MINISTRY OF HEALTH OF UKRAINE

KHARKIN NATIONAL MEDICAL UNIVERSITY

EMERGENCY SURGERY

Textbook for the 6th year students of medicine

Under the editorship of
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Emergency surgery. Textbook for the 6th year students of medicine: / Authors: V.V. Boyko, V.M. Lisovyi, V.A. Kapustnyk, L.Y. Goncharenko, I.A. Taraban, V.V. Makarov and others.; Eds. Corresponding Member of NAMSU Doctor of Medicine, Professor V.V. Boyko, Corresponding Member of NAMSU, Doctor of Medicine, Professor V.M. Lisovyi
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The typical, comprehensive, updated syllabus on Surgery (2015) for students of higher medical education of accreditation level 4 envisages learning a large volume of the course of Clinical Surgery (namely "Abdominal Surgery" by the 4th year students and "Thoracic, Cardiovascular, Endocrine Surgery" by the 5th year students) after the course of "General Surgery" learned by the 3rd year students.

The 6th year students doing the course of Surgery expand and consolidate the earlier obtained theoretical knowledge and improve their practical skills in the most complex and most dangerous for the human health component of surgery, Emergency Surgery.

The Medical License Integrated Examination "KROK 2. General Medical Training" (Surgery) and the Integrated Graduate Practical-Oriented Examination in Surgical Diseases assess the effectiveness of studying Surgery at a university concerning the issues of surgical pathology diagnosis, especially urgent, and readiness to provide emergency medical aid for patients with surgical emergencies.

The textbook for the 6th year students combines the teaching materials of the department, which were designed to study 25 topics in practical classes and 10 topics for self-study on the questions of Emergency Surgery. Each topic in the textbook contains sections corresponding to professionally-oriented tasks of the medical activity, approved by the branch standards of higher education - "Educational-professional program" and "Educational-qualification characteristic" (Kyiv, 2003), which were supplemented and updated in 2010.

The textbook, prepared by the teaching staff of the Department of Surgery No. 1 of KNMU, is designed to provide the students with the necessary teaching materials in all topics of practical training and self-study work, which will improve the training of future surgeons in accordance with the requirements of the national standards of higher education.

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PREFACE

Studying the discipline "Surgery" on the principles of the credit transfer system of training in accordance with the updated typical comprehensive syllabus in Surgery (2015) is continued as the topic Emergency Surgery learned by the 6th year students, which includes 25 classroom topics, and 10 topics for self-study.

Each topic outlined in the textbook contains sections corresponding to professionally-oriented tasks of medical activity, approved by the branch standards of higher education - "Educational-professional program" and "Educational and qualification characteristic" (Kyiv, 2003), supplemented and updated in 2010 .

Issues of etiopathogenesis, classification of diseases in accordance with IDC 10 (2001), clinical symptoms, standard protocols of additional laboratory and instrumental examination with characteristic features of evidence-based medicine for each disease, emergency care at the pre-hospital stage in surgical patients are featured; the choice of therapeutic tactics is substantiated; pathogenetically grounded conservative therapy, indications and types of surgical interventions are presented.

Considerable attention is paid to modern mini-invasive intervention - videoendoscopic, videothoracoscopic, X-ray endovascular.

Each unit is accompanied by the list of recommended literature. On the issues related to anatomy and topographical anatomy of various units of the textbook it is recommended to consults the atlases by O.O. Shalimov, V.M. Voilenko et al. mentioned in the recommended literature.

The textbook was prepared by the teachers of the Department of Surgery No. 1 of KhNMU. The textbook is intended to provide the students with the necessary learning materials in all topics of practical classes and self-study, which will contribute to the improvement of training of future surgeons in accordance with the requirements of the national standards of higher education.

ABBREVIATIONS

AAp - acute appendicitis
AA - aortic aneurysm
AAB - acid-alkaline balance
AAbS - acute abdomen syndrome
AAD - acute abdominal disease
AAG - aortoarteriotography
AAO - acute arterial occlusion
AAS - acid-alkaline state
AAT - acute arterial thrombosis
ABG - arterial blood gases
ABPI - ankle-brachial pressure index
ABS - acid-base state
ABT – antibiotic therapy
AC - abdominal cavity
ACA - aminocaproic acid
ACC - acute cholecystitis
ACE - angiotensin converting enzyme
ACS - acute coronary syndrome
ACTH - adrenocorticotropic hormone
ADH - antidiuretic hormone
ADVT – acute deep vein thrombosis
AE - acute erosions
AEM - arterial embolism
AHED - anticholinesterase drugs
AI - aortic insufficiency
AIB - anterior interventricular branch
AIO - acute intestinal obstruction
ALA - acute lung abscess
ALIES - acute limb ischemia syndrome
ALIS acute lung injury syndrome
AMA - antimitochondrial antibodies
AMI - acute myocardial infarction
anti-HAVAg IgG - class IgG antibodies to hepatitis A virus
anti-HAVAg IgM - class IgM antibodies to hepatitis A virus
anti-NCV IgG - class IgG antibodies to hepatitis C virus
AP - acute pancreatitis
APE - acute pleural empyema
APM – artificial pacemaker
APA - anatomical and physiological area
APS - antiphospholipid syndrome
APTT - activated partial thromboplastin time
APTT- activated partial thromboplastin time
ARD - acute respiratory disease
ARVI - acute respiratory viral infection
ASVT - acute superficial venous thrombosis
AU - acute ulcers
AV - artificial ventilation
AVB - atrioventricular block
AW – airway
BAP - bone alkaline phosphatase
BC - breast cancer
BCA - brachiocephalic arteries
Bd - "direct" unconjugated bilirubin
BE - bronchiectasis
BEI - butanol extracted iodine
Bi - "indirect" unconjugated bilirubin
BMD – bone mineral density
BOIVC - balloon occlusion of the inferior vena cava
BP - blood pressure
CA - coronary artery
CABG - coronary artery bypass grafting
CAD – coronary artery disease
CAG - coronary angiography
CARS - compensatory anti-inflammatory response syndrome
CBD - circulating blood deficiency
CBV - circulating blood volume
CfA - circumflex artery
CI - calcium index
CIC - circulating immune complexes
CIEP - counterimmunoelectrophoresis
CKD - chronic kidney disease
CMV – cardiac minute volume
CNS - central nervous system
COADLL - chronic obliterating arterial diseases of the lower limbs
CPE - chronic pleural empyema
CPK - creatinine phosphokinase
CPK-MB - creatinine phosphokinase MB fraction
CPR - cardiopulmonary resuscitation
CRP - C-reactive protein
CS - complement system
CT - computed tomography
CVI- cerebrovascular insufficiency
CVILL - chronic venous insufficiency of the lower limbs
CVP - central venous pressure
D- duodenum
DBD - dyshormonal breast disease
DFA - diffuse fibroadenomatosis
DFCM - diffuse fibrocystic mastopathy
DFS - diabetic foot syndrome
DIC - disseminated intravascular coagulation
DM - diabetes mellitus
DNA - deoxyribonucleic acid
DSRA - distal splenorenal anastomosis
DVT - deep vein thrombosis

ECG - electrocardiogram
echoCG - echocardiogram
EEPI – end-expiratory pressure increase
EF - ejection fraction
EMA- emergency medical aid
EMF - electromotive force
EPH - edema, proteinuria, hypertension
EPST - endoscopic papillosphincterotomy
EPT - electropulse therapy
ERCPG - endoscopic retrograde cholangiopancreatography
ERF - external respiration function
ESR - erythrocyte sedimentation rate
FCG - phonocardiogram
FGDS - fibrogastroduodenoscopy
FNPAB – fine-needle puncture aspiration biopsy
GGTP - γ-glutamyltranspeptidase
GHBA - gamma-hydroxybutyric acid
GPP - generalized purulent peritonitis
GPVR - general peripheral vascular resistance
GSD - gallstone disease
GTRH - gonadotropin-releasing hormone
HAVAg - hepatitis A antigen
HBeAg - infectious antigen
HBOT - hyperbarooxygenotherapy
HBsAg - surface antigen
HCV RNA - virus C RNA
HDV - special (defective) virus
HES - hydroxyethyl starch
HF - heart failure
HH – hiatal hernia
HLA - chronic lung abscess
HLIS - chronic limb ischemia syndrome
HLM - heart–lung machine
HPDA - hepatopancreatoduodenal area
HR - heart rate
HT - hemothorax
HTH - hormone therapy
HU - Hounsfield units (densitometry index)
IB - intestinal bleeding
ICA - internal thoracic artery
IIS –intestinal ischemic syndrome
ISD - initial surgical debridement
IVCS -inferior vena cava syndrome
IVI - intravenous immunoglobulins
LA - left atrium
LAAOS - limb acute arterial occlusion syndrome
LAPS – limb acute pain syndrome
LBT - lymphocyte blast transformation
LCA - left coronary artery
LDL - low density lipoprotein
LHE - laparoscopic cholecystectomy
LI – large intestine
LII – leukocyte intoxication index
LITA - left internal thoracic artery
LM - lactation mastitis
LMIT – leukocyte migration inhibition test
LMWH - low molecular weight heparins
LP - Liver/Pancreas Antigen antibody
LV - left ventricle
MAC – Medical Advisory Commission
MASS - Morgagni–Adams–Stokes syndrome
MCA - mesenteric-caval anastomosis
MI - mitral insufficiency
MI - mitral insufficiency
MIMR - minimally invasive myocardial revascularization
MJ - mechanical jaundice
MNR - international normalized ratio
MODS - multiple organ dysfunction syndrome
MOF - multiple organ failure
MRA - magnetic resonance angiography
MSCT - multislice computed tomography
MVL - maximal ventilation of the lungs
MVO - maximum venous outflow
MVS - mitral valve stenosis
NLA - neuroleptanalgesia
NSAD - non-specific abdominal disease
NSAID - non-steroidal anti-inflammatory drugs
NTproBNP - brain natriuretic peptide
NYHA - New York Heart Association
OI - oscillographic index
OPS - ozonated physical solution
P - pancreas
PAR - pressure-adjusted rate
PAWP - pulmonary artery wedge pressure
PBI - protein-bound iodine
PCA - portacaval anastomosis
PCT - polychemotherapy
PE - pulmonary embolism
PE - pulmonary embolism
PH - pulmonary hemorrhage
PHT - portal hypertension
PLS - perfusion lung scan
PPI - proton pump inhibitors
PT - pneumothorax
PTC – percutaneous transhepatic cholangiography
PTFE - polytetrafluoroethylene
PTS – post-thrombotic syndrome
PTT - physiotherapy treatment
PU - perforated ulcer

QCT - quantitative computed tomography
RA - road accident
RDS - respiratory distress syndrome
RDSA - respiratory distress syndrome in adults
RES - reticuloendothelial system
RGOA - right gastroomental artery
RI - rheographic index
RITA - right internal thoracic artery
RMV - respiratory minute volume
RNA - ribonucleic acid
RPS- retroperitoneal space
RR – respiratory rate
RV - right ventricle
SCD - sudden cardiac death
SCLC - small cell lung cancer
SDA - subdiaphragmatic abscess
SFMC - soluble fibrin monomer complexes
SH – strangulated hernia
SI - systolic index
SIR - systemic inflammatory response
SIRS - systemic inflammatory response syndrome
SLA - antibodies to soluble liver antigen
SP - subpectoral phlegmon
SPV - selective proximal vagotomy
SRA - splenorenal anastomosis
SSD - systemic scleroderma
SSI - surgical site infection
SSS – sick sinus syndrome
SVA - segmental venous ability
SVCS – superior vena cava syndrome
TBV - total blood volume
TCVD - transient cerebrovascular disorder
TG - thyroid gland
TI - tricuspid insufficiency
TIA - transient ischemic attacks
TLBAP - transluminal balloon angioplasty
TMLR - transmyocardial laser revascularization
TNF - tumor necrosis factor
TPMG - tomopneumomediastinography
TS - tricuspid stenosis
TSH - thyroid stimulating hormone
TSI - thyroid stimulating immunoglobulins
UC - ulcerative colitis
UFH - unfractionated heparin
UL - urolithiasis
URT- upper respiratory tract
US – ultrasonography
UVI - ultraviolet irradiation
VA - vermiform appendix
VCL - vital capacity of the lungs
VTE - venous thromboembolism
VTEC - venous thromboembolic complications
VTED - venous thromboembolic disease
VV- varicose veins
WHO- World Health Organization
WPW - Wolff-Parkinson-White syndrome

HISTORY OF SURGERY IN UKRAINE

The study of the historical aspects of surgery development in Ukraine plays an important role in developing the knowledge of the future doctors. Consideration of this range of issues allows not only to improve the educational level, but also to study the degree of surgical science development in our country at different stages of social and economic development of society, to understand the difficulties that the surgeons of antiquity and modern time had to pass through.

The knowledge of the history of surgery development will help the future physicians develop not only a sense of deep respect for the doctors of the past, but also take the basics of clinical thinking, diligence, dedication and immense responsibility.

The most important task of Ukrainian medicine is to preserve and restore the health of the population. The decree of the Cabinet of Ministers of Ukraine "On the Measures for Further Improvement of Health Care and Development of Medical Science in the Country" as well as "On Measures for the Further Improvement of Public Health" and a number of other resolutions, which envisage expansion of the network of medical and preventive institutions, improvement of provision of medical aid to the citizens, are of great importance for health development in Ukraine.

An important role in solving these problems is played by outpatient hospitals, the doctors of which render aid to 70% of patients, administer treatment and preventive measures aimed at morbidity reduction.

A great deal of work in carrying out these tasks is done by outpatient surgeons. Therefore, getting acquainted with ambulatory surgical care is an important point in treatment of the patient.

New hospitals in Ukraine are based on scientifically processed standards that ensure their compliance with the current level of organization and technology of medical treatment, as well as compliance with sanitary and hygiene requirements.

The hospital is the main sanitary-hygienic institution of the health service, performing functions of prevention, diagnosis, treatment, rehabilitation, health education, medical personnel training.

Every year Ukrainian hospitals treat millions of patients. Now the tendency to increase the size of hospitals and to complicate their structure is clearly evident.

Important structural subdivisions of the surgical hospital are the admission department and the surgery unit. The study of the peculiarities of the work of these structural subdivisions is important for understanding the principles of organization of work on inpatient surgical assistance for the population.

Deontological problems have always been the focus of the leading Ukrainian surgeons. The traditions, laid by M.I. Pirogov in his articles and in “The Diary of the Old Doctor” were worthily carried on by the entire galaxy of prominent Ukrainian surgeons (O.M. Bakulev, B.V. Petrovsky, V.I. Burakovsky, O.O. Vishnevsky, S.S. Yudin, M.M. Petrov, M.M. Amosov, and others). The problems of surgical ethics and deontology have been discussed at the International Congresses. Thus, at the International Congress of Pediatric Surgeons in Scandinavia (1996), one of the problem lectures was devoted to new ethical aspects of surgery.

Society development and medicine progress put forward the fundamental problems and issues, the knowledge of which is mandatory for the practitioner, especially the surgeon. Organ transplantation, new possibilities of resuscitation and prolonged substitution of vital body functions, actively developing fetal surgery, correction of combined developmental defects, all this and much more issues requires new legal and ethical approaches.

The rapid technologization and computerization of medicine, along with the undeniable advantages, have a serious disadvantage: such concepts as clinical thinking of the doctor, his spirituality, moral categories take the back seat.

History of Surgery in Ukraine

In Kyivan Rus, medicine led by doctors, did not exist. In the towns, there were persons among the representatives of different occupations who were engaged in treatment. For the majority of them, this was not the main, but only an additional kind of activity and earnings. Only with the increase in the population of the towns (at the time of the greatest growth the population of the ancient Kyiv

reached 100,000 inhabitants) there was a demand for medical care, which contributed to emergence of a significant number of people for whom medicine became the main profession, more often inherited. The basis of knowledge of these doctors was the age-old experience of folk empirical medicine with elements of mystical nature. Already at that time, some of them "specialized" in treatment of wounds, fractures, bloodletting, others in charming the toothache, eye treatment, obstetrics, etc. The early collections of laws ("Ruska Pravda", XI cent.) mentioned doctors, elements of their activities regulation and rewards for treatment.

