuses into thyroid gland, this had been detected during the scanning as "cold nodes", diminished or completely resorpted. At case of developed nodular forms of goiter therapy by thyroid hormones leads in 55-65% of cases to diminishment of sizes as single and multiple nodular goiters. The optimum dose of administration of thyroid hormones is determined, from one side, by absence of clinical picture of overdose of hormones, with other – the level T3, T4 and TTH in the serum of blood.

Existent types of operative intervention and indication to them:

Hemistrumectomy is the method of choice of operative interference.

Indications to operative treatment:

1. Nodular and mixed forms.
2. Big sizes of goiter.
3. Symptoms of compression of surrounding organs.
4. Suspicion on the malignant regeneration of goiter.

Possible postoperative complications:

– damage, paresis of recurrent nerve;
– hypoparathyrosis, tetany;
– early up to 6 month of hypothyrosis;
– late hypothyrosis;
– relapses of disease.

Diagnostic and treatment of possible complications of euthyroid goiter. Distinguishing the next following complications of basic disease:

1 – hemorrhage in a goiter.
2 — calcification of goiter.
3 — inflammation.
4 — malignant regeneration.

Literature: 1) Bailey and Love's. Short practice of surgery. H.K.Levis and Co LTD, 1992. 2). Guidance on the endocrine diseases: V.N. Stick, T.A. Moyseenko, O.I. Moskalenko; under red. prof. B.H. Stick. Kharkov, Fact. – 1999. – 352 C. 3). Pudnizky L.V. Diseases of thyroid. «Peter». Saint Petersburg. – 2006. – 128 C.

IV.3 Inflammatory, dishormonal diseases andtumors of thebreast

***Mastitis** — one of diseases of mammary gland, which most often meets both in out-patient, and in-patient practice. Lactational mastitis (LM) makes about 95% of acute inflammatory diseases of mammary gland. LM occupies one of the first places in the structure of postdelivery purulent-inflammatory complications.*

Actuality of topic is also dictated by high rate of dyshormonal diseases of mammary gland among women, and by its progressive increase during the last decades, from one side, and also by constant growth of combined endocrine genital pathology and diseases of mammary glands - from other. To date the dyshormonal diseases of mammary gland (DHDMG, mastopathy) are diagnosed more than in the half of women of childbearing age.

In addition, it is impossible to forget that DHDMG can increase probability of development of cancer of mammary gland or to complicate its primary diagnostics.

The cancer of mammary gland (CMG) in Ukraine takes the first place among all of localizations of malignant tumors in women, and rate of disease continues to increase with every year. Being almost 30% morbidity from malignant tumors and 16-18% the death rate from them, CMG is considered one of the most frequent localizations of cancer in women.

Mastitis

Mastitis is acut inflammation of mammary gland.

Depending on the functional condition of gland and features of inflammatory process lactational and nonlactational mastitis are distinguished. 95% of acute inflammatory diseases of mammary gland is attributed to lactational mastitis.

Lactational mastitis is inflammation of mammary gland, developing in postdelivery period on a background of lactation. More often it develops on 2-3 week after delivery.

Epidemiology. Acute lactational mastitis develops in 2,4-18,0% of childbearing age woman and takes one of the first places (26-67%) in the structure of postdelivery purulent-inflammatory complications. More often develops after the first births. After the second births mastitis develops in 20% women, and after the third — only in single cases.

Etiology. Basic etiologic factors: microtrauma of nipples of mammary gland (cracks and excoriation of nipples, damage of gland skin), lactostasis.

An infection into mammary gland can be invaded by endogenous or exogenous way, more frequent it is exogenous. The cracks of nipple (50%), eczemas of nipple, small wounds which develop at rearing by breast, serve as entrance gates. Endogenous infections more often invade by lymphogenic way, but sometimes by galactogenic and hematogenic.

In 85% cases lactostasis precedes mastitis. At most patients its duration does not exceed 3-4 days. At the incomplete elutriation there are a lot of microbal bodies in ducts, which cause lactic-acid fermentation, turning of milk and damage of epithelium of mammary ducts. Turned milk obturates mammary ducts, lactostasis develops.

On development of lactational mastitis also has influence: toxicosis of the first or second half of pregnancy, anaemia, nephropathy, threats of abortion or premature births.

A certain role in pathogenesisy plays sensibilization of organism by various medical preparations, staphylococcus; autoimmune reactions.

In development of mastitis a basic role is played by staphylococcus Aureus which in 97% cases is revealed in pus and milk. These cultures are characterized by the expressed pathogenicity and resistance to most antibacterial preparations. In other cases mastitis can be caused by *epidermal staphylococcus, E.coli, streptococcus, Enterococcus, Proteus* and *P. aeruginosa*.

Also followings factors influence development of LM: decline of immunological reactivity of organism, failure to follow of the personal hygiene, excessive negative pressure, created in the oral cavity of child during feeding (principal reason of development of cracks of nipples of mammary glands).

Classification of mastitis on ICD-10 is used:

I. ***Depending on origin***:

1. Lactational (post-delivery).
2. Nonlactational.

II. ***Depending on flow of inflammatory process***

1. Acute.
2. Chronic.

III. ***On character of inflammatory process***:

1. Nonpurulent:
   – serosal;
   – infiltrative;
2. Purulent:
   – abscessing;
   – infiltrative-abscessing;
   – phlegmonous;
   – gangrenous.

IV. ***Depending on the side of lesion***:

1. One-sided (left or right-side).
2. Two-sided.

V. ***Depending on localization of abscess in a gland:***

1. Subtotal.
2. Hypodermic.
3. Intramammary.
4. Retromammary.

VI. ***On spread of process***:

1. Limited (1 quadrant of gland).
2. Diffuse (2-3 quadrants of gland).
3. Total (4 quadrants of gland).

***It is needed to define at questioning of patient*:**

1. Anamnesis of disease:
   – terms of onset and first symptoms of disease;
   – sequence of development of process (growth of edema or tumor, change of skin of gland, nipple, increase of axillar lymphatic nodes);
   – presence of pain syndrome, character of pain (one-side or bilateral, increase or appearance before menstruation);

– presence of excretions from nipples (one-side or bilateral), their character (colostric, serosal, with blood and other);
– previous medical treatment and its results; operations on mammary glands (concerning mastitis, benign tumors, cancer);
– traumas of mammary glands (for the exception of traumatic necrosis);
– disease of lungs, bones and other organs, that can be involved in distant metastases in the cancer of mammary gland.

2. Previous and concomitant diseases of genitals, liver, thyroid gland and other organs and systems which can cause number of hormonal violations in organism and stimulate tumor processes in a mammary gland.

3. Gynaecological and reproductive anamnesis:
– character and time of the first menstruation, climax, menopause, date of the last menstruation;
– age of patient at the the first, last pregnancy, number of births artificial and spontaneous abortions; in absence of pregnancies - reason;
– character of breast-feeding, its duration, amount of milk, presence of excretions from nipples after completion of feeding.

4. Sexual function:
– regularity of sexual activity;
– type of contraception (biological, mechanical, chemical, hormonal).

5. Social and living conditions and professional factors marriage status; presence of stress situations; professional harmfulness.

6. Diseases on the father and mother: endocrine, exchange, oncologic.

Clinical physical examination

On morphometric signs (height, weight, structure of skeleton) a somatotype patient is determined, the degree of fatty tissue is taken into account.

Skin are examined: elasticity, color, pigmentation. An elastic velvety skin testifies to the normal or increased estrogenic activity. A dry, hard, pale skin can testify about hypofunction of thyroid, ovaries, about the presence of anaemia or expressed avitaminosis. Pigmental spots can appear at pregnancy, dysfunction of liver, adrenals and others.

Character of hair distribution is evaluated: growth of hair on pubis (on a womanish or masculine type), thighs, middle line of abdomen, breast.

It is necessary to get the conclusion of gynaecologist about the condition of external genitals. Hypogenitalism, poorly expressed pigmentation, pale, dry mucous membranes of vulva and vagina, absence or smoothed out of rugosity of vaginal wall, specify on the decreased level of estrogenic activity.

Examination and palpation of thyroid: its sizes and presence of compressions.

***Examination of mammary glands of patient is conducted in position both hands up and down and in supine position. Thus pay a regard to the following signs*:**
– increase or diminishing of sizes of glands, their form, degree of development, symmetry;
– displacement upwards or aside, presence of mobility or fixing; violation of configuration of glands (pulled in, thrusting out);
– condition of nipple and areola (pulled in, detumors, ulcers);

– presence of excretions from a nipple, their character (colostric, brown, greenish-brown, ointment-like, serosal, with blood);
– condition of skin covers of gland; local or diffuse hyperemia of gland, distribution of it on nearby areas; local or total edema on the type of «lemon crust»; dilation of blood vessels; presence of the nodal indurations, ulcers of skin, fistulas, disintegration of tissues etc.

Examination of supraclavicular, subclavicular and axillar areas allows to reveal smoothed out of one of them, that can testify about the presence of enlarged lymphatic nodes. The special attention is paid to the presence of edema of upper extremity and neck which it can be caused by the block of lymphatic outflow.

Palpation of mammary glands

It is recommended to do palpation on 8-14th day of menstrual cycle, when it can be most informing, because at that time their edema and tenderness diminish.

Palpation is conducted in vertical and horizontal position of patient in the following positions:

– hands on thighs, with tension of muscles of thorax (fixing of gland is eliminated);
– hands on the back of head (palpation of lower quadrants, submammary folds, the changes of contours of gland, pulled in of skin;
– hands on shoulders of doctor, standing opposite (palpation of margins of pectoral muscle and axillar space).

It is necessary always to palpate both mammary glands, starting from healthy.

At first conduct superficial palpation by stroking by all palm in direction from periphery to the center. Superficial palpation determines localization of indurations.

Deep palpation of mammary glands is then performed, starting from upper-external quadrants, in direction clockwise for left side and anticlockwise for right gland.

Palpation determines diffuse or focal character of changes.

At presence of tumor node, such its descriptions are used:

Localization — central, external quadrants (upper or lower), internal quadrants (upper or lower). It is necessary to mark the presence of tumor in area of nipple, areola, in axillar, subclavicular, sternal processes of gland, and also in a submammary fold.

Form (rounded, tubular, irregular).

Sizes (diameter for round, maximal size - for the nodes of irregular form).

Consistency (dense, densely-elastic, softly-elastic, irregular).

Margins (contours) of tumor: clear, unclear.

Surface: smooth, uneven.

Connection with surrounding tissues, skin, nipple, muscle, mobility.

Palpation of regional lymphatic nodes (axillar, subclavicular) is then conducted, and also supraclavicular lymphatic nodes on each side. Thus the number of lymphatic nodes, their sizes, form, consistency, presence of tenderness, mobility, connection with surrounding tissues and in relation to each other are determined.

Complaints

As lactational mastitis is postdelivery pathology, the first signs of disease can appear in the first week after births, but at most woman mastitis develops on second - third week after births. Not uncommon is mastitis in a period up to 10 months after births. At most women lactostasis develops on 3-4 day prior to the origin of mastitis. Patients complain on pain and feeling of heaviness in a mammary gland, edema of gland, decline of lactation, weakness, chill and increased temperature of body, often — on head-ache.

The differences of flow of inflammatory process at mastitis from other acute purulent surgical infection are related to the postdelivery increase of functional activity and features of anatomic structure of gland:

– lobular structure;
– a lot of natural cavities (alvaeols and sinuses);
– wide network of mammary and lymphatic ducts;
– a lot of fatty tissue.

At acute mastitis distinguish two stages of inflammatory process: unpurulent (serosal and infiltrative forms) and purulent (abscessing, infiltrative-abscessing, phlegmonous and gangrenous forms).

Acute inflammatory process begin with the accumulation of serosal exsudate in intercellular spaces and leukocyte infiltration. In this stage a process yet can be reversed. However inflammation is not limited and has ability to spread on the nearby areas of mammary gland. From serosal and infiltrative forms lactational mastitis quickly passes to purulent with the simultaneous involvement of new areas of gland tissue. Purulent inflammatory process more frequent is intramammary, with involving of two and more quadrants of gland, quite often the protracted flow with frequent recurrences. Among purulent forms more frequent infiltrative-abscessing and phlegmonous forms occur.

In 10% cases LM has the latent flow which is conditioned by long antibiotic therapy.

On occasion as a local response to autosensibilization of organism to the organ-specific antigens (of milk or damaged tissue of gland) gangrene of mammary gland develops. Then an inflammatory process flows especially malignantly, with vast necrosis of skin and rapid distribution on cellular spaces of thorax.

Purulent mastitis is always accompanied by regional lymphadenitis.

The clinical presentation of acute mastitis depends on the form of inflammatory process.

Non-purulent forms

Serosal (initial) form is widespread. For this form inflammatory exsudate is characteristic without some focal changes in tissues of gland. Disease begin acutely with appearance of pain, feeling of heaviness in a mammary gland, chill, fever up to 38 °C and higher. Objectively: a gland is enlarged in volume, an insignificant hyperemia appears in the area of inflammation. Palpation in area of hyperemia is painful. The amount of milk is diminished. In blood - moderate leukocytosis and increased ESR. At the favorable flow of disease serosal form can accept abortive character; at inadequate or ineffective treatment this form makes progress with development of next phases and complications.

Infiltrative form of mastitis is continuation of first form and can be its short presentation. Usually flows as an aseptic variant, and at inadequate treatment passes to different purulent complications. Patients produce the same complaints, as well as at serosal mastitis, above-mantioned symptoms are preserved, but painful infiltrate without clear margins, areas of softening and fluctuation is found in tissues of gland. High temperature of body and chill, both at serosal and at infiltrative forms is conditioned by lactostasis, at which through the damaged mammary ducts milk, having a pyrogenic action, is absorbed into blood. During the desensitizing therapy and liquidation of lactostasis at most patients a temperature goes down to 37,5 °C. At absences of treatment and inadequate therapy serosal and infiltrative forms mastitis in 3-4 days pass to purulent.

Purulents forms

Abscessing form is characterized by appearance of focus of softening and disintergration with forming of the limited purulent cavity. At this form the condition of patients is worsened, general and local symptoms become more expressed, intoxication increases; temperature of body higher 38 °C; an edema and hyperemia of mammary gland increases. Objectively: painful infiltrate is palpated in a mammary gland (abscess); at 50% it occupies one more quadrant; for 60% of patients an abscess is located intramammary, rarer - subareolar or hypodermic, the positive symptom of fluctuation is marked; often in a center of infiltrate the area of softening influence is present.

Infiltrative-abscessing the form of mastitis flows heavier, than previous form. Characterized by fever up to 38 °C and higher, by the expressed hyperemia, edema, pain; dense infiltrate, consisting of great number of small abscesses of different size on the type of "bee honeycombs" is determined in tissues of gland (therefore the symptom of fluctuation is positive only in 5% cases). At 50% patients infiltrate occupies not more than two quadrants of gland and is located intramammary.

Phlegmonous form is characterized by worsening of the general condition and expressed signs of intoxication. Pain increases in a mammary gland, a weakness grows, an appetite goes down, the pallor of skin is marked, the temperature of body ranges from 38 °C to 39 C and higher. Objectively: a mammary gland is enlarged in a volume, edematous, hyperemic with cyanotic tint; a nipple is often pulled in. At palpation: of iron dense, painful, tissues are edematic, at 70% patients the positive symptom of fluctuation. At 60% patients 3-4 quadrants are involved in an inflammatory process. In a clinical blood test: WBC count is increased, hemoglobin of blood is decreased, shift of blood formula to the left. An proteinuria, presence of cylinders in a clinical urinalysis.

Gangrenous form the condition of patients is extremely severe, necrosis of skin and sublayong tissues is marked. This form is more often observed in patients, lately appealing for mrdical care. A purulent process flows with the rapid disintegration of tissues and distribution on the cellular spaces of thorax and accompanied with the expressed system inflammatory response. At most patients temperature is higher than 39 °C. The general and local symptoms of disease are acutely expressed.

All quadrants of mammary gland are engaged in an inflammatory process. On inspection: skin of mammary gland is of blue-red color, epidermis exfoliation with multiple bubbles, filled with hemorrhagic fluid, there are foci of necrosis. In a clini-

cal blood test is considerable leukocytosis, hemoglobin of blood is decreased. In a clinical analysis of urine an protein, cylinders and increase of number of red corpuscles are revealed.

Rare forms of mastitis

Acute nonlactation mastitis meets considerably rarer than lactation. An infection gets in tissue of mammary gland through mammary ducts or damaged skin. The hematogenic and lymphogenic ways of penetration are possible also. More frequent women suffer in age from 15 to 50 years, disease is not associated with a lactation.

Clinical symptoms of acute nonlactation and lactation mastitis are similar. At the same time, a purulent inflammatory process at nonlactational mastitis flows less heavily, more often as an abscess. At the same time, there is predisposition to the chronic flow and recurrence. Immunological reactivity of organism is suppressed rarely, problems, associated with violation of functional activity of mammary gland, are absent.

Mastitis of pregnant and mastitis of infants are distinguished separately among nonlactational mastitis.

Mastitis of pregnant is rare pathology, develops on a background of alteration of structure of mammary glands in the period of pregnancy.

Mastitis of infants arises up on 2-3 week of life, both in girls and in boys on a background of the physiological edema of glands, because of entering into blood of infant of sex hormones (estrogens) of mother. An infection is inoculated through the damaged skin or hematogenic. Intertrigo and purulent-inflammatory processes of skin or soft tissues often precedes disease.

Chronic mastitis occurs in women of any age. It develops in most cases as a result of the acute purulent inflammatory process in mammary gland, more often at treatment by the repeated introductions of antibiotics directly in the focus of inflammation. Thus very dense infiltrate with little abscesses is formed in tissues of gland, limited by the thick fibrotic capsule. The edema of tissues around infiltrate is not present, hyperemia is absent in this area. At palpation of infiltrate is little painful, not adhesed to skin.

Clinical diagnostics, as a rule, does not present difficulties. For diagnosis of acute mastitis it is necessary to take into account: complaints, anamnesis of disease and life, general and local clinical symptoms and functional condition of glands, the generally accepted laboratory and instrumental methods of examination.

According to standard charts, plan of additional examination (laboratory and instrumental) of patients with mastitis, mastopathy, benign tumors and cancer of mammary gland includes: Clinical blood test. Biochemical blood test. Clinical urine analysis. Blood type. Coagulogram. ECG. X-ray of organs of thoracic cavity. Determination of function of the external breathing. Level of sex hormones (at mastopathy).

With the purpose of early diagnostics, monitoring of the condition or clarification of diagnosis also are used:

- bacteriologic examination of milk from both mammary glands (qualitative and quantitative determinations of microbal bodies in 1 ml of milk);
- normally breast milk is sterile;
- cytologic exam of milk (count of hemocytes as markers of inflammation);
- determination of pH milk, activity of reductase;

– bacteriologic examination of pus, obtained during an operation or puncture, or in postoperative period — wound exsudate with determination of susceptibility of microflora to the antibiotics.
– US of mammary gland (evaluates the condition of glandular structures, character and localization of inflammatory process);
– US of mammary gland with simultaneous puncture of infiltrate by needle with wide lumen for the receipt of content (this method has a value only at abscessing mastitis).

Differential diagnostics

***Lacto stasis*:** the same as at mastitis, the body temperature is increased, a mammary gland is enlarged in volume, palpation is painful in all departments, but there never is an edema and hyperemia. After emptying of gland pain calms down, small size painless fragments with clear contours and fine-grained structure begin to be palpated.

***Mastitis-like form of cancer of mammary gland*:** also can have signs, similar to acute or chronic mastitis, however it is necessary to pay regard to substantial discrepancy: as a rule, patients with the cancer of mammary gland are more senior; association absents with delivery and lactation; process, as a rule, is one-sided; the clinic of inflammation usually does not develop so acutely, as at lactational mastitis; the presence of lymphadenopathy at cancer and absence of it is possible at nonpurulent forms mastitis; presence of specific symptoms of cancer of mammary gland, revealed at examination and palpation.

Nodal form of cancer of mammary gland with abscessing – a difference from acute mastitis are the points listed above + presence of the palpated (and/or instrumental) found node with the signs of internal destruction. In both last cases decision value has biopsy of tissue of gland.

***Formulation of clinical diagnosis*:**

1) specify at formulation of clinical diagnosis;
2) basic diagnosis;
3) complication;
4) concomitant pathology (2 and 3 – at presence of such).

Example of formulation of clinical diagnosis: Acute right-side nonpurulent lactational intramammary mastitis. Infiltrative form. Acute left-side purulent lactational intramammary total mastitis. Abscessing form.

Treatment of patients with acute mastitis

Choice of medical tactic. It is necessary to begin treatment of patients with mastitis since appearance of the first complaints about pains and edema of mammary gland. Lactostasis and nonpurulent forms of mastitis (serosal, infiltrative) are treated conservatively, at purulent forms (abscessing, infiltrative-abscessing, phlegmonous, gangrenous) apply operative treatment in in-patient conditions.

Conservative treatment of mastitis is possible at presence of the following cases:
– the condition of patient is satisfactory;
– duration of disease less than three days;
– temperature of body below 37,5°C;
– the local symptoms of purulent inflammation absent;
– infiltrate is moderately painful, occupies no more than one quadrant of gland;

– indexes of analysis of blood normal or unimportant changes.

In absence of positive dynamics during two days of conservative treatment, (that, as a rule, beginning of purulent inflammation specifies), operative intervention is indicated.

The scheme of conservative treatment of lactostasis and nonpurulent forms of mastitis.

1) Immobilization of mammary gland.

The obligatory expression of breast milk or sucking by breast pump from both mammary glands each 3-4 hours (8 times per day); at frist from a healthy gland, after - from a patient,

Intramuscular introduction of 2,0 ml of drotaverin (Nospanum) 20 minutes before expression of breast milk (3 times per a day, during 3 days through the equal intervals of time), and 0,5 ml of oxytocinum 5 minutes before expression of breast milk, that improves the lactation.

Antibiotic therapy under control of the susceptibility of flora (in staphylococcus infection it is expedient to appoint cefalosporines of the I generation (cefazolin); in the association of Staphylococcus with Escherichia, Clebsiella or Proteus - preparations of the II generation (cefuroxim, cefoxitin); at joining of the secondary infection — antibiotics of III and the IV generations (III generation: ceftriaxon; IV generation: cefepim).

Daily retromammary novocaine blockades with the wide spectrum antibiotics in half of daily dose.

Application of physical therapy treatment (ultraviolet irradiation, solluz, UHF-therapy or ultrasonic therapy and others; it is necessary to apply physical therapy treatment in positive dynamics after several days from the beginning of conservative therapy).

Desensitizing therapy (i/m introduction of antihistaminic preparations 2-3 times per day).

Semi-spirituous bandages on a mammary gland.

General therapy, symptomatic therapy and vitamins (vitamins of group B and C, polyvitamins).

Lactation is stopped if necessary, but only after liquidation of lactostasis.

Indications to ablactation:

1. severe flow of inflammatory process (gangrenous or total phlegmonous mastitis, sepsis);
2. two-sided mastitis;
3. recurrence of disease;
4. presence of reasons in which breast feeding of child after mother's recovery is impossible.

Ablactation by the tight bandaging of mammary glands is extremely dangerous (the production of milk after bandaging proceeds some time, that always results in lactostasis, and disturbance of circulation of blood in mammary gland - more often to development of severe purulent forms of mastitis), it is therefore recommended to use the special preparations.

The most effective for ablactations are inhibitors of secretion of prolactin: carbeholin (dostinex, USA) and bromokriptin (parlodel, Switzerland).

Expression of milk strengthens the secretion of prolactin, however its acute stopping can cause the recurrence of lactostasis. It is necessary to take into account these effects at ablactation.

Technique of implementation of retromammary novocaine blockade.

Position of patient: supine. A mammary gland is displaced by hand forward and up. Skin in the area of transitional fold is infiltrated by 0,25% solution of novocaine, than a long needle is entered parallel to thorax under posterior fascia of mammary gland into fatty tissue, in the area of projection of areola. Then through this needle 100,0-150,0 ml of 0,25% solution of novocaine with antibiotics is entered by syringe, controlling the position of needle in retromammary space:

– the acute end of needle stays still;
– no infiltration of tissue of mammary gland during introduction of solution;
– mammary gland must evenly rise by solution filling retromammary space.

After a blockade pain calms down, mammary ducts dilates, that facilitates milk expression, the high concentration of antibiotic is created in mammary gland. Milk is expressed through 20-30 minutes after blockade.

Surgical treatment of acute purulent mastitis

Basic principles of surgical treatment of acute purulent lactational mastitis:

– Choice of rational access to the purulent focus taking into account the necessity of maximal maintainance of function and original appearance of mammary gland.
– Radical debridement of purulent focus.
– Adequate draining, including - with the use of the flowing drainage system.
– Closing of wound, on possibility, by primary sutures, and at contra-indications - imposition of the secondary sutures or application of dermoplasty.
– Prolonged lavage of wound in postoperative period by solutions of antiseptics through the flowing drainage system.

A basic operation is opening and drainage of abscess

Types of anaesthetizing which can be used:

Local anesthesia:	General anesthesia:
superficial;	intravenous with the independent breathing;
infiltration;	inhalation mask with the independent breathing;
regional;	intubation anesthesia with APV;
loco-regional anesthesia;	intravenous with APV;
epidural;	inhalation anesthesia with APV;
spinal	combined (intravenous + inhalation) with APV.

At opening of abscess depending on the form of disease are applied: radial incision, paraareolar incision, on the lower margin of mammary gland - on Bardengaer at a retromammary form.

7-10 cm incision is done in radial direction, not reaching 2-3 cm to the areola of nipple. A finger, entered in the cavity of abscess, divides present strands and bridges. At presence of abscess both in the upper and in lower quadrants of mammary gland, incision can be done in lower quadrant and from it to empty an abscess, located in the upper quadrant of mammary gland. At difficulty of emptying of two abscesses from one incision it is necessary to do the second radial contra-incision.

After opening of abscess and pus removal cavity is carefully washed by solution of antiseptic. An operation is finished by draining of wound by glove-tube drainage («open drainage») and imposition of bandage with ointments of «Levosin» or «Levomekol».

Modern operation is radical excision of abscess together with a purulent capsule on the type of sectoral resection of mammary gland with the subsequent adjusting of the flowing drainage of wound through two incisions and closing of it by primary sutures at the location of abscess within the limits of one quadrant.

Closing of wounds by primary sutures after the radical debridement with the use of the flowing drainage system allows to reduce the terms of healing, improve the functional and aesthetically beautiful results of treatment.

Contra-indication to imposition of primary sutures is an anaerobic component of infection and large defect of skin, when to put together the margins of wound is impossible without tension.

In phlegmonous-gangrenous and purulents mastitis, threatening life of woman, the rotined removal of mammary gland (mastectomy) is indicated.

Treatment of chronic mastitis is surgical. An operation is conducted in obedience to principles of treatment of purulent lactation mastitis, infiltrate is excised within the limits of healthy tissues.

Complication at treatment of mastitis

General complications

The nonpurulent forms of mastitis well respond to treatment and, as a rule, have a favorable prognosis.

Acute purulent lactational mastitis can be complicated by development of phlegmon (distribution of process in the cellular spaces of thorax) and sepsis. At inadequate treatment an inflammatory process accepts the prolonged flow quite often; in 6-23% cases there are recurrence which needs the repeated operations; in 5-7% patients mammary fistulas are formed; severe sepsis develops in 5% cases. Lethality at purulent mastitis reaches 1%.

The consequences of the purulent mastitis can be considerable dysfunction and change of original appearance of mammary gland. Presence of any defects, including rough postoperative scars, negatively influences the psychoemotional condition of woman, and in a number of cases requires difficult plastic operations. Sometimes consequences and complications of mastitis result in the loss of ability to work.

Violation of lactation, changes of quality composition and infection of milk by pathogenic microflora at a purulent-inflammatory process of mammary gland is considered to influence normal development of child and can result in his infectious diseases.

Postoperative complications

Mammary fistula can be formed on a background of preserved lactation in 1% of women, operated with the use of the flowing drainage system and primary sutures of wound. As a rule, it closes independently during a month. If closing does not take place, lactation must be stopped. If the inflammatory phenomena is absent in mammary gland, milk fistula is not contra-indication to the natural feeding of child.

Suppuration of skin and hypoderm. Wound heals by primary tension in 90% cases, in other cases its suppuration or partial dehiscence of margins can occur. The

first signs of suppuration of the wound appear on 3-4 days of postoperative period, but sometimes before.

At the first signs of suppuration between the walls of wound it is possible to enter a thin tube (subclavian catheter) and through it carefully to wash wound by 3% penozone and 1% solution of dioxidin. In most cases for treatment of inflammation three-four daily washings are enough. If this did not result in improvement, it is necessary to drain wound (partly to remove the stitches) and make its systematic washing by antibacterial solutions.

Progress of purulent inflammation – more often is complication of nonradical necretomy. The condition of patients after operation is not improved, there are pains in mammary gland, though less intensive. The temperature of body go down insignificantly, but more often is higher than 37,5-38,0 °C. Painful infiltrate is palpated in area of purulent focus. Tendency absents in normalization of indexes of analysis of blood, leukocytosis and high leukocyte index of intoxication is preserved, ESR rises. Laktostasis does not pass. Purulent exsudate discharges through drain tube. In these cases in absenc of positive dynamics during two, maximum three days after operation on background of intensive antibacterial therapy the repeated operative intervention is indicated. Under the general anaesthesia it is necessary to take out all stitches for the wide revision of wound and perform the secondary debridement.

Prophylaxis of lactostasis and mastitis

It is necessary for the effective prophylaxis of mastitis:

1. To carry out the careful medical looking after:
   – all primipara;
   – women with pathology of pregnancy or births;
   – women, having anatomic changes of mammary glands;
2. Not to apply the tight bandaging of mammary glands, which was before used for ablactation.
3. To carry cotton brassiere (synthetic fibers irritates nipples and can result cracks). A bra must support well from below, but not to compress mammary gland.
4. To take into account physiological mechanisms stimulating the expression of milk. The early feeding of infant (in the first 30 minutes after birth) activates release into blood of prolactin and stimulates production of milk.
5. Application of circular shower on a mammary gland 20 minutes prior to feeding is possible.
6. To observe correct technology of expression of milk (hand method is most effective in the plan of prophylaxis of lactostasis). It is necessary to pay the special attention to expression of milk from the external quadrants of gland, where more often there are lactostasis and purulent inflammation.

Rational sanitary-hygienic and disease measures have large value in prevention of origin of mastitis:

1. Systematic exposure and sanation of transmitters of pathogenic staphylococcus among pregnant and medical personnel.
2. Planned active immunization of pregnant by a staphylococcus anatoxin in women consultations.
3. Preparation of mammary glands to feeding of child.
4. Punctual observance of sanitary-hygienic and disease rules of maternity permanent departments.

5. Urgent isolation of the exposed patients with postdelivery mastitis in separate wards.

6. Following of the personal hygiene by woman after delivery, and also correct techniques of the feeding and expression of milk.

7. Sanitary-educational work on warning of postdelivery mastitis.

Mastopathy, benign tumors and cancer of mammary gland

> ***Mastopathy*** *is fibrotic-cystic disease, which is characterized by violation of correlations of ephithelial and connective tissue components, wide spectrum of proliferative and regressive changes of tissue of mammary gland (determination of WHO, 1984).*

Last years more often the term «dyshormonal diseases of mammary glands» is used, because it reflects essence of pathological processes developing in organ.

Etiology of mastopathy

Predisposition of mammary glands to development of pathological dyshormonal processes is determined by dynamic instability of their morpho-functional condition, related to the high sensitiveness to various hormonal influences and to cyclic character of changes in the organs of the reproductive system.

Principal reason of origin of dyshormonal dysplasia of mammary glands is violation of balance of estrogens and progesteron, as a result the relative and later absolute hyperestrogenemia develops. Different gynecological and extragenital diseases also can assist this process.

Mastopathy and risk of transformation into the cancer of mammary gland (CMG) is generally recognized in our time, CMG on a background the of benign diseases of mammary glands develops in 3-5 times more often. However mastopathy is not obligatory precancer and not all forms of benign dysplasia can be considered as facultative precancer. If some of them rarely pass to malignant tumors (adenoma, fibroadenoma), other, for example, nodal forms with proliferation of epithelium, can in 30-40 times increase the risk of origin of carcinoma, and the separate types of tumors (phylloid fibroadenoma) can be transformed in a sarcoma.

Cancer of mammary gland (carcinoma of mammary gland on ICD) is an heterologous malignant tumor from glandular epithelium. In the structure of diseases of mammary glands CMG makes about 2%.

Etiology of CMG

Among causal factors first of all it is necessary to mention endocrine disturbances. This hypothesis is confirmed by the prevailing lesion in women as compared to men, connection with sexual life, time of the first pregnancy, amount of births, abortions, application of hormonal and chemical contraceptives, feature of lactation.

A hypothesis in relation to viral genesis of cancer of mammary gland is based on experimental data. In mice the «factor of milk» (Bittner), which was found to be one of oncogenic viruses, was studied. In humans the role of this virus is not well-proven. The role of the inherited factor in development of cancer of mammary gland is great enough. Increase of frequency of cancer of mammary gland in the female relatives of patients is the confirmation of it. Predisposition to the cancer of mammary gland is passed on inheritance.

The changes of phenotype of epithelium of mammary glands under regulator influence of endocrine factors belong to cancerogenesis at the level of cell. At the level of organ cancerogenesis is presented by those changes, resulting in diffuse and localized hyperplasia of mammary glands, known as «mastopathy» or «fibroadenomatosis».

Clinical classification of dyshormonal dysplasia (Methodical recommendations of MH of RSFSR (1985)

1. ***Diffuse mastopathy.***

– diffuse mastopathy with predominance of glandular component (adenosis);
– diffuse mastopathy with predominance of fibrotic component (fibroadenomatosis);
– diffuse mastopathy with predominance of cystic component;
– mixed form of diffuse mastopathy (fibrous-cystic mastopathy).

2. Nodal mastopathy.

3. ***Benign tumors and tumor-like processes:***

– adenoma;
– fibroadenoma;
– intraduct papilloma;
– cyst.

4. ***Special forms:***

– phylloid;
– tumor andother**.**

Clinical and anatomic classification of breast cancer

I. Nodal formII.
II. Diffuse form:
a) edematic-infiltrative;
b) mastitis-like;
c) erysipelas-like;
d) corset.
III. Rare forms:
a) Cancer of Pedzhet (eczema-like, psoriasis-like, ulcerous, tumor forms);
b) Atypical and other forms (primary-multiple cancer).

International clinical classification of breast cancer on TNM

T (tumor) is PRIMARY TUMOR
Tis — preinvasive carcinoma (carcinoma in situ).
T0 — tumor in a mammary gland is not determined.
T1 — tumor not more than 2 cm in the greatest size.
T2 — tumor from 2 to 5 cm in the greatest size:
T3 — tumor more than 5 cm in the greatest size:
T4 — tumor of any size with direct distribution on thoracic wall or skin.
Tx — it is not enough data for the assessment of primary tumor.
N (nodus) — REGIONAL LYMPHATIC NODES
N0 — the lymphatic nodes of axilla are not palpated on the side of lesion;
N1 — the unfixed lymphatic nodes are palpated;
N1a — lymphatic nodes consider as non-metastatic;
N1b — lymphatic nodes consider as metastatic;

N2 — the lymphatic nodes of axilla are matted together or with other structures and considered as metastatic;

N3 — subclavian or internal mammary lymphatic nodes consider as metastatic or there is an edema of hand.

NX — it is not enough data for the assessment of the condition of regional lymphatic nodes.

M (metastasis) — DISTANT METASTASES

M0 — there are not signs of distant metastases;

M1 — present distant metastases;

MX — it is not enough data for determination of distant metastases.

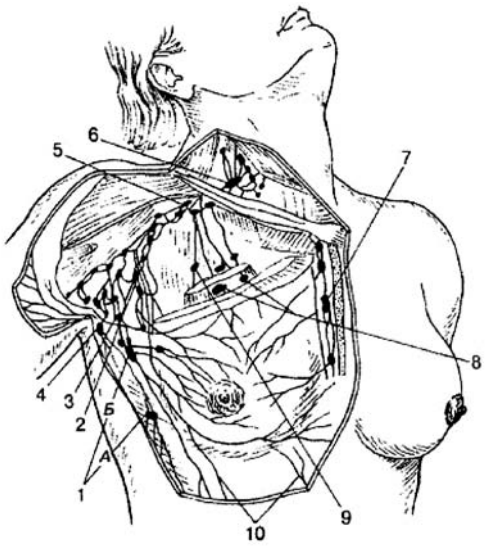


Fig. 38. Main groups of the regionary lymphatic nodes of mmamry gland: *1* – paramammarial (А – Bartels node, Б – Zorgius node); *2* – lateral axillary; *3* – central axillary; *4* – subscapular; *5* – subclavicular; *6* – supraclavicular; *7* – parasternal; *8* –Rotter node; *9* – retrosternal; *10* – lymphatic vessels in the epigastrium

GROUPING OF CMG ON THE STAGES

Stage	Primary tumor (T)	Lesion of lymphatic nodes (N)	Distant metastases (M)
0	Tis	N0	M0
I	T1	N0	M0
IIA	T0	N1	M0
	T1	N1	M0
	T2	N0	M0
IIB	T2	N1	M0
	TZ	N1	M0
IIIA	T0	N2	M0
	T1	N2	M0
	T2	N2	M0
	T3	N1,N2	M0
IIIB	T4	N1-3	M0
	T1-4	N3	M0
IV	T1-4	N1-3	M1

Features of inspection of patient with suspicion on mastopathy, benign tumors and cancer of mammary gland are presented earlier.

On determination of typical clinical symptoms of mastopathy and CMG during questioning and physical examination of patients it is needed to follow presented below data about the clinical presentation of disease (Fig. 38).

Features of physical diagnostics of CMG

At external examination and palpation of MG it is necessary to pay attention to the presence of skin and specific palpatory symptoms, pathognomonic for CMG:

«*Lemon crust*» symptom (lymphatic edema of papillary layer of derma because of blockade of lymph outflow from skin, with the point indrawings of skin in the places of localization of hair follicles).

«*Area*» symptom (rigidity of skin, infiltrated by tumor).

«*Umbilication*» symptom — an indrawing of skin, reminding umbilicus, in penetration of tumor into skin (caused bbby infiltration and shortening of Couper ligaments).

Yanishevskiy symptom — appearance of retraction of skin above tumor at raising of hands up (decline of elasticity and mobility of skin during infiltration of Couper ligaments).

Payr symptom — skin above tumor hardly undertakes into fold, these folds are uneven and «round» tumor.

Palm symptom — tumor is well determined by palpation of tissues glands between the fingers of hand. If after it palpate a gland flatways, pinning it against a mammary wall, a node disappears in mastopathy — negative symptom of palm. In cancer and fibroadenoma a tumor node does not change — positive symptom of palm.

Konig symptom — nodal tumor is well palpated in vertical position of patient. In transition to horizontal position node stops to be determined, is «lost» in surrounding tissues. Pathogenesis is similar with the symptom of palm.

Benzadon symptom — retraction of nipple at the compression of it by two fingers with the simultaneous drawing off deep into of tumor by the fingers of other hand (can be conditioned by the lesion of Couper ligaments or infiltration of excretory ducts).

Krauze symptom — protrusion of nipple and areola at CMG.

Pribram symptom — at traction of nipple cancer tumor is displaced after it.

Ri symptom — sign of tumor, fixed to the thoracic wall. At taking of hand on the affected side at 90 a node stays still.

Cholsted symptom — the mild compression of tumor between fingers in the case of colloid cancer gives the impression of break of capsule of tumor and spreading of jelly-like material, the size of tumor does not change.

There are also specific symptoms of metastatic lesion of lymphatic nodes at CMG:

Zorgius symptom — at the external margin of major pectoral muscle the enlarged node of Zorgius is palpated of sizefrom a pea to the hazel-nut.

Truaz'e symptom (node of Truaz'e) — a lymph nodus is located in the medial department of supraclavicular triangle in the place of confluence of internal jugular and subclavian veins. The lesion of this node specifies on preceding it metastases in

parasternal and mediastinal lymphatic nodes, that means considerable distribution of cancer.

The clinic of diffuse mastopathy is made from three basic symptoms:

1) pains in mammary glands — mastalgia;

2) presences of tumors in mammary glands;

3) pathological excretions from nipples.

These symptoms can be observed in different combinations and have a different degree of presentation depending on the form of diffuse mastopathy, phases of menstrual cycle and individual features of woman.

The difference of CMG from mastopathy consists in that the single symptom of CMG for a long time is presence of tumor in mammary gland, while pathological excretions from nipples are rarely present, and development of lymphadenopathy and pain syndrome more often specifies on advanced disease.

Description of separate types of benign

Diffuse mastopathy

1) **A diffuse mastopathy with predominance of glandular component (adenosis)** is the transitional condition between a norm and pathology, that is characterized by hyperlasia of glandular lobules, venous stagnation and edema of stroma. Young nullipara women (22-25 years) are ill more often. Basic complaints are a presence of the pain expressed more or less, edema and infiltrations in one or both mammary glands which enlarge in a few days to beginning of menstruation.

An adenomatosis can make spontaneous reverse development in the case of pregnancy and lactation. At unfavorable outcome, it is possible passing to the next stage - development of diffuse fibroadenomatosis or the small cystic form of diffuse cystic mastopathy.

2) **Diffuse mastopathy with predominance of fibrotic component (diffuse fibroadenomatosis - DFAM).** Diseases occur mainly in women 21-35 years. A pain syndrome in this form of mastopathy, as a rule, is moderately expressed.

In anamnesis of more than in 60% of patients there are more late menarche (after 15-16 years), disturbance of menstrual cycle, oligomenorrhea at short cycle (18-21 day) or with the increase of interval (35 and more days); periodic amenorrhea at stress conditions. Mastopathy in these patients often combines with the chronic inflammatory diseases of genitals, dysfunctional uterine bleeding, sterility. In anamnesis can be number of artificial abortions.

3) **Diffuse mastopathy with predominance of cystic component (small-cystic disease of mammary glands, illness of Reklyu).** Disease more often occurs in the malnourished patients under 25 years. Among patients many of them does not live by sexual life or suffers sterility. This pathology can undergo spontaneous reverse development at the of pregnancy and lactation. Pains, infiltrations in mammary glands, arising up to 8-10 days prior to beginning of menstruation, syndrome of predmenstrual exertion with the expressed vegetative and vascular symptoms are characteristic. After the beginning of menstruation pain diminishes or disappears, however complete softening of glands does not come.

4) **The mixed form of diffuse mastopathy (diffuse fibrotic-cystic mastopathy - DFCM)** is pathology which often meets in clinical practice. Parous women are ill,

as a rule, more senior 30 years. For the clinic of disease a pain syndrome is characteristic with an irradiation in shoulder, neck, hand, which arises up in premenstrual period; a presence of different size of infiltrates is in one or both glands. Often symptoms are colostrum or greenish excretions from nipples.

Nodal mastopathy

This form of mastopathy more often occurs in patients in age from 35 to 50 years. It develops, as a rule, on a background of diffuse changes of mammary gland and is considered as the next stage of development of disease. A morphological picture at this form of dyshormonal hyperplasia is as different, as well as difficult. The processes of hyperplasia of glandular lobules, cyst forming, fibrosis, proliferation of cellular elements covering cysts and ducts can prevail. Morphological changes in nodes are more expressed, than in diffuse mastopathy. At finding in the foci of proliferation atypical epithelium, this form of mastopathy is considered as precancer.

A clinical presentation is characterized by appearance in one or both mammary glands on a background of the diffuse mastopathy of one or few foci of infiltrates of irregular form with enough clear margins. Nodal infiltrates are usually less painful than diffuse proliferates, surface of them can be grainy, lobular, sometimes smooth, that testifies the presence of cysts. Nodal infiltrates are more expressly determined at the clinical inspection of patients in position upright. At pinning of infiltrate by palm against thoracic wall it stops to be determined (negative symptom of Konig). Skin symptoms are negative. On palpation of nodal mastopathy absence of substantial changes in association with a menstrual cycle is characteristic.

On mammogram the nodal form of mastopathy is characterized by presence of single or multiple infiltrates with uneven, unclear contours, without clear margins on background of the diffuse changes. Alteration of structural picture of gland is not observed. Mastopathy nodes unlike cancer have heterogenous structure. With the purpose of differentiation of cancer tumor from a nodal mastopathy the repeated examonations before and after menstruation are performed. After menstruation form, sizes and consistency of cancer nodes remain unchanging. Mastopathy nodes diminish and divides into separate fragments.

Treatment of nodal mastopathy is surgical. Sectoral resection with urgent histological examination of preparation is performed. Indication to operative intervention is determined by not so much principles of therapeutic tactic, how many by the risk of possible diagnostic error. It is thus impossible to be limited only by the removal of node. For complete recovery of woman it is necessary to conduct all complex of therapeutic measures, as at the proper form of diffuse mastopathy.

Benign breast tumors

An adenoma *(from greek aden — a gland) — a homologous benign tumor from a glandular epithelium. Grows expansively. Sometimes cysts appear in a tumor, in these cases it is talked about cystadenoma.*

On correlation of parenchyma and stroma of adenoma it is divides on:

– simple adenoma (parenchyma prevails above stroma);
– fibroadenoma (approximately equal correlation of parenchyma and stroma);

– adenofibroma (expressed predominance of stroma, reminds a fibroma on a structure).

Simple tubular adenomas in a mammary gland occur rarely. It is clinically difficult to distinguish them from fibroadenoma. It is a node of the rounded or oval form, of softly-elastic consistency, with clear contours, smooth surface, well mobile. Adenomas meet mainly in young women, often appear or significantly increase during pregnancy. A tumor is badly differentiated at mammography. US, aspiration or trepan-biopsy allow to specify a diagnosis.

Fibroadenoma *is an of homologous benign tumor, which occur more often than all. Young women up to 30 years, sometimes girls 16-18 years are ill, as a rule. Quite often fibroadenomas develop on background an adenosis or diffuse fibroadenomatosis.*

In the clinic of disease complaints in the presence of dense painless tumor in a mammary gland are characteristic, the exposure of which often causes in patients strong anxiety, insomnia and cancerophobia.

On histological structure tumor is presented as tubular fibroadenoma.

Depending on the diameter of ducts, forming a tumor, pericanlicular and intracanalicular fibroadenomas are distinguished.

Pericanlicular fibroadenoma is characterized by concentric increase of connecting tissue round the basal membrane of ducts. The lumen of ducts is narrowed, but preserved.

Intracanalicular fibroadenoma is characterized by lengthening of glandular ducts, prolapsed into their lumen of fascicles of collagen fibers, because of what the lumen of ducts becomes narrowed.

The fibroadenomas become malignant rarer than mastopathy — only in 0,5-1,5% cases when cancer in situ develops usually.

On palpation fibroadenoma is determined as the rounded form, dense, painless node, with smooth (sometimes — uneven) surface, is unconnected to skin, easily displaced in relation to surrounding tissues — the so-called symptom of «swimming» of tumor on gland. Not at any other disease of mammary gland this symptom can be revealed. The positive symptoms of Konig and palm are characteristic (in horizontal position and at palpation a node does not disappear under palm).

The phylloid tumor (phyhlloid fibroadenoma, cystofibrosarcoma) is a rare tumor. Occurs more often in women in age 40-50 years. Sizes can vary from small to giant. Rapid growth of tumor is characteristic.

At palpation has rounded or oval form with clear contours, smooth or polycyclic surface, densely-elastic or heterogenous consistency at which dense areas alternate with the foci of softening (the symptom of fluctuation is determined).

Intraductal papilloma. *It is a small (1 cm) homologous benign papillar tumor, arising up in the duct of mammary gland. In literature occurs also under the names «Bleeding mammary gland», illness of Mink.*

The basic complaint of patients is a presence of bloody, brown or greenish-brown excretions from nipples. It is explained that the superficially located papillary excrescences are easily injured at compression of mammary glands. Thus there is

destruction or tearing away of papillary excrescences and microbleedings, quite often an inflammatory process develops on this background. The amount of the selected blood is various. Sometimes it discharges involuntarily drops or even stream, in other cases — only at compression of gland. A lot of women complain on pain in a mammary gland in a premenstrual period, that, probably, is caused by the accumulation of secret in the cystic changed ducts.

Palpation. If a papilloma is located in a large duct, directly after a nipple or areola, at careful palpation of mammary gland in area of areola it is possible to define rounded tumor of softly-elastic consistency. At compression on it with blood drops appear only from one duct on nipple.

The most informing method of diagnostics of intraductal papilloma is ductography. Implementation of cytologic exam of excretions from nipples is absolutely obligatory.

Breast cyst

In it self is not independent nosology unit, but comes forward as a display of diffuse or nodal fibrotic-cystic mastopathy. However large solitary cysts have a characteristic clinical presentation, require specific diagnostic and more often — medical tactic.

Patients produce complaints about tumor in mammary gland, which is gradually increased in sizes during months or even years, or suddenly. Pain at cysts, as a rule, poorly expressed or absents. In those cases, when cysts reaches considerable size and pressure rises in them, the pain feelings can appear.

At palpation on background of the symptoms of diffuse fibrotic-cystic mastopathy the rounded form little painful tumors with a smooth or uneven surface are found. Consistency of cyst can be different and depends on intraductal pressure. Cysts can be soft, then fluctuation in them is revealed, but more often they have densely-elastic consistency. Non-infected cysts can be mobile in relation to surrounding tissues, reminding a fibroadenoma.

The «gold standard» of diagnostics of cystic tumors is ultrasonic exam and FNAB (fine-needle aspiration biopsy).

Instrumental diagnostics of mastopathy and cancer of mammary gland

Instrumental diagnostics is based on the radial and morphological methods of examination.

Presently for diagnostics of CMG is accepted «triple diagnostic standard» including:

1) physical examination;

2) mammography;

3) FNAB.

Test can be «triple negative» or «triple positive». Possibility of error of triple diagnostic test does not exceed 1%.

Mammography is X-ray investigation of mammary glands. It is the most effective method of early diagnostics of pathology of mammary glands, which allows to expose the foci of tumor from 0,3 cm in a diameter and indirect signs of onset of pathological process, and also to differentiate malignant and benign tumors.

Non-contrast mammography is performed on the special X-ray apparatus — mammographs. It is necessary to conduct mammography on 6-9 day after menstru-

ation. It is begun with standard X-ray in two projections at the dosed compression of organ. Most informing is combination of direct (axial) and oblique (under the angle of 45°) projections.

The X-ray image of CMG largely depends on the morphological and histological structure of tumor. According to X-ray signs two forms of cancer are distinguished: nodal and diffuse. On finding of pathological focus on the X-ray, the direct and indirect signs of nodal form of CMG are evaluated. Among direct signs tumor node and microcalcinates are characterize.

A tumor node in nodal local infiltrating cancer has the most characteristic, pathognomonic in typical cases picture. It is a focus of darkening of wrong stellar, polygonal or ameboid form, heterogenous structure, with unclear contours and characteristic striped «rays» on periphery, around focus there is alteration of structure of gland. Often there is a brightening area round a tumor, caused by substituting of glandular-fibrotic structures by fatty tissue.

Approximately in the half of cases of local infiltrating cancer microcalcinates are determined as small calcifications, located inside or in direct closeness to tumor node. It should be noted that exposure of microcalcinates even in absence of tumor node is enough suspiciously in regard to cancer. So, at presence of more than 15 microcalcinates on 1 cm^2, probability of cancer reaches 80%.

Restrictedly growing nodal cancer looks as a focus of the rounded or irregular form, heterogenous structure (from uneven growth of tumor) with unclear, sometimes polycyclic contours. On occasion a node is surrounded by pseudocapsule (because of compression of surrounding structures), that mimics the picture of benign tumor. With appearance of striated mammary ducts, caused by cancer lymphangiitis, a characteristic symptom «tail of comet» appears.

There are followings indirect signs: changes of skin - compression, infiltration, indrawing; changes of vascular picture (symptom of hypervascularization); alteration of tissues round a tumor - irregular form shadow strips appear which form the reticulated picture; «cancer path» between tumor and skin or nipple; changes of nipple. An important differentially-diagnostic sign is disparity of sizes of tumor at palpation and mammography, that is conditioned by the infiltrating growth of tumor and perifocal inflammation due to which on palpation tumor seems greater, than on images.

For the increase of low natural contrasty of tissues of mammary gland with the purpose of differential diagnostics of number of diseases methods with the artificial contrasting are used.

Pneumocystography is contrasting of cavity of cyst by air. After puncture of cyst and removal of its contents air in a volume, equal to the volume of contents, is entered into cavity. Then X-ray in two mutually perpendicular projections is performed.

Ductography is X-ray exam of the contrasted mammary ducts. It is a method of choice at presence of pathological discharge from a nipple. Bloody and serosal character of discharge is an absolute indication to ductography. Contra-indication is acute inflammatory process and clinically diagnosed CMG due to danger of dissemination of tumor cells into ducts. A method allows to reveal parietal excrescences in ducts (cancer or papillomatosis), to specify localization of focus, assess spread of process.

For the exposure of small (2-3 mm) parietal excrescences along the whole length of the duct system, the method of the double contrasting is used - the filling of ducts by the iodinated contrast, and after its removal - by air.

Computer tomography is used for the receipt of additional information in the local edema-infiltrating form of cancer, recurrences of tumor and localization of new tumors in retromammary space. A method is especially valuable for the diagnostics of metastases in the lymphatic nodes of axillar areas, and also for the evaluation of depth of penetration of tumor to thoracic wall.

Ultrasonic diagnostics complements and specifies the picture of pathological process, revealed clinically and by roentgenologic exam, and for examination of mammary glands in women to 35 years, the US is the method of choice. A method is widely used for differential diagnostics of solid tumors and cysts, and also allows to visualize small cysts (0,3-0,5 cm) at diffuse mastopathy, which come to light neither on palpation nor on X-ray.

For diagnostics of CMG ultrasound is effective at nodal forms and less informing at diffuse, where mammography takes advantage.

Application of Doppler US allows to increase efficiency of differential diagnostics, giving possibility to estimate the features of vascularization and rate of blood flow in tumor.

Thermography is a method of exam of skin temperature on which, due to heat conductivity of tissues, the changes of temperature are projected in the thickness of organ. High percent of false-negative results (to 30%) causes only additional value of thermography in diagnostics.

FNAB (fine needle aspiration biopsy) is a method of sampling of cellular material for cytologic exam obligatory at any suspicion on CMG

> ***FNAB*** *— technically simple, enough reliable and informing method (80 % coincidences of cytologic and histological diagnoses). Stereoscopicmammograph or ultrasound guided biopsy allow to insert needle into the focus of 1 mm.*

Indication to FNAB:

- exam of not palpating tumors;
- aspiration of content of lactic cysts; differential diagnostics of benign and malignant tumors.

Contra-indications are absent.

Cytologic verification has value only in the case of positive result (that, if CMG is revealed). The negative result of FNAB cannot be the basis of planning of therapy, because does not exclude the presence of cancer.

Thick cutting needle (trepan-biopsy) biopsy allows to get the column of tissue for histological exam, and also for determination of sex chromatin and hormonal receptors.

Exfoliative cytology (smear-imprint) of excretions from nipples is used for the exposure of malignant cells at differential diagnostics of proliferative processes of the ductal system.

Incisional biopsy is method of sampling of material for histological exam, at which small part of the tumor, sufficient for the receipt of conclusion, is undertaken only.

Excisional biopsy (usually - with urgent histological exam) is the final and decision stage of diagnostics of CMG (in 99% cases gives reliable conclusion about nature of process). It is usually performed in the volume of sectoral resection under anesthesia, if necessary (at confirmation of diagnosis of CMG) must be converted into radical intervention (about possibility of such tactic a patient must be warned). So pathological tumor is removed fully.

Indications to the express-biopsy: revealed by instrumental methods not palpating tumors, suspicious on CMG; doubtful or negative results of biopsy at presence of clinical and instrumental signs of cancer; atypical location of tumors which are not revealed on X-ray; nodal mastopathy with proliferation of epithelium; intraductal papillomas with proliferation of epithelium; solitary cysts with parietal excrescences, proliferation and atypical epithelium or cyst with a tendency to the increase and filling after the third puncture.

For diagnostics of CMG a large value has determination of tumor markers — macromolecules, appearance and change the concentration of which depends on an origin and growth of malignant tumor in patient. Tumor markers can be divided into two types:

Cellular tumor markers — antigens connected with cellular membrane, receptors of the hormones and factors of growth (determined by the methods of immunohistochemistry; molecular genetic changes (revealed by the genetic typing).

Humoral tumor markers — macromolecules which can be revealed by radioimmune or immune enzyme methods in the biological fluids of organism of patients (more often - in the serum) in much larger concentrations, than in healthy people.

Immunological methods can be used as additional method for prognosis and monitoring of efficiency of immune therapy. They determine the amount of T and B-lymphocytes, their subpopulation, concentration of globulins and heterospecific gamma-globulins.

Evaluation of hormonal type is extraordinarily important for the choice of medical tactic in patients with mastopathy. Hormonal type in the I phase (7-9 day) and in the II phase (20-22 day) of menstrual cycle can be assessed by colpocytologic (at 1 stage), radio immune and immune enzyme methods. Results are evaluated taking into account the age-dependent and anatomic and functional features of patient.

For instrumental diagnostics of metastases of CMG the condition of regional lymphatic collectors can be determined by the methods of axillography, computer tomography, ultrasound of axilla; by visualization of veins along which the lymphatic nodes are located (axillar and thanssternal phlebography). In addition, there are methods of direct radiological or radioisotope imaging of lymphatic ducts. There are radiological methods of the direct contrast lymphography of upper extremity (a contrast is entered into lymphatic vessel) and indirect contrast mammolymphography with introduction of contrast subcutaneously. An increase and change of form of lymphatic nodes, defects of filling in them, deviation of lymphatic vessels, presence of collateral ways, partial blockade of outflow, can be revealed at these examinations. At presence of 2-3 signs, accuracy of method reaches 80-90%. Au^{198} or Ts^{99} are used, which are entered directly near tumor or in the interdigital intervals of hand. With the purpose of cytological verification the biopsy of lymphatic nodes is carried out.

X-ray method (bones, lungs), computer tomography, MRI (mediastinum, cerebrum, extraperitoneal lymphatic nodes and others), radionuclide scan of skeleton and liver, US of abdominal cavity and small pelvis, complex of biochemical tests are used in diagnostics of distant metastases.

Differential diagnostics. In pathological processes, associated with tumors or nodes in tissue of gland and / or pathological excretions from nipples, it is necessary to differentiate with cancer. A primary purpose is a division of malignant and benign processes.

Mastopathy and benign tumors of MG which has foregoing symptoms also belong to this pathology. Basis of differential diagnostics in both mentioned cases is cytologic (or histological) verification.

Some specific diseases of mammary gland can give an analogical clinical presentation.

Tuberculosis of mammary gland is more often secondary. There are the following forms of tuberculosis of mammary gland: nodal, ulcerous, sclerotic and fistular.

For tuberculosis of mammary gland the indrawing of nipple, presence of dense infiltrate without clear contours and enlarged lymphatic nodes are characteristic. Diagnosis is made on the basis of anamnesis (tuberculosis of lungs, lymphatic nodes and other) and histological exam of puncture biopsy or distant sector of mammary gland.

Syphilis of mammary glands occurs rarely. At primary lesion in area of nipple, areolas a chancre appears (the limited ulcer with infiltration of bottom). The lymphatic nodes of axillas are enlarged, but not dense. At a secondary syphilis there are papules and rash on skin. Tertiary syphilis flows as single syphilitic gumma. At first a dense node appears in the mammary gland, than it increases, infiltrates skin, an ulcer, reminding disintegrating cancer tumor or tuberculosis, appears.

A diagnosis is specified by the reaction of Wassermann, cytological exam of smears from the margins of ulcer.

Actinomycosis of mammary gland also occurs rarely; it can be primary and secondary. Diseases begun with appearance of invasion of actinomycetes of small granulomas (nodes) and abscesses. Further nodes change into dense infiltrate, which soften in some places. Long not healings fistulas appear after drainage of abscesses. It is diagnosed on the basis of anamnesis (actinomycosis of other organs) and exposure of accumulations of actinomycetes in excretions from fistulas.

***Formulation of clinical diagnosis*:**

1) basic diagnosis,

2) complication,

3) concomitant pathology.

Example of formulation of clinical diagnosis:

1). Diffuse fibrotic-cystic mastopathy with pain syndrome.

2). Cancer of the left mammary gland T3N1M0, the stage IIIA, mastitis-like form.

Principles of treatment of mastopathy

Treatment of patients with mastopathy must be individualized, complex, long, etiopathogenic, to take into account the concomitant hormonal and metabolic features of patient.

The choice of medical tactic at mastopathy and benign tumors consists of the following: the diffuse forms of mastopathy are treated conservatively, and nodal forms, cysts and of benign tumors, are indications for operative treatment.

Principles of conservative treatment of mastopathy

***Principles of non-medicinal treatment of mastopathy*:**

1) Normalization of sexual and reproductive function. All women of proper age are to recommended to normalize sexual life; at birth of child to breast-feed no less than year.

2) The correction of diet must be directed on normalization of function of liver, intestine, removal of metabolic and hormonal disbalance:

- products, containing vegetable cellulose (greenery, bread of rough grade);
- fruit and green-stuffs, rich in vitamins, beta-carotene and potassium, (citrus, wild rose, carrot, cabbage, raisin);
- fish, sea products, containing the unsaturated fat acids, phospholipides, iodine.

It is possible to recommend the use of vitamin-mineral food additions.

The rational regimen of physical activity assists to normalization of metabolism, overcoming stress situations, improvement of the psycho-emotional condition.

Limitation of influence of detrimental physical factors. Patients must avoid a long stay in the sunshine, to limit the visit of solarium et cetera.

So, carrying of brassiere of inproper form or size can become reason of chronic compression of breast, especially in women with a large and lowering breast. Quite often at the removal of these reasons of pain in mammary gland diminishes or even passes fully.

Medicinal treatment of mastopathy

***Non-specific therapy of general action*.**

It is expedient to appoint vitamins in connection with their ability to normalize metabolism and hormonal homoeostasis of organism, antioxidant action. For treatment of mastopathy most often apply vitamins A, C and E and vitamins of group B (especially — B6).

Non-steroid anti inflammatory facilities can be with the purpose of braking of synthesis of prostaglandins, that has under itself a physiopathology ground.

> ***Hepatoprotectors*** *— normalizations of metabolic processes and structural and functional integrity of cellular membranes of hepatocytes. As a result, there is activation of exchange of sex steroid hormones.*

Psychotropice preparations (Corvalolum, Valocordinum, novo-passit, mixture of Qater) are used in the dyshormonal dysplasia at presence of neurogenic factors in etiology. At presence of stress, depression, emotional tension, preparations from the group of tranquilizers are appointed(trioxasin, radedorm).

Fytotherapy is most effective in young women at an adenosis, diffuse cystic mastopathy. It is expedient to use collections of herbares, possessing anti-inflammatory, sedative, immune stimulating, hepatoprotector and spasmolytic effect. Apply a valerianic root, wild rose, corn snouts, train, immortelle. At pains in mammary glands

application of diuretics — birch buds, leaves of cowberry, which are added to the indicated collection of herbares, is expedient in the second phase of cycle.

Combined fytotherapy facilities: mastodinon, klamin, climadinon.

Antireticular cytotoxic serum is used for a mastopathy due to the ability to restore immunological reactivity, regulate correlation of ephithelial and connective tissue at presence of antiproliferative activity, to stimulate the processes of cell metabolism, regeneration.

Treatment of dimexide is effective at the expressed pain syndrome, especially in patients with diffuse fibroadenomatosis or DFKM.

Hormonotherapy

Gestogens repress the gonadotropic function of hypophysis, have antiestrogenic effect, diminish influence of estrogens on tissue of mammary gland.

In a clinic progesterone is used — hormone of yellow body and its analogues and derivates (pregnin and other).

Combination of estrogens and progestins. Estrogen-gestogens synthetic preparations (oral contraceptives) repress a steroidogenesis in ovaries, modulate anovulatory cycles with the low level of gonadotropins, that diminish peak levels of the release of ovarial hormones.

Antiestrogenic preparations (Tamoxifenum) — block estrogenic receptors in tissues-targets, including – hypophysis and mammary gland, not allowing estrogens to contact with cellular receptors and at the same time reducing their biological activity. Suppress growth and cause regression of estrogen-dependent tumors.

Androgens (methyltestosteron) are antagonists of estrogens, show the antiproliferative action on organs-targets.

Blockers of hypophysis hormones (nemestran, danasol) are preparations of different origin, which have suppressing influence on synthesis and / or secretion of hormones of adenohypophysis.

***Operative interventions*,** as already mentioned before, are performed mainly in the cases of nodal forms and cysts (at mastopathy), and benign tumors. Operations are performed also at development of complications of the mentioned diseases (suppuration) or at suspicion on malignancy.

The volume of operative interventions and methods of operations at mastopathy and benign breast tumors depends on the concrete type of pathology.

Nodal mastopathy, adenoma of mammary gland, intraductal papilloma

Sectoral resection with urgent histological exam of preparation is peerformed. Indications to operative intervention are determined not so much by principles of therapeutic tactic, how many by the risk of possible diagnostic error. So for complete treatment it is necessary to conduct all of complex of therapeutic measures, as at the proper form of diffuse mastopathy.

***Fibroadenoma*.** The presence of fibrotic capsule and expansive character of growth of tumor allow to conduct operative intervention in the volume of enucleation of fibroadenoma with subsequent obligatory histological exam of specimen.

Phylloid tumor. Sectoral resection or quadranectomy of mammary gland are usually performed. After an economy sectoral resection the recurrences of tumor are not uncommon, that compels to perform the repeated operations. On this basis, at considerable sizes amputation of mammary gland is recommended.

Lactocele. Minimally invasive treatment is an removal of content of cyst or with simultaneous introduction to the cavity of air or sclerosants (96% ethyl spirit, synthetic glue compositions).

An indication for the manipulation is presence of single cavity cyst without the signs of proliferation of intracystic epithelium from data of instrumental and morphological exams.

Contra-indications to application of method are signs of proliferation and atypia of epithelium of cysts, multi-cavity cyst, cystic disease of mammary glands, treatment in occasion of CMG in anamnesis, before exposed at excisional or aspiration biopsy atypical proliferation of epithelium.

Excisional biopsy in the volume of sectoral resection with urgent histological exam is indicated in the followings cases: presence of proliferation or atypical proliferation of epithelium without the obvious signs of malignancy; exposure in the cavity of cyst of intraductal papilloma with proliferation of epithelium; cysts with a tendency to the increase and accumulation of fluid after three punctures; exposure of cyst in woman, which was treated before for CMG, or at which before atypical proliferation of epithelium was found at biopsy.

At the histological proven benign character of process excisional biopsy is simultaneously and treatment measure.

Technique of diagnostic and medical manipulations

Technique of ductography:

1. Mammary gland is massaged for the removal of secret.
2. In the external opening of duct, where discharge appeared from, through the special non-sharp needle 0,5 ml of water-soluble contrast is entered (urotrast, verographin)
3. Make images in two projections with the external compression of organ for the best distributing of contrast.

Technique of FNAB: Anesthesia is not used. To clean the place of puncture by antiseptic. To fix node by two fingers. To enter a needle into node, paying attention to its consistency (hard, soft, elastic, dense), presence of characteristic «crunch» at the calcification of tumor. To perform puncture, moving forward needle in various directions through tissue of node for the receipt of material from different areas. Before removal of needle from node, to release the piston of syringe — it went back into neutral position. To remove syringe with needle and close the place of puncture by dressing. To take off needle from syringe. To fill syringe with air. Again to put needle on a syringe, to squeeze out obtained materialon object-plate. To do smear, distributing material on object-plate, pressing on the second object-plate, and to send preparation to cytologic laboratory.

Technique of aspiration of lactocele:

Anesthesia: local infiltration is usually performed by local anesthesia.

- under control of US cyst is punctured by needle (the diameter of needle depends on character of content);
- conduct the removal of cyst contents;
- air is entered into cyst cavity in volume, equal to the volume of aspirate or sclerosing fluid - 96% ethyl spirit (1/5 from the volume of aspirate) or glue synthetic composition.

After puncture it is necessary to perform US control exam of efficiency of manipulation, dynamics of obliteration and scarring of cyst walls.

Description of formsof CMG

The initial form of cancer of mammary gland (mentioned in some classifications) – it rather the stage disease, during which no clinical signs are present. Diagnostics is based exceptionallyon special methods of exam — mammography, ultrasound and radionuclide scan-outs, morphological examination.

Nodal form occurs the most often. It is presented as dense uneven node in mammary gland, mainly in an upper-external quadrant. In medial and lower quadrants tumor is observed rarer. Tumor for a long time is presented as painless node with clear contours, which is displaced aside. Characteristic indrawings or picture of «lemon crust» can appear.

Edematic infiltrative (diffuse) form is presented by the increase of mammary gland, tumor infiltration of skin with development of «lemon crust». Palpable infiltrate does not have clear margins. The flow of this form of cancer is less favorable, than nodal, involvement of regional lymphatic nodes is observed often and develops quickly.

Mastitis-like form has signs of edematic infiltrative form with the symptoms of inflammation — hyperemia, increase of local temperature, rapid growth of tumor and infiltration of both skin and near-by tissues. It is very unfavorable form of cancer of mammary gland. Regional and distant metastases develops early.

Erysipelas form is a variant of inflammatory infiltrative cancer. The signs of itare: intracutaneous distribution of cells of tumor into the lymphatic vessels of skin. Clinically it is presented by hyperemia, as at erysipelas inflammation.

Corset cancer — actually the late stage of local distribution of infiltrative cancer of mammary gland. Infiltration of mammary gland, skin and deeper tissuesprevails. Gland shrivels gradually, and infiltrate spreads on thoracic wall, as though enchaining a patient in corset.

Disease of Pedzhet — special, rare formof cancer of mammary gland. Tumor develops from the epithelium of large ducts of gland and spreads on nippl and deeper into gland. The first symptoms of illness look like the «eczema of nipple»: nipple is thickened, the surface of it is covered by crust. Erosions appear gradually, and by the time and ulcers. An areola and skin of gland is engaged into process. It is necessary to mark that as the whole process flows slowly and its local symptoms prevail above metastasing.

The initially-multiple cancer of mammary gland is conditioned by simultaneous development of two or more tumors in one patient. At it few variants are possible: presence of two (or more) tumors in one gland; simultaneous lesion of right and left glands; presence of tumor in one gland and other organs, lesion of both mammary glands with the simultaneous presence of tumors in other organs and tissues.

Treatment of mammary glandcancer

Choice of medical tactic.

Now cancer of mammary gland is a disease at which only operative treatment is able to provide the best results. Therefore in patient with CMG operative treatment

is absolutely indicated. The additional methods of treatment are chemo-, irradiation and endocrinotherapy. These methods of influence on tumor are not radical and are used in a complex with operative treatment on indications. Independently afore-mentioned methods can be used only in those cases, when a patient can not undergo operative treatment (severe condition, inoperable stage of cancer or refuse of patient from an operation).

The pathogenic grounded effective conservative therapy of patients with CMG in our time do not exist

Surgical treatment. In our time surgical intervention is basis of medical tactic at CMG, and capable on the early stages at favorable terms fully to cure a patient.

Radical operations

Radical mastectomy on Holsted-Mayer. This intervention for a long time was classic operation in CMG. It includes the en bloc removal of all gland with major and minor mammary muscles, axillar, subclavian and subscapular fatty tissue together with lymphatic nodes within the limits of anatomic areas. The disadvantage of this operation is considered deformation of thorax, dysfunction of upper extremity, often development of lymphostasis. Now an operation is applied only on occasion — at the close contact of tumor with fascia or at moderate penetration into major mammary muscle.

More radical variant of operation is the ***extended radical mastectomy on Urban-Kholdin***, providing for in addition tothe Holsted operation resection of thoracic wall with the margin of breastbone, cartilages of II-IV ribs, ligation of a.thoracica interna and removal of parasternal lymphatic nodes. It is not used also, because it does not improve survival of patients and is extremely traumatic with high rate of postoperative complications.

Modified (limited) radical mastectomy on Peyti-Dayson. Unlike the Holsted operation, at this intervention major mammary muscle is preserved, that diminishes blood loss, creates more favorable conditions for the function of upper extremity, protects vascular-nervous bundle by muscular tissue. Indication is tumor of T_{1-2} $N_{0-1}M_0$ stages. There is less percent of postoperative complications, more rapid healing of operating wound, the best functional and cosmetic results in comparison with classic mastectomy. Unlike the last, it enables subsequent reconstruction of gland.

Mastectomy on Madden (amputation of mammary gland with regional lymphodissection) is an operation in which mammary gland, axillar, subscapular and subclavian lymphocollectors are removed with the preservation of mammary muscles. Used as an alternative of operation of Peyti.

Since 70th implementation of organpreserving operations at the cancer of mammary gland of small sizes began. Such operations are the element of the combined treatment and necessarily are accompanied with radiotherapy.

Organpreserving operations at the breast cancer

Quadrantectomy with lymphodissection. Quadrantectomy is an operation of wide excision of tumor with surrounding tissues with the removal of axillar lymphatic nodes of I, II and III levels. An operation is performed at localization of tumor of the stages of $T_{1-2}N_0M_0$ in an upper-external quadrant. An analogical operation with

the preservation of mammary muscles is namedradical resection of mammary gland and is performed at the same indications.

Lumpectomy (lump - is a node, tumor) or tumorectomy consists of removal of tumor node or marked tumor which is not palpated on 2-3 cm from its edge. Axillar lymphodissection is performed in all patients with the subsequent controlled distance radiotherapy or intratissue radiotherapy with the use of radioimplant. Indexes of non-recurrence survival are worse in comparison to quadrantectomy.

As palliative intervention at disintegrating tumors with an ulcer and bleeding amputation of mammary gland (with or without removal of fascia of m. pectoralis major) is used. Other name of operation is simple mastectomy.

Auxiliary therapy

Radioherapy. Radiotherapy (RT) is usually used as an element of the combined or complex treatment. Rentgentherapy practically lost the value as the method of radial influence, it was changed by the controlled distance gamma therapy.

Postoperative (adjuvant) RT is indicated in patients after the organpreserving operations; after radical mastectomy at the sizes of primary tumor more than 5 cm, presence of metastases in four and more regional lymphatic nodes removed during an operation; at non-radical character of intervention.

Terms of postoperative RT. It is considered most expedient to begin radial therapy not later 6-8 weeks after operative intervention.

As an independent method of treatment RT (or chemoradiotherapy) is used in the case of presence of contra-indications for operative treatment, at inoperable and diffuse forms, refuse of patient from treatment. Independent RT does not give proof convalescence, progress comes in relatively short time.

Chemotherapy. CMG is tumor, sensible to plenty of modern antiblastom facilities which behave to the different groups of cytostatics.

Most active groups of preparations at CMG:

1. Alkylating agents (thyophosphamide, cyclophosphan, melphalan, sarcolysin);
2. Antracyclins (adriamycin, farmorubicin, novantron);
3. Antimetabolits (5-fluoruracil, methotrexatum, xeloda):
4. Vinkaalkaloids (vincristin, vinblastin, navelbin);
5. Taksans (paklitaxel, docetaxel).

In treatment of CMG usually polychemotherapy (PCT) - different combinations of the most active preparations - is used.

Polychemotherapy has an aim to prevent recurrence and metastases, increase survival of patient. PCT can be conducted on the following types:

– adjuvant (postoperative) polychemotherapy (APCT).

– neoadjuvant (preoperative) polychemotherapy (NAPCT).

PCT as an element of chemoradiotherapy in locally spread and disseminated CMG does not result in recovery of disease. However much diminishing of tumor mass in an organism gives weakening of clinical symptoms which improves quality of life of patients.

Hormonal treatment.

As CMG is hormondepending tumor, a hormonotherapy (HT) is one of the most important methods of treatment. A substantial role for planning of HT and prog-

nosis of its efficiency is played by determination of factors, testifying the degree of hormone sensitiveness of tumor and hormonal status of organism:

1) ablation HT: a purpose is liquidation of organs-producers of estrogens: surgical or radial castration.

2) medicinal HT is used on the different stages of the combined and complex methods of treatment of resectable forms of CMG, and also in the cases of generalization of tumor process. The following groups of preparations are used.

Antiestrogens like the hormonotherapy of mastopathy.

LHRH-agonists are preparations-agonists of gonadotrope releasing hormone (GRH) of hypothalamus (gozerelin, buserelin, leypromid).

Inhibitors of aromatase (aminoglutetimid, fadrosol, letrosol, anastrosol, voroso) are the second line of hormonotherapy in women in postmenopause. On the mechanism of feed-back they reduce the production of androgens by adrenals - an effect is determined as a «pharmacological adrenalectomy».

Progestins (megestrol acetate, medroxiprogesteron-acetas) are preparations of the II line of HT in women with hormone-positive tumors in postmenopause.

Estrogens and androgens have a greater amount of side effects, in this connection are used rarely, can be used as IV-V line of endocrinotherapy. Indication is treatment of patients of reproductive period after ovarioectomy (androgens) or in deep menopause (estrogens) at depletion of possibilities of chemohormontherapy.

Complication of treatment and rehabilitation

Postoperative complications. The following complications take place besides general surgical (bleeding, suppuration, Infiltrates et cetera):

Subcutaneous seromas, conditioned by lymphorrhea because of removal of lymphatic collectors, bad fixing of skin fragments and inadequate draining of wound. The removalof drawbacks of surgical technique and puncture of fluid result in liquidation of this complication.

The thrombophlebitis of upper extremity usually develops on background of lymphorrhea and joining of inflammation. Treatment is traditional.

The early edema of extremity (in the first month after an operation) are usually conditioned by thrombophlebitis and develops after its liquidation. Reason is also a lymphostasis because of insufficient collateral blood flow, disturbance of transcapillar exchange, delay of Na and water in a extracellular sector, decline of oncotic pressure of blood due to the loss of albumin with wound discharge. Treatment: compression of extremity by elastic bandages or special rubber gloves, diuretics, vasoprotectors, correction of hypoproteinemia.

The late edema of extremity is conditioned by lymphostasis which causes sclerosis and fibrosis of cellular tissue. Recurrent erysipelas, thrombophlebitis, changes in the walls of vessels and nervous elements (plexus, sympathic neuroganglions), including related to the irradiation, is instrumental in the origin of late edema. Conservative treatment is ineffective; microsurgical method is used - imposition of lympho-vein anastomosises.

Dysfunction of extremity in an early postoperative period is conditioned mainly by pain syndrome, removal of muscles and tension of skin. In patients, which actively use medical physical exercises on the specially developed program, in 4-6 months

function is restored in full. At the incorrectly planned rehabilitation program or non-following of recommendations contraction of humeral joint, arthrosis and pain syndrome is formed. Medical physical exercises must begin within 1-2 days of post-operative period, in future can combine with a massage.

Physical therapy procedures are contra-indicated in the patient with CMG!

Prophylaxis of cancer of mammary gland

Primary prophylaxis of CMG (genetic, immunobiological and other) to date, unfortunately, presents theoretical interest only.

Large attention is taken to prophylactic measures which are the components of anticarcinogenic program and allow to diagnose CMG on that stage, when it is local, curable disease.

Basis of early diagnostics of nodal pathology of mammary gland to date is considered a self-examination, physical examination (examination by mammologist) and mammography. The last method is the basic type of screening exam of mammary gland.

Last modified recommendations, based on the study of influence of different methods of inspection on the timeliness of diagnostics and the results of treatment of pathology of mammary gland, above all things - CMG, are presented below:

Recommendations in relation to the mode and types of prophylactic

Inspection of mammary gland:

- from 20 years - monthly self-examination of mammary glands (SEMG);
- 20-40 years – SEMG + physical examination of mammary glands (PEMG) 1 time per 3 years;
- 40-49 years – SEMG + PEMG, mammography on individual indications (at the high risk of neoplasia);
- 50 years and more senior - mammography annually.

The program of prophylaxis and early diagnostics consists of two stages:

I stage: Monthly self-examination of mammary glands in all women after the special teaching.

At the same time, the last large studies showed that self-examination cannot be used as screening method of CMG due to its low diagnostic value. In the women, practicing self-examination, must be given those recommendations in relation to regular mammography examination.

II stage: the deep special inspection of the selected groups of patients - examination of oncologist, instrumental inspections, if necessary - morphological verification.

One of the most important directions of primary prophylaxis of CMG is an active exposure, supervision, long and effective pathogenic treatment of patients with dyshormonal dysplasia of mammary glands, above all things - proliferative forms.

IV.4 Diabetic foot

Diabetes mellitus *(DM) – one of the most widespread chronic diseases, which acquires the character of uninfectious epidemic gradually. According to information of WHO, more than 150 million of patients with DM are in the world. The expert foresee that prevalence of DM to 2010 will be more than 230 million patients.*

One of complications of DM is a syndrome of diabetic foot or diabetic foot.

Syndrome of diabetic foot (SDF) or diabetic foot is an aggregate of pathological changes on foot and lower extremities, which are characterized by development of trophic ulcers, osteoarticular lesions and purulent-necrotizing processes in patients with DM on background of neuropathy and angiopathy.

According to WHO data, amputations of l/e in patients with DM are made in 17-45 times more frequent, than in patients without violation of carbohydrate exchange, and are one of the most severe complications which determine the high risk of mortality.

Analyzing foregoing information it is possible to draw a conclusion, that actuality of this theme consists of unsatisfactory treatment of patients with SDF and with the necessity of development of measures for the improvement of diagnostics, prevention and decrease of rate of amputations in these patients.

Topografic and anatomic features of foot of man

The foot of man consists of bones, muscles, arterial, venous and lymphatic vessels, nerves and tendon-ligament apparatus.

Bones of foot consist of pre-metatarsus, metatarsus and bones of toes.

Bones of pre-metatarsus (tarsus) consist of seven short spongy bones which are located in two rows.

Bones of metatarsus (ossa metatarsalia) are short (monoepiphyseal tubular bones and remind bones on hand. Bone of metatarsus consists of basis (proximal end), body (middle part or corpus) and head (distal end).

Biomechanics of foot

A foot is difficult organ which provides balance and motion of body. It serves as not only the foundation of rest of body, influences every motion and has two primary functions:

1). Mobile adapter.

2). Rigid lever.

3). Classification of syndrome of diabetic foot.

Classification of lesions of feet in DM must answer the followings requirements:

- To reflect etiopathogenic lesion of lesion;
- To have prognostic significance;
- To determine tactic of treatment;
- To enable comparison of epidemiology trials on rate of different forms of lesions, and also to evaluate comparative efficiency of different methods of treatment;
- To be simple and accessible for the wide use;
- To have clinical significance. It supposes evaluation of prognostic of risk of amputation, efficiency of conservative or surgical treatment, and also possibility of effective prophylaxis of development of SDF.

The most valuable is the etiopathogenic classification (Dedov I. I., 2005)

There are following clinical forms:

I. Neuropathic (infected) (45% to 60%)

– with osteoarthropathy,

– without osteoarthropathyII. Neuro-ischemic (mixed) (30-45%);

III. Ischemic (5-10 %).

This classification reflects etipathogenesis of lesion, determines tactics. Ischemic variant of lesion has a number of clinical and diagnostic features. Clinical features are: proximal type of obliterative or stenosing lesion of arteries of l/e, presence of complaints about claudication intermittens, presence of all of types of sensitivity of the affected extremity, absence of mediocalcinosis of arteries of shin and foot. While in neuro-ischemic form the lesion of arteries has distal, diffuse character, only 15% of patients have complaints on claudication intermittens, mediocalcinosis of arteries of shin and foot limits possibilities of Doppler ultrasound for assessment of the condition of blood circulation. Etiopathogenic classification does not take into account severirt of lesion, that from the prognostic point of view is the substantial drawback. So the combined classification is used, where except the form of lesion it is necessary to specify the degree of severity of wond process.

Combined classification on the depth of lesion and condition of blood stream (Dedov I. I., 2005):

0 degree – there is not skin defect (there are not ulcers), however there are signs of high risk of development of SDF (areas of hyperceratosis, cracks, dryness of skin in area of feet, expressed deformation of foot, decline of sensitivity, absence of artery pulsation on foot).

I degree A — superficial ulcer, on background of normal blood stream. Patients with superficial ulcers without the signs of infection, part of patients has ulcers in anamnesis. Treatment of this category of patients consists of local treatment of wound defect and rest of the affected area of foot. Prognosis is favorable.

I degree B — superficial ulcer with the signs of decrease of blood flow. This category of patients requires more detailed inspection to evaluate the condition of blood circulation. Prognosis depends on the degree of decrease of blood flow.

II degree A — ulcer involving soft tissues, without the signs of ischemia. Antibacterial therapy, local treatment and extremity rest are indicated. Prognosis favorable.

II degree B – as II degree A, but with the signs of ischemia of extremity. Prognosis is unfavorable.

III degree A – ulcer involving tendons and bone tissues, with the signs of deep infection. Patient requires immediate hospitalization into surgical department, conservative and surgical treatment. Prognosis is rather unfavorable.

III degree B — as the III degree A, but with the signs of ischemia. Prognosis bad.

IV degree — gangrene of part of foot, more often in combination with the decrease of blood flow or thrombosis of arteries of toe\foot. Amputation in this category of patients is inevitable.

V degree — gangrene of all of foot.

Classification of chronic obliterative diseases of arteries of l/e on Fontain-Pokrovskiy:

I degree — the stage of clinically significant stenosis, revealed by non-invasive methods of diagnostics (US, full-duplex scan-out).

II degree — the stage of claudicaation intermittens:

II degree A — distance of the painless walking more than 200 meters;

II degree B — distance of the painless walking more than 200 meters.

The III degree — presence of rest pain.

The IV degree — the stage of critical ischemia.

The condition of critical ischemia is determined as a presence of rest pain, chronic character of ulcers, gangrene which are confirmed by objective information confirming the presence of obliterative lesion.

Pathogenesis of syndrome of diabetic foot

Diabetic polyneuropathy

Diabetic neuropathy is heterogenic concept, which unites number of specific for diabetes and unspecific, unassociated with violation of carbohydrate exchange of clinical syndromes.

Diabetic neuropathy has the followings stages:

Stage 0 — there are no symptoms of neuropathy;

Stage 1 — changes of the peripheral nervous system, exposed by the special quantitative neurological tests, the clinical symptoms of neuropathy are absent here;

Stage 2 — the stage of clinical symptoms, when together with the changed quantitative neurological tests, signs and symptoms of neuropathy are present;

Stage 3 – characterized by the severe dysfunction of nerves, resulting in severe complications, including the development of diabetic foot.

All types of lesions of the nervous system are subdivided into five groups:

1. Metabolic.
2. Immuno-vasculitis.
3. Compression.
4. Hypoglycemia (insulin) neuropathy.
5. Ischemic type due to obliterative diseases of arteries of l/e.

The most frequent form of lesion of the peripheral nervous system is diabetic polyneuropathy.

Diabetic polyneuropathy — lesion of the peripheral somatic and vegetative nervous system, related to DM.

Clinical classification of diabetic neuropathy:

1. Focal neuropathy.
2. Diffuse neuropathy.

From data of epidemiology trials, about 60-65% of patients with DM of 1 or 2 type has symptoms of one or another form of neuropathy. 40-45% patients has distal symmetric form of y neuropathy. About 30% has signs of various tunnel syndromes.

Diagnostics of lesions of the peripheral nervous system

Diagnostics of lesions of the peripheral nervous system is conducted on the basis of anamnestic data, examination of function of nerves by the evaluation of different types of sensitivity and electro-physiological methods of investigation.

The objective assessment of neurological symptoms is conducted according to the following scales:

– the visual analog scale;

– the scale of neurological symptoms;

– the general scale of neurological symptoms;
– the Hamburg questionnaire on a pain syndrome (HQPS).

Four most often symptoms are analyzed: shooting pain, burning, numbness, paresthesias. In digital expression the scale of symptoms varies from 0 (no symptoms) to the maximal value 14, 64 (all of symptoms, almost constantly).

Violations of neurological function are assessed according to the followings scales:

– scale of neurological violations (tabl. 1);
– scale of neurological disorders.

Table 1

Scale of neurological violations (NDS according to Yang)

Parameters	Normal	Decreased	Absent
Vibratory sensitivity			
Pain sensitivity			
Temperature ensitivity			
Reflexes			

Research is conducted on l/e with the calculation of sum of marks.

A maximal value on both extremities makes 10 marks.

Mild neuropathy – sum of marks 3-5.

Moderate neuropathy – sum of marks 6-8.

Severe neuropathy – sumof marks 9-10.

The most objective method of evaluation of the functional condition of the peripheral nervous system is an electro-myography, allowing to examine important electro-physiological features.

Influence of neuropathy on the condition of peripheral blood circulation flow.

SDF is leading cause of amputations of l/e in the patients with DM. The large number of amputations is made in patients with diabetic neuropathy, without violation of circulation of blood. Diabetic neuropathy can become cause of foot gangrene in an absolutely normal pulsation on the arteries of foot. Cause of gangrene of foot in preserved blood flow is development of septic thrombosis because of deep infection of foot.

Sensory neuropathy is characterized by considerable decrease or loss of pain, temperature, vibratory sensitivity. The exposure of signs of ischemia is based on the pain feelings of patient during physical exercise (walking) or in condition of rest. This principle is based on classification, offered by Fontain in 1954 and confirmed by TASK in 2000. However, in Fontain classification there are no exceptions, when a patient could not specify on the pain feelings.

It can be at presence of severe neuropathy or, when patient is in the unconscious condition. From 100% patients with DM and amputations only 30% had syndrome of claudication intermittens in anamnesis.

Diabetic neuropathy hidden the flow of occlusion lesions of arteries.

Thus, the careful inspection of patients is needed with DM to reveal neurological deficit and violations of peripheral blood circulation, non depending on the presence of subjective symptoms.

Diabetic osteoarthropathy

It is known that the different forms of lesions of locomotor system are revealed in DM: osteoarthropathy, kidney osteodystrophy and osteoporosis.

In osteodystrophy more often there are such lesions as:

– lesions of pre-metatarsus and metatarsus;

– lesions of talocrural joint;

– development of generalized osteoporosis.

The generalized lesion of skeleton meets rarely and presents as an osteoporosis of spine and proximal departments of thigh.

Diabetic osteoarthropathy (Sharko arthropathy) is relative painless, making progress and destructive arthropathy of one or a few joints, associated with a neurological deficit (Fig. 36).

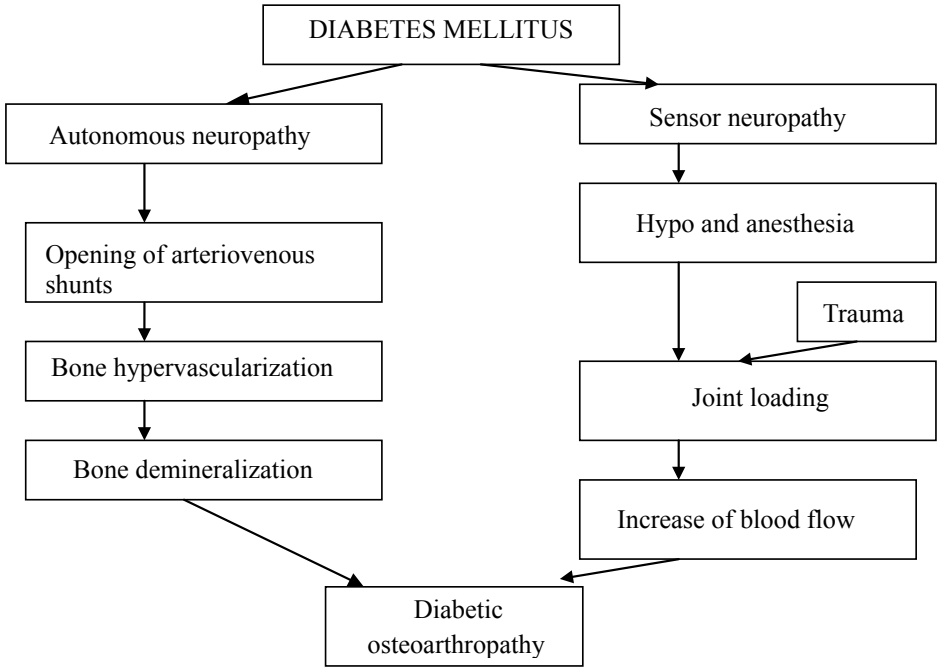


Fig 36. Pathogenesis of diabetic osteoarthropathy

Three stages of development of osteoarthropathy:

I — initial stage – characterized by acute destruction of joint with osteochondral fragmentation, tension of joint capsule, distortion of ligament apparatus and subluxation.

II — the stage of consolidation — is accompanied by resorbtion of most bone fragments and their fixation to sublaying bone.

III — stage of reconstruction – remodelling of bone. Result of it is diminishing of sclerosis, and also partial renewal of architectonics of joint.

Clinically in development of osteoarthropathy select two stages:

I – acute, and II — chronic. On the basis of this division the differential is made for the choice of therapy.

Methods of diagnostics of bone lesions in patients with the syndrome of diabetic foot.

Besides the routine methods laboratory and instrumental methods for determination of the condition of bone structures are used.

Instrumental methods of diagnostics of bone changes in osteoarthropathy

X-ray. On X-ray osteoarthropathy joint looks as severe form of degenerative or atrophy arthritis. Changes are interpreted as hypertrophic or atrophy consequences of damages of bone structures of l/e. Atrophy and osteolysis are revealed in the bones of metatarsus and pre-metatarsus. Disintegration of joint, subluxation, osteopenia, pathological fractures, edema of soft tissues, atrophy are changes, characteristic for the active phase of osteoarthropathy.

Hypertrophic changes which prevail in the chronic stage more often are in the bones of metatarsus and pre-metatarsus. These changes include thickening of cartilage, subchondral sclerosis and formation of regional osteophytes.

Late stage of Sharko arthropathy is characterized by the followings changes: proliferation of new bone (outcome of neuropathic fractures), osteoporosis, atrophy, pathological fractures and disintegration of joint.

Scintigraphy. Combined application of methods of scan-out with techentium and indium in 90% cases allows to diagnose osteomyelitis in patients with Sharko arthropathy.

MRT. During MRT, as a rule, it is needed to conduct differential diagnostics between osteoarthropathy and osteomyelitis.

In addition, MRT exactly describes the margins of infectious process, that is extremely important for determination of volume of operative intervention, allowing to avoid the repeated amputations.

A magnetically-resonance angiography is the non-invasive method of assessment of blood flow in vessels of feet, especially when proximal occlusion limits possibilities of angiography.

Key in diagnostics of osteoporosis is an exposure of the systemic diminishing of bone mass. To the methods, which are based on the fact that a bone and soft tissues take in an ionizing radiation with different intensity, belong:

– Method of one-photon absorptiometry;
– Method of quantitative computer tomography (QCT);
– Ultrasonic method;
– Double energetic X-ray absorptiometry (DEXA).

INDM of 1 type the density of bone tissue decrease on 10-12%, in DM of 2 type, decrease of bone density is more expressed — from 10 to 26% depending on age, degree of obesity and duration of disease.

Determination of biochemical markers of bone metabolism sufficiently informs both about the systemic presentation of osteopenia in patients with DM and local changes in bone tissues in patients with diabetic osteoarthropathy.

Diabetic angiopathy

The vascular system is one of the «targets» of DM. At this disease both a microvasculature (diabetic microangiopathy) and arteries of large and middle calibre are involved (diabetic macroangiopathy).

Diabetic microangiopathy – is specific for DM lesion of vessels of microvasculature. However its significance is different in the vessels of various organs. Diabetic microangiopathy is able fully to violate the function of retina of eye and kidney glomerulus (with development of diabetic retinopathy and nephropathy). In regard to the microvasculature of tissues of extremities information of studies is disputable.

The basic changes of microvasculature are studied in DM. They are: distrophic changes of endotheliocytes, increase of permeability of vascular wall for the albumens of, activatiion of pericytes and smooth muscular cells of vessels, thickening of basal membrane and hyalinosis of arterioles. Changes, characteristic for a diabetic microangiopathy, are revealed in 82,3 – 88,6 % patients.

Large attention was paid for the possible pathogenic role of the exposed morphological changes of microvasculature in development of complications of DM, in particular, destructive lesions of feet (DLF).

The results of the transcutaneous measuring of oxygen tension of in l/e in patients with peripheral atherosclerosis both with DM and without DM showed that it is determined by the degree of violations of main blood flow and does not depend on the presence of DM. It was acknowledged, that diabetic microangiopathy is not able in itself to cause necrosis of tissues and trophic ulcers of feet.

Thus, it is possible to draw a conclusion: the morphological changes of small vessels take place in majority of patients with DM 1 and 2 types which can develop early enough. However their influence on the function of vessels in skin and soft tissues of extremities remains not fully clear.

Diabetic macroangiopathy

Morphologically this complication of DM is atherosclerosis. Except of the vessels of extremities, coronal and cerebral arteries are involved as well. An atherosclerotic lesion has a number of features in the patients with DM: more distal lesion (in lower extremities more often is popliteal artery and arteries of shin), bilateral and plural localization of stenosises, development of process in more young age, the same morbidity in men and women. All of it allows to talk about the specific form of atherosclerotic lesion in DM and to define it as diabetic macroangiopathy.

Diabetic macroangiopathy results in development of critical ischemia of tissues of extremity. As a result necrosis of skin and hypodermic tissues without some additional mechanical damage — only due to acute violation of oxygen and nutritives delivery into the distal departments of extremity (above first of all, into skin). In some patients it is possible to expose in anamnesis some damaging factor, breaking the integrity of skin. Such factor can be skin damage by nails, formation of cracks due to skin dryness, mycosis.

The considerable decrease of blood flow blocks the reparation capabilities of tissues and results in further expansion of area of necrosis. The result is development of typical circulatory ischemic dry necrosis of skin as a scab, locating in the «end» areas of foot with poor vasculature.

Pathogenesis of wound process in the patients with DM

From 600 000 to 2,5 million people in the world suffer from chronic trophic ulcers of feet and shins. Among them 15% has ulcerous defects feet due to DM.

The most frequent locations of trophic lesions of l/e are:

– toes (dorsal and plantar surfaces) – 51-52%;
– area of projection of heads of metatarsus bones on planta – 28-37%;
– dorsum of foot – 11-14%;
– multiple ulcerous defects – to 7%.

As a result of conservative treatment, primary healing of ulcerous defect is succeeded in 63-81% cases, a necessity for amputation on different levels appears in 14-24% patients, mortality is about 5-13%, development of recurrences of chronic ulcers occurs in 43-50% of cases.

Phases of wound process. In the process of healing of acute wounds it is possible to distinguish several phases: coagulation, inflammation, matrix synthesis and accumulation, angiogenesis, fibroplasia, epithelisation, retraction and reconstruction (renewal). End-point of the uncomplicated cicatrization is simple scar with small fibrosis, minimum, if a wound is retracted, and return of almost normal architectonics of tissues and function of organ. If a wound does not heal in a normal order and temporal sequence, if the process of cicatrization does not result in structural renewal, such wound is considered chronic. Cicatrization of chronic wound includes those processes, what cicatrization of acute wound, namely inflammation, fibroplasias, epithelisation. At the same time, a chronic wound differs from acute that cicatrization goes along with formation of abundant granulations and often with expressed fibrosis, resulting in cicatrical deformation and loss of function of organ.

Phase of inflammation (exudation). The damage of tissues initiates cellular and vascular response, the result of which is wound cleansing from necrosis, determining the terms of cicatrization and regeneration. This inflammatory response consists of two components:

1) vascular response, showing up local vasodilatation and increase of capillary permeability;

2) leucocyte infiltration, caused by specific chemical factors, present in a wound.

Phaseof proliferation of fibroblasts. Fibroblasts appear in wound during 2-3 days and for the first week become basic cellular population. During the phase of proliferation of fibroblasts collagen is synthesized the level of which constantly increases during 3 weeks until rate of its synthesis will not become similar to the rate of degradation. The increase of concentration of collagen in a wound during the phase of proliferation correlates with the increase of force wound retraction. Fibroblasts are the basic source of collagen and connecting tissue of wound.

Ripening phase. Balance between synthesis and disintegration of collagen is achieved to the third week, and reconstruction of wound begins. This process proceeds about two years. For this period of increase of amount of collagen does not take a place, but reorganization of collagen filaments goes in more dense latticed structure, whose closeness determines local mechanical terms. Elasticity of wound in this phase quickly increases.

Epithelisation. Reepithelisation of wound is the confirmation of the proper care is sequence of such processes, as mobilization, migration, mitosis and cellular embryonisation of epithelial cells. Advancement of margins of epithelium is carried out by means of mitosis and takes place until the opposite margin of wound will not be attained.

In this point migration of cells is inhibited due to the phenomenon of contact inhibition.

Retraction of wound. Very typical for cicatrization of chronic wound is the phenomenon of its retraction. Although playing useful role in diminishing of size of wound and, finally, closing the defect, this process is disorderly and can result in changes of structural integration, loss of function and cosmetic defect. Narrowing of wound begins already during the first week after damage.

Formation of granulation tissue. Besides the phenomenon of retraction, chronic wound differs from acute on volume of granulation tissue. The last consists of great number of capillars and supporting matrix, includingfibroblasts, cells of inflammation, endothelial cells, pericites and myofibroblasts. The first stimuli for neovascularization of granulation tissue are factors of growth.

In the process of cicatrization granulation tissue rich in cells and vessels transforms into collagen acellular matrix. Probably cause of disappearance of granulation cells from a wound is apoptosis. Violation of this process leads in future in development of hypertrophic scar. It is often marked in burns. Concrete signal to the apoptosis is unknown.

Examination of patient with suspicion on SDF

Maximally early diagnostics of initial signs of lesion of the peripheral nervous system, vascular system, soft tissues and bone structures of l/e are basis of prophylaxis of amputations in persons with DM.

Diagnostic methods, used for the patients with DM for diagnostics of lesions of l/e, can be divided into two large groups: out-patient tests; in-patient tests. At questioning of patient:

1) complaints and anamnesis regarding SDF;
2) complaints and anamnesis regarding DM;
3) complaints concerning other organs and systems.

Diagnostics in out-patient departments

1) Complaints and anamnesis regarding SDF.

Diagnostics must begin with collection of complaints and anamnesis of disease. Patients with SDF, as a rule, have the followings complaints:

– Pain is in feet and l/e;
– Weakness and fatigue in l/e in physical exercise;
– Paresthesias and chill of feet;
– Claudication intermittens;
– Pallor of skin of l/e;
– Cyanosis of feet and toes of feet;
– Numbness of l/e;
– Decline of skin temperature of l/e;
– Atrophy of skin and development of trophic disorders (fragility of nails, hyperkeratinisation, absence of hair, fissures on toes and between them, « marbleness» of skin of l/e and feet;
– Presence of ulcerous defects on feet;
– Fever;

– Deformation of feet and talocrural joints.

On collection of anamnesis the special attention must be paid to the following:

– Subjective symptoms of decompensation of DM;
– Duration and character of course of basic disease;
– Presence of ulcerous defects on feet and shins in the past;
– Presence of concomitant pathology which was instrumental in development of SDF;
– Collection of family anamnesis (early development of atherosclerotic symptoms in relatives);
– Conditions of life of patient.

On the basis of complaints and anamnesis it is possible make conclusion whether this patient in the group of risk of development of SDF.

Complaints and anamnesis regarding DM

DM is an endocrine-exchange disease, development of which is caused by the absolute or relative deficit of insulin; or DM is the condition of chronic hyperglycaemia which can develop because of influence of many exogenous and genetic factors (WHO, Geneva, 1981).

Most typical complaints of DM:

– Polydipsia (thirst);
– Polyuriya;
– Loss of weight;
– Fatigue;
– Disability;
– Itching of skin and genitals;
– Anorexia; - Heart-ache;
– Worsening of sight (evelopment of cataract);
– Pain in l/e.

On collection of anamnesis in patients with suspicion on DM, or in patients with DM it must be paid attention to the following:

– In detail to inquire for the presence of diseases in relatives and cause of their death (death of relatives could be due to undiagnosed DM);
– To expose a presence in the relatives of obesity or atherosclerosis;
– To expose probable causes or contributing factors, such as, acute or chronic infections (especially viral), psychical trauma, dietary violations.

Complaints concerning other organs and systems. The complaints of those organs and systems which are most often affected in patients with DM need special attention.

Diagnostics of lesions of l/e in patients with DM in out-patient department (in policlinic).

Examination of l/e of patient

On examination of l/e of patient especially pay attention to dorsal and plantar surfaces of foot, and on the condition of interdigital spaces.

Changes which can be revealed on examination of foot in patients with SDF:

1. Discolorations:

– Red in cellulitis or early stage of Sharko arthropathy;

– Pale or cyanotic in ischemia;
– Bronze in severe ischemia (related to pain and absence of pulsation of arteries).

2. Deformations:
– Claw-shaped, hammer toes;
– Drop foot, inversions of foot;
– Salient heads of metatarsus bones;
– Sharko foot of (deformed foot, developed as a result of the numerous repeated fractures of bones of foot, occurred unnoticed for a patient because of complete anaesthesia).

3. Oedema:
– Bilateral – related to cardiac insufficiency or neuropathy;
– One-sided – can be caused by infection or Sharko arthropathy.

4. Nails:
– Atrophic in polyneuropathy and ischemia;
– With the change of colour in mycosis (white-yellow) with subnail ulceration.

5. Callosity:
– On foot with neuropathy callosity develops on the plantar surface of heads of metatarsus bones and apexes of toes.

6. Ulcers:
– neuropathic (usually plantar);
– neuro-ischemic (usually on the tips of foot).

During every examination the size of ulcer must be measured (diameter, depth).
– Chaps blisters.
– Palpation of l/e of patient

On palpation of l/e of patient with SDF conduct the followings manipulations:
– Check up the pulsation of arteries on foot:
– The pulsation of posterior tibial artery and dorsal artery of foot is weak or absent on an ischemic foot;
– The pulsation of arteries is preserved on neuropathy foot.

A. For diagnostics of chronic occlusive diseases of arteries of extremities (obliterative atherosclerosis, endarteriitis, thromboangitis, syndrome of Lerishe, disease of Takayasu, the syndrome of diabetic foot) determination of pulsation of main arteries of extremities in projection points (it enables to diagnose the approximate level of occlusion) is conducted:

1. dorsal artery of foot (a. dorsalis pedis) – on the dorsum of foot in its middle part between I and II metatarsus bones;
2. posterior tibial artery (a. tibialis posterior) — behind medial ankle-bone;
3. popliteal artery (a. poplitea) – in a popliteal space with a little bended in knee-joint;
4. common femoral artery (a. femoralis communis) – below the inguinal ligament on thigh.

The functional clinical tests in the chronic occlusive diseases of arteries of extremities allows clinically determine the level and degree of occlusion and development of collateral blood circulation. They are:

1. Oppel («symptom of plantar ischemia», Samuels, Goldflamm) — pallor of skin of foot and rapid appearance of fatigue during exercise in talocrural joint of affected extremity in position of l/e lifted up on 45-70 degrees in horizontal position of patient;
2. Lenyel-Lavastina ("symptom of white spot") – in pressing on two toes simultaneously on the dorsum of hallux of healthy and affected feet after taking away hands on affected extremity a pale spot disappears considerably longer, than on healthy leg;
3. Kozachesku – dermographism from an inguinal ligament to lower third of shin: more long phase of white dermographism in the distal departments of affected leg to the certain proximal level as compared with healthy extremity;
4. Moshkovich — an exposure of level of "reactive hyperaemia" in vertical position of patient after in horizontal position patient will hold for some the time lifted up on 45 degrees affected leg during 5-7 minutes;
5. Kollenz and Villenski — exposure of degree and rate of filling of superficial veins in transition of patient from horizontal to vertical position;
6. Panchenko "knee phenomenon" — appearance of pareshesias in position sitting with affected leg on healthy leg during 5-7 minutes;
7. Alekseyev — measuring of skin temperature of extremity before and after the long walking (to 2000 m), which reveals its decline in the affected extremity;
8. Shamova – "time of reactive hyperaemia" — determination of time of appearance of "reactive hyperaemia" after the compression of arteries of shin of the affected extremity by the cuff for measuring of arterial pressure for 5 minutes (normally – through 15-20 seconds, in pathology – considerably later, sometimes through 2 and more minutes).

B. Temperature of skin:
– neuropathy foot is warm;
– ischemic foot is cold.

C. Signs of infection :
– crepitus, fluctuation;
– softening of tissues on deep palpation.
– Humidity of skin:
– neuropathy foot is usually dry (hyperhidrosis is replaced by an anhidrosis).

After examination and palpation of l/e apply the next tests of assessment of blood circulation.

1. Skin temperature.
2. Thermoesthesia. Use two glass test tubes, filled with warm and cold water, or apparatus "Type Therm", which is a cylinder, made from two materials, having permanent difference of temperatures.
3. Condition of peripheral innervation and blood flow by monofilament and graduated tuning fork. For diagnostics of distal polyneuropathy apply monofilament of weight 10.
4. Use the graduated tuning fork for diagnostics of distal polyneuropathy and monitoring of lesions of peripheral innervation (determine an increase or decline of vibratory sensitivity during treatment).

5. In out-patient department diagnose possible ischemia of extremity by ankle-humeral index determined by portable Doppler apparatus and sphygmomanometer. The cuff of manometer is positioned on the middle third of shin. The sensor of Doppler is positioned in the point of projection of posterior tibial artery or dorsal artery of foot. Systolic arterial pressure is measured in one of the mentioned arteries. Then systolic arterial pressure is measured on humeral artery. Ankle-humeral index (AHI) is calculated as a relation of systolic pressure in the artery of lower extremity to the systolic pressure in a humeral artery.

AHI = systolic AP in the artery of l/e: systolic AP in humeral artery

Normally AHI is 1,0. Decline of this index to 0,8 and below talks about a presence of occlusive disease of arteries of l/e. Increase of AHI to 1.2 and higher confirms severe diabetic neuropathy and accompanying Monkeberg atherosclerosis. In single occlusion of artery an ischemic index can be not decreased (approximately 1,0). For the exposure of occlusion «stress-test» is used in this case.

1) determine an ischemic index at rest;

2) patient walks for 4th minutes (or until claudication intermittens appears), than ischemic index again is determined, then through 1 min, then through every minute until the index of ischemic index will not go back to initial (normally it takes a place after 1 minute).

«Stress-test» is contra-indicated in patients with IHD. In these patients «effect of hyperemia» is used: the second cuff of sphygmomanometer is positioned proximally to the first, pumped in to stop outflow of blood (for 4 minutes or till appearance of pain in leg). Normally change of metabolism results in dilation of arteries, that is similar to the action of the physical loading. Then the second cuff is taken off and ischemic index is determined. The decline of this index testifies occlusion of artery.

3). At the signs of diabetic macroangiopathy, in out-patient terms, laboratory tests are performed:

– Biochemical test of serum of blood (cholesterol, creatinine, urea);
– Prothrombin index and coagulation time.
– glycolized haemoglobin, daily proteinuria. And in absence of albumen in urine – microalbumenuria.

4). For the evaluation of the condition of bone structures of foot and exposure of signs of diabetic osteoarthropathy X-ray of feet and talocrural joints in two projections (direct and lateral) must be performed.

5). During evaluation of the condition of the nervous system it must be remembered, that there are three factors of the peripheral nervous system, which result in formation of ulcers on diabetic foot:

1. Sensory neuropathy – due to violation of pain sensitivity there are often microtraumas, they can long time remain not noticed, that results in joining of infection.
2. Motor neuropathy: disbalance of muscles, thinning of intercostal muscles, deformation of joints («clawhand»), increase of pressure on foot – these biomechanics factors result in appearance of new points of pressure on foot, in these places ulcers develop subsequently.

3. Autonomous neuropathy results in violation of blood flow in feet (to inadequate blood flow through a microvasculature), that worsens perfusion of tissues and promotes the risk of ulceration.

For diagnostics of neuropathy it is necessary to use certain tests.

It is necessary to take into account:

– Colour of skin feet;
– Condition of skin, subcutaneous fat and appendages of skin;
– Presence, location and features of ulcers.

For peripheral autonomous neuropathy is characteristic hyperemic warm foot with impaired sweating (it is excessive in the initial stages, subsequently it is decreased with dry skin, chaps), edema of shins, feet.

For an ischemic foot the pale or cyanotic colouring, dry skin, absence of hair, thickening of nails is characteristic, often with their mycotic lesion, atrophy of subcutaneous fat.

I. In out-patient terms it is necessary to make diagnostics of sensory violations.

1. To study vibratory sensitivity – by tuning fork of frequency of 128 Hertz.
2. To study proprioceptive sensitivity. By the fingers of hand of doctor, hallux of patient is moved upwards-downward, patient must correctly define direction of toe motions.
3. To study pain sensitivity – by pin beginning from distal toes. For peripheral neuropathy violation of sensitivity is characteristic on feet on type of "socks" or "stockings", however in DM also lesion of separate nerves with absence of sensitivity in the proper area of innervation (mononeuropathy).
4. To study of touch sensitivity (by the piece of cotton wool).
5. To study of discriminatory sensitivity.
6. To study temperature sensitivity.

II. Diagnostics of motor violations.

1. Assess the volume of muscles of shin, feet.

For chronic peripheral sensory-motor neuropathy little muscular atrophy, thinning of intercostal muscles, is characteristic.

2. Study resistance of muscles.
3. Expose the presence of contraction.
4. Check up the tendon reflexes (patellar, axillar, plantar).

So the complex of out-patient diagnostic measures in patients with SDF must include the following:

– collection of complaints and anamnesis;
– examination and palpation of l/e;
– determination of touch, temperature and vibratory sensitivity;
– determination of ankle-humeral index;
– determination of the condition of carbohydrate, albumin, lipid exchange, coagulant system of blood by laboratory tests;
– X-ray of feet in two projections.

The use of these simple methods of research will allow already on the ambulatory stage to expose patients, included in the risk group of development of SDF, and also persons, already having the one or another lesion of extremities, to define the clinical form and to begin treatment.

Diagnostics of lesions of l/e in patients with SDF in the hospital. They are:

– Determination of severity of lesion of foot;
– Choice of adequate method of conservative or surgical treatment;
– Monitoring of medical process.

In the list of obligatory tests of patients with the different forms of SDF, being in the conditions of the specialized clinic, the followings positions include:

– Determining of size and depth of ulcerous defect;
– Bacteriologic culture of wound exudate and tissues of ulcerous defect with determination of microbiological spectrum and susceptibility of microorganisms to the wide spectrum of modern antibiotics;
– Full-duplex scan-out of arteries with determination of degree and level of occlusive lesion (for the choice of method of vascular operation – X-ray or MR-angiography);
– Transcutaneous determination of oxygen saturability of tissues (transcutaneous oxymetry, TcPO2) and prognosis of possibilities of conservative therapy;
– X-ray, MR-tomography, scintigraphy, CT of bone structures of l/e for the exposure of signs of osteomyelitis and for the differential diagnostics of combined lesions of feet and shins;
– Clinical blood and urine tests;
– Biochemistry of blood (lipid spectrum, general protein, albumin, creatinine, potassium, alkaline phospatase, ionized calcium, bone isoenzyme of alkaline phospatase, tartratresistant acid phospatase) for determination of expressed of atherosclerotic process, diabetic nephtopathy, bone resorbtion and osteosynthesis;
– Reologic properties of blood (coagulogram);
– Condition of fundus of eye.

For determination of degree of severity of SDF it is necessary to evaluate an ulcerous defect and condition of surrounding tissues. For this purpose it is necessary to measure area and depth of trophic ulcer, to conduct a bacteriologic culture of wound exudate and soft tissues. Material for culture must be taken not only from a surface but also from the depth of the affected tissues, because a microbiological spectrum can be different, at the ischemic or neuro-ischemic (mixed) form of SDF in hospital it is necessary to conduct more exact diagnostics of occlusive lesion of peripheral arteries for the timely vascular operation, and also to define the degree of ischemia of soft tissues of extremities.

Method of full-duplex scan-out is enough widely spread for diagnostics of vascular diseases of different location in patients with DM. Lesions of arteries of middle calibre are more frequent in DM (popliteal, posterior and anterior tibial, dorsal artery of foot). The long extent of occlusion, multi segmental lesions are typical is characteristic. During evaluation of the condition of vascular wall (complex of intima-media) pay attention on its thickening, cleavage, sometimes absence of embryonization on layers. In case if occlusion is more than 70%, local and tense, the vascular intervention is possible for renewal of blood flow in the affected extremity.

The method of transcutaneous determination of oxygen saturability of tissues (TcPO2) got wide distribution in practice of the specialized departments of both surgical and therapeutic type due to the high informing, non-invasiveness and relatively

small cost (as compared with a full-duplex scan-out and angiography). Method allows to assess the degree of ischemia of soft tissues. A threshold value of TcPO2 is 30 mm Hg. Oxygenation of tissues below this value confirms the severe ischemia and prognoses unfavourable outcome of conservative treatment of such extremity. Transcutaneous oxymetry must be conducted in the patients with the neuropathic form of SDF, at which development of local ischemia of soft tissues is possible in area of ulcerous defect due to an inflammatory edema and development of thrombosis of capillaries.

The presence of lesions of bone structures in patients with the different clinical forms of SDF determines actuality of inspection, directed on verification of origin of bone pathology (osteoarthropathy, osteomyelitis, et cetera) and determination of tactic of its treatment. For this purpose, besides traditional X-ray, MR-tomography, computed tomography, scintigraphy are possible.

For the differential diagnostics of neurological disorders apply electromyographic methods. They allow to expose the level of lesion of neuromotor apparatus, character of lesion, define the level of compression of nerve in tunnel syndroms, and also the conditions of neuro-muscular transmission.

Severe deformation of feet in patients with diabetic osteoarthropathy results in formation of atypical points of pressure on a plantar surface. A large value has a timely exposure of these points at development of measures of prophylaxis of recurrences of ulcerous defects. Substantial help herein can have computed pedobarography. This method can be instrumental in the correct choice of orthopaedic devices and evaluation of efficiency of their application in patients, including the group of risk of development of SDF.

Methods of laboratory diagnostics, which must be applied in patients with SDF.

- A clinical blood test must be performed in all of patients with the ulcerous lesion of l/e not rarer than 1 time per 7-10 days, but at the severe inflammatory process – one time per 3-5 days.
- In clinical urine analysis it is necessary to pay attention to signs of possible diabetic nephropathy (proteinuria, erythrocyteuria, cylinderuria). The expressed nephropathy and the results of bacteriologic culture of wound exsudate, will determine the choice of antibiotics and its dosage.
- Biochemical parameters of serum of blood (lipid spectrum), signs of diabetic nephropathy (creatinine, urea, potassium, general protein, albumin), ionized calcium, bone isoenzyme of alkaline phospatase, tartratresistant acid phospatase for determination of bone resorbtion and osteosynthesis.
- Determination of reology of blood (coagulogram) is special important in patients with the ischemic form of lesion. The results of this test play an important role in the choice of medicine of correction of ischemia and determination of duration of its application.

Differential diagnostics of different clinical forms of SDF

Differential diagnostics of basic clinical forms of SDF is based on information of the followings methods of examination:

- careful collection of complaints and anamnestic information;
- examination of l/e;

– evaluation of the condition of peripheral innervation;
– evaluation of the condition of peripheral blood flow;
– determination of the condition of bone structures of feet and talocrural joints;
– diagnostics of possible local infectious process and its bacteriological analysis (character of local infectious process depends on the degree of ischemia of tissues. For the neuropathy form of lesion of extremity, the preserved arterial blood flow, staphylococcus aureus or epidermal are characteristic. An infectious process has local character more often. In the occlusive diseases of peripheral arteries not only aerobic but also anaerobic microorganisms almost always are found in the microflora of wound, that does an infectious process more resistant to the conducted therapy).

Differential diagnostics of basic (neuropathic and ischemic) clinical forms of SDF is presented in a table № 2.

Table 2

Differential diagnostics of basic forms of SDF

Stages of diagnosis	Neuropathic form	Ischemic form
Complaints, information anamnesis	Night pains in the feet of aching character, paresthesias, sensitivity of feet to the cold, cramps in the muscles of shins, painless ulcerous defects on plantar surface. Large duration of diabetes and presence of other late complications.	Pains in feet and shins at walking, night pains, decreased at lowering of feet, very painful tip necrosises. Duration of DM can be little, a presence in anamnesis of diseases of the cardiovascular system (high arterial pressure, heart attack, stroke) and dyslipidemia.
Examination	Skin is dry, rose, warm. Deformations of toes, feet, talocrural joints, bilateral edema. Hypercer-atinizations are in points of maximal loading pressure|. Thickening and deformation of nails. Ulcerous defects on plantar surface in the areas of excessive pressure.	Atrophic skin, cold, cyanosis, petechial hemorrhage, often cracks of interdigital intervals. Deformations are not typical. Atrophic nails. Tip necrosises on heels and toe-tips.
Condition of peripheral innervation	Decrease of temperature, pain, touch, vibratory sensitivity	Preserved peripheral sensitivity.
Condition of peripheral blood flow	Pulsation of peripheral arteries is preserved, ankle-humeral index > 1,0, oxygenation of soft tissues > 30 mm of Hg	The pulsation of peripheral arteries is acutely decreased or not determined, ankle-humeral index < 0,8, oxygenation of soft tissues < 30 mm of Hg
Condition of bone structures and talocrural joints	X-ray picture of diabetic osteoarthropathy (osteoporosis, osteolysis, paraossal calcifications, spontaneous fractures, dislocations and subluxations of joints)	Not typical changes of X-ray picture (artrosis and arthritises, in severe cases signs of osteomyelitis)
Bacterial culture of wound discharge	Aerobic flora	Combination of aerobic and anaerobic microorganisms

Risk groups of development of SDF

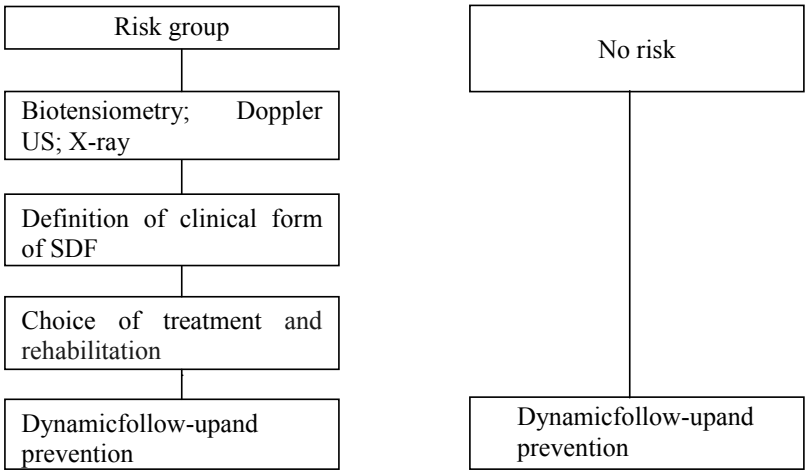


Fig. 37. Algorithm of examination of patients with DM for the exposure of group of risk of development SDF

Algorithm of examination of patients with DM for the exposure of group of risk of development SDF.

Forming of risk groups and determination of degree of risk of development of SDF allows to select the contingent of patients, needing most attention with the purpose of prophylaxis of development of purulent-necrotizing lesions of foot.

The estimation of degree of risk of development of SDF is presented in a table 3.

Table 3

Determination of degree of risk of lesion of lower extremities

Risk degree	Description	Measures
Low	Feels 10º monofilament/vibration of tuning fork. No deformations. Pulse on arteries of feet preserved. No ulcers and amputations	Examination of feet 1 time per a year Self-control Ordinary shoes
High	No sensitivity to monofilament / vibration of tuning fork and/or presence of deformations and/or pulse on arteries of feet absent. No ulcers and amputations	Examination of feet 1 time per 6 months Teaching the care of feet Podiatric care Consultation of orthopaedist on the selection of shoes and / or special adaptations Consultation of oncologist
Very-high	Ulcer and/or amputation in anamnesis at presence of deformations. No sensitivity to monofilament\vibration of| tuning fork and/or presence of deformations and/or pulse on arteries of feet absent.	Examination of feet 1 time per 3 months Teaching the care of feet Podiatric care | Consultation of vascular surgeon Making of orthopaedic shoes of and/or special adaptations

It is possible to consider the followings conditions the risk factors of development of SDF: peripheral polyneuropathy; diseases of peripheral vessels; deformations of feet; expressed decline of sight and complete blindness; diabetic nephropathy, especially on the stage of chronic renal insufficiency; single elderly patients; alcohol abuse; smoking.

Inspection of patient with lesions of feet must include determination of character, volume of purulent-necrotizing focus; vascular status (degree of ischemia); presences of neuropathy; evaluation of the general condition of patient (table 4).

Table 4

Volume of examination of patients with the syndrome of diabetic foot

Types of examination	Obligatory examination	Additional examination
General clinical tests	Clinical blood and urine test Biochemical blood test Coagulogram Electrocardiography X-ray of organs of thorax and abdomen	Echocardiography Immunological tests
Methods, directed on assessment of purulent-necrotic focus	X-ray of foot in two projections Bacterial examination (quality and quantitative) with definition of susceptibility to antibiotics Cytological exam of wound	Radioisotope exam of foot Computed tomography of feet, shins Gas chromatography and massspectrometry Morphological exam
Methods of diagnosis of degree of ischemia of foot and description of lesion of vascular arterial flow	Pulsation of arteries; Doppler US of arteries of l/e with determination of LPI	Full-duplex scan-out of arteries X-ray angiography with the obligatory contrasting of distal arterial vasculature of l/e| (on indications) Determination of transcutaneous tensions of oxygen on a foot laser Doppler flowmetry
Methods of diagnosis of neuropathy	Examination of pain, touch and vibratory sensitivity (dull needle, monofilaments of Sem Weinstein| (5,07), tuning fork) Determination of tendon reflexes (knee and Achilles)	Electro-myography Examination of vibratory sensitivity (biotesiometr)

Treatment of patients with SDF

Treatment and rehabilitation must be multidisciplinary and to unite the following specialists: endocrinology, orthopaedist surgeon, psychologist, trained nurses, technician on making of the orthopaedic correcting devices and shoes. For patients with severe purulent-necrotizing forms of SDF in the command of specialists must be included surgeons of purulent and vascular departments, endocrinologist, internist, anaesthesiologist, specialist in resuscitation.

Treatment depends on form of SDF.

Treatment of neuropathy form of SDF must include the following:

a) compensation of DM;

b) immobilization or unloading of the affected extremity with possible application of orthopaedic devices of correction;

c) local treatment of ulcer (podiatric care) with the use of modern devices and methods;

d) systemic antibacterial therapy taking into account the susceptibility of microorganisms;

e) surgical treatment of purulent-necrotizing lesions of foot with possible subsequent plastic closure of wound (wounds).

Treatment of diabetic neuroosteoarthropathy must include:

a) compensation of DM;

b) in the acute stage the prolonged immobilization of extremity with obligatory application of orthopaedic devices of correction (plaster circular or splint, removable joint-immobilizer, orthesis) and additional devices;

c) in the chronic stage, complex orthopaedic shoes with holdings elements;

d) at presence of ulcerous defect treatment according to the proper algorithm taking into account the necessity of the strict unloading of foot.

Treatment of neuroischemic and ischemic forms must include:

a) compensation of DM;

b) antibacterial therapy taking into account the suceptibility of microorganisms;

c) application of conservative treatmentand vascular interventions methods to decrease the severity of ischemia of foot;

d) local treatment of ulcer (podiatric care) with the use of modern devices and methods;

e) surgical treatment of purulent-necrotizing lesions of foot with possible subsequent plastic closure of wounds.

Compensation of DM is conducted by a doctor-endocrinologist.

Treatment of patients with SDF is conducted by physician-podiatrist, who organizes the process of treatment.

Correction of metabolic violations

Level of glycaemia on an empty stomach, at which the diagnosis of DM is made, is 7,0 mmol/L. And in obedience to recommendations of European Diabetes Policy Group), optimum level of glycaemia is considered on empty stomach is 6 mmol/L, glycated haemoglobin of HbA1c 6,5%.

Criteria on screening of persons with the high risk of development of DM:

- Age (more than 45 years);
- Obesity (body mass index more than 30 kg/ m^2);
- Heredity on DM;
- Ethnic origin;
- Gestational diabetes in anamnesis or weight of foetus over 4 kg;
- Arterial hypertension;
- Dyslipidemia;
- Preceding diagnosis — intolerance to glucose or changed glycaemia on an empty stomach.

At DM of the I type basis of effective treatment is intensifyed insulin therapy which supposes: frequent injections of insulin, self-control of level of glycaemia (not rarer than 2 times per day) and planning of feed with the count of bread units. Adaptation of doses of insulin is conducted taking into account the choice of insulin of «action», that means insulin responsible for a certain interval time. For example, insulin of "action" responsible for night-time there is insulin of the prolonged action, done for the night. The dose of insulin changes gradually, depending on initially entered dosage. The significant increase of dose of the entered insulin can require the considerable change of the mode of physical activity, for example, putting the patient to the bed regimen, or regimen of unloading of extremity. It is necessary to increase the dose of the entered insulin also in the case of fever.

At treatment of DM of II type two approaches are applied according to the following principles:

1. Traditional approach, oriented to strategy of consecutive prescription of antidiabetic preparations;
2. intensive approach, oriented to achievement of special target values of glycaemia.
3. Conservative treatments of DM of 2 type is directed mainly on the removal of symptoms, related to the high level of glycaemia. Monotherapy is initially appointed by a diet. Further at presence of active symptoms of hyperglycaemia one or another antidiabetic preparation is added. Attaining of normoglycaemia only on dietotherapy is succeeded in the minority of patients (diet is effective for 45% patients with first exposed DM of 2 type during first 6 years from the onset of disease, and less than for 20% in 12 years).
4. Intensive tactic of treatment is oriented to achievement of special target values of glycaemia. As a rule, treatment is conducted by combination of antidiabetic preparations, influencing on the separate links of pathogenesis of DM of 2 type, including the removal of postprandial hyperglycaemia (development of insulin resistance of muscular tissue at which its capacity for utilization of glucose goes down, due to that hyperglycaemia and compensatory hyperinsulinemia develops).