Visiting foreign doctors from the countries of the West and East practiced along with the doctors-craftsmen from the indigenous population at certain princely courts as well as in large towns. They taught our doctors the medical methods of their countries and, in turn, took over our treatment methods, including the use of medicinal herbs.

The due attention to medicine was paid by Princess Olga. She founded a hospital in Kyiv, and the care for the sick was entrusted to women. In 996 Prince Volodymyr the Great and in 1096 Prince Yaroslav the Wise fixed the right of monasteries to treat patients.

By Greek examples, the monasteries and large churches, chiefly Kyiv-Pechersk Monastery, arranged shelters for the sick and disabled. The individuals who specially devoted themselves to care and treatment of patients were nominated among the monks. Prayers were considered the foremost drug; by they also practiced folk medicine. "Kyiv-Pechersk Patericon" brought to us information about the monk Agapit, who in the XI century treated at Kyiv-Pechersk Lavra.

The first prominent woman doctor of Ukraine was the granddaughter of Vladimir Monomakh - Evpraksiya Mstislavivna, who was born in 1108, was educated at the princely court and received encyclopedic education. She began her medical activities when a little girl and successfully treated patients from all over Kyiv. After marrying the Byzantine prince in 1152, she continued her medical education, studying under the most famous doctors of the Byzantine Empire. She described her experience and knowledge in the scientific treatise "The Ointments" and in the first in Rus scientific work written in Ukrainian, which is a kind of encyclopedia of the medical knowledge of those times. Now this book is stored in Lorenzo di Medici library in Florence, Italy.

During the rise of Kyivan Rus, there were also special medical works, which provided the data on the treatment of diseases, based on centuries of empirical experience of our people and the written sources of ancient scholars who came to us from other countries.

The most ancient case of surgical treatment mentioned in the written sources is tumor excision performed on Svyatoslav Yaroslavovych in 1076.

In 1237-1243 all the lands of Kyivan Rus were captured by Tatar-Mongols. The catastrophic decline in the economic and cultural life of the people did not contribute to development of medicine at that time. The western Ukrainian lands, Volyn and Galicia, suffered the least.

During the nomadic invasion, the number of maimed, sick, those who needed care and surgical aid, increased significantly. However, the deep recession of the economic and cultural life hampered medicine development. Communication with Byzantium, the southern and western Slavic peoples ceased.

In the XIII century medicine continued its development in Lviv, occupied by Tatar-Mongols, and first community hospitals were organized. And it was exactly in Lviv in 1445 that the first on the territory of Ukraine pharmacy was opened. In the XVII century similar pharmacies were opened in Chyhyryn, Kyiv, Nizhyn.

Medical treatment was out of the interests and control of state power in Ukraine. Population masses were served not by specially educated doctors, but by doctors-craftsmen named of barbers.

A copy of the Charter of Barbers dated the XVIII century is stored in Kyiv Central Archive. It defined the scope of their activity: "The skill of the barbers must consist in shaving, bloodletting, chopped and shot wounds curing, and especially tooth extraction and treatment of French pox (now called syphilis) and skin diseases". As we can see, all surgery, traumatology, treatment of venereal, skin diseases, dental practice were in the competence of the barbers.

In addition to guild barbers, a large number of people who did not belong to the guild practiced medicine in large cities. They were called private traders. These two groups were in constant competition.

After serfdom abolition, medical aid to the rural population was concentrated in local councils (zemstvo), where the leading role belonged to the landlords. In Ukraine, zemstvo was originally introduced in the east and only in 1905 in the western part.

Significant distribution of various epidemic diseases in the villages, high mortality of the population, especially children, forced the established councils to pay attention to the measures improving the medical care of the population. Doctors were invited to serve the rural population. They were supposed to provide medical care in all medical fields, including surgical care. Later these doctors became well-known surgeons, such as O. Bogayevsky, B. Kozlovsky, L. Malynovsky.

This surgery contributed to the penetration of surgical care to the broad masses of the population. In small zemstvo hospitals, quite complex surgical operations were sometimes performed.

An important role in the further improvement of medical assistance for the population of Ukraine was played by opening of educational medical facilities for training medical personnel.

In the XV century doctors training started at Krakow University, and later - at Zamostia academy. The Academy organized a hospital for 40 beds. Zamostia Academy worked for 190 years.

Despite the modest capabilities of the faculties of medicine in Krakow and Zamostia, they played a positive role in expanding scientific medical knowledge among the population. The number of graduates of these schools, especially Ukrainians and Belarusians, was small. Some of them, having received the degree of medical licentiate, continued their studies at the universities of Italy, where they received a doctor's degree. Among them were Yuri Kotermak-Drohobych, Georgy Francisko Skorina, and Philip Lyashkovsky.

The first higher educational institution in Ukraine was Kyiv-Mohyla Collegium, which was founded in 1671 by Petro Mohyla by combining the school of Kyiv brotherhood with the school of Kyiv-Pechersk Lavra.

In 1701 it became Kyiv Academy. Many of its pupils became famous scientists, founders of medicine, including such as Nestor Maksimovich-Ambodik, Andrievsky, Shumlyansky. The number of hospitals increased, State Military Hospital was opened in Kyiv as well as state hospitals in Kremenchug, Poltava, Rivne, Lubny.

The Imperial Kharkiv University was opened on January 17, 1805 under the Charter of Russian Universities in 1804. It had a faculty of medicine, which united 6 departments, Department of Surgery being among them. This marked the beginning of development of Kharkiv surgical school, one of the oldest in Ukraine and the former USSR.

Recruitment of students for the Faculty of Medicine was announced in the year of its discovery, its first dean was Professor of Surgery Pavel M. Shumlyansky, who also taught theoretical, clinical, operational surgery and desmurgy.

From 1820 to 1833 the Department of Surgery was headed by Professor **Mykola Ivanovich Yelinsky**. Under his leadership, the number of beds supervised by the department expanded to 7. Professor M.I, Yelinsky taught clinical, theoretical and operative surgery.

From 1834 to 1837 the department was headed by professor **Petro Oleksandrovych Butkovsky**. He taught clinical surgery, and also conducted a course of mental illness, pharmacology, formulation, and diet.

In 1838, the Department was headed and led until 1853 by Professor Titus Lavrentievich Vanzetti. With it the number of guarded beds expanded to 21. Professor T.L. Vanzetti read clinical and surgical surgery and ophthalmology. In 1870, ophthalmology was allocated to a separate department, and in 1874, Department of Operative Surgery was established. At TL Vanzetti for the first time began to systematize the history of the disease. In the summer of 1838 Prof. TL Vanzetti, with 4 students, traveled for 2.5 months to military settlements in Kharkiv and Ekaterinoslav provinces, where 83 operations were performed.

From 1853 to 1858, Surgery Department was headed by Professor Petro Andriyovych Naronovich. He read the course of clinical and surgical surgery.

From 1859 to 1897, the department was headed by Professor Wilhelm Fedorovich Grube. In 1867 the clinical base of the department expanded to 25 beds. V.F. Grube read clinical lectures 3 times a week 3 hours each. At lectures he inspected the patients supervised by the students, and after the lectures he performed operations. Each student was obliged to be in charge of at least two patients in the semester. The students participated in reception of ambulatory patients, performed small outpatient operations.

In 1834, a **university was opened in Kyiv**. Six years after the foundation of Kyiv University, the faculty of medicine was opened. And it was only in 1844 when therapeutic and surgical clinics for 20 beds each were organized. They immediately had a positive effect on the activities of the faculty. And most importantly, the leading departments of the faculty of medicine were headed by prominent medical scientists (among the chairmen of surgery department of Kyiv University there were **V.O, Karavayev, M.M. Volkovych** and others).

In 1865 another a university was opened **in Odessa**. Such outstanding surgeons and scientists as K. Serapin, M. Lysenkov, K. Sapezhko, A. Shchoholiv and others worked at the Faculty of Medicine of Odessa University.

After 1917, the construction of a network of medical and prophylactic institutions was started in Ukraine. After the end of the Civil War, special attention was paid to organization of surgical assistance for the miners of Donbas, workers of heavy industry of Dnipropetrovsk region, Kryviy Rih and, first of all, to provision of traumatology aid. The leading role in this work was played by Kharkiv Orthopedics and Traumatology Institute. It has long been the methodical center for the organization of traumatology aid in Ukraine.

Much work was done in the country for organization of emergency medical care, including at surgical pathology. Already in the 1930s, the level of solution of this problem was rather high. In pre-war years, special attention was paid to organization of emergency surgical care in districts of the regions. Surgical departments, led by experienced surgeons, were created. In the diagnostically and operatively complicated cases, the district surgeon was able to invite a consultant from the regional center via sanitary aviation or transport the patient to a specialized institution.

In 1930 in Kharkiv, and in 1934 in Kyiv, institutions for blood transfusion were organized. They worked at t questions of blood conservation, investigated viability of red blood cells, white blood cells. The importance of determining erythrocytes resistance as a biological criterion for storage of preserved blood was established.

New surgical schools were organized in the country, they were connected with the names of such famous surgeons as M. Volkovich, O. Krymov, O.O. Vishnevsky, B.V. Petrovsky, I. Ishchenko, M. Kolomyichenko and others.

Ukrainian surgeons along with the surgeons of other nationalities took an active part in the heroic struggle of our people with fascist Germany. Thanks to their selfless work, 72% of the wounded returned to the ranks of soldiers. The organization of medical care was staged. This principle was first developed during the First World War by Professor of Military Medical Service V. Oppel.

During the Great Patriotic War a major role in the managing the surgical service was played by the chief surgeon of the Soviet Army M. Burdenko, the Soviet Navy - Yu Dzhanelidze, the fronts and armies - M. Yelansky, P. Kupriyanov, M. Akhutin, S. Banaytis, I. Ishchenko. However, the most difficult work was done by thousands of ordinary physicians and surgeons, who, under the bombardment of the enemy, performed extremely complicated operations. Hundreds of thousands of the wounded owed their lives to these brave people.

Soviet women (40% of the front line doctors, 43% of doctor assistants, 40% of instructors and nurses) played an important role in the activity of the military-sanitary service, in the organization of treatment and service of the wounded.

During the war years, enormous experience was gained in various areas of military medicine.

After the war, many scientific schools were formed in Ukraine, they made a significant contribution to development of not only Ukrainian but also world surgery. All this contributed to development of surgical techniques, expansion of the range of surgical interventions.

In 1957, an outstanding surgeon **M.M. Amosov** created the first department of cardiac surgery in Kyiv, and in 1983 he organized Research Institute for Cardiovascular Surgery. Heart surgery was started at Kyiv Research Institute of Clinical and Experimental Surgery, Thoracic Surgery Clinic of Lviv Medical Institute, Kharkiv Research Institute of General and Emergency Surgery, and the surgical clinic of Donetsk Medical Institute.

M.M. Amosov made a huge contribution to the development of pulmonary surgery in Ukraine. He organized specialized divisions in Kyiv in 1952, and in 1955 - the first in Ukraine department of thoracic surgery. The scientific and methodical center of tracheobronchial surgery was Department of Pulmonology of Kyiv Institute for Advanced Training of Physicians (now Department of Thoracic Surgery and Pulmonology of P.L. Shupyk National Academy of Postgraduate Education). Later, the department was headed by an outstanding female surgeon, Professor Olga Matviyevna Avilova, a pioneer in the reconstructive surgery of the trachea and bronchi.

Formation of anesthesiology service in Ukraine began in the 1950's. Anesthesiology centers were organized at leading surgical hospitals. In 1957 Department of Thoracic Surgery was reorganized into Department of Thoracic Surgery and Anesthesiology of Kiev Institute for Advanced Training of Physicians. It was headed by professor A.I. Troshchynsky.

The activity of an outstanding surgeon, doctor of medical sciences, professor, member of the Academy of Sciences of the Ukrainian SSR Aleksandr Alekseevich Shalimov, one of the founders of Ukrainian and Kharkiv surgical school, is worth mentioning as a separate point (Photo 1).

Photo 1. **Academy member, O.O. Shalimov**

He received medical education at Kuban Medical Institute, after which in 1941 he was sent as chief physician and surgeon to Nerchinsk-Zavodsky District Hospital of Chita Region. In 1944 he was appointed the chief physician and head of surgery department of Petrovsk-Zabaikalsky interdistrict hospital. From 1946 he was the head of surgery department of Bryansk Regional Hospital. In 1949 he was appointed the chief surgeon of Oriol Regional Department of Health.

At the end of 1952, O.O. Shalimov was elected an assistant of Department of Hospital Surgery of Kursk Medical Institute. However, he was more interested in practical work and in 1953 he was appointed the chief surgeon of Bryansk Regional Department of Health and simultaneously the head of surgery department of the regional hospital. In 1955, he defended his thesis for the degree of a candidate of medical sciences on creation of an artificial esophagus in scar obstruction.

In 1957, he was elected Associate Professor of Department of Intermediate level Surgery of Kharkiv Medical Institute. In 1958, he defended his doctoral dissertation on surgical treatment of cancer of pancreatic head and ampulla of Vater, and in 1959 he headed Thoracic Surgery and Anesthesiology Department at Institute for Advanced Trainingt of Physicians in Kharkiv.

From 1965, he headed Kharkiv Research Institute of General and Emergency Surgery and Department of Thoracoabdominal Surgery at Institute for Advanced training of Physicians. In 1970, he was elected the head of Department of Thoracoabdominal Surgery at Kyiv Institute for Advanced Training of Physicians and was appointed Director of Kyiv Research Institute of Hematology and Blood Transfusion. In May 1972 he became Director of the newly established Kyiv Research Institute of Clinical and Experimental Surgery.

O.O. Shalimov published more than 830 research works, including more than 20 monographs, he authored more than 100 inventions. The main directions of his research were surgery of the digestive organs, cardiovascular system. O.O. Shalimov paid much attention to development of surgical methods of treatment of complicated peptic and duodenal ulcer. He developed and implemented a number of original surgical techniques for resection of the stomach, modern methods of plastic bile duct surgery. He made a great contribution to development of surgical treatment of pancreatic diseases. He developed and implemented in practice the original methods of surgical treatment of various forms of chronic pancreatitis. Under his leadership, transplantation of pancreas in patients with diabetes mellitus was performed for the first time in the Ukrainian SSR. For his research

in the field of diagnosis and treatment of pancreatic diseases in 1977, he was awarded the State Prize of the Ukrainian SSR.

In 1978 Oleksandr Oleksiyovych was elected a full member of the Academy of Sciences of Ukrainian SSR (now, National Academy of Sciences of Ukraine), in 1993 – a member of Academy of Medical Sciences of Ukraine.

A high position in the research of O.O. Shalimov was held by cardiac and vascular surgery. He participated in development of an artificial blood circulation device, flat dilators for mitral commissurotomy. O.O. Shalimov proposed and introduced into practice several new surgical techniques of treatment of obliterating vascular diseases, developed and implemented a number of instruments for vascular surgery.

For development and introduction into clinical practice of the techniques for cryodestruction of malignant neoplasms, he was awarded the State Prize of the Ukrainian SSR in 1985.

By a decree of the Presidium of the Supreme Soviet of the USSR dated July 22, 1982, Oleksandr Shalimov was awarded the title of the Hero of Socialist Labor with the award of the Order of Lenin and the gold medal "Sickle and Hammer".

Monographs of O.O. Shalimov "Diseases of the pancreas and their surgical treatment" (1970), "Atlas of operations on the esophagus, stomach and duodenum" (1975, co-authored), "Surgery of the esophagus" (1975), "Atlas of operations on the liver, biliary tract, pancreas and intestines" (1979, co-authored) and others are valuable tools for any surgeon.

By the Decree of the President of Ukraine No. 974/2005 dated June 20, 2005, for outstanding personal merits to Ukraine in development of health system, the rise of the prestige of Ukrainian surgical school and medical science in the world, long-term ascetic medical activity, the honorary director of the Institute of Clinical and Experimental Surgery of the Academy of Medical Sciences of Ukraine, Doctor of Medical Sciences, member of the Academy of Medical Sciences of Ukraine, member of the National Academy of Sciences of Ukraine Oleksandr Oleksiyovych Shalimov was awarded the title of Hero of Ukraine with awarding of the Order of the State.

O.O. Shalimov combined t academic, pedagogical and practical work with social activities. He was a member of the board of the All-Union Scientific Societies of Surgeons, Gastroenterologists and Cardiologists, a member of the International Association of Surgeons, the Chairman of the Board of Ukrainian Republican Scientific Society of Surgeons, the chief surgeon of Ministry of Health of Ukrainian SSR, the editor-in-chief of the *Clinical Surgery*, a deputy of the Supreme Council of Ukrainian SSR of the 8-10th convocations. He was an honorary citizen of Kyiv and Kharkiv.

He spent his last years and worked in Kyiv. He died on February 28, 2006 and was buried on the Central Avenue of Baykove Cemetery in Kyiv.

During the recent decade the diagnosis and surgical methods of treatment of various pathologies, methods for prevention of surgical diseases have improved, the network of specialized medical institutions has expanded, the scientific research has become more fundamental. We owe much this progress to our predecessors, the prominent surgeons of the past.

History of Surgery Department No. 1 of KhNMU

For a long time, the desire of Kharkiv University to open a hospital surgical clinic faced push-back from the most backward members of Kharkiv City Council (Duma) and the administration of the already opened Oleksander hospital (in Blagoveschenska street), who apparently did not understood or did not want to understand the importance of the tasks to organize and improve medical education in the country and develop surgical aid in Kharkiv.

In this regard, only considerably later, i.e. during the Russian-Turkish War of 1877-78, Kharkiv Public Society of Care for Wounded and Diseases Soldiers gave Kharkiv University their consent to open hospital clinics (surgical and therapeutic) in The Red Cross Hospital, located on the territory of the garrison military hospital (Kultury street). In addition, the university in its solemn assembly hall further organized a surgical department for 30 beds for the established hospital surgical clinic. This was the beginning of the **surgical hospital of the Faculty of Medicine of Kharkiv University**.

Professor of theoretical surgery, a well-known surgeon, a wonderful teacher, an outstanding public figure, a student of V.F. Grube and M.I. Pirogov, **Ivan Kondratovich Zarubin** was temporarily appointed the head of the department (Photo 2). He headed the department from 1877 to 1894. The 1877-1878 report of the Faculty of Medicine states that Professor I.K. Zarubin in the improvised clinic "paid 6-hour visits with the 5th year students every week". The report also noted that the number of patients in the hospital was 248, and in the hall - 132.

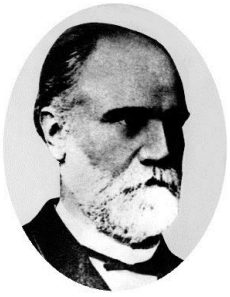


Photo 2. **Professor I.K. Zarubin**

Organization of this clinic and its 1.5 year activities on the basis of the hospital and within the walls of the assembly hall of the university definitely contributed to improvement of surgical care for the wounded and surgical training of the medical graduates.

On May 12, 1878, Kharkiv City Council approved the provision for hospital clinics (surgical and therapeutic), which were organized in the city Olexander Hospital opened on October 1, 1869. However, the conditions at which it was allowed to organize these clinics with the opportunity to teach surgery and therapy on the basis of this hospital, were described by Kharkiv University as "difficult conditions".

Hospital surgical clinic started its work in difficult conditions, in unapproved premises, without sufficient funds for the purchase of surgical instruments and equipment, without a lecture hall, a dressing room and with a rather primitive operating room. Prof. I.K. Zarubin with an intern, doctor Nebykov, began to hold classes with the 5th year students, remaining, as well, the head of Department of Theoretical Surgery.

In 1882, I.K. Zarubin was finally appointed to the professorship of the hospital surgical clinic. This made it possible to concentrate the efforts of the head of the clinic on organization of the pedagogical process in the hospital clinic.

In the 1882-83, Professor I.K. Zarubin, in addition to regular clinical classes, delivered the course of lectures about abdominal diseases to the 5th-year students (2 hours per week). The number of patients in the hospital increased from 170 to 220 per year. The ambulatory department started its work. A sufficient amount of surgical instruments was purchased. In these and subsequent years, the number of students ranged from 72 to 250.

Professor I.K. Zarubin was the author of more than 20 scientific papers covering various questions of surgery: fractures of the skull base, restoration of the lower lip, aneurysms of the innominate artery, thoracocentesis, hypertrophy of the prostate gland, hypospadias, gunshot wound of the knee joint, ulcer of the sole, etc. He was the author of manuals on General and Specialty Surgery "General Surgical Pathology" and "Special Surgical Pathology". These manuals for students in the form of lithographic lectures came of print in four editions.

Being a prominent clinical surgeon, excellent teacher, I.K. Zarubin was engaged in extensive social work for the Red Cross and Society of Research Sciences, for almost 20 years he was the dean of the faculty of medicine.

In spring of 1889 due to the expiration of the 30-year term of service, Honored Professor of the Surgical Clinic I.K. Zarubin was appointed director of this clinic, and the vacancy of professor was filled by professor of surgical pathology **Maxym Semenovych Subotin**. In that situation the hospital surgical clinic was represented by two professors. I.K. Zarubin conducted classes with 48 students at Olexander Hospital, and M.S. Subtitan taught intermediate level surgery to 24 students in 25-bed "poor uniform material of the military hospital" and in his private hospital. The name of M.S. Subotin in these years was associated with development of the method of active aspiration at pleura empyema, he wrote "General Surgical Pathology" and a number of other works.

In January 1890, Professor M.S. Subotin was transferred to St. Petersburg Military Medical Academy and the surgical clinic worked in Alexander Hospital under the direction of Professor I.K. Zarubin until his retirement in May 1894.

At the same time (in May 1894) the post of the head of the hospital surgical clinic was occupied by a disciple of Professor V.F. Grube Professor Apollinariy Grygorovych Podrez (Photo 3),

who headed the department from 1894 to 1900. Professor A.G. Podrez was a brilliant surgeon-innovator, an outstanding teacher and tireless worker of science.

Having seen the extremely unsatisfactory conditions for the proper course of hospital surgery in Olexander city hospital, he repeatedly appealed to the Faculty of Medicine with requests to change these conditions in accordance with the requirements of practical medicine and the tasks of clinical teaching. The constant preoccupation of the head of the clinic was to provide normal conditions for teaching the students, treating the patients, performing research.

In 1895, Professor A.G. Podrez started delivering a course of genitourinary diseases (once a week for 2 hours). He organized ambulatory reception 2 times a week with the participation of students, regular evening professor's rounds of the patients. Until 1898, owing to a private donation, a lecture hall was arranged in Olexander Hospital.

However, the majority of the requirements of Professor A.G. Podrez were not satisfied due to lacking funds allocated to the university, because of "reluctance of the city to somehow ease the task of teaching". A.G. Podrez himself in the presentation to the Faculty of Medicine "On the Impossible Condition of the Clinic" stated that in it he found "his heavy cross".

Photo 3 **Professor A.G. Podrez**

One way or another, but with the advent of Professor A.G. Podrez the activity of the hospital clinic considerably revived in all aspects: the number of patients increased (to 731 in 1897) as well as the staff, the conditions for conducting classes improved, the number of operations increased while the number of postoperative complications decreased, research work intensified.

Professor A.G. Podrez wrote 46 research works, including on the technique of craniotomy, peptic ulcer surgery, peritonitis treatment with laparotomy, genitourinary surgery, surgical treatment of the hip joint tuberculosis, and others.

In 1897 A.G. Podrez was the first in the world to perform a successful surgical intervention for a gunshot wound of the heart. In 1898, for the first time in Ukraine, he transplanted the ureter into the rectum. In the same year, Professor A.G. Podrez also for the first time, opened a question of urgent operation for strangulated hernia.

During the period of leadership of professor A.G. Podrez, a privat-docent M.M. Filippov began to deliver the lectures on pediatric surgery at the hospital for children's non-communicable diseases. Thus, the start of the pediatric surgery clinic was marked, which, after the October Revolution, became an independent university department.

A senseless death interrupted the active and useful wok of the outstanding surgeon, teacher, scientist, professor A.G. Podrez, he tragically died in his prime at the age of 48.

After his death in 1900, teaching hospital surgery was delegated to Professor of Surgical Pathology **Mykola Andriyovych Sokolov**, who headed the department from 1900 to 1909 and was a representative of the Moscow surgical school.

Professor M.A. Sokolov began teaching hospital surgery "in the terrible conditions of Olexander Hospital". In 1902, he was appointed to the position of the professor of the hospital surgical clinic. At that time, the consent of the higher authorities for the construction of a new building for hospital clinics was obtained. In 1904, the department moved into a two-story building (before it occupied one floor) on the territory of Olexander Hospital, where there was an operating room, a dressing room, a small laboratory, an X-ray unit, 5 small wards of 30 beds, a classroom, a professor's office. This created relatively satisfactory conditions for conducting classes, medical and research work. In addition to the professor, the teaching was provided by full-time doctors Kosenko and Livshits, part-time doctors Stoklytsky, Burlakov and Lyubovsky. Under the direction of Professor M.A. Sokolov urgent surgical aid was intensified. Along with the teaching and medical work, research was conducted at large.

Of the 35 scientific papers of Professor M.A. Sokolov, the main ones were devoted to surgical treatment for echinococcus infection, spleen diseases, the questions of restorative surgery, and others.

He proposed modification of Klapp's suture in surgery for varicose veins of the lower extremities. M.A. Sokolov took an active part in creation of the journal "Herald of Surgery and Related Sciences", its publication started in 1900.

After the death of M.A. Sokolov, from 1909 to 1920 Department of Hospital Surgery was headed by Professor **Yulian Romanovich Pensky**, who was previously in charge of Department of Operative Surgery and Topographic Anatomy. Yu.R. Pensky significantly increased the staff of the department: associate professor B.G. Sharetsky, assistant V.P. Nedokhlibov, residents Popov, Gutovsky, Kapustin, Klemchinsky, Katz worked with him. The clinic intensified practical training of students. Particular attention was paid to urgent surgery. ENT operations, including laryngeal cancer, were performed. In 1912, at the 12th Congress of Russian Surgeons, Professor Yu.R. Pensky made a report "On early operation for acute appendicitis". In 1914, he and his colleague, doctor Trofymov, for the first time in southern Russia, performed pericardiotomy for adhesive pericarditis. In 1893, together with M.M. Kuznetsov, Yu.R. Pensky proposed suture of the liver during its resection. He also developed surgery for resection of the gastric pylorus, ligation of the middle artery of the dura mater, formation of an artificial bladder from the intestinal loop, and others.

It should be noted that the hospital surgical clinic, which from the first years of its existence was headed by gifted professors of surgery, excellent organizers of medical practice and pedagogical process, highly erudite, talented scientists, brilliant clinicians and operators, quickly gained popularity and trust of the population of Kharkiv. Over the years, it remained the main center for surgical care, and first of all emergency care, in Kharkiv. In spite of the extremely unsatisfactory conditions of the premises, equipment, and state, it became an authoritative school of surgical training of students and doctors who always with deep gratitude mentioned the experience necessary in their daily practical medical work, which they acquired in the walls of the Surgical Clinic.

Only in 1900, the newly built City Hospital No. 2 undertook urgent assistance, mainly in the factory district of Kharkiv, and Ukrainian Institute for Emergency Surgery and Blood Transfusion, which was organized in 1933, shared this difficult and highly responsible activity with it. In addition, urgent surgical care was absolutely necessary for a full-fledged pedagogical process in hospital surgical clinic.

After the death of Yu.R. Pensky, from 1920 to 1933, the department was headed by Professor Ivan V. Kudintsev, who before this was the head of Surgery Department at Higher Medical Courses for Women of Kharkiv Medical Society, organized in Kharkiv in 1910.

This clinic was also located on the territory of Olexander Hospital, but in the old building, where in 1878 Hospital Surgery Department of the Faculty of Medicine of Kharkiv University started its work. In 1920, Higher Medical Courses for Women were united with the Faculty of Medicine of Kharkiv University, and these two medical educational institutions were transformed into Kharkiv Medical Academy, and subsequently (in 1921) the Academy was renamed into Kharkiv Medical Institute.

Professor I.V. Kudintsev expanded the premises of the surgical hospital clinic, which occupied the lower floor of the new clinical building, after which the clinic could place up to 80 surgical patients.

The department consisted of highly qualified teachers (doctors and candidates of medical sciences): professors A.A. Chugayev and Ya.B. Voytashevsky, privat-docent S.A. Krichevsky and A.Ya. Krasnov, assistants M.M Milostanov and V.P. Nedokhlibov and others.

Professor I.V. Kudintsev paid a lot of attention to the diseases of the genitourinary system. He was the author of works about opening pulmonary abscesses, adrenal function, treatment of the prostate hypertrophy, narrowing of the urethra canal, and others.

Under the guidance of a brilliant clinician, Professor I.V. Kudintsev, the clinic paved the way for large-scale organization of urgent surgical care, fighting for early hospitalization of urgent patients and implementation of the most advanced surgical tactics. A highly competent school of urgent surgeons was established in the clinic. A significant role in this was played by his colleague Associate Professor, and later Professor of the Department A.A. Chugayev.

The second professor **Anatoly Andriyovych Chugayev**, a highly skilled surgeon and teacher, was in charge of qualified aid for patients with urgent surgical diseases. His was the author of one of the first monographs on urgent surgery, a two-volume book "Emergency Surgery" (1927), which was reissued for many times and was a desktop book of surgeons.

Department of Hospital Surgery, headed by prof. I.V. Kudintsev prepared several professors: A.A. Chugayev, B.Ya. Voytashevsky, V.P. Nedokhlibov, M.M. Mylostanov, AA Zymnytsky, who in the future were in charge of surgical departments in Kharkiv and other cities. Prof. V.P. Nedokhlibov occupied the chair of surgery at Kharkiv Medical Institute, prof. V.Ya. Voytashevsky - Urology, N.N. Milostanov - Military Field Surgery at Ukrainian Institute of Advanced Training for Physicians, A.A. Zimnitsky - General Surgery at Dagestan Medical Institute.

The staff of the department, headed by Professor I.V. Kudintsev took an active part in organizational work on implementation of new forms of clinical surgery teaching, editing the curriculum, maximum involvement of the students in practical activity. Night shifts of the students in the clinic, systematic rounds of the professor, a system of traineeship for doctors were introduced in the clinic.

After the retirement of professor I.V. Kudintsev due to his 65th anniversary, Department of Hospital Surgery was headed by Professor **Alexander Vasyliovych Melnykov**, who headed it from 1933 to 1939 and was one of the talented students of the outstanding anatomist V.M. Shevkunenko and clinician S.P. Fedorov

A brilliant clinical surgeon, lecturer and speaker, a great scientist with a wide range of scientific interests and research, prof. O.V. Melnikov contributed to the high prestige of the department. The period of his management was particularly fruitful. He significantly intensified all types of work of the department and, especially, research.

Particular attention was paid to development of issues related to acute diseases of the abdominal cavity and anaerobic infection, which the staff of the department began to investigate deeply both clinically and experimentally. A special laboratory for investigation of anaerobic infection was organized.

The number of beds was increased to 100 (albeit at the expense of the classroom which was in the surgical building from its first days) and the equipment of the operating room was modernized.

In the clinical work a significant place was occupied by scheduled surgery.

While working in this clinic O.V. Melnykov paid much attention to training of personnel, having knitted together a large team of doctors and scientists. Under his leadership a number of doctoral and candidate's theses were defended, professors and heads of surgical departments were trained.

The positions of associate professor were occupied by Professor A.A. Chugayev, a great specialist in urgent surgery, and M.M. Levin, who defended his doctoral dissertation and wrote a monograph entitled "Disease of the Stomach Operated for Ulcer" (1938).

A number of heads of departments of surgery of medical institutes came from the clinic: M.M. Levin - Kharkiv, K.P. Hess-de-Calve - Crimea, G.B. Monashkin - Dagestan.

A number of candidate's theses were defended (T.D. Pysareva, Y.E. Yelkin, A.S. Zolotarev, S.M. Pravdnikov, etc.). The residents who worked in the surgical departments of the hospital were also involved in the research. a large number of scientific articles were published during the work O.V. Melnikov.

O.V. Melnykov published a number of works on the questions of clinical surgery, oncology and proposed new original methods of operations.