Presently in therapy of DM of 2 type 5 basic groups of peroral antidiabetic preparations are used:

– group of biguanids (siophor, methformin);
– group, developed, on the basis of sulfourea (or derivates of sulfourea): glibenclamid, gliclazide, glimepiride, glipizide;
– group of thiazolidindions (rosiglitazone, pioglitazone);
– group of inhibitors of α-glucosidase (acarbosa);
– group of meglitinids (repaglinide, nateglinide).

Meglitinids are to the comparatively new class of medications. They have rapid beginning and short duration of action, that allows, from one side, effectively to control postprandial hyperglycaemia, from other – does not increase the risk of origin of hypoglycaemia.

Presently, the best results of treatment of patients with DM, obtained at the combined therapy. The following combinations of peroral antidiabetic preparations are possible: sulfourea and biguanids, sulfourea and thiazolidindions, methform-

in and meglitinids, methformin and thiazolidindions, sulfourea and inhibitors of α-glucosidase.

If control of glycaemia is not obtained at combination of peroral antidiabetic preparations, the combined therapy of biguanids and insulin or monotherapy by insulin is appointed.

The aims of insulin therapy at DM are following:

1. Prevention of hyperglycaemia coma and cases of cetoacidosis;
2. Relief of symptomsof hyperglycaemia (dryness, thirst, polyuria);
3. Recovery of weight loss with the improvement of the general condition;
4. Reduction of frequency and severity of infections;
5. Prevention of micro- and macrovascular complications.

Medicinal correction of ischemia of l/e (preparations applied in patients with SDF, for the correction of ischemia of l/e).

Preventive treatment:

1. proper pharmacotherapy is used: control of glycaemia, antilipidemic preparations, antihypertensive therapy, antiplatelet therapy, therapy of hyperhomocysteinaemia.

2. organizational work is conducted on the change of mode life of patient: regular physical exercises, no smoking, decline of weight.

Medicinal therapy on the correction of metabolic and hemodynamic violations is conducted taking into account the special target values of level of glycaemia, cholesterol and triglycerides, and also arterial pressure, associated with the low risk of cardio-vascular pathology.

As antiplatelet preparation the small doses of aspirin are most widely used. Clopidogrel is applied at aspirin intolerance. Also, preparations of pentoxyfylline are most often used (trental, agaspurin, vasonit).

Pentoxyfylline influences the deformability of red blood celss, reduces fibrinogen, possesses insignificant antiplatelet activity.

Naftidrofuril (serotonin antagonist) and buflomedil result in the increase of distance of the painless walking (now these preparations are not widely used in clinical practice). Cilostozol, inhibitor of phosphodiesthase, belongs to preparations with antiplatelet activity.

If reconstructive operations are impossible in the patients with critical ischemia treatment by preparations of prostoglandin E1 (Vazaprostan) or by more stable analogue of prostacycline (I leprous) is justified. The prolonged course (during few weeks) of intravenous infusions of these preparations results in diminishing of intensity of pains of rest, and also in healing of ulcerous defects.

Perspective is possibility of gene therapy by preparation on the basis of vascular endothelial factor of growth (VEGF). Studies confirm forming of new collateral vessels at the use of preparations of VEGF.

Pharmacotherapy remains one of important components of treatment in patients after angioplastic operative interventions, especially in persons with the high risk of development of thrombosis.

Methods of unloading of the affected extremity

Mechanical loading – one of factors, which results in formation of trophic ulcer. Therefore, an obligatory condition of the quickest healing of trophic ulcer is a complete reduction of loading on a wound.

The applied methods of unloading depend on what from the situations resulted in ulcer development:

– a wound is located not on an area of bearing (shin, back of foot);
– a wound is located on an area of bearing of one foot (any surface of toes, which is always injured at walking);
– wounds are located on both feet.

Methods of unloading which are used:

– At the ulcers of shin with participating in their development of venous component limitation of vertical position is necessary (in combination with treatment of venous insufficiency);
– At ulcers which are located on the back of foot or in the projection of Achilles tendon, street shoes hinders healing (carrying of home shoes which does not injure a wound is recommended);
– At ulcers which are located in the anterior part of foot, apply the special unloading adaptation, or orthopaedic shoes (an orthopaedic shoe is made in orthopaedic workshops).

Unfortunately, unloading adaptation does not eliminate traumatisation of middle part of foot and heel area. In this situation an individual unloading bandage is made from polymeric fixative («plaster-like») materials.

Technique of making of unloading bandage:

1. After standard treatment of wound and imposition of aseptic bandage on a wound a cotton tubular bandage is put.
2. A few layers of synthetic materials, forming a gasket of 1 cm of thickness, are laid on.
3. A bandage made from fixing material («synthetic plaster»: CELLACAST, Dinacast or Scotch Cast, Soft Cast) is laid on.
4. With the special disk bandage is cut in a horizontal plane, overheadpart retires.
5. In the plantar area of bandage opening is formed in area of wound.
6. During walking the special shoe which retains a bandage and hinders its contamination is used.

The «gold standard» of unloading of extremity, widely applied abroad, is making of unremovable unloading bandage from polymeric fixative materials (Total Contact Cast), allowing to walk, and also daily to treat wounds through the proper openings. The analogue of this technology is making of orthesis on the type of removable «knee-boot».

Orthopaedic correctors which can be made in place from the special silicon are used in mild lesions (for example, ulcer on the tip of toe, associated with rostral deformation).

Methods of unloading which must be avoided:

– application of crutches (unloading often appears incomplete and loading rises on other foot);
– simple limitation of walking («home regimen»). At wounds on area of bearing (or between toes) this method does not provide the valuable unloading, and a wound does not either heal or heals too long (2-3 months and more).

At ulcers of feet both to perform unloading of the affected extremities is more difficult. To use two unloading «shoes» for patient is not possible. Strict bed regimen – patients hardly follow. Therefore, the bed regimen must be combined with applying of unloading bandages on both feet or moving on an wheel-chair (in hospital).

Antibiotic therapy in patients with SDF

Antibiotic therapy in patients with SDF is indicated in cases:

– presence of the infected wound;
– high risk of infecting of wound (circulatory ischemic necrosis, long existent ulcers, large size of wound).

All wounds are divided on sterile, colonized by microorganisms, and infected (the signs of wound infection is not always obvious).

During a few hours after development, a wound is colonized by microorganisms, but local forces of immune defence are able to restrain their reproduction and invasion in deeper tissues. The colonized wound on principle differs from infected. Wound infection is a pathological process, caused by the invasion of microorganisms and developed at certain terms, such as: weakening of protective force, high invasiveness of bacteria.

Patients with SDF have hyporeactivity of the immune system (especially - in elderly patients) which results in decline or disappearance of signs of wound infection. Therefore antibiotic therapy have to be oriented on the local displays of wound infection.

The signs of wound infection strongly differs at acute and chronic wounds.

In first case it is hyperemia, edema, local hyperthermia, purulent exsudate.

In second case (chronic wound):

– tenderness of wound;
– bleeding of granulation tissue;
– unpleasant smell from a wound;
– increased size of wound;
– abundant exudation (even with serosal character of exsudate);
– slow healing of wound;
– atypical colour of granulation tissue;
– formation of «pockets» is in the bottom of wound.

In practice at the ischemic form of SDF antibiotic therapy is required in 90-100% patients. It is related to that on background of ischemia the risk of development of wound infection is high, its signs are obscure, and progress – rapid.

The choice of optimum preparation or combination is difficult enough and must be based on information about the agents of wound infection and their supposed susceptibility to the antibiotics, and also about the features of pharmacokinetics of preparations and location of infectious process (not every antibiotic penetrates to bone tissue). At prescription of antibiotic «blindly», probability of success does not exceed 50-60%.

For achievement of antibacterial effect at SDF application of modern medications with a bacteriolytic action is needed. Penicillins of first generation is not used due to their insufficient efficiency. Preparations with bacteriostatic action are used rarely, mainly, doxicyclin, erythromycin.

Modern macrolides (roxytromycin (rullid), azitromycin (summamed) at high efficiency in treatment of number of infections do not suppress purulent flora, in this connection they are not preparations of choice at SDF.

Aminoglikosides is used rarely, as possess a nephrotoxic action and able to cause kidney insufficiency even in patients with the early stages of diabetic nephropathy.

Before the receipt of results of bacteriologic examination treatment is conducted by the empiric preparations, which operate on majority of microorganisms.

Basic agents of wound infection.

The basic agents of wound infection (infected superficial neuropathic ulcer of the I–II degree) in 90% cases are staphylococci or streptococci.

Mixed-infection develops in deep wounds (3-5 agents). The agents of it can be: gram-positive cocci, gram-negative bacteria (*Escherichia, Klebsiella, Proteus* and other), anaerobic microorganisms (at presence of deep wound or ischemic form of SDF), *P. aeruginosa* and other *Pseudomonas* at excessive humidity of wound.

At high probability of infecting by gram-positive cocci preparations of choice are inhibitor-protected penicillins and cefalosporins (including, II generation), fluorchinolones is less effective . At the mixed flora (anaerobic and aerobic microorganisms) the spectrum of action must be extended to combination of antibiotics: cefalosporin or fluorchinolon with metronidazole or clindamycin, lincomycin). The moderate influence on an anaerobic flora has inhibitor-protected penicillins and cefalosporins of the III generation.

At presence of osteomyelitis combination of preparations, accumulating in bone tissue, is used (lincozamid + cefalosporin/fluorchinolone in high doses).

In absence of clinical effect of antibiotic therapy during 2-3 days, changing of preparation is needed. Cause of ineffectiveness can be stability of agent to the applied facilities, presence of anaerobic flora (often in associations with aerobes, that results in errors during the selection of culture of agent), by low bioavailability of preparation (for example, at peroral application), and also low dosage, negative medicinal interaction. At the receipt of results of microbiological culture, indicative in the presence of resistent microflora, the reserve antibiotics are used. Natural susceptibility of microorganisms to the basic antibiotics which are used at treatment of patients with SDF are presented in a table № 5.

Acquired resistance of microorganisms to the antibiotics

The most known mechanism of antibiotic resistance of microorganisms is making by the staphylococcuss β-lactamase, destroying penicillins and cefalosporins of the I–II generations. The decision of this problem appeared with creation of inhibitors of β-lactamase (clavulonic acid, sulbactam, tazobactam). Inhibitor-protected penicillins is not exposed to the hydrolysis by these enzymes.

However, the number of gram-negative bacteria (Enterobacteriacaeae) synthesizes extended spectrum β-lactamase (ESBL), on which the applied inhibitors do not operate on. So other groups of antibiotics are required in these cases, namely – preparations of reserve.

At the severe forms of wound infection, threatening life or extremity (phlegmons, deep abscesses, moist gangrene, sepsis etc.), antibiotic therapy must be conducted only parenterally, in the hospital, and to combine with the surgical draining of purulent focus and other components of treatment – detoxication, correction of carbohydrate exchange.

Table 5

Natural susceptibility of microorganisms to the basic antibiotics, used at treatment of SDF

Group	Name of antibiotics	Gram +	Gram -	Anaerobes
Cefalosporins of the I – II generation	Cefazolinum, cefamezin, cefamadol, cefalexin	+ +	+ -	0
Cefalosporins of the III generations	Cefotaxim, Ceftriaxon, Ceftazidim and other.	+ +	+ +	+
Cefalosporins of IV generation	Cefepim	+ +	+ +	+
Fluorchinolones of I generations	Ofloxacin, ciprofloxacin, pefloxacin and other	+ +	+ +	0
Fluorchinolones of II generations	Levofloxacin, moxifloxacin	+ +	+ +	+ -
Inhibitor-protected penicillins	Ampicillin/sulbactam, piperacillin/tazobactam, ticarcyllin/clavunat	+ +	+ +	+ +
	Amoxicillin/clavulanat	+ +	+	+
Carbapenems	Imepenem/cilastatin mero-penem	+ +	+ +	+ +
Glycopeptides	Vancomycinum	+ +	0	+ -
Linkozamids	Klindamycin, lincomycin	+ +	0	+ +

++ expressed action; + moderate action; ± insignificant action;
0 – absence of action; Klindamycin is 5-6 times more effective than lincomycin;
metronidazol has the expressed action (+ +) only on anaerobes, on gram$^+$ and gram$^-$ does not have an action.

Minimum duration of antibiotic therapy is usually 7-14 days. Treatment is finished in a few days after disappearance of all of signs of wound infection.

At the ischemic form of SDF minimum duration of antibiotic therapy is 14-21 days, with possibility of prolonged therapy at recurrence of inflammatory changes in wound.

In the prolonged treatment by antibiotics it is necessary to apply eubiotics (colibacterin, bifikol and other).

Measures on minimization of side effects of antibiotics are:

- application of preparations strictly on indications and taking into account the susceptibility of microflora;
- preliminary collection of medicinal anamnesis (allergy);
- screening of the conditions, doing dangerous application of one or another preparations (diabetic nephropathy, lesions of liver parenchyma – chronic hepatitis et cetera).

Surgical treatment of patients with SDF

Active surgical treatment of purulent-necrotizing lesions of feet is used in the patients with DM including:

- debridement of wound;

– additional methods of physical treatment of wound (pulsating stream, ultrasound);
– local treatment of purulent focus by modern bandaging facilities ;
– early restorative operations.

Surgical interventions in purulent-necrotizing processes of foot are most effective in absence of the expressed edema of extremity, at elimination of foot ischemia, during stabilizing of the general condition of patient, correction of carbohydrate exchange and adequate antibiotic therapy.

Aim of debridement of purulent-necrotizing focus is its wide opening, removal of necrotizing tissues, adequate draining and prevention of further distribution of infection. Principle of saving of tissues must be one of basic in surgery of diabetic foot with the purpose of maximal possible maintenance of functions of foot.

The obligatory component of surgical treatment must be implementation of early restorative operations regardless of form of SDF. The removal of defect of foot must be carried out after compensation of the general condition of patient, treatment of infectious process and ischemia of extremity.

Necessity for surgical or orthopaedic operative treatment at SDF arises up in the case of infecting of ulcer and development of progressive phlegmon or moist gangrene; occlusion or stenosis of major vessels, resulting in an ischemia or dry gangrene; at the severe deformation of feet, threatening the ulcer development or hindering the carrying of shoes.

Treatment of the infected ulcers and phlegmons of foot

Infecting of ulcer is recognized on purulent excretions and redness round an ulcer, presence of abscesses and osteomyelitis.

In infecting of ulcer a smear undertakes from an ulcer for determination of agent and susceptibility to the antibiotics and the antibiotics of wide spectrum of action are appointed. After the receipt of result of bacterial culture antibiotic therapy is conducted depending on the exposed susceptibility. antibiotic therapy proceeds to calming down of inflammation.

Besides the infected ulcers of the I stage, the III–V stages are subjected for surgical treatment. Deep ulcers with abscesses or osteomyelitis are treated by draining of abscess, excision of nonviable tissues and resection of bone. When a wound clears up from an infection, a total contact tire is laid on.

Local treatment of ulcerous defect. At local treatment of ulcerous defect it is necessary:
– bandaging must be performed in the conditions of dressing room;
– to provide comfort position of patient;
– to use only sterile surgical instruments (scalpels, scissors et cetera).
– to delete hyperkeratinisation which surrounds wound defect;
– to clean the wound surface of ulcer from necrotizing tissues, blood clots, foreign bodies (sand, dust, fibres of tissue et cetera).
– in the case when an ulcerous defect is closed by dense scab or fibrin application of ointments with proteinase and collagenase activity is possible (Iruxolum) before the complete clearing of surface;
– after surgical care of wound, the cavity / surface of trophic ulcer must be carefully washed by liquid antiseptics (1% and 0,5% solutions of Dioxydinum,

solution of Miramistin, solution of Chlorhexidine, 0,02% solution of Furacilinum), sterile saline solution;

– at treatment of wound surface do not to apply an ethyl spirit, spirit solutions of antiseptics, solutions of permanganate of potassium and peroxide, because they cause the chemical burn of wound surface and have toxic action on granulation.

Choice of dressings.

General requirement to the modern mean of closing of wound is: minimal traumatisation (unadhesion to the wound) and possibility with its help to create an optimum wound environment.

In the first phase of wound process it is necessary to apply noninvasive bandages and sponges with a high absorbancy, allowing as quick as possible to obtain the complete purging of cavity or surface of wound from necrotizing masses and exsudate. On this stage of treatment combining of general antibiotic therapy with local application of antibiotics is possible (ointments "Levomekol", "Dioksikol") and proteolytic enzymes (Iruxolum). In that case, when there is a deep wound defect of small diameter, causing difficulties at the mechanical cleaning, it is necessary to use preparations as powder, granules or gel, allowing to facilitate and accelerate the process of delete of necrotic tissues and avoid violation of outflow of exsudate. Changing of bandages in the phase of exudation must be conducted not rarer than 1 time per day, but at large volume discharge – each 8 hours.

In the second phase of wound process, at infection reduction, in the case of neuropathy form of SDF or other combined pathology, not associated with violation of arterial blood flow, it is possible to turn to the use of hydrocolloid bandages which allow to support an optimum environment in a wound, stimulate growth of granulations. Bandaging facilities, applied for treatment of neuropathy form, are absolutely contra-indicated in patients with the ischemic form of lesion. In the patients with the ischemic form of SDF in the second phase (stage of granulation) it is necessary to use liquid antiseptics (solutions of Dioxydinum 1%, Furacilinum 0,02%, Povidone-Iodum) in combination with non-traumatic dressings. On this stage it is necessary to conduct bacteriological control of wound discharge, because conducted antibiotic therapy can lose efficiency due to acquired resistance of microorganisms or joining other, more often mycotic flora. In the period of proliferation the switch of patient to the out-patient regimen is possible, where changing of modern hydrocolloid dressings can be conducted each 3-5 day.

On the stage of epithelization antibiotic therapy is possible to stop. Liquid antiseptic facilities, non-traumatic dressings, transparent semi-permeable bandages, allowing to avoid traumatisation of young ephithelial and cicatrical tissue and control the wound process, are used. Changing of dressings can be conducted each 2-4 days. On this stage it is necessary the same to follow the regimen of unloading of the affected extremity, the mechanical loading can cause the recurrence of ulcerous defect.

In the period of proliferation, in the patients with the neuropathy form of SDF (not associated with violations of arterial blood flow), in the USA and countries of Europe dressings with the factors of growth (Dermagraft and Apligraf, gel of Becaplermin) are applied.

However, the necessary condition of achievement of the desired therapeutic effect at their use is absolute sterility of wound.

It is possible to apply for fixing of dressings: reticulated bandage, special fixative bandage (Peha-haft, Pehalast), hypoallergic ventile plasters (Curafix, Omnifix).

The ulcers of the IV–V stages require amputations of foot or higher, depending on the level of blood supply. Sometimes amputations on foot are performed in patients with the ulcer of the III stage, depending on the depth of lesion and presence of Sharko joint. Also, the following operations are performed on foot: amputations of toes, transmetatarsal amputation, amputation through the joints of Shopar and Lesfrank. Advantage of these operations is that they save the supporting function of foot.

The most threatening complication of diabetes is a gangrene of l/e. Gangrene makes progress to the level, where blood flow due to developing of inflammatory reaction is able to prevent death of tissues. Process temporally stops on this line. If gangrene is dry, and skin is intact – gangrene arose up only as a result of arterial occlusion, antibiotic therapy is not needed. If there is an infection or gangrene is moist, antibiotics are used to decrease a process or prevent septicaemia. If gangrene involves only the segment of skin and superficial tissues lumbar sympathectomy and, if needed, vascular operation can stop a process.

At the gangrene of distal tips of toes, but good blood flow of proximal part, amputation can be made after formation of demarcation and calming down of inflammation.

If gangrene reaches to foundation of toe, amputation is made through foundation, and a wound is left open and well drained.

Transmetatarzal amputation is performed, when a gangrene involves one or more toes, but does not pass to the foot, and circulation on a foot is sufficient for healing.

Amputations through the Shopar and Lesfrank joints are made in involving of distal departments of foot.

A level of amputation below of knee is operation of choice, when a gangrene or ischemia on a foot hinders local amputation. Maintenance of knee and thigh are very important for prosthesis. Even when blood flow below of knee is bad, often there is healing of stump at the sparing technique of amputation and splintage of stump for the good dressings.

Trans-knee amputation is performed in impossibility of amputation at the level of shin and gives the better stump for prosthesis, than amputation higher than knee.

Amputation higher than knee is performed in patients with the severe vascular lesions resulting in gangrene, in particular, if a foot and shin is infected. It is also performed, if amputation below the knee was erroneous.

Indications to major amputation (level shin/thighs) in patients with SDF it is necessary to define after inspection of volume of purulent-necrotizing lesion of extremity and (or) decision of question about possibility of vascular reconstruction. Methods of examination, applied for the choice of major amputation:

1. clinical information;
2. ultrasonic Doppler of arteries of l/e (full-duplex scan-out of arteries);
3. transcutaneous oxygenation of tissues (if possible);
4. X-ray of foot.

The obtained results of tests are compared with clinical information.

Indications to major amputation:

- extensive circulatory ischemic necrosises of front and middle departments of foot and heel area;
- moist gangrene of foot;
- long existent, extensive trophic ulcers of plantar surface of feet in combination with the severe destructive forms of osteoarthropathy without the signs of consolidation;
- critical ischemia of extremity with the severe pain syndrome, ineffective conservative therapy, impossibility of surgical vascular correction.

Amputations for patients with SDF with the extremely low level of blood circulation must be considered after the decision about possibility of vascular reconstruction and definition of volume of necrotizing lesion of extremity. Amputations at any level must be performed at possibility by flap method. If large amputation is planned the priority is the maintenance of knee-joint.

Maintenance of functional active extremity is one of basic tasks in treatment of purulent-necrotizing lesions of l/e in the patients with DM.

Vascular operations for patients with SDF

Diabetic atherosclerosis of l/e is characterized by low possibility of collateral circulation, involving deep arteries of thigh.

Classic indications to the vascular operation are: claudication intermittens, rest pain, development of ischemic ulcers and gangrenes. Patients with complaints on claudication intermittens usually have aorto-iliac or femoro-popliteal occlusion.

The type of treatment depends on kind and extent of occlusion, location, risk factors and condition of patient.

Transcutaneous balloon dilation, endartherectomy and laser recanalization are used in short stenosis in major vessel.

The by-pass synthetic prosthesis appliance is the operation of choice for treatment of aorto-femoral lesion. On femoro-popliteal segment the by-pass is more frequent made by autovein.

Endartherectomy is deleting of atheromatic plaque from an artery intima. Today this operation is used only with the by-pass.

In patients that have an ulcer or phlegmon, before a vascular operation aggressive treatment of ulcer, dissection and draining of abscesses or amputation of foot, application of antibiotics of wide spectrum of action, is performed.

The receipt of pulsating blood flow on a foot allows to stop distribution of infection and obtain healing of ulcer or conduct amputation more distally.

Orthopaedic operations in patients with SDF

The amount of amputations and hospitalizations is the last years increased concerning the orthopaedic problems of SDF. There is clear connection between autonomous neuropathy and development of SDF. Patients with the neuropathy form of SDF have: valgus deformation of the I toe, rigidity and deformations of the V toe, ingrown nail, Sharko joint.

Surgical interventions are used for the correction of biomechanics abnormalities, which preceded ulcering.

Ostectomy is used to delete bone prominences which directly put pressure on skin and result in ulcering. Prominences located laterally and medially are removed through lateral and medial incisions, More central prominences are removed through an ulcerous defect.

For treatment of ulcer under metatarsal heads metatarsal osteotomy or resection of head of metatarsal bone is performed.

If ulcer of toes takes place due to hammer or sharp-clawed deformations of toes – a plastic operation on soft tissues, removing of deformation or proximal interphalangeal arthroplasty, is needed.

If an ulcer developed on prominent middle phalanx and combines with valgus deformation of the I toe, the arthroplasty of the first proximal phalanx of toe is needed.

Mechanically and neurologically unstable foot (foot of Sharko) which is clinically characterized by the loss of foot arch and tendency to expansion of foot laterally in a forehand and by valgus deformation of back-end; with the purpose of stabilizing and prevention of ulcers treats arthrodesises are used. In presence of ulcer on such foot amputation of foot is inevitable.

Rehabilitation and prevention in patients with SDF

Rehabilitation of patients of DM with SDF must be early, to begin on the stationary stage and based on interdisciplinary approach.

Basic measures of prophylaxis in patients with SDF:

1. Adequate therapy of DM, directed on achievement of recommended today «aims of treatment» in the maximallygreater number of patients.
2. Teaching of patients to the measures of prophylaxis of lesions of feet.
3. Exposure of patients with DM with the significant risk of lesion of l/e (risk groups).
4. Part of patients from the group of risk needs prophylactic podiatric care (in a cabinet «Diabetic foot»).
5. Treatment of the conditions, predisposing to development of SDF: timely vascular operations in illnesses of arteries and veins, orthopaedic shoe for the groups of high risk etc.
6. Creation of possibility for an urgent appeal in a cabinet «Diabetic foot» in the case of damage of l/e.

It is known that formation of neuropathy ulcer takes a place only after mechanical or other damage of skin of foot. At the ischemic form of SDF a damage also often becomes crucial moment for development of necrosis of skin. In this connection the special recommendations are developed for patients allowing to minimize the risk of such damages.

Complex of prophylactic measures, allowing in sufficient degree to reduce the risk of lesion of l/e, it is possible to present as «prohibitive» and «permissive» rules.

Next positions behave to the «prohibitive» rules (eliminate factors, able to injure tissues feet):

1. To forbid to use acute objects during care of skin of feet (by scissors, callosity knives, shavings blades, tongs).

2. In patient with decreased sensitivity of feet bad sight help of relatives or specialists is possible.

3. If feet freeze, it is not necessary to warm them by hot-water bottles, devices or radiators. If patient has decreased temperature sensitivity – he will not feel a burn.

4. It is impossible to use hot foot-baths. In addition, foot-baths must not be protracted, it does skin more vulnerable.

5. It is not recommended to walk barefoot (including at home).

6. It is necessary to give up an uncomfortable (pressing, rubbing) shoe and not carry a shoe on a high heel, because circulation of blood is here violated in a foot and the areas of enhanceable pressure appear on its plantar surface.

7. If there are callosities on feet, it is impossible to try to delete them by a callosity plaster or keratolysis ointments and liquids, as these facilities damage a skin.

8. It is necessary to pay a regard to elastics socks: too tight elastics cause impression on the skin of shins, that hampers circulation of blood.

"Permissive" recommendations:

1. Treatment of nails must be made by safe method. The edge of nail needs to be filed horizontally, do not touch corners. In other case ingrown nail, which causes pain feelings, inflammatory process and requires the prolonged special treatment, can evolve.
2. The most suitable mean for removal of callosity and areas of hyperkeratinisation is pumice. Do not to try to removal callosities during one washing of feet.
3. It is necessary to oil the dry areas of skin by fat cream. It prevents formation of fissures - possible entrance gate for infection.
4. It is necessary to wipe leg after washing, not grinding, especially in interdigital spaces. Increased humidity in these areas is instrumental in development of intertrigo and mycotic diseases
5. If feet freeze, use the warm socks of the proper size and without tight elastics. It is necessary to watch, that socks were not hammered together in a shoe.
6. It is needed to accept rule each time to check up the internal surface of shoe before to put on: whether some extraneous objects, able to wound a leg.
7. Daily the patient of DM must attentively examine feet, especially plantar surface and interdigital spaces. Elderly people and persons with an overweight can have here certain difficulties. They can be recommended to use a mirror, set on the floor, or (at the decline of sight) to ask relatives about a help. This procedure allows in good time to discover wound, crack, shabbiness.
8. Wound or crack, discovered at examination of foot, it is needed to wash disinfectant solution. For this purpose it is possible to use 1% solution of, solutions of miramistin, chlorhexidine. The washed wound is necessary

If the damage of skin did not relieve after 1 2 days - it is necessary to appeal to the doctor, desirably – to the specialized medical establishment (cabinet « «Diabetic foot»).

The following of these simple rules will allow patients to reduce the risk of gangrene and subsequent amputation.

Chapter V. Surgical pathology of the arterial system

V.1. Chronic occlusive diseases of major arteries of extremities

The chronic occlusive diseases of arteries of extremities (CODAE) take considerable pleaseamong the diseases of vessels, have different names, different etiopathogenesis and morphology changing, but clinically is enough similar. The indicated diseases, mainly, are characterized by the heavy, making progress evolution and are often resulted in the long loss of ability to work and invalidation of patients. If to take into account that it is suffered by them, in considerable part, men of middle ages and young, and the conservative and surgical methods of treatment are not enough effective, that is confirmed by frequency of amputations of the affected extremities — social importance of problem and necessity of persistent searches of effective methods of treatment of this difficult pathology becomes obvious.

Earlier by the chronic occlusive diseases of arteries of extremities suffered of about 12% population of earth, in our time - 18%. The amount of amputations at this group of patients makes 16-37%.

In USA annually performs about 62 thousand amputation of extremities due to occlusive diseases of arteries and a loss makes near 9 billion dollars a year. The cardio- vascular diseases confidently lead in the unencouraging rating of reasons of death rate and disability in world, including, and our compatriots. Statistics establish insistently, that illnesses of the system of circulation of blood annually take away half a million lives of citizens of Ukraine, and specific gravity of death rate from these diseases makes almost 65%. This in 12 times more than in the countries of Europe, USA, Canada.

According to results of Medical Statistic of Ukraine, for the first half-year of 2000 year the general index of patients with the ischemic diseases of extremities made 322 patients on 10000 populations.

Anatomy and physiology features of the arterial system of extremities.

Main arteries of upper extremity: blood circulation is performed through a subclavia artery (on the right is branch of brachiocephalicus trunk, on the left are branches of arc of aorta) which passes to axillaries artery, which passes to humeral; a humeral artery in upper third of forearm divides on ending branches - radial and ulnaris arteries, which forming superficial and deep arterial arcs of manus.

Palpation of pulse on the arteries of upper extremity is accessible:

- *axillaries artery* — near the internal border of roller which formed by the muscles coracobrachialis and short head of biceps muscle;
- *humeral* – on a medial surface in middle third of shoulder in internal humeral fissure which delimits the internal border of biceps muscle, and also,on a medial surface above an elbow bend;
- *radial* - in lower third of forearm on a border with a wrist joint on the internal border of brachioradialis muscle;
- *elbow* – in lower third of forearm on the elbow border of wrist joint.

Magisterial arteries of lower extremity: an external iliac artery (branches of general iliac artery in small pelvis) passes below than inguinal ligamentum to the short general femoral artery from which the deep artery of thigh departs in upper third of

thigh, and it prolong as superficial femoral artery which in popliteal pit has the name popliteal; the last in upper third of shin divides on anterior and posterior tibialis; a fibular artery walks away from a posterior tibialis artery. An anterior tibialis artery passes to the dorsal artery of foot and an artery tibialis posterior — in the middle and lateral arteries of sole.

***Palpation of pulse on the arteries of lower extremity*:**

- *general femoral* – on a thigh under an inguinal ligament in the projection of vascular bed (in the middle of inguinal ligament);
- *popliteal* - in popliteal pit in the middle;
- *dorsal tibial artery* – behind internal epicondylus (between it and the Achilles ligament);
- *posterior artery of foot* - in the meddle of back surface of foot laterally from the ligament of long extensor.

> **The chronic occlusion diseases of arteries of extremities (CODAE)** *are characterizedby general pathological substrate – local or diffuse, gradually increasing stenotic process in main arteries, which ends with complete occlusion of vessels and are the cause of community of two most important clinical signs, – increasing ischemia and the making progress trophic disorders of tissues of distal parts of extremities the eventual result of whichdevelopment of irreversible necrobiotic changes -gangrene of extremity.*

It grounds to observe of these diseases in one group, but at the same time, diseases are different by etiology, character of morphological changes in vessels, by clinical evolution.

In a clinic accepted next classification CODAE.

A. By etiopathogenetic principles:

1. Obliterated atherosclerosis.
2. Obliterated endarteritis.
3. Obliterated thrombangitis (illness of Burgher).
4. Other rare forms (the Renault illness and other).

The second and third above-mentioned forms (obliterated endarteritis and obliterated thrombangitis) in western English-language literature are consider as one disease — obliterated thrombangitis or disease of Vinevarter-Burger in which both the arterial and vein beds are damaged. The term of «obliterated endarteritis» is not mentioned.

At the same time, in Medical classification of diseases (MCD-10 2000) «obliterated endarteritis» is selected as nosology form.

B. By localization of pathological process:

I — peripheral and main arteries of extremities (obliterated atherosclerosis, endarteritis, thrombangitis);

II — terminal parts of abdominal aorta, it bifurcation and iliac arteries (**the Lerysh syndrome);**

III — branches of arch of aorta (**the Takayasu syndrome**).

C. On the degree of violation of circulation of blood with development of chronic ischemia in tissues of extremity by Fontaine – A. A. Shalimov(1954, 1979)**:**

The I stage is complete compensation: symptoms are - a sensitiveness to the cold of foot, promoted fatigueability, paresthesia, inconstant pallor of skin of distal segments of extremity which will be replaced on the bright rose coloring; by A.A.Shalimov is easy degree.

The II stage — insufficiency of circulation of blood at the functional loading: a leading symptom is the symptom of the «intermittent claudication» , that characteristic by the irregular walking with the stops through certain distance due to appearance of ischemic pain which occurs in dorsal muscles of shin and passes after of shortrest; strengthening of paresthesia, numbness; resistant pallor of skin and cyanosis of toes,lowering of skin temperature, the initial appearance of trophic disorders - fragility of nails, hyperkeratosis; by A.A.Shalimov - degree of «transient ischemia».

The III stage — arterial insufficiency of extremities at peace: a basic symptom is permanent and nightly pain («pain of rest», «rest pain») in a foot, separate fingers, which violates sleep of patient; more expressed symptom of the «intermittent claudication» (strengthening of intensivety of pain and diminishing of distance between the forced stops); making progress of trophic disorders as thinning and atrophy of skin, diminishment of volume of shin due to atrophy of muscles,fall of hairs, appearance of fissures on fingers and between them, «marble» of skin covers of distal segments of extremity; foot cold at palpation; by A.A.Shalimov - degree of «resistant ischemia».

The IV stage — the expressed destructive changes of tissues of distal segments of extremity: making progress trophic ulcers, focal necrosis, gangrene, joining of infection; permanent intolerant pain in toes, foot and sometimes and all leg, that deprives sleep (characteristically forced position of patient in a bed with the lowered sick leg), which is often removed only after administration of narcotics. The patients become mentally unstable, aggressive; making progress the other trophic disorders as a «parchment» skin, atrophy of muscles of shin, deformation of nails; a skin has bluish discoloration at joining of infection - purple color, edema of rear of foot; sharply expressed symptom of the «intermittent claudication» (a patient can pass no more than 7-10 m without stop); by A.A.Shalimov - degree of the «complicated ischemia».

Etyopathogenesis and pathomorphology of CODAE

Obliterated atherosclerosis of arteries of extremities which is, as a rule, evidences of general atherosclerosis, takes leading place at CODAE, affecting about 90% patients of this group inage more than 40 years, more frequent men.

In spite of enormous interest to this disease, multilateral its study, until now its pathogenesis not enough clear, and etiology – not completely known too. There is suspicion to examine atherosclerosis, as polyetiologic disease, in development of which play hyperlipidemia, high blood pressure, and surplus mass of body, not mobile way of life, psychical overloading, diabetes mellitus, fat food. The indicated factors are examined as factors of risk of disease, have the special value hyperlipidemia, high blood pressure, and smoking. Genetic predisposing in combination with the factors of risk also acts certain part.

For the atherosclerotic affectation of arteries of extremities the following *morphological features* are characteristic:

1) a pathological process, mainly, develops in the internal wall of artery (intimae), where forming atheromatous plaques containing a cholesterol, lipids, calcium, and, compare with endarteritis, does not spread on other layers of wall and periarterialy;

2) affectation of arteries of large and middle caliber (abdominal aorta, iliac, femoral, rarerpopliteal arteries), more frequent the disease begins in proximal section, and then lowering to dystally;

3) character of affectation, as a rule, segmental;

4) more frequent is one-sided character of affectation.

Thus, *occlusive character of affectation at atherosclerosis — proxymal, descending, segmental, one-sided. Often combines with the atherosclerotic affectation of other organs (heart, brain).*

The indicated morphological features provide more favorable terms for compensation of the disordered main circulation of blood of extremity (formation of collaterals) and allows to speak about relatively more the of high «quality» flow of atherosclerosis by comparison with endarteritis (more rare and more late evolution of irreversible necrobiotic changes in extremity), and also possibilities for implementation of reconstructive vascular operations.

Obliterated endarteritis arises up in age 20-40 years, strikes almost exceptionally men (95%). The arteries of extremities are affected mainly, mostly lower, however the disease has the systemic character, because also suffer and the vessels of heart, kidneys, cerebrum, intestine.

Etiology is remains not completely know. On today, smoking is considered one of principal reasons of development of disease (hypersensitiveness to nicotine). In the origin and development the diseases matter also and a lot of other factors: long repeated coolings of extremities, is special – in a moist environment, traumas, thus not only extremities, but and cranial-cerebral trauma, allergic processes with the autoimmune reactions, nervous mechanisms(degenerative and inflammatory changes in sympathy nervous ganglions, in peripheral nerves), endocrine factors, in particular, hyperfunction of suprarenal glands - that, big variety of pathogenetic mechanisms.

Morphological features of endarteritis (unlike atherosclerosis):

1) a pathological process begins in the sub endothelial layer of artery, where on a background a long spasm proliferation of cells, which brings the gradual narrowing of the vessels, intima is thickened due to hyperplasia of subintimal tissues, then adding an inflammatory process which carries character of panarteritis with transition on a paravasal cellulose and subsequent fibrosis of muscles coat;

2) distal arteries (mostly feet) with a next affectation more of proximal segments (arteries of shin, popliteal artery);

3) as a rule, character of affectation is diffuse;

4) affect both lower extremities (the successive affectation is possible), and then-upper extremities.

Thus, **occlusive character of affectation at endarteritis is distal, ascending, diffuse, besides.**

Indicated morphological features of obliterated endarteritis, stipulating practically complete absence of possibility for development of collateral circulation of

blood, prevalence of affectation of main arterial bed of extremity, result of the quickly progress of malignant development of disease at young people, which formation of the irreversible destructive changes in tissues of distal section of extremities (trophic ulcers, necrosis, gangrene) which can develop during a few years. The patients may often lose lower extremities at first, and then upper in age up to 40 years.

In spite of likeness with obliterated endarteritis it considers that **obliterated thrombangitis (disease of Burgher)** has differences in morphology and mechanisms of pathogenesis.

If obliterated endarteritis, as was already specified, is morphologically characterized by the gradual narrowing of internal lumen of artery on a background a long spasm, due to hyperplasy of subintimal tissues and sclerosis of muscles coat, that, at first a proliferate-dystrophy process in an arterial wall and already secondary the protracted and flabby inflammatory reaction in a walland in a paravasal cellulose; veins, as a rule, are not changed, obliterated thrombangitis from beginning develops as the expressed inflammatory-allergic process which at once affected all layer of wall of artery and develops on the type of panarteritis with involving of paravasal cellulose and, main - with the impair of veins. Acute migrant thrombophlebitis of hypodermic veins and acute thrombosis of accompanying deep veins is the important clinical sign of thrombangitis.

It is equal as endarteritis; thrombangitis is characterized **by distal, ascending, diffuse character of occlusion process** and malignant development with the serious affectation of other organs.

Clinical picture of basic CODAE

The clinical evidences at CODAE, foremost, depend on the degree of chronic ischemia of tissues of distal sections of extremity, symptomatic and evolution of which, depend from many factors: anatomy, morphology, biochemical and patophysiology condition from which most important are following: caliber of the affected vessel, level and expressed of occlusion (complete or partial), speed of its progress, volume of affectation, intensity of vascular spasm, condition of the system of homeostasis, central circulation of blood and cardiac activity, speed and efficiency of evolution of collateral circulation of blood in the affected extremity.

The classification of degrees of chronic ischemia of extremities proposed higher reflects basic symptoms and clinical evolution of CODAE.

Absence of pulsation on the certain segments of main arteries of extremities is the most essential objective symptom CODAE (Fig. 38).

It is necessary to remember in the plan of differential diagnostics, that at obliterated atherosclerosis due to affectation of

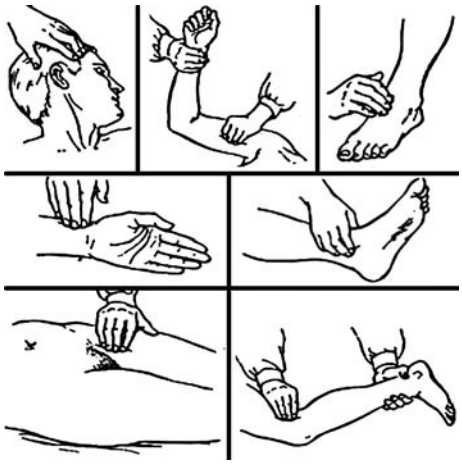


Fig. 41. Determination of the main arteries pulsation on the extremities and neck

proximal segments of arterial bed of lower extremities absence of pulsation is determined on a foot, shins, in popliteal pit, and at the Lerish syndrome and on a general femoral artery, while at obliterated endarteritis and thrombangitis due to impair of distal segments of arterialbed of extremities a pulsation is absent more frequent on a foot andshin, rarer in popliteal pit, and, as a rule, is determined on a general femoral artery.

By palpation the lowering of skin temperature is determined, violation of sensitiveness,by auscultation systole noise above the stenotic area of vessel is determined (at atherosclerosis). At the IV degree of ischemia limitation of volume of active motions in distal joints, may be present symptom of Krakovsky ("symptom of the bend fingers or claws like foot").

In 1982 for denotation of group of patients with pains at peace, with trophic ulcers and distal necrosis first the entered term "critical ischemia of extremities".

Clinic criterias of chronic critical ischemia of lower extremities are: permanent ischemic pain, which increases at night time, there is necessity in permanent anesthetize during more than 2 weeks; presence of trophic ulcers or gangrenous changes, which located usually on nail phalanges of toes, calcanea region, dorsal surface of foot.

Clinic of the Lerish syndrome at stenosis or occlusion of terminal part of abdominal aorta, its bifurcation and iliac arteries (first described by Lerish in 1923), as a rule, atherosclerotic genesis, unlike the occlusion affectation which are localized distally than inguinal ligament, is characterized more frequent by the bilateral impair of lower extremities, pain at walking is localized not only in feet and shins but also in thighs, buttocks, lumbar region and even in the lower parts of abdomen, the heavy form of the «intermittent claudication» develops quite often, when patients can not without the stop pass more than 15–20 m; characteristic changed color of skin of the affected extremities in the color of ivory and at the half of patients impotence (symptoms, described by Lerysh) is marked, hairs are absent not only on a shin but also on the distal sections of thigh; at auscultation in area bifurcation of aorta (from the side of the back or abdomen), on femoral arteries is hearkened the systole noise; a main objective symptom is absence of pulsation on a general femoral artery. The clinical feature frequently is relatively favorable, makes progress slowly, rarer, than at more distal occlusion, and a gangrene develops enough lately (due to good terms for development of collaterals).

A. A. Shalimov distinguishes 4 form of affectations:

I — high occlusion of the abdominal aorta with affectation of visceral or kidneys arteries;

II – occlusion of the terminal part of aorta or both general iliac arteries (typical syndrome of Lerish);

III – one- side occlusion of iliac artery;

IV — occlusion –stenotic affectation of bifurcation of aorta and external iliac arteries with saving of blood circulation in general and internal arteries.

The Takayasu illness is pathology which carries different names (unspecific aortoarterit, «illness of absence of pulse», «syndrome of arch of aorta»). First description of this disease belongs Davis (1839). In 1908 the Japanese ophthalmologist Takayasu brought exhaustive description of this syndrome which from that time has the name of the Japanese researcher.

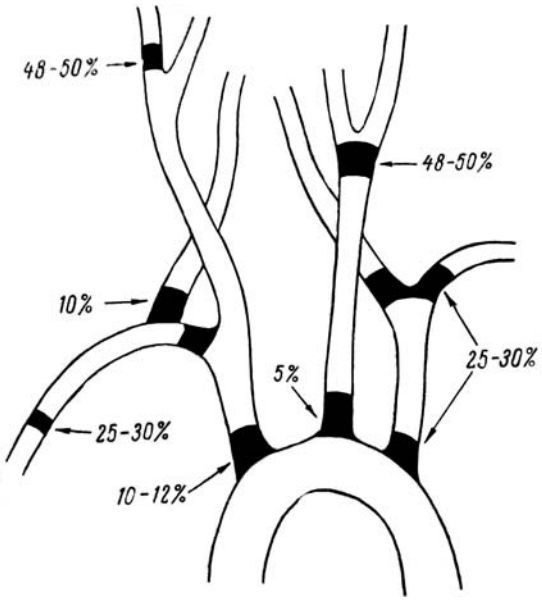


Fig. 39. Frequency of brachiocephal arteries damage

Frequency of affectation by the occlusion process of brachiocephal arteries is different: more frequent than other affected internal carotids and bifurcation of general carotid — 45–50%, than subclavia arteries — 25–30%, brachiocephal trunk – 10–12%, vertebral arteries — 10% and rarer than all suffers a general carotid — 5% (Fig. 39).

Etiologic factors which cause violation of blood stream in brachiocephal arteries can be parted on 3 groups:

I – are affectation of vascular wall;

II – anatomic features of vessels,

III – extravasal factors.

The first group of reasons prevails – 82 %, to the second and third groups is according 10 and 8 % (B.V.Petrovsky, 1970)

I. ***Pathological processes in an arterial wall***:

1)atherosclerotic occlusion favorite localization of which is the bifurcation of general carotid, often with transition on an internal carotid, mouths of vertebral arteries and brachiocephal trunk, the affectation carry segmental character, suffer mainly blood supply of head and cerebrum;

2) unspecific arteritis – affected, mainly, subclavia arteries (80-90%) and carries widespread character;

3) syphilitic aortitis and aneurism arising up on its background, is localized mainly in the ascending part of arch of aorta, can be as the reason of violation of blood stream in brachiocephal arteries (more frequent - in a brachiocephal trunk)

II. ***Anatomic peculiarities*** — the change of ordinary motion of artery due to excessive length of vessel with formation of corners and loops, displacement of mouth of artery, different anomalies can serve as the reason of violation of blood stream in brachyocephal arteries.

III. ***Extravasal factors.*** The compression of brachiocephal arteries is possible as a result of anatomic features of structure of cervical-axiliary channel, peculiarities of structure, fixation and variants of anatomy of scalenus muscles and big pectoral muscles, specific of motions of head, neck and upper extremities, innate anomalies of bone skeleton, consequence of traumas, inflammatory processes and new formations.

More frequent than all subclavia artery in all its departments is subject of the outward compression. The possible variants of this compression are following:

1) syndrome of additional neck rib;

2) syndrome of anterior scalenus muscle, (at the people of certain professions are auto locksmiths, artists, house-painters and others);

3) bones-clavicular syndrome;

4) hyperabduction syndrome.

The permanent trauma of subclavia artery at the above mentioned syndromes, causing at first as a result of permanent irritation and long spasm and than moderate changes in a wall vessels, in future brings gradual stenosis and occlusion of artery, its thrombosis is possible also.

The clinical picture of disease is determined by the anatomic features of affectation (one or a few branches of aorta are involved), by localization and long of occlusion and related to this violation of circulation of blood.

Distinguish ***proximal and distal forms*** of the Takayasu disease depending on a place, where occlusion of vessel happened: at the place of branching of the arch of aorta or more distally. The indicated circumstances result in the considerable variety of clinical picture of this pathology.

Symptomatic of disease consists of signs of insufficiency of blood supply of cerebrum and face, and also arterial insufficiency of upper extremities.

At stenosis or occlusion of carotids the disorder of the central nervous system characteristic by head pain, dizziness, loss of consciousness; at the heavy degrees of ischemia there can be the transient paralyses of central origin, disorder of vision as the temporal loss of a different duration. At considerable duration of disease there is atrophy of skin and muscles, which results in the expressed asymmetry of face.

At stenosis or occlusion of subclavia arteries insufficient arterial blood supply of muscles of upper extremity results in the intensive fatigueability and sensitiveness to the cold of hands, to appearance of pain at active motions, at the promoted physical loading (carrying or elevation of weight), to the considerable lowering of muscles force, appearance of pain at peace.

At objective research detects absence of pulsation distally than place of occlusion of vessel (on a neck, on upper extremities, branches of external carotid), at auscultation is systole noise distally of occlusion, which can be conducted and be heard on a neck, in a subclavicular region and axillaries pit.

Peculiarities of investigation of patient with suspicion on CODAE.

In spite of difficulty of recognition of vascular diseases, the correct diagnosis of majority from them can be detected at the ordinary clinical inspection of patient. Important, that it was careful and it is enough by detailed. The special methods, as a rule, only specify details. On different stages of clinical inspection it is possible to expose symptoms important for diagnostics of vascular disease.

Questioning of patient allows to define the basic complaints of patients.

Above all things detects the symptoms related to insufficient blood circulation of some organ. Their appearance or strengthening in the moment of functional activity of organ is characteristic.

Pain in feet, which arises up at walking and disappears at peace. It is the pathognomonyc symptom of stenosis or occlusion of arteries of lower extremities or bifurcation of aorta and named as «intermittent claudication».

Similar **arm ache** evidence of affectation of arteries of upper extremities.

A *abdominal pain* on height of digestion allows to suspect chronic violation of visceral circulation of blood.

Head ache can be the symptom of insufficient cerebral circulation of blood.

By character, intensivety and localization of pain can judge about the type of violation of circulation of blood, degree of ischemia and speed of its development.

A muscles weakness also is the permanent satellite of the disordered circulation of blood in extremities and often accompanies a pain syndrome.

Paresthesia especially localize also can be the symptom of the disordered circulation of blood. For them periodicity and strengthening in the moment of functional activity is also characteristic. For correct estimation of these symptoms information about their duration is important, to the general dynamics of development, efficiency of the used drags.

Clinical physical inspection.

***Examinatio*n** allows detects violations of trophic of tissues important for diagnostics of vascular diseases.

Muscles hypotrophy usually develops at chronic violation of arterial blood supply.

The dystrophic changes of skin and its appendages (thinning of it, fall of hairs, change and fragility of nails and others) also often accompany chronic insufficiency of circulation of blood in this area.

Discoloration skin (pallor, cyanosis) at vascular pathology is ordinary local and has a big diagnostic value.

Auscultation must be performed in all case, is special at the inspection of patient with the atherosclerotic affectation of the arterial system. In a norm over a main artery it is possible to hear out the conducted tone of blow of pulse wave. There is systole noise at narrowing or pathological expansion of artery, and at the overshoot of blood from arterial to venous bed – systolic-diastolic noise.

The physical clinical inspection of the patients with CODAE ending with conducting of functional clinical tests, which allowto definea level and degree of violation of main and to estimate development of collateral circulation of blood. The following tests are used:

- **Oppel** («symptom of plantar ischemia») - a patient lies on back and elevates lower extremity up. A doctor watches after the color of feet and marks time of their going pale;
- **Samuels-Goldflam** – control appearance of paleness of skin of foot (Samuelsa) and rapid appearance of fatigueability during work of ankle joint (Goldflam) of the affected extremity in position of lower extremities lifted up on 45-70 at horizontal position of patient;
- **Leniel-Lavastyn** («symptom of white spot») — after pressure by two fingers is simultaneous on the back surface of big fingers of healthy and sick leg, appearing on the affected extremity a pale spot disappears considerably longer, than on a healthy leg;
- **Kosachesku** (with determination of dermography from an inguinal ligament to lower third of shin) — the phase of white dermographymore long at times in the distal section of leg to the certain proximal level in is comparable with healthy**;**
- **Moshkovich -** determination of level of «reactive hyperemia» in vertical position of patient since he in horizontal position will hold for some the time lifted up on a 45 sick leg during 5-7 minutes;
- **Kollensa-Vilensky** — detection of degree and speed of filling of superficial veins in transition of patient from horizontal one in vertical position;

– «**knee phenomenon» of Panchenko** — appearance of symptom of «crawl of small ants» or «sat leg» out at finding of patient in sitting position with the sick leg putting up on healthy during 5-7 minutes;
– **functional test Alekseev —** measuring of skin temperature of extremities before and after long walking (to 2000 м), that exposes its diminishes on the affected extremity;
– **time of reactive hyperemia on Shamov** — determination of time of appearance of «reactive hyperemia» after the compression of arteries of shin of the staggered extremity during 5 minutes by the cuff(in a norm — through 15-20 sec, at pathology — considerably more later, sometimes through 2 and more minutes).

It is necessary to know also the diagnostic criteria of obliterated thrombangitis by Olyn (2000):

– age up to 45 years;
– smoking is permanent or in anamnesis;
– presence of ischemia of lower extremities, which manifest by intermittent claudication, pain at peace, ischemic ulcers or gangrene confirmed by noninvasive researches of vessels;
– exception of autoimmune diseases, hypercoagulative states and diabetes mellitus; absence of proximal sources of emboli by result of ULTRASONIC or arteriography investigations;
– proper changes of vessels by result of angiography.

According to standard charts, the plan of inspection (laboratory and instrumental) of the patient with CODAE includes the following: Clinical blood test. Biochemical blood test. Clinical analysis of urine. Blood type. Coagulogramm. EKG. General roentgenography of the chest. Determination of function of the external breathing. Special researches of vessels (information of angiography, ULTRASONIC of vessels and etc).

Instrumental methods of investigation.

Except for the generally accepted obligatory laboratory researches (clinical blood, urine, biochemical blood and coagulogramm), the following methods of research are used:.

1. Ultrasonic research of vessels (ultrasonic dopplerography) is a research method, gives possibility to inspect the lumenof vessel, to measure the thickness of its wall, presence of obstacles to the blood stream (emboli, atherosclerotic plaques), but also to define direction, character andspeed of blood stream, to define age of blood clots and others. On today is one of basic methods of estimation of the state of main vessels.

2. Angiography – roentgencontrasting research of vessels on the special x-ray photography set (angiograph) is a basic method which allows more exactly to diagnose localization and length of affectation of arterial bed and to decide a question about possibility and character of operative reconstruction of the affected arteries.

A few types of angiography are used:

At localization of disorder of lower extremity distally than inguinal ligament, when the pulsation of femoral artery is determined, transcutaneus **femoral arteriography** is executed by **Seldynger**, using for this purpose the special punction needle of Seldynger and transitional tube which connects a needle with automatic

syringe. Performs puncture of the femoral artery at once below than inguinal ligament, injects a 40-45 ml liquid contrasting matter (urotrast, triombrast, omnipak) and make the videotape recording or X-ray films of passage of contrast on different segments of arterial bedof extremity (on femoral, popliteal, to the tibial arteries and other).

At the Lerish (occlusion of aortoiliac segment) **syndrome**, when the pulsation of femoral artery is not determined, execute **translumbal puncture abdominal aortography by Dos Santos** , for what the special long punction needle is used. The place of puncture at position of patient on an abdomen is a left costal-vertebral corner, direction of needle - to the spine, a needle puncturing an aorta, inject a 60–80 ml contrasting matter and make the record or X-ray film of passage of contrast on an abdominal aorta, to the general, external and internal iliac arteries and to the arteries of different section of extremity (femoral, popliteal, tibial) (Fig. 40, 41).

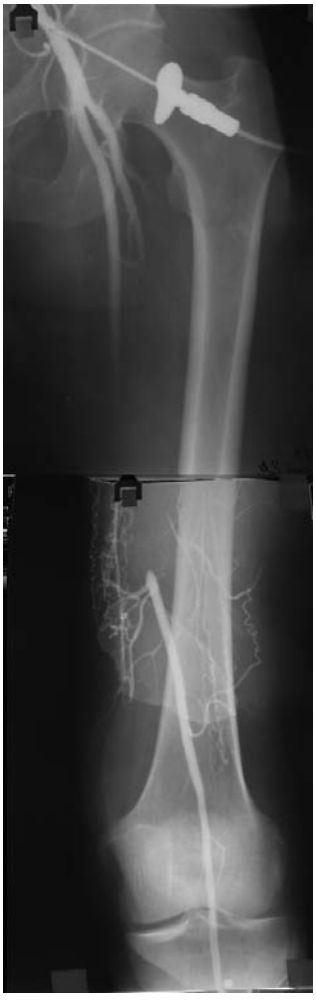


Fig. 43. Femoral arteriography by Seldynger Segmental occlusion of right superficial femoral artery

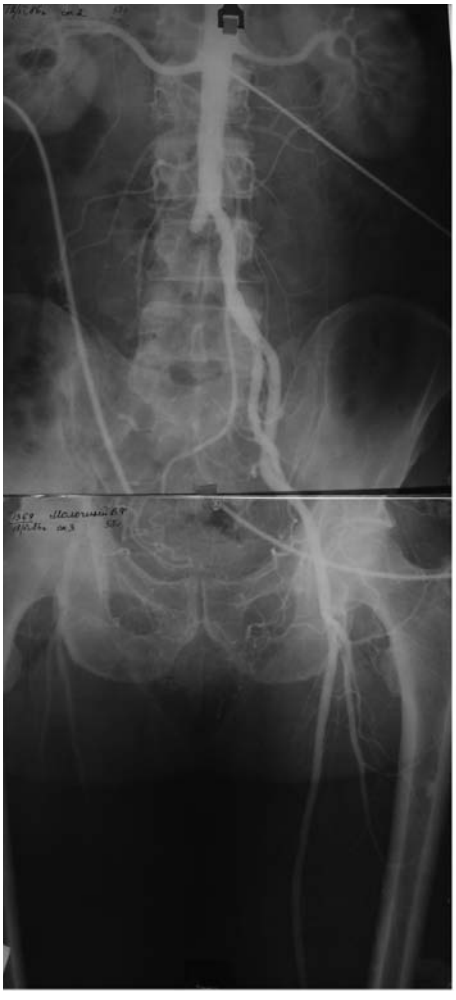


Fig. 44. Translumbal puncture abdominal aortography Occlusion of aortoiliac segment

At localization of affectation in the branches of arch of aorta - **at the Takayasu disease** execute c**atheterization pectoral aortography:** by the Seldynger needle a femoral artery puncturing below than inguinal ligament, a flexible metallic guide is entered at its lumen, a needle is removed, and the guide inserted in an iliac artery, a catheter is entered, an guide is deleted, and passed a catheter toward to arch of aorta, enter a contrastand do the record of passing of contrast on an arch and pectoral aorta, on general, internal and external carotids, on subclavia, axillaries, humeral, radial and elbow arteries.

***Basic angiography signs of occlusive affectation of arteries*:** equal narrowing of lumen, segmental stenosis or segmental occlusion, total occlusion of distal arteries (symptom «amputations», «stump of vessel»), expansion and bending of segment of artery, unequal narrowing,presence of expressed number of collaterals.

Two above-mentioned methods have the prevailing value in diagnostic of CODAE , other, resulted below – only auxiliary methods which are used, mainly, in policlinic terms**.**

Oscillography – is based on the record of pulse vibrations of arterial wall after compression of extremity by inflateble cuff.Is characterizes a main blood stream. Depend from kind of oscillation estimate the passage of artery tone of its wall. Determine: maximal, middle and minimum B/P (for middle third of shin the Mx 120-140, Md 180-105, Mn 105-80 mm Hg column. a basic index is oscillography index (OI) is the height of the most high indent norm of which in middle third of shin 10-15 mm (and on a radial artery) measured in mm, on a popliteal artery — 20-25 mm, on a femoral artery – 30-35 mm.

Reography longitudinal segmental — registers pulse vibrations of resistance of tissues of extremity to electric. Due to that a blood has small electric resistance, at the influx of blood (during a systole) resistance of tissues of extremity diminishes, and at the outflow (during a diastole) — is increased. Is characterizes a general (main and collateral) blood stream in extremity. A kind and form of reovasogramm depends on character and speed of pulse wave which changes depending on peripheral resistance and systole volume of blood. A normal reographic wave has a high steep anacrotic indent, segment which goes to down — dicrotic indent. *A reography index (RI)* — it is a basic index are relations of size of basic reographic wave (anacrotic indent) in mm to sizes of the calibrated impulse of apparatus. In a norm on the foot RI makes 0,9-1,0, on a shin — 1,5–1,6, on a thigh — 2,5–2,7.

Radioisotope method detection of speed of blood stream;

1) clirens method – investigation muscle blood stream of extremity by introduction to dorsal groop of shinmusclesisotope I ^{131}-gypurana (1-2 mkKu) and determination of time discharged of half dose of isotope (N – 11-16 mines, at pathology is lengthening of time);

2) research of speed of blood stream in extremity by introduction to the vein of albumen of serum, marked I^{-131}, and than determination of time of appearance of isotope on a foot (norm -20-30 c). Determine, also, time of satiation of feet by isotope (norm 3-5 min). At pathology is lengthening of time of the indicated indexes.

Capyllaroscopy — under a microscope the bed of nail of finger of foot is examined: in a norm is rose basis, capillaries in the form of womanish hairpin for hairs,

at pathology is pale background, deformation of capillaries, their coiled, precipice, they have the appearance of commas, points.

Thermometry (-graphy) — estimation of temperature of surface of extremity by special electrothermograph or registration of its infra-red radiation by heat vision set. In a norm - on distal sections of extremity (foot) temperature of 29-31 °C, at pathology — 22-24 °C. Control mast be done on symmetric areas.

Differential diagnostics. A differential diagnosis is performed with the diseases of veins (postthrombophlebitic syndrome and other), with neurological pathology (polyneuritis), with diabetic angiopaty, and also between the basic occlusion diseases - atherosclerosis, endarteritis and thrombangitis. In spite of community of many clinical symptoms, there are enough distinctive differential signs, including – angiography, knowing which a doctor can determined a correct clinical diagnosis.

Table 6

Differential signs of obliterated atherosclerosis and endarteritis arteries of lower extremities

№№ п/п	Signs	Atherosclerosis	Endarteritis
		Clinicalsigns	
1.	Beginning of disease	It is ordinary after 40 years	It is ordinary in 15-20 years
2.	Pain syndrome in the initial stages of disease	Symptom of the «intermittent claudication»	Symptom of the «intermittent claudication and often pain at peace
3.	Cooling, trauma, infectious diseases in anamnesis	Rarely	Often
4.	Concomitant diseases of vessels of heart and brain, hypertensive illness	Often	Rarely
5.	Diabetes mellitus	It is approximate at 20% of patients	It is usually absent
6.	Hypercholesterinemy	It is approximate at 20% of patients	Rarely
7.	Absence of pulse on a popliteal artery	It is absent often (at 80% of patients), the gangrene of foot is here observedrarely	It is absent rarer (at60% of patients), as a rule, is accompanied by a gangrene
8.	Absence of pulse on a femoral artery	It is absent at 15-20% of patients, the gangrene of foot is here observedrarely	It is absent at 5% of patients, a gangrene here is at most patients
9.	Character of affectationof extremities	Prevailing affectation of lower extremities, the one-sided is more frequent	Not uncommon affectation of lower and upper extremities, the bilateral is more frequent

№№ п/п	Signs	Atherosclerosis	Endarteritis
10	Occlusion type of affectation	Proximal, segmental, descending	Distal, diffuse, ascending
11.	Vascular noise above a femoral artery	Often	Rarely
12.	Acute thrombosis of the affected main artery	Often	Rarer
13.	Clinical feature	Relatively«benign», slow progressing of ischemic disorders, more later and rarer is development of gangrene	More «malignant», heavy pain syndrome, pain at peace can disturb already in a prodromal stage, rapidprogressing of ischemia, a gangrene develops quickly and often
Angiographysigns			
14.	Equal widespread narrowing of main artery	It is not	As a rule
15.	Unequal undermined internal contour of artery	Often	It is not
16.	Symptom of «amputation»	Often	It is not
17.	Segmental occlusion of big main arteries of pelvis and thigh	Often	It is usually absent
18.	Occlusion of arteries of shin and foot	It is extremely rare (it is ordinary at the aged people with diabetes mellitus)	It is determined as a rule
19.	Calcinosis walls of artery	Often	Rarely

***Formulation of clinical diagnosis*:**

At formulation of clinical diagnosis specify

1) basic diagnosis,

2) complication of basic disease,

3) concomitant pathology (if it present).

Example of formulation of clinical diagnosis:

1) *Basic* - Obliterated atherosclerosis of vessels of lower extremities, atherosclerotic occlusionof right superficial femoral artery, left popliteal artery, chronic ischemia of lower extremities of the

II B degree on the right, IIIA degrees on the left;

2) *complication* - the acute thrombosis of left popliteal artery, acute ischemia of the III degree;

3) *concomitan*t – IDH, coronar cardiosclerosis, hypertensive disease of the IIB stage.

Treatment of the patients with CODAE

The choice of medical tactic is determined by character of affectation (by etiology, morphological features), stage of disease, age and general condition of patient, presence of concomitant diseases.