O.V. Melnykov worked part-time at Kharkiv X-ray and Cancer Institute. He, together with M.P. Mikhedko, published a monograph devoted to malignant neoplasms of the stomach and, together with M.I. Lifshits, issued a two-volume work "Clinic of Malignant Tumors" (1931). During these years O.V. Melnikov launched extensive research work on experimental surgery and oncology at Ukrainian Institute of Experimental Medicine.

Professor O.V. Melnykov was an active organizer and participant in the All-union and republican congresses of surgeons and oncologists, at which he spoke with program reports, deep in content and bright in form.

He can by right be recognized as the founder of clinical oncology in Ukraine.

After moving to Leningrad in 1939 where he occupied the chair of intermediate level surgery of Navy Medical Academy. From 1939 to 1941 Department of Advanced Level Surgery was headed by Professor **Volodymyr Ivanovych Yost**, who, along with the educational work, paid a lot of attention to development of gastric surgery issues.

At the beginning of the Great Patriotic War in 1941 V.I. Yost joined the ranks of the Soviet Army. In this regard, Department of Advanced Level Surgery was again entrusted to Professor I.V. Kudintsev (until the evacuation of Kharkiv Medical Institute to Orenburg).

During the Great Patriotic War, the chair of surgery was temporarily occupied by professor **Mark Myronovych Levin** (from 1941 to 1944). At surgery department of the regional 120-bed hospital, M.M. Levin with his colleagues did a lot of work on providing surgical care to the population of Orenburg and the region. In addition to teaching work, he also performed a great advisory and operative work in a number of military hospitals in Orenburg. During these years a large number of doctors were trained for the Soviet Army at this clinic.

In 1944 professor V.I Yost became the head of the department again.

After the return of the medical institute to Kharkiv from 1944 to 1952, the clinic of surgery was headed by Professor **Isay Solomonovych Kogan.**

In the postwar years, the staff of the Department of Advanced Level Surgery paid special attention to investigation of general and local purulent infection, the use of antibiotics in surgery, restorative treatment of the wounded. According to the materials of these research T.M.Karavanova, V.P. Riumshina, O.S. Kudintseva, V.S. Voronchenko, V.O. Golubeva defended candidate theses, and G.G. Karavanov - doctoral.

From 1952 to 1953, the acting head of Department of Advanced Level Surgery was Associate Professor, Doctor of Medical Sciences, **S.A. Katz** (from department of general surgery of the Faculty of Pediatrics and The Faculty of Sanitation and Hygiene of medical institute).

From 1953 to 1959, the department was headed by **Borys Yevmenovych Pankratiev**.

During this period, the operating unit (with allocation of an operating room for urgent surgery) was reconstructed. A new lighting system was brought to the operating room and a kit for thoracic surgery was purchased. The classroom was renewed.

In these conditions, thoracic surgery was launched and the volume of assistance for health authorities was considerably expanded by means of advisory assistance for outpatient clinics, district enterprises, prevention of industrial and agricultural injuries, etc.

The 5^{th} and 6^{th} year students had an opportunity to get acquainted with the issues of urgent, purulent and traumatic care. Job placement was organized for the 6^{th} year students at 20 surgical departments of the city and district hospitals of Kharkiv region. The staff of the department also conducted active work in the student's scientific society: for 3 years, the students delivered 14 papers on different sections of surgery.

The main research problem of the department during this period was the role of neurogenic factor in traumatic and surgical shock, terminal state, clinical death, and the problem of resuscitation (B.E. Pankratiev, V.F. Postnikova, etc.). much attention was also paid to the study of the changes in the intra- and extramural nervous system and to the role of the nervous system in the pathogenesis of a number of diseases: peptic ulcer disease, cholecystitis, acute intestinal obstruction, acute peritonitis, and others. (B.Ye. Pankratiev, V.F. Postnikova, V.T. Zaitsev, I.G. Mitasov, G.I. Dudenko, A.F. Chebakova, etc.)

To study these issues, a laboratory for neurohistological research was established; a freezing microtome and a functional device for the microphotography of histological specimen were purchased.

The method of artificial hypothermia with cooling of only the central nervous system (head and spine) was developed in the experiment and a special rubber "semi-suit" (helmet and back "cape") was purchased.

This period was very fruitful for research. Assistants of the department A.F. Chebakova, Yu.A. Lebedenko, G.I. Dudenko, I.G. Mitasov defended candidate's theses on the topics of urgent and purulent surgery. Professor B. Ye. Pankratiev, associate professors P.S. Gontar, K.E. Odintsova performed research on surgical treatment of lung abscesses, cancer of pancreas and papilla of Vater, treatment of coronary failure with the help of Novocain blockade.

Professor B.Ye. Pankratiev was a member of the editorial board for preparation of a nine-volume manual on clinical surgery published in 1964.

From 1959 to 1965, the chair of hospital surgery was occupied by Professor **Tetyana Ivanivna Tikhonova**, who for the previous two years was the second professor of this department.

During this period, the clinic expanded to 200 beds (120 surgical, 40 traumatology and 40 urological) with a well-equipped large operating unit owing to additional floors of the surgical building of City Clinical Hospital No 1.

The clinic provided urgent surgical aid 3-4 times a week with round-the-clock shifts and had a sufficient clinical and medical staff.

During these years, Department of Advanced Level Surgery provided a multi-faceted pedagogical process with the 5^{th} and 6^{th} year students of the Faculty of Medicine and 6^{th} and 7^{th} year students of the evening department of the same faculty, teaching not only hospital surgery but also military field and maxillofacial surgery, oncology.

From 1951 to 1964 the course of military field surgery was taught by associate professor V.I. Kudintsev, and from 1965 to 1970 by associate professor F.F. Usikov. In addition to practical classes, the teachers of the department annually took part in preparation, organization of field training with deployment of health support battalion and active participation of the students.

In addition to the clinic the teaching was carried out at the outpatient hospital, regional oncology hospital and traumatology department of the Institute of Orthopedics and Traumatology. Job placement for the 6^{th} year students was organized at 25 district medical institutions of Kharkiv, Poltava and Sumy regions.

Professor T.I. Tikhonova successfully coped with these huge tasks owing to the consolidated work of the creative team of surgeons-teachers: V.T. Zaitsev, G.I. Dudenko, O.V. Bernesnev, F.P. Vorobyov, F.S. Tkach, F.F. Usikov, G.D. Dotsenko, B.D. Amelichev, B.F. Senchenko, L.N. Sharlay, Yu.A. Lebedenko and others.

Department of Advanced Level Surgery performed a fruitful and innovative research, the beginning of which was laid by the previous head of the department Professor B.Ye. Pankratiev On the initiative of T.I. Tikhonova the laboratory of the department was modernized, modern biochemical and physiological methods of research were set up. They provided an adequate level of research at the department.

The research topics were chiefly devoted to acute abdominal diseases, including in patients of the elderly and old age, determining the function of the pancreas and adrenal cortex in acute surgical pathology of the abdominal organs.

The department members defended a doctoral dissertation (associate professor A.N. Gubska, a (course of maxillofacial surgery), and candidate theses (V.T. Zaitsev, B.D. Amelichev, M.I. Koval, B.F. Senchenko).

Subsequently, a galaxy of future professors, heads of surgical departments came out of the department, V.T. Zaitsev, G.I. Dudenko, I.G. Mitasov, A.V. Bernesnev, F.S. Tkach (surgeon-endocrinologist).

Professor T.I. Tikhonova was the author of 80 scientific works, she prepared 3 doctors and 9 candidates of medical sciences.

In 1965, T.I. Tikhonova was again assigned the chair of General Surgery of the Faculty of Medicine, which she occupied until the end of her academic activity (in 1975).

During her work at Kharkiv Medical Institute, professor T.I. Tikhonova was the dean of the Faculty of Medicine (1959-1960), and in 1960-1962 she was a vice rector of the Institute for educational work (after the death of professor M.M. Bokarius).

In 1965 (until 1970), the department was headed by Professor Yuriy Maksymovych Orlenko. Under his leadership, the staff of the department constantly improved teaching and methodical work. The classes with the 5th and 6th year students of the Faculty of Medicine, and the 6th and 7th year students the evening department were conducted by experienced teachers: associate professors F.F. Usikov, L.N. Sharlay, G.B. Gavrilov, assistants V.A. Likhachev, V.E. Oleksiyenko, G.D. Dotsenko V.L. Dubrovin, B.I. Gaiday, I.P. Pluzhnik, I.M. Lytvynov, P.S. Isaev, G.P. Oliynyk.

Professor Yu.M. Orlenko did a significant work on organization and improvement of the medical process in the clinic. A unified system of active post-operative management of the patients was introduced; it took into account early sitting up and feeding of the patients after surgery, which was facilitated by the organization of two intensive care units. The method of treatment for acute pancreatitis was developed.

Anesthesiology service, which was organized at the clinic, made it possible to carry out major surgical interventions (including on the lungs) using modern methods of general anesthesia, and improved operational technique.

Photo 4. **Professor V.T. Zaitsev**

The department staff took an active part in research work. Two doctoral (F.F. Usikov, A,V. Beresnev) and 4 candidate (G.D. Dotsenko, F.I. Dudenko, A.D. Sheinin, E.O. Orinigalieva) dissertations were defended, a lot of research articles were published.

A new stage of the department activity began in 1970 with the advent of the chairman Volodymyr Terentiyovych Zaitsev (photo 4), who headed the department for 30 years (until July 1999).

Demanding to himself and his subordinates, he headed all kinds of work of the department. The latest achievements of modern surgery in the diagnosis and treatment of surgical pathology, especially urgent were introduced. The range of surgical interventions expanded. Practical medicine began to receive effective help. The teaching staff of the department, namely professors L.N. Sharlay, V.M. Mylostanova, G.D. Dotsenko, assistant B.I. Gayaday, I.M. Dyukarev, V.L. Dubrovina, L.Y. Galimova (hereinafter - Goncharenko), V.I. Shcherbakov actively participated in improvement of the educational process, introducing progressive teaching methods. Much attention was paid to pedagogical skill improvement, qualification of all medical and paramedical personnel of the clinic. To this end, compulsory clinical discussions of patients scheduled for surgery, weekly review conferences were introduced. All clinical staff was involved in research activities.

In 1974, V.T. Zaitsev was appointed the director of Kharkiv Research Institute of General and Emergency Surgery, and the department moved to the institute, the clinic of which was a multidisciplinary specialized surgical institution. From 1977, the clinical base of the department expanded at the expense of surgical departments of a new city clinical hospital of emergency medical aid named after. Professor O.I. Meshchaninov, thus bed fund of the department increased up to 500.

The new conditions allowed teaching of advanced level surgery to the 5th year students and primary majoring in surgery of the 6th year students at a high modern level in accordance with the current curriculum.

In 1974, V.T. Zaitsev defended his doctoral dissertation, he was awarded the academic title of professor. Associate professor V.M. Milostanov for many years managed the job placement of the students. The staff of the department was replenished by young teachers - assistants O.S. Kudynenko, Yu.I. Migrin, O.V. Kuznetsov, G.I. Gerbenko, N.F. Polovynka, who passed the student's scientific society of the department, postgraduate course or clinical residency at the department. Assistant V.I. Lupaltsev, who took a postgraduate course under the supervision of Professor O.O. Shalimov defended his candidate thesis, became assistant professor, and then, after defense of the doctoral dissertation under the supervision of Professor V.T. Zaitsev was appointed the Head of Department of Surgery of

the Faculty of Pediatrics and the Faculty of Dentistry of Kharkiv Medical Institute (1984). Assistant L.Y. Goncharenko, who for many years supervised the work of the student's scientific society, held the position of the associate professor of the department and replaced the position of the director of teaching of the department previously occupied V.M. Mylostanova (1985).

A large number of postgraduate students and clinical residents, not only Ukrainian but also foreign ones, were trained by the department every year.

The staff of the department successfully combined enormous pedagogical and educational work with medical and research activity.

The main direction of the department research was the study of various issues of urgent pathology of the digestive system (complication of peptic ulcer, acute appendicitis, obstructed hernia, acute intestinal obstruction, peritonitis), as well as vascular, thoracic and other subjects.

A significant role in optimization of the educational process at the department and the research activity was played by creation in 1986 of the Scientific-Training and Production Association "Surgery", which united Kharkiv Research Institute of General and Emergency Surgery, Kharkiv City Clinical Hospital of Emergency Medical Aid and Department of Advanced Level Surgery of Kharkiv Medical Institute.

It is impossible not to dwell on the personality of Professor V.T. Zaitsev and not to emphasize the special role he played in the history of the department.

All his career was connected with Kharkiv and his medical institutions. After graduating with honor of the Faculty of Medicine of Kharkiv Medical Institute in 1954, he covered a long way from a clinical intern, an assistant and associate professor to the head of department, which he was in charge of for 30 years, and to the director of Kharkiv Research Institute of General and Emergency Surgery, which he led for 25 years.

V.T. Zaitsev was a highly skilled clinician, surgeon, scientist, and educator.

As a surgeon he possessed all the arsenal of modern surgical interventions on the abdominal and chest organs, he was constantly engaged in surgical activities, performing annually about 300 different operations, including those in patients with concomitant pathology (oncological, urological, gynecological).

An outstanding surgeon, he turned to life thousands of patients.

The research of V.T. Zaitsev covered a wide range of issues, from the study of oxygen supply of tissues in operations on the organs of the chest and abdominal cavities to the use of modern methods of cybernetics for diagnosis and the choice of surgical intervention in surgical patients.

However, the greatest place in the scientific developments of V.T. Zaitsev was occupied by more complex issues of urgent surgery and, above all, acute pathology of the abdominal cavity.

A special place in the scientific interests of V.T. Zaitsev was occupied by surgery for peptic ulcer and duodenal ulcer, its complicated forms. His doctoral dissertation for the first time in our country proved the necessity of an individual approach to the choice of the method of surgical intervention in patients with peptic ulcer and the effectiveness of using organ-saving operations in this complex pathology. The cycle of these works included development of original organ-saving methods of operations, the main achievement of which was a significant reduction of the immediate and remote postoperative complications, while simultaneously preserving their radical character.

He published about 450 scientific works, including 24 monographs, 3 textbooks and about 20 other teaching materials. He had 40 patents for inventions, 2 patents of Ukraine.

For the cycle of works "Development, theoretical substantiation and clinical introduction of new organ-saving methods of surgical treatment for bleeding stomach and duodenum ulcers" V.T. Zaitsev was awarded the State Prize of Ukraine in the field of science and technology in 1990.

As a student of academy member O.O. Shalimov, V.T. Zaitsev implemented the scientific plans of his teacher and at the same time created his own scientific and practical school of surgeons for emergency surgical care.

Under the direction of V.T. Zaitsev, a large number of practicing doctors, about 100 scientists (21 doctors and 63 candidates of medical sciences) were trained, not only for Kharkiv and the region,

but also for the entire Soviet Union as well as for many countries of Asia, Africa, Latin America. A large number of Ukrainian and foreign clinical residents studied and perfected their surgical skills.

His highly useful pedagogical activity was marked with an honorary badge "Excellence in Higher Education".

V.T. Zaitsev took an active part in creation and became the first head of the Scientific-Training and Production Association "Surgery", organized Kharkiv City Clinical Hospital of Emergency Medical Aid in 1986 and Department of Advanced Level Surgery of Kharkiv Medical Institute, which optimized medical, scientific and pedagogical work of these institutions.

V.T. Zaitsev performed a great social work, he was the head of Kharkiv Scientific Society of Surgeons, the Deputy Chairman of Society of Surgeons of Ukraine, a member of the International Association of Surgeons, a member of the editorial board of the journals "Clinical Surgery" (Kyiv) and "Experimental and Clinical Medicine" (Kharkiv State Medical University) a member of the expert council of the SAC of Ukraine.

His multifaceted activities were repeatedly celebrated by government awards: in 1983 he was awarded the title of Honored Worker of Science and Technology of Ukraine, 1990 - the winner of the State Prize of Ukraine. He was awarded with two Orders of Labor Red Banner and a medal "For Valiant Labor".

In 1995 V.T. Zaitsev was elected a full member of the International Academy of Sciences of Ecology and Safety of Life.

Great experience in the educational and scientific fields, self-discipline and demand, persistence in achieving the goal, justice and a friendly, respectful attitude to people gave him the respect and immense authority among doctors, scientists, teachers, students and the public.