The conservative methods of treatment are used at all forms of occlusion diseases in an initial stage — at the I-II stages of chronic ischemia, at the refuse of patient from operation, and also at the extremely grave general condition of patient.

Conservative therapy. It must be complex, directed on different parts of pathogenesis and removal of symptoms of disease. Basic its tasks: prophylaxis of progressing of basic disease; liquidation of influencing of unfavorable factors (risk factors - smoking, cooling, stresses and other.); liquidation of angiospasm; stimulation of development of collateral circulation of blood; normalization of neurotrophic and exchange processes in tissues of the affected extremity; improvement of microcirculation and reologic peculiarities of blood; normalization of disorders of the system of hemostasis; symptomatic treatment.

To the patients is recommended the feeding with low content of cholesterol.

For the prophylaxis of progressing of atherosclerotic process - administration of hypolipidemyc and antysclerotic preparations (lipocain, metyonin, lipostabil, linetol, myskleron, dyosponin, prodektin, ascorbic acid, iodine preparations) are necessary.

In the last period for a prophylaxis and treatment of atherosclerosis, including at the atherosclerotic affectation of arteries of extremities, is recommended administration *of statins (symvastatin, atorvastatin and other)*, which have expressed antiatherogenic characteristics – suppress synthesis of cholesterol, lowered level of lipids, diminished systemic inflammation, improved the function of endothelium of vessels, have antythrombotic action. All of it predetermines the decline of level of cholesterol, stabilizes an atherosclerotic plaques, intensive diminishes systemic and local inflammation of wall of vessels.

Liquidation of angiospasm and stimulation of development of collateral circulation of blood in ischemic extremity achieved by medicinal, physical therapy and balneological facilities:

1) application of novocaine blockades (paranephral, sympatic, extradural irrigational (introduction 2-3 times per days during 2-3 weeks through the catheter of mixture with of a 25 ml 0,25% solution of novocaine, 0,3 % solution of dycaine 2 ml., vitamin B_1- 1ml, 2-3 ml of 96% of alcogol), which blocking pathological impulses and has influence on trophic function of nerves system and capillary blood circulation;

2) introduction of solution of novocaine intravenously droply (of20-30 ml 0,5% solution)and endarterially;

3) introduction of vasodilatators of 3th groups:a) myotrop actions (no-shpa, papaverin, nicoshpan, nicoverin,galidor and other), b) operating in area of the peripheral cholinreactive systems - through the vegetative nervous system (bupatol, mydocalm, andecalyn, calicrein-depo, delminal, dyprofen, spasmolytin, nicotine acid and other). c) ganglioblockers actions (blocking N-cholinreactive systems of vegetative knots) - benzohexoniy, pentamin, dimecolin and other; it is necessary to remember that at the initial stages of disease all 3 groups of spasmolytics are effective, and in the IV stage - only 1th groups, because preparations 2^{th} and 3^{th} groups

strengthen the atony of capillaries, multiplying violation of circulation of blood in the affected extremity.

Normalization of neurotrophyc and exchange processes in tissues of staggered extremity– application of complex of vitamins (B_1, B_6, B_{15}, E, PP);

Preparations as *solcoseril and actovegin* — activating processes of oxidation in tissues, assist to renewal of reparations processes in tissues, influence on the exchange-trophic function of tissues even in the conditions of the loosened blood stream (inserted endarterially on 8 ml, intravenously — 6-20 ml on 250 ml normal saline or solution of glucose, intramuscular for 4 ml on the course of treatment in an amount 20-25 introductions).

Improvement *of microcirculation and reology properties of blood* by application *of hemocorrectors* - preparations of lower molecular dextran (reopolyglucin, reomacrodex, jelatinol, reogluman) and derivative polyvinilpirolidone (hemodes), which decrease viscosity of blood, due to hemodilution, cellular aggregation, hinder of the intravascular thrombosis (forming the negative charged monomolecular layer, which repulse the negatively charged elements of blood, thrombin, fibrin), increase VCB (volume of circulated blood), promoting colloid-osmotic pressure and assisting to transition of interstitial liquid in a vascular bed).

Normalization of hemocoagulation (at its increase*) is performed* by application of anticoagulants of direct (heparins) and indirect (pelentan, fenilin, sincumar, varfarin and other) action, and also, desagregants (acetyl salicylic acid, trental, sermion, dipirydamol).

It is necessary to mark efficiency of protracted endarterial infusions by multicomponent infusion mixtures which include above mentioned medicinal preparations, by cateterisation of femoral artery or its branches (a. epigastrica superior and other), by the method of regional perfusion.

Physiotherapy treatment — the Bernara current, UHF, electrophoresis with novocaine and spasmolitics, and also barotherapy in the Kravchenko chamber and electro-impulsive barotherapy in the Shmydt chamber.

Symptomatic treatment is directed on the removal of pain syndrome, inflammatory phenomena, straggle against an infection, stimulation of healing process of trophic ulcers, etc.

Operative treatment

Reconstructive and physiological directions — two major ways of development of modern vascular surgery.

Becoming and development of vascular surgery became possible due to introduction and wide distribution **of angiographic methods of research.**

Developed the special vascular tools and operative accesses to the basic main arteries and vascular stitches.

Since the II degree of ischemia with appearance at the pain symptom of the «intermittent claudication» it is necessary to execute angiography research for the decision of question about possibility and character of operative interference:

1) at obliterated atherosclerosis and proximal character of affectation of main arteries possible implementation of reconstructive operations:

a) at the segmental affectation – one from different kinds of endarterectomy or prosthetics of the affected area,

b) at more widespread affectation - operation of the bypass shunting;

2) at obliterated endarteritis with widespread impair of distal parts of main arterial bed — implementation of «symptomatic» operations is indicated — abdominal or pectoral sympathectomy, epynephrectomy, peryarterial sympathectomy (the Lerish operation);

3) at atherosclerosis and at endarteritis at the IV stage of ischemia, at presence of destructive changes, operations of necrectomy are executed, amputations of extremities at different level, exarticulation.

There are 4 types of operative interferences at CODAE on this time:

I — reconstructive (restoration) operations,

II — operations on the sympathetic nervous system,

III — operations on endocrine organs,

IV — necrectomy, amputations, exarticulation.

Reconstructive operations. Indications to this type of operation are the magisterial arteries with segmental occlusion on level of iliac-femoral-popliteal segments with saving of blood stream higher and below than place of occlusion, that, mainly, this are patients with obliterated atherosclerosis. Restoration of ability for passage of occlusion artery in most cases can be attained by the following three types of reconstructive operative interferences:

- **by endarterectomy** (or intimthrombectomy), at which recanalization of artery is arrived after deleting of changed intimae (with atherosclerotic plaque) and obturated blood clots (at their presence);
- **by the bypass shunting** of occlusion segment of artery by synthetic prosthetic or autovein;
- **by prosthetic** of artery by the resection of the affected segment with subsequent replacement of resected area by synthetic prosthetic or autovein (autoartery).

Indication to endarterectomy is local (segmental) occlusion of main arteries in aorto-iliac, femoral-popliteal, aorto-brachiocephal and subclavia-axillar segments mainly at the atherosclerotic affectation. Terms for its implementation is good ability for passage of arterial blood distally than place of occlusion and complete ability for passage of blood proximally from planned desobliteration.

The following types (types) of endarterectomy are executed:

1. The «*opened*» method consists of wide longitudinal section of artery above the affected area, deleting of blood clot together with changed intimae and stitching incision of artery, as a rule, with thepatch from autovein (segment of large hypodermic vein), for the prophylaxis of stenosis (Fig. 42).

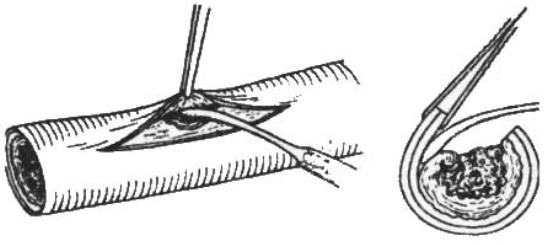


Fig. 42. Opened endarterectomy

2. «*Half-closed*» method — an artery above an occlusion area is dissected not on all length, and only in a few places as the small openings, out of which makes deleting of intimae and blood clots by the special instruments. These openings are stitched also by «patches» from autovein (Fig. 43).

3. *Eversional endarterectomy* for a desobliteration of aorta-iliac segment at the Lerysh syndrome, got yet the name *of endarterectomy by method «turns» inside out*, at which makes laparotomy, the affected segments of aorta and iliac arteries are picking out , cross, adventitia these vessels release from changed intimae and blood clots by the turn inside out; an external membrane liberated from the pathologically changed tissues inturn, forming thin, but enough strong auto prosthesis, which is restore into former place (Fig. 44).

Advantages of endarterectomy are physiology (restoration of blood stream through the anatomic normally located artery), deleting of mouths of collaterals, absence of foreign bodies. Failings - operation is long, possible implementation only at atherosclerosis and on bid arteries.

Operation **of the bypass shunting** is indicated at widespread (on the considerable extent) occlusion of arterial bed of extremity at atherosclerosis (Fig. 45, 46).

3 types of shunts are thus used:

1) vascular polymeric prosthetic;

2) autovein,

3) autoartery.

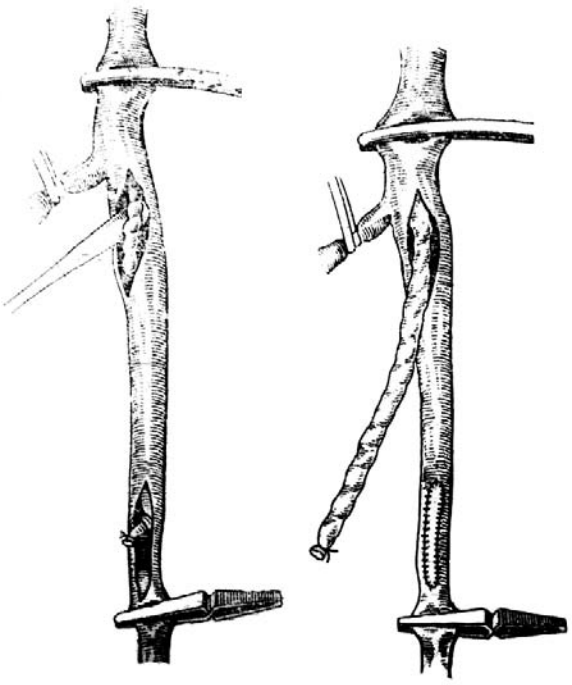


Fig. 43. Half-closed endarterectomy

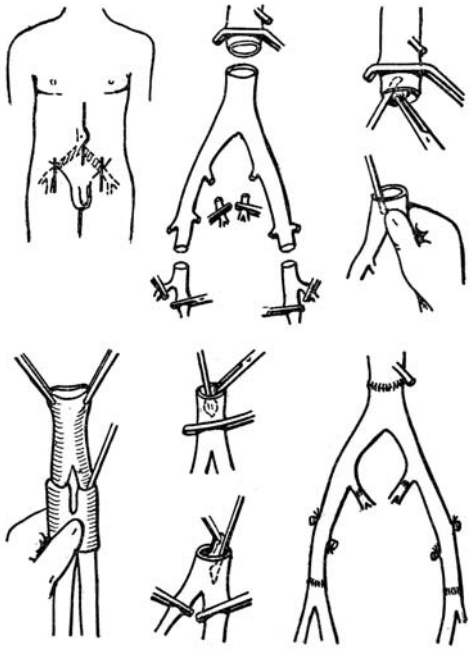


Fig. 44. Eversional endarterectomy for a desobliteration of aorta-iliac segment at the Lerysh syndrome

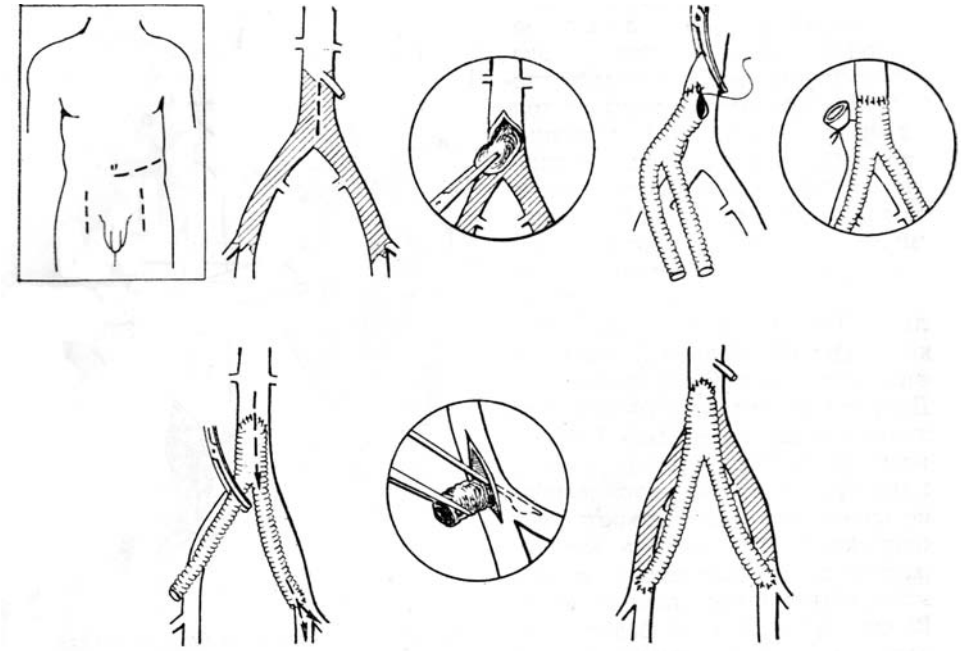


Fig. 48, 49. Bypass shunting

The widest distribution was got by vascular alloprosthetics (from polymeric materials) and auto venous shunts.

Synthetic vascular prosthetic are made from the 3th groups of synthetic materials:

a) polyamide fibers (nylon, capron) – they may be the cause expressed reaction of surrounding tissues, may loss with time durability, inclined to the thrombosis;

b) polyester (lavsan, terilen, dacron) — with the best qualities; in) polytetrafluorethylen (teflon, ftorlon and other.) are optimum. On construction they are tubular and bifurcation.

To prosthetic the hard requirements are produced: they must be indifferent (that, not to be the cause the reaction of surrounding tissues), strong, elastic, to have sufficient biological permeabilityto provide the process «growing» in, «implantations» of prosthetic at which is formed new intimae of prosthetic (due to vessels, germinate in pores), and, at the same time, not to be result in the expressed bleeding from prosthetic with formation of haematoma around prosthetic, that threatens by infection or subsequent scar deformation of prosthetic. Unfortunately, a common complication is thrombosis of prosthetic lumen.

For prevention of the indicated complications, new modifications of vascular prosthetic were offered: semibiological prosthetic saturated with a collagen-heparin complex (prevent a thrombosis and formation of haematoma); electricconducted vascular prosthetic with silver framework whichcreates a negative electric charge on a surface, diminishing possibility of thrombformation;synthetic selffixed endoprosthesis (priority of invention and introduction in the clinic of which belongs to the research workers of Institute of general and urgent surgery of Kharkov).

Synthetic prosthetic are used for the plastic arteries of big sizes- aorto- iliac segment and branches of arch of aorta.

For shunting of femoral-popliteal segment autovein is used - large hypodermic vein. Biological affinity of autovein, simplicity of its withdrawal, presence of unchanged intimae and elasticity does autovenous plastic arts as optimum type of operation.

For shunting can be used also autoartery such as deep artery of thigh, internal iliac artery and other.

Advantages of operation - less traumatic, than endarterectomy, collaterals is not damaged, lymphatic, vein and nervous formation, anastomose on a type «end of shunt in the side of artery» can be applied enough wide.Failings - possible development in the postoperative period of thromboses of shunts.

Prosthesing operations - *resection of segment of artery with substitution of it by alloprosthetic or autovein (depending on a caliber of vessels) is used rarely in connection with trauma (Fig. 46).*

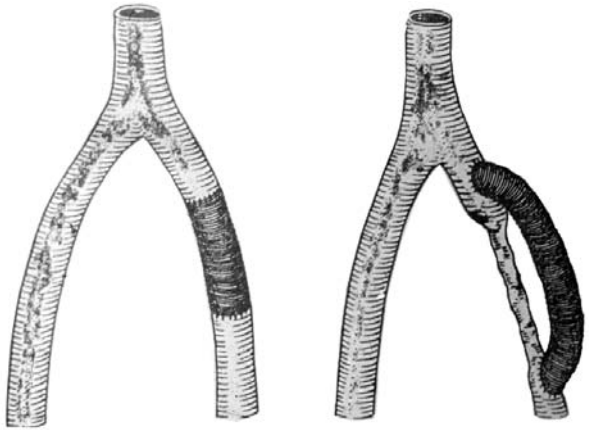


Fig. 46. Prosthesing operation and bypass shunting

Except for restorative reconstructive operations on vessels at CODAE used and ***operationson the nervous and endocrine systems*****,** which are directed on diminishment of spasm of arterial bed and improvement of collateral circulation of blood. A indication to them for independent application is absence of possibilities for implementation of reconstructive operation (mainly — at endarteritis), and also — as additional operative interference at reconstructive operations, which improves effectiveness of the last. It is operation of Diez — lumbar sympathectomy on the side of affectation (deleting of sympathy lumbar knots L2-L4 by extra peritoneal access on Lerysh),the B.V. Ognev's operation – pectoral left-side sympathectomy (deleting of pectoral sympathy ganglion D3), peryarterial sympathectomyis the Lerish operation, operation of V.A.Oppel – epynephrectomy (resection or deleting of adrenal gland).

At the existent variety of methods of operative treatment of CODAE choice of type of operation for every patient must be individual, although it is here needed to follow basic principles: at atherosclerosis endarterectomy and bypass shunting is basic operations, which can be accompanying by sympathectomy and epynephrectomy; at end-

arteritis are operations on the sympathy and endocrine systems (Diez, Ognev, Oppel); at thrombangitis are those interferences, that at endarteritis adding by venectomy.

At all forms of occlusive diseases of arteries of extremities in case of the IV stage of chronic ischemia with development of irreversible destructive changes in tissues of distal sections of extremities are necessary to execute necrectomy, amputations, exarticulation.

It is necessary to mark that the CODAE treatment can not be limited by surgical restoration of main blood stream, and certainly must include the complex of the conservative measures directed on all links of pathogenesis of disease, that, operative treatment must be examined only as stage in treatment of patient.

New in the CODAE treatment. Presently in conservative therapy widely spread lowmolecular heparins (fraxiparin, clecsan), desagregants - ticlid (ticlopedin), plavix (clopidogrel).

In last years new effective preparations for the CODAE treatment appeared. This ***enelbin 100 retard*** (selective blocker of serotonin 5-HT2 receptors), which has spasmolitics, desagregants,antyserotonin actions, improves microcirculation and reologic properties of blood also, stimulates intracellular metabolism, increases the ATF level, that improves utilization of glucose, reduces the products of milk acid in cells at an ischemia – the same improves blood circulation of peripheral tissues and their supply by oxygen and nutritives, results of diminishment of intensity of ischemic pain in extremity, multiplies distance ofwalking.

Especially effective is administration ***of preparations of prostaglandin E1*** – vasoprostan and alprostan, having expressed spasmolitics, antiaggregates, angioprotectors and antiatherogenic effects: a blood streamis strengthened by direct expansion of vessels, hemostasis is stabilized due to activating of fibrinolysis, aggregation of thrombocytes and activating of neutrophiles, microcirculation and reology of blood is improved, normalizations of metabolism in ischemic tissues due to the improvement of utilization of oxygen and glucose, antisclerotic actiondue to oppression of mitotic activity and surplus proliferation of cells of wall of vessel, and also reducing the synthesis of cholesterol and its deposit in the wall of vessels.

Dosage: vasaprostan - intravenously droply for 40-60 mcg (2-3 ampoules), dilute in 50-200 ml normal saline in a day (for 3-4 hours) during 2-6 weeks (depending from intensivity of ischemia), at endarterial introduction - 20 mcg 1 times per days during 15-20 days;alprostan - from 50 to 200 mcg (0,5 - 2 ampoules) 1 times per days or for 50-100 mcg 2 times per days in a 100-500 ml normal saline solution or 5 % glucoses (for 2-6 hours), course of treatment 2-4 weeks.

In 2001 year proposed the method of volume pnevmopressing (pnevmovacuum-compression), carried out by the complex «Bioregulator», a medical effect achieved due to the mechanical compression of tissues, tensions of skin cover, change of reologic properties of blood and acceleration of microcirculatory processes. Physiological action of pneumopressing is the multisides, conditioned by neuro-reflectory reactions, directly by mechanical influence on tissues and organs, and also changes in an organism at humoral level: microcirculation in tissues and organs is actively stimulated, trophic of tissues gets better.

In a whole world greater distribution is got **the miniinvasive methods of operative interferences,** including — in vascular surgery.Arose up and successfully develops a new region in medicine is intervention **radiology.**

Methods, directed on restoration of ability for passage of main arteries - extremities, coronar, kidney, brachiocephal at the atherosclerotic affectation (stenosis, occlusion), named as **«transcutaneus transluminal angioplasty».** For their implementation different modern technologies are used.

It is the following miniinvasive interferences: method of roentgenendovascular balloon dilatation of arteries by the catheter Gruntzig; the method of mechanical recanalization with the help mechanical catheter Kenzy; laser angioplasty (or thermal recanalization), sometimes in combination with mechanical; deleting (cutting away) of atherosclerotic plaque and its deleting by the catheter of Sympson; ultrasonic angioplasty; transcutaneus endarterectomy.These methods are performing under the roentgen TV and angioscopic control.

For the prophylaxis of development of restenosis in last period above mentioned operations ending by **stenting of vessel** - implantation in the reanalyzed area of endoprosthesis (on construction it is more frequent than all the reticulated wattled cylinder from a thin metallic wire) (Fig. 47, 48).

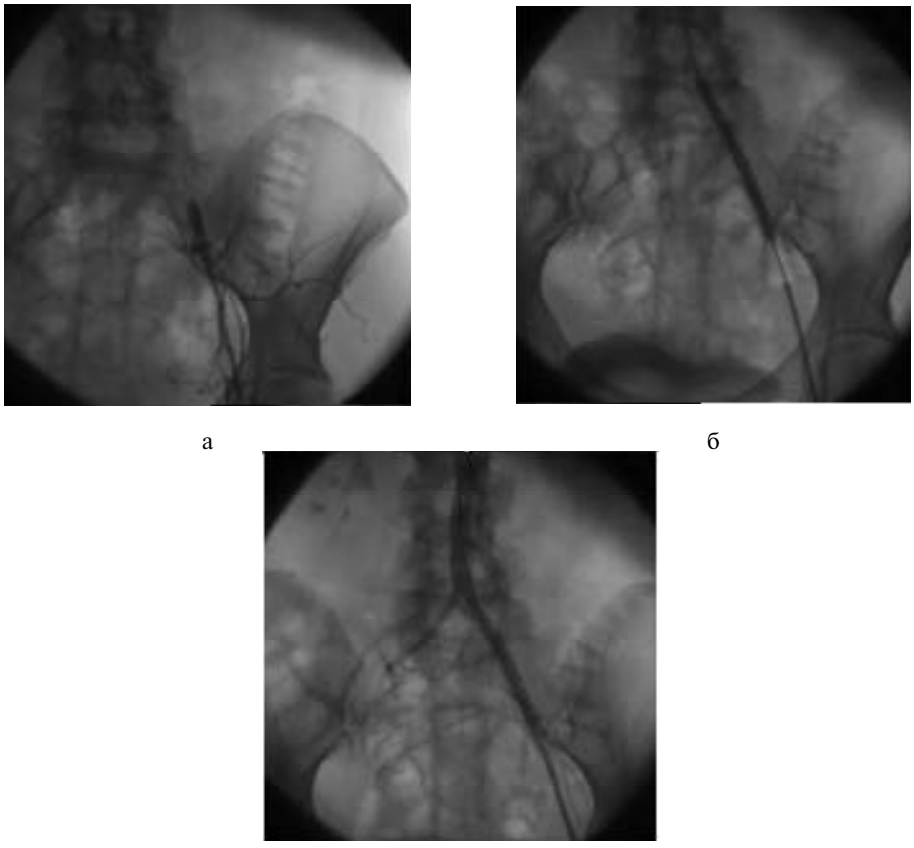


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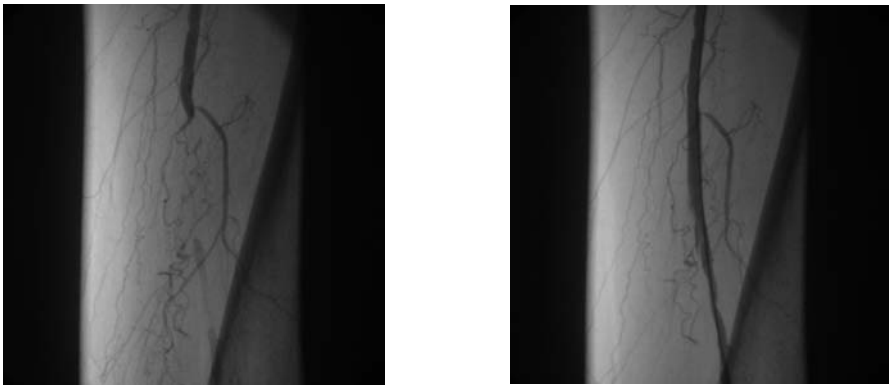
Fig. 47. Transluminal balloon angioplasty with the stenting of common iliac artery, a - critical stenosis of the left common iliac artery, b - its endovascular balloon dilatation, c - artery stenting



a b

Fig. 48. Transluminal balloon angioplasty with the stenting of the superficial femoral artery, a - atherosclerotic occlusion of the right superficial femoral artery (a symptom of "amputation stump"); b - the same artery after angioplasty

Literature: 1. Jay D. Cofman, Robert T. Eberhardt. Peripheral arterial disease: diagnosis and treatment.- Human Press, 2003, 356 p. 2. Jonatan B. Towne, Larry H. Hollier. Complications in vascular surgery.- Informa Health Care, 2002, 628 p. 3. Ivan P. Casserly, Ravish Sachar, Jay S. Yadav. Manual of peripheral vascular intervention.-Lippencott Williams & Wilkins, 2005, 374 p. 4. Sanjay Rajagopalan, Debabrata Mukherjee, Emile R. Mohler. Manual of vascular diseases.-Lippincott Williams& Wilkins, 2004, 437 p. 5. Robert W. Hobson, Samuel E Wilson, Frank J. Veith. Vascular surgery: principles and clinical practice.- Informa Health Care, 2003, 1292 p.

V.2. Arterial thrombosis and embolism of the extremities

The statistical information of WOH 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* – so-called «*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 (Botalo'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. 49). 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.

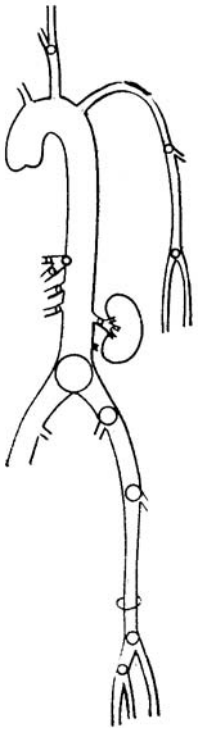


Fig. 49. "Surgical floors" of arterial embolisms

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 with block of it, arises reflectory spasm of the periph-

eral 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 by appearance 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 skin on 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, roentgenologic, 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 succusia 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 entrancein 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 of gangrene 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.

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. 50), 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. 51).

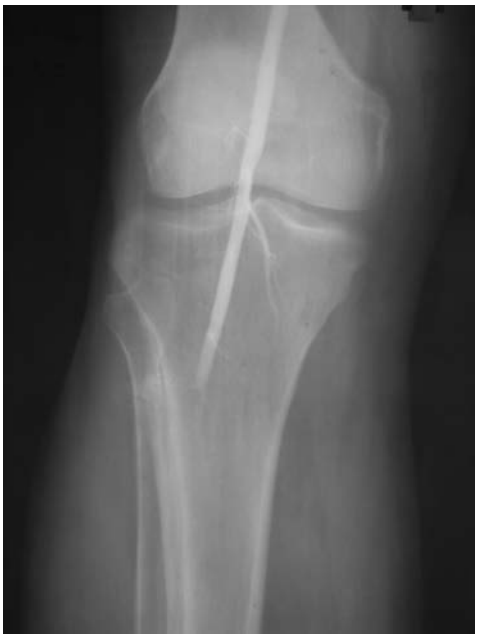


Fig. 50. Popliteal artery embolism in its bifurcation

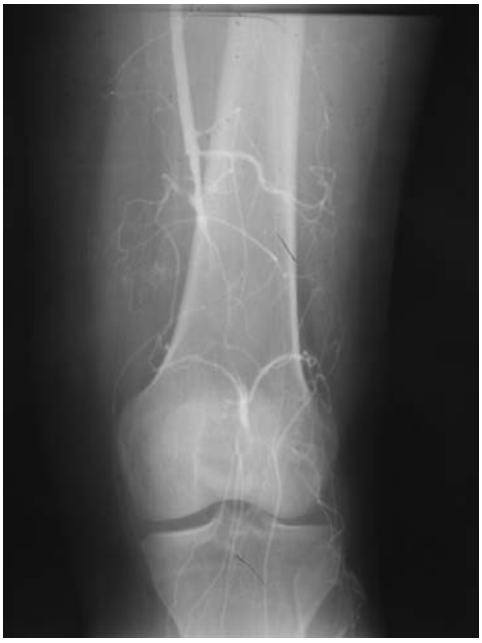


Fig. 51. Acute thrombosis of the superficial femoral artery in the lower third in its atherosclerotic lesions

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 7

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 medicin directed *on warning of making progress of acute ischemia* and extension the same of viability of tissues of extremity. *For the removal of pain* is intravenous 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 contra-indications 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:

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. 52);

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. 53).

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. 54).

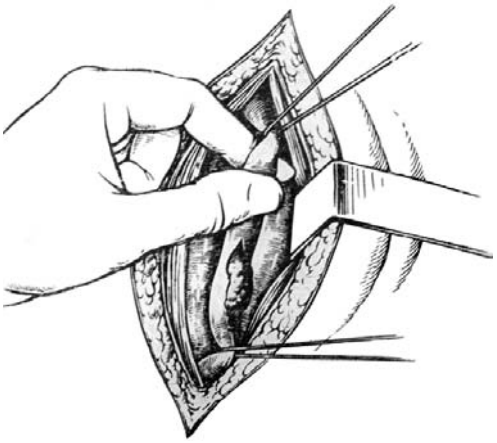


Fig. 52. Direct embolectomy

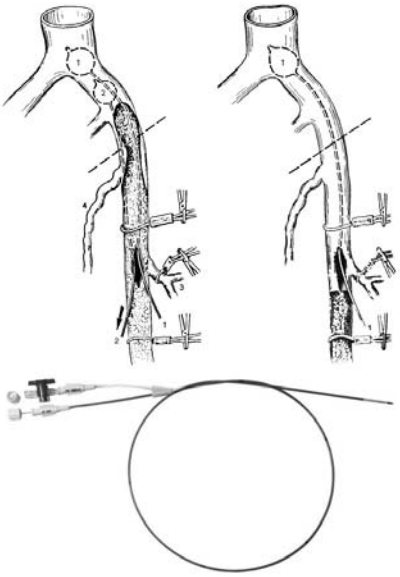


Fig. 53. Indirect embolectomy by catheter of Fogartyi

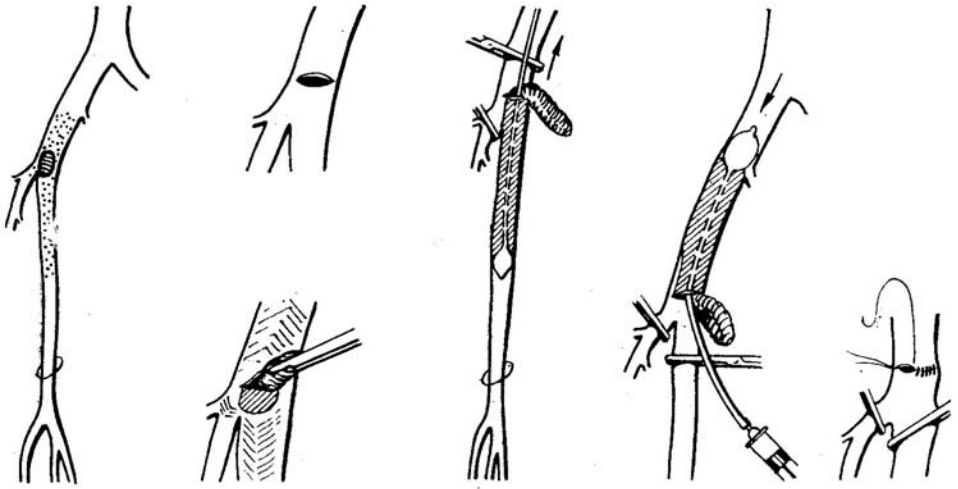


Fig. 54. Scheme stages of operations embolectomy, a, b - direct embolectomy from the common femoral artery; c- indirect embolectomy of the external iliac artery; d – artery suture

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 it radical correction for the prophylaxis of the possible relapse of AE.

It is necessary to mark, that in this region of urgent vascular surgery became uses small invasive operations are inculcated is implementation *of trascutaneous aspirating embolectomy* (Fig. 55).

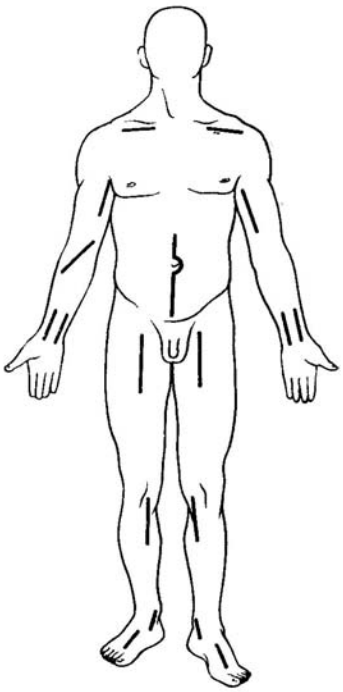


Fig. 55. Typical surgical approaches during embolectomy operations in arterial embolism of the upper and lower extremities

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 than continuous intravenous infusion a medical dose of preparation, which can achieve a 1 500 000–3 000 000 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-amyno-capronic acids, 1% of solution of chloride calcium, native or 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, pipolfene and 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 XX ages of 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 OPD therapeutics'and vascular surgeon. Very necessarily is to control the indexes of coagulogramm. If it necessary a surgeon appoints ULTRASONIC of main arteries of extremities.

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V.3. Abdominal ischemic syndrome

Rate of development of abdominal ischemic syndrome (AIS) is high enough: in 75,5% at autopsy of deathes due to ischemic heart trouble, atherosclerosis of cerebral arteries or vessels of lower extremities, atherosclerosis of abdominal aorta and its visceral branches is also found out; in 50-57% of cases abdominal ischemia is diagnosed only in development of acute disturbance of mesenterial blood flow – intestinal infarction. All of other cases, as a rule, are misdiagnosed with the ordinary diseases – gastroduodenitis, hepatitis, pancreatitis and others. Early diagnostics and treatment of abdominal atherosclerosis in gastroenterology deparmtnets are not conducted.

Rate of stenotic lesions in visceral branches of abdominal aorta according to post mortem data are 19,2-70%, angiographies – 4,1-53,5%. At the same time operative interventions in this pathology make only 2% from the number of all of operations on abdominal portion of aorta and its branches.

Anatomy of vessels, supplying gastrointestinal tract

Arteries supplying gastrointestinal tract goes out from aorta (three main branches): celiac trunk, superior and inferior mesenteric arteries (Fig. 56-59).

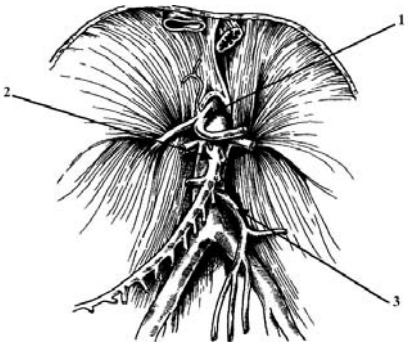


Fig. 56. Main branches of abdominal aorta: 1 - celiac trunk; 2 – upper mesenteric artery; 3 – lower mesenteric artery

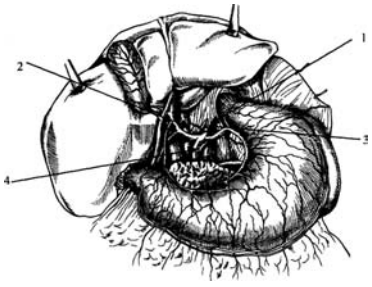


Fig. 57. Celiac trunk and its branches. 1 - celiac trunk; 2 - common hepatic artery; 3 - the left gastric artery; 4 - splenic artery.

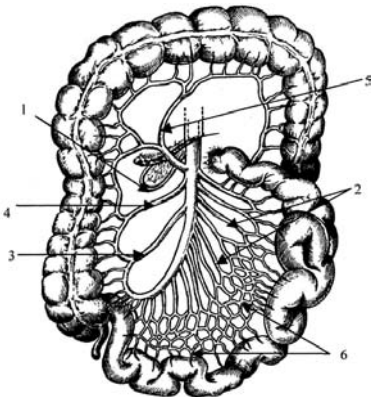


Fig. 58. The superior mesenteric artery: 1- lower pancreatic-duodenum atery. 2 - branches to the iliac and caecum intestines; 3 - colon iliac artery; 4 - right colon artery; 5 - middle colon artery

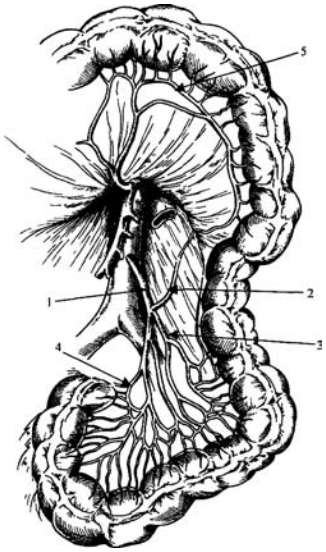


Fig. 59. Lower mesenteric artery: 1 - lower mesenteric artery; 2 - left colon artery; 3 - sigmoid artery; 4 - upper rectal artery; 5 - arc Riolan

The chronic ischemia of organs of digestion is conditioned by occlusion of visceral branches of abdominal aorta and develops due to the deficit of blood flow in some part department of gastrointestinal tract on the different stages of digestion. It begins usually with functional disorders and results in organic changes. First report about the infarction of intestine which was preceded by chronic bowel ischemia, was done by Despre in 1834. Term "angina abdominalis" was firstly used in clinical practice by G. Bacelli (1905). The first successful operation on upper mesenteric artery (trombendarterectomy) was performed in 1957 by R. Shaw and coauth. Underlying cause of abdominal ischemia is atherosclerosis.

Causes of disturbance of visceral blood circulation can be various. Character and degree of them depend on aetiology of lesion of visceral branches of aorta. A few classifications of causes of chronic ischemia of visceral organs were offered.

Y. L. Shalkov (1971) offered classification in which selected 3 groups of etiologic factors:

1. Mechanical:
   a) narnumbering of arteries due to obliterative atherosclerosis, arteriitis, partial atherothrombosis;
   b) extravasal compression of arteries by scars, tumour;
   c) disturbance of blood flow in vessels with aneurysm;
   d) disturbance of blood flow due to congenital defects of visceral arteries.
2. Functional:
   a) due to the angiospasm;
   b) due to low blood pressure.
3. Other:
   a) polycytemia;
   b) other diseases of blood, in which conditions are created for development of disturbance of visceral circulation of blood.

Classification of abdominal ischemic syndrome on causal factors (V. I. Burakovskiy and coauth., 1989):

I. Functional disturbances of visceral branches of abdominal aorta: arteriospasm, hypotension of central origin, hypoglycemia, medical diseases, polycytemia

II. Organic changes of visceral branches of abdominal aorta:

1) extraarterial compression factors:

a) congenital anomalies of location and position of arteries, falciform ligament, medial crura of diaphragm, elements of celiac plexus;

b) acquired: tumours, aneurysm of abdominal aorta, periarterial and retroperitoneal fibrosis.

2) anomalies of development of visceral branches: aplasia, hypoplasia of arteries or aorta in intervisceral segment, fibromuscular dysplasia, congenital arteriovenous fistula and hemangiomas.

3) disease of visceral branches: atherosclerosis, arteriits, dissecting aneurysm of descending part of aorta, traumatic arteriovenous fistula and aneurysm.More frequent celiac trunk and mesenteric arteries are involved.

Classification of abdominal ischemic syndrome on the stages of disease (V. I. Burakovskiy and coauth., 1989):

I. Stage of compensation:

a) asymptomatic flow;

b) the stage of microsymptoms - ischemia as a result of functional overload of organs of digestion.

II. The stage of subcompensation - ischemia as a result of the functional loading on the organs of digestion.

III. The stage of decompensation - ischemia in functional rest of organs of digestion.

IV. The stage of ulcerative and necrotic changes in the organs of digestion: special group of gastric and duodenal ulcers, enteritis, colitis, post-infarction strictures of small and large bowel.

Examination of patient with suspicion on AIS consists of collection of complaints, anamnesis, inspection, palpation, percussion and auscultation of abdomen.

***It is necessary to define during questioning of patient*:**

1. Complaints: pain after meals; nausea, possible heartburn, belch, feeling of repletion of abdomen, vomiting, often preceded by pain. Patients complaints on abdominal distension, constipation, replaced by diarrhoea sometimes.

2. Anamnesis of disease:

– time of onset and first symptoms of disease;
– sequence of development of process;
– presence of pain syndrome, character of pain;
– preceding treatment and its result;
– operations.

3. Anamnesis of life:previous and concomitant diseases of organs of abdominal cavity. Social description and professional factors:

– married or single or divorced;
– presence of stress situations;
– professional harmfulness;

– disease of father and mother: endocrine, exchange, oncologic.Clinical presentation of patients with an abdominal ischemia differs by large variety and not always has the expressed specificity. All of symptoms it is possible to divide into four groups of symptoms:
  • Pain abdominal syndrome.
  • Dysfunction of gastrointestinal tract.
  • Progressing decline of weight.
  • Depressed astheno-ippochondric syndrome.

A abdomen-ache is the basic complaint of patients with a chronic abdominal ischemia. Pain can be spastic (31,3%), aching deeply inwardly or with an irradiation into the back (27,6%), or dull (41,2%). Begins through 15-30 min after-meal and proceeds 1-3 hours mainly depending on an amount, and also composition of the eaten meal. Location of pain is various. The more than half (52,7%) of patients marks the strictly certain area of tenderness, located in one anatomic area (Fig. 60). Other part has pain which spreads on a few anatomic areas. More frequent than all it the pain feelings in epigastric area with an irradiation into the back, or pain which spreads on all anterior abdominal wall.

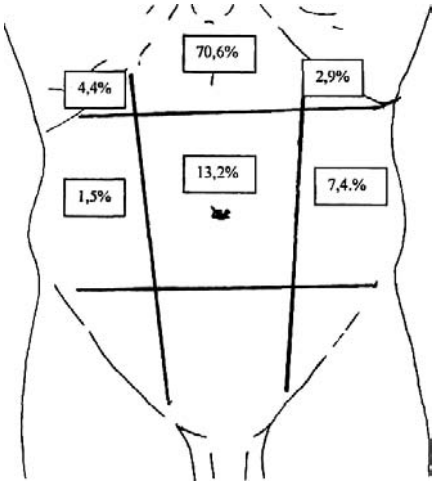


Fig. 60. Rate of location of pain in anatomic areas

For determination of connection of area of tenderness with location of lesion of visceral branches it is possible to select next features. In the case of stenosis of celiac trunk pain is more frequently located in epigastric area. Narnumbering of upper mesenteric artery causes pain mainly in umbilical and right iliac areas. The lesion of inferior mesenteric artery causes pain in the left departments of abdomen. Certainly, the different concomitant diseases of organs of abdominal cavity can change the location of areas of pain in patients with a chronic abdominal ischemia.

The number of factors influences the decrease of pain or its disappearance. Among them: reception of spasmolytics (53,9%), knee-elbow position (28,7%), local application of heat (13,3%). In severe cases (4,2%) it is necessary to prescribe analgetics. Knee-elbow position is more typical for «extravasal» compressions.

In the initial stages of disease the use of light meal can be not accompanied by pain reaction, while reception of meat, milk and other meal, which is hardly digested, causes a very intensive pain syndrome which causes disgust for patients of these products.

Taking to account that it is very rare pathology, doctors have the first idea about possibility of malignant process in an abdominal cavity. It is possible suspicion on gastritis, peptic ulcer, chronic calculous cholecystitis.

At careful collection of anamnesis some patients mark appearance of abdomen-aches at the intensive physical loadings (at run, weight lifting, physical work et cetera), breath-holding during abdomen-aches, appearance of pain in a night-time in horizontal position with certain periodicity. In the special cases there is an irradiation of pain in the left shoulder and breastbone.

As far as disease progresses patient begins to lose weight which convinces a doctor in a presence of malignant tumor. Intensity of pain attacks is such, that they are ready fully to give up a meal. In an interpain period patients mark feeling of discomfort in bdomen. The objective methods of investigation of organs of abdominal cavity find no pathology, adequate to pain syndrome.

Mean time from the moment of appearance of the first symptoms to establishment of correct diagnosis makes 7,5+0,4 years.

Dysfunction of gastrointestinal tract in patients with chronic abdominal ischemia presents with different displays and degree of severity. Most frequent are fullness, distension in abdomen, disturbance of stool.

As a rule, dyspepsia symptoms coincide with activation of digestion, and their severity depends on an amount and composition of the meal.

Seasonal character of dysfunction of intestine is determined in fourth part (26,1%) of patients. The phenomena dyspepsia increases in cold and becomes less expressed in warm time of year.

Table 8

Description of dyspepsia symptoms in patients from AIS

Symptoms	Rate (%)
Fullness in abdomen	39,8
Feeling of distension in abdomen	36,8
Nausea	25,7
Belch	21,1
Heartburn	11,1
Vomit	11,7
Constipation	36,8
Diarrhoea	14,6
Duty of закрепу and liquid chair	5,3

The progressive loss of weight is observed in 88,0% patients with an abdominal bowel ischemia. It is caused, from one side, by the waiver of reception of meal in connection with a pain syndrome, and from other, by the decline of basic function of intestine in connection with the ischemia of its wall. The presence of pain abdominal syndrome makes patients to take meals in a less volume, less high-calorie, easily mastered, more monotonous. Forced limitation of amount of meal, which is taken, causes a necessity to carry out its more frequent reception, so-called fractional feed.

Before entering hospital the loss of weight in patients is (7,9+0,5) kg. Thus it is in direct dependence on duration of disease, severity of pain syndrome and dyspepsia.

The features of clinical presentation of patients with a chronic abdominal ischemia allow to select a few clinical forms of this disease.

1. Pain form (52,6%) — more frequently occurred in stenosis of celiac trunk.
2. Small intestine enteropathy (5,8%), in which prevail disturbance of secretory and absorption function of intestine; proves instability of stool, flatulence, feeling of overeating, belch, heartburn, vomiting; observed in disturbance of superior mesenteric blood circulation.
3. Large intestine colonopathy (13,5%), that presents with disturbances of motor function of colon as constipation, can result in cicatrical stenosis of the left departments of colon as a result of destructive forms of ischemic colitis. Occurs in stenosis of inferior mesenteric artery.
4. Mixed form (23,1%) is possible to add to any combinations of afore-mentioned forms and concomitant diseases of organs of digestion.

AIS has a few stages of development.

In the 1th stage, relative compensation, the lesion of visceral branches are diagnosed during an inspection of patients with other arterial pathology (occlusive diseases of aorto-iliac area, renal arteries, aneurysm of aorta). In these patients it is possible to reveal the dyspepsia phenomena. These patients present the group of high risk of development of acute ischemic colitis after the reconstruction of aorto-iliac segment.

Development of occlusive lesion of visceral branches of abdominal aorta, in absence of collateral circulation of blood, results in the 2th stage, so-called subcompensation. It is characterized by appearance of pain after-meal and the severe dyspepsia phenomena which can be corrected by diet and conservative therapy.

Subsequent progress of occlusive process for these patients results in development of the 3th stage - decompensation of visceral circulation of blood. Clinically it is expressed by permanent pain which increase after-meal, progressive weight loss, oppressing of psychic status.

Clinical physical examination

According to morphometric signs (height, weight, structure of skeleton) somatotype of patient is determined.

Skin is inspected, here attention is paid to elasticity, colour, pigmentation. Character of hair covers is evaluated, presence of hair on feet, middle line of abdomen, breast.

Examination and palpation of thyroid: its size and presence of compressions are determined.

Examine supraclavicular, subclavicular and axillar areas to reveal the enlarged lymphatic nodes.

However, except anamnesis of disease and recurrent abdominal pains after-meal, there are not specific symptoms of chronic bowel ischemia.

On palpation of abdomen tenderness can be revealed in the half (55,9%) of patients. Usually their location coincide with the areas of spontaneous or after-meal pain. On results palpation it is possible to suppose involved artery. Reacting of patient on palpation of upper and right departments of abdomen allows to suppose

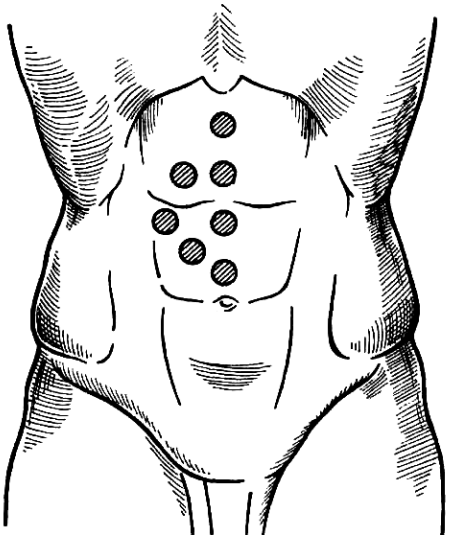


Fig. 61. Location of areas of auscultation of visceral arteries for patients with AIS

the lesion of celiac trunk or superior mesenteric artery. Tenderness on palpation of the left and inferior departments of abdomen specifies on possibility of lesion of inferior mesenteric artery. However these information is very relative.

Auscultation allows to define systolic murmur less than in the half of patients (47,3%). absence of systolic noise in the projection of visceral arteries does not exclude their lesion. It is better to conduct auscultation in areas, located on a midline upper than umbilicus and on 2-4 cm more right (Fig. 61). The most informing examination is performed in supine position of patient before meals and on breath out. It is necessary to distinguish noise of systole of visceral arteries from cardiac and aortic. Cardiac noises are conducted on all of main arteries. For control it is possible to conduct auscultation of carotid or subclavicular arteries. In stenosis of abdominal department of aorta noise is conducted on femoral arteries.

Leading clinical symptom – pain.

On the basis of findings of questioning and clinical physical examination of patient a preliminary clinical diagnosis is put.

Laboratory and instrumental diagnostics

For today does not exist clear clinical and laboratory parameters which would characterize the flow of AIS. More often changes in laboratory tests characterize complications of basic disease or concomitant pathology.

Instrumental methods of diagnostics of AIS.

They can be divided into 4 groups:

1) investigation of functions of absorption and excretion of intestine;

2) uninvasive methods;

3) contrast angiography;

4) intraoperative diagnostics.

Investigation of functions of absorption and excretion of intestine in diagnostics of chronic abdominal ischemia is not basic.

Uninvasive methods of investigation:

Functional test with the meals. Provocation of abdominal pain after meals: to an ordinary triple feed (lunch which consists of three dishes) add 500 ml of milk or 200 g of the boiled meat. The products fried, pepper are excluded. A test is considered positive, if during 20-40 min after the meal there is typical abdominal pain.

Nitroglycerine test consists of the reception of nitroglycerine during pain attack (it is possible after a functional test with the food loading). If after its use abdomen pain diminish or fully disappear during 15-20 min, a test is considered positive.

Thermography is used for registration of the thermal field. For patients with a chronic abdominal ischemia the unevenness of the thermal field of abdomen is determined as areas of decreased irradiation. Thus the difference of temperatures between the determining area of ischemia and unchanged part of abdomen is about 2,7 °C. The method can be applied both for diagnostics of abdominal ischemia and for control of result of the performed surgical intervention.

Ultrasonic investigation without doppler is possible to use for diagnostics of concomitant diseases of organs of abdominal cavity (chronic pancreatitis, cholecystitis, tumour et cetera). These information is of interest for the differential diagnostics of abdominal ischemia.

Duplex scan is the most modern method of diagnostics and can be widely used for an exposure of hemodynamically significant lesions (stenosis, occlusion) of major arteries of patients with a chronic bowel ischemia.

Application of duplex scan allows to conduct investigation of celiac trunk and upper mesenteric artery for an exposure of hemodynamically significant lesions.

Acupuncture test of «Redoraku-Acabane», being the untraditional method of investigation, allows to find out the changes of the different systems of organism. It is based on determination of electric resistance of skin of hands and feet in 54 representative points. Findings are marked in the special map which was offered by author of method J. Nakatani (1977), with subsequent their analysis. Thus on a map the so-called «physiology corridor» is built in relation to which the information of acupuncture meridians is analysed. In patients with chronic abdominal ischemia the rule in the location of meridian of «small bowel» and «triple heater», to which the meridian of "colon" is joined sometimes, is noted. These meridians are disposed below the «physiology corridor» which indirectly specifies the presence of ischemia of organs of abdominal cavity.

Contrast angiography. Contrast angiography is the basic method of investigation in a diagnostics of AIS. Indications: presence of abdominal angina with suspicion on stenosis of visceral branches. Information of the investigation is of great value for a surgeon. Therefore conducting of investigation is expedient in patients, who agree to surgical treatment and if they do not have contraindications to them.

For contrasting of visceral branches of aorta an abdominal aortography is used. As celiac trunk and mesenteric arteries goes on different levels and different projections of front half-round of aorta, the stenotic areas of these vessels can accumulate on the contrasted aorta. In this connection the number of authors recommends to conduct films, except in front and lateral projections, in atypical cases – in oblique projections.

During the analysis of the obtained aortograms it is necessary to evaluate the condition of abdominal aorta, all of visceral branches, and also renal arteries, as the combined lesions of these vessels are possible.

According to character of lesions the cause of stenosis can be assumed. For example, absence of atherosclerotic changes of aorta allows to assume the extravasal narnumbering of celiac trunk. A. V. Pokrovskiy (1979) considers the length of stenosis the characteristic sign of atherosclerotic lesion of celiac trunk on 1-2,5 cm from its origin. Presence of unclear and uneven contours is possible as well. In nonspecific aortoarteriitis contours of involved vessel are more even.

A lot of authors divides the angiographic signs of disturbance of blood flow in visceral arteries on direct and indirect (Fig. 62).

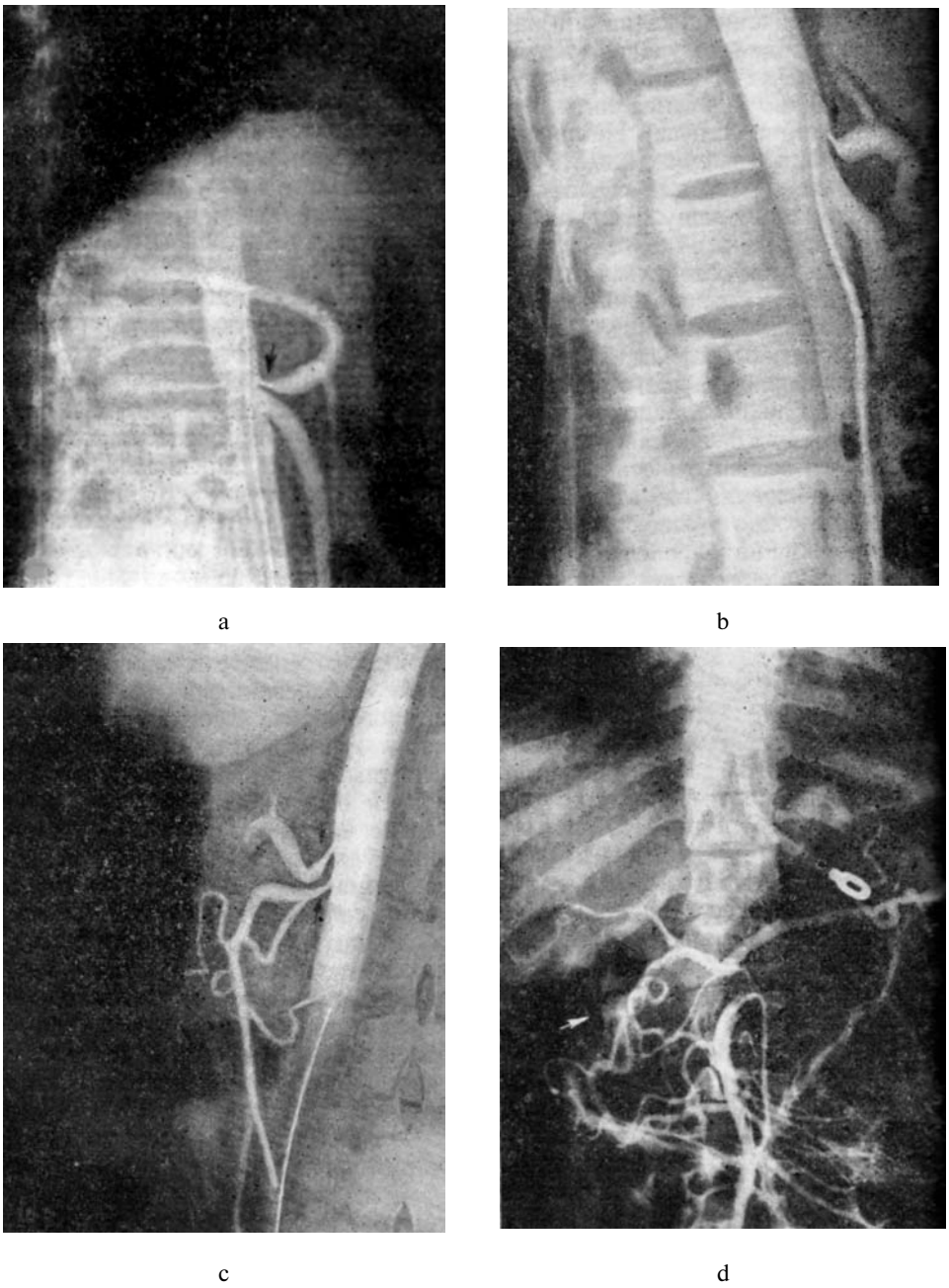


Fig. 62. Abdominal aortograms in patients with abdominal ischemic syndrome: a, b — tenosis of the celiac trunk with fibro-muscular hyperplasia and nonspecific coronary arteritis, c — atherosclerotic stenosis of the celiac trunk and superior mesenteric artery; d — retrograde filling of branches of the celiac trunk with the expansion of abdominal-mesenteric collaterals

Stenosis with poststenotic dilation or occlusion of vessel, different deformations of wall of artery are the direct signs. As well as in other arteries, stenosises can be centric, when narnumbering of lumen develops evenly from every quarter, and eccentric, when stenosis prevails from the one side.

The indirect angiographic signs of lesion of lumen of one or few vessels reflect alteration of visceral blood circulation. They are: development of collateral blood flow from superior mesenteric artery to the circulation of celiac trunk through pancreatoduodenal arteries and gastroduodeal artery, compensatory dilation of superior mesenteric, and sometimes of inferior mesenteric artery and other.

The angiographic film of extravasal stenosis of celiac trunk is characterized by next signs: incurving of celiac trunk, eccentric stenosis as an tooth on the upper contour of vessel. Quite often the celiac trunk in the area of narnumbering is pinned against an aorta, while he must go parallell to the superior mesenteric artery. The artery is arcuated up under an angle distally from the stenosis. The area of narnumbering of vessel can be disposed in its origin or in few millimetres from it on small extent. A degree of stenosis can be different. The simultaneous compression of celiac trunk and superior mesenteric artery, which is observed in 8% patients, is possible.

The evaluation of angiographic signs of ischemia of organs of digestion take into account the condition of abdominal aorta, location of origin, size and form of celiac trunk and mesenteric arteries, condition of collateral blood flow, presence of simultaneous lesion of other branches of abdominal aorta (kidney, iliac arteries).

The degree of vessel stenosis is estimated according to diameter of stenotic lumen toward the diameter of the distally located lumen of artery in percents.

The evaluation of location, form and length of vessel narnumbering, presence of deformation and poststenotic dilation, angle of artery origin from an aorta and change of this angle distally is done.

In a number of cases, for clarification of character of stenosis of celiac trunk, it is expedient to perform angiographic investigation in the phases of deep breath in and breath out. It allows with greater accuracy to suppose extravasal character of narnumbering of celiac trunk.

Intraoperative diagnostics allows finally to diagnose the lesion of visceral branches of abdominal aorta. Intraoperative diagnostics includes:

1) revision of organs of abdominal cavity;

2) inspection and palpation of visceral branches and abdominal aorta in the place of their origin;

3) direct measuring of arterial pressure distally from stenosis;

4) determination of volume blood flow by electromagnetic flowmetry;

5) evaluation of linear blood flow by the method of dopplerometry.

The revision of organs of abdominal cavity is conducted in those cases, when an operation is carried out from laparotomy approach, or there is a necessity of opening of peritoneal cavity in thoracophrenolaparotomy approach. It is performed to find possible organic pathology of organs of abdominal cavity for implementation of reconstructive operation and control inspection before wound closure.

Inspection and palpation of visceral branches is performed during all the stages of their dissection. At first an celiac trunk or superior mesenteric artery are palpated after dissection of muscular portion of medial diaphragmatic crus. In this case it

is possible to define the hyposthenic pulsation of artery and shaking of systole. In critical stenosis or occlusion of artery pulsation usually is not determined. On this stage it is possible to conduct dopplerometry for registration of linear blood velocity. Flowmetry is not always possible, it requires sufficient mobilization of artery for probe application on vessel.

The presence of whitish tissue as a thin plait, dense on palpation, is revealed in compression by the arched ligament of diaphragm. As far as the dissection of tissues is performed the increase of pulsation and disappearance of shaking of systole are possible.

The hypertrophied abdominal neuroganglion has the appearance of whitish spidery formation which envelops an artery and is dense enough on palpation.

The hypertrophied fibrotic tissue is the rough scar process. An artery in this case is embedded in fibres, dense on touch. The dissection of it is associated with certain difficulties.

In all cases of extravasal compression after dissection of artery from surrounding tissues there is increase of pulsation, absence of shaking of systole, presence of soft wall of artery and aorta. The volume of surgical intervention is terminated thereon. It should be noted that in 21,7% patients with the extravasal compression of celiac trunk mismatch of angiographic data to the intraoperative findings is marked. Greater lesion is found, that was seen on angiography. It is due to that compression of celiac trunk by the middle arched ligament, its crura and fibrotic tissue takes place not only from the anterior and superior sides but also on each side by the medial crura of diaphragm. In these cases an celiac trunk goes not strictly away from the anterior surface of aorta, but from the left anterior-lateral surface.

In intravasal involvement of arteries character of pathology is determined, in most cases, already during their dissection. In the case of atherosclerotic lesion the dissection of artery is done without difficulties. An external wall of artery is not changed, with clear contours, easily moves away from surrounding tissues, through the thin wall yellow atheromatous deposits are often seen. On palpation a characteristic hardening is determined in lumen.

In the case of fibromuscular dysplasia artery dissection, as well as in atherosclerosis, is done without difficulties. However external diameter is reveaaled as a «rosary of necklaces», or tubular narnumbering.

Quite another findings are revealed in non specific aortoarteriitis. An aorta and visceral branches are in fibrotic-inflammatory periprocess. The walls of aorta and arteries are thick, deformed and are in the perivasal net of veins and arteries of small diameter. During the dissection of arteries, as a rule, there is diffuse bleeding of tissues.

In majority of cases information of intraoperative revision coincide with the results of the histological investigation.

The direct measuring of arterial pressure distally from arteriostenosis place allows to estimate the degree of disturbance of visceral blood circulation and allows to provide the evaluation of pathogenity of performed intervention.

Electromagnetic flowmetry allows to define volume blood velocity on visceral arteries.

Dopplerometry, being the less informing method of investigation, than flowmetry, however, is widely used in intraoperative diagnostics.

Other methods of investigation, such as laboratory tests, X-ray, endoscopy of gastrointestinal tract and others, does not have a decision value in diagnostics of chronic abdominal ischemia. They allow to find out different functional and possible organic changes which develop in the conditions of chronic ischemia of organs of digestion. It is thus impossible to exclude other concomitant diseases of organs of abdominal cavity and retroperitoneum.

Thus, the most informing are abdominal angiography and duplex scan.

Differential diagnostics.

Diseases with which it will be necessary to differentiate an abdominal ischemia are very various. They are primary diseases of organs of GIT which clinically presents with abdominal pain and dysfunction of intestine. That's why the careful clinical inspection of patients is needed with the use of modern methods of investigation.

Late diagnosis of AIS is conditioned by the number of reasons. Main of them is absence of attention of doctors to the abdominal ischemia due to rareness of this pathology. In addition, as every chronic disease, an abdominal ischemia has the features of development, which are expressed in not quite typical clinical displays in the initial stages. It explains the protracted anamnesis of disease, and also circumstance that a correct diagnosis is put, as a rule, already in the stage of decompensation.

Treatment was more often conducted for chronic pancreatitis, gastroduodenitis, cholecystitis and colitis. Some of patients were operated on the organs of abdominal region: cholecystectomies, appendectomies. There were a lot of diagnostic laparotomies. All of it characterizes severity of pain syndrome in those patients.

However it is necessary to underline that the presence of chronic abdominal ischemia does not exclude the possibility of concomitant disease of organs of abdominal cavity. This circumstance requires a careful analysis and critical evaluation of clinical prfesentation, and also laboratory investigations.

Chronic abdominal ischemia it is necessary to differentiate with a chronic pancreatitis. Differential diagnostics presents certain difficulties. Both pathologies clinically presents with pain in epigastric area, related to the food intake, dysfunction of intestine and information of coprologic investigation (steatorrhoea, creatorrhoea et cetera).

The following diseases needs differentiation with AIS: acute pancreatitis, pancreatic cancer, chronic gastroduodenitis, peptic ulcer, chronic cholecystitis, partial chronic arteriomesenterial obstruction, acute appendicitis, Crohn disease, enterocolitis, retroperitoneal tumors, abdomenalgia of various origin, irritation bowel syndrome, non-ulcer dyspepsia syndrome.

Substantiation and formulation of clinical diagnosis of patient (taking into account classification of disease, presence of complications and concomitant pathology):

1. basic;
2. complication (basic disease, if present);
3. concomitant pathology (if present).

Example: *Abdominal ischemic syndrome. Obliterative atherosclerosis of visceral branches of aorta, with the primary lesion of superior mesenteric artery. Pain form. Stage of subcompensation.*

Treatment of patient with an abdominal ischemic syndrome

Choice of medical tactics

There are two types of treatment of AIS: operative and conservative. One of the most essential questions is a choice of medical tactics and indications to surgical treatment. The hemodynamic changes of visceral branches, except of development of chronic abdominal ischemia, present the risk of development of acute disturbances of mesenterial blood circulation.

Indications to surgical treatment are based on that to date there is no effective conservative treatment able to stop progress of chronic abdominal ischemia. Conservative therapy has symptomatic character and that is why efficiency of it is very relative. As a result of progress of occlusive process and presence of risk of thrombosis of visceral arteries these patients have a risk of acute disturbance of mesenterial blood circulation, which in most cases (75-95%) ends with a lethal outcome.

During determination of indications to surgical treatment in patients with a chronic abdominal ischemia it is necessary to take into account the stage of disease, character of lesion of visceral branches, prevalence and combination of lesions, and also presence of concomitant diseases.

The stage of chronic abdominal ischemia is basic in determination of indications to the operation. Surgical intervention is indicated in II (subcompensation) and III (decompensation) stages of visceral blood circulation.

Operative treatment in the I of stage (relative compensation) is indicated only in those cases, when a patient is undergoing basic reconstruction concerning the lesion of abdominal aorta, arteries of lower extremities and their branches. Reconstructions of aorto-iliac segment or renal arteries with one-moment revascularization of visceral artery for the prophylaxis of acute disturbance of mesenterial circulation of blood in an early postoperative period. Otherwise possible increase of haemodynamic disturbances in visceral arterial blood flow as a result of redistribution of blood. Implementation of reconstruction is also indicated because mobilization of visceral artery is performed from the same approach.

Concomitant diseases also must be taken into account during determination of indications to surgical treatment of patients with chronic abdominal ischemia. It is necessary to distinguish the followings groups of concomitant diseases:

1) combination of lesions of visceral branches in other vascular areas;

2) concomitant diseases of organs of digestion.

Chronic diseases of organs of digestion, such as gastritis, duodenitis, peptic ulcer, colitis et al, is not contra-indication to the operation. Surgical intervention is indicated in the period of remission of these diseases. Thus the arterial reconstruction of visceral artery for some patients can result in treatment of chronic pathology of organs of digestion.

Basic directions of conservative treatment:

- diet;
- vasodilatatots;
- symptomatic therapy, directed on diminishing of structural changes and improvement of the functional condition of organs of digestion;

– correction of hyper- and dyslipidemia for diminishing of progress of atherosclerosis by medicnes which contain essential phospholipids, statins;
– antioxidants; antiagregant therapy - clopidogrel;
– correction of diabetes mellitus; treatment of complications.

***The diet of patients with AIS*:**

– 5-6-time feeding;
– limitation or exception of the high-calorie refined products and dishes (sugar, pastry wares, margarine, fat, flour wares and others);
– the source of carbonhydrates must be green-stuffs, in particular a little bit of potato (baked or boiled);
– lipotropic products (non-fat cheese, buckwheat, wheat);
– irreplaceable components of meal (seasonal green-stuffs, fresh juices, greenery, decoction of wild rose, pharmacy yeasts);
– products which contain a valuable albumen (fish, non-fat sorts of meat, birds, rabbist, eggs, cheese);
– functional food stuffs and food additions (soy-bean lecithin, sea products, algae, spirulin, vitamin-mineral mixtures);
– replacement of animal fat (except fish) by vegetable - by soy-bean, olive, sunflower, peanut, corn oil;
– enriching of meal by lecithin, cholin, inositol, magnesium, iodine, by vitamins A, B, E;
– use of food fibres, especially pectin (bread from grain of rough grade, millet, cabbage, wheat, green-stuffs, fruit and others).

For decrease of severe oxidative stress in AIS it is recommendede to enrich a meal by antioxidants (vitamins of C, E, selenium, methionine).

In AIS primary and secondary exocrine insufficiency of pancreas develops. That's why in treatment of patients with AIS enzyme medicines are absolutely needed.

All types of surgical interventions for chronic abdominal ischemia are divided into reconstructive, decompressive and prophylactic. The last ones are performed on visceral arteries in asymptomatic lesions in the case of the combined lesions of aorta or renal arteries, when there are indications for their reconstructions.

There is other classification (Wylie E. et al., 1980) in obedience to which authors divide all operations into three groups:

1) extravasal decompression;
2) direct reconstruction;
3) indirect reconstruction.

Extravasal decompression means the section of the middle arched ligament of diaphragm, fibrotic tissue et cetera (Fig. 63).

Direct reconstruction means endarterectomy, resection of area of stenosis with an anastomosis «end to end» or reimplantation into aorta – «anatomic» interventions without application of other plastic materials (Fig 64).

An indirect reconstruction means application of different bypass interventions with the use of different prosthetic material (auto-, allo-, synthetic) or extraanatomic reconstructions (spleen-renal, mesentericorenal, spleen-mesenteric and other anastomoses) (Fig. 65).

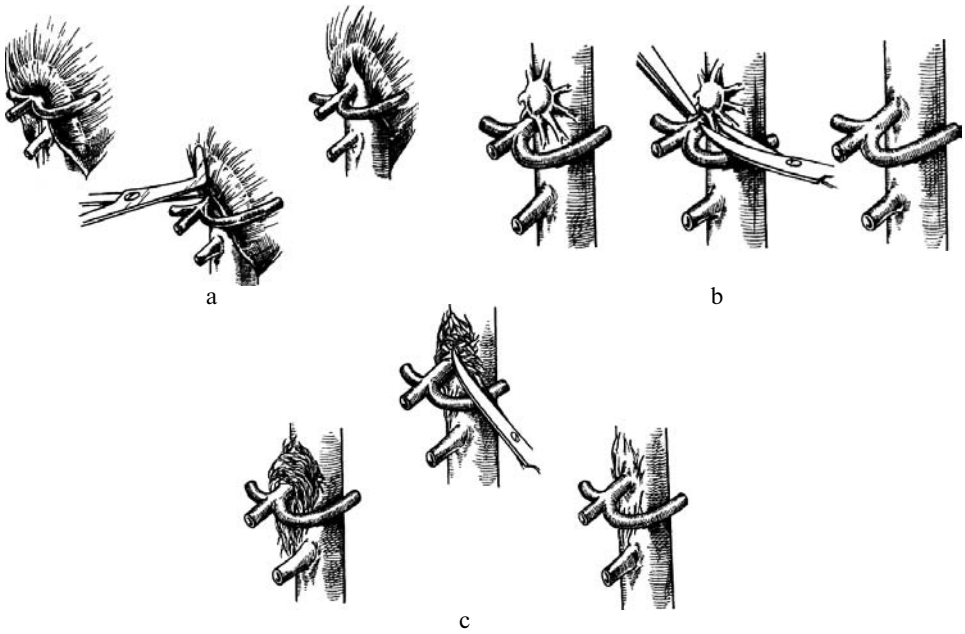


Fig. 63. Scheme of lesion and surgery of different types of extravasal compression: a — stages of thecutting middle diaphragm bow-shaped ligament; b — Remove of the hypertrophy solar plexus ganglion and sight of the abdominal trunk after surgery; c — abdominal trunk release from fibrous tissue

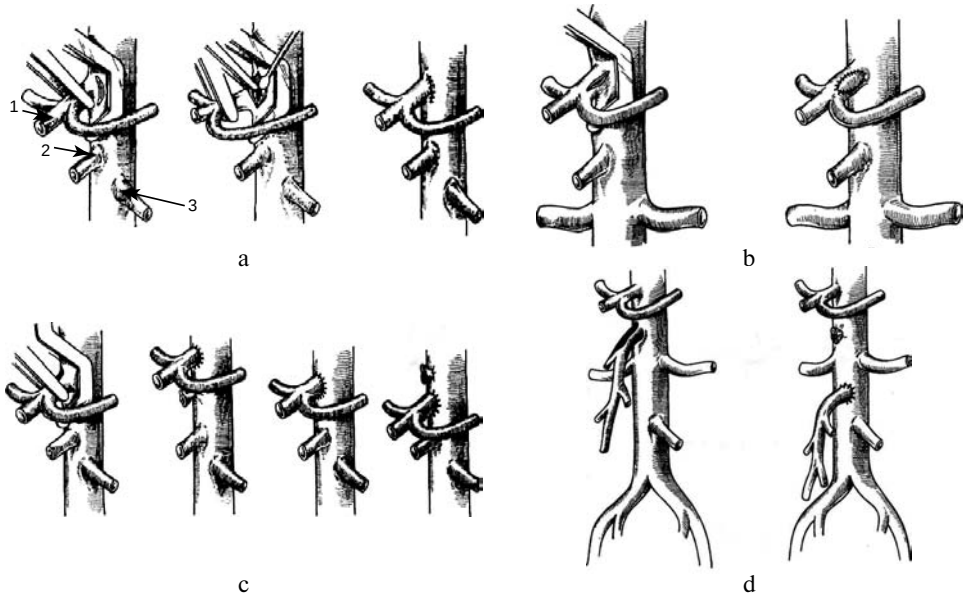


Fig. 64 Surgery of the direct reconstruction with AIS:
a – intraaortal endarterectomy from celiac trunk (1 — celiac trunk, 2 — superior mesenteric artery, 3 — left renal artery); b — direct endarterectomy from celiac trunk with overlay patches; c — reimplantacion of celiac trunk to the aorta: I — above the orifice; II — in the old orifice; III — below the natural orifice; d — reimplantation superior mesenteric artery to the infrarenal aorta

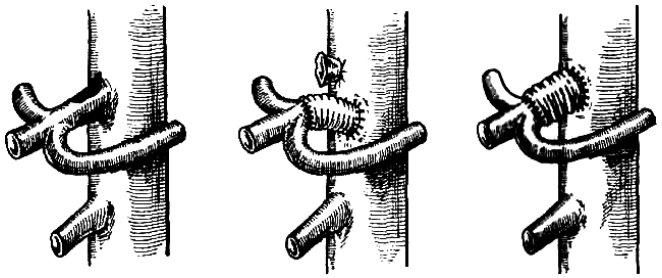


Fig. 65. Varieties reconstruction of the abdominal trunk with prosthesis

The choice of that or other type of surgical intervention is determined by character of lesion taking into account the accepted tactics of treatment of such patients in every concrete clinic (Fig. 66).

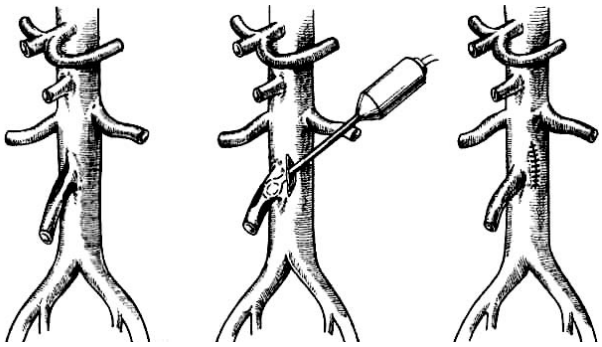


Fig. 66. Scheme of ultrasonic endarterectomy surgery

Preoperative preparation does not differ from surgical patients with the diseases of gastrointestinal tract.

In persons with the III stage of disease, if a cachexy takes a place, it is necessary to conduct the correction of water-electrolyte and albumin balance. In the proper situations it is necessary to enter water-salt solutions, vitamins, albumin, components of blood.

If non specific aortoarteriits is in the active phase, the proper antiinflammatory therapy is needed, and in some cases, hormonal preparations.

In patients with combined (ischemic heart disease, cerebral, lower extremities and other ischemia) and concomitant (arterial hypertension, peptic ulcer, colitis et cetera) pathology the proper therapy is conducted.

General anaesthesia is usually applied

Surgical approach. In the reconstruction of celiac trunk and superior mesenteric artery left-side thoracophrenolumbotomy is used in VII-IX intercostal spaces or middle line laparotomy. In the case of necessity a thoracophrenolumbotomy can be extended to pararectal line (Fig. 67).

A choice of optimum surgical approach is the important stage which determines success of future operation greatly.

1. For surgical intervention on celiac trunk or upper mesenteric artery it is recommended to perform thoracophrenolumbotomy in VII-IX intercostal spaces depending on the constitutional features of patient.

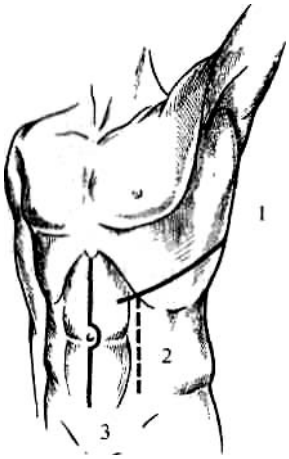


Fig. 67. Approaches in the reconstruction of visceral branches: 1 — thoracophrenolumbotomy; 1, 2 — thoracophrenolaparotomy; 3 — middle line laparotomy

In the case of necessity incision can be extended to pararectal line downward (thoracophrenolaparotomy).

Thus a patient is concluded on a right side. The left upper extremity is taken beforehand and a bit upwards, fix on the special elbow-rest.

2. A middle line laparotomy for reconstruction of the celiac trunk and superior mesenteric artery is less comfortable, than thoracophrenolumbotomy. It is indicated, when the extravasal compression of celiac trunk takes a place.

3. In those cases, when a necessity is for the one-time united reconstruction of abdominal aorta and arteries of lower extremities, execute complete middle laparotomy or thoracophrenotomy.

Thoracophrenolaparotomy *consists at first of typical thoracophrenolumbotomy in VII-IX intercostal space depending on the volume of intervention and constitutional features of patient. After crossing of costal arch incision is continued downward pararectally to the inguinal area if necessary.*

Surgical correction of pathology of visceral arteries. Depending on a kind and character of lesions of visceral arteries the proper surgical interventions are applied. If cause of disease is an external compression of vessel, extravasal factor is eliminated only. When cause of stenosis is the change of vessel wall, which is observed in atherosclerosis, arteriitis, fibromuscular dysplaisa the different types of reconstructive operations are performed. Reconstructive operations are performed in aneurysm of visceral arteries.

Due to this all types of surgical interventions on visceral arteries are divided on:

1. reconstructive;

2. relatively reconstructive (decompressive).

Decompressive operations. If cause of chronic abdominal ischemia is extravasal compression of celiac trunk, this patient undergoes extravasal decompression which consists of section or resection of compressing factor. These interventions can be divided on:

At the mixed character of extravasal compression the mentioned interventions can be combined.

Reconstructive operations. In those cases, when cause of chronic abdominal ischemia is actually lesion of arterial wall (atherosclerosis, arteriitis, fibromuscular dysplasia) there is a necessity for a reconstructive operation.

Approach to the isolated reconstruction of celiac trunk and superior mesenteric artery is left thoracophrenolumbotomy. If there is a necessity for intervention on infrarenal part of aorta and inferior mesenteric artery, middle line laparotomy or left pararectal approach are performed. In more difficult cases thoracophrenolaparotomy may be used.

All reconstructive operations can be divided into the following types:

1) endarterectomy:
2) reimplantation;
3) prosthesis;
4) by-pass;
5) minimally invasive methods.

Type of reconstructive operation is determined by aetiology of disease, length of lesion, and location of pathological process. Reconstruction is possible only in the satisfactory condition of distal vessels. Final decision about the necessity of arterial reconstruction and its type is always accepted after intraoperative diagnostics, which includes inspection, palpation, and also instrumental investigations (doppler ultrasound).

Before the clamping of artery solution of heparin, taking into account weight of patient, is infused to prevent clotting process in artery (patient of weight about 70 kg usually needs 5000 units of heparin).

1. *Transaortal endarterectomy is indicated in local atherosclerotic lesion in the orifice of artery, which spreads no more than on 1,0-1,5 cm from the wall of aorta.*

A reconstruction ends with imposition of single layer sutures on the aorta opening.

Endarterectomy with longitudinal arteriotomy can be also used, in this case longitudinal patch is put to close the aorta opening.

2. *Reimplantation of visceral branches. In those cases, when there is a lesion of artery orifice and to perform adequate endarterectomy is not possible because of aortoarteriitis or fibromuscular dysplasia and arterial wall is in the cicatrical-changed conglomerate, reconstruction consists of resection of artery with reimplantation of it into the aorta.*

Reimplantation of visceral artery into the aorta is possible only if aorta wall is not changed, but it is far not in all of cases, especially in atherosclerosis and aortoarteriitis. In these cases reconstructive operations with the use of plastic materials (auto-, allo-, xeno- and synthetic prosthetic appliances) are used.

3. *Prosthesis of visceral arteries. Prosthesis of visceral arteries is performed with distal anastomosis with an artery «end to end». This method of reconstruction is used for the extended lesions of arteries (more than 1,5 cm), when to perform reconstruction with the use of only artery tissue is not possible.*

Depending on the location of proximal anastomosis we divide prosthesis of visceral arteries into two types:

1) proximal anastomosis is done in old orifice of arteries;
2) an aortic anastomosis is formed in other aorta location.

4. *By-pass of visceral branches. The method of by-pass of visceral branches means implementation of distal anastomosis of prosthesis with an artery «end to side». Antegrade and retrograde by-pass are distinguished.*

5. *Angioplasty in treatment of patients with chronic abdominal ischemia. It has large possibilities in the reconstruction of blood flow of visceral arteries. An angioplasty advantageously differs from the traditional surgical methods of treatment of patients with chronic abdominal ischemia. It is less traumatic, less blood loss as compared to conventional surgery, reduction of hospital stay.*

However efficiency of these interventions is of short duration and early recurrence is marked during 3-6 months. Metallic stents are used during angioplasty in the last years to obtain more prolonged effect.

Surgical prophylaxis of bowel ischemia in reconstructive operations for aneurysm of abdominal aorta. In most cases the operation is accompanied with the protracted crossclamping of aorta. It predisposes to development of number of postoperative complications, one of which is bowel ischemia. With the purpose of its prophylaxis reimplantation of inferior mesenteric artery is performed.

Electromagnetic flowmetry, Doppler ultrasound and measuring of arteriotony in circulation of inferior mesenteric artery, allow to carry out control the condition of visceral blood circulation during an operation.

Information of arteriotony can be the direct indication for reconstruction of inferior mesenteric artery. If retrograde pressure in inferior mesenteric artery is less than 50% from systemic or less than 50 mm Hg reimplantation of inferior mesenteric artery into prosthesis is considered obligatory.

Results of treatment

In accordance with principles of international classification of diseases and taking into account etiologic and pathogenic mechanisms, an abdominal ischemic syndrome is included to the VIIth class of organs of cardiovascular system.

Very important for today there is a social aspect of ischemic disease of organs of digestion.

Limitation of vital functions, functional insufficiency of the system of digestion, decline of social and labour activity develops in patients with ischemic disease of organs of digestion.

Surgical treatment allows to obtain positive result in the majority of patients (93%). It allows to improve quality of life of patients and increase life expectancy as compared to patients which underwent conservative treatment only. Conservative treatment of patients with chronic abdominal ischemia does not allow to obtain compensationof blood circulation in visceral organs.

Acute blockage of blood flow in mesenteric vessels can become complication of AIS

Acute occlusion of mesenteric vessels is relatively rare disease. The infarction of intestine is caused by blockage of superior mesenteric artery and its branches as a result of embolism (60-90%) or acute thrombosis (10-30%). The blood clots of heart are the basic source of embolism in its rheumatic and atherosclerotic lesions, myocardium infarction, endocarditis, rarer blood clots are of aorta origin. Acute thrombosis usually develops on the backgroundof the pathologically changed arteries as a result of atherosclerosis, aorto-arteriitis, obliterative endarteriitis, fibromuscular dysplasia, nodular periarteritis, diabetes mellitus. The premonitory symptoms of chronic insufficiency of mesenteric blood circulation often (in 40-50% patients) precede acute occlusion of mesenteric arteries. That's why the intestine infarction develops mainly in older patients (middle age is more than 50 years).

Acute occlusion of mesenteric vessels often results in the intestine infarction, as collateral blood circulation does not have time to develop. Rate and terms of intestine gangrenes after occlusion of mesenteric vessels it is not enough studied, necrosis does not develop always. In occlusion of only inferior mesenteric artery necrosis of intestine develops rarely.

Intestine infarction can be result not only of acute arterial occlusion but also venous thrombosis, combination of arterial and venous occlusion, prolonged spasm of branches of mesenteric artery; in patients with severe cardiac insufficiency, endotoxemic shock, in allergy as a result of disturbance of microcirculation in intestinal wall, in young women – due to the reception of hormonal anticonceptives.

Acute bowel ischemia causes progressive changes in absence of collateral blood flow, which during 2 - 4 - 6 hours become irreversible. Viability of bowel can be restored, if during this period disturbances of blood flow are removed. However even if the resection of bowel will not be needed, there are consequences of ischemic attack as fibrosis with disturbance of absorption and motor and secretory functions of intestine in normal macroscopic appearance.

The clinical presentation of acute occlusion of mesenteric arteries is similar to the syndrome of acute abdomen of other aetiology. Very characteristic is acute onset and at the same time, especially in an initial period, very little objective data.

Abdominal pain is the first and basic symptom — appears suddenly. Pain is severe, usually permanent, in an umbilical area, and then in all areas of abdomen, quite often irradiates into the back. Very often muscular defence of abdomen is absent at first, but there always is acute tenderness on superficial palpation of abdomen. Tenderness is more often to the left of belly-button. Muscular defence develops in late terms during development of peritonitis. Through little time pain can disappear or diminish on some time (occult period).

Distension of abdomen is expressed in various degree, usually in more late stage. Nausea, vomiting, sometimes with blood, pallor, hyperhidrosis, stool with mucus and blood are present in the beginning of disease; a peristalsis is usually present. Body temperature rises only in some patients.

Through 3 - 12 (24) hours from the onset of the disease on background of some diminishing of pain the symptoms of paralytic intestinal obstruction develop with disappearance of peristalsis, distension of abdomen and severe general condition, with endotoxemic shock, as a result of diminishing of volume of circulatory fluid, intoxication. In the final stage of peritonitis with the classic presentation of paralytic intestinal obstruction, which is characterized by severe intoxication, changes of intestine are usually irreversible.

In the acute thrombosis of mesenteric vessels, which develops on background of chronic insufficiency of mesenteric circulation of blood, development of clinical symptoms is more gradual. So the irreversible changes of intestine develop in more late terms that mean favourable possibilities for timely diagnostics and surgical treatment.

Characteristic and permanent is high leucocytosis - 15-20x10^9/L leucocytes, which appears in patients already in few hours from the onset of disease (in combination with the severe pain syndrome and little objective data). On X-ray in the early stage usually no pathological changes are found (unlike mechanical, strangulation intestinal obstruction, perforated peptic ulcer), and there is clinical presentation of paralytic

intestinal obstruction in the late stage, that has non specific signs of any intestinal obstruction, regardless of its aetiology.

Clinical presentation described higher is characteristic for patients with acute occlusion of superior mesenteric artery. Acute occlusion of celiac trunk is observed rarely and is characterized by appearance of pain in the upper part of abdomen, in the back, in right hypochondrium, by early nausea, bloody vomiting. Disease must be differentiated with perforated gastric ulcer, acute pancreatitis, cholecystitis, the myocardium infarction with the syndrome of pseudoabdominal angina.

Early timely diagnostics of disease is difficult even in typical cases. Approximately in 6% patients it is diagnosed in proper time. Difficulties of diagnostics, from one side, consist in rareness of disease, and from other - in a very short period from the moment of development of intestine infarction and appearance of the first clinical symptoms to the development of irreversible changes in intestine. On differential diagnostics it is necessary to take into account next features.

1. Presence of possible sources of embolism (atherosclerosis, cardiosclerosis, myocardium infarction, heart-disease and other).

2. Sudden origin of abdominal pain, usually severe with indefinite location.

3. Disparity between severe abdominal pain syndrome, severe general condition (endotoxemic shock) and little information on physical examination and X-ray.

4. High leucocytosis in an early period of disease.

5. Stool and vomiting with the admixture of blood.

In acute development of syndrome of acute abdomen in the patients of elderly and senile age with heart-diseases, myocardium infarction, above all things it is necessary to exclude occlusion of mesenteric arteries.

Differentiation of the acute arterial and venous thrombosis of mesenteric vessels is extraordinarily difficult. Venous thrombosis can be supposed in presence of venous thromboses of other location, signs of portal hypertension, tumour of pancreatoduodenal area.

Angiography and laparoscopy are the modern methods of early diagnostics of intestine infarction. On laparoscopy intestine infarction is diagnosed on discoloration of bowel, presence of hemorragic exudate, edema of mesentery. Exact early diagnosis is possible only by angiography. Abdominal aorto-arteriography and selective angiography, that is usually performed by the retrograde cannulation of aorta through a femoral artery, allow to diagnose the disease in the first 5-6 hours. In the severe condition of patient in the late stage of the disease angiography is not indicated, and better quickly to conduct laparotomy.

Only agonizing condition of patient makes to decline operative intervention in acute occlusion of mesenteric arteries. It is necessary justified to count conscious hyperdiagnostics of intestine infarction with immediate hospitalization in the case of doubtful diagnosis. At suspicion on the intestine infarction laparotomy is obligatory, even if it can remain only trial.

Treatment of acute occlusion of mesenteric vessels - surgical. Purpose of operation – to restore blood supply of intestine, if it is possible, and to perform the resection of necrotic loops of intestine. The choice of method of operation is determined by the condition of patient, character of occlusive lesions of vessels, terms from the onset of disease and condition of intestine.

In absence of signs of gangrene of bowel in the first 6 hours from the onset of disease operation on vessels is performed with the purpose to remove their blockage. In the developed necrosis of intestine it is expedient to combine the resection of intestine with an operation on mesenteric vessels, if allows the condition of patient, as a removal of blockage and restoration of blood circulation prevent progress of thrombosis, improve postoperative course, prevent dehiscence of anastomosis.

In the limited intestinal infarctions and developed collateral circulation an operation on vessels is not always expedient. In these cases the resection of the affected intestine is performed.

At presence of intestine gangrene maximal radicalism is needed. If an operation on vessels is not performed, the resection of intestine must be performed within the limits of supply of occluded vessel. At high occlusion of main trunk of mesenteric artery a radical resection is needed, even if the area of necrosis of intestine is limited. However resection of all small intestine and right colon, as a rule, is incompatible with life. At the severe condition of patient the resection of intestine or exploratory laparotomy are performed.

V.4. Aortic aneurysmes

The frequency of aortic aneurysms (AA), according to the postmortem autopsies varies from 0,9 to 1,1%. In the structure of mortality aortic aneurysm has the 13th place. Approximately 0,6% of women and 1,2% of men die of rupture. In studies of domestic and foreign authors it was found that within 3-5 years after diagnosis of aneurysms of the thoracic aorta occurs fatal dissection. According to A. Hirst about 50% of patients die within the first 48 hours dissection, 84% — within the first month, 90% — within 3 months, and only 8% remain alive at 1 year. Massive bleeding occurs in the retroperitoneal space, free peritoneal cavity, in the presence of fistulas in the lumen of the digestive tract or the inferior vena cava, while mortality is over 90%.

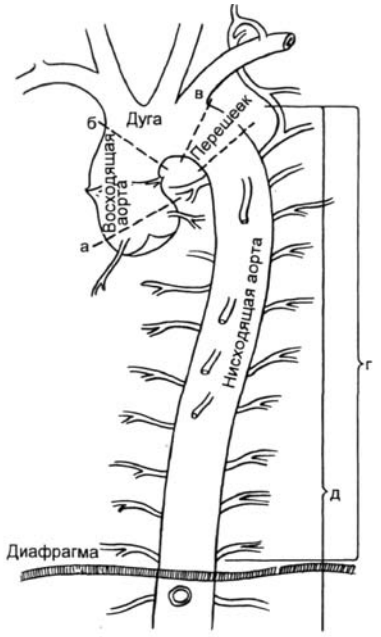


Fig. 68. Localization of aneurysms of the thoracic aorta. a - aneurysm of Valsalv sinuses, b - aneurysm of the ascending aorta; c - aneurysm of the aortic arch; d - aneurysm of the descending aorta; e - aneurysm of thoracoabdominal aorta

Aneurysms of the aorta

The aneurysm of the aorta is an increase in the diameter of the aorta more than doubled in comparison with normal or a local bulging of the wall (Fig. 68).

The average diameter of the abdominal aorta is 18-20 mm. Aortic root diameter should not exceed — 31 mm, ascending aorta — 32 mm, the aortic arch — 32

mm, the proximal portion of the descending aorta — 28 mm, middle portion of the descending aorta — 27 mm.

1. The following classification of aneurysms of the aorta is used

Classification of aneurysms of the abdominal aorta (A. A. Shalimov, N. F. Druk, 1979):

I. Infrarenal

II. Suprarenal

III. Infrarenal and suprarenal forms are:

1) asymptomatic
2) typical uncomplicated
3) aneurysm associated with occlusive process in the branches of the aorta
4) aneurysm, complicated by dissection, or rupture

Surgical classification of abdominal aortic aneurysms (V. I. Burakovsky et al., 1989):

I. Suprarenal aneurysm isolated, diffuse without engaging in the process of bifurcation of the abdominal aorta and diffuse with involvement in the process of bifurcation of the abdominal aorta.

II. Infrarenal aneurysm without involvement in the process of bifurcation and involvement in the process of bifurcation of the abdominal aorta.

III. Suprarenal and infrarenal forms are: 1) uncomplicated 2) complicated

IV. Complicated forms of abdominal aortic aneurysms: 1) Part rupture - a tear or dissection of wall of the aneurysm with formation of subadventisial hematoma, sometimes with distal fenestration of bifurcation 2) Complete rupture with haemorrhage in the retroperitoneal space, free peritoneal cavity, the organs of the gastrointestinal tract and the system of the inferior vena cava (directly into the trunk of the inferior vena cava, the left renal vein, the right iliac vein).

Classification of A. V. Pokrovsky (1968):

Etiology:

1. Congenital.
2. Acquired:
   a) inflammatory (specific and nonspecific)
   b) non-inflammatory (atherosclerotic, traumatic).

Morphology:

1. True.
2. False.
3. Dissecting.

In form:

1. Saclike.
2. Diffuse.

On clinical course:

1. Uncomplicated.
2. Complication (rupture).
3. Dissecting.

Localization:

Type I — aneurysm of the proximal segment of the abdominal aorta involving visceral branches.

Type II — aneurysm of infrarenal segment without involvement of the bifurcation.

Type III — infrarenal segment aneurysm involving the bifurcation of the aorta and iliac arteries

Type IV — total lesion of the abdominal aorta.

In accordance with the localization isolated aneurysms of the thoracic aorta are classified:

- aneurysm of sinuses of Valsalv includes the initial segment of the aortic fibrous ring of the aortic valve to sinotubular sulcus - the line on which are projected the upper points of commissures of semilunar valves of ascending aorta. These aneurysms are usually congenital in nature;
- aneurysm of the ascending aorta - the level of sinotubular sulcus to the mouth of brachiocephalic trunk;
- at the suggestion of D. Cooley (1990), aneurysm of the ascending aorta, accompanied by the expansion of the fibrous ring of the aortic valve, sinuses of Valsalv and the loss of sinotubular sulcus were in a separate category called "annuloaortal ectasia";
- aneurysm of the aortic arch, including the segment of the thoracic aorta from the mouth of an artery anonyma to the level of orifice of the left subclavian artery;
- aneurysm of the descending aorta, located in the segment of the thoracic aorta between the left subclavian artery and aortic aperture;
- aneurysm of thoracoabdominal aorta, beginning in the descending aorta and extend to its ventral division (Fig. 72).

To describe the last type of aneurysms classification of Crawford E.S. (1986) is used: in I type of thoracicoabdominal aneurysms lesion begins in the proximal half of the descending aorta and ends above the level of orifice of the renal arteries; in type II aneurysm extends from the proximal half of the descending thoracic aorta below the renal arteries orifice; in type III lesion begins in the distal half of the descending aorta and extended to various length in the abdominal aorta; in type IV aneurysm begins at the aortic orifice and includes all the abdominal aorta.

On the etiology aneurysms of thoracic aorta are divided into two major groups: congenital and acquired.

I. ***Congenital disease***.

1. Cardiovascular system (bi-or single-winged aortic valve stenosis of the aortic valve, coarctation of the aorta, aortic isthmus tortuosity).

2. Connective tissue (Marfan's syndrome, Ehlers-Danlos syndrome, etc.).

II. ***Acquired disease.***

1. Noninflammatory, degenerative: atherosclerosis or degeneration of media; postoperative (places of cannulation, aortic suture line of anastomosis or prosthetic aortic patch).

2. Inflammatory: Infectious and non-infectious aortitis; mycotic; infection of the prosthesis of the aorta.

3. Post-traumatic.

4. Iatrogenic (catheterization of the aorta, balloon contrapulsation, etc.).

5. Idiopathic (medio-necrosis of Erdgeym).

6. Hormonally-caused (medio-necrosis of pregnant).

Features of the examination of patients with suspected aneurysm of the thoracic aorta.

The clinical picture in aneurysms of the thoracic aorta is very variable and depends on the location and size of the aneurysm.

In inspection of patient:

1). Complaints to the underlying disease: in approximately 75% of cases the disease is completely asymptomatic.

The most common subjective symptoms in aneurysms of the ascending aorta are retrosternal pain as a result of the destruction of the aortic wall, or compression of surrounding organs, or stenocardia character with lesions of the coronary arteries. Compression of superior vena cava accompanied by the edema of the upper limbs, head and neck. Also symptoms of heart failure may be present: palpaitation, shortness of breath, dizziness, decreased physical activity with a concomitant lesion of the aortic valve.

In aneurysms of arch and descending thoracic aorta pain syndrome may occur as behind the breastbone, and in the interscapular region. At this location of aneurysms symptoms associated with compression of surrounding organs and tissues are more typical. Thus, compression of the esophagus manifests by dysphagia, recurrent nerve compression - by the developed dysphonia, and compression of the vagus nerve - by noted bradycardia and excessive salivation. Compression of the trachea and left main bronchus may be accompanied by dyspnea, stridor, the development of lungs edema.

The lesion of the aortic arch branches is accompanied by chronic vascular clinic - brain disease, symptoms of transient or acute disorders of cerebral circulation in hemispheric or fan type.

Aneurysms of thoracoabdominal aorta is often accompanied by abdominal pain, especially in the epigastric region, they are characterized by symptoms related to involvement in the process of the branches of the abdominal aorta and the development of ischemia related area. These are signs of chronic abdominal ischemia in the lesion of celiac trunk, superior mesenteric artery, severe renovascular hypertension with renal artery constriction, in some cases, ischemia of lower extremities.

Rarely occlusion of intercostal arteries causes ischemic lesions of the spinal cord up to paraparesis and paraplegia.

2). **History of the disease**: in most cases the development of AA occurs slowly, symptoms increases gradually. The course of the disease progresses, however, there are cases of acute disease development.

3). **Medical history of life**: History taking may indicate the presence of congenital diseases among relatives (Marfan syndrome), trauma of the chest. It is important to ascertain the fact of the disease in the history of syphilis, transferred an episode of acute systemic inflammation (nonspecific aortoarteriitis). The presence of surgical interventions on the aorta (aneurysm can develop in the field of vascular suture).

***Clinical physical examination (with the characteristic features of this disease)*:**

1). Evaluation of the general condition of the patient

Consciousness is usually clear. Constitutional peculiarities – usually asthenia.

2). Information about the appearance of the patient:

Aneurysm of the thoracic aorta:

Visual inspection of the patient with an aneurysm of the thoracic aorta usually is of little value, except for patients with Marfan syndrome, which is based on the anomaly of the connective tissue has a characteristic appearance: tall, narrow face, disproportionately long limbs and spider-fingers.

3). «***Locus morbi***»:

Attention is drawn to the visible pulsation in the supraclavicular areas and jugular fovea, that in some cases is a sign of aneurysm of the thoracic aorta. Rarely in giant aneurysms (mainly syphilitic) is uzuration and destruction of ribs, sternum, so that the pulsation of the aneurysm is visible just under the skin. In lean patients with large thoracicoabdominal aneurysms pulsation of anterior abdominal wall can be seen. Compression of the cervical sympathetic trunk leads to syndrome of Bernard — Horner.

Palpation is effective in determining the asymmetry of the peripheral arterial pulsations, which may indicate involvement in the process of aortic arch branches and iliac arteries. Asymmetry should also be determined in the supraclavicular and jugular areas.

In aneurysms of thoracoabdominal aorta pulsation in the epigastric and mesogastral areas helps to determine the size of the aneurysm, its relationship with the organs of the abdominal cavity, and to make differential diagnosis between thoracoabdominal aneurysm and infrarenal aneurysm of the abdominal aorta.

If by the transversely located to the course of aortic aneurysm hand it is possible to separate it from the costal arch, it usually means that aneurysm does not extend above the level of a orifice of the renal artery from the abdominal aorta.

The most important among the physical methods is auscultation. The presence of systolic murmurs over the aorta is determined in 70% of cases with aneurysms of the thoracic aorta. At auscultation of the aortic valve it is possible to reveal the aortic valve insufficiency (diastolic, or when listening systolic-diastolic noise). Weakened breath above the surface of the lungs.

4) Examination of musculoskeletal system: patients with an aneurysm of the thoracic aorta kyphoscoliosis, abnormal motility and deformity of joints.

5) Leading clinical symptom: in aneurysm of the thoracic aorta: chest pain.

6) Based on examination findings and clinical physical examination the patient may have the following clinical diagnosis: Aneurysm of Thoracic Aorta.

Additional methods of research:

1. Clinical analysis of blood.
2. Coagulogram
3. Measurement of blood pressure in all four extremities
4. X-ray imaging.
5. Computed tomography.
6. Angiographic study.
7. Transthoracic echocardiography.
8. Transesophageal echocardiography.

1). Clinical analysis of blood: changes are nonspecific: possible anemia, moderate increase in ESR.

2). Coagulogram - the phenomenon of hypercoagulability.

3). Measurement of blood pressure in all four extremities performed by cuff method or by using Doppler. Normally blood pressure on the extremities should be equal to pressure on the hands or exceed its not more than on 10-20 mm Hg. Gradient of blood pressure over 10 mm Hg between the hands may be an indication of the lesion of the branches of the aortic arch or stenosis of the aortic arch with aneurysm of descending portion. Blood pressure gradient between the upper and lower extremities (with decrease in blood pressure in the legs) can occur in aortic stenosis with aneurysms of the descending or thoracoabdominal department.

4). Radiological examination: X-ray signs of aneurysm of the thoracic aorta is the presence of homogeneous formation with clear margin, not separated from the shadow of the aorta and pulsating synchronously with it. You can also detect the deviation of the trachea, bronchus, esophagus by aneurysm. Calcification along the contour of the aorta in most cases, evidence of aneurysmal lesions of the aorta.

5). Computed tomography (CT) allows to determine the size, localization of aneurysm of the aorta. It is very important during CT to find information about the extravasations of blood and condition of surrounding tissues of the aorta. CT clarifies the dimensions of the walls of the aorta and their changes, the contents of the lumen of the aorta (blood clot, detached intima), and the involvement of the branches of the aorta, the involvement of bone structures (sternum, spine), location, degree of involvement and the size of the adjacent major vessels, the presence of aortovenous fistulas, the presence of fluid in the pericardial cavity.

Dissecting aneurysm of the aorta is determined as two lumen and two contour wall of the aorta.

6). Angiographic study. For full assessment of the thoracic aorta study is conducted in 3 projections - direct, left and oblique lateral view. Localization, length of aneurysm, the presence of thrombotic masses are defined.

The main angiographic sign of dissecting aneurysm of the aorta is a double contour.

7) Transthoracic echocardiography. In most patients the root of the aorta and the proximal portion of the ascending aorta can be visualized, the dilation of this segment thickening of the wall, the detached intima, compression of the left atrium are identified. Echocardiography also provides information about severe arterial insufficiency, pericardial effusion, systolic and diastolic dysfunction of the left ventricle of the heart. Visualization of the aortic arch and descending thoracic aorta is usually more problematic and may require additional access (suprasternal, left and right parasternal and subclavian).

8). Transesophageal echocardiography. It allows to specify the exact location and size of the aneurysm in the ascending and descending portions of the thoracic aorta, the presence of thrombi in the lumen, to assess aortic valve function and severity of aortic regurgitation, reliably differentiate aortic aneurysm from the dissection and intramural hematoma.

Differential diagnosis: aneurysm of the thoracic aorta - tumor of mediastinum, lung.

Rationale and formulation of clinical diagnosis (including the classification of disease, presence of complications and comorbidities):

1) primary - aneurysm of the thoracic aorta
2) complications (if present)
3) comorbidities (if present)

Treatement of patient with an aneurysm of the thoracic aorta

The choice of treatment: the treatment of patients with aortic aneurysm is only surgical.

Types of surgical interventions

Aneurysm of thoracic ascending aorta - surgical treatment is indicated in increasing of hemodynamic disturbances caused by aortic valve insufficiency and / or progressive dilation of the ascending aorta. The operation consists in prosthesis of ascending aorta and aortic valves.

Aneurysm of the aortic arch – alloprosthesis of the aortic arch and its branches.

Aneurysm of the descending thoracic aorta - excision of the aneurysm with alloprosthesis.

***Possible postoperative complications*:**

– Postoperative hemorrhage;
– Dynamic intestinal obstruction;
– Thromboembolic complications;
– Cardiac - pulmonary

Emergency conditions

Aortic dissection is the separation of the aorta media into two layers by outflow of intraluminal blood through the rupture of the intima, and development of pathological communication between the true lumen of the aorta and formed channel in the middle layer of the aorta called the false lumen.

In aortic dissection several periods are marked:

– acute (up to 48 hours after dissection);
– subacute (up to 2-4 nedel);
– chronic (up to 2 months from the date of dissection).

***There are two classifications of types of aortic dissection localization and length*:**

De Bakey (1965) divided the dissection of aorta as follows:

Type I — dissection begins in the ascending portion, passes through the arch in the descending portion, and then can spread to any extent;

Type II — dissection involves only the ascending aorta;

IIIa type — dissection starts below the orifice of the left subclavian artery and is distributed within the descending aorta;

IIIb type — the dissection is the same as in IIIa, but extends through the aortic aperture into the abdominal aorta.

Second original classification was the classification of Stanford University, proposed by Daily P.O. et al. in 1970:

– Type A — any dissection involving ascending aorta;
– Type B — any dissection below the level of orifice of the left subclavian artery.

The clinical picture of aortic dissection is characterized by acute, almost fulminate onset on background of increasing blood pressure with the development of intense pain, as expressed so that, despite of hypertension, the patient appears to be in collaptoid status (lethargy, cold sweat, weak peripheral pulsation). Decrease of blood pressure leads to the suspension of dissection of the aorta and reduce the severity of pain with the stabilization of general condition, but then this process may increase again and again.

Location of pain depends on where dissection starts: behind the breastbone - in I and II types, in the interscapular region - in the type III.

There is manifestation of ischemia of other organs and systems: coronary insufficiency, stroke, or transient cerebrovascular accident, paraparesis or paraplegia, mesenteric thrombosis, acute renal failure, acute arterial occlusion of lower extremities.

Diagnosis. During diagnosis it is necessary to pay attention to the difference between the pulsations of the arteries of the upper or upper and lower limbs, signs of cardiac tamponade (paradoxical pulse, the weakening of the heart tones) and aortic valve insufficiency (diastolic noise).

Patient is admitted to ICU of vascular profile were following activities are performed:

– medical therapy;
– implementation of diagnostic studies;
– preparation for probable operation.

In patients with suspected aortic dissection central venous catheter of large diameter is introduced and infusion of β-blockers and vasodilators to slow down the rhythm and reduce blood pressure is quickly begun. The heart rate should range between 60-70 beats per minute, and systolic blood pressure must be maintained within 90-100 mm Hg. Introduction of the arterial catheter into the radial artery, catheter of Swann - Gantz into the pulmonary artery, ECG – monitoring, the bladder catheterization with determination of the hourly urine output, X-ray of the chest are performed. Collection of medical history and physical examination is carried out taking into account the neurological status and patency of arteries of limbs. The latter is important to select the type of connection of by-pass machine. Pain syndrome control is conducted taking into account heart rate and blood pressure, but if they are reduced to specified limits, but the pain persisted, then narcotic analgesics are injected. It is obligatory to provide oxygen inhalation, to determine blood group and to monitor the level of hemoglobin, hematocrit, electrolytes, gas composition and the basic biochemical parameters of blood. Avoid infusion of large amounts of fluids - the danger of raising blood pressure due hypervolemy.

Before starting the diagnostic activities differential diagnosis of acute aortic dissection and acute myocardial ischemia is needed. The final and rapid decision of this problem is vital in these patients, because immediate administration of thrombolytic in coronary insufficiency will lead to fatal outcome in patient with aorta dissection.

The purpose of diagnostic strategies in acute dissection is especially the identification of dissection membrane and its differentiation from intramural hematoma, i.e. confirm or exclude a diagnosis of the actual dissection. The second key moment in the diagnosis of acute dissection is to determine the localization process. In case of detection of the acute proximal dissection membrane operation is absolutely indicated: no additional diagnosis in the absence of complicating factors is needed, because the status of the aortic arch and the orifices of the coronary arteries can be adequately assessed intraoperatively.

Patients with unstable hemodynamics and the absence of an immediate effect on the primary drug treatment for suspected proximal dissection is transferred to the operating room, where, after the start of general anesthesia in conjunction with the preparation for surgery (connecting of monitoring lines) transesophageal echocardiography is performed. Upon confirmation of the diagnosis operation is performed. In the case of exclusion of diagnosis of dissection or in the difficulty of visualization

of the aorta after the stabilization of patient he can be transferred to the intensive care unit for further treatment or to other diagnostic rooms (CT, angiography).

The same transesophageal echocardiography is method of choice of diagnosis in relatively stable patients with acute aortic dissection. Next place in diagnosis is contrast-enhanced CT, spiral CT with three-dimensional reconstruction due to its high availability, speed, accuracy and the advantages. Application of MRI in acute aortic dissection is limited, although the accuracy of this method in diagnosis even is superior to transesophageal echocardiography. The use of MRI is possible in rare cases of acute dissection only in fully stable patients who are able to spend considerable time without medication support and monitoring. In addition, this technique is sometimes absolutely necessary in acute dissection to detect the condition of the aortic arch (failure of visualization of transesophageal echocardiography) in the case of suspected retrograde dissection in III and B-type of dissection.

Frequency of use of angiography in acute dissection is gradually decreased due to traumatic method in extremely severe patients. Nevertheless, angiography remains the method of choice for diagnosis of the state (patency) of branches of the aorta in the dissection. The need to perform coronary angiography in acute dissection is unclear. On the one hand, some authors believe that in 10-30% of patients with lesions of coronary arteries and proximal aortic dissection intraoperative assessment is sufficient to address the issue of one-stage revascularization of the myocardium. On the other hand, it was proved that the performance of one-stage coronary artery bypass in patients with proximal aortic dissection did not affect the intra-hospital survival, although long-term survival in patients with concomitant coronary artery disease without myocardial revascularization was significantly lower.

Indications for surgical treatment:

- in acute proximal aortic dissection emergency surgery is indicated because of the extremely high risk of aortic rupture or cardiac tamponade. In expressed cardiac tamponade and hypotension during the preparation for an operation to restore vital body functions pericardiocentesis with gradual drainage to prevent possible rupture of the aorta due to acute increase in blood pressure is indicated. Neurological deficit is not a contraindication for surgery, because most patients have its complete regression after the intervention;
- in chronic dissection of proximal aorta elective surgical intervention regardless of the size of the aorta, symptoms caused by enlargement of the aorta, complications occurred as a result of acute dissection or performed before surgery is indicated;
- in acute distal aortic dissection surgical treatment is indicated in the case of hypertension or pain refractory to drug therapy, recurrent pain, increase in aortic diameter over 5 cm, complications associated with impaired patency of the branches of the aorta (renal failure, paraparesis / paraplegia , ischemia of limbs and / or intestines), rupture of the aorta;
- in chronic distal aortic dissection operative treatment is indicated in patients with symptomatic disease, with the size of the aorta more than 5 cm and with an increase in the diameter of the aorta more than 1 cm per year.

Operations on the proximal thoracic aorta in degenerative diseases

There are two fundamental issues necessary for the planning of operational tactics during these interventions: how much is preserves sinotubulyar fissure and how severe are the aneurismatic changes of the aortic arch. If sinotubulyar fissure is preserved linear prosthesis of ascending aorta is sufficient, depending on the state of the aortic valve, supplemented by separate prostheses. In the reverse situation, with the expansion of the root of the aorta, aortic valve and the aorta ascending portion prosthesis by valve-containing conduit is indicated.

If the distal portion of the ascending aorta is of normal size or moderately dilated at an level of artery anonyma, direct distal anastomosis with the proximal portion of the arch is indicated. If aneurysmal expansion of the arch is greater use of deep hypothermia with circulatory arrest and retrograde perfusion of the brain is performed.

Linear prosthesis of ascending aorta

Intervention begins with midline sternotomy, the pericardiotomy on its entire length of anterior surface, its margins are taken on handles (Fig. 69). The by-pass machine is connected according to scheme: right atrium (or cava vein) - proximal part of the aortic arch (rarely femoral artery). Aorta is cross-clamped immediately proximally to the level of orifice of brachiocephalic trunk, the cavity of the aneurysm is dissected and the aorta is transected immediately above the upper attachment points of commisuras of aortic valve and proximally to aortic clamp. Full transection of aorta facilitates tight control of the anastomosis and reduces the risk of false aneurysm in the delayed period. The aortic valve is revised. The first is performed the distal anastomosis with a linear prosthesis by prolen thread 3-0 continuous suture, first the posterior, then the anterior wall are sutured.

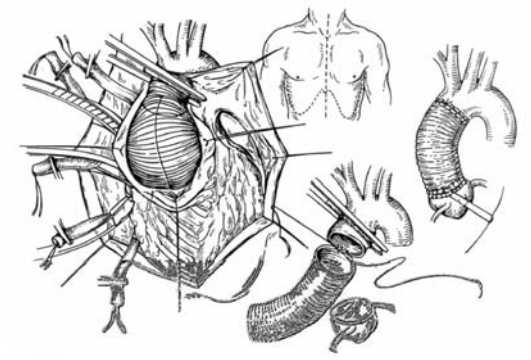


Fig. 69. Prosthesis of aortic valve and ascending aorta by valve-containing conduits in the modification of Svensson

Proximal anastomosis is performed in the same way. The patient is transferred to the Trendelenburg position, the air is evacuated, parallel artificial circulation (AC) is started, during which the control of tightness of anastomoses is performed. After the renewal of heart contractions and the gradual stabilization of hemodynamics in parallel the volume of perfusion is reduced, and then the aorta is decannulated. Next right part of heart is decannulated, hemostasis is checked and prosthesis is covered by remnants of aneurysmal sac. Pericardiotomy opening is closed by patch. Drain is inserted into the pericardial cavity and anterior mediastinum through counterpuncture below the xiphoid process. The margins of the sternum are approximated by steel wire. The sternum is completely closed by musculo-fascial sutures and the skin is sutured.

Separate prosthesis of aortic valve and ascending aorta is indicated in patients with combination of aortic valve defect and aneurysm of ascending aorta without increasing of aortic ring. After opening of the cavity of the aneurysm and the tran-

section of the aorta, as described above, aortic valve cusps are excised and prosthesis is implanted with the standard technique. Further techinque of the operation is the same as the linear prosthesis of ascending aorta. If you have more severe annular dilation, especially in elderly patients, it is possible to use technique developed by M.N.Wheat (1964), when transection of the proximal aorta is performed immediately above the fibrous ring, the walls above the orifices of the coronary arteries are left. In this situation, initially proximal anastomosis is imposed with linear prosthesis, so that the orifices of the coronary arteries are incorporated into the anastomosis and hiding them. To reinforce the proximal anastomosis aortic valve prosthesis is taken into sutures.

Prosthetic of aortic valve and ascending aorta by valve-containing conduits

It is indicated in patients with expansion of aortic valve ring, sinotubular fissure or Valsalva sinuses. Since the beginning of the AC, clamping of the aorta and cardiac arrest ascending aorta is transected in the distal part, aortic valve is excised with leaving of 1-2 mm rim of fibrous ring, stitches are put on the commissures and the diameter of the valve ring is measured to match conduit.

Further tecnique depends on the choice of method of intervention. There are three classic methods of prosthesis of ascending aorta by valve-containing conduits which differs by techniques of inclusion of the coronary arteries into blood flow.

In the method of Bentalla De Bono orifices of the coronary arteries directly, in its natural position in the wall of the aneurysmal sac, are anastomosed with the openings in the wall of the conduit.

It was also proposed to cut out the orifice of the coronary arteries on areas and to produce mobilization of the primary portions of the coronary arteries that allows to increase the length of the vessels and replant them easily into the wall of the aortic prosthesis.

The third method is the classical technique of Cabrol, when the orifice of the coronary arteries is anastomosed with 8 - or 10-millimeter linear prosthesis, which is sutured side-to-side to the conduit. Initially, the last anastomosis was placed on the anterior surface of the aortic prosthesis, then the technique has been improved and anastomosis is performed to the posterior surface of the conduit.

Cabrol technique greatly facilitates access to coronary anastomosis and suture line on the valve ring when the additional hemostasis is needed, but subsequently due to the side-to-side anastomosis between the prosthesis to the coronary arteries and conduit it is high probability of inflection of the first prosthesis up to the development of occlusion of any of the branches to the coronary arteries.

To resolve the above disadvantages L. G. Svensson (1997) proposed to include into the blood flow the left coronary artery by prosthesis into conduit, and the right coronary artery is replanted on the area (Fig. 70). The latter method allows you easily to visualize all anastomosises, eliminates the risk of coronary branch inflection, makes it possible complete transverse transection of the aorta (no need of wrapping of conduit for

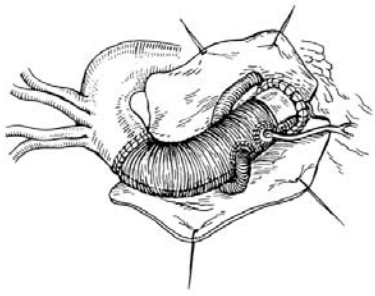


Fig. 70. Linear prosthetic of ascending aorta

reliable hemostasis), which reduces the risk of false aneurysms of anastomosises in the long run.

Aortic arch prosthetics

After the femoral artery cannulation a longtitudinal sternotomy is performed, v.cava is cannulated, the cooling of patient starts and the extracorporeal (artificial) circulation begins (Fig. 71).

The aortic arch is exposed along the anterolateral surface close to the adventitia in order not to damage n.n. vagus, diaphragmaticus laryngeus recurrens and preserve the integrity of the left parietal pleura. During the stage of cooling an end-to-side anastomosing between the linear 10-mm prosthesis and aortic prosthesis is performed for the arterial perfusion. The patient is moved to Trendelenburg's position and the aortic lumen is opened 1 cm above the beginning of brachiocephalic arteries. Total aortic transection is carried out between the ascending aorta and aortic arch (Fig. 71a).

If the beginning of descending aorta is involved in aneurism, this part is transected below the aneurism and an end-to-end anastomosis is created between the descending aorta and aortic prosthesis. Then aortic branches are resected on one area and reimplanted to the prosthesis (Fig. 71b).

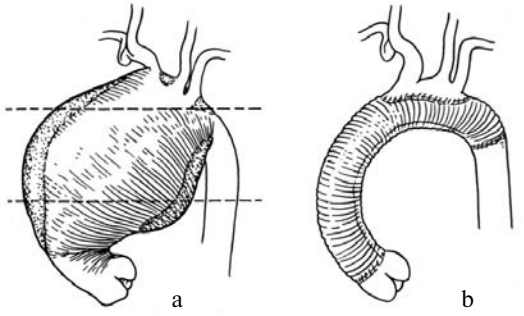


Fig. 75. An ascending aorta and aortic arch prosthetics in a patient with aneurism extending to the aortic isthmus: a — aneurism site; b — design of operation.

Operations on a proximal thoracic aorta at acute aortic dissection

At any acute dissection extending to the ascending aorta, after the connection of extracorporeal circulation device the patient is cooled down to a state of the profound hypothermia, a circulation is stopped and a retrograde brain perfusion begins.

At dissection of an ascending aorta with transition to an arc, associated with the significant intimal tears in aortic arch or its dilating, an aortic arch prosthetics is required. In this situation aorta is intersected at once behind the left subclavian artery, and a distal anastomosis is imposed with the linear aortic prosthesis. Brachyocephalic arteries are resected on the same area. Stitches are reinforced with teflon stria stacked in an oval shape around the anastomosis area. The created area with orifices of aortic branches is replanted to the aortic prosthesis with the continuous sutures.

At the dissecting lesion of the aortic valve (especially at its extension to the beginning of aorta or interventricular septum, presence of low fenestration, congenital or acquired defects of the aortic valve, congenital diseases of a connecting tissue) the prosthetics of the aortic valve is indicated.

Principles of carrying out of operations on descending thoracic and thoraco-abdominal aortic segments

Crossclamping of aorta and stopping of blood circulation in descending thoracic and thoraco-abdominal segments do not demand extracorporeal circulation. Nevertheless there is an actual problem of an acute systolic left ventricular overload prevention at the moment of aortic crossclamping and protection of organs and tissues supplied by branches of an aorta departing below the crossclamping level from ischemic damage. The most effective measures to reach this goal are following: an auxiliary artificial circulation according the scheme "left departments of heart - femoral artery", monitoring of cerebrospinal fluid pressure and its drainage, crossclamping of aorta in the condition of a moderate hypothermia.

Monitoring principles at operations on distal aorta are similar to the featured above for interventions on proximal segments of aorta.

Posthetics of a descending thoracic aorta at degenerative diseases

The patient's position – edgewise, shoulders are turned anglularly (80-90°), and a pelvis - about 60° to a table. Left femoral artery is exposed first. At operation on a proximal portion of a descending aorta an incision is made in the fourth or fifth intercostal space from the edge of a breastbone to the inner edge of scapula, bending around a lower edge of the scapula, with the subsequent resection of the V^{th} rib. At a lesion of a distal portion of an aorta the same incision is made in the fifth or sixth intercostal space with a resection of the VI^{th} rib, and after the subsequent transection of a costal arch the dissection of a diaphragm is started. After the opening of pleural cavity one-lung ventilation is started, an aorta is bypassed above a prospective place of proximal anastomosing and connected to the device for auxiliary artificial circulation. For the better mobilization of aorta the arterial ligament is transected. The aorta is crossclamped between the left carotis and subclavial arteries and the left subclavial artery is clamped separately. A distal clamp is put on aneurysmal sac below the place of a proximal anastomosis applying. The aorta is intersected proximally form the aneurysm, displaced from esophagus and an "end-to-end" anastomosis is applied with an aortal prosthesis. At this stage of operation it is necessary not to traumatize vagus, laryngeal recurrens and phrenic nerves. It is very important not to trap an esophageal wall in the stitches on the back wall of an aorta. Such technical error can lead to formation of an aorto-esophageal fistula. After the finishing of a proximal anastomosis a prosthesis is clamped below it and a blood flow in the left subclavian artery is recovered.

A distal clamp on aneurysmal the sac is moved on an intact area of descending aorta. The remaining portion of an aneurysmal sac is opened, orifices of intercostal arteries are oversewn. An aorta is then transected over a clamp and a distal direct "end-to-end" anastomosis is applied (Fig. 72).

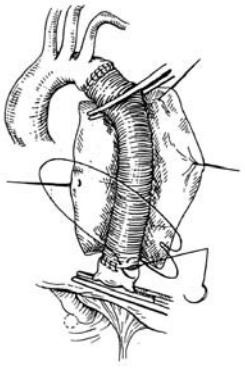


Fig. 72. Prosthetics of a descending thoracic aorta.

Prosthetics of thoraco-abdominal portion of aorta at degenerative diseases

The position of a patient – edgewise with shoulders turned anglularly (80-90°), and a pel-

vis - about 30° to the table. The left femoral artery is exposed. The incision line runs from a point between p.spinosus and medial surface of a scapula bending around the inferior angle of scapula, extends frontally to the VI-VIIth intercostal space, and further goes to an umbilicus or pararectally downwards to a pubis.

After the connection of atrio-femoral bypass a proximal anastomosis for types I and II of thoraco-abdominal aneurysms is formed on descending thoracic aorta. At aneurysms of types III and IV a proximal anastomosis is imposed to medial or to the inferior third of descending thoracic aorta. Aorta then is clamped over a celiac trunk, its lumen opened and surgeons spot the amount and location of orifices of intercostal arteries which should be replanted to the prosthesis. Routinely the replanted arteries are located at a level of Th_{VII}-L_{II}. When this is done, a clamp on aortic prosthesis is removed and the perfusion of spinal cord recovers. An aortic clamp then is moved to the infrarenal segment and the lumen of supra- and infrarenal aorta is opened. At aneurysms of type I and rarely III of thoraco-abdominal aorta the specified "end-to-end" anastomosis is imposed with an aortal prosthesis. At types II, IV and in the rest of cases of type III aneurysm of thoraco-abdominal aorta the anastomosis is formed with a lateral window of prosthesis, and a distal "end-to-end" anastomosis is imposed with the infrarenal aorta. The left renal artery is replanted to the prosthesis on the separate place after blood flow renewing in the right renal artery and visceral branches (Fig. 73).

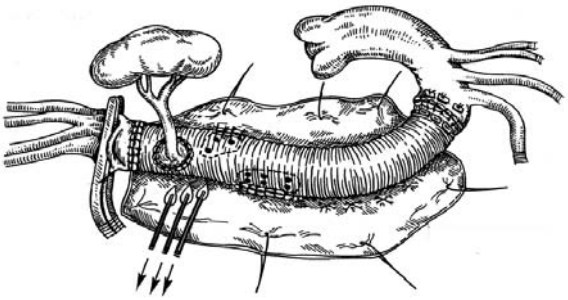


Fig. 73. Prosthetic repair of thoraco-abdominal aorta

Operations at acute distal aortic dissection

Basic principles of performance of such operations are identical to mentioned above. The indication to reconstruction of thoraco-abdominal aorta is big diameter of aorta in abdominal portion or disturbance of function of the vital organs. The descending thoracic aorta (with pre-established atrio-femoral bypass) is intersected below a subclavian artery. Stratified intimal and adventitious layers of aorta are sewed with continuous suture as at proximal acute dissection. After that proximal and distal anastomoses with the aortal prosthesis are formed.

Operations at chronic distal aortic dissection

Operations are performed in respect of the same principles, as at aneurysms of descending thoracic and thoraco-abdominal aorta. If the intercostal arteries demand replantation or visceral and renal arteries are separated with a stratified membrane in a place of anastomosing then its excision is required. At carrying out of the second stage of an intervention by an "elephant trunk" technique after the connection of atrio-femoral bypass a crossclamping of a descending thoracic aorta is carried out. Then a descending aorta is dissected over a clamp below a suture line on the aortic isthmus and distal end of proximal prosthesis is clamped. Further operation continues as mentioned above with one exception - there is no necessity in intersecting of a descending thoracic aorta in a proximal segment.

Endovascular interventions at aortic aneurysms and dissection

A development of endovascular techniques of treatment of cardiovascular diseases during the last decade has led to attempts of using of intravascular technologies, namely – an endoprosthetics with stenting, in the surgery of thoracic aorta aneurysms and dissection. Application of endovascular prostheses fixed to the aortic wall by stents for closure of a proximal windowing now is considered to be challenging at acute distal dissection as well as the use of stents for restoration of passability of branches of abdominal aorta.