After the death of professor V.T. Zaitsev in 1999, the department of Advanced Level Surgery was headed by his student Valery Volodymyrovych Boyko (Photo 5), whose main position is Director of Institute of General and Emergency Surgery of the National Academy of Medical Sciences of Ukraine. Being a second year student, he became involved with surgery, attended student's scientific society at Department of Advanced Level Surgery, and graduating from Kharkiv Medical Institute in 1985, he devoted his life to the chosen specialty.

Having started his labor way as a practical surgeon in Kharkiv Research Institute of General and Emergency Surgery, V.V. Boyko defended his candidate's degree in 1990 under the supervision of professor V.T. Zaitsev, and in 1992 defended his doctoral dissertation, becoming at the age of 30 the youngest doctor of medical sciences in the country.

From 1990 V.V. Boyko worked at the Department of Advanced Level Surgery as assistant, then associate professor, and from 1996 - a professor at the department. From 1997 to 1999 he was the vice-rector for research of Kharkiv State Medical University, since November 1999 he has been the director of Kharkiv Research Institute of General and Emergency Surgery, and since January 2000 he has been Head of Department of Advanced Level Surgery of Kharkiv State Medical University.

V.V. Boyko is a highly educated surgeon, a prolific scientist, a creative and able-bodied person: he is the author of more than 2000 scientific works, of which 95 monographs on various surgical pathologies, 4 teaching aids (including for English-medium students), more than 250 teaching materials for students, professors and surgeons, about 600 patents of Ukraine for inventions. Among monographs are such significant works as "Purulent peritonitis. Pathophysiology and Treatment" (2002), "Polytrauma". Manual for doctors in 4 volumes (2010), "Quantum-biological theory" (2003 – a monumental work at the junction of biology and physics) and others.

Photo 5. **Professor V.V. Bovko**

V.V. Boyko is an experienced teacher and research supervisor. Continuing and developing the traditions of Kharkiv Surgical School of Academicians O.O. Shalimov and V.T. Zaitsev, he pays great attention to training of young medical, scientific and pedagogical staff. Under his supervision, 24 doctoral and 66 candidate dissertations, and more than 20 master's degree papers have been completed and defended.

Professor V.V. Boyko picked up the slack from professor V.T. Zaitsev in the leadership of the Scientific-Training and Production Association "Surgery", which fruitfully works to provide emergency and planned surgical care to the population of Kharkiv and the region, as well as training of medical, scientific and pedagogical personnel.

In 2002 on the initiative of V.V. Boyko a new educational and research association "Polytrauma", which included City Clinical Hospital of Urgent and Emergency Medical Aid, Department of Hospital Surgery of Kharkiv State Medical University, Institute of General and Emergency Surgery, Traumatology Department of Kharkiv Medical Academy of Postgraduate Education was created. This association has increased the efficiency and quality of emergency aid in this complex and extremely difficult category of patients and the quality of training of doctors specializing in the field of shock and polytrauma.

In 2005 V.V. Boyko became the laureate of the State Prize of Ukraine for the fundamental research on the impact of hyperthermia on the state of immunity and on development of new high-tech treatment technologies for purulent and purulent-septic diseases in cardiovascular and abdominal surgery.

Professor V.V. Boyko takes an active part in international and community activities: he has been elected to the European International Association of Surgeons, as well as Association of Injury Surgery and Intensive Care. Since 2000 he has headed Kharkiv Scientific Society of Surgeons, and since 2009 - the Association of Surgeons of Kharkiv and the region, a member of the editorial board of Ukrainian Journal of Clinical Surgery, journals "Experimental and Clinical Medicine", "Medicine Today and Tomorrow", the Deputy Editor-in-Chief of "Kharkiv Journal of Photobiology and Photomedicine", editor-in-chief of the journal "Kharkiv Surgical School", member of the Editorial Board of a new journal "Research Journal of Ministry of Health of Ukraine" (founded in 2013 in Kyiv).

V.V.Boyko was awarded with 6 medals (including foreign ones - America, England) and three orders.

In 2008, Professor V.V. Boyko was awarded the honorary title "Honored Worker of Science and Technology of Ukraine".

In 2012, V.V. Boyko was elected a member of the Academy of Sciences of Higher School, and in 2017 - a Corresponding Member of the National Academy of Medical Sciences.

Since 2001, the Department has been teaching English medium students.

In 2009 and 2010 credit-module system of teaching students was started.

Since 2015, teaching at the department is conducted on the principles of credit transfer system of training in accordance with the updated standard curriculum "Surgery" (2015).

The united creative team of the department is currently represented both by experienced teachers - veterans of the department, and talented youth. These are the head of the department Doctor of Medical Sciences, Corresponding Member of the National Academy of Medical Sciences of Ukraine, member of the National Academy of Higher Education, Professor Valery Boyko; 13 professors, doctors of medical sciences; 8 associate professors, candidates of medical sciences (in charge of the educational work is associate professor L.Y. Goncharenko); 26 assistants, of which 17 candidates of medical sciences and 9 - without a degree. 14 teachers are in charge of postgraduate training for interns and doctors, 19 lecturers are engaged in English medium instruction.

Almost all teachers participated in the work of the student's scientific society as students, took master's course, and then did postgraduate studies at their department and work on candidate theses under the supervision of professors V.T. Zaytsev and V.V. Boyko and will be worthy followers of the great educational mission of their teachers - professors Alexander A. Shalimov, Volodymyr T. Zaitsev, Valeriy V. Boyko, associate professors and assistants of the department in the third millennium.

Every year the department trains up to 15-20 young surgeons (Ukrainian and foreign), some of which, are preparing for the future pedagogical activities, and improve their surgical skills.

At present, the staff of the Department of Surgery No.1 of Kharkiv National Medical University, the department, rich in its traditions, world-renowned surgeons and brilliant educators,

continues active and fruitful work to improve and optimize the educational process for training of young medical personnel for Ukraine and many countries of the world, on development of Ukrainian surgery, working out modern surgical techniques and implementation of the latest advances in medical science in the practice of health care.

Surgical service organization in Ukraine

Organization of ambulatory care for surgical patients. The composition and structure of the surgical department in an outpatient hospital can be quite different and depend on the number of population served. In large city hospitals, the structure of the outpatient surgery department includes several offices of outpatient surgeons: a surgeon, an urologist, an oncologist, a traumatologist. The main tasks of these specialists are reception, examination of patients, diagnosis and treatment. In addition, these specialists administer the treatment to the patients discharged from the hospital, and are in charge of preventive work. Thus, the doctor of the outpatient surgery department provides qualified specialized aid to the population, is responsible for medical and preventive measures aimed at reduction of surgical morbidity.

A surgical office consists of the doctor's office, where the patients are received, a dressing room, and an operating room. In the dressing room and operating room there is everything necessary to serve outpatient surgical patients: dressing and operating tables, dressing stands, cabinets for medicines and solutions, sterilizers, and drums with sterile material.

The ambulatory surgical patients make a diverse group. The largest group includes the patients with occupational and off-the-job injuries (wounds, injuries), purulent diseases of the skin and subcutaneous tissue, surgical diseases (felons, soft tissue abscesses, hemorrhoids, hernia, etc.); followed up patients, the patients referred to for a preventive examination.

During the reception, the surgeon fills in various documents, among them a patient's card (registration form No. 25/-87), which is filled in with the main data about the patient's illness, laboratory examinations, administered treatment, diagnosis; statistical leaflet; follow-up check-list (form No.131/u-86).

A significant factor that distinguishes the work of a surgeon working on an outpatient basis from the work in a hospital is inability to return any time to the patient to check the data obtained or to identify additional clinical information. Therefore, the examination of the patient in an outpatient setting should be comprehensive, and the decision about the administered treatment should be final. This can be achieved within a short period of the patient's visit only if certain conditions are observed and certain methods of examination are used. Such condition is sufficient knowledge of the surgeon about the nature of various diseases, causes, clinical presentation, symptoms and signs, detection of which is possible in an outpatient settings. Considerable attention during the examination should be paid to the sequence of the examination by organs and systems. Passing to each subsequent diagnostic stage is only feasible after obtaining complete information at the previous step. Standard diagnostic protocols allow easier orientation among the possible individual characteristics of the disease in a particular patient.

It should be borne in mind that during the examination of the patient, reasonable doubts about the truth of the information received, related to the unconscious or conscious reluctance of the patient to correctly orient the doctor about his condition, may appear. In such cases, there are the following options in the patient's behavior: *simulation* - an attempt to describe the signs of the disease, which in reality do not exist; *aggravation* - more common syndrome associated with exaggeration of the existing symptoms; d*issimulation* - conscious (less often unconscious) shading or negation by the patient of his pathological subjective sensations.

The examination of an outpatient consists in questioning and identifying objective signs. The questioning should be as short as possible, targeted and relevant to those facts that may directly influence the diagnosis.

When examining a surgical patient, not only the diagnosis, but also the indications for the operation are determined. A number of symptom-complexes ("acute stomach", shock, coma, acute respiratory failure, etc.) by themselves can be indications for urgent measures and referral of the patient to the hospital. In such cases, it is important not to attempt to accurately establish the diagnosis

as the cause of a life-threatening condition, but to render the necessary first aid and transport the patient to a medical in-patient facility.

The process of the objective examination of a patient in an outpatient facility consists of questioning (complaints, disease history, previous history), assessment of the general condition and the study of pathological data using inspection, palpation, percussion, auscultation.

If necessary, to confirm the diagnosis, the necessary laboratory tests are made; the volume of them is quite large: blood count, urinalysis, blood glucose test, urea test, bilirubin test, coagulation test, bacteriological examination, histological examination, etc.

Outpatient facilities perform a fairly large volume of instrumental examination: fluoroscopy, radiography, various types of endoscopy (rectoscopy, cystoscopy, esophagoscopy and colonoscopy), ultrasound.

Treatment of surgical patients in an outpatient facility is carried out both surgically and conservatively. In his work, the surgeon detects diseases at the early stage, many of which can be treated conservatively on an outpatient basis, and if necessary, refers the patient to the hospital for planned surgical treatment.

In his work, an out-patient surgeon renders *surgical aid*. Ambulatory operative interventions can be both scheduled and urgent.

Urgent surgical interventions, often for acute purulent soft tissue diseases (abscesses, felons, etc.), cut and contused wounds are performed by the surgeon during reception hours.

To perform *scheduled outpatient operations*, each surgeon is assigned fixed hours and days. They are usually performed in the morning before the reception. These include removal of small benign soft tissues tumors, excision of bursa in patients with bursitis, removal of the ingrown nail, secondary suturing, etc.

Indications for hospitalization of patients can be urgent and scheduled.

The indications for urgent hospitalization are injuries, which cannot be treated on an outpatient basis; acute surgical infection requiring major surgical intervention or constant observation of the patient; acute diseases of the chest and abdominal organs; acute vascular diseases (acute thrombosis, embolism); diagnostic hospitalization (when observation of the patient in the outpatient facility does not allow to exclude acute illness), etc.

The indications for scheduled hospitalization are diseases requiring major surgical intervention; chronic diseases the ambulatory treatment of which is ineffective; diagnostic hospitalization requiring the use of special equipment or special conditions; small surgical intervention in persons with severe concomitant pathology, etc.

It is necessary to remember that in order to reduce the bed-day in the hospital, scheduled patients who are referred to a hospital for operative treatment should be maximally inspected in the outpatient facility.

Prevention is the main principle of health care. A very important part of the work of ambulatory surgeons is *preventive medical examination*, a method of active dynamic monitoring of the health of population groups in order to identify, register and treat the earliest forms of chronic surgical diseases. The patients subject to medical examination are selected by doctors during the visits of the patients and during preventive checkups.

The detected patients should be registered and constantly observed, undergo active treatment, which involves regular scheduled inpatient or outpatient treatment, including operative, when indicated.

Preventive examination is carried out according to nosological forms, which have high morbidity, lead to disability, mortality. According to the order of the Ministry of Health of Ukraine, surgeons perform preventive examination of patients with the following diseases:

1. Post-thrombotic syndrome (visiting a patient by a surgeon 2 times a year).
2. Varicose veins of the lower extremities (in case of refusal of the operation - one visit a year).
3. Condition after stomach resection (supervision for 5 years after the operation, 2 visits a year for 2 years, further – if indicated).

4. Chronic osteomyelitis (2 visit a year).

5. Atherosclerosis of arterial vessels of the lower extremities (2-4 visits a year); obliterating endarteritis (1 visit in 2 months); thrombangitis obliterans (monthly visits); Raynaud's syndrome (1 visit a quarter).

In addition to the above groups of the patients, the patients with the following diseases are also subject to *registration* when refusing from scheduled surgical treatment (as revised by the WHO):

1. Inguinal hernia (without obstruction).
2. Femoral hernia.
3. Umbilical hernia.
4. Hernia of the abdominal wall (middle line).
5. Diaphragmatic hernia.
6. Benign breast tumors.
7. Anal fissures.
8. Pararectal fistulas
9. Hemorrhoids.

It is necessary to remember that the followed-up patients have often to be transferred to disability group due to loss of working capacity.

Disability *can be temporary and constant*.

Temporary disability is considered to be the state of health, when the pathological disorders caused by the disease, are temporary, reversible. Temporary disability occurs in 60-80% of all diseases; of all days of treatment within the period of temporary disability more than 80% of patients undergo ambulatory treatment in outpatient facilities. The doctor, along with the diagnosis and treatment, must solve the question whether the patient can continue his work or if he needs to be discharged from employment for a certain period of time.

In this regard, temporary disability is divided into: a) complete; b) partial.

Complete temporary disability pertains to the cases when the patient cannot or should not work and needs a special regimen.

Partial temporary disability pertains to the cases when the patient cannot perform his professional work, but without harm to his health can do another easier job.

The legislation of our country provides for the following ***types of temporary disability***:

a) in case of disease associated with loss of ability to work;

b) at sanatorium-resort treatment;

c) in the case of a family member's disease, if there is a need for care for the patient;

d) at quarantine;

e) at position change due to an occupational disease;

e) at prosthetic care with referral to the hospital of the prosthetic and orthopedic enterprise.

In our country, examination of temporary disability is carried out in medical facilities in accordance with the provisions on the examination of temporary disability, approved by the Ministry of Health of Ukraine, *with issue of the sick leave certificate*.

Examination of temporary disability is carried out by a physician who, if necessary, discharges the patient from employment for a term of 3 days, no more than for 6 days. From the 7^{th} day of temporary disability, in order to extend the sick leave certificate the doctor must refer the patient to the head of department for consultation, and then, in case of prolongation of the disease, refer the patient to the *Medical Advisory Commission (MAC*).

MAC consists of the chief medical officer for disability examination, head of the department and the attending physician. In accordance with the "Regulation on the examination of temporary disability in medical institutions", MAC does the following:

1) solves complex and conflicting issues of examination of temporary disability;

2) refers patients with persistent disability and unfavorable prognosis for the Medical Labor Expert Commission (MLEC, now MSEC Medical Social Expert Commission);

3) gives a leave for sanatorium treatment, provided by the instruction on the procedure for issuing sick leave certificates;

4) transfers the able-bodied patients to easier job due to a disease;

5) issues a conclusion about the necessity to be transferred to another job that corresponds to the disability status for the patients with a stable disability who are not referred to MLEC;

6) issues sick leave certificates for 2-month periods at temporary position change of temporarily disables persons;

7) issues sick leave certificates for special treatment in another city.

The issues of *permanent or stable disability are addressed by Medical Social Expert Commission (MSEC)*, which is administered by the social welfare authorities (formerly the Medical-Labor Expert Commission (MLEC). The organization of MSEC work is regulated by the "Regulation on the medical-labor expert commissions".

The primary MSEC consists of 3 expert physicians: an internist, a surgeon, a neurologist, as well as a representative of the relevant social welfare department and a representative of the trade union organization.

MSEC deals with:

1) determining permanent or prolonged disability;

2) establishing disability groups;

3) determining the conditions and nature of work available to disabled persons which correspond their condition of health;

4) indicating the measures that promote restoration of health and working ability;

5) investigation of the causes of disability in the area of the commission activity.

Disability *is considered to be such a stable working ability disorder, which leads to the necessity to terminate professional work for a long period or requires a significant change in its conditions.*

Disability is a dynamic concept, because it is directly dependent on the state of health, which can improve or deteriorate.