The main problem of descending thoracic aorta stenting is a leakage of endoprostheses – it happens in 12-53% of all cases.

Ruptured aneurysm of thoracic aorta

The clinical signs are following: a collapse happens often before a preceding or persisting pain. Other symptoms depend on rupture localization. Aneurysms of ascending aorta may cause a massive hemopericardium. A bleeding in a mediastinum is possible at aneurysm of both ascending portion and an aortic arch. A rupture localized in a descending portion or aortic arch result in hemothorax or massive esophageal or pulmonary bleeding. Small relapsing vomiting by scarlet blood can precede extensive outbreak from esophagus or tracheal lumen.

Ruptured thoraco-abdominal aneurysms cause bleeding to the abdominal cavity or retroperitoneal space, rarely - to the stomach or duodenum that manifests with bloody vomiting.

The most infrequent localization of an aneurysm rupture of ascending aorta and thoraco-abdominal portion is an outbreak to the superior or inferior vena cava. The clinical presentation in these situations is characterized with a fulminant heart failure.

The algorithm of diagnosis and management is similar to activities at acute dissection of aortic aneurysm.

Lethality at ruptured aneurysms of thoracic aorta reaches 94% (according to Bickerstaff L.K. et al. 1982).

The list of diagnostic and therapeutic manipulations:

– Auscultation; a radiopaque aortography

Complications of aortic aneurysms:

– a rupture of aortic aneurysm – demands an urgent operative treatment;
– ischemic stroke (aortic arch aneurysm) - a conservative therapy or surgical treatment.

Disability examination and prophylactic medical examination of patients with thoracic aorta aneurism. Patients with persisting aortic aneurysms have an unfavorable outcome. Almost all patients die within 3 years after the establishment of the diagnosis from aneurysm rupture. Operated patients undergo a dispensary observation by the vascular surgeon.

Particularities of examination of the patient with a supposed aneurysm of abdominal aorta

Questioning of the patient:

1). Complaints concerning a basic disease:

The clinical pattern of an uncomplicated abdominal aorta aneurysms is various and depends on the character of a basic disease, location and dimensions of dilating,

involving in process of visceral and renal arteries. It is important that the majority of patients with aortic aneurysm are asymptomatic.

Uncomplicated aneurysms are characterized by a classical triad of symptoms: an aching abdominal pain, presence of pulsing formation in an abdominal cavity and systolic bruit over it. The abdominal pain is the most frequent (90%) complaint. Routinely it is aching and is located in mesogastrium to the left from the umbilicus, sometimes with an irradiation to the lumbar, inguinal range or the inferior extremities. The abdominal pain is caused by a pressure of aneurysm on neuroplexes and spinal cord roots that is quite often regarded as implication of neurologic, urologic or abdominal diseases. Increase of pain indicates at enlargement of aneurysm and a threat of rupture, however even at major aneurysm an abdominal pain may be absent. A frequent symptom (40%) is the sensation of the abdominal pulsation and distension.

A disease may be presented with several syndromes:

1. Sciatic or radicular syndrome. It is caused by a compression of spinal roots in lumbar department therefore causing back pain with an irradiation downward, sensitive and locomotory disorders of the inferior extremities.

2. The abdominal syndrome is manifesing with eructation, vomiting, unstable stool, decreasing of appetite and a weight loss. It is caused either with a duodenal compression or with the involving of visceral branches of an abdominal aorta in the pathological process.

3. The urologic syndrome: a pain or distention in lumbar area, hematuria, dysuria. It is a result of disturbances of urinary flow owing to compression of pelvis or ureter.

4. The syndrome of a chronic lower limb ischemia arises at involving in pathological process of iliac and femoral arteries. Thus an intermittent claudication, rest pain and trophic disturbances of the inferior extremities are possible.

2) *The disease anamnesis*: in most cases of development of aotic aneurysm is slow, the symptomatology increases gradually. The disease is progressing, however, there are cases of acute onset of disease.

3) *The life anamnesis*: a presence of congenital diseases (Marfan's syndrome, fibromuscular dysplasia), and acquired (atherosclerosis, syphilis, rheumatic disease, tuberculosis), and also an abdominal trauma is defined, as well as the information about the undergone operations on aorta.

Clinical physical examination of patient:

1) An evaluation of general condition of patient

Consciousness as a rule is clear. Constitutional features – ectomorphic body building.

2) Examination of cardiovascular system: a palpitation increasing at physical exercise stress is characteristic. A tachycardia is permanent, does not variate at body postural change, persists while sleeping.

3) Examination of respiratory organs: no changes.

4) «***Locus morbi***»: Pulsing formation in the abdominal cavity is routinely spotted in a medial or upper half of abdomen, more often to the left. It has dense elastic consistence, spherical or oval in shape, has fixed position and usually painless. The pulsing waves of aneurysm are transmitted extensively. It is necessary to realize that

if aneurysm can be revealed by palpation it usually has significant dimensions and the probability of its rupture is high.

At auscultation of abdomen the third basic symptom is found in 75% of patients - a systolic bruit. It is caused by a turbulent blood flow and oscillation of the aneurysm walls, but also can be a result of stricture formation in its visceral branches.

5) Leading clinical symptoms of **aneurysm of abdominal aorta** are: ***abdominal pain*** and the ***presence of pulsing formation***.

6) On the basis of the gained data the following clinical diagnosis is established.

The aneurysm of abdominal aorta

Classification of aneurysms of the abdominal aorta (A.A. Shalimov, N.F. Druk, 1979):

I. Infrarenal

II. Suprarenal

III. Infrarenal and suprarenal forms are:

1) asymptomatic

2) typical uncomplicated

3) aneurysm associated with occlusive process in the branches of the aorta

4) aneurysm, complicated by dissection, or rupture

Surgical classification of abdominal aortic aneurysms (V. I. Burakovsky et al., 1989):

I. Suprarenal aneurysm isolated, diffuse without engaging in the process of bifurcation of the abdominal aorta and diffuse with involvement in the process of bifurcation of the abdominal aorta.

II. Infrarenal aneurysm without involvement in the process of bifurcation and involvement in the process of bifurcation of the abdominal aorta.

III. Suprarenal and infrarenal forms are:

1) uncomplicated

2) complicated

Complicated forms of abdominal aortic aneurysms:

1) Part rupture - a tear or dissection of wall of the aneurysm with formation of subadventisial hematoma, sometimes with distal fenestration of bifurcation

2) Complete rupture with hemorrhage in the retroperitoneal space, free peritoneal cavity, the organs of the gastrointestinal tract and the system of the inferior vena cava (directly into the trunk of the inferior vena cava, the left renal vein, the right iliac vein).

Classification of A.V.Pokrovsky (1968):

Etiology:

1. Congenital.
2. Acquired:
   a) inflammatory (specific and nonspecific)
   b) non-inflammatory (atherosclerotic, traumatic).

Morphology:

1. True.
2. False.
3. Dissecting.

In form:

1. Saclike.
2. Diffuse.

On clinical course:

1. Uncomplicated.
2. Complication (rupture).
3. Dissecting.

Localization:

Type I — aneurysm of the proximal segment of the abdominal aorta involving visceral branches.

Type II — aneurysm of infrarenal segment without involvement of the bifurcation.

Type III — infrarenal segment aneurysm involving the bifurcation of the aorta and iliac arteries

Type IV — total lesion of the abdominal aorta.

According to standards of examination and management patients with aneurysms of abdominal aorta have to unedrgo:

1. Clinical analysis of blood.
2. Coagulation tests.
3. Ultrasonic examination.
4. X-ray examination.
5. Computed tomography. 6. Angiography.

1) Clinical analysis of blood: changes are non-specific: the anaemia, a leukopenia against a lymphocytosis, the ESR is slightly increased.

2) Coagulation tests - a hypercoagulation phenomena.

3) US: ultrasonic methods of examinations, especially duplex scanning, are available, safe and economic, allow to make fast screening of the patient and to gain data concerning location and dimensions of aneurysm, a state of its walls, presence of thrombi. Duplex scanning gives an opportunity to differentiate an aneurysm from the aortic deviation, fused kidney or retroperitoneal tumor etc.

4) The X-ray: at suspicion concerning abdominal aortic aneurysm an anteroposterior or lateral X-ray examination of abdomen can be utilized. A shade of aneurysmal the sac is found together with the falciform calcification of its wall. Sometimes an erosion of bodies of II-IV of lumbar vertebrae can be revealed.

5) The computed tomography gives a high-precision information about the structure of aneurysm, an extrinsic and intrinsic contour, presence of initial intimal tears or thrombothic masses. It allows to estimate a state of nearby organs and anatomic formations. The examination is done before and after the introduction of contrast medium with the subsequent computed reconstruction of the image of an aorta that gives a possibility to evaluate a syntopy, changes of its dimensions and configuration and important morphological details (thrombi, calcification, initial teares, dissection, etc).

6) Angiographic examination. The radiopaque angiography now is rarely utilized in diagnostics of abdominal aorta aneurysms. At analysis of angiographic patterns it should be clear that the diameter of aorta above 3 cm is abnormality. As the aneurysmal sac often contains thrombotic masses, the dimensions of aneurysm seen on aortogram in most cases is less than real one and in 13% of patients the aortic lumen seems to be normal. The basic angiographic sign of aneurysm dissection is double contour of aorta.

***Differential diagnosis*:**

Tumors of stomach, pancreas and masses of lymph nodes, located near the aorta, can be pulsing and feign an aneurysm. However the tumor routinely has the dense consistence, an uneven surface, not mobile and has no extensive pulsing. A tumor does not produce any pathologic sound. Ultrasonic, radiological and endoscopic examination of stomach and duodenum or laparoscopy are carried out in doubtful cases.

Misdiagnosis is possible in the presence of a tumor or a fused kidney, and also at nephroptosis and floating kidney when it is located close to aorta and is pulsing together with it, however the kidney can be dislocated at palpation and any sound over it is absent. Ultrasound, intravenous excretory urography, scintigraphy and angiography help to make the correct diagnosis.

The major lymphosarcoma of a mesentery of small intestine can be erroneously assumed as abdominal aortic aneurysm. Ultrasound and CT can facilitate diagnostics.

The deviation of an abdominal aorta quite often revealed in hypertensive patients can be taken for an aneurysm. The correct diagnosis is established with the help of ultrasound and CT.

Substantiation and formulation of the clinical diagnosis (taking into account classification of disease, presence of complications and concomitant diseases):

1) basic disease - an aneurysm of abdominal aorta;

2) complications of a basic disease (if they are present);

3) concomitant diseases

Treatment of the patient with aneurysm of abdominal aorta

***A choice of treatment policy*:** all patients with aneurysms of abdominal aorta are treated by surgery only.

Indications and tactics of surgical management of abdominal aortic aneurysms

Therapeutic methods of treatment of patients with abdominal aortic aneurysms are not used. At revealing of disease the patient should be referred to a specialized vascular surgery department. At abdominal aortic aneurysm an early operation is indicated before the development of various complications.

Elective surgical management of patients with abdominal aortic aneurisms is contraindicated in the presence of recent disturbances of coronary or cerebral circulation, the expressed compromise of liver or kidneys function, circulatory insufficiency of grades IIB or III. However 3 months after a myocardial infarction and 6 weeks after the stroke it is possible to operate under condition of a stable hemodynamics and the absence of serious neurologic problems. Old infarctions or strokes do not present a contraindication to surgery.

At the threat of dissection or already dissected aneurysm of an abdominal aorta surgery is a single way of saving patient's life and should be performed urgently under any circumstances.

Surgical management

A modern technique of operation on infrarenal aortic aneurysm is a resection of aneurysm with the subsequent prosthetics of aorta by a linear or bifuractional synthetic graft.

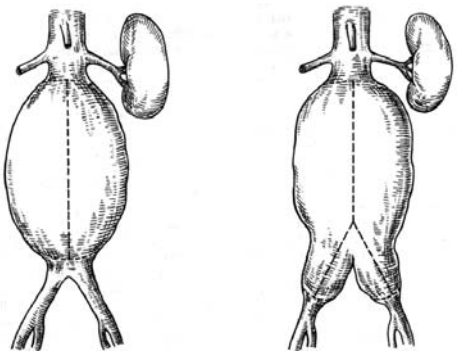
A complete excision of aneurysmal sac is dangerous due to damage of inferior v. cava, iliac veins, ureter etc. Therefore such procedure is not applied generally.

At infrarenal aneurysms the optimal surgical access is a complete median laparotomy allowing a wide exposure of all infrarenal aorta and iliac arteries. In selected cases an extraperitoneal or pararectal access can be used. A thoracophrenolumbotomy is indicated in patients with aneurysms of types II and IV.

A techniques of an infrarenal aortic aneurysm resection with the prosthetic repair in the sac cavity

After the complete median laparotomy a rear peritoneum and Treitz's ligament are dissected. Small intestine and duodenum are dislocated to the right and upwards. The aorta is exposed proximally from aneurysm as well as its terminal portion and iliac arteries. After the intravenous infusion of 5000 U of heparin the aorta is clamped above the aneurysms under the control of arterial pressure.

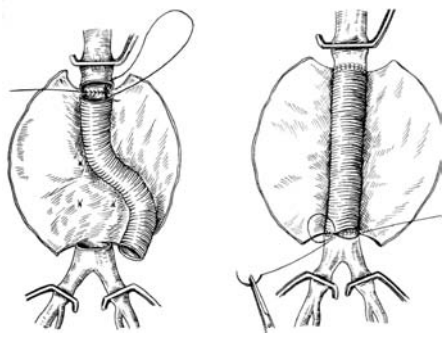
The inferior mesenteric artery is clamped. After the crossclamping of iliac arteries an aneurysmal sac is opened widely along the forward wall with the removal of thrombotic masses and stitching of orifices of lumbar arteries from within. At the proximal end of incision the lateral wall of aorta is dissected (Fig. 74). A back wall of aorta is left intact.



A B

Fig. 74. An incision of a forward wall of aneurysmal sac. A - an incision of a forward wall of aneurysmal sac at intact iliac arteries; B - a shape of incision at involving of common iliac arteries

Depending on the extent of aneurysm a terminal aortic portion or common iliac arteries is dissected. A linear or bifurcational prosthesis is anastomosed with an aorta from the side of aneurysm cavity with a monofilament continuous suture (Fig. 75).



C D

Fig. 75. Anastomosing. C - applying of a proximal anastomosis. D - applying of a distal anastomosis

At a lesion of iliac arteries the ends of bifurcational prosthesis are moved to thighs and an "end-to-side" anastomosis is applied with femoral arteries. After the restoration of blood flow along the inferior mesenteric artery the wall of aneurysmal the sac is partially excised and sewed over a prosthesis for its isolation from the intestine (Fig. 76). A retroperitoneal space is drained through a counteropening.

One of new methods of treatment the abdominal aortic aneurysm is a remote endovascular prosthetics of aorta with a graft introduced through a femoral or iliac artery and fixed by stents. Such interventions reduce operation risk for serious patients and to gain good results.

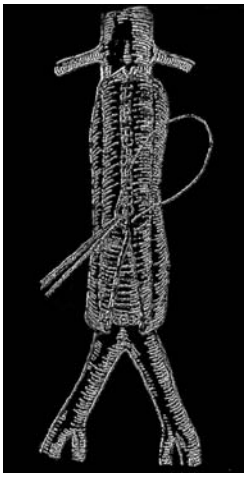


Fig. 76. Suturing of aneurysmal the sac over the aortic prosthesis

Postoperative complications:

– an intracavitary bleeding;
– a dynamic intestinal obstruction;
– thromboembolic episodes;
– acute cardiopulmonary insufficiency.

Urgent conditions

Abdominal aortic aneurysms may complicate with rupture (complete or incomplete) and thrombosis of aneurysmal sac. The aneurysm rupture is the most frequent and serious complication and in the absence of urgent surgery results in a hundred-percent lethality.

The incomplete rupture is a slight tear of an aneurysm wall with the formation of a subadventitious hematoma. An incomplete rupture of aneurysm always converts to complete and this is a natural outcome of the disease.

The clinical presentation of complicated abdominal aortic aneurysm is manifold and depends on defect dimensions and location, but at all variants the basic clinical sign is the pain syndrome. Intensifying of pain against the appearance of the considerable painfulness can be a precursor of rupture. Usually patients with an incomplete aortic rupture suffer from acute pain without signs of collapse or increasing anaemia. The pain is located in the middle of abdomen, more often to the left, also irradiates to loin, inguinal range and perineum. A pulsing formation producing a systolic bruit is spotted in the abdominal cavity.

In the majority of patients aneurysms are breaking to the retroperitoneal space (65-85%) or to the duodenum (26%) and to the abdominal cavity (14-23%) and rarely - to the inferior vena cava. It is very important to realize that the majority of patients with abdominal aortic aneurysm rupture do not die immediately. Only 13% of them perish within first 6 hours, 45% survive during 24 hours, 36% live from 1 to 6 days, and 6 % - even to 9 days from the moment of rupture.

The presentation and clinical course of ruptured abdominal aortic aneurysm depend on location of wall defect. The typical a triad of signs is ***pain***, ***pulsing formation*** in the abdomen and ***hypotension***.

At outbreak of aneurysm (Fig. 77) to the retroperitoneal space patients note sudden appearance or intensifying of abdominal or loin pain. The pain location and ir-

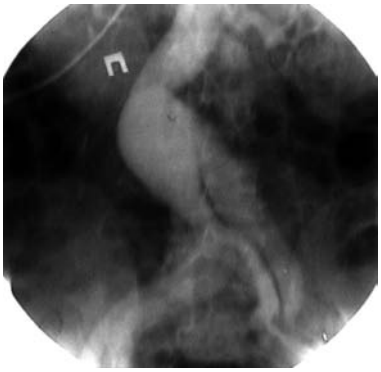


Fig. 77. Outbreaking of abdominal aortic aneurysm

radiation depends on the direction of hematoma extension. So, if the formed hematoma spreads from top to bottom and reaches a small pelvis, the pain irradiates to the inguinal area, hip and genitals. At a high location of hematoma pain irradiates upwards, often to the heart area. It is important that in most part of patients the intensity of a pain syndrome mismatches with data gained at physical examination of abdomen. The signs of peritoneal irritation are absent or slightly positive, the abdomen is distended and almost painless at palpation.

Signs of an internal bleeding of various degree play a significant role in the establishment of the diagnosis of an abdominal aortic aneurysm rupture. At the majority of patients the hemorrhagic syndrome is expressed moderately as the bleeding to retroperitoneal space is comparatively slow. The acute implications of a hemorrhage (collapse and loss of consciousness) are observed only in 20% of patients.

Extensive haematomas in lateral abdominal, inguinal and femoral areas serve as late signs of the bleeding.

It is important to emphasize, that in the majority of patients with a ruptured abdominal aortic aneurysm the correct diagnosis is possible just on the basis of routine clinical data: 80% of patients suffer from abdominal and loin pain, collapse and pulsing formation in the abdominal cavity; in 65% of cases the prompt augmentation of the dimensions of formation is observed; 70% of patients present an anaemia.

At an **intra-abdominal rupture** when symptoms of an acute bleeding promptly educe, all patients complain of acute abdominal pain, nausea, vomiting, serious collapse. A paleness of the patient and cold sweat also attract attention. The pulse is frequent, thready, the arterial pressure is low. The abdomen is distended, painful in all areas, symptoms of peritoneal irritation are expressed. A free fluid is located in the abdominal cavity. Owing to a hypotension an oliguria or anuria is routinely observed.

The aneurysm outbreak to the organs of gastrointestinal tract is usually located in the duodenum. The basic symptom of this complication is a sudden sharp abdominal pain in the epigastrium or mesogastrium. Signs of a profuse gastrointestinal bleeding are also expressed: a fulminant collapse and hematemesis. Later symptoms are tarry stool and anaemia. At physical examination of patient a painful pulsing formation in the abdominal cavity with the systolic bruit over it is found.

At aneurysm outbreak (Fig. 81) to the inferior vena cava patients usually complain of dyspnea, palpitation, edema of the inferior extremities, pain in the inferior half of the abdomen and the presence of pulsing formation inside. The progressing heart failure is accompanied with the enlargement of liver and edema of lower extremities. A pathognomonic sign is a sudden appearance of rasping bruit which is conducted along the venous blood stream and a tremor like a "fremissemet cataire" ("purring thrill"). The specified symptoms steadily progress, result in a serious heart failure and death of patients in some days after the appearance of the first one.

Chronic clinical course of aortic dissection is observed in 10-20 % of patients.

Diagnosis and differential diagnosis

The exact knowledge concerning clinical symptomatology and careful examination of patients allow to establish a correct diagnosis of the complicated abdominal aortic aneurysm in most cases. Nevertheless, about 60% of patients with this disease are operated for erroneous indications and a considerable part of patients perish in therapeutic, neurologic and other departments from unrecognized complications. Often an acute pancreatitis, intestinal infarction or obstruction, myocardial infarction, renal colic or acute radiculoneuritis are incorrectly diagnosed.

Special examination techniques among which non-invasive procedures are preferable help to establish an exact diagnosis. Highly informative are ultrasonic scanning and computed tomography which do not demand a special preparation of patients. In some cases a radiopaque aortography may be a helpful examination (for the evaluation of state of visceral branches of aorta), however in serious condition of the patient it is not used.

A magnetic resonance tomography, made in traditional and angiographic modes, allows to get a dimensional image of an aortic aneurysm and its branches by building-up of multivariate reconstruction of vessels and gives an opportunity to choose an optimal surgical tactics.

A laparoscopy revealing blood in abdominal cavity or hematoma of a mesentery and retroperitoneal fat may help to make a correct diagnosis in some cases of complicated aneurysm.

At abdominal aortic aneurysm rupture an immediate surgery is a single way to save the patient. Its success depends on observance of the following requirements:

1) in the presence of a hemorrhagic shock before the operation it is not necessary to emphasize on complete restoration of systolic arterial pressure above 80 mm hg;

2) for the same reason it is necessary to exclude all actions resulting in decrease of the intra-abdominal pressure promoting a bleeding (a gastric lavage, cleansing enema, a urinary bladder catheterization);

3) introduction to general anesthesia and intubation of the patient (without relaxants!) is possible only after the complete readiness of surgeons for an emergency laparotomy and hemostasis as general anesthesia leads to abdominal muscle relaxation and may increase bleeding.

Operation at a retroperitoneal rupture of abdominal aortic aneurysms is technically identical to elective operations. The primary goal is a bleeding arrest by cross-clamping of aorta above aneurysm and then a complete program of actions for homeostasis restoration is performed.

On occasion for a temporary stopping of a bleeding a thoracic aorta is clamped over a diaphragm through a separate incision. A further surgical intervention consists of resection of aneurysm and prosthetics of aorta with a graft inside the sac (Fig. 78).

***The list of medical diagnostic and therapeutic manipulations*:**

– radiopaque aortography (catheterization using Seldinger's technique).

Diagnostics and treatment of postoperative complications:

– After the operation a patient is left on assisted ventilation for several hours. Extubation is made under condition of complete stabilization of homeostasis and normal gas exchange.

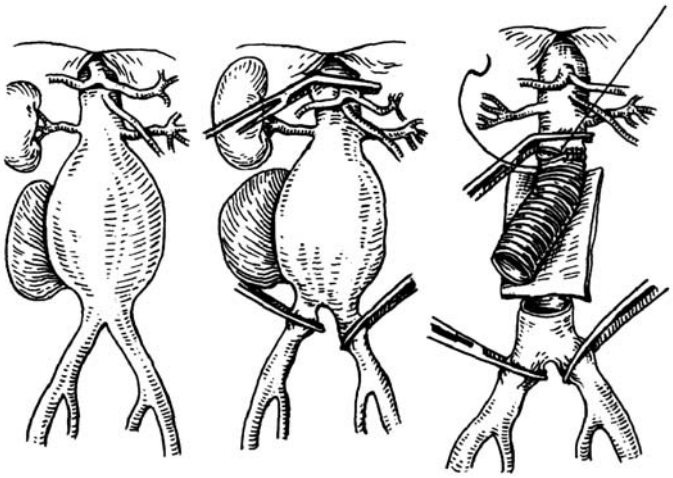


Fig. 78. Operation at retroperitoneal abdominal aortic aneurysm rupture.

It is essential to avoid using heparin because of probability of extensive hematomas formation, increase of intoxication and renal failure.

Examination of disability and prophylactic medical survey of patients with aortic aneurysm.

Patients with aneurysms of abdominal aorta have an unfavorable prognosis without surgery. Survival rate of the operated patients is five times greater than of those who avoided surgery. The majority of operated patients return to normal life and their life expectancy is almost the same as in other people of their age. Patients should undergo a dispensary survey by the vascular surgeon after surgery.

Chapter VI. Surgical pathology of the venous and lymphatic systems

VI.1. Varicose veins of lower extremities

The varicose veins of legs (varicose illness) represents diseases of a venous wall and valves which lead to chronic venous insufficiency. According to the literature, 10 - 17 % of population of people is ill varicose illness. The ratio of men and women makes 2 : 4. More frequent the left leg is affected and also the great saphenous vein did in comparison with the lesser (short) saphenous vein.

The following classification of varicose illness{disease} is used in clinic:

1. By the etiological factor: a primary and secondary the varicose veins of legs;
2. By an anatomical principle recognize dilatation:
   – V. Saphena magna; the great saphenous vein (GSV).
   – V. Saphena parva;
   – the small saphenous vein (SSV).
   – vv. subcutaneus;
3. Under the form of varicose dilatation recognize following forms:
   – Cylinder;
   – Sinuous, scattering;
   – mixed;
4. Stages of development of disease:
   – Compensation;
   – Subcompensation;

Decompensation;

5. Under the expression of disturbances of a venous circulation recognize I, IIA, IIB and III degrees;
6. Under the state of the valval apparatus of veins recognize: without disturbance of functions of valves; with a failure of valves;
7. Under the character of complications:
   – Complicated by bleeding;
   – Complicated by thrombophlebitis;
   – Complicated by ulcer of crus.

It is necessary to remember factors which provide a normal venous hemodynamic. The venous system of the inferior extremities consists of two rather isolated systems, where the fascia is an interface: superficial or subcutaneous veins are placed over fascia and deep veins are placed sub- fascia, connection between which is carried out by communication (perforated) veins. Feature of veins of the inferior extremities is presence in them of venous valves which oppose retrograde blood flow and support it to the heart. Propulsion of the blood is promoted also by heart beats, by reduction of muscles of shin (a muscular-venous pomp) and femur, sucking action of a thoracic cavity. Also the important role belongs to a tonus of venous wall. In norm the blood flows from periphery to the center, and on communication veins - from superficial in deep. The causes of the varicose veins of legs are precisely unknown. Following theories are offered: genetically, endocrine, neurotrophic, mechanical, different physiologic and pathological factors which promote rising of intra-abdominal pressure.

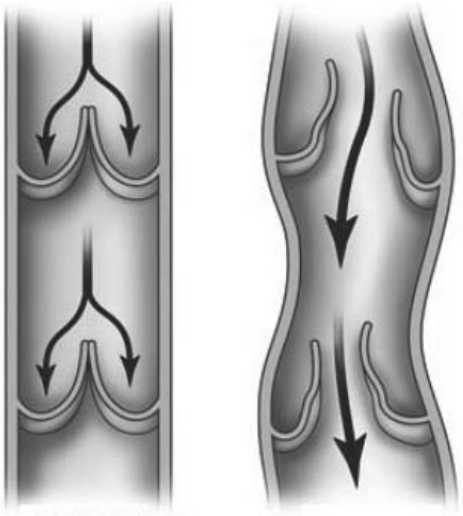


Fig. 79. Retrograde blood flow in the incapable of valves with varicose veins

There are promoted and produced factors. The former are physiological conditions of a blood flow in the inferior extremities, age's changes of venous wall, congenital delicacy of elastic and muscular fibers. To the second are mechanical difficulties of outflow, shunt of blood from system of deep veins in superficial (Fig. 79).

The listed factors lead to a valves failure of superficial veins. In pathological conditions at a lesion of the valval apparatus begins retrograde filling of veins of the legs from top, and the blood flow in perforated veins goes from deep to superficial veins (the blood stream is kinked).

Features of patient examination with suspicion on the varicose veins of legs.

At inquiry of the patient:

Patients complain on sensation of heaviness, completeness and spreading, fatigabilities in the leg. It is sponginess pastoseness or edema, a burning pain in a field of a phlebectasia, night spasms of muscles of crus, a pain. At the collecting of the anamnesis it is necessary to specify the cause of disease, a working condition and heredity.

Clinical physical examination

On the view there are separate varicose units, their conglomerates that collapsed in horizontal position. There are typical localizations of varicose units which fit with localizations of perforated veins: a point of sapheno-femoral anastomosis, boundary of middle and inferior third of femur, top thirds of crus on a medial surface, an epimalleolar field. Also on the view it is possible to find out complications of varicose disease which are display of a stage of a decompensation. The clottage of varicose veins and a thrombophlebitis, a varicose ulcer, dermatitis, a pigmentation of skin, lymphangitis, bleeding from a varicose ulcer.

The clinical characteristic of a chronic venous failure I degree. The chronic venous failure I degree answers a stage of compensaition. It is characteristic the absence of complaints. Patients mark sensation of heaviness and completeness after an exercise stress. On the view of patients in vertical position probably slightly expressed dilating of branches of the vv. Saphena magna et parva. The valval apparatus is not damaged due to functional tests.

The clinical characteristic of a chronic venous failure II A degree. For a chronic venous failure II A degree which answers a stage of sub-compensation, characteristic are complaints on sensation of heaviness, completeness and spreading, sponginess pastoseness**,** edema in the leg, burning pain in a field of a phlebectasia which arise in the end of day and pass till the morning. On the view of the patient in vertical position it is possible to note an appreciable the varicose veins of legs. A functional test of the valval apparatus finds out a failure hypodermic and communicative veins.

At a chronic venous failure II B degree a subjective symptomatology is increased. Sensation of heaviness in legs is constant, fast fatigability, an edema and spasms of crus. The trophic changes of soft tissues with the phenomena of induration, hyperpigmentation, hemosiderosis, dermatitis, loss of hair, and induration of hypodermic tissue are characteristic also. Varicose veins are visible well. There is a failure hypodermic, communication and deep veins of the leg due to functional tests.

The clinical characteristic of a chronic venous failure III degree. For a chronic venous failure III degrees, which educes in a stage of the expressed clinical forms of decompensation, characteristic formation on a background above the described changes vasotrophic ulcers which are localized, mainly, in the inferior third of crus (Fig. 80).

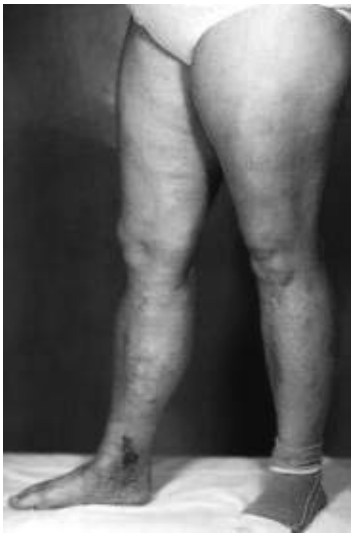


Fig. 80. Trophic complications of the varicose disease

According to standard schemes the plan of auxiliary inspection (laboratory and instrumental) at the patients with varicose veins of legs includes:

Functional tests for definition of a state of valves superficial, communication and deep veins of the inferior extremities.

Assays for definition of a state of valves of superficial veins. *Test Brodie - Troyanov - Trendelenburg* is carried out in position of the patient, laying on a back (supine). The investigated extremity is lifted for emptying the varicose veins. A soft rubber tourniquet applies on the top third of femur or the spot of saphenofemoral junction squeezed by finger. The patient stands up. After decompression of the great saphenous vein, it is filled within some seconds by retrograde blood flow. This is treated as positive test which indicate the failure of the saphenofemoral junction and valves of the great saphenous vein trunk.

The Hakkenbruch's test. At the patient who stands in vertical position, press by fingers the venous trunk and ask to cough. At a failure of veins valves is present, especially saphenofemoral junction, the jerk of blood returning wave is passed to fingers which lay on vessel.

The tests for definition of communication veins valves state

Pratt's test (Fig. 81) is carried out in position of the patient, laying on a back. The investigated extremity is lifted for emptying the varicose veins. Then an elastic bandage applies on the extremity from toes to groin. A soft rubber garrot applies on the top third of femur. The patient stands up. The garrot is unwind little by little from the top and, simultaneously, from an inguinal field applies an-

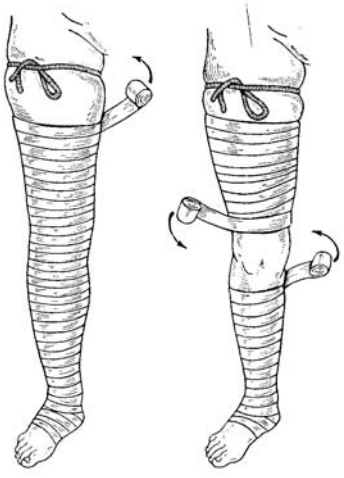


Fig. 81. Pratt'stest

other bandage so that between them there was an interspace 5–6 cm. Thus in places of a communication veins valves failure there is a venous distention. Sheynis' test (triple-garrot). Test carried out in position of the patient, laying on a back (supine). The soft rubber garrots apply on the top third of femur, above- and under- the knee joint. The patient changes in vertical position. Filling of superficial veins between garrots testifies to presence of a communication veins valves failure in these segments. Thalman's test (modification Sheynis' test). A rubber garrot in length 2-3 m is used for performance this test. After empting superficial veins garrots applies on the patient leg. Then the patient changes in vertical position and the garrot is taken out. Observing of filling of veins, a communication veins valves failure is assessed, as at Sheynis' test.

Assays for definition of deep veins valves state. *Mayo – Pratt's test.* Test is carried out in position of the patient, laying on a back. The soft rubber garrot applies on the top third of femur and leg is bandaged tightly from toes to the top third of femur. The patient is suggested to walk during 20-30 minutes. Appearance of a strong distend pain in an extremity is connected with disturbance deep veins patency. Absence of unpleasant sensations testifies to good function of deep veins.

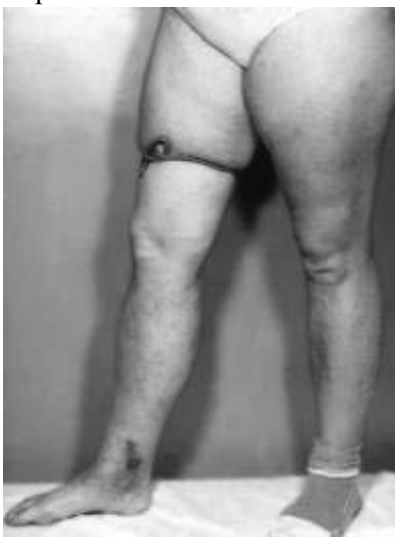


Fig. 82. Test Delbet - Perthes («march test»)

Test Delbet - Perthes («march test») (Fig. 82). The patient is in standing position. On the top third of femur a garrot is applied. Then the patient goes during 10-15 minutes. The saphenas are collapsed if there are deep and communication veins patency and their valves apparatus is safety.

Phlebography apply to finding-out of a state of deep and main veins and their valval apparatus, definition of features of a blood flow in veins and its direction in communication veins. At diseases of deep veins of the inferior extremities apply a distal vertical functional phlebography. For this purpose one of dorsal vein of foot or a great saphena anteriad of medial ankle are punctured or denuded and catheterized. To prevention filling superficial veins contrast solution introduce after applying a tourniquet on the inferior third of shin. An x-ray table is placed under a 45 - 90° angle. The phlebography carries out in some stages: a shin, a femur during the muscle contraction at standing up on toes of foot. Intraosseous phlebography carries out through heel bone more often when intravenous phlebography is impossible (an oedema of foot, trophy ulcers). For finding-out of a state of a femoral vein and its valves, the phlebography carries out by subcutaneus puncture of the femoral vein. Contrast mass is injected holding of breath (Walsalva's test). At an insufficiency of valves there is retrograte blood flow as the result shunting the contrast solution in distal parts of the femoral vein, a popliteal vein, veins of the shin.

Differential diagnostics. The varicose veins of legs is carry out with diseases which have a similar clinical picture: congenital venous dysphasia, a femoral hernia.

Differential diagnostics of varicose disease and congenital dysplasia of veins. The last represent capillary, cavernous or ramal hemangiomas. Differential diagnostics is based on the collecting of anamnestic data about disease exhibitions, especially at young age, however the basic method in recognition is the phlebography.

Differential diagnostics of varicose knot of groin and a femoral hernia is carrying out using the data of the patient inspection. The opportunity of manual reduction of hernia protrusion in the abdominal cavity in vertical position is characteristic for a femoral hernia but it is impossible to make at presence of varicose knot of groin. Also the collecting of anamnesis data is important (were or not a strangulation of hernia, when the swelling was occurred). Also a phlebography is possible for differential diagnostic.

Substantiation and formulation of the clinical diagnosis at the patient (on classification of disease, presence of complications and an accompanying pathology basis)

1) The basic diagnosis - primary (secondary), mixed varicose of the great saphenous vein of dextral leg in the compensate stage without valves failure;

2) Complication - the acute thrombophlebitis;

3) Accompanying - a chronic venous failure I stage

Treatments of the patient. Today at treatment in treatment of a The varicose veins of legs it is possible to secure{discharge} following methods of treatment: conservative, sclerosing and operative.

Conservative treatment of a the varicose veins of legs is directed on the prevention of the subsequent development of disease or if there is a contraindication to application of other methods of treatment. Hard bandaging of the leg, wearing of convenient footwear is necessary to recommend patients. During a sleep - to give to legs the elevated position. It is recommended to limit intake of fluid, salt, to normalize weight. Prescribe drugs which improve microcirculation in tissues (Troxevasinum, Escin), venotonics (Detralex)), diuretic drugs, cardiac glycosides, a physiotherapy, exercise therapy, at not complicated forms of varicose disease the hydrotherapeutic procedures, especially swimming, are recommended.

Sclerotherapy: the introduction in varicose nodes sclerosing solutions (Varicocidum, vistarin, Trombovar) Drugs cause a destruction of intimae, the walls of vein adhere, obliterations of their lumen comes. The indication to sclerotherapy: for an obliteration of separate nodes or fields of the varicose veins in an initial stage of disease or negative Troyanov's test; for obliteration of separate nodes and small veins which have remained after excision of the cores; in the form of the combined treatment (sclerotherapy lateral branches of superficial veins before operation). Contraindication to carrying out sclerotherapy are thrombophlebitis, obliterating and purulent diseases.

Surgical treatment of the varicose veins of legs (Fig. 83). Surgical treatment of the varicose veins of legs is applied at: progress of subjective signs, a decompensation of varicose disease with a failure of valves, complications of varicose disease (an acute thrombophlebitis, a trophic ulcer, a bleeding from varicose nodes). Contraindications to surgical treatment can serve: an obstruction of deep veins; decompensate heart diseases, diseases of the liver and kidneys with the expressed disturbance of

their function; an obesity III degrees. Dermatitises, eczemas concern to relative contraindications.

The saphenectomy operation sequence. Today there are combined methods for surgical treatment of varicose disease which consist of bonding several classical operations have been offered earlier as independent. As a rule, the operation begin from Troyanov- Trendelenburg 's operation. From the small cut of a skin below inguinal [Poupart's] ligament separate the great saphenous vein, ligate it in the saphenofemoral junction and transect between two ligatures. Simultaneously ligate all veins which flow in to the great saphenous vein in this place. On the femur the great saphenous vein is delete by Babcock's method. For this purpose, into a lumen of the crossed great saphenous vein introduce special olive-tipped probes which pass into distal direction. Then above an oliva of a probe make a small cut of a skin, transect a vein and its end fix by the ligature on a probe. Then a probe extract together with a vein. On the shin saphenas delete from separate small skin cuts which placed each other on 10 - 15 cm. This method has offered Narath. At the valves failure of femur and popliteal deep veins carry out extravasal methods elimination of valves failure which principle consists in formation extravasal spiral which narrows a vein in a place of the big valve, or sew with atraumatic suture by Kisner. After operation an active regimen is recommend for patients. Walking is allowed next day after operation. The patients are recommend bandaging an extremity by elastic rollers during 1,5 - 3 months for prophylaxis of postoperative complications.

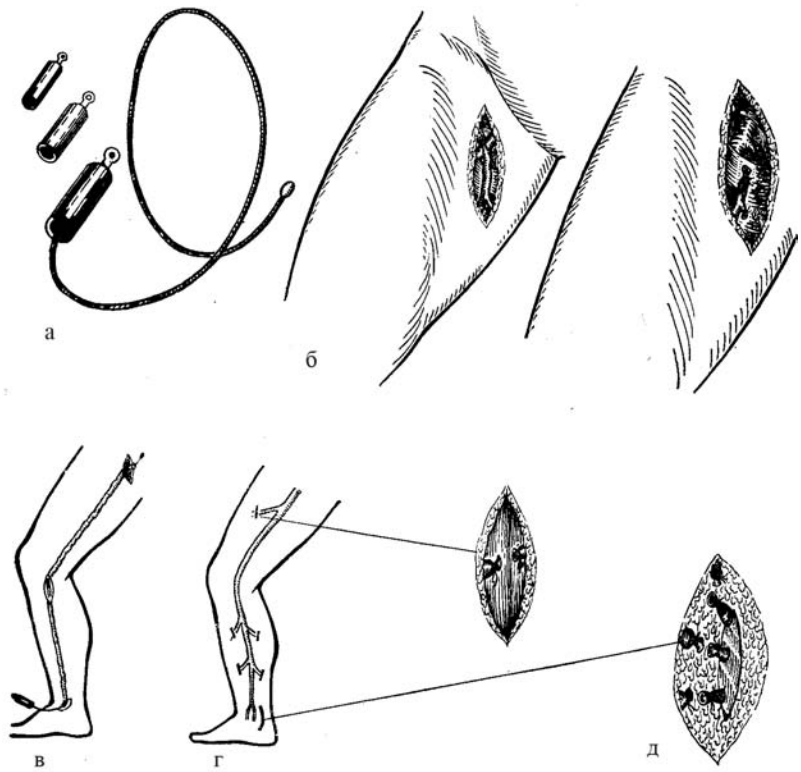


Fig. 83. Babcock- Narath operation: а - Babcock's probe, б - Troyanov- Trendelenburg's operation, в - removing of the main trunk of the great saphenous vein by Babcock's probe, г - removal of the narrative varices and varicose side branches by Narath, д - Kokket's operation

The cause of varicose disease relapse after saphenectomy is veins which run into the great saphena in the region of the saphenofemoral junction. Therefore the ligation of the given veins during performance of primary operation is a great importance for the prevention of disease relapse.

The ligation of insufficiencies communication veins which are localized, more often, on inside surface of the shin is obligatory. At absence of trophic disorder the over fascia ligation of communication veins is validated. If there are trophic distresses of the skin and subcutaneus fats it is necessary to perform sub fascial ligation of the communication veins by Linton. Operation carry out from a cut on an internal surface of an shin in length 10 - 15 cm. Cutting a skin, a hypodermic fat, a crural fascia, find communication veins then ligation and cut them. Continuity of a deep fascia restore suturering by doubling its edges. If the indurations of skins and a hypodermic fat on the internal and external sides of shin are presence it is necessary to carry out subfascial ligation of communication veins through a section on a back surface of an shin (by Felder).

For prophylaxis of the varicose veins of legs wearing elastic stockings is recommended, bandaging of extremities, wearing convenient footwear on a low heel, to avoid a long standing and hard physical exercise, working in hot and wet rooms, normalization of weight.

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VI.2. Acute thrombosis of the main veins of extremities

The acute occlusion diseases of main veins of extremities are the important and not yet finally dissolved problem of medicine. Actuality of it are spread of diseases of veins which are observed in 7–8 times more frequent, than diseases of arteries of extremities, patients with this pathology make the considerable percent of policlinic surgical visits.

In a whole world statistic mark permanent growth of acute venous occlusive pathology, scientists connect it with growth of cardio-vascular and oncology diseases, with appearance and quick distribution of difficult operative interferences on a heart and big vessels, transplantation of organs which requiring protracted intravenous insufflations, wide administration of coagulants and etc.

Presently according to results of investigations of scientists the acute thromboses of deep veins and it complication affected 1,5 % population, greater part of which is in work age; on 100 000 populations 160 cases of acute vein thrombosis are marked.

Acute thrombosis of the deep veins and thromboemboly of pulmonary artery begin to occupy the leading value among postoperative complications: ATDV develops after orthopedic operations at 50–75% of patients, after prostatectomy – at 40%, in abdominal and thoracal surgery – at 30% of patients (Bergquist D. and al., 2005).

ATDV takes the third place among the reasons of death from the cardiovascular diseases after the myocardial infarction and ischemic insult of cerebrum (Heit J.A. et al., 1999).

With above mentioned pathology of veins may meet all doctors of basic specialities and each of them mast be able to recognize this pathology, to appoint the necessary investigation and in the case of necessity administrate the conservative treatment or to point him at operation.

The most widespread classification of the acute vein thromboses, accepted in our clinic, is classification of L.I. Klioner (1969), the criteria of which are: localization of primary thrombosis, ways of its distribution, etiology, clinical feature, degree of trophic disorders and disorders of homodynamic.

I. ***On localization of primary thrombosis process and way of its distribution***:

A. Thrombosis of system of vena cava inferior:

1) thrombosis of veins, draining muscle of shin;

2) iliac-femoral thrombosis;

3) thrombosis of subrenal, renal, suprarenal segments of vena cava inferior or thrombosis of all trunk of vena cava inferior;

4) cava-ileofemoral thrombosis;

5) combinated total thrombosis of all deep vein system of vena cava inferior;

Б. Thrombosis of system of vena cava superior:

1) thrombosis of trunk of vena cava superior at the level of mouth of vena azigos, above and below than it;

2) thrombosis of all trunk of the vena cava superior;

3) thrombosis of trunk of the vena cava superior and jugulars veins;

4) thrombosis of the vena axilaris and vena subclavia;

5) total thrombosis of all deep vein of upper extremity.

II. ***On an etiologic factor:***

1) after the infectious diseases;

2) after traumas;

3) after operations;

4) after births;

5) at varicose dilatation of superficial veins;

6) at an allergy;

7) at presence of intravascular congenital and acquired factors (septums, diaphragms, adhesions, atresias);

8) at presence of extravascular congenital and acquired factors (compression of veins by arteries, by tumors, by aneurism).

III. ***On the clinical flow:***

1) acute thrombophlebitis;

2) subacute thrombophlebitis;

3) postthrombophlebitic syndrome;

4) acute thrombophlebitis on a ground a postthrombophlebitic syndrome.

IV. ***On the degree of trophic disorder and disorders of hemodynamic:***

1) mild form;

2) middle form;

3) heavy form.

We consider it necessary the division of acute vein thromboses on *the acute thromboses of superficial veins and acute thromboses of deep veins of extremities*. To the thromboses of superficial veins in modern time uses a term «thrombophlebitis of subcutaneous veins», which is often meet at patients with varicose dilatation of veins of lower extremities (in 34-65 % cases) and which we are name «acute varicothrombophlebitis». Infections, traumas, malignant tumors are assisting in development of thrombophlebitis of superficial veins (symptom of Fysher).

A. A. Shalimov selects 3 stages of vein thrombosis: acute (7-14 days), subacute (to the 3rd months) and chronic (after 3rd months).

A. Acute thrombosis of superficial veins of extremities

The main superficial veins of lower extremities may be affected as unchanged and varicose dilated. Because of intravenous injections (taking away of analyses, intravenous insufflations, including by permanent vein catheters) there are the thromboses of superficial veins of upper extremities.

Peculiarities of investigation of patient with suspicion on the acute thrombosis of superficial veins of lower extremity.

At questioning of patient:

1). Characteristic complaints of the basic disease: on discomfort, general weakness, subfebrile temperature (a chill can be marked sometimes), on the pain and presence of painful infiltration around of affected superficial vein. The pain may increases during motion or walking.

At acute varicothrombophlebitis patients feel pain in area localization of one or a few varicose nodes on shin, pain on a thigh in area of thromboses of trunk of vena saphena magna. The patient notes that the affected nods became hard and can not disappear in horizontal position.

2). Complaints from other organs and systems: for their detection need perform the detailed questioning on organs and systems.

3). Anamnesis of disease: as usually, development of acute thrombosis of superficial veins of lower extremities frequently takes place acutely. Predisposing factors for appearance are: infectious diseases, operations, traumatic damages, presence of malignant tumors, allergic diseases.

4). Anamnesis of life: usually patients long time mark presence of varicose - dilatation veins on extremities. Often such patients have hard physical work.

Clinical objective inspection (characteristic features at this disease):

1). Estimation of the general condition of patient: the common state, as a rule, is relative satisfactory. Consciousness clear. A subfebrile temperature is marked.

2). External condition of patient: patient some adynamic due to pains in extremity. Pains are more expressed at walking.

3). Inspection of the condition of the heart-vessels system: moderate tachycardia, presence of varicose dilated veins on lower extremities.

4). Inspection of respiratory system: without characteristic features.

5). Inspection of the condition of organs of abdominal cavity: without characteristic features.

6). Inspection of the state of bones–muscles system: without characteristic features.

7). «***Locus morbi***»:

At examination of the affected lower extremity hyperemia and edema of skin is determined, painful "string like" infiltration in area of segment of vein, usually expressly delimited from surrounding tissues.

At the thrombosis of segment of trunk of vena saphena magna pathological focus locating on anterio-medial surface of shin and thigh. At the thrombosis of segment of trunk of vena saphena parva focus of affectation locating on dorso-lateral surface of shin.

Acute thrombosis of the unchanged superficial veins of extremity, especially in absence of reason (operations, traumas and al.) in anamnesis it is necessary to interpret as *the Fysher's symptom*, which may indicate on big probability of presence (it is special in age more than 40 years) malignant tumor formation of any localization.

The diameter of the affected extremity, as a rule, does not change; it means that the general edema of extremity is not characteristic.

At the acute thrombosis of varicose dilated veins hyperemia and edema of skin in the projection of the affected vein is marked, thrombosed varicose nodes are hard, painful at palpation.

At the post-injection thrombosis of superficial veins of upper extremity by palpation detects hyperemic "string like" hard infiltration.

8). Leading clinical symptoms: pain and painful induration in the projection of hypodermic veins of extremity.

9). On the basis result of findings of questioning and objective clinical inspection of patient it is possible to propose one of the following clinical diagnoses:

a). Acute thrombosis of the vena saphena parva (magna) in area of shin (thigh);

b). Varicose illness of right/left lower extremity, stage of decompesation, complicated by acute thrombophlebitis of the shin (thighs);

c). Acute post-injection thrombophlebitis of subcutaneous veins of forearm.

***According to standard charts the plan of additional investigation (laboratory and instrumental) of patient with the acute thromboses of main veins of extremities includes*:**

1). Clinical blood test.

2). Clinical analysis of urine.

3). Biochemical blood test.

4). Coagulogramm.

5). Urgent ultrasonic research of veins of lower extremities with Dopplerography.

1). *Clinical blood test*: the changes are unspecific: it is possible leucocytosis, insignificant increase ESR.

2). *Clinical analysis of urine*: without characteristic features.

3). *Biochemical blood test*: without characteristic features.

4). *Coagulogramm*: disorder coagulation of blood to side of hypercoagulation.

5). *Urgent ultrasonic research*: gives possibility with absolute exactness to detect a presence, localization and extent of thrombosis. The special attention is spared to the proximal border of thrombosis, due to possibility of tearing off of blood clot and the TELA development.

***Differential diagnostics*:** it is performed with other pathological processes, more frequent of inflammatory nature, localized in a skin and subcutaneously, such as: lymphangitis, erysipelas inflammation, nodular erythema, allergic dermatitis, strangulated femoral hernia.

Ground and formulation of clinical diagnosis (taking according classification of disease, presence of complications and concomitant pathology):

1) basic - Acute thrombosis of varicose - dilated veins (varicothrombophlebitis) of right/left lower extremity

2) complications –(if it present)

3) concomitant pathology- (if it is)

Treatment of patients with the acute thrombosis of superficial veins of extremities

Medical treatment must be directed on the dissolving of the following tasks:

1) prevention of distribution of thrombosis on deep veins and appearance of TELA;

2) quick liquidation of the inflammatory process in the walls of vein and surrounding tissues;

3) excluding of relapse of thrombosis of superficial veins.

Choice of medical tactic:

At the acute thrombosis of superficial veins of lower extremities *surgical treatment is indicated urgently* for the excluding relapsing of thrombosis and TELA.

The brief postponement of operation (on 1-2 days) for performing of conservative therapy is possible, directed on the removal of expressivity of periphlebitis.

Conservative therapy is administrated at the refuse of patient from operation or at presence of serious contra-indications (extremely heavy general condition and others... and also as preoperative preparation).

At postinjection thrombophlebitis of subcutaneous vein of shoulder conservative therapy is also indicated.

Surgical treatment of acute thromboses of superficial veins of lower extremity

Surgical treatment can be *palliative and radical.*

The target of palliative operation is: prevention of transition of thrombosis on deep veins through saphenofemoral or saphfenopoplyteal mouths and prevention of the TELA development**.** The classic operation Troyanov-Trendelenburg or its modification is proposed for it.

The Troyanova-Trendelenburga operation includes: bandaging and resection of area of vena saphena magna immediately at the place of inflow of it in a femoral vein (the area of vein is excised between two ligatures).

Modern modification of this operation: high legation of vena saphena magna with the obligatory bandaging of all communicants near the mouth and excision of trunk of subcutaneous vein within the limits of wound. At distribution of thrombosis higher than mouth of vena saphena magna is performing thrombectomy from a femoral or even external iliac vein.

Radical surgical treatment foresees not only the removal of threat of development of thrombosis of deep veins and TELA but also recovery from thrombophlebitis, and from varicose disease at varicothrombophlebitis on a shin or thigh by projection accesses by Narat with the use of method of «tunneling», with the obligatory bandaging and dissection of vena saphena magna at the place of inflow of it in a fem-

oral vein is the Troyanov-Trendelenburg operation (at the thrombosis of segment of vena saphena parva is bandaging and dissection of it at the place of inflow in a popliteal vein).

At acute varicothrombophlebits traditional radical operation is including – venectomy by Bebkokk-Narat, which begins by the modern modified operation Troyanov-Trendelenburg, with obligatory removal of all varicose dilated veins (with thrombosis and without thrombosis) with the obligatory bandaging of perforant veins with valvular insufficiency.

The pathogenetic grounded conservative therapy of acute thrombosis of superficial veins of lower extremities is directed on dissolving of inflammatory and local thrombotic processes, prevention of distribution of thrombosis on deep veins and the TELA development.

1) Regime – 2 – 3 days bed with transition on the semibed mode with the obligatory elastic bandaging of the staggered extremity from a foot to the groin, that provides more intensive blood stream in deep veins, hindering to development in them of thrombosis and warns the TELA development.

2) A diet is prohibition of reception of alcohol, acute dishes.

3) Medicinal therapy: nonspecific anti-inflammatory medicines is diclofenak and its derivative (voltaren, ortofen, artrotek) and ketoprofen and its derivative (oruvel, ketonal, fastum), desagregant and hemocorrectors - intravenous inuflation of reopolyglucin, trental, small doses of acetylsalicylate acid; local anticoagulants: lioton-gel, hepathrombyn and athers; direct anticoagulants - heparyn, fragmyn, fraxyparyn is used at the persistent relapse of thrombophlebitis at patients with pathology of the system of homeostasis; venotonics - venoruton, troxevasin, detralecs, diovenor, which are the protectors of vein wall and have a anti-inflammatory effect; polyenzymes mixtures for oral application - vobenzim, flogenzym is used from ability of hydrolytic enzymes suppresses process of inflammation, to render unoedematic and immunomodulating action.

If during performing of conservative therapy appears distribution of thrombotic process (in spite of treatment) a indication to urgent operation on vital indication is necessary due to real threat of the TELA development.

Need performs minimal operative interference under local anesthesia as the Troyanov-Trendelenburg, or radical operations as venectomy by Bebkokk-Narat.

Rules of conduct of postoperative period, possible postoperative complications:

Application of the elastic bandaging, administration of direct anticoagulants as heparin (fraxyparin), desagregants, nonspecific anti-inflammatory drags and antipain are necessary in a postoperative period. For patients are recommended from the first day after operation moderately active motion regime.

Complications of acute thrombosis of subcutaneous veins

Abscess formation, transition of thrombosis on the deep veins of extremity, TELA.

B. Acute thrombosis of deep veins of lower extremities (ATDV)

The acute thrombosis of deep veins of extremities is the disease dangerous by possible complications. The considerable diameter of main veins gives possibilityfor formation into them of blood clot of big size, and an intensive blood stream forming

predisposing for easy it tearing off and the TELA development with a possible fatal outcome. In the big percent of cases the thrombosis of deep veins of lower extremities lead to development of postthrombophlebitic syndrome (PTFS) which very common are the cause of chronic vein insufficiency often with development of trophic ulcers, that considerable reduces ability to work and quality of life of patients.

Peculiarities of investigation of patient with suspicion on the acute vein thrombosis of deep veins of lower extremities.

At questioning of patient:

1). Complaints on the basic disease: on the edema, blush discoloration of extremities, pains in area of shin, in popliteal fossa, on a thigh, increasing at motion, local increase of skin temperature, high temperature, general weakness.

2). Complaints from the side of other organs and systems: head pain, tachycardia.

3). Anamnesis of disease: more frequent of acute thrombosis of deep veins of lower extremities takes place acutely, without some visible reason. Between the cases which support appearance of acute process, the important role regard to previous infection diseases, operations, traumatic damages, malignant tumors, allergic diseases, the tumors of uterus and appendages serve as a frequent reason at women, complicated births and postnatal period. The flow of disease is acute, progressing.

4). Anamnesis of life: specific peculiarities as usually are not present.

***Clinical physical investigation (specific features at this disease)*:**

1). Estimation of the common state of patient: common state more frequent of middle heavity. Consciousness clear. The forced position due to pain, febrile temperature is marked.

2). Collection of information about original condition of patient: patient some adynamic due to pains in extremity.

3). Inspection of the state of the cardio-vascular system: it is described in «Locus morbi».

4). Inspection of respiratory system: without characteristic features.

5). Inspection of the state of organs of abdominal cavity: without characteristic features.

6). Inspection of the state of bones-muscles system: without characteristic features.

7). «***Locus morbi***»:

From the side of heart is tachycardia.

Objective symptoms are similar to subjective. Basic symptoms: edema of extremity and level of it – depending on localization of thrombosis, cyanotic discoloration of skin and increase of skin temperature of the affected extremity, dilatation of subcutaneous vein network; a main symptom is a painfulness at palpation in the projection of the affected deep veins: on a shin – on a back surface, in popliteal fossa, on the anterio-medial surface of thigh, in a femoral triangle, in the inguinal-iliac region of abdomen.

At the thrombosis of deep veins of shin are detecting some of pathognomonyc symptoms: Homans (1941) - appearance of several pain in muscles gastrocnemius at dorsal flexion of foot; *Meyer* - painfulness at palpation on the internal border of tibia in middle third of shin; *Payr* - painfulness at palpation of medial surface of foot; *Opyts-Ramynes* - painfulness at compression of gastrocnemius muscle by a hand

(Fig. 84); positive test *Lovenberga* (Lowenberg, 1954) - appearance of several pain in a gastrocnemius muscle at compression of shin in the middle its third by the cuff of sphygmomanometer already at minimum pressure in 30-40 mm of Hg c.; Moses (1946) - appearance of pain at compression of shin by hands in front-back direction.

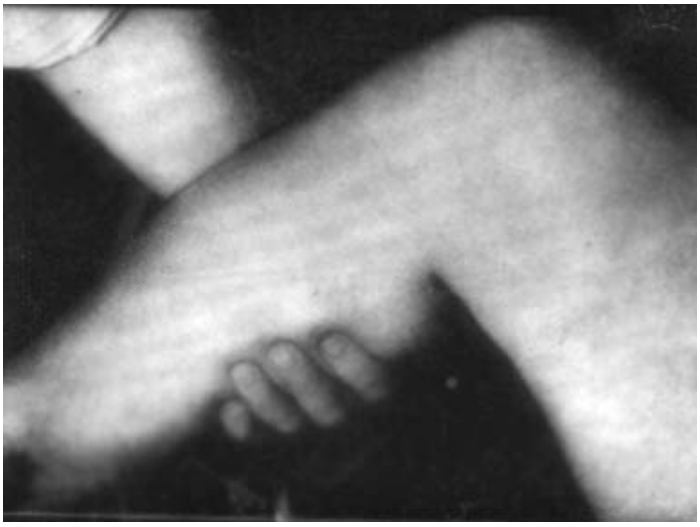

Fig. 84. Opyts-Ramynes symptom

It is necessary to mark that clinical symptomatic ATDV very variable in expression of symptoms and, especially, on the level of localization of edema.

So, at the thrombosis of deep veins of lower extremity on level up to mouths of deep vein of thigh («critical point») due to relatively high effectivety of collateral compensation of circulation of blood, symptoms and edema of extremity will be expressed moderately: at the thrombosis of one or two deep veins of shin an edema will be determined only at the level of foot and ankle joint, at the thrombosis of popliteal vein – in area of foot and shin, at the thrombosis of superficial femoral vein below than mouth of deep vein of thigh the edema will spread on the distal part of thigh.

At the acute thrombosis of all deep veins of shin, similarly as well as at the thrombosis of popliteal vein present expressed disorder of outflow of vein blood, a shin becomes an edema, tense, the diameter of it compare with a healthy shin on 4-5 sm more; a pain syndrome is expressed, a patient is disturbed by sense of dilatation of shin, tensions in a shin, on a foot and in lower third of shin there is cyanosis of skin. After 2-3 days the edema diminishes, but appear the extended subcutaneous veins on a shin, acute painfulness of gastrocnemius muscle at palpation and all symptoms described higher are safed.

At distribution of process on a superficial femoral vein, patients mark pains on the medial surface of thigh according to the projection of Gunter's channel, to which approximately corresponds the extent of thrombotic area of vein; vein circulation of blood in area of knee-joint can be affected, as result of it - pains, the contours of joint became smoothed, determined increase of its volume, appear disorder of function; the edema and distention of subcutaneous vein network in the distal part of thigh is marked.

The most expressed clinical picture is observed at **the acute ileofemoral vein thrombosis of lower extremities** (at sharp occlusion of general femoral and vena iliaca externa, and sometimes and vena iliaca communes) due to ineffectivety of collateral circulation of blood, which is selected in a separate nosology form.

The stage of the expressed clinical feature is characterized by the classic triad of signs: pain, edema and discoloration of skin

Pains are intensive, is localized in an inguinal region, on anterior-medial surface of thigh, can spread on shin muscles. The cause of pain is extension of walls of main vein below of place of thrombus formation because of intravenous hypertension, and also development of periphlebitis; patients mark sense of heaviness, tensions in extremities and its rapid increase in a volume due to edema.

Edema of extremity – most pathognomonyc symptom of acute ileofemoral vein thrombosis, which has spread character, taking all lower extremity (from a foot to the inguinal ligament), as result disorder of outflow from veins of extremity.

Change of coloring of skin - the blush discoloration have diffuse character is more frequent, rarer is the «spotted cyanosis», the pallor of skin can be marked sometimes. The cause of it is disorder of venous outflow from distal part of extremity, with formation of hypertension and stagnation of the blood, dilatation due to it venuls and capillars.

Strengthening of «picture» of subcutaneous veins on a thigh and especially in an inguinal region is the frequent and very important symptom of acute ileofemoral thrombosis, not being at the same time an early symptom (appears on 3-5 days after beginning of disease). It leads to diminishment of edema of extremity due to improving outflow of the blood through dilated superficial venous network.

Some patients can have the symptoms of «psoitis» characteristic by painfulness in an iliac region at the maximal flexing of thigh.

8). Leading clinical symptoms: pain, edema, discoloration of the skin.

9). On the basis of findings of questioning and clinical physical inspection of patient it is possible to propose the following clinical diagnosis: ACUTE THROMBOSIS OF DEEP VEINS OF LOWER EXTREMITY (shins, thighs, ileo-femoral)

10). Relatively rarely there is the thrombosis of deep veins of upper extremities (humeral, axilar, subclavicular), known as the Pedjet-Shretter syndrome. Meet more frequent at people the profession of which is related to the intensive dynamic loading on upper extremities.

Specific clinical forms of acute thrombosis of the main pelvis veins include **"white and blue pain phlegmasia"** *(phlegmasia alba dolens, phlegmsia cerulea dolens)* (Fig. 89) .

According to standard charts the plan of additional inspection (laboratory and instrumental) of patient with the acute thromboses of main veins of extremities includes:

1). Clinical blood test.

2). Clinical analysis of urine.

3). Biochemical blood test.

4). Coagulogramm.

5). Determination of the D-dimer level in plasma (D-dimer-test, dimertest) - a test on activity of thrombotic process.

6). Urgent ultrasonic investigation of veins of lower or upper extremities with Dopplerography, organs of abdominal region and small pelvis.

7). Roentgenoscopy of organs of thorax.

1. Clinical blood test: the changes are unspecific: it is possible leucocytosis, insignificant increase ESR.

2. Clinical analysis of urine: without characteristic features.

3. Biochemical blood test: without characteristic features.

4. Coagulogramm: the indexes of the coagulative system more frequent correspond to hypercoagulation.

5. Increase of the D-dimer level in plasma more than 500 mcg/l.

6. Ultrasonic research of vein vessels can with absolute exactness to expose a presence, localization, terms and extent of thrombosis; the special attention is spared to the proximal border of thrombosis, in connection with possibility of tearing off of blood clot and the TELA development. The ultrasonic investigation of organs of small pelvis can expose a tumor, as reason of thrombosis.

7. Phlebography in acute venous thrombosis rarely performed in case of unclear diagnosis or to determine the cause of pulmonary embolism.

Differential diagnostics: it is conducted with other pathological processes in symptomatic of which are: an edema and pains in extremity such as: insufficiency of blood circulation, lymphostasis, traumatic edema, anaerobic phlegmona, tumor of bones and soft tissues, tumors of organs of small pelvis, artrosoartritis, sharp arterial impassability**.**

Ground and formulation of clinical diagnosis (according classification of disease, presence of complications and concomitant pathology):

1) basic - sharp thrombosis of deep veins of shin (popliteal, thigh, iliac-femoral segment) of right/left lower extremity;

2) complications (basic disease if they are);

3) concomitant pathology (if it is).

Treatment of patient with the acute thrombosis of deep veins of lower extremities

1). Treatment must be dane in surgical department and more preferable in the specialized vascular department.

2). Treatment must be directed on the decision of the following tasks: to stop distributions of thrombosis; to prevent TELA; to prevent progressivety of edema and prevent a possible vein gangrene and loss of extremity; to restore ability passage of veins with that in future to avoid evolution of the PTFS.

Choice of medical tactic.

Presently in connection with presence of effective thrombolitics and anticoagulants even at localization of acute thrombotic process in the big main veins of extremities in case of timely hospitalization of patient the method of choice is *the conservative method of treatment.*

Operative treatment is used at the massive thrombosis of iliac and vena cava inferior with presence of flattering thrombes and impossibility to execute thrombolisis (high danger of development of massive TELA with a fatal outcome)

Pathogenetic grounded conservative therapy

1) Regime: first 3-5 days strong bed mode (up to diminishment of pains and possibilities of walking, diminishment of temperature, diminishment of probability of the TELA development), than moderate active (dosed walking) at the obligatory elastic bandaging of extremity.

2) A diet - limitation of acute dishes, reception of alcohol and food allergens.

3) Medicinal therapy.

More effective and pathogenic grounded therapy is thrombolitic therapy, which directed on restoration passage of affected vein. Administration of it in case of massive acute deep veins thrombosis (ATDV) with affectation ileocaval segment aicnd presence of flattering thrombes with time of presence up to 5 days is absolutely indicated.

Most effective there is thrombolitic therapy by the tissue activator of plasminogen – actelise in a dose 100 mg during 2 hours with following transition on direct anticoagulants. Use and other kind of thrombolitics such as streptokinase, cabicinase, urokinase.

The indexes of coagulogramm as concentration of fibrinogen A and B, thrombin time, activity of fibrinolisis are the control of thrombolitic therapy.

After ending of thrombolitic therapy for supporting condition of hypocoagulation is administrating heparin and it derivates (cleksan, fraxiparin, enoxaparin and others). It prevents the relapse of thrombosis, ascending thrombosis and TELA.

Control of result of direct anticoagulant therapy performs under control of time coagulation by method of Li Uait or Burger. Middle day's dose makes about 30000 UN (duration of therapy from 2 to 5 days).

Than mast be performed transition on indirect anticoagulants (varfarin, phenilin, sincumar). Control – level of prothrombin index with frequency 1 time per 2-3 days, which mast be lowered up to 35-45%.

Dosages: varfaryn 7,5 mg 1 times per days, phenilin 0,3 2-3 times per days, sincumar 0,4 2-3 times per days (taking into account a different sensitiveness of organism to the indirect anticoagulants, it is recommended to pick up doses individually). Absence of ascending thromboses and the ATDV relapses is considered the criterion of efficiency of anticoagulant therapy, the TELA development, and also quick regress of the basic symptoms ATDV.

During conducting of thrombolitic and anticoagulant therapy serious complications are possible: hemorrhagic (nose-bleeds, small subcutaneous haematomas, microhematuria up to massive bleeding). Not only the indexes of coagulogramm, but also regular analysis of urine, supervision of the wounds (appearance of hemorrhagic discharge).

In case appearance of hemorrhagic complications antidotes of direct anticoagulants are protaminsulfate and protamichloride (intravenous infusion 5-10 ml on normal saline). To antidotes after over dosage of indirect coagulants regard vicasol (intravenous insufflations of 2-3 ml on normal saline).

Opposite complication which is result of a «ricochet effect» is possible, when rethrombosis develops after the sharp stopping of administration of anticoagulants (gradual abolition of preparations is needed).

Hemocorrectors and **desagregants** for the improvement of microcirculation of blood, diminishment of it viscosity and aggregation of elements of blood (reopolyglucin, pentoxyfillyn, trental, aspirin in small doses);

Nonspecific anti-inflammatory drags (NAID) - diclofenac and it derivates (voltaren, ortofen, artrotec) and ketoprofen (oruvel, ketonal, fastum).

Venotonics – venoruton, troksevazyn, detraleks and others being protectors of vein wall, making better vein homodynamic and rendering an anti-inflammatory effect.

Symptomatic therapy - anesthetic, cardiotrophycs and other.

Existent methods of operative treatment

The tasks of operative interferences at the acute vein thrombosis of deep veins are:

1). Restoration of passage of vein bed;

2). Prevention of massive pulmonary embolism.

Next types of interferences are applied.

1. *Ideal thrombectomy* (which can be considered **radical operation** at ATDV) at the early admition (to 5-7 days) of patient and at localization of thrombi in big magisterial veins (ileofemoral segment, iliac veins, vena cava inferior). Thrombectomy is direct and indirect. *The direct (opened) thrombectomy* (Fig. 85) is performed on the easily accessible segment of deep magisterial vein – in area of general femoral vein (at once below than inguinal ligament in area of femoral triangle) by phlebotomy, removing of thrombotic mass with following plastic of vein. *Indirect (half-open) thrombectomy* (Fig. 86) is performed with help the Fogarty catheter at localization of thrombosis in the difficult for access segments of main veins (in iliac veins and in a vena cava inferior).

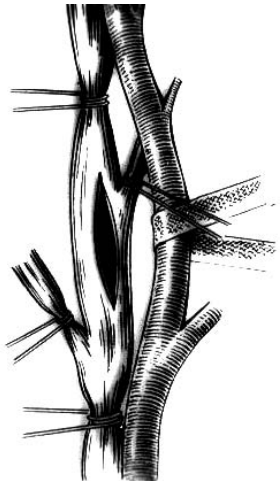

Fig. 85. Ideal thrombectomy

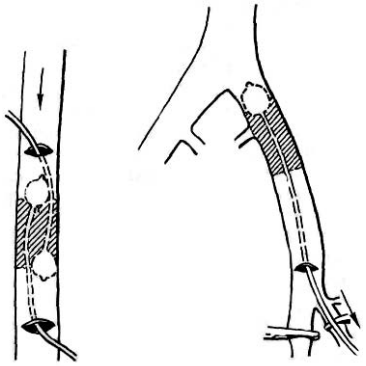

Fig. 86. Indirect thrombectomy with help the Fogarty catheter

2. Palliative operations directed on the TELA prevention.

It is plication of the vena cava inferior– method of partial occlusion of lower hollow vein (F. C. Spenser, 1959 and M. Ravitch, 1964). The operation includes

dividing of the lumen of vein on same channels with diameter slightly more than 3 mm with help special stitching sets or separate stitches.

More effective is endovascular implantation of cava filters..

Bandaging of main veins is the type of operative interference, directed on blocking of only that part of vein bed, where pulmonary embolism threatens from (presently is not practically used).

***The peculiarities of postoperative period, possible postoperative complications*:**

In postoperative period from the 1 day after operation used moderately active motion with obligatory conducting of the elastic bandaging, anticoagulant therapy (in beginning by direct, than indirect anticoagulants), desagregants, hemocorrectors and, symptomatic therapy.

Possible complications:

А). Bleeding during operation;

Б). Suboperative TELA;

В). Postoperative rethrombosis;

Г). Inflammatory complications of wounds;

Д). Damages of nerves and lymphatic vessels.

Diagnostics and treatment of possible complications of acute thromboses of main veins of lower extremities

Thromboemboly of pulmonary artery is the most threatening complication of acute thromboses of main veins of extremities (TELA). Source of TELA more frequent are thrombes forming in ileofemoral segment of the deep veins of lover extremities (to 40%), rarer - in the deep veins of shin. To tearing off and bringing of thromboembol assist every active movement and tension of patient, most often – when a patient after the long bed regime first gets up or sits down (sometimes – during the act of defecation).

Pathophysiology basis of TELA are obturation by tromboembol basic trunk or branches of pulmonary artereries with following development of hypoxemia and hypertension in pulmonary artery. Develops impossibility for perfuse of blood by right part of the heart through decreased network of lung vessels. Due to it appears overload of the right part of the heart, arises acute or subacute insufficiency of the right ventricle. The block of the branches of the lung artery leads to insufficiency of the oxygen in arterial system – general hypoxemia. The lung arterial hypertension is consequence not only lung artery obturation but and arterial vasoconstriction too. Irritation of the arteriopulmonar reflectory zone from one side is lead to vasospastic reaction from other side of the lung and bronchospasm and coronar artery spasm. The clinical evidences of TELA, depends from size of blocked pulmonary artery: basic trunk and main branches, or middle and small branches.

Clinical picture. *General symptoms*: several pain in a chest, trouble, caused by fear of death, increase of temperature of body (Mykhael's symptom), loss of consciousness, general weakness, diminishment of diuresis.

Heart-vessels symptoms: increasing tachycardia (Maler's symptom), arterial hypotension (up to development of collapse), cyanosis of face, neck and upper half of trunk, distention of neck veins, increase of the central venous pressure (CVP), pain in area of heart and increase of liver.

Pulmonary symptoms: dyspnoea, cyanosis, cough, hemoptysis, noise and friction of pleura.

Rarer there can be symptoms: *cerebral* (loss of consciousness, convulsion, hemiplegy - at the aged persons because of hypoxia of brain), *abdominal* (pains in the right subcostal region due to enlargement of liver as result of right part of heart insufficiency), *kidney* (secretory anurya because of hypoxia of kidneys).

Additional methods of research

Electrocardiography (EKG) – signs of acute overloading of right part of the heart, disorder of the right ventricular conduct system – sinus or paraxismal tachycardia, atrial fibrillation, blockade of right pedicles of the Gys and others changings.

Roentgenography of the chest may show specific signs of lung emboli: enlargement of right part of the heart; extension and deformation of the lung hillum; triangular shadow of the lung or transparent of the lung parenchyma in involved to affectation zone (Westermark's symptom); limitation of diaphragmal excursion and elevation of it on side of affectation (Zveifel's symptom); pleural effusion.

Angiopulmonography is the most informative method of investigation, which may show next specific signs of emboli: central filling defect of pulmonary artery (amputation of the arterial branches); absence of vessel strictures below of obturation; extension of the truncus or main branches of the lung artery; retention of the radiopaque in main lung arterial bed on side of affectation.

Because in 65-85% of cases the ileocaval segment of vein system is the TELA source, for the detection of emboli focus need perform and ileocavagraphy.

The TELA treatment

All methods of the TELA treatment can be divided into 3 groups:

1) conservative method;

2) conservative therapy in combination with surgical interference;

3) urgent operative treatment.

Before more effective method of treatment on vital indications consided operation of Trendelenburg – direct embolectomy from the lung artery with help apparatus of artificial blood circulation.

Presently method of choice is *conservative thrombolitic* therapy: after performed of angiopulmonography and confirmation of the TELA diagnosis a catheter is inserted in the basic trunk of pulmonary artery, through which insufflating thrombolitic (streptase, urokynasea, actilise) with the following angiographic control to the complete lysis of tromboembol.

After ending of thrombolitic therapy need transit to therapy by direct anticoagulants (heparin or it derivates) with following therapy by indirect anticoagulants (on a standard chart).

To complex of conservative therapy includes hemocorrectors, desagregants, broncholitics, cardiotonics, antibiotics, antihistaminic drags and analgesics, need conducted correction of PH balance and other symptomatic therapy.

Only at ineffective thrombolitic therapy appear indication to surgical intervention. Presently together with traumatic direct embolectomy from the pulmonary artery through thoracotomy with help apparatus of artificial of blood circulation, pos-

sible performs endovascular operative interference - indirect embolectomy through a femoral vein by the Grunfild catheter.

Prophylaxis of TELA consists in timely diagnostic and effective treatment of ATDV of lower extremities, in prohibition of active movement after long bed regime with the obligatory elastic bandaging of lower extremities, in prophylactic application in the postoperative period of anticoagulant therapy with the permanent control of coagulogramm.

For the prophylaxis *of repeated TELA* – endovascular implantation of cava-filters is necessary (umbellate on Mobin-Uddin, 1967, conic of Grunfield, 1973).

VI.3. Pulmonary embolism

Presently in Europe rate of pulmonary embolism (PE) is 11–25 %. In particular, 100 000 cases of PE are annually registered in France, 65 000 patients are annually hospitalized for pulmonary embolism in England and Scotland, and in Italy — 60 000. From data of the largest clinics of the USA, PE is observed in 3% population of country. From data of Fremingem study, PE is 15,6% from all of intrahospital mortality, among them there were 18% and 82% surgical and therapeutic patients respectively. From data of numerous pathoanatomical studies, in 50–80% of cases pulmonary embolism is not diagnosed, and hypothetical diagnosis is made only in many cases. PE occupies one of leading places in obstetric practice: death rate from this complication ranges from 1,5 to 2,7% on 10 000 births, and makes 2,8–9,2% in the structure of maternal death rate. Not receiving adequate treatment, many patients die in the first hours from the onset of disease.

Pulmonary embolizm (PE) is occlusion of lumen of main trunk or branches of pulmonary artery by clot-embolus, origined from a blood clot which was formed in the veins of greater circle of blood circulation or right cavities of heart, and added to the lesser circle of blood circulation with the flow of blood. The mechanical blockade of trunk or branches of pulmonary artery by clot-embolusis accompanied with the generalized spasm of pulmonary arterioles, that results in acute limitation of blood flow in lungs.

Physiopathology basis of PE is occlusion of pulmonary arteries by clot-embolus with subsequent development of hypoxemia and pulmonary hypertension. Blood circulation in lung is acutely violated, an overload of right half of heart with development of acute or subacute right ventricular insufficiency, vast neuro-reflectory violations develop.

The clinical flow of PE depends on what vessel is occluded (basic trunk and main branches, or its middle and small branches), and also from completeness of occlusion.

On clinical flow four forms of PE are distinguished (N. B. Rzaev, 1970):

fulminant, at which death of patient occurs suddenly (during 10 minutes) from acute asphyxia or cardiac arrest;

acute – with sudden appearance of severe pain behind breastbone, expressed shortness of breath, development of collapse; in absence of treatment patients die during 24 hours;

subacute – develops more gradually and clinically presents as the lung infarction, the basic display of which is bloody expectorations;

chronic – develops gradually and clinically presents as chronic pulmonary and heart insufficiency.

Depending on the volume of lesion of lung vessels PE can be:

- supermassive — lesion of more than 70% of vessels of lungs (sudden loss of consciousness, diffuse cyanosis of overhead half of body, arrest of blood circulation, cramps, stop of breathing);
- massive — lesion of more than 50% of vessels of lungs (loss of consciousness, shock, hypotension, insufficiency of function of right ventricle);
- submassive — lesion from 30 to 50 % of vessels of lungs (shortness of breath, normal arterial pressure, the function of right ventricle is disturbed in less degree);
- unmassive — lesion less than 30% of vesselsof lungs (shortness of breath, the function of right ventricle does not change).

Features of examination of patient with suspicion on PE

A clinical presentation depends on size and location of clot-embolus.

General, cardio-vascular and pulmonary symptoms are defined.

***General symptoms*:** severe pain in thorax, anxiety caused by fear of death, fever (symptom of Michael), loss of consciousness, general weakness, decrease of dieresis.

Cardio-vascular symptoms: increasing tachycardia (c-symptom of Mahler), hypotension (up to development of collapse), cyanosis of face, neck and overhead half of trunk, dilation of neck veins, increase of CVP, pain in area of heart and liver enlargement.

Pulmonary symptoms: shortness of breath, cyanosis, cough, bloody expectorations, noise of friction of pleura.

It is possible to select several clinical syndromes:

1. Pain in thorax at PE is observed at 52–86,9 % patients. Pain is localized behind breastbone, without irradiation, has angina character, is accompanied by fear of death. PE is shock, shock from the obstruction of vessels. Syndrome of «weir», which is conditioned by violation of hemodynamics in the lesser circle of blood circulation due to clotting and reflex spasm of vessels. 3 variants of pain syndrome at PE are distinguished: angina-like; pulmonary-pleura; mixed.

2. Syndrome of acute respiratory insufficiency. Sudden disorders of breathing are characteristic: from feeling of shortage of air to the acutely expressed shortness of breath with appearance of cyanosis, development of bronchospasm, senses of fear.

Development of shortness of breath is explained by the reflex reaction of respiratory center on development of pulmonary hypertension and irritation of receptors of pulmonary vessels, and also by the increase of functional alveolar dead ground due to reduction of pulmonary perfusion.

3. Syndrome of acute vascular insufficiency. It is explained by pulmonary-depressor reflex - the reflex of falling of arterial pressure in the greater circle of blood circulation in response to acute increase of pressure in lesser circle. Other reason is diminishing of blood delivery to the left ventricle and decline of the cardiac output

because of considerable occlusion of pulmonary circulation and mechanical obstruction of blood flow.

4. Syndrome of acute cardiac insufficiency (right- and left ventricular). One of clinical presentations of PE is a syndrome of acute pulmonary heart.

The physiopathology mechanisms of development of acute pulmonary heart are: thromboembolic occlusion of pulmonary artery causes the reflex spasm of arterioles, acute increase of pressure in the system of lesser circle of blood circulation that leads to overstrain of right ventricle.

5. Syndrome of acute rhythm disturbances of heart. Tachycardia is present in 70–100 % cases, extrasystole, atrial fibrillation and flutter.

6. Syndrome of acute coronary insufficiency. At part of patients with PE on ECG, except the classic signs of acute pulmonary heart, the changes of segment QT and indent T can appear in the left thoracic load, that confirms the acute ischemia.

7. Cerebral syndrome. Acute disturbance of cerebral blood circulation. It ispresented by the loss of consciousness, cramps, hemiplegia, involuntary selection of urine, stool.

8. Abdominal syndrome. Acute pain in the right upper quadrant of abdomen, belch, vomiting, symptoms of irritation of peritoneum.

None of the mentioned syndromes is pathognomonic only for PE. At the same time, at absence of such symptoms as shortness of breath, tachycardia, painsin a thorax, the diagnosis of PE is doubtful. The value of these symptoms substantially increases at presence of the signs of deep vein thrombosis.

At questioning of patient:

1). *Complaints on basic disease*: sudden onset, quite often inexplicable shortness of breath is the most characteristic symptom of pulmonary embolism, it is the reflection of acute respiratory insufficiency and has inspiratory character, ortopnoe is not observed; a shortness of breath is of different degree of severity: from feeling of shortage of air to very severe.

A pain syndrome occurs in several variants. In 42–87% of patients there is acute knife-like pain behind breastbone. The duration of pain syndrome can vary from few minutes to few hours.

In embolism of main trunk of pulmonary artery quite often there is severe retrosternal pain, which is conditioned by irritation of receptors in the wall of pulmonary artery. In massive pulmonary embolism acute pain with a wide irradiation reminds pain syndrome in dissecting aneurysm of aorta. Sometimes pains can have angina character, that is associated with decrease of coronary blood flow because of decline of stroke and minute volumes of heart.

In development of massive infarctions of right lung there can be acute pains in right hypochondrium, related to acute right ventricular insufficiency and acute congestion and swelling livers, combining with an intestinal paresisi, symptoms of irritation of peritoneum.

At the infarction of lung acute pains in thorax, increasing at breathing and cough, are present.

At embolism of small branches of pulmonary artery pain can absent or be veiled by other clinical symptoms.

2). *Anamnesis of disease:* in most cases development of PE takes a place suddenly on background of acute deep vein thrombosis (diagnosed or undiagnosed).

3). *Anamnesis of life*: Collection of anamnesis can specify the presence of thrombosis of veins of lower extremities, varicose illness of veins of lower extremities, previous operative interventions, other.

Clinical physical examination (characteristic features in this disease):

Assessment of the general condition of patient depends on location and massiveness of pulmonary embolism and can be both the middle degree of severity and extremely severe, preagonal, the loss of consciousness can be present.

«***Locus morbi***» is an examination of the cardio-vascular and respiratory system. Position of patient is forced - semi-sitting. There is the expressed cyanosis of skin and visible mucous membranes. Consider that suddenly appearing cyanosis in combination with the shortness of breath, tachycardia, pain in thorax is by the reliable sign of PE, and development in patient of cyanosis of face, neck and upper half of trunk supposes massive pulmonary occlusion with a severe prognosis.

In majority of patients with PE increase of tachycardia is observed because of acute decompensated pulmonary heart. Expressed tachypnoe, dyspnoe.

From the objective symptoms of acute right ventricular cardiac insufficiency in PE it is observed: dilation of neck veins, positive venous pulse, enlargement of liver.

About the overstrain of right ventricle it is possible to judge on appearance of epigastral pulsation, strengthening of cardiac beat, displacement of right border of heart, sometimes pulsation in second intercostal interval on the left. At auscultation the accent of II tone and its splitting (rhythm of «gallop») upon pulmonary artery is found, a systole, and sometimes and diastole noises are auscultated there. Central venous pressure is considerably increased (CVP).

Leading symptoms: pain in thorax, acute cardiac and respiratory insufficiency.

On the basis of findings of questioning and clinical physical examination of patient it is possible to propose a preliminary clinical diagnosis: PE.

In accordance with standard charts the plan of additional examination (laboratory and instrumental) of patient with suspicion on PE in the specialized vascular surgery must include:

1) Clinical blood and urine test, biochemistry of blood.
2). Coagulogram.
3) ECG.
4) X-ray of thorax.
5) Echocardiography
6) Ultrasound Doppler of main veins of lower extremities
7) Perfusional scintigraphy of lungs
8) Angiopulmonography.
1) Clinical blood test - increase of WBC, ESR.
2) Coagulogram — the phenomena of hypercoagulability
3) *Electrocardiography diagnostics* (Fig. 87). Changes are characteristic for acute right ventricular insufficiency. The changes of complex QRS, characteristic for the turn of heart about longitudinal axis clockwise, appear. In lead I appears or deepens indent S, and in lead III an indent Q (syndrome of SI, QIII) is present. In the III

lead the high indent R appears also. The deviation of transitional area to V5 or V6 is characteristic, the incomplete or even complete blockade of right crus of His bundle develops quite often. On occasion the small elevation of segment S–T and inversion of indent T is present in III, AVF, V1–3 leads. The signs of overload of right atrium are characteristic: an increase of amplitude of indent P in leads II, III, AVF. All the mentioned changes of ECG, developed in acute pulmonary heart, as a rule, have transient character and disappear in few days. It is necessary to take into account that sometimes (even at massive embolism of pulmonary artery) specific on ECG symptoms can absent.

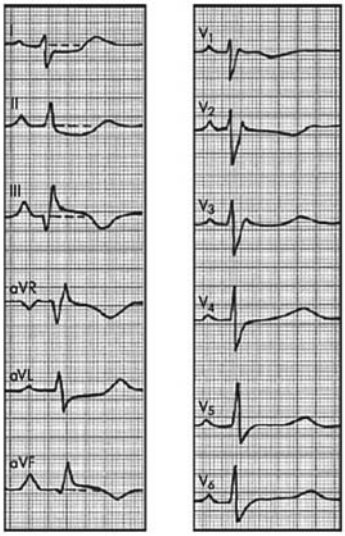

Fig. 87. ECG signs of PE

4) *X-ray signs of PE* (Fig. 88) are non specific in the first days. Most characteristic symptoms of acute pulmonary heart are: dilation of vena cava superior and cone of pulmonary artery, smoothing out of waist of heart. There can be increase of right cavities of heart, bulging of arch of pulmonary artery, absence of pulsation and expansion of lung radix, depauperated pulmonary picture in the area of branching of thrombosed trunk of pulmonary artery (symptom of Westermark, 1958), presence of wedge-shaped shade, high standing of diaphragm dome and limitation of its mobility on the side of lesion (symptom of Tsveyfel).

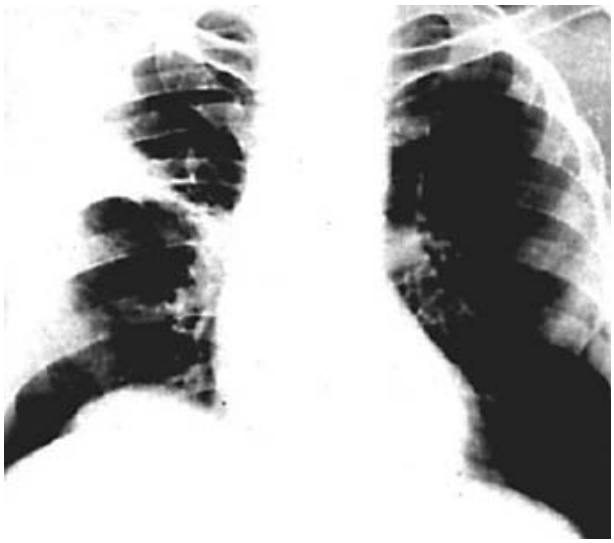

Fig. 88. PE X-ray picture(symptom of Westermark)

5) Echocardiography allows to reveal blood clots in the cavities of right heart, to evaluate the hypertrophy of right ventricle, degree of pulmonary hyperten-

sion. Echocardiography is very useful for the evaluation of regression of embolic blockade of pulmonary blood flow in the process of treatment, and also for differential diagnostics with similar on resentation diseases (heart attack, exudative pericarditis, tamponade of heart, dissecting aneurysm of thoracic aorta, thrombosis of vena cava superior).

6) Ultrasonic Doppler of main veins of lower extremities – is performed to reveal the source of PE - blood clots in veins of lower extremities.

7) Perfusional scintigraphy of lungs (PSL) is based on visualization of peripheral vascular circulation of lungs by the macroaggregates of protein, labeled by Tc^{99} or I^{131}. Defects of perfusion that corresponds to the area of blood supply of the affected vessel can be found. Specificity PSL rises at comparison of results with X-ray information. Absence of disturbance of perfusion of lungs allows with a sufficient confidence to reject PE.

8) Angiographic exam (angiopulmonography) (Fig. 89) - is gold standard in diagnostics of PE, confirming or eliminating of diagnosis. The most specific angiographic sign of PE is defect of filling in the lumen of vessel or «amputation», break of contrasting of vessel.

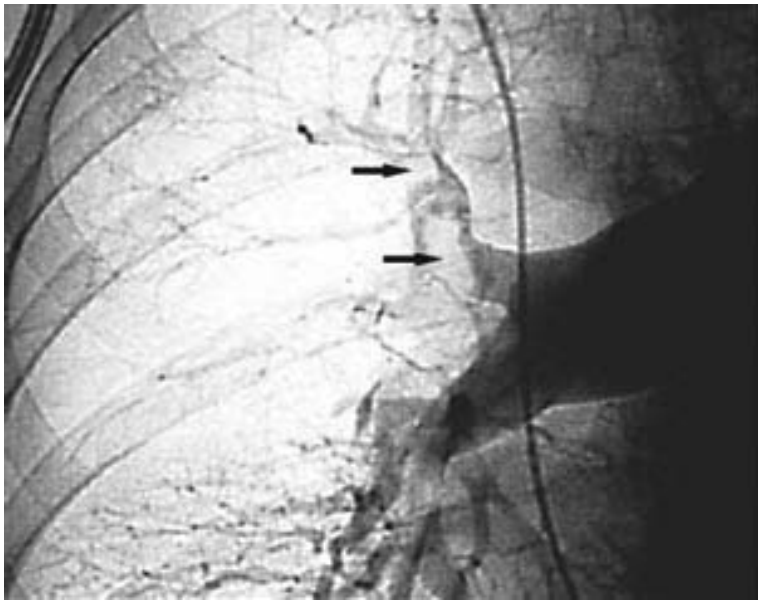

Fig. 89. Angiopulmonography: thrombs in pulmonary artery

***Differential diagnostics of PE*:** heart attack, dissecting aneurysm of aorta, croupous pneumonia, exsudate pericarditis, spontaneous pneumothorax.

Substantiation and formulation of clinical diagnosis (taking into account classification of disease, presence of complications and concomitant pathology):

1) basic – acute right ileofemoral venous thrombosis;

2) complication (of basic disease) — PE, acute form;

3) concomitant pathology (if present).

Choice of medical tactic

The basic medical measures directed on renewal of patency of the lumen of pulmonary artery are thrombolysis or removal of blood clot, however for their application time which can be insufficient is needed. From 3 patients, died because of PE, two died during first 2 hours after embolism. Therefore in the acute phase of severe PE very large value has measures, directed on life suppport (hemodynamics and interchange of gases).

First medical urgent aid

1. Life support in the first minutes (massage of heart, ventilator support, inhalation of oxygen) at massive PE. The indirect massage of heart not only provides blood delivery to vitally important organs, but allows to fragment blood clot or «push» it through pulmonary trunk and decrease the degree of obstruction of pulmonary vascular circulation.

2. Neuroleptanalgesia, narcotic analgetics (morphinum, omnoponum, promedol and other), non-narcotic analgetics with spasmolysants (trigan, baralgin), analgin in combination with antihistaminic preparations (Dimedrolum) are used against fear and pain (removes pain, fear, cathecholaemia, diminishes requirement in oxygen).

3. Removal of vasovasal intrapulmonic reflex — myotropic spasmolytics (euphyllinum 2,4% 10–20 ml, Nospanum, papaverine), beta-2-adrenomymetics (alupent, asthmopent, salbutamol), anticholinergic medicines (platyphyllinum); medicines, diminishing the blood inflow to the right atrium (nitroglycerine, isoket, 5-nitro).

***Conservative therapy*:**

The basic pathogenic grounded conservative therapy is thrombolytic therapy, directed on renewal patency of pulmonary artery, and also anticoagulant therapy, directed on creation of hypocoagulation and stopping of growth of blood clot.

Thrombolytic therapy.

The direct and indirect activators of endogenous fibrinolysis are used for thrombolysis: indirect thrombolytics (streptokinase, streptase, cabikinase, celiase); direct thrombolytics (urokinase), tissue activators of plasminogen (actilise, metalise).

Treatment of streptokinase is begun with intravenous introduction of 250 000 units («inactivating dose») of preparation in 300 ml of 5% solution of glucose during 30 minutes, after this during 8 hours infusion of medical dose of preparation 750 000 units proceeds (at a rate of 100 000 units/hour). For the prophylaxis of allergic reactions simultaneously with streptokinase 60–90 mg of prednisolone is injected.

Urokinase: during the first 15–30 min 4400 units/kg of mass of patient is entered intravenously, after this - 4400 units/kg per hour during 12–24 hours.

Since 90th of XX century actilise became the gold standard of thrombolytic therapy. The disadvantage of it is short period of semiejection (4–5 minutes), that requires permanent infusion of preparation during 90 minutes.

Scheme of application of actilise: intravenously 10 mg during 2 minutes, in subsequent 60 minutes — 50 mg, than during 2 hours 40 mg (100 mg during 3 hours). Intravenous introduction of 100 mg of preparation during 2 hours is possible.

Thrombolytic therapy by actilise in patients with severe PE have significant advantages as compared with streptokinase and urokinase, because it gives more rapid clinical effect.

Metalise (tenecteplase) modern thrombolytic medicine of the third generation, providing bolus thrombolysis. Time of semiejection by comparison to alteplase was increased on 20 minutes. It is entered during 5–10 seconds as a single intravenous bolus. Metalise is injected in an individual dosage (depending on weight of patient) that does preparation most safe. It is used on the prehospital stage, that improves prognosis substantially. It should be remembered that aspirin, heparin must be entered for the prophylaxis of rethrombosis on background of bolus introduction of Metalise, because damage of intima of revascularized artery is preserved for a long time.

During thrombolysis the following complications are possible:

1) pyrogenic and allergic reactions (urokinase, actilise and metalise practically do not have antigen properties);

2) hemorrhagic complications - in 45–50% cases;

3) probability of recurrence of PE is high at treatment by the plasminogen activator, because fragmentation of other venous blood clots in thromboembolic disease also takes place.

Rate of infusion of thrombolytic preparation must support thrombin time in 2–5 times longer than control. The effect of thrombolysis is assessed on regress of clinical symptoms, electrocardiography changes.

After completion of thrombolytic therapy heparin or low-molecular heparins are prescribed.

Anticoagulanttherapy

Presently basic preparation for treatment of PE is heparin (both non-fractioned and low-molecular). It supresses growth of blood clots, is instrumental in their dissolution and prevents thrombi development. In saddition, it has antiserotonin and antibradykinin action, due to what plays an important role in the elimination of vaso- and bronchoconstrictor effects.

10–20 thousands units of heparin are entered intravenously, than — 5000 units each 4 hours. More effective is to apply the high doses of heparin: intravenously - 20 000 units (300–500 units/kg) with subsequent infusion of 5000 units/hour. Control of heparin therapy: APTT 4 times per day (achievement of non-coagulation during more than 100 seconds). Then gradual decline of dose on 500–1000 units/hour. Daily dose must be 30 000–60 000 units.

Duration of course is 5–7 days, as there are lysis and organization of blood clot in these terms. 3 days prior to abolition of heparin indirect anticoagulants are appointed (phenylin, varfarin), because they in the onset reduce the level of protein C, that can cause thromboses. Duration of treatment by indirect anticoagulants must make no less than 3 months.

Low-molecular heparins (LMH) are used during last years with success - fraxiparine, clexane, fragmin. Positive moments in the use of LMH are high bioavailability, rapid absorption in hypoderm, small rate of introduction — 1–2 times per days, good tolerance, rare development of complications.

With the medical purpose fraxiparine is entered 2 times per day with an interval of 12 hours in next doses depending on body mass of patient.

Treatment proceeds during all period of increased risk to the moment of complete renewal of motiion activity of patient, but no less than 10 days.

Contra-indications to introduction of LMH are: acute bacterial endocarditis, thrombocytopenia at presence of bleeding or predisposition to it (except for the

coagulopathy of consumption), gastric and duodenum (acute period) peptic ulcer, vascular hemorrhage of cerebrum. It is necessary to apply preparation with caution in pregnant and patients with renal-hepatic insufficiency.

At treatment of LMH with carefulness it is necessary to appoint preparations, containing an acetophene, non-steroid anti-inflammatory preparations due to their possibility to potentiate action of LMH.

3. Infusion therapy. The guided, slow introduction of fluid (colloid solutions) is indicated under control of CVP. At an increase CVP more than 15 cm of H_2O it is necessary to stop the infusion. The infusion therapy improves the condition of hemodynamics and oxygenation of tissues.

4. Inotrope support. Dobutaminum in the dose of 5–10 mg/kg/min provides the adequate pre-loading, increases a cardiac index, elimminates hypotension, reduces heart rate, diminishes resistance of pulmonary vessels. Analogical effects has Dopaminum in the dose of 5–17 mcg/kg/min, however at its introduction pressure can increase in pulmonary artery, and tachycardia develops in the number of patients. Noradrenalinum provides the inotrope function of heart effectively, promotes cerebral and coronal perfusional pressure.

5. Mezaton is used for renewal of vascular tone and rapid increase of arterial pressure at collapse.

6. Supporting antithrombotic disaggregant therapy: reopolyglucinum (400 ml), trental (5 mcg/kg), nicotine acid (2 mcg/kg per day) during 5–7 days, new effective disaggregant plavix (clopidogrel).

7. Following of the strict bed regimen. Giving of correct position abed is necessary.

Surgical treatment

At pulmonary embolism of trunk of pulmonary artery in inefficacy of conservative thrombolytic therapy the operation is used: embolectomy in the conditions of artificial circulation of blood.

As alternative presently there is minimally invasive operation: open surgical intervention - thrombectomy from a pulmonary artery by the catheter of Fogarty from peripheric access. After the angiopulmonography, determination of location and size of thrombus-clot under X-ray guidance a probe is entered and mechanical destruction of blood clot is made with subsequent introduction of thrombolytics.

Prophylaxis of development of PE

The non-medicinal measures of prophylaxis include: early activation of patients in postoperative period, in the heart attack, stroke of cerebrum; bandaging of shins and thighs by elastic bandages; remittent pneumatic compression of cuffs, imposed on a shin.

The medicinal correction of the system of hemostasis is carried out by the small doses of heparin which is appointed hypodermic 5000 unita each 6-8 hours. Treatment is begun before 2 hours to the operation and continue during 7-10 days after it or up to the discharge of patient from department. If necessary it is continued to enter a heparin in ambulatory terms. Application of heparin diminishes the risk of nonlethal PE on 40%, lethal - on 65%, DVT - on 30%. For patients with the high risk of development of hemorragic complications (after operations on head and spinal cord) in place of heparin daily infusion of low-molecular dextrane (reopolyglu-

cinum) is used which is appointed intravenously at a rate of infusion 10 ml/kg during the first 24 hours, after operation - 500 ml/day during 2-3 days. An aspirin appeared ineffective in prevention DVT.

In the last decade for the prophylaxis of postoperative DVT LMH is widely used, application of which does not require regular laboratory control, and rarer, than at the use of standard heparin, is accompanied by development of bleeding and thrombocytopenia. Thus the dose of preparation is determined depending on the degree of risk of development of DVT: at high risk dose is increased as compared to such in the case of moderate risk. With prophylactic purpose LMH is appointed hypodermic 1 time per day taking into account body mass of patient: fraxiparine 0,3-0,6 ml, clexane 0,2-0,4 ml, fragmin 2500-5000 units. Duration of prophylactic application of preparation must be no less than 10 days (sometimes up to 3 months) (Fig. 90).

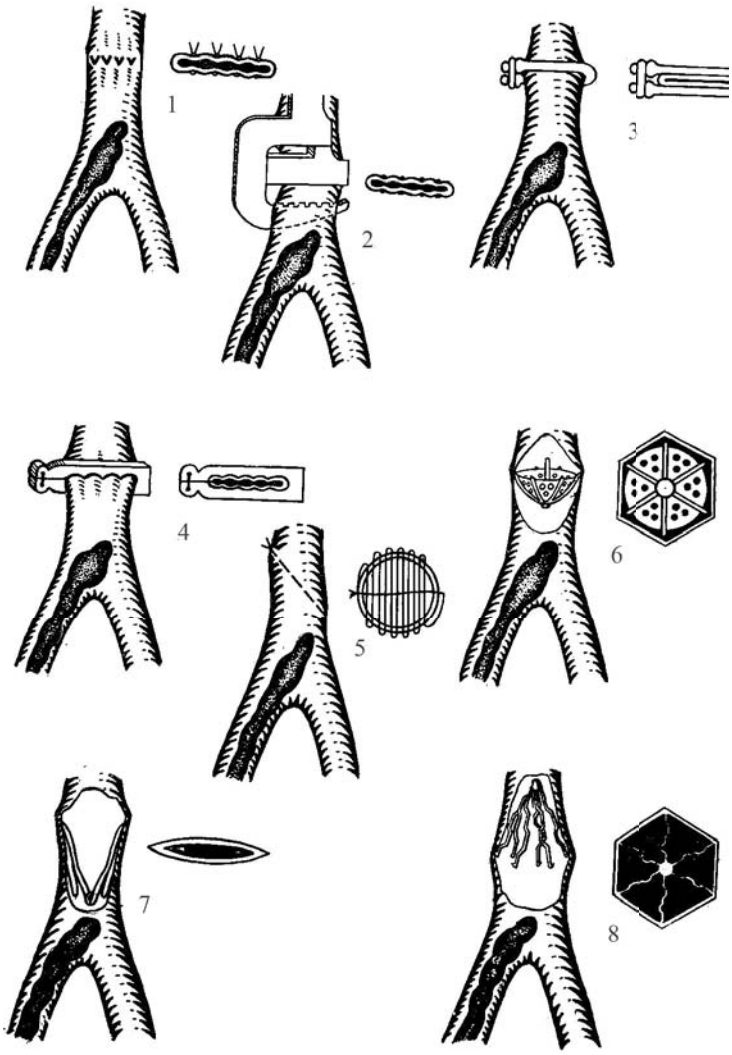

Fig. 90. Methods of partial occlusion of vena cava inferior and different types of cava-filters

The basic methods of surgical prophylaxis is implantation of cava-filters. Transcutaneous implantation of cava-filters is most widely used presently, indications for it are: contra-indications to anticoagulat therapy or severe hemorragic complications at its application; recurrence of PE or proximal distribution of phlebothrombosis on background of adequate anticoagulant therapy; thromboembolectomy from a pulmonary artery; extensive «floating» blood clot in ileocaval venous segment; DVT/PE in patients with low cardiopulmonary reserve and severe pulmonary hypertension; high risk of development of DVT/PE (major surgical interventions, fractures of bones and spine) in patients with PE in anamnesis; PE in pregnant as adding to therapy by heparin or at contra-indications to application of anticoagulants; unsuccessfulness of the before used methods of treatment of DVT/PE; thromb-endarterectomy in patients with post-embolic pulmonary hypertension; DVT/PE in patients after transplantation of kidney or heart.

Examination of disability and follow-up in patients with PE

At I and the II degree of embolism and adequate treatment a prognosis is favorable, at III and especially the IV degrees lethality is high, because adequate help is late as a rule. At recurrent chronic embolism of pulmonary artery treatment by the anticoagulants of indirect action is indicated, in case of occurring of indications is setting of cava-filter. Patients must be on clinical supervision.

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VI.4. Postthrombophlebitic syndrome

*The **Postthrombophlebitic** syndrome (PTPS) is complex of symptoms, developing at patients which carried the acute thrombosis of deep veins, and is cause of vein circulatory disorders in extremities due to incompetence of valvular system of deep veins after a thrombosis in the areas of recanalization, absence of ability to passage of blood in system of deep veins and valvular insufficiency of hypodermic and perforated veins in connection with the overload by the vein blood stream of basic ways of collateral vein outflow.*

It is characterized by pain, by the edema, by the second varicose dilatation of hypodermic veins, by the trophic changes with hyperpigmentation of skin, induration of soft tissues of distal segments of extremity, by trophic ulcers.

According to statistical information, in different countries by this disease suffers 1, 5 — 5 % populations. PTPS is the reason of chronic vein insufficiency at 96 % patients with the affectation of the system of vena cava inferior and only at 4 % of patients chronic vein insufficiency is conditioned by other factors. The basic contingent of patients with PTPS is made by the persons of young and middle ages from 20 to 50 years. Thus at women PTPS develops more frequent, than at men. It is explained by more frequent origin at the women of the acute vein thromboses, related to births, abortions.

In Germany of PTPS meets more than at 1 million persons, in the USA - at 6-8 million patients (from them at a 500 thousand - trophic ulcers). In the countries of UIS PTPS meets at 28% of patients with different types of vein pathology, in 43-79% of cases it resulted in development of trophic ulcers. These patients, as a rule, invalids of the II-III groups, they make the considerable percent of surgical policlinic receptions, in connection with difficulties and small efficiency of treatment of trophic ulcers of this etiology surgeons name this contingent of patients with the «cross of surgeon».

Anatomy of veins of lower extremities

Veins of lower extremities are the well developed thick network with the numerous variants of fork. The general lumen of veins of lower extremities in 10-12 times is greater, than arteries. Distinguish hypodermic, deep and communicant veins.

Hypodermic veins on periphery are forming the thick network which anastomoses between itself and with deep veins. A large hypodermic vein (v. saphena magna) is the most long vein of man - collects a blood from a 2/3 surface of extremity. It rises on the anterior-internal surface of lower extremity and in inguinal region confluences in a femoral vein. In upper third of thigh a large hypodermic vein has two large branches: v. saphena accessoria medialis et lateralis. Near the place of inflow of them in a large hypodermic vein in it inflows v. pudenda externa superficial, v. epigastrica superficialis et v. citcumflexa ilium superficialis. Before the inflow they can meet in one trunk or are separate little trunks (from 1 to 5-6 and anymore) and act important part in development of postoperative relapses of varicose expansion of veins.

A small hypodermic vein (v. saphena parva) collects a blood from the external surface of foot and back surface of shin. In upper part of shin it passes between the heads of gastrocnemius muscles in duplicator of fascia of shin and falls in a popliteal vein. There are a lot of variants of inflow and anastomose of small hypodermic vein.

The deep veins of shin on two accompany of the same name arteries. This: 2 back tibialis, 2 anterior tibialis and 2 fibular. In upper third of shin to popliteal fossa they meet in a large popliteal vein which passes in popliteal fossa and on a thigh proceeds as superficial femoral vein, in upper third of thigh it unites with the deep vein of thigh, forming short general femoral vein which higher than inguinal ligament in small pelvis passes to the external iliac vein. The last unites with an internal iliac vein, forming general iliac vein. Connection of right and left general iliac veins are forming lower hollow vein.

The communicant veins are placed under fascia shins and thighs, in muscles and between them. Part of them perforating fascia and connects subcutaneous veins with deep, name them perforant (direct and indirect). Direct perforant veins predominant in lower part of shin. The communicant veins provide functioning of muscles pump of vein outflow of blood.

Very important anatomical peculiarities in venous system are presence in it structure *valves of vein.*

In superficial and deep veins valves make way for a blood only in central direction, preventing the retrograde current of blood in vertical position and at the physical loadings, the same protecting veins from the considerable increase of intravenous pressure.

The valves of communicant veins of lower extremities make a way for a blood only from superficial veins to deep, preventing the overload of more weak superficial veins. In addition, slammed at the retrograde current of vein blood on deep veins, valves fragment the vein column of blood on separate segments, considerably diminishing the negative influencing of hydrostatical pressure of column of blood, is special on the walls of veins of distal parts of extremity.

Deep and superficial veins of upper extremity is widely anastomose one with other, thus these anastomoses does not have valves and a blood stream can be freely carried out from superficial in deep veins and back.

The normal functioning of the vein system of lower extremities is conditioned by effective activity of row of physiological mechanisms: valvular apparatus, by the mechanism of action of shin muscles which name a «muscular pump» and «vein heart»; by the «pump» function of heart; by negative intrathoracal pressure which «sucking» the vein blood; fascias vaginas of extremity, which execute the role of «prop» for deep veins.

The walls of veins, as well as arteries, have three layers, but a muscles layer and adventitia is less developed, a wall is thinner, that is the cause of easy collapse of vein vessels.

Etiology, pathogenesis, the PTPS classification. Pathogenesis formation of trophic ulcer at patients with PTPS

In basis of origin of chronic vein insufficiency at PTPS lie two pathophysiology mechanisms:

1) violation of valvular system of deep and perforant veins,
2) permanent vein hypertension in lower extremity.

In pathogenesis of PTPS distinguishes two stages: the stage of occlusion and stage of recanalization.

As it is already marked, development of regional vein hypertension which in the stage of occlusion of deep veins is conditioned by the presence of blockade (blood clot) of vein circulation lies in basis of the PTPS pathogenesis, and in the stage of recanalization - insufficiency of valvular system and, as a result, pathological influencing of considerable gravitation of vein blood column, origin of pathological retrograde blood stream in deep and perforant veins; relapsing thromboses are characteristic.

Vein hypertension increases loading on communicant and subcutaneous veins, on lymphatic collectors which at the beginning of disease compensate disordered magisterial vein hemodynamic; but after some period decompesation of blood and lymph circulation (on the level of microcirculation) that is the reason of development: 1) secondary varicose dilatation of subcutaneous veins, 2) increases of edematic process due to the expressed regional depositing of vein blood - chronic venous stasis («vein bog»), 3) deep violations of microcirculation with the lowering of transcapillary exchange, which leads to the ischemia of tissues and making progress of trophic violations in tissues of distal parts of lower extremity - to development and making progress of hyperpigmentation, indurative cellulites and trophic ulcer (most often in area of internal part of ankle). Thus, the expressed clinical picture of syndrome of chronic vein insufficiency of lower extremity develops at a patient.

Pathomorphology. In main veins there are the considerably expressed sclerotic changes with involving in the process of valves. Histological determine the sclerosis of all vein membranes with atrophy and necrobiosis of the functional active elements (smooth muscles and elastic membranes). Intimae of such vessels are sharp and unevenly thickened. Thus there are both the areas of hypertrophy and areas of the sharp thinning up to disappearance of structural elements. The valves in such veins are absent also (S.A.Borovkov, 1978).

Pathogenesis of trophic ulcer formation at patients with PTPS

The characteristic display of PTPS is induration of soft tissues in lower third of shin, conditioned by development of fibrose changes in a skin and subcutaneous cellulose (lipodermasclerosis). The changes are especially sharply shown in the area of medial surface of ankle. A skin in this area adopts the brown or umber colours, becomes dense and immobile.

On the changed area of skin often there is a weeping eczema which is accompanied by an itch. Quite often the area of hyperpigmentation and induration of skin affected of the lower third of shin as a ring (“armour” fibrose). On this area of shin the edema is absent, higher it is expressly expressed.

Trophic vein ulcers which are usually disposed on front-internal surface of lower third of shin above an internal part of ankle in the area of lipodermasclerosis are heavy complication of disease. They are characterized by the persistent relapses. More frequent there are ulcers single, rarer are plural with sclerotic borders. The bottom of ulcer as usually flat, covered by languid granulations, discharge is scanty

with an unpleasant smell. The sizes of ulcers vary from 1-2 cm up to the ulcers which occupy all circumference of lower third of shin. In case adding of infection they become very painful, and may complicated by microbe eczema, pyodermia, dermatitis.

The most widespread theory of pathogenesis of vein ulcers of shin and changes of skin and hypodermic cellulose preceding to them appearance of skin changings connecting with the increase of pressure in the vein ends of capillaries of microcirculatory bed of skin, by violation of diffusion in capillaries, increase of permeability of their walls, output of albumen (including fibrin) to interstitial space. It results in accumulation of fibrin around of capillaries as a cuff which compresses them (it is confirmed by the histological study biopsy of the skin). Assume that concentration of fibrin around the capillaries of skin is a barrier which hinders to diffusion of oxygen and nutritives to the cells of skin and hypodermic cellulose. As a result of it there is tissue hypoxia, violation of feed of cells and focal necrosis. Endothelia of capillaries and cellules, which hold out in a fibrin cuffs, can operate as chemo attractants and activators of leucocytes and thrombocytes, which discharge anti-inflammatory interleukins, active oxygen radicals, and factor of activation of thrombocytes. Hereupon there are favorable terms for the thrombosis of capillaries, hypoxia of tissues, damages of cells of skin and hypodermic cellulose by oxygen radicals. As a result there are micro necrosis of tissues and chronic inflammation, which are result formation of trophic vein ulcers.

Some authors consider that vein hypertension as incident of chronic vein insufficiency support discharge by the endothelial cells of interleukins and adhesion molecules. In this connection leucocytes adhesion to endothelium of capillaries of the microcirculatory system of skin (regional standing of leucocytes), that is the first step to creation of favorable terms for the delay of them in the capillaries of the microcirculatory system of skin and difficulty of circulation of blood in a microcirculatory bed. In this connection there is stagnation of blood, thrombosis of capillaries, opening of arteriovenous shunts because of difficulty of outflow of vein blood on capillaries, ischemia of tissues. The fixed in capillaries of skin leucocytes get to interstitial space, produce active oxygen radicals and lysosomal enzymes which cause necrosis of skin and formation of ulcer.

In the process of development of trophic ulcer distinguishes three phases: exudation, reparation and epithelization. *The phase of exudation is* accompanied by the expressed perifocal inflammation, by the focal necrosis of tissues, by the considerable semination of ulcer by microorganisms, dense wound's discharge with an unpleasant festering smell. Sometimes an inflammatory process from an ulcer spreads on lymphatic vessels, with formation of eczema, erysipelas inflammation, lymphangitis, thrombophlebitis. Frequent intensifications of local infection can result in obliteration of lymphatic vessels and appearance of the secondary lymphostasis of distal parts of extremity.

In the phase of reparation the surface of ulcer gradually clears up from necrotic tissues, is covered by fresh granulations. Peryfocal inflammation and purulent discharge diminish.

The phase of epithelization is characterized by appearance of strip of fresh epithelium on the edges of ulcer. The surface of it is covered by clean, fresh granulations with the insignificant serous discharge and than formation of scar.

In a clinic used classification of the PTPS by V. C. Saveliev and al. (1972)

On localization:

– lower (femoral-popliteal) segment,
– middle (iliac-femoral) segment,
– upper (lower hollow vein) segment;

On a type:

– localize,
– spread;

An in due form:

– edema,
– edema-varicose;

On stages:

– compensation,
– decompesation without trophic violations,
– decompesation with trophic violations.

A.A.Shalimov (1984) selects **the following forms PTPS:**

1) edema-pain,
2) varicose-ulcerous,
3) mixed.

A. N. Vedensky (1986) selects *two forms* of disease – *sclerotic and varicose and three stages*:

The I stage -occlusion and beginnings of recanalization, is characterized by expressed vein hypertension and insufficiency of vein hemodynamic without development of trophic violations;

II stage - complete or partial recanalization, destruction of valvular apparatus and development of pathological retrograde blood stream in deep and perforant veins, is characterized by some diminishment of expressed of pathological symptoms (fatigueability and edema of extremity), by appearance of initial signs of trophic disorders in tissues - hyperpigmentation and indurative cellulites;

III stage - the expressed chronic lymph and venous insufficiency, is characterized by development of heavy trophic changes of soft tissues of extremity – trophic ulcers.

Classification of chronic vein insufficiency (E.G. Jablokov and al., 1972)

Degree	Basic symptoms
0	It is absent
I	Syndrome of «heavy feet», passing edema
II	Permanent edema, hyper- or hyperpigmentation, lipodermasclerosis, eczema
III	Vein trophic ulcer opened or beginning to live

Variants of clinical features

Sclerotic form. For this form is characteristic absence of the pathologically extended veins of extremity on the stage of disease and insignificant expansion of main hypodermic veins in the areas of localization of perforant veins in the II and III stages. The basic trunks of large and small hypodermic veins do not have the signs

of pathological expansion. The indurative process in a hypodermic cellulose and hyperpigmentation of skin of shin is more expressed, comparative with other forms of postthrombophlebitic illness. At a sclerotic form there is so-called armour fibrosis of cellulose, which can be localized in lower third of shin. Taking into account it, in this part of shin the edema is not noticeable, but under him it is sharply expressed.

Varicose form. Patients have the skin of feet of the ordinary coloring, the edema of shins is expressed insignificantly, but hypodermic veins extended and varicose changed. Their localization usually specifies on a level of affectation of main veins. So, in case of involving to process of ileo-femoral segment usually there are expansions simultaneously of large and small hypodermic veins and insufficiency of communicant veins of distal parts of shin. At complete occlusion of main veins of pelvis or limited occlusion of iliac vein varicose expansion of superficial veins is localize, mainly, in upper third of thigh and lower part of anterior abdominal wall. At occlusion of distal part of lower hollow vein varicose dilatation can be noticed on both lower extremities and lateral surfaces of the abdominal wall and thorax.

Edema-pain form. It develops at once after the acute phenomena of thrombosis of main veins with the appearance of pain, edema and moderate cyanosis.

Pain carries segmental character, may be local on projection of nerves, vascular bunch of shin and thigh. Nevertheless, when patients occupy horizontal position with heaved up extremity, distribution of pain, feeling of weight and fatigueability of the affected extremity gradually disappears. The degree of edema is in direct dependence on heaviness of hemodynamic changes in main veins. So, at the limited affectation of femoral and popliteal segments there are the moderate increase of volume of shin and small edema of third of thigh. At the same time, spreading of process on an iliac-femoral segment sharply increase of the volume of all extremity and serves as the reason of edema on a buttock. At complete or limited occlusion of pelvic veins also there can be the diffuse edema of extremity. In case of affectation of distal part of lower hollow vein develops the extraordinarily expressed edema of both extremities up to elephantiasis. Although at this form of disease of expansion of hypodermic veins is absent.

At the satisfactory state of compensation of vein outflow the edema form of disease sometimes disappears during a few months. At same patients compensation is so well developed, that not a visible foundation is for the diagnosis of postthrombophlebitic disease, although there is segmental obliteration in deep veins.

Ulcerous form. At patients with this form of disease it is possible to mark all above mentioned symptoms, and in addition sharply expressed. The edema of leg even after the stay of extremity in a state of rest do not disappear fully; it is increases a degree and distributions of varicose expansion of hypodermic veins, there are pigmentation of skin and infiltration of hypodermic cellulose with diffuse their distribution on all lower half of shin. Trophic ulcers which are formed on the medial surface of lower third of shin are accompanied by an itch and trophic changes of skin and hypodermic cellulose.

Clinical symptomatic at *ileofemoral PTPS* is the same, as well as at the affectation of deep veins of lower extremity, but has some features. Pain more frequent is local-

ized in an inguinal region, on the front internal surface of thigh or dorsal muscles of shin. Sometimes pain in extremities is accompanied by pains in a lumbosacral region. The edema at such localization of PTPS characteristic not only prevalence on all extremity on the affected side, but sometimes and on genital organs and buttock, however, the most expressed edema is always on a thigh. Due to several edema the thigh can be multiplied the circumference up to 10 cm and more. At some patients with a long-term existent ileofemoral thrombosis may be marked considerable varicose expansion of hypodermic veins of anterior abdominal wall on the side of affectation and above pubic region.

***At questioning of patient*:**

1). *Complaints* from the basic disease: swelling, sense of heavily, varicose expansion of hypodermic veins and pain in the affected extremity which increases at the long stay up. Pain is dull, and only some times may be intensive, decreases in lying position of patient or with elevated leg. Quite often patients are disturbed by cramps of dorsal muscles of shin during the long standing and in a night-time. The edema usually arises up by the end of day, after nightly rest with the elevated position of feet they diminish. Also patients can complain on hyperpigmentation of skin covers and on the presence of ulcers of shin.

2). Complaints from the side of other organs and systems (if they are).

3). Anamnesis of disease: patients mark the presence before acute thrombosis of deep veins of lower extremities. The disease makes progress slowly, the stage of flow is traced. It is specified, whether applied for medical help and where, or was engaged in self-treatment, what medicines were adopted by an effect from them. Remote anamnesis - from what time is ill, where and by what treated oneself, with what effect, information of previous auxiliary methods of inspection - laboratory, roentgenologic, instrumental.

4). *Anamnesis of life:* infectious diseases, trauma, operative interferences, births at women, malignant formations, allergic diseases, harmful habits, terms of work, professional harmfulness and others.

Clinical physical inspection (characteristic features at this disease):

1) Estimation of the common state of patient.

Consciousness clear. Personal constitutional peculiarities, fatness at this disease are not traced.

2) Collection of information about original appearance of patient (examination of skin, hypodermic fatty layer, palpation of lymphatic nodes, thyroid and mammary glands) – without features.

3) Inspection of the state *of organs of breathing:* (examination of thorax and upper respiratory tracts, palpation of thorax, percussion and auscultation of lungs; determination of the special symptoms) - without features.

4) Inspection of the state *of organs of abdominal cavity:* (examination of abdomen, palpation and sucussion of the stomach, palpation of intestine, livers, spleens, pancreas, kidney, organs of small pelvis, auscultation of stomach) – more frequent without features (sometimes there can be varicose expansion of hypodermic veins of anterior abdominal wall on the side of affectation and above the pubic).

5) Inspection of the state *of bones-muscles systems:* (examination and palpation) - without features.

7) «***Locus morbi***» is inspection of the state of the cardio- vascular system: examination and palpation of area of heart and superficial vessels, determination of pulsation of main arteries of extremities and neck in projection points, determination of percussion borders of heart, auscultation of heart and vessels (pathology can not be determined).

From the side of affected extremity: the edema of foot and shin, expressivity and spreadness of which allow to judge about the level of affectation; very common symptom is compensatory evolution of the secondary *varicose dilatation of hypodermic veins of shin* and thigh, anterior abdominal wall is a frequent symptom; presence of dermatitis; trophic *disorders on* a skin and in the hypodermic cellulose of a different degree of expressed – from the spots *of hyperpigmentation and induration (indurative cellulites)*, dermatitis *and eczema up to the heaviest disorder – trophic ulcers* (Fig. 91)(ordinary localization of trophic violations is distal third of shin, mainly in a medial malleolus region, sometimes they circularly affected distal third of shin as a ring); at palpation – painfulness on way of projection of deep veins in the affected segments (shin - on the middle of back surface and along the medial surface of tibia, on a anterior-medial surface on projection of vascular bunch on a thigh).

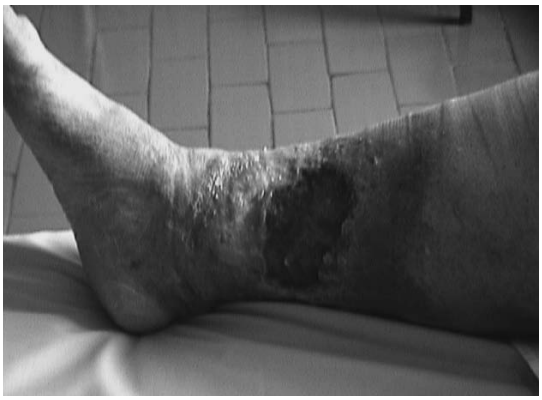
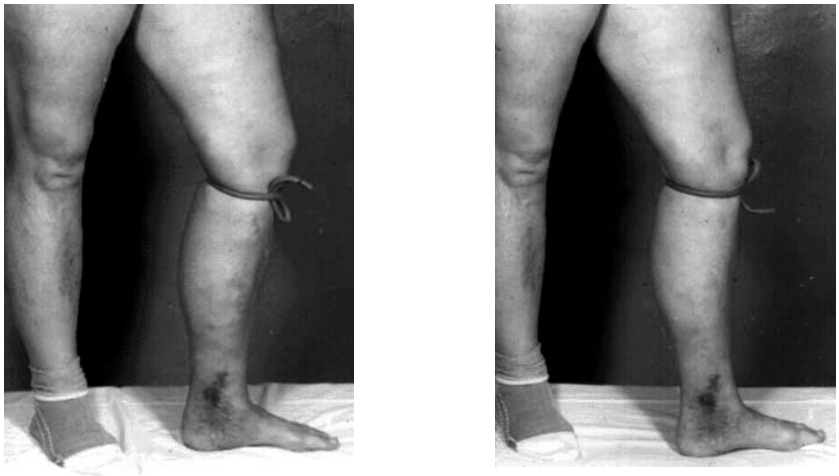


Fig. 91. Trophic ulcer by PTPS

The picture of ability to passage of blood thought the deep veins may be estimated by the "March test" of Delbe-Pertes and the Pratt -1 test.

"March test" of Delbe-Pertes (Fig. 92). To the patient in vertical position at the maximally filled hypodermic veins below knee-joint applied tourniquet, which mast compresses only the superficial veins. Then ask a patient to walk during 5-10 mines. If here hypodermic veins and varicose knots on the shin are disappeared or became empty, it means that deep veins are passable (positive test). If veins after loading do not become empty, tension of them by palpation does not diminished, and vice versa increased, distended pains in a shin appear at a patient, the result of test indicates on impassability of deep veins (negative test). Sometimes the negative result of test can be conditioned by considerable incompetence of valves of perforant veins or can depend on the wrong conducting of test (squeezing of deep veins by the applied tourniquet), from the presence of strong sclerosis of superficial veins, which hinders to decline of their walls. In such cases it is necessary to repeat a test.

The Pratta-1 Test. After measuring of circumference of shin (level it should be noted, that to conduct the repeated measuring thereon level) of patient lay on the back and empty veins from a blood with help elevation of extremity. On a leg (beginning from below) an elastic bandage is tightly laid on, reliably to compress the hypodermic veins. Then to the patient suggest walking during 10 mines. Appearance of pain in muscles of shin specifies on impassability of deep veins. The increase of circumference of shin after walking at the repeated measuring confirms this supposition.



a b

Fig. 92. "Marsh test" Delbe-Perthes: a - protruding above the skin varices exercise; b - the disappearance of varices after loading

8) *Leading clinical symptoms (syndromes)* characteristic for PTFS: pain in extremity, edema.

9) On the basis of findings of questioning and clinical physical inspection of patient to propose *a preliminary clinical diagnosis* to the patient (example): postthrombophlebitic syndrome, localize type (lower segment), edema-varicose form, stage of decompensation without trophic disorders.