When referring the patient to MSEC, "MSEC Referral" is issued (according to the order of the Ministry of Health No.183 dated 07.04.2004 "Instruction on determining disability groups" and Resolution of the Cabinet of Ministers of Ukraine No. 83 dated 22.02.1992 "On the approval of the Regulation on Medical Social expertise and the Regulations on Individual Program for Disabled Rehabilitation and Adaptation"). It is filled in by the attending physician with all information about the patient, his disease. When establishing the disease, MSEC is governed by the instruction on determining disability groups (see above). This instruction is based on the three-group classification of disability.

The reasons for establishing of *Group 1 disability* are disorders of the organism functions caused by chronic diseases or a severe anatomical defect, when patients cannot serve themselves and need constant help and supervision.

At *Group 2 disability* there is a complete permanent or prolonged disability as a result of significant organism dysfunctions, which, however, do not require constant care or supervision.

The reason for determining *Group 3 disability* is reduction of the capacity for work due to organism disorders caused by chronic diseases or anatomical defects, herewith the patients cannot work in a specialty, but can do easier work.

Structure and work of admission department of a surgical inpatient hospital

The hospital care begins at the admission department. The admission department is an important medical and diagnostic department intended for registration, reception, initial examination, anthropometry (Greek *anthropos* - a person, *metreo* - to measure), decontamination of the admitted patients and qualified (urgent) medical aid. The success of the further treatment of the patient (and his life in urgent situations) depends to a certain extent on the professional, prompt and organized action of the staff of this department. Every admitted patient should feel an attentive and friendly attitude in the admission department. Then he will trust in the institution where he will be treated.

The main task of the doctor working in the admission department of a surgical hospital is to confirm or establish the presence of surgical pathology, as well as to determine the degree of severity

of the patient's condition and solve the question of the form of the surgical care (emergency or scheduled surgery, reanimation, conservative treatment, etc.).

Thus, the main functions of the *admission department are as follows:*

1. Admission and registration of patients.
2. Medical examination of the patients.
3. Emergency medical care.
4. Determining the hospital department for the hospitalization.
5. Decontamination of the admitted patients.
6. Filling in the respective medical documentation.
7. Transportation of the patients.

The structure of the admission department of surgical inpatient hospital. The work of the admission department proceeds in the strict sequence:

1) registration of the patient;
2) medical examination;
3) decontamination.

The premises of the admission department are located in the same sequence.

The structure of the admission department of the surgical inpatient hospital includes, as a rule, the following offices:

1). *Waiting room.* There are patients who do not need bed rest, and persons accompanying the patients. There should be a table and a sufficient number of chairs. The information about the mode of operation of the offices, hours of meetings with the doctor, a list of foodstuff allowed to bring to the patient is posted on the walls.

2). *Registry.* The admitted patients are registered and the necessary documentation is issued there.

3). *Examination room* (one or more) is intended for medical examination of patients with the purpose of establishing a preliminary diagnosis and determining the type of decontamination, anthropometry, thermometry and, if necessary, other investigations, for example, electrocardiography (ECG).

4). *Sanitary inspection room* with a shower (bath), a room for changing clothes.

5). *Diagnostic room* for patients with an unidentified diagnosis.

6). *Isolation unit* for patients with a suspected infectious disease.

7). *Procedure room* for emergency aid.

8). *Operating (dressing) room* render emergency surgical care.

9). X-ray room.

10). Laboratory.

11). The office of the doctor-in-charge.

12). The office of the head of admission department.

13). Toilet.

14). Room for storing the clothes of the admitted patients.

In multidisciplinary hospitals the admission department can also have other offices, such as a traumatology, intensive care unit, cardiology (for patients with myocardial infarction), and others.

Admission and registration of the patients. The patients can be brought to the admission department in the following ways:

1) by ambulance: in case of accidents, injuries, acute diseases and aggravation of chronic diseases;

2) according to the referral of the district doctor in case of ineffectiveness of the ambulatory treatment, before the examination by the Medical Social Expert Commission (MSEC), as well as according to the referral of the military commissariat;

3) due to transfer from other health care institutions (in agreement with the administration);

4) "self-referral", a visit in case of deterioration of the patient's health in the street near the hospital.

Depending on the method of delivery to the hospital and the patient's condition, three types of admission are distinguished:

1) scheduled admission;

2) emergency admission;

3) admission at "self-referral".

If the patient is brought to the admission department in a state of moderate severity and, moreover, in severe condition, before the registration, the nurse is obliged to provide the first medical aid to the patient, to invite the physician and quickly do all medical administrations.

The doctor of the admission department inspects the patient and decides on the necessity of his admission to this medical institution. In case of admission, the nurse registers the patient and fills in the necessary medical documentation. After registering the patient, the nurse sends him to the examination room inspection for examination by the doctor and necessary diagnostic and medical procedures.

If the patient is delivered to the admission department from the street in unconscious condition without identification, after examining him by the doctor and providing emergency medical care, the nurse fills in the necessary documentation. After that, she is obliged to inform the police station and the accident office. She provides the descriptive information of the admitted patient (sex, approximate age, height, body build), describes his/her clothes. In all documents until the personality has been established, the patient should be listed as "unknown".

The nurse is obliged to give the phone call to the relatives and make the corresponding entry in the "Register of Telephone Calls" in the following cases:

1) the patient has been brought to the hospital due to a sudden illness that arose out of his house;

2) the patient died in the admission department.

Basic medical documentation of the admission department. 1. "Register of Admissions and Refusals from Hospita lization" (Form No. 001/u). The nurse records the surname, name, patronymic of the patient, year of birth, passport and insurance policy data, home address, place of work and position, telephone (home, office, close relatives), date and time of admission, where from an who by he was delivered, the type of hospitalization (scheduled, emergency, "self-referral"), the diagnosis of the referring institution, the diagnosis of the admission department, which department the patient has been sent to.

When the patient refuses to be hospitalized, the information about the reason and the aid provided shall be entered in the register: medical aid, referral to another hospital, absence of indications for hospitalization, etc.

2. "Medical inpatient record" (also referred to as "case report form", form No. 003/u). The nurse fills in the title page of the medical inpatient record as well as the demographic data section and the title page of the "Statistical card of the discharged from the hospital" (form No. 066/u). "The register of inspection for pediculosis" is filled when pediculosis has been revealed, in addition, the designation "P" (*pediculosis*) is made in the medical inpatient record.

3. Emergency message to the sanitary-epidemiological station (for referral to the sanitary-epidemiological station at the place of detection) is filled in the presence of an infectious disease, food poisoning, pediculosis in the patient.

4. "Register of telephone calls". The nurse writes in the text of the telephone call, the date, the time of the call, who answered the call.

5. Alphabetical journal of admitted patients (for information service).

Decontamination of patients. After the diagnosis has been made, the doctor-in-charge decides on decontamination of the patient. If the patient is severely ill, he is delivered to a resuscitation unit or an intensive care unit without decontamination.

Decontamination is carried out at the sanitary inspection room of the admission department. There are one-stage and two-stage methods of decontamination. In hospitals with a small number of beds, the patients are decontaminated a one-turn system is used, that is, women and men take turns. At a two-turn system, both men and women are decontaminated in different premises simultaneously.

The sanitary inspection room usually consists of an examination room, changing room, bathroom and shower room, and a room where the patients are dressed. Some of these rooms can be interconnected (for example, an examination room and dressing room).

In the examination room the patient is undressed, examined for pediculosis and prepared for decontamination. There is a couch, a table, chairs, a thermometer on the wall (the air temperature in the examination room should not be lower than 25° C).

If the linen is clean, it is put in a sack, and the outwear is hanged on a hanger and handed over to the room for storing the clothes. The list of things (receipt note) is issued in two copies: one is handed over to the storing room, the other is stuck to the medical record. The available articles of value and money are handed over to a senior nurse against a signed receipt for storage in a safe.

If the patient has an infectious disease, the linen is placed in a tank with bleach or chloramine B for 2 hours and sent to a special laundry. When the closes are infested with lice, they are pre-treated with disinfectant solution and sent to a fumigation chamber for special treatment. The bags with such clothes should bear the appropriate inscription "Pediculosis".

Stages of decontamination:

1. Inspection of the skin and hair of the patient.
2. Cutting hair, nails, shaving (if necessary).
3. Taking shower or hygienic bath.

Types of patient transportation to the hospital departments. *Transportation is moving or carrying the patients to the place of medical care and treatment.* The method of transporting a patient from the admission department to the respective hospital department is determined by the admitting physician. The means of transportation (wheelchairs, stretcher) are provided with sheets and blankets. The latter should be changed after each use. The patients who move independently, are accompanied by a junior nurse or orderly. The patients who cannot move are transported to the hospital department on a stretcher or on a wheelchair.

Hygiene and infection control of the admission department. Compliance with the hygiene and infection control in the admission department is an integral part of the hygiene and infection control of the hospital and provides the following measures:

1) obligatory decontamination of the admitted patients;

2) emergency information of the sanitary and epidemiological service (by telephone and filling in a special form) and provision of all necessary measures at detection of a patient with an infectious disease, food poisoning, pediculosis;

3) regular thorough wet cleaning of premises and surfaces of objects, application of different methods of disinfection (boiling, disinfectant solutions and ultraviolet radiation).

The structure of the operation unit. The operation unit is the cleanest, "holy" place of the surgical hospital. It is the operation unit that requires the most strict aseptic rules. Gone are the days when the operating room was in the department. Currently, the operation unit should always be located separately, and in some cases, it is constructed in special buildings connected by a passage to the main hospital complex.

At present, two basic variants of operation unit organization are used. The first variant, used for a long time, provides the presence of an operation unit in each surgical department. In this case, in order to prevent air pollution, the operation unit is located in a deadlock zone of the department or in a separate wing of the building. According to the second variant, the operation units of several surgical departments are united into one operation complex, for which a separate wing of the ground or underground floor is allocated or it is located in a specialized outbuilding that is connected with the hospital directly or with the help of a closed passage. In the latter case, the height and size of the premises in the operating room will not depend on the planning of the main building where the hospital is located. The second variant is optimal, because it provides a complete isolation of the operating rooms from the hospital.

All operation units are divided into *general* and *specialized* (traumatological, cardiosurgical, burn, neurosurgical, etc.).

Both general and specialized units include septic and aseptic divisions (operating rooms with auxiliary and service rooms). In this case, the auxiliary rooms of the operation unit are organized separately for an aseptic operating room and for a septic operating room.

When designing specialized operation units, there may be cases where the operation unit has only aseptic divisions (neurosurgery, cardiac surgery, etc.).

On the basis of the presence of one division (aseptic) or two divisions (aseptic and septic), the operation units are divided into *aseptic and combined*.

The number of operating rooms in the operation unit depends on the structure, size and specialization of the surgical departments, surgical activity, complexity and duration of operations, the length of the hospital stay of the operated patients, the number of operating days, etc.

The number of operating rooms in central district and inter-district hospitals in rural areas and in city district hospitals should be calculated as 1 operating room for every 30 surgical beds and 25 beds in emergency medical care hospitals. In specialized surgical departments, when calculating the number of operating rooms, it is necessary to take into account department specialization. The number of operating rooms in regional and clinical hospitals is determined in each specific case at the design stage.

The number of operating tables and types of operating rooms in a specialized operation unit depends on the type and capacity of the structural subdivisions of hospitals and are determined at the design stage. Optimally, operating rooms should be designed for 1 operating table.

The number of operating rooms in the septic division of the operation unit is determined depending on the local conditions (the number of beds with purulent pathology). In the operation units of general hospitals, the approximate ratio of septic and aseptic operating rooms should be 1:3, but not less than one septic operating room per an operation unit.

With the number of operating rooms over 6, it is recommended to provide a dispatching post.

The operation unit design is based on division into impassable aseptic and septic divisions, rational zoning of the internal premises and space in accordance with the functional division into the following zones:

1. Sterile zone including operating rooms.

2. *Zone of strict regime:*

- a group of premises for personnel preparation for an operation consisting of preoperative and dressing rooms for personnel for special and working clothes;
- a group of premises for patient preparation to the operation consisting of premises for the preparation of a patient for surgery or anesthesia room;
- a group of premises for placement of equipment intended for life support of the patients, consisting of premises for artificial circulation machine and hypothermia unit;
- a group of postoperative wards, consisting of the wards themselves and rooms (work-place) of the nurse-in-charge;
- a group of auxiliary premises, which includes a gateway at the entrance to the operating room (in the absence of anesthesia room).

3. *Limited regime zone:*

- a group of premises for diagnostic investigations;
- a group of premises for operation of instruments and equipment for the operation, including sterilization room, washing room, room for disinfection of anesthetic respiratory equipment;
- a group of staff rooms, including a surgeon's room, a protocol room, an anesthesiologist room, an anesthetist's room and a room for orderly;
- a group of auxiliary premises, including gateways at the entrance to the septic and aseptic divisions, a central control room for monitoring the patient's condition, plaster room, post-operative service rooms (washing and disinfection of bed pans, washing and drying waterproof bedlinen, etc.);
- a group of storage rooms, which includes a storage room for blood, a storage room for a mobile X-ray machine and a photolaboratory, a room for anesthesia and respiration equipment, a room for preparing disinfection solutions and storage of disinfection materials, a room for temporary storage of wheeled beds.

4. *General regime zone:*

- a group of premises for staff, which includes a department head office, a senior nurse's room, a shower for the staff;

- a group of storage rooms, which includes a room for cleaning items, a room for plaster storage.

The number of beds in the postoperative wards should be calculated as two beds per one operating room. In the presence of anesthesiology and resuscitation units, intensive care units, postoperative wards are not designed, and their number is determined by the number of beds in the department of anesthesiology and resuscitation.

In the absence of centralized air conditioning, provision should be made for premises for local air conditioners, the area of which is determined by the location of the equipment.

The most rational is placement of an operation unit in a separate medical-diagnostic outbuilding, which adjoins the building with wards or is connected with it by heated passages. It is possible to place an operation unit on the upper floor of the building with wards.

The admission department in a large hospital or emergency medical care hospital should be provided with an operating room for urgent operations.

To perform emergency operations in general hospitals, it is necessary to provide additional on-duty operating rooms.

Postoperative wards should be placed in a separate isolated part at the operation unit or at anesthesiology and resuscitation department, or separately in the surgical department.

The design of the premises of the operation units, their capacity depend on the type and number of beds of the hospital, the capacity and structure of its subdivisions.

Updated technologies in surgery

Modern methods of diagnosis and treatment in surgery

Technology development, its wide merger with medical science greatly influence improvement of methods of diagnosis and treatment in practical medicine and surgery, in particular. These methods allow shortening the terms of the examination, accurate establishing the diagnosis, administering a highly effective treatment with minimal invasion and often without a large "incision". Minimally invasive interventions have been used in all areas of surgery. This led to a maximum economic effect, reduction of the hospital stay, as well as pre- and post-operative period.

Among the new diagnostic and therapeutic technologies, ***videoendoscopy, X-ray vascular, ultrasound and computer tomography techniques are currently the most widely used.***

Videoendoscopy techniques

Videoendoscopic techniques are used to diagnose and perform surgical interventions.

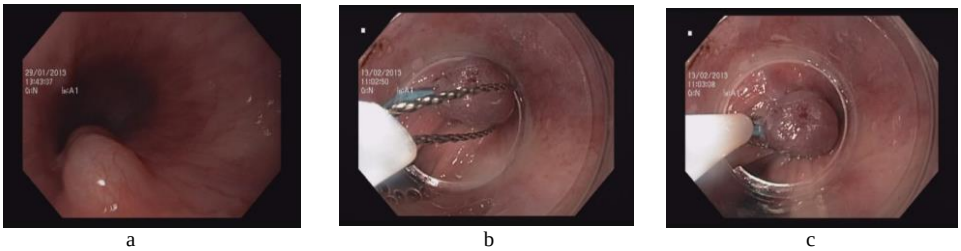
Such endoscopic techniques as video esophagogastroduedenoscopy, video bronchoscopy, video colonoscopy, sigmoidoscopy, video laparoscopy, video cholangioscopy, video thoracoscopy are used in surgery.