In accordance with standard charts the plan of additional inspection (laboratory and instrumental) of patient with PTPS includes:

- Clinical blood test: changes unspecific are possible leucocytosis with shifting of formula to the left side, the ESR increases.
- Clinical analysis of urine: the changes can not be present.
- Biochemical blood test: possible increase of transaminases, C-reactive albumin.
- Coagulogramm: often hypercoagulation (shortening of time of coagulation, increase of prothrombin index, tolerance of plasma to heparin, increase of content of fibrinogen A and B with the simultaneous lowering of fibrinolytic activity or lengthening of time of fibrinolysis, diminishment of free heparin).
- US of vein vessels and dopplerography:

Ultrasonic dopplerography allows to detect expansion of lumen of veins, increase of echogenic of walls, stage of organization of process and degree of violation of ability passage of veins, and also degree of expressed of valvular insufficiency.

Ultrasonic classification of hemodynamic violations at PTPS (V. G. Leluk, 2003):

Initial degree - at background research a retrograde blood stream is absent, amplitude of inducted retrograde blood stream less amplitude of base, length of it no more than a 200 mc.

Moderately expressed degree - at background research a retrograde blood stream is absent, appearance of the prolonged retrograde blood stream (more than 200 mc), that exceeds base amplitude.

Heavy degree — there is a spontaneous retrograde blood stream amplitude of which is considerably increases at loading tests, or a spontaneous retrograde blood stream is absent, after a loading test appears high-peak dual direction blood stream

Phlebography: contrasting research of vein vessels of extremity allows to describe the condition of deep, superficial, perforant veins and collateral blood stream, and also to detect all pathological changes in the vein system of lower extremity, which determines a necessity and volume of operation.

Depending on the PTPS localization is executed *distal (ascending) and proximal (pelvic) phlebography*.

The angiographic signs PTPS are: absence or unclear of contrasting of main deep veins on a shin and thigh, narrowing of their lumen, contrasting of superficial, often varicose changed superficial veins (what must not be at competence of valves of communicant veins) (Fig. 93, 94).

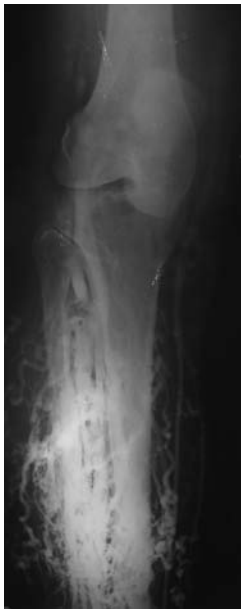


Fig. 99. The distal phlebography - non clear contrast of deep vein trunk and contrasting of the superficial varices in the localization of PTPS in the lower segment

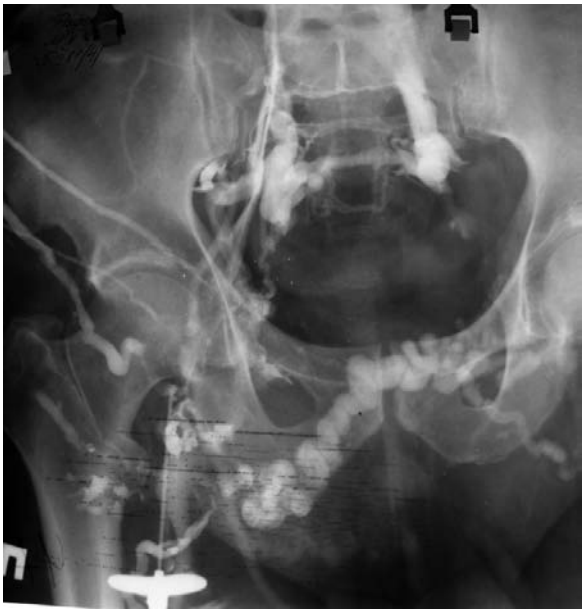


Fig. 100. The proximal (pelvic) phlebography - occlusive-constrictive process in general right femoral and iliac arteries, availability of collateral venous circulation by superficial varicose veins of the anterior abdominal wall in the localization of PTPS in the middle segment

Foremost, ***differential diagnostics*** must be conducted between varicose illness of lower extremities with primary varicose dilatation of veins and second varicose dilatation of veins of lower extremities at PTPS.

Varicose illness of veins of lower extremities more frequent diagnose at women. It arises up unnoticed and slowly makes progress, develops higher and below than knee-joint and can have cylindrical, saccular, serpentine types, the edema in the stage of compensation diminishes after rest. The function of valves of deep veins is not affected; deep veins are passable (positive result of the "March test"). Trophic

ulcers are signs inherent to the stage of decompensation of vein circulation of blood of lower extremity.

The varicose form of PTPS characteristic by the presence of the acute thrombosis of deep veins with the expressed edema, which is preceded dilatation of hypodermic veins. At PTPS violation of function of valves of deep veins is present, the negative result of the "March test" testifies on impassability passage of deep veins with quick develops of sclerotic changes in hypodermic cellulose and trophic changes of skin covers. An eventual conclusion is determined by phlebography.

Certain difficulties always arise up at differential diagnostics with *lymphostasis.* At patients with lymphostasis the edema is diffuse, takes an ankle joint, foot, and sharply may spreads to the shin and thigh. The edema as usually elastic and diminishes after rest. Important there is that the symptoms of lymphostasis in majority of cases appear after erysipelas inflammation.

At PTPS the edema tense, localization of which depend from level of affectation, and may spreads on all lower extremity and infrequent their distribution on a foot. It is slightly diminishes after rest and combine with the trophic changes of skin.

Postthrombophlebitic illness must be differentiated with *innate angiodysplasias,* which show up the thickening and lengthening of the affected extremity, pigmented spots and angiomatose dilatations of hypodermic veins with atypical localization (foot, buttocks and external surface of extremity). Rarer there are a pulsation and systolic and diastolic noises in the area of angioma and artery- venous fistulas, formation of ulcers and decompensation of heart. Confirm a diagnosis artery- and phlebography, pigmentation of skin, determination of tension of oxygen in a blood.

On the II stage of PTPS, in mostly cases on the medial surface of lower third of shin, appear hyperpigmentation of skin and induration of hypodermic cellulose, the trophic changes develop on a background of dermatitis. In addition, localization of changes of skin not always is typical. In this connection, there is a necessity in conducting of differential diagnostics with such skin diseases, as indurative erythema and illnesses of the nervous system.

It is also necessary to conduct the differential diagnosis PTPS with the diseases which are accompanied by development of chronic ischemia of tissues of extremity with the making progress symptoms of trophic disorders (obliterated atherosclerosis, endarteritis and thrombangitis).

The ground and formulation of clinical diagnosis of patient (taking into account classification of disease, presence of complications and concomitant pathology) - an example:

1) *basic* - Postthrombophlebitic syndrome of femoral- popliteal segment, localize type, edematic form in the stage of compensation.

2) *complication* - the acute vein thrombosis of femoral-popliteal segment (if it is).

3) *concomitant pathology* (if it is).

Treatment of patient with a postthrombophlebitic syndrome.

Choice of medical tactic: exists the conservative and surgical treatments PTPS.

The conservative is the basic method of the PTPS treatment (although V.S.Savelev considers that the surgical method of treatment in the stage of occlusion can be only effective, because conservative treatment gives a positive effect only approximately at 8% of patients).

However in connection with that the results of operative treatment can not be named enough effective, the surgical method of the PTPS treatment is applied only at the heavy progressive form of disease.

Leading place in **conservative treatment** is taken *by compression therapy* of extremity by *the elastic bandaging* (by elastic bandages) or «compression knitted fabric» (carrying of the elastic stockings), is desirably permanent with the removal of them for the night (Fig. 95).

Most effective in this case there is application of compression *zinc-gelatinous of bandage - the Unna «knee-boot»*, which is applied on 1,5-2 months, which results in considerable diminishment of edema of extremity and healing of trophic ulcers (in connection with the removal of «vein bog» and improvement of trophic of tissues); its recipe: Zinci oxydati, Gelatini aa 60,0, Glycerini 200,0, Aqvae destillatae 180,0.

Medicinal therapy directed on diminishment of edema, improvement of exchange processes in tissues of extremity and vein hemodynamic, correction of hemocoagulation, struggle against dermatitis, with an infection in an ulcer, on stimulation of healing process in ulcer - diuretics, vitamin therapy, desagregants, hemocorrectors, if it necessary - anticoagulants, and also vein tonics - venoruton, troksevasin, diovenor, ziclo 3 fort, detralex; at presence of trophic ulcer are ointment preparations of opposite microbes, sanitation action *in the I phase of wound process* mainly on hydrophilic basis - levomycol, levosin, levomicol-m, dioxycol, oflocain, miramistin 0,5%, nitazid and metrogil -jelly (for influence on a nonclostridial anaerobic flora), argosulfan (silver salt of sulfatiasole), titriolin-cream (the operating beginning is the extract of butter of tea tree), liquid form of iodinedizerin; for stimulation of granulations in II- phase — methylurazil (5 %), pantestin, streptonitol, iruxol (with an enzymatic component), other indifferent ointments which contain an antibiotics or antiseptics (furacilin, streptoid, syntomicin emulsions, tetracyclin, gentamicin and others); in the III-h phase of epithelization and scarring - methyluracil 10 %, curiason (drops and jellies); universal (for all phases) preparation for local application on a hydrophilic basis - complar (dimexid, dioxydine, anesthesine, extract of ariny).

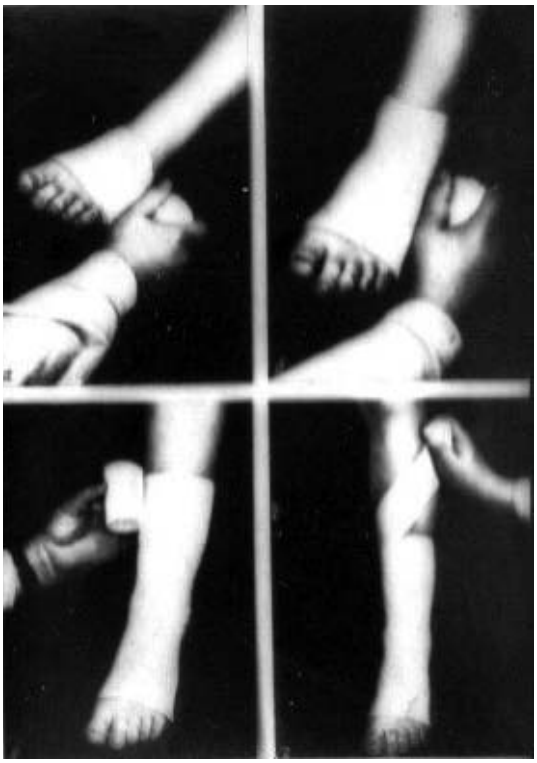


Fig. 95. Elastic bandaging

Expedient administration of physiotherapy treatment – ionic galvani-

zation with iodine potassium, courses of sulphuretted hydrogen, radon and other baths, electrophoresis with lidase and others.

The PTPS treatment is extraordinarily difficult. Conservative therapy expected only on the postponement of heavy consequences of violation of vein hemodynamic and often is symptomatic. The disease makes progress steadily.

Therefore **different methods of the operative treatment** of **PTPS** are used in clinical practice.

Surgical treatment. All reconstructive operative interferences at PTPS dividing on two groups:

1) executable in stages of occlusion - directed on normalization or improvement of blood stream on deep veins and on formation of additional ways of vein outflow

2) executable in stages of recanalization executable - directed on the decrease of hydrostatical pressure in deep veins, which prevent passage of blood to the superficial veins and which diminish an arterial influx in extremity.

I group:

1. Operations which fully remove violation of main vein blood stream:

a) complete thrombintimectomy;

b) prosthetic of segment of occlusion vein (by the segment of healthy vein, synthetic prosthetic graft).

2. Operations which improve a main vein blood stream:

a) partial thrombintimectomy;

b) bougienage of occlusion vein;

c) fascial plastic - the Askar-Zelenin operation (dissection from back access on the shin of deep fascia and sewing together of it as duplication - a muscles sheath narrowing and due to it compresses the deep veins in the period of muscles contraction, which almost fully redacting relative valvular insufficiency of deep veins, improves the suction function of shin muscles).

d) operations directed on formation of additional ways of vein outflow - operations of bypass shunting Palm-Esperon (in a femoral-iliac segment) and Uoren-Tayr (in a thigh-popliteal segment) (Fig. 96).

II group:

1. Operations which reduce hydrostatical (gravitation) pressure of column of vein blood, which prevent the retrograde reflux of vein blood and its depositing in the distal sections of extremity:

a) creation of artificial valves – extravasal by Psatakis from the m.gracillis and intravasal by Karavanov — from the mouth of v. Saphena magna;

b) the resection of deep veins in different segments — the Bauera (resection of popliteal vein, 1948) operation, the Lynton-Khardy (resection of femoral vein, 1948) operation, distal resection of dorsal tibial veins in lower third of shin (A. N. Vedensky, 1976).

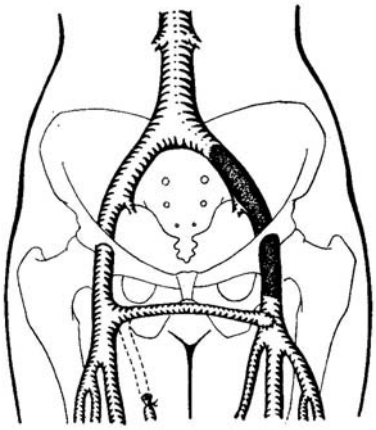


Fig. 96. Operation of bypass shunting Palm-Esperon

2. Operations, which prevent outflow of blood to superficial veins:

a) veinectomy on Bebcokk-Narat;

b) operation of Lynton — subfascial bandaging of communicant veins from medial access (1938)

c) operation of Felder — subfascial bandaging of communicant veins from back access (1955)

d) operation of Kokket - overfascial bandaging of communicant veins in the area of internal ankle.

3. Operations directed on diminishment of arterial influx to extremity - narrowing of main arteries, their resection (Fig. 97).

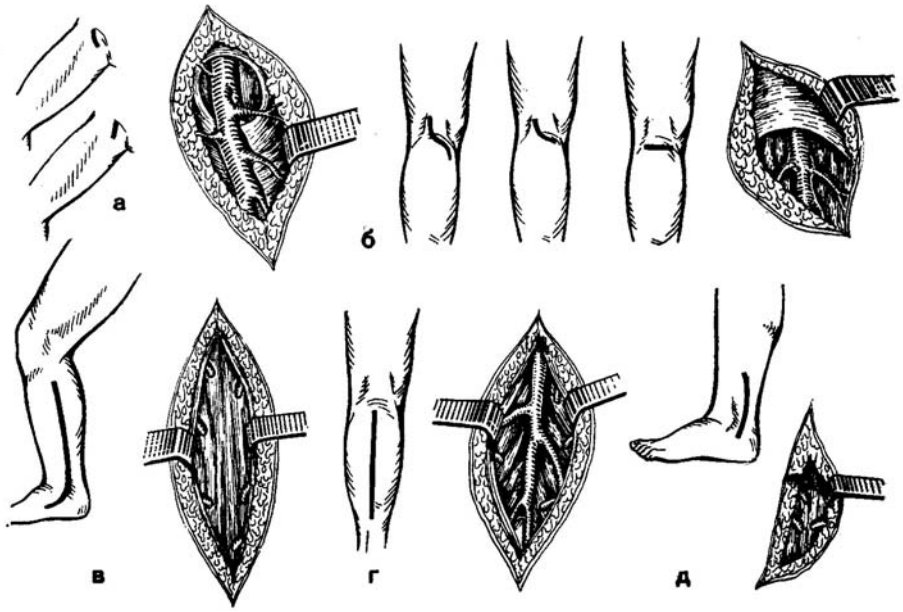


Fig. 97. Operations, which prevent outflow of blood to superficial veins в - Lynton, г - Felder, д – Kokket

Rules of conduct of postoperative period, measures on a prophylaxis, diagnostic and treatment **of possible postoperative complications** at this disease.

Stability of results of operative treatment in a great deal depends from the periodic conducting of courses of conservative therapy, health resort treatment, organizations of rational work and rest hours, including employment.

Except for the medicinal therapy PTPS, the extraordinarily important value has application of elastic bandages and organization of the rational regime of the physical loadings. The control of degree of edema of extremity is a reference point for the correct choice of the regime.

Diminishment of surplus mass of body, normalization of activity of intestine, limitation of the physical exertions is basic medical recommendations at PTPS. A patient during day rest mast occupy the raised position of feet, it is better – lying position. Salutary influence on circulation of blood in lower extremities renders swimming. Overheating on the sun, hot general and foot-bathes are contra-indicated.

The prophylaxis of PTPS consists of timely and effective treatment of acute vein thrombosis, in long application of compression therapy and supported anticoagulant therapy (indirect anticoagulants) in the period of rehabilitation.

At PTPS possible appearance of **such urgent states**, as thromboemboly of pulmonary artery, bleeding from varicose veins, for which the urgent medical help are needed.

At PTPS it is necessary to be able to execute doctors, diagnostic and medical manipulations: functional tests on determination of ability to passage of blood through the deep veins ("March" and Pratt-I), elastic bandaging of extremity, imposition of the zinc-gelatinous bandage (bandage of Unn).

The technique of conducting and estimation of tests of "March" Delbe-Pertes and Pratt-I is represented on p.12.

The elastic bandaging of extremity mast be performed daily after nightly rest and diminishment of edema on the extremity raised up. Begin with circular rounds on a foot. Then 2–3 eightvivid rounds on an ankle joint, farther passing bandage on a shin and thigh up to the inguinal ligament (usually 2 elastic bandages are needed). A bandage is taken off for the night.

The PTPS may has such **complication as** acute thrombosis of deep veins in the damaged segment, which needs urgent hospitalization (it is desirable in the specialized vascular department) and intensive treatment

Principles of examination disability and prophylactic medical examination at PTPS.

Patients with PTPS with the expressed chronic insufficiency (with trophic ulcers) often either have the reduced ability to work or lose it quite, and that is why need registration of disability of the III or II groups and to be on the clinical supervision at a surgeon with the systematic conducting of courses of the planned conservative therapy (it is desirable in the clinic) and if preset indication - surgical treatment.

Literature: 1) Andrew D. Blann, Gregory Y.H.Lip, A.Graham G.Turpie. Thrombosis in clinical practice.-Published by Taylor & Francis, 2005, 326 p. 2) Ghaye B., Willems V., Nchiimi A., Koukam L. and al. Relationship between the extent of deep venous thrombosis and the extent of acute pulmonary embolism as assessed by CT angiography.- Br J Radiol., 2009, Mar; 82 (975): 198-203. Epub 2009 Feb 2. 3) James E. Dalen. Venous Thromboembolism.- Informa Health Care, 2003, 365 p. 4) P. Colm Malone, Paul S. Agutter. The Aetiology of deep venous Thrombosis: a critical, historical and epistemological aurvey.- Spring, 2008, 265 p. 5) Robert W. Hobson, Samuel E. Wilson, Franc J. Veith. Vascular surgery: principles and practice.- Informa Health Care, 2003, 1292 p. 6) Sanjay Rajagopalan, Debabrata Mukherjee, Emile R. Mohler. Manual of Vascular Diseases.- Lippicott Williams & Wilkins, 2004, 437 p. 7) R.Wayne Alexander, Robert C. Schlant, John Willis Hurst at al. Hurst's the heart, arteries and veins.- McGraw-Hill, Health Professions Division, 1998, 2697 p.

VI.5. Lymphedema of extremities

Lymphedema in a translation from Latin means the edema of tissues because of stagnation of lymph, and is a chronic disease, developed because of insufficiency of the lymphatic system, disturbance of lymph transporting, as a result disturbance of absorption of proteins and metabolites produced in skin, hypoderm, and origin of lymphatic edema.

According to the literature data lymphedema of extremities (LE) or elephantiasis meets in 0,25%of patients admitted to the hospital. Elephantiasis in 3 times more

frequent develops in women. This fact is related to pregnancy, hormonal changes, increased insolation etc.

For understanding of questions of diagnostics and treatment of patients with lymphedema of extremities it is necessary to know the anatomo-physiological features of the lymphatic system of extremities, aetiology and pathogenesis of disease.

There are superficial and deep lymphatic vessels. The first collect a lymph from a skin and hypoderm, second - from underlying tissues. Superficial lymphatic vessels go alongside subcutaneous veins, the deep vessels are located alongside the proper deep blood vessels. In lower extremity they fall in popliteal and deep inguinal lymphatic nodes which take lymph from superficial lymphatic nodes.

The wall of lymphatic vessels contains muscular elements which are instrumental in the proximal direction of lymph flow. Valves which especially much are present in the vessels of distal departments hinder its reverse flow.

Development of lymphedema takes place because of constitutional (hypoplasia, hyperplasia, insufficiency of valves) or acquired character (inflammation, most often the recurrent erysipelas, trauma, operation, parasitic infection and other) of disturbance of lymph outflow on the different levels of the lymphatic system, that results in the loss of normal resorption and transport ability of lymphatic vessels. They are not able to take an protein rich fluid which is left in interstitial tissue, that results in an accumulation in it of pathological proteins and others, that in same time results to increase of fibroblasts and to development of sclerosis.

Thus, stable insufficiency of the lymphatic system results in development of connecting tissue which compresses lymphatic capillaries and vessels, increasing the phenomena of hypoxia, lymphostasis, lymphectasi and cellular infiltration are growing. All of it is instrumental in stimulation of development of connecting tissue. Acutely expressed fibrosis of skin, hypoderm and fascia develops as a result. Disturbances of lymphatic flow produce the condition for development of inflammatory processes, mainly erysipelas that is also instrumental in further progress of disease.

The following classification of lymphedema of extremities is used in clinic:

Forms: primary (congenital and idiopathic), secondary (of inflammatory and non-inflammatory origin).

Stages: lymphedema (Fig. 98), fibredema (Fig. 99).

***On questioning of patient*:**

The complaints of patients with lymphedema extremity has local character. General condition, as a rule, is not disturbed. Patients complain on thickening and deformation of extremity, feeling of edema and weight in it, sometimes complaints on “shooting” pain and indisposition are present.

Clinical physical inspection.

In a clinical flow of the disease the stages of lymphedema and fibredema are distinguished.

«***Locus morbi***». In the stage of lymphedema, which is characterized mainly by functional disturbances of lymph outflow, clinical symptoms consist of the small edema of foot and lower third of shins which appear at the end of day, after the long staying on feet. An edema has soft consistency, skin temperature is normal. Skin is not united with hypoderm and fascia, mobility of it is preserved, that allows to make

skin fold. The edema of extremity disappears after giving the leg elevated position, after which the circumference of the affected extremity corresponds to healthy one or insignificantly increased.

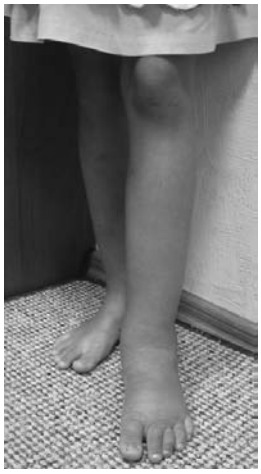


Fig. 98. Lymphedema

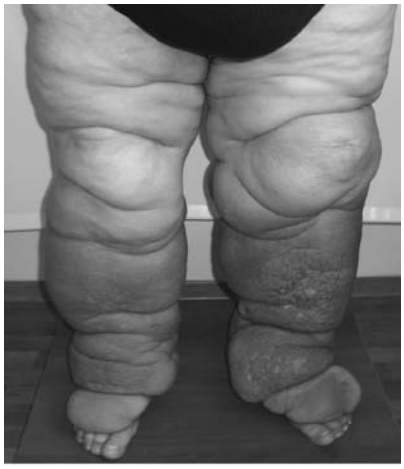


Fig. 99. Fibredema

Stable organic changes develop in the stage of fibredema. The edema acquires permanent character here. It does not disappear fully at protracted horizontal position of patient, asymmetry of extremity acquires stable character and by the time increased. An edema acquires dense, woody character, a fovea at pressing by finger does not develop, a skin does not undertake in fold, mobility of skin in relation to underlying tissues is absent. In these cases irreversible fibrotic changes in skin, hypoderm and fascias take place.

Basic clinical symptom is stable edema of extremity

Standard plan of additional examination (laboratory and instrumental) for patients with lymphedema of lower extremities includes:

Clinical blood test, urine, biochemistry of blood, coagulogram; ECG, X-ray of thoracic and abdominal cavity.

The final diagnosis of lymphedema is made on the basis of lymphography.

This method consists of that for 10–15 minutes after preliminary local anaesthesia in area of the first interdigital interval of foot intracutaneously 1–2 ml of lymphotropic dye (indigocarmin and other) is injected. Then at the level of middle third of back of foot between I and II metatarsus bones transverse or oblique incision of skin of 1,5–2 cm is done. The coloured lymphatic vessels are found in hypoderm, in one of which by needle or thin catheter 5–10 ml of water-soluble contrast (thryombrast and other) is injected , and than X-ray is performed. On lymphograms the ways of lymph outflow, presence and level of obstacle at the secondary forms of lymphostasis usually are well visible.

Differential diagnostics of lymphedema of extremities must be conducted not only with the diseases of extremities, but with the diseases of other organs. For the best mastering of differential diagnostics the followings tables are recommended:

Differential diagnostics of edema at lymphedema of extremities with edema at the diseases of heart ("cardiac" edema) and kidneys ("nephrotic" edema)

Symptoms	Lymphedema (LE)	"Cardiac" edema	"Nefroticheskiy" edema
Location of lesion	Primary – more often two-sided, secondary – more often one-sided	Always two-sided	Always two-sided
Location of edema	Rdema of back of foot + shin(thigh)	Shin, area of talocrural joint	Shin, area of talocrural joint
Character of edema	Soft in the beginning, dense in the late stages	Soft, dense becomes at the protracted flow of insufficiency of blood circulation	Soft
A tint of skin in the area of edema	Pale	Rose	Pale
Skin temperature	Normal	Decreased	Increased
Day's|day-night| dynamics of edema	In the early stages disappears at the morning, in the late stages no dynamics	No dynamics	No dynamics
Additional criteria	At primary LE an edema arises up to 35 years, at the secondary after 40 years	Expressed signs of basic disease	Expressed signs of basic disease

Differential diagnostics of lymphedema of lower extremities with postphlebitic syndrome and varicose illness

Signs	Lymphedema (LE)	PTFS	Varicose illness
Etiology	Erysipelas, congenital pathology of the lymphatic system	Acute thrombosis of deep veins of extremity	Congenital endocrine, mechanic causes and etc
Anamnesis	Making progress flow	Deep venous thrombosis after delivery, operations, traumas and others	Slow development, in women, as a rule, after delivery
Pain syndrome	Absent	Permanent, of different intensity inside of extremity, decrease at posture drainage, increases at walking and at the evening	Permanent, moderate pain in a shin and foot, increasing at the evening
Edema	Even, dense, with a clear border level of the underlying disease	Permanent, of different degree in hypoderm, subaponeurotic tissues	Quickly passing, involves subcutaneous fat

Signs	Lymphedema (LE)	PTFS	Varicose illness
Pigmentation	Absent	Present	Possible
Induration	Present	Present	Possible
Dermatitis	Absent	Present	Possible
Cellulitis	Absent	Present	Possible
Ulcers	Absent	Present	Possible
Varicose veins	Absent	Secondary	Primary
Test of Troyanov-Trendelenburg	Impossible due to absence of varicose veins	Positive	Positive
Delbe–Pertes	Impossible due to absence of varicose veins	Negative (deep veins are occluded)	Positive (deep veins are passable)
Pratta I	Impossible due to absence of varicose veins	Positive	Positive
Symmetry of lesions	Characteristic	Rarely	Often
Phlebography	Without pathology	Lesion of valves of veins, varicose dilation of superficial and deep veins, reflux of contrast	Varicose expansion of superficial veins, rarely reflux on communicants, deep veins is not changed
Lymphography	Permanent lesion of the lymphatic system, most expressed in superficial vessels	Expansion and disruptions, lesions of valves, lymphothrombosis mainly of the deep system	Changes appear lately, not always
Lymphatic nodes	Can change, up to formation of conglomerates	Single, dense, of different size	Not changed, firm at the complicated form

Formulation of clinical diagnosis:

a) basic – lymphedema of lower extremities, stage of lymphedema /fibredema;

b) complications – (if present);

c) concomitant pathology - (if present).

Treatment of lymphedema of extremities.

Choice of medical tactic. Conservative treatment of lymphedema ofextremities is effective only in the stage, when stable organic changes of soft tissues of the affected extremities areabsent, that means the stage of lymphedema.

At development of stable, irreversible changes, when hypoderm is substituted by dense connective tissue, and fascia becomes sclerotic (stage of fibredema) the unique rational method of treatment is operative intervention.

The complex of conservative measures includes: tight bandaging of the affected extremities by elastic bandages; prescription of preparations, which improve the nutrition of tissues (vitamins), peripheric circulation of blood (Nospanum, Galidor,

other), microcirculation (reopolyglucinum, trental, detralex, troxevasin, escusan); antihistamine medicines (suprastinum, dimedrolum, other); non steroid anti-inflammatory preparations (butadion, indomethacinum, diclofenac, movalis, other); diuretic (furosemid, other); preparations which hinder formation of scar tissue (corticosteroids, lidase, other).

Also it is appointed the draining massage of the affected extremity, medical physical exercises, physical therapy and balneological treatment.

For the prophylaxis of recurrences of erysipelas and progress of the disease the courses of antibacterial therapy and ultraviolet irradiation of extremity are prescribed.

However conservative treatment has short effect and does not cure the patient.

A lot of methods of surgical treatment of lymphedema of extremities is offered, that confirm the unsatisfactory results of treatment.

At lymphedema, which is accompanied with development and induration of connective tissue in hypoderm, the complete removal of the changed hypoderm together with deep fascia which covers muscles is used. During this operation skin is perforated by small incisions and replant on «naked» muscles. Excess of skin is excised. Skin flaps are sutured by catgut to the muscles and together. Extremity is tightly bandaged and immobilized by plaster splint.

One of the most rational and pathpgenetically grounded methods of operative treatment are operations which are directed on renewal of outflow of lymph. Direct lymphovein anastomosises are performed between superficial lymphatic vessels and branches of subcutaneous veins in area of femoral triangle or popliteal fossa. 1-1,5 hours before the operation lymphotropic dye is injected for colouring of lymphatic vessels. Maximally possible number of lymphatic vessels is dissected and the nearest branches of subcutaneous veins. Lymphatic vessels are divided, their central ends coagulated, and distal anastomosed with veins on type «end to side» or «end to end». 6—10 lymphovein anastomosises are performed. If an operation is performed in the initial stages of disease, it allows fully to normalize lymph outflow from the affected extremity, in the late stages allows considerably to decrease the edema of skin and hypoderm.

Chapter VII. Modern mini-invasive technologies in surgery of great vessels and heart

The last achievements of scientific and technical progress and development of new technologies and methods allowed to carry out minimally invasive procedures with great success to the wide circle of patients. Presently one of new directions is combination of surgical and endovascular revascularization of myocardium. This approach became especially actual due to the development of minimally invasive surgery.

Videoendosurgical manipulations, executable on the closed thorax, correspond to principles of minimally invasive surgery to the utmost. The real thoracoscopic cardiac surgery was actually pointless in the past in connection with technical imperfection of existing endoscopic instruments. With the purpose of conducting of endoscopic reconstructive microsurgery the new intellectual generation of instruments was required with the improved dirigibility and with higher exactness. Nowadays in the arsenal of cardio surgeons there are all necessary instruments and methods for implementation of endoscopic reconstructive microsurgery.

Many cardio surgeons speak out about necessity of bringing in attention of wide circle of doctors to new possibilities of coronary surgery which became a powerful social factor in life of any society. It disposes enormous possibilities, conduces to warning of heart attack of myocardium and its complications. Its prospects are obvious in the future, and the role of the specialized centers which will be engaged in the decision of these questions will be unchangingly growing on condition of clear organization, financing and timely lineion of patients to surgical treatment.

Endovascular methods of treatment are a separate enormous section of problem of treatment of atherosclerotic lesion of vessels. In 1977 Gryuntcig offered a balloon catheter which by means of puncture of general femoral artery is entered into coronary channel and at blowing extends the lumen of the narrowed areas of coronary arteries. This method quickly got wide distribution at treatment of chronic IHD, unstable stenocardia, severe disorder of coronary circulation of blood. The methods of endovascular treatment and surgery of atherosclerosis do not compete, but complement each other. The number of angioplasties with the use of stent in the economic developed countries grows steadily. Each of these methods has the indications and contra-indications.

Stenting of main vessels

During the last 30 years, on the basis of diagnostic angiography there was one of the stormiest developing industries of modern minimal invasion medicine - intervention radiology. Percutaneous transluminal angioplasty, offered by Ch.T. Dotter, became the beginning of new era in treatment of atherosclerotic lesion of peripheral arteries and background of further development of percutaneous interventions in different areas of medicine. Due to talent and ingenuity of scientists, standing at the sources of intervention radiology, it grew from the applied diagnostic speciality into independent lineion of medicine, offering the unique minimal invasion methods of treatment. The basic indication to establishment of stents are stenosises of main arteries over 60%. Presently stents are set practically in all main arteries which can be affected by atherosclerosis: carotids, coronary arteries, kidney arteries, arteries

of lower extremities. In addition, stents for arteries with an aneurismal lesion are invented. Before interference the angiography of the affected area is necessarily executed which allows to estimate : presence of stenosis; degree of stenosis; character of plaque; diameter of vessel; extent of lesion. An angiography can be executed right before the intervention interference.

Coronaroangioplasty is a balloon angioplasty and stenting of coronary arteries. At a balloon angioplasty in an angiographic department in the affected coronary artery a catheter is entered with a balloon on the end, under roentgenologic controlit is set in the place of narrowing of coronary artery and is blown up for 1-2 minutes. Thus, an atherosclerotic plaque is crushed, and the lumen of vessel is increased. At stenting after that in the lumen of vessel a metallic frame cylinder opens up with the special medical coverage - stent which supports the form of vessel. Medical coverage on the walls of stent serves as the prophylaxis of thrombosis inside of it. After implanting of the stent conducting of control coronarography within 4-6 months is needed. This method of treatment is acceptable at the lesion of one or two arteries, at small atherosclerotic plaques. In the case of plural and severe lesions of coronary arteries it is necessary to resort to operative treatment. Advantages of Percutaneous transluminal angioplasty: 1) higher efficiency in comparison to conservative treatment; 2) does not need general anesthesia and thoracotomy (for patients, at which aorta coronary bypass is intolerable|); 3) does not require the prolonged rehabilitation period. Drawbacks: risk of development of complications dangerous for life during conducting of balloon angioplasty and stenting makes about 2%. Coronary stenting is a method of intravascular prosthetics of coronary arteries at different pathological changes in the structures of their wall. For the reconstruction of coronary arteries stents are used. Stent is metallic frame which is the little metallic tube made of wire. Stent is entered into an artery after its expansion and set in a place of the lesion of artery with the purpose of prevention of restenosis. Stent supports the walls of artery. More than 60 different constructions of coronary stents are presently offered. Depending on a design stents are divided into:

– wire (made of one wire);
– tubular (made of cylindrical tube);
– rings (made of separate links);
– reticular (as the wicker net).

Depending on the technique of the implantation there are distinguished self-straightening stents and those which are straightened with the help of a balloon-catheter. This method is carried out|, as well as angiography, by puncture of femoral artery. The operation is conducted under the local anaesthesia. A balloon with a stent is set through the puncture in the femoral artery on the special explorer catheter in the place of narrowing of coronary artery . At blowing of balloon the stent falls out and restores the lumen of the artery. The heart gets the necessary volume of blood along the restored artery which in its turn brings to decrease or disappearance of pains behind the breastbone at the physical loadings.

Advantages of stenting: lasts a little longer than Percutaneous transluminal angioplasty; use of the stent diminishes the necessity of the repeated operations; duration of hospitalization is shorter - 3-4 days; stenting can be applied to the patients, who can not undergo aorta coronary bypass, but Percutaneous transluminal angio-

plasty is not indicated; in comparison to Percutaneous transluminal angioplasty it diminishes the necessity of implementation of urgent aorta coronary bypass; finally, less traumatic, than aorta coronary bypass. Drawbacks of the stenting: thrombosis of the stent; restenosis in stent. Three types of the stenting are defined:

1) bailout (saving, urgent) stenting - conducted at the acute occlusion of the coronary artery by fragments of atherosclerotic plaque after Percutaneous transluminal angioplasty instead of urgent aorta coronary bypass;

2) planned (elective, primary) stenting - stenting is conducted initially regardless of results of Percutaneous transluminal angioplasty (at different forms of CHD);

3) stenting on indications (provisional) – is used according to the results rezul'tatam| of angiography of angiography after Percutaneous transluminal angioplasty, when its results are not optimum, with the purpose of prevention of the acute occlusion of coronary artery or development of its restenosis.

Endoscopic aortocoronary shunting

In 1995 a new method — minimally invasive coronary surgery entered in clinical practice of cardiac surgery. It is a new section of coronary surgery. It is based on implementation of operations on a working heart without application of artificial circulation cardiopulmonary bypass of blood and use of minimum access.

Presently in clinical practice several methods of minimally invasive surgery are used at CHD:

Without artificial circulation of blood with the use of "stabilizator" for shunting. (In order to stabilize the certain area of heart and impose a shunt on this area, the special stabilizing systems are used).

Application of minimum surgical cuts, including endoscopic operations (endovideoassisting , minimally invasive revascularization myocardium).

With the use of robotics (in this case a robot, guided by a surgeon, carries out forming of anastomosis between the coronary artery and internal thoracic artery. This method is in the stage of mastering).

Transmyocardiac laser revascularization of the myocardium — implies creation of numerous artificial transmyocardiac_channels from epicardium to endocardium of the left ventricle by the high-powerful CO2 laser. The founder of this method is a former leading employee of laboratory of pathomorphology and experimental medicine of Kharkov scientific research institute of general and urgent surgery Kononov A.J.. In 1979 - 1982 experiments on revascularization of ischemic myocardium were conducted by him first in the world by means of the distant minimally invasive method by a laser catheter and cannula for the improvement of circulation of blood of myocardium. Presently this method is used mainly for patients, when medication is noneffective and there is no possibility to carry out the line revascularization of the myocardium. In other words, transmyocardiac laser revascularization of the myocardium is used as a «step of despair» at the unsatisfactory prognosis of disease and noneffectiveness of other methods of treatment of CHD.

The first large clinical experience of minimally invasive operations of line revascularization of the myocardium is presented by F.Benetti and co-authors, and also by V.Subramanian in Rome in November, 1994 at the international conference «Arterial conduct-books for revascularization of the myocardium».

Most capacious determination of the term minimally invasive revascularization of the myocardium was given by A.Calafiore (1996): - revascularization of the myocardium through thoracotomy no more than 10 cm, without cardiopulmonary bypass, with the use of only arterial conduct-books "In situ". Since 1996 the coronary surgery was literally overflowed by the wave of minimally invasive interferences (A. Calafiore and soavt., 1996., F.Benneti and co-authors 1996., J.Grandjean and co-authors, 1996.).

The accumulated experience of implementation of operations on a working heart created pre-conditions for development of minimally invasive revascularization of the myocardium, including usage of endoscopic methods. The pioneers of introduction of endoscopic technique in coronary surgery are F. Benetti (1995) and P. Nataff (1996), having defined basic directions of its application.

As founders of the method of minimally invasive coronary surgery F.Benetti and P.Nataff mark, operations with endoscopic support have very short history so far, in spite of the absolute perspective.

Indications for this type of surgical treatment meanwhile are quite limited: in the leading clinics of the world this method is used in 10-20 % of all operations at IBS. Indications to the operation are based on information of clinical examination and aortocoronography.

Minimally invasive revascularization of the myocardium is conducted to the patients with stenosises of coronary arteries more than 50% in diameter, in case when conservative methods, ballooning or stenting can not be conducted, or they do not help already. This operation is conducted at the considerable narrowing of one of coronary arteries.

According to the data of some German specialists in case if the narrowing of the right coronary artery or its branches occurred to the patient, conducting of minimally invasive operation is possible, without wide dissection of thorax and without application of artificial blood circulation apparatus. In case if other arteries of heart are affected and their narrowing is more than 70-75%, and also in cases of associated pathology of heart (presence of postinfarction heart aneurysm of the left ventricle, congenital or acquired heart-disease requiring surgical correction), a classic operation is indicated - aortocoronary shunting, with connecting of artificial blood circulation apparatus.

By the result of the 2th world congress on a minimally invasive revascularization of the myocardium (Spain, Barcelona, 1998) the indications to minimally invasive revascularization of the myocardium were determined:

1. Repeated operations.
2. Single-, twovascular lesion of coronary arteries in the group of patients of the hyper-risk for operations with the use of cardiopulmonary bypass.
3. Restenosis of coronary arteries after translumen balloon angioplasty.

Technical aspects of minimally invasive revascularization of the myocardium

Nowadays more than 10 different methods of minimally invasive endoscopic coronary surgery are developed, with the use of one or another miniaccesses, that allows to shunt one, two and even three coronary arteries, and then from a little access to sew down the distal end of this artery linely to the affected coronary vessel, restoring the same circulation of blood in the heart.

The most widespread miniaccesses which are used at shunting of coronary arteries are: right- and left-side minithoracotomy with excision of costal cartilages or without their excision (Bennetti F. and co-authors, 1995, V.Subramanian and co-authors,1996); subxiphoid access (Grandjean and co-authors,1996.); parasternal left-side minithoracotomy with excision of costal cartilages (P.Nataff and co-authors,1996., Vlasov P. and co-authors, 1998.). Several accesses, allowing to make revascularization of several CA, were also developed: upper and lower ministernotomic access for plural mammaro - coronary shunting of front interventricular branch, diagonal branch, right coronary artery; (Vlasov g.P. and co-authors 1998).

At miniaccess laboured valuable discharge of the internal thoracic artery on a large extent and ligation of all its branches. Special endoscopic instruments, entered into the thoracic cavity, allow to manage with this task. The use of endoscopic technique allows to decrease access to 4-5 sm.

One of the greatest problems of minimally invasive coronary surgery is a shortage of arterial shunts for fully arterial revascularization of the myocardium, and also question of method of their mobilization. In a great deal this problem is decided by the method of endoscopic selection of arterial transplants.

Last years in leading cardiac surgery centers as an anastomosis it is accepted to use the internal thoracic artery, then sewing of bypass into the aorta is not made, but distal part of the internal thoracic artery is sewn underneath , under the place of stenosis, into the coronary artery. If such variant is impossible, because of the anomaly of development of internal thoracic artery, the arteries of antebrachium (radial artery or ulnar artery) are used. These arteries are the most suitable material for imposition of anastomoses. Usually these anastomoses work tens of years unlike the venous. Considering that the artery is more lasting, and adjusted to function exactly in the conditions of high blood pressure which is maximum in the aorta. It should be noted that many cardio surgeons do not practically apply anastomoses from the veins of lower extremities .

Mobilization of internal thoracic artery in a skeletonized kind is carried out much simpler, provides winning in length of transplant up to 25%, that, in its turn, allows to solve a problem of shortage of length and to avoid its pull.

Nowadays, methods of endoscopic mobilization of both internal breast arteries and right gastroepiploic artery are already developed and inculcated (Nataff P. and Vlasov G. P.).

During the endoscopic selection of internal thoracic artery separate endobronchial intubation by means of double-lumen tube «Carlens» and conduct one-lung (right) ventilation. A patient is laid on the right side with inclination of 30 degrees and with his left arm turned off and up. Arterial shunts are selected endoscopically through three thoracoports, conducted into the left pleural cavity. Thus the chamber is on the right of the endoscopic instruments in the 3^{d} intercostal space along the front axillary line. The second port is entered in 4-5 intercostal space along the middle axillary line and the third port - in 6-7 intercostal space along the front axillary line. This kind of location of ports, according to Vlasov G.P. and co-authors, considerably improves possibility of visualization and gives more possibility for implementation of surgical manipulations.

The location of ports in relation to intercostal intervals is determined in every case individually and depends on the constitutional features of patients.

Selection of the left internal thoracic artery is conducted along the whole length with clipping and crossing of lateral branches. Mobilization of internal thoracic artery is always begun in the middle part from the longitudinal dissection of parietal pleura in the projection of artery in proximal and distal directions. Further the dissection of internal transverse thoracic muscle in the projection of artery in distal direction is made. Taking into account the expressed susceptibility of internal thoracic artery to the spasm, after excision of its distal part in the lumen of artery the solution of papaverin is introduced, that allows to obtain an adequate antegrade blood stream. According to the data of separate authors (Vlasov G.P.,1998), in any case of endoscopic selection of VGA it was not fixed any meaningful damage of artery or development of the profuse bleeding, in case of tearing away of little lateral branches, after the stoppage of the active bleeding by a temporal compression by gauze swab the damaged branch of clipped. Average time of mobilization of left internal thoracic artery makes 40 ±10 min.

Application of endoscopic method in mobilization of internal thoracic artery, allows in the case of necessity of implementations of double-vascular revascularization of the myocardium together with left internal thoracic artery, to make also a selection of the right internal thoracic artery. Thus both conduct-books are selected through the left-side access without the change of location of ports. It is necessary to mark that mobilization of right internal thoracic artery must be conducted in the first place, otherwise, mobilized by the first left internal thoracic artery, considerably hampers further manipulations. Technique of selection of right internal thoracic artery does not differ from such during mobilization of left internal thoracic artery.

After careful endoscopic control of hemostasis access to the coronary artery is made- left-side parasternal access of 4 sm length with the resection of areas of cartilages of the 4th or 5th ribs or left-side front minithoracotomy with the length of 4-5 sm. The pericardium is dissected longitudinally and fixed on holders. On the area of coronary artery in the place of the supposed anastomosis tourniquets are put, and after conducting of test with cross-clamping of coronary artery the anastomosis is put by a continuous blanket suture by the filament of prolen 8/0.

The accumulated experience of endoscopic selection of internal thoracic artery allows to conclude that this method is fully safe and applicable in overwhelming majority of cases. However a number of factors hampers mobilization of conduct-book considerably, making it not only noneffective but also, sometimes, risky. To such hampering factors they refer narrow intercostal intervals, considerable fatty deposits in anterior mediastinum and expressed comissural process in the pleural cavity.

Mobilization of right gastroepiploic artery can be also conducted with the use of endoscopic support, that has advantage over the selection of artery through minilaparotomy.

Endoscopic mobilization of right gastroepiploic artery is executed through three laparoports. Thus through the Veresh's needle, conducted through the umbilical ring, into the abdominal cavity the air is blasted, whereupon, always through the umbilical ring the first port for a video camera is conducted. The second and third ports for endoscopic instruments are set in the left mesogastrium. At a necessity of

providing adequate traction of the stomach in epigastrium additional port can be set for the Bebcock's forceps.

Mobilization of right gastroepiploic artery is conducted from a bulb of the duodenum to the splenic corner of the stomach. Pair short branches are clipped and crossed. It is necessary to mark that front and back branches should be clipped separately, that allows to avoid deformation of conduct-book. For the exception of «excessive» mobility of right gastroepiploic artery during the mobilization it is desirable to leave 1–2 intersections from short branches.

After excision and clipping of distal part of right gastroepiploic artery through 1–1.5 sm opening, made in the central tendon of diaphragm, an artery is conducted in the cavity of pericardium with control of its possible torsion. Average time of mobilization of right gastroepiploic artery is approximately 65 min. For prevention of spasming of artery the solution of papaverin is introduced into its lumen.

Due to wide introduction of method of endoscopic selection of arterial conduct-books into clinical practice, possibility to extend indications to minimally invasive revascularization of the myocardium appeared. To the patients with lesion of front interventricular branch and diagonal branch several charts of minimally invasive revascularization of the myocardium can be used: sequential shunting of two coronary vessels with the help of the left internal thoracic artery and use of "U- like" composite shunt from left internal thoracic artery and radial artery, as a free transplant, and also separate shunting of front interventricular branch and right coronary artery with the help of internal thoracic artery and right gastroepiploic artery.

Shunting of 3th CA (front interventricular branch, diagonal branch and right coronary artery) one left internal thoracic artery is also possible, with the use of both antegrade and retrograde blood stream along it. Thus the sequential shunting of front interventricular branch and diagonal branch is conducted, and by distal part of left internal thoracic artery, above the inserted radial artery, revascularization of right coronary artery is conducted.

Advantages of minimally invasive revascularization of the myocardium:

Mini-access (5-6 cm) does not disturb the stability of breastbone, allows not only to injure the thorax minimally but also recover from anesthesia quicker - a man wakes up already on the operating table. Except for it the patient goes back to the usual physical loadings already in the early postoperative period.

The operation is conducted on a working heart.

Sensitivity and little traumatic characteristics of endoscopic technique allows to select arterial conduct-books along the whole length with crossing of all of lateral branches of artery without technical complications.

Less trauma of blood, because at aorta coronary bypass with IK the artificial circulation of blood apparatus is used, that is most negative.

Decline of risk of development of harmful effects of the AC.

Minimally invasive revascularization of the myocardium extends indications to the operation for patients with low ejection fraction, acute myocardial infarction, by heavy concomitant diseases and for elderly patients .

Special value minimally invasive revascularization of the myocardium acquires in the case of the repeated operations. Possibility to yield up longitudinal resternoto-

myi, cannulization of aorta and vena cava, and also implementation of cardiolis, considerably allows to reduce the risk of the repeated surgical interference.

Cosmetic effect (no traces of the undergone interference within 6 months).

At a minimally invasive revascularization of the myocardium, development of which is impossible without endoscopic support, more and more supporters appear. Variety of accesses, minimum traumatic characteristics, the refusal of the cardiac arrest and application of artificial circulation of blood allow, at first, to reduce the price of the operation by dozens of times- one artificial circulation of blood apparatus costs 150-170 thousand usd., not speaking about disposable oxygenators, filters, cardioplegic solution, heat exchangers, great number of expensive drugs, and also protracted postoperative treatment and care. Secondly, a new method gives a chance to return to normal life people for which a traditional operation was intolerable: the patients with the acute myocardial infarction, and also patients having heavy concomitant diseases are insular diabetes, chronic renal insufficiency, stomach ulcer, diseases of lungs et cetera.

In opinion of many leading cardio surgeons the developed original method of endoscopic coronary surgery - aortocoronary and mammacoronary shunting — will allow to give up at all even minimum cuts, replacing them with several small punctures.

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Chapter VIII. Allografts and laser surgery in angiology

For becoming of vascular surgery as important constituent of general surgery had been necessary same significant such as: invention of vascular stitch in 1902 A. Carrel; wide introduction in practice of angiography investigation; development of methods of substitution of the segments of a vessels affected by pathological processes on auto- and heterotranspplantants, include and invention of synthetic materials for production artificial vessels and others.

Allotrasplantants (modern terminology - *explantants*) take strong place in vascular surgery, actually without them impossible reconstructive operations, and due to greater distribution in the world of chronic and acute stenotic-occlusion affectation of main vessels (according information of UNISEF, if earlier by those diseases suffered near 12% of population of Earth, now near 18%(!)) actuality of this problem becomes clear.

History of substitution of vessels begins from 1882 year, when Th. Gluck (1883) for connection of ends of artery first applied metallic and bones tubes. Allexis Carrel in 1910 offered synthetic vessels for it replacement. However a lot of years all attempts to replace arteries by the artificial vessels of various constructions and from different materials (tubes from ivory, feathers of birds, glass covered from within by a paraffin, aluminium, silver, polyethylene and others) were unsuccessful and practically remained on the stage of experimental researches.

The basic type of replacement of vessels in our time is allotransplantants from synthetic tissues. The first clinical application of textile explantants belongs to 1952 and related to appearance of the work A.B. Voorhees and coauthors which offered implant from synthetic material of "Winion-M".

At the end of 50th angiosurgers began to apply polyamides - nylon and kapron, and then - dacron, lavsan and tephlon.

In 1955 W.A. Edwerds and J.S. Tapp was offered elastic goffered grafts.

In 1960 M. De Becky and coauthors had most experience of the use of dacron explantants, and in 70th De Becky offered «velour» explantants.

Tubular and bifurcation grafts began to be produced (S.A. Wesolowski and D.A. Cooley firm "Meadox Medicals")

In 80th in the USA a new synthetic material of polytetrafluorethylen (PTFE) began to be used for making of vascular grafts «Gore-Tex».

To this time the *«ideal»* artificial substitute of vessels is not created. The researches precede their work directed on improvement, foremost, on diminishment of such failings, as thrombosis of synthetic grafts, appearance of periexplantants haematomas, infected of grafts.

Requirements to artificial vascular grafts-explantants

1) Vascular implantants must be done from chemically neutral synthetic, which is arreactive or is the cause the minimum local reaction of tissues of organism without some harmful influence on him, that their biological indifference.

2) Vascular grafts must be intrusion for living tissues, that in a great deal determines theirs capacity for «implantation», to creation of «new intimae».

3) Permeability of wall of vascular prothetic mast to be such, that at implantation prothetic blood loss through his walls did not worsen the state of patient, there was no considerable periimplantant haematoma.

4) The wall of vascular graft must be rough, that is achieved in number of different ways (promoted villous formation, combination of synthetic fibers with different properties, velour surface of prosthetic and other.).

5) Vascular prosthetic must be mechanical characteristics which provide the most advantageous for a blood stream on graft.

To these mechanical properties it is possible to deliver the following:

a) a tube mast be without stitches and not have a stitch in place of branching of bifurcation prosthetic;

b) prothetic mast to be elastic and apt at a passive pulsation at once after implantation;

c) tube is to be apt at the design under act of current of blood;

d) a tube must not bend under an acute angle;

e) tube must not be twisted on an axis;

f) tube is to be strong and not change the properties independently from time of stay in an organism;

g) prothetic must have a thin wall and it is easily to be pricked by a atraumatic needle;

h) prothetic must be well compared with a vessel.

Except for the above mentioned mechanical properties, matter and some other:

– prothetic must easily and simple to be sterilized;
– it mast be simple in circulation;
– prothetic mast to be proof to the microbes.

Thus, to produced prothetic implantant the basic requirements are: they are to be indifferent (that, not to cause the reaction of surrounding tissues), strong, elastic, to own sufficient biological permeability, to provide the process «growing» in, «implantations» of prothetic at which is formed new intimae, and in that-exactly time not to result in the expressed bleeding from prothetic with formation of vast haematoma around prothetic implantant, that threatens of infection, or subsequent scar deformation of prothetic. Unfortunately, a thrombosis of the graft is frequent complication.

Are basic synthetic materials for vascular allotransplantants are: lavsan, fluorlon, teflon, dacron.

Exist a 3 basic type of synthetic textile vascular prothetic:

1) bounding,
2) weaving,
3) wattled.

Considerable elasticity and flexibility are positive qualities of bounding grafts, good of «establishment» them at a tissues of organism through high permeability their walls, but through the last such prothetics give the considerable bleeding and formation of considerable periexplanting haematomas.

Their durability and low permeability of their wall is basic advantage of weaving prothetic implantats, that provides small bleeding at their application; but their failing are rigidity and inflexibility.

Vascular prosthetic appliances of wattled construction occupy intermediate position between bounding and weaving are they give comparatively small blood loss during implantation, elastic, tensile in longitudinal and transversal directions, but they are used rarer as a rough.

Corrugating of vascular implant have big matters for prevention of flexibility and incompressibility under different corners without a tendency to be twisted.

The most considerable group of patients to which indicated **application of artificial vascular grafts** are patients with **obliterated atherosclerosis of** a different localization with segmental occlusion of main arterial vessels. It is the Lerishe syndrome that illness Takajasy.

Aneurism of different origin (above all things are atherosclerotic) of aorta and big arteries is an absolute indication to application of explantants.

Coarctation of aorta is direct indication for grafting, and also **traumatic damage** of big arteries and aorta.

Vascular explantants are used at implementation of reconstructive operations of the bypass shunting at the Lerishe syndrome, at the Takajasy disease (at damaged brachiocephal branches of arch of aorta), at coarctation and aneurism of aorta, at traumatic the damaged aorta and big arteries and others.

In different vascular pools it is expedient to apply special explantants, such more adapted exactly for this area.

As operations on an ascending aorta are conducted in the conditions of artificial circulation of blood, at complete heparinisation of patient, used vascular explantants are to be finely porous and corrugated, in order to avoid those bandings at forming of the proper to the arch of aorta.

At the reconstruction of descending, thoracoabdominal and abdominal aorta is mainly used explantants with finely porous. However possible it is to apply high porous explantants after their previous impregnation by a blood.

Finely porous explantants absolutely indicated in those cases, when it is necessary to avoid minimum bleeding even, for example at patients with the factors of risk – initial anemia, by kidney and hepatic insufficiency, at the exhausted patients and others like that.

At the reconstruction of femoral-popliteal area in the case of absence of adequate autoveinr is more preferable explantant from polytetrafluorethylen.

At the reconstruction of kidney arteries and visceral branches of aorta explantants is material of choice with PTFE.

For the reconstruction of brachiocephalic trunk it is possible to use any high-quality explantant. In case bifurcation – more recommended plastic material with corrugation for the prophylaxis of bend. For a reconstruction of corotis, subclavia and inguinalis arteries the especially important use of thromboresistens substitutes of vessels. In two last cases during conducting of explantants in subclavia-costal space it is necessary to apply reinforced grafts for avoidance of their squeezing at motion of upper extremities.

Introduction in clinical angyology inflateble catheters for embol- and thrombectomy (T. Fogarti, 1968 p.), author developments of Ch. Dotter, which gave beginning to development of transluminal angioplasty are basic factors of a appearance of new direction at the allotransplantology of vessels as distant endoprosthetic and intraoperative prosthetic and shunting of vessels by auto fixing endoprothetic.

In 70th in Kharkov Institute of general and exigent Surgery the created and developed base inventions, which behave to this type of vascular alloplasty, and also devices, for his realization (Kononov A.J.,2000).

The most frequent complications of alloprosthetic of vessels are:

1) thromboses of explantants;

2) acute bleeding during operation at imposition of anastomosis of graft with an aorta or artery and in the first days of postoperative period;

3) formation of aneurism in the area of anastomosis of prothetic with an artery (more frequent - false);

4) development of wound infection in the area of vascular graft.

For the prophylaxis of the above mentioned complications and lowering risk of their appearance development various technologies.

For the prophylaxis of thrombosis created explantants with antythrombotic properties which have antythrombotic coverage with heparin; electric conductive vascular grafts with silver framework which creates a negative electric charge on implantant, it diminishing possibility of thrombi formation.

The Czech scientists (M. Chvapil V. but Krajicek, 1966) created thromboresistens combined explantants, which consist of insoluble elastic synthetic framework with big porosity, inside of which inserted tubas from collagen, saturated with solution of heparin.

For the prophylaxis of bleeding is produced explantants, which covered for hermetic collagen and gelatinous overages (explantants «Gelvin », «Uni-Graft»,and others).

For the prophylaxis of infection were creation of explantants with antimicrobial properties (grafts from the antimicrobial synthetic tissues of letilan in combination with a lavsan – Lebedev L.V. and coauthors, 1963; implantants with the antibacterial preparation heomicine – Shalimov A.A. and coauthors, 1977).

From 1993 in Saint Petersburg serially produce explantants «Vitaflon» with a high biological sluggishness and thromboresistant.

In Moscow from 1996 serially produce new antimicrobial, thromboresistant with high porosity explantants «BASECS».

Laser surgery in angiology

Actuality of theme.

Development of medicine, the search of new ways of improvement of results of treatment of patients with a different pathology, include and surgical, introduction in medical practice of new methods of treatment. In a present tense there is intensive introduction of laser radiation in biological researches and in practical medicine in most world countries. Unique properties of laser ray opened the wide prospects of that application in different industries of medicine: to surgery, therapy, and diagnostics. Clinical researches show the efficiency of different types of laser radiation (ultraviolet, infra-red and other) for local application on pathological focuses and for action on all organism.

The target studies of theme:

To get theoretical knowledges and know answers for control questions on a theme and decide to the test of task in a format “Step 2” on a theme on the final module control.

Educational tasks for the independent extra class working of theme

III.1. Minimum base level of knowledges and abilities, necessary for mastering of theme.

1) Physical bases of work of laser
2) Regions for using of laser radiation

Concrete aims for the independent extra class working of theme

– Using the base level of knowledges, to study theoretical material on the theme of employment and know answers for control questions on a theme:
– Physical bases and types of laser radiation
– To know the types of photobiology processes
– To know the methods of delivery of laser radiation to the patient
– To know a indication and contra-indication to application of laser technologies in angyology.

Informative block for the independent extra class working of theme

A laser or quantum generator is the technical device, which produces light in a narrow spectral range as, high gerent monochromatic, polarized bunch of magnetoekectric waves. The light streams of low intensity are applied in laser therapy. No more than a 100 мVt/cm kv, that answers the radiation of the Sun on a terrene in a clear day. Therefore name such type of laser co-operation is low intensivety laser radiation (Low Level Laser Therapy). One of important descriptions of laser radiation is it spectral characteristic or wave-length. By photo biologic activity of possess light in the ultraviolet, visible and infra-red areas of spectrum. Photo biologic processes it is enough broad church and specific. The photo physics and photochemistry reactions which arise up in organism under act of light lie in basis of them. Are the photo physics reactions conditioned, mainly, by heating object and distribution of heat in biological tissues. Are the photochemistry reactions conditioned by excitation of electrons in atoms, taking in light objects. Action of low intensive lasers are the rapid calming down of inflammatory and stimulates reparative processes, improves microcirculation of tissues, normalizes the state of the immune system, rise the resistance of organism. It is lead to, that the lower intensive laser radiation has the expressed therapeutic action. Depending on the device of laser of it radiation can take place as impulsive or continually. To the first regards ruby laser, to the second is gas. The semiconductor lasers can work in both modes. The laser radiation has the personal peculiarities- it is coherent, monochromatic and orientation.

Optical quantum generators are the source of laser radiation. They are hard mass, gas, liquid and semiconductor. A laser can work in impulsive or in continual regimes.

Depending on character of co-operation of laser light with biological tissues ***the following types of photo biologic processes are distinguished:***

1) ***photo destructive action*** at which the thermal, hydrodynamic, photochemistry effects of light cause destruction of tissues; this type of laser using in laser surgery;

2) ***photo physics and photochemistry action*** at which the light absorb by biotissues excites atoms and molecules in them causes the photochemistry and photo physics reactions. On this type of co-operation application of laser radiation is used as therapeutic;

3) ***imperturbable action***, when a biosubstance does not change the qualities in the process of co-operation with light; it is such effects, as dispersion, reflections and penetrations; this kind is applied for diagnostics (for example laser spectroscopy).

Four basic methods of delivery of laser radiation to the patients are distinguished:

1) external or transcutaneous action: organ, vessels, nerves, pain areas are exposed to the rays through an unharmed skin in the proper area of body. If a pathological process is localizing in the superficial layers of skin, laser action is directed on it directly. The transcutaneous action is based on that the laser radiation of infra-red rays well penetrates through tissues on the depth of to 5–7 cm and arrives at damaged organ.

2) action of laser radiation on the points of acupuncture; at laser acupuncture there is possible application of continual radiation or impulsive radiation with application of different frequencies for a different pathology.

3) a internally empty way performs either through a endoscopes sets or by the special attachments, both red and infra-red radiation is here used.

4) the intravenous irradiation of blood is conducted by punction of cubital or subclavia veins; a thin light diode is entered in a vein, through which is exposed to the rays blood.

By basis of mechanism of co-operation of low energy radiation with biologic object are the photo physics and photochemistry reactions which connected with resonance absorption by tissues of light and violation of weak molecular connection, and also perception and transfer of effect of laser irradiation by liquid medium of the organism.

Basic indications to application of laser technologies in angyology are: endarteritis obliterant and atherosclerosis of extremities, diabetic angiopathy of the vessels of lower extremities, phlebitis, postthrombophlebitic syndrome, varicose illness of lower extremities.

Contra-indications to application of laser therapy are:

Absolute: oncological diseases, bleeding, diseases which reduce coagulative function of blood.

Relative: heart-vessels diseases in the stage of decompesation, cerebral sclerosis with the expressed violation of cerebral circulation of blood, acute violations of cerebral circulation of blood, disease of lungs with respiratory insufficiency, hepatic and kidney insufficiency, first half of pregnancy, active TB processes.

At varicose illness of lower extremities endovascular laser obliteration (coagulation) is used in the stage of trophic disorders, and also insufficiency of perforant veins, at the refuse of patients from radical operation. Indirect influence on perforant veins laser's radiation allows attaining their reliable obliteration. There is regress of displays of vein insufficiency in a postoperative period. Endovascular laser radiation through catheterization of the vein by Seldinger is lead to coagulation of inner surface of the vein with formation of aseptic thrombi. In a postoperative period is achieved complete replacement of the vein by scar tissues.

In complex treatment of endarteritis obliterant and atherosclerosis of extremities, diabetic angiopathy of lower extremities, varicose illness with trophic ulcers, phlebitis and postthrombophlebitic syndrome used anti-inflammatory action of laser radiation and ability of lasers to improve microcirculation of tissues by the internal irradiation of blood.

Also the laser radiation finds application in diagnostics of affectation of vessels. Laser Dopler fluorometry is used for estimation of ischemia of lower extremities at patients with the obliterant diseases of vessels.

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