Video esophagogastroduedenoscopy *is a method to investigate the mucosa and lumen of the esophagus, stomach and duodenum with a flexible device fitted with a video camera - video esophagogastroduedenoscope.*

The indications for esophagogastroduodenoscopy include:

1) with diagnostic purpose - diagnosis of suspected foreign bodies, tumors, diverticula, strictures of the esophagus, stomach and duodenum, biopsy of tumors, in ulcer and its complications (to clarify the location and characteristics of ulcerative lesions), varicose veins of the esophagus, hiatal hernias, suspected tumor of pancreatic head, at choledocholithiasis, strictures of the bile ducts, retrograde administration of water-soluble contrast agents to the biliary tract (endoscopic retrograde cholepancreatography - ERCPG);

2) with therapeutic purpose - to remove foreign bodies, to treat cicatrical constriction of the esophagus with a bougie, to remove benign esophageal formations that cause its stenosis (Fig. 1), to apply hemostatics, to perform diathermocoagulation and clipping of the bleeding vessel, endoscopic ligation and sclerosing of the varicose veins of the esophagus and cardia in portal hypertension, to perform polypectomy, endoscopic papilosphyncterotomy (EPST).



a b c

Fig. 1. Esophagovideoscopy:
a - submucosal tumor up to 1.5 cm in the diameter in the middle-thoracic division of the esophagus;
b, c - endoscopic removal of the tumor (histologically - leiomyoma)

Video esophagogastroduedenoscopy plays a great role in treatment of gastroduodenal bleeding (Fig. 2) - to determine the degree of hemostasis and treatment strategy, to stop the bleeding. The endoscopic study is conducted in dynamics to evaluate the results using the classification of Forrest (1974), which distinguishes the following stages: 1 – acute hemorrhage; 2 - signs of recent hemorrhage; 3 - lesions without active bleeding.

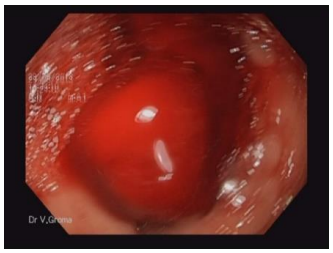


Fig. 2. Gastroscopy demonstrating gemorrhage from the

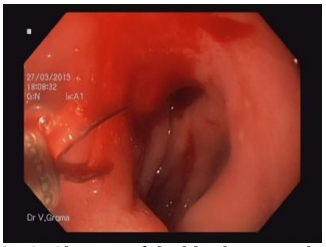


Fig. 3. Clipping of the bleeding vessel i in the ulcer

Local hemostasis "at the height of" bleeding with the use of irrigation of the bleeding focus with hemostatic solutions (aminocaproic acid, thrombin, hemofobin), film forming agents (sodium alginate, oxicellsole, lifusolel), diathermocoagulation, cryoelectrocoagulation or laser photocoagulation of the bleeding sites, clipping the bleeding vessels (Fig. 3) is successfully used during urgent endoscopic investigation.

Contraindications to fibrogastroscopy include decompensated cardiovascular diseases, aortic aneurysm, fresh burns of the mouth and esophagus, inflammation of the chest and mediastinum organs, stenosis of the pharynx and larynx, hemophilia.

Preparation of the patient includes the following: scheduled investigation is conducted in the morning on an empty stomach, urgent investigation is performed after washing the stomach. Anesthesia consists of both local anesthesia (spraying the throat with 10% solution of lidocaine for 5 minutes before the investigation), and general anesthesia. An easily excitable patient is prescribed sedation.

Complications of fibrogastroscopy include mucosal rupture and perforation of the esophagus; rupture of the stomach wall.

At esophagus rupture, urgent surgical intervention is indicated. It consists of drainage of the mediastinum, at large ruptures - suturing the rupture.

At gastric wall rupture, suturing of the rupture with treatment and drainage of the abdominal cavity is done.

Colonoscopy *is a method of investigating the cavity and mucous membrane of the colon with the help of a colonoscope.*

Colonoscopy is performed with the use of a fibrocolonoscope, which differs from the fibrogastroscope in length (200 cm) and diameter (13 mm).

Indications for colonoscopy:

- bleeding from the anus, when bleeding from the rectum is excluded;

- suspected tumor, polyps of the large intestine;
- suspected non-specific inflammatory processes (Fig. 4).

Contraindications:

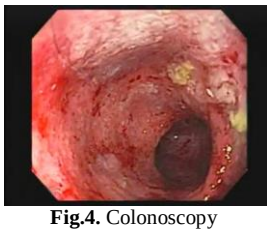


Fig.4. Colonoscopy demonstrating longitudinal ulcers in ulcerative colitis

- severe general condition of the patient;
- decompensated cardiovascular diseases;
- bronchial asthma;
- ascites;
- enterocolitis.

The patient's preparation consists in the following:

- preliminary thorough examination of the rectum (digital investigation, rectoscopy, irrigography);
- easily digestible diet, fasting the day before the investigation;
- administration of laxatives (fortrans, endofalk);
- cleansing enema (1-2 hours before the investigation);
- introduction of 2% promedol solution, 1.0 ml 15 - 30 minutes before the study.

Complications include perforation of the intestinal wall, which occurs when the colonoscope is manipulated carelessly in the zone of physiological narrowing, pathologically altered tissues, with excessive filling of the intestines with air.

Endoscopic retrograde cholangiopancreatography (ERCP) is performed in patients with suspected choledocholithiasis (Fig. 5), common bile duct stricture to clarify the diagnosis (at jaundice, when the signs of pathology are detected with ultrasound).

The clinical presentation of *choledocholithiasis* is characterized by a complex of symptoms and signs: severe pain in the right hypochondrium with characteristic radiation to the right shoulder blade, right supraclavicular area, right shoulder; fever; jaundice; itch; dark urine and discolored feces.

Charcot's triad has been described: pain in the upper middle or right abdomen, chills, and fever, which indicates cholangitis.

Ultrasonography shows the presence of small stones in the gallbladder, increased width of the common bile duct and the presence of stones in it.

Complications of ERCPG include acute pancreatitis, septic cholangitis, perforation of the duodenal wall or common bile duct.

Endoscopic papilosphyncterotomy (EPST) has proven to be a highly effective method of diagnosis and treatment for choledocholithiasis (Fig. 6).

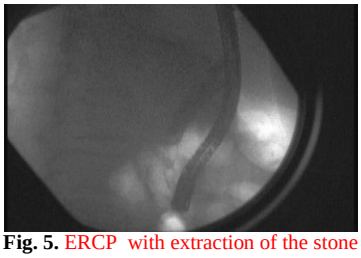


Fig. 5. ERCP with extraction of the stone from the common bile duct

Fig. 6. EPST with stone removal from the common bile duct

Indications for EPST:

1) patients with high operational risk, followed by laparoscopic cholecystectomy;

2) patients with cholecystocholedocholithiasis, cholangitis, stenotic papillitis at possibility of complete correction of disturbance of outflow into the duodenum;

3) patients with post-cholecystectomy syndrome for correction of patency and treatment of the biliary tract, if it is the only and final method.

Contraindications:

1) severe disorders of blood coagulation;

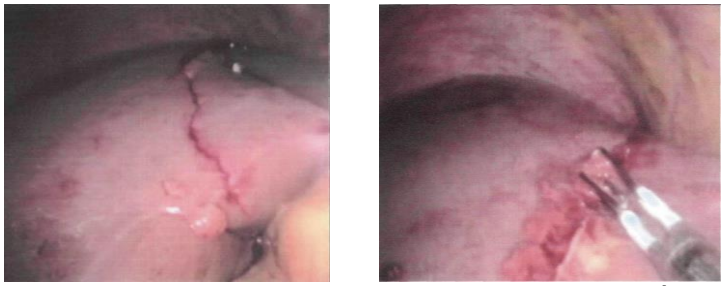
2) acute non-biliary pancreatitis;

3) tubular stenosis of the common bile duct exceeding 2.5 cm;
4) large (more than 2 cm) stones in the common bile duct;
5) accompanying lesions of the proximal hepatocholedoch division;
6) papilla location in duodenum diverticulum;
7) chronic duodenostasis.

When preparing the patient for EPST, intramuscular injection of 2 ml of 1% promedol solution and 1-2 ml of a 0.1% solution of atropine is required.

For adequate correction of choledocholithiasis, the length of the sphincter incision should be 1-2 cm. For stone extraction, cylinder catheters, a Dormia's basket, Dormia's basket probe are used. EPST is finished with control cholangiography.

Laparoscopy *is a method to perform examination, diagnostic manipulations and therapeutic interventions in the abdominal cavity with the help of videolaparoscopic technique* (Fig. 7).



a b

Fig. 7. Laparoscopy:
a - rupture of the right liver lobe; b - coagulation of the liver rupture site

Absolute indications for emergency laparoscopy are:

1. Inability to exclude the presence of acute surgical disease of the abdominal cavity requiring emergency surgical treatment.
2. Acute closed abdominal trauma and suspected intraabdominal bleeding or rupture of a cavitary organ.
3. To perform the minimum volume of surgical intervention in patients with subcompensation of basic life support systems.
4. To determine the organ viability in conditions after strangulated hernia reduction and at mesenteric thrombosis as well as effectiveness of restoring mesenteric circulation by thrombolytics or by vascular operations. In these situations, a "dynamic" (repeated) laparoscopy is applied after 6-12-24 hours.

In scheduled surgery the laparoscopic surgeon faces more complex tasks.

Indications for scheduled laparoscopy.

1. Ascites of unclear origin.
2. Differential diagnosis of tumor and cirrhotic lesion of the liver.
3. Presence of the liver cysts with the purpose of laparoscopic surgical treatment.
4. Differential diagnosis of liver diseases with a characteristic pathoanatomical picture: polycystic disease, sarcoidosis, cirrhosis, hemochromatosis, acute fatty dystrophy...
5. Splenomegaly of unknown origin.
6. Suspected carcinomatosis of the abdominal cavity.
7. Differential diagnosis of jaundice in case of impossibility to perform ERCP.

Contraindications for laparoscopy can be relative and absolute, local and general.

General absolute contraindications include:

1. Agony of the patient.
2. Acute myocardial infarction.
3. Acute cerebral circulation disorder.

4. Functional decompensation of the cardiorespiratory system due to chronic heart and lung diseases.

Relative general contraindications include:

1. Chronic diseases of the heart and lungs with functional subcompensation.
2. Heart arrhythmias.
3. Bronchial asthma.
4. Blood coagulation disorders (hypocoagulation).
5. Acute thrombosis of the pelvic veins and veins of the lower extremities.

Absolute local contraindication is the presence of multiple scars on the anterior abdominal wall resulting from previous operations for peritonitis, intestinal fistulas, and eventration.

Relative local contraindications can be:

1. Hernias of the hiatus or anterior abdominal wall.
2. The history of operations on the abdominal organs.
3. Previous radiotherapy to the abdominal cavity.
4. Pronounced intestinal pneumatosis.

Preparation of the patient for the manipulation consists in the following: fasting the day before laparoscopy or light dinner, cleansing enema. Before the study, 0.5-1 ml of a 0.1% atropine solution, 2 ml of 2% promedol solution are injected.

To perform laparoscopy and intra-abdominal manipulations, the following instruments are necessary: trocar for introduction of gas into the abdominal cavity (pneumoperitoneum), trocars for intra-abdominal manipulators and maintenance of pneumoperitoneum, optical system for examination of the abdominal cavity, means of illumination, a device for video recording, a device for bloodless separation of the tissues, a device for maintaining the required intra-abdominal pressure, a device for washing the abdominal cavity and evacuation of the liquid contents.

Laparoscopy technique. The investigation is tolerated similarly to gastroscopy. The place of Veress' needle and the first trocar introduction is determined by topographic and anatomical data of the anterior abdominal wall. At present, the point in the middle line is often used, 1-2 cm below or above the navel. Abdominal cavity inspection starts with the place of Veress' needle and a trocar insertion for the possibility of intra-abdominal damage. Then laparoscopy is started. In case of intra-abdominal manipulation or surgery, up to four troachars can be introduced under the visual control.

Complications of laparoscopy:

- damage to the vessels of the anterior abdominal wall;
- puncture of the small or large intestine;
- bleeding into the abdominal cavity after liver biopsy.

Laparoscopic cholecystectomy (LCE). Laparoscopic methods are now widely used in cholelithiasis treatment.

Given the prevalence of this surgical intervention, we give a more detailed description of it.

LCE consists of several stages: operative approach (introduction of the instruments to the abdominal cavity and pneumoperitoneum), identification of the duct and artery, separation of the gallbladder with its extraction from the abdominal cavity and closure of the surgical wounds.

The most commonly used laparoscopic approach for gallbladder removal is Reddick-Olsen four-point technique.

LCE technique. The first stage is mobilization of the gallbladder. In the presence of adhesions around the gallbladder, they are separated by coagulation, using a sail-shaped stretching technique. It is necessary to make the dissection as close as possible to the gallbladder wall, without damaging the fat tissue, since cutting the omentum with a coagulator does not always result in a good hemostasis. Puncture emptying of the gallbladder should be carried out in all cases of its overfilling, tension, acute inflammation and empyema. The puncture is performed with a thick needle through a point on the abdominal wall, which corresponds to the projection of the gallbladder bottom. The puncture of the gallbladder wall is better to perform not perpendicularly, but along the tangent line, which helps to lengthen the puncture canal in the wall and to reduce the leakage of the residual content through it. The intrahepatic location of the gallbladder, in any relation to its bottom and the edge of the liver, results in

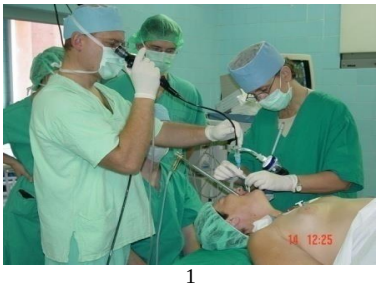
complexity of the gallbladder separation from the bed. Intrahepatic bile ducts and vessels are often located very close to the remaining large pit in the liver parenchyma, which can lead to their injury.

The next LCE stage is to position the gallbladder to provide a convenient approach for manipulations in Calot's triangle.

After providing optimal exposure to the region of the gallbladder cervix, cholecystectomy begins.

After providing such an exposure, the next step of the operation is to cut the peritoneum on the neck of the gallbladder, leaving 1-1.5 cm from its edge. If revision does not reveal any alarming signs of damage, you can proceed to the next LCE stage - excretion of the gallbladder body from the liver bed.

The next stage is extraction of the gallbladder from the abdominal cavity and suturing of the trocar wounds. The extraction can be carried out through an extended opening in the abdominal wall of the umbilical port, epigastric port or through a specially prepared contraperture in the right hypochondrium.



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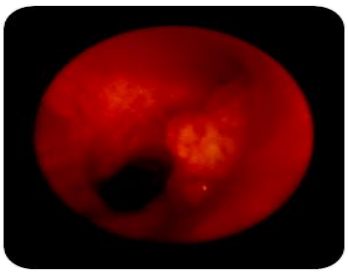


2

Fig. 8. Bronchoscopic examination:
1- bronchoscopy; 2 - tumor the right main bronchus (endophotograph)

The final stage of the laparoscopic operation involves treatment of the abdominal cavity and closure of the trocar wounds. The gas is removed from the abdominal cavity, the ports are pulled out.

Complications of LCE: leakage of bile along the drainage, postoperative pancreatitis, bleeding from the drainage. To diagnose and eliminate the complications, relaparoscopy or an open operation can be performed. The combination of endoscopic methods with laparoscopy has allowed achievement of good results.

At suspected choledocholithiasis, two-stage treatment is performed. Initially, endoscopic diagnosis and correction of the hepatocholedoch pathology are performed by EPST with extraction of the stones followed by laparoscopic cholecystectomy, 3-5 days or 1-2 weeks later. In choledocholithiasis, which cannot be cured by endoscopic transpapillar interventions (large stones), laparoscopic cholecystectomy with choledocholithotomy and external drainage of the common bile duct are performed.

Bronchoscopy *is a method of studying the lumen and mucous membrane of the bronchi (main and lobar) and the trachea using a bronchoscope with a video camera (Figure 8) in respiratory diseases, which allows performing a number of diagnostic and therapeutic manipulations. Flexible bronchoscopes are used.*

Indications for bronchoscopy:

1) examination of the bronchi and trachea to clarify the cause and location of the disease;
2) lavage and scraping of the mucosa for cytological test;
3) biopsy;
4) bronchography (method of bronchiectasis diagnosis);
5) aspiration from the bronchial tree;
6) medication administration (antibiotics, proteolytic enzymes, etc.);
7) coagulation of polyps in the trachea and bronchi;
8) diagnosis and removal of foreign bodies.

Contraindications include decompensated diseases of the cardiovascular system; cervical spine injuries; aortic aneurysm; acute diseases of the tonsils, pharynx and larynx.

Preparation of the patient:

1) bronchoscopy is performed on an empty stomach;
2) administration of sedatives;
3) administration of 0.5 ml of atropine or metacin solution;
4) spraying the throat and bronchial lumen with 10% lidocaine solution 5 minutes before the investigation and during the bronchoscope introduction.

Complications arise with negligent manipulation of the bronchoscope, especially in pathologically altered bronchi and during anesthesia:

1) edema of the infraglottic space;
2) vocal cord injury;
3) injury to the back of the pharynx and bronchus;
4) purulent inflammation of the tracheobronchial tree.

Thoracovideoscopy is a direct examination of the pleural cavity using endoscopic techniques (Fig. 9).

As a diagnostic method it was first used by a Swedish internist Jacobæus in 1910 to diagnose the condition of the pleural cavity in patients with tuberculosis. Later, it was most frequently used in phthisiology to incise the adhesions aftert formation of therapeutic pneumothorax.

Introduction of effective anti-TB drugs in the 1950's decreased the interest in this method. Only a small number of hospital continued to use the method of thoracoscopy, mainly as a diagnostic tool for pleural pathology, chest injury and inflammatory diseases of the pleura and lungs.

Only in the early 90's, development of video technologies led to the revolution in this method of diagnosis and treatment. Since then, not only the surgeon can observe what is happening in the pleural cavity, but also all his assistants, which allows not only to conduct a diagnostic examination, but also to perform a complete surgical intervention.

Videothoracoscopic surgery has been developing rapidly at present, and only now the appropriateness of various interventions is analyzed properly, since the ability to perform thoracoscopic surgery does not mean a final, successful outcome.

Indications for videothoracoscopic operations.

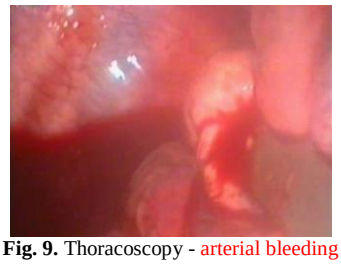


Fig. 9. Thoracoscopy - arterial bleeding from the lung rupture

Diagnostic indications:

1) pleural effusion of unknown origin;
2) nodule formation in the lungs of unknown origin;
3) interstitial lung disease;
4) clarification of the histological type of the tumor in the presence of a tumor process;
5) evaluation of the tumor process spread;
6) staging the tumor process spread to the lymph nodes;
7) evaluation of intrathoracic infectious process.

Therapeutic indications:

1) spontaneous pneumothorax;
2) bullous disease;
3) pneumoreduction;
4) prolonged absence of parenchymal balloon;
5) benign tumor of the lung;
6) lung resection at a metastatic tumor (if indicated);
7) lung resection at a primary tumor (if indicated);
8) removal of mediastinal cysts;
9) removal of mediastinal tumors (if indicated);
10) esophagomyotomy;
11) drainage of the pericardium at pericardial effusion;

12) resection of the esophagus in cancer (if indicated);
13) removal of benign tumors of the esophagus (leiomyoma);
14) thymectomy at myasthenia;
15) ligation of the chest lymphatic duct at chylothorax;
16) sympathectomy and splanchnicectomy;
17) drainage of the pleural cavity at fragmented effusion;
18) drainage and decortication at early empyema;
19) pleurodesis.

Contraindications for videothoracoscopic operations include:
1) excessive obliteration of the pleural cavity;
2) impossibility to carry out one-lung ventilation;
3) involvement of the hiatal structures;
4) pre-operative chemo- and chemo-radiation therapy;
5) pronounced coagulopathy.

Many operations that deal with general thoracic surgery can now be performed using the method of video support or videothoracoscopy. In the western literature, the term VATS (video-assisted thoracic surgery) is widely used. VATS is applied to the operations on the lungs, mediastinum, pleura and esophagus for treatment of cancer, pneumothorax, purulent-inflammatory diseases, cysts, esophagus patency disorders.

X-ray endovascular surgery

Endovascular surgery or interventional radiology *(initiated by Dotter, 1964) is a new branch of medicine that uses minimally invasive intravascular catheter technologies (that is, without cuts and blood loss) in the diagnosis and treatment of a wide range of vascular diseases.*

It is performed through a puncture of the vessel on the thigh or in the axillary region (the puncture diameter is only a few millimeters). All surgical interventions are carried out with sparing methods through the vessels and inside the vessels and are aimed at reducing or restoring (angioplasty) the blood flow. In order to see the result of surgical intervention inside the vessel, special X-ray equipment is used, angiograph, and all the actions of the surgeon are performed under the control of the image on the screen of the X-ray apparatus or television.

It is used both for diagnosis and treatment of many diseases, such as:
1) stenotic atherosclerosis of the aortoiliac segment and arteries of the lower extremities;
2) stenotic atherosclerosis of brachiocephalic arteries;
3) acute thrombosis of the veins of the lower extremities and post-thrombotic disease;
4) cerebrovascular accident (stroke);
5) coronary artery disease (angina pectoris, myocardial infarction, ciliary arrhythmia, etc.);
6) vasorenal hypertension;
7) chronic hepatitis and liver cirrhosis;
8) chronic pancreatitis;
9) chronic disturbance of the intestinal circulation (abdominal ischemic syndrome);
10) fibromyomas of the uterus;
11) varicose veins of the testicle (varicocele);

Indications for the use of endovascular surgery:

1. Angiography of all vascular basins – investigation of the arteries and veins of the human body introducing contrast substance through a thin catheter. It allows detection of many diseases associated with vascular changes (for example, atherosclerosis, liver cirrhosis, tumors), as well as the source of internal bleeding.

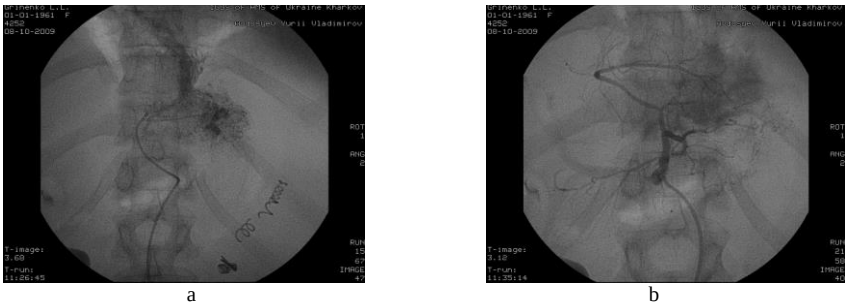
2. Embolization of vessels in various diseases and injuries to decrease blood flow (at internal bleeding, injuries of parenchymal organs, liver, spleen, uterus, stomach ulcer, lung), by introducing special particles, emboli, through the catheter (Fig. 10).

3. Prevention of pulmonary artery thromboembolism (PATE) at venous thrombosis, varicose disease, in which thrombi appear in the lumen of the veins of the lower extremities. At the risk of pulmonary embolism, a special device, cava filter, in installed in the inferior vena cava through a

catheter. It catches the thrombi at the level of the vena cava, preventing them from migrating to the heart and pulmonary artery.

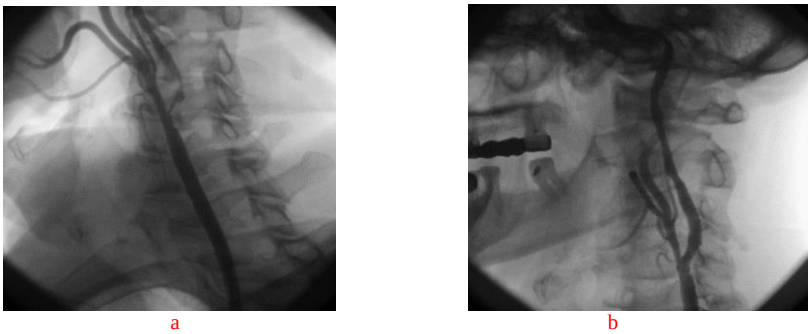
4. Regional infusion therapy. Many drugs (for example, antitumor, thrombolytics) are more effective when administered directly in the diseased organ.

5. Balloon angioplasty with stenting - increasing artery lumen of the narrowed place with establishment of stent to prevent restenosis.



a b

Fig. 10. Arteriography of the esophagus and cardia of the stomach:
a - bleeding (extravasation of the contrast substance, b - absence of extravasation after the X-ray occlusion of the bleeding vessel



a b

Fig. 11. Arteriogram of the left common carotid artery:
a - its stenosis; b – the left common carotid artery after balloon dilation and stenting

Endovascular treatment - transluminal balloon angioplasty (TLBAP) and stenting (Fig. 11) consists in introduction into the narrowed section of the artery of a special balloon catheter, by means of which the plaque is crushed and vessel lumen is dilated. To prevent restenosis in the dilated area, a special device, stent, which "reinforces" the artery inside and prevents its collapse and growth of the crushed plaque and restenosis, is introduced.

Advantages of endovascular surgery:

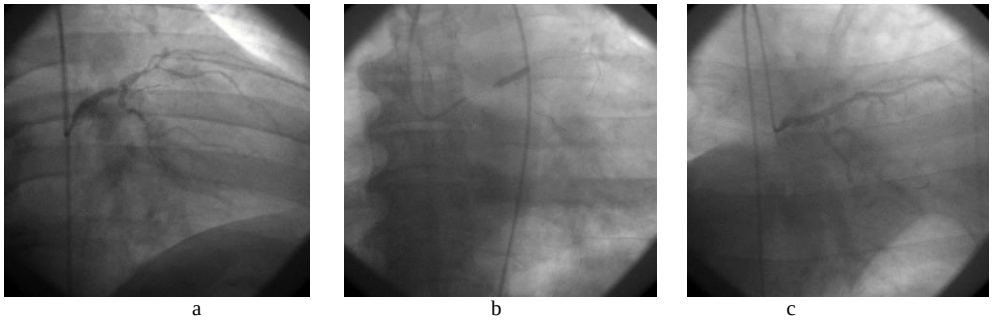
1) Endovascular diagnostic and therapeutic interventions may be performed on an outpatient basis or require only a few days of hospitalization;

2) general anesthesia in most cases is not needed;

3) surgical risk, pain syndrome, recovery time are reduced when compared with traditional surgery;

4) endovascular interventions in most cases are less expensive than traditional surgical procedures.

If the lumen of the vessel is fully closed, ***rotational atherectomy*** is used. It consists in removal of the atherosclerotic plaque using a rotablator. This method uses a special bur, covered with diamond



a b c

Fig. 12. X-ray endovascular balloon angioplasty with stenting:
a - coronary angiography of the left coronary artery (LCA);
b - balloon dilatation of the anterior interventricular branch of LCA; c – final result

crystals, by which the atherosclerotic plaque is destroyed and the lumen of the vessel is restored. The operation is successful in 50-65% of cases.

With complete obstruction of the lumen, the method of ***intravascular extraction atherectomy*** is also used. It uses a special catheter with two systems - cutting and aspiration. The latter is connected to a vacuum device that allows removal of the incised fragments of the atherosclerotic plaque.

In addition to the above techniques, endovascular surgery uses various laser technologies. These are destruction of an atherosclerotic plaque with the use of a pulsating garnet laser, a combination of balloon angioplasty with simultaneous exposure to laser radiation on the vessel wall.

Endovascular methods have been widely used in cardiac and vascular surgery at coronary artery disease, vasorenal hypertension, cerebral insufficiency, atherosclerotic occlusive lesions of the arteries of the extremities or compression of the arteries from the outside.

Percutaneous intravascular angioplasty is especially widely used in the treatment of *coronary artery disease*. The above-mentioned types of intravascular angioplasty are also used: balloon dilatation (expansion) with stenting, rotablation (rotational atherectomy), laser destruction of the atherosclerotic plaque.

Coronography. Planning cardiac operations is impossible without coronary angiography findings. This is the study of cardiac vessels with contrast substances. Coronary angiography indicates the exact location of the ***atherosclerotic plaque*** (Figure 12a) and helps to determine the tactics of surgical intervention.

Today, the most commonly used is ***X-ray endovascular balloon angioplasty with stenting coronary arteries*** (Figure 12, b, c).

Indications for endovascular angioplasty at coronary artery disease.

1. Stenosing lesion of one or two vessels, provided that the other arteries of the heart are in good condition.
2. In case of angina which is poorly treated with medical treatment.
3. If the signs of ischemia (blood supply insufficiency) of the heart are proven by heart investigations.
4. In patients with angina and a high risk of major surgical intervention.
5. Repeated angina after myocardial infarction.

Balloon angioplasty is *contraindicated* if the patient has multiple arterial lesions or the atherosclerotic plaque is small and takes less than 50% of the lumen of the vessel, as well, if in the presence of pain in the heart, insufficient blood supply to the heart muscle is not been proven by instrumental methods.

Operation technique. To perform balloon angioplasty, special cylinder catheters 1.5 - 4 mm in the diameter, with a pressure of up to 15 atmospheres, are used. The operation is carried out through the femoral artery. The balloon is filled with the contrast substance so that the position of the balloon

and the result of its application can be seen with X-ray control. The catheter with the balloon is introduced to the heart through the femoral artery under X-ray control. It is introduced into the desired artery, and is inflated in the place of the vessel narrowing. The time for the balloon inflation is from 15 seconds to 2 minutes. With this, the plaque on the vessel wall is compressed and flattened, which leads to the lumen dilation. The electrocardiogram is constantly monitored, thus when the balloon inflates, there is no insufficiency of blood supply to the muscle supplied by this vessel. The balloon can be inflated several times until the lumen of the vessel increases by 70%. If there are several such vessels, a vessel with a greater degree of narrowing is selected.

Before balloon angioplasty it is necessary to prepare the patient. To prevent possible thromboembolic complications, 2-3 weeks before the surgery, the patient is prescribed drugs that reduce thrombocytopoiesis (aspirin, ticlid, clopidogrel). During the operation, heparin is used for 18-48 hours. From the second day after the operation, aspirin is administered at a dose of 325 mg per day, as well as calcium antagonists and β-blockers.

Complications of balloon angioplasty include rupture of the inner wall of the artery with thrombus formation, tearing off the parts of the inner membrane and blocking with them of the distal arteries. In this case, a stent (a special metal net frame) should be placed or intracoronary thrombolysis (thrombus dissolution) is conducted. Sometimes spasm of the artery or its residual narrowing is possible. This complication is eliminated by medication or by repeated balloon angioplasty with the use of a larger balloon.

Ultrasound investigation methods (ultrasonography)

These are based on reflection by the transducers of ultrasonic oscillations from the investigated medium (organs) and their transformation into echograms. For this purpose, transducers with different frequency are used. The method is used to diagnose many diseases in surgery.

In abdominal surgery, ultrasound techniques are the most simple, affordable, and have high resolution. They are widely used in diseases of the liver and bile ducts, spleen, pancreas, etc., allowing to diagnose the disorders of the shape, structure of organs, the state of the blood flow in the organs and major vessels, the presence of free fluid in the abdominal cavity (hemoperitoneum, ascites, peritonitis, cysts, tumors, cirrhosis of the liver, stones in the gallbladder and common bile duct, and others (Fig. 13).

Ultrasonography is effective in both scheduled and urgent surgery. In polytrauma it is used to specify the nature of the injury to the parenchymal organs (spleen, kidney, liver), to determine the treatment tactics. Ultrasound can determine disorders in the organ integrity, i.e. a defect in its outlines (rupture of the capsule) and parenchyma, which looks like hypo- and anechoic zones. Detection of subcapsular hematomas looking like anechoic crescent-shaped formations (in case of trauma of the spleen), and their dynamic investigation with the purpose of controlling the growth of the hematoma or an already formed rupture of the capsule is possible. ultrasonography allows to determine the size of the organ, to visualize the vessels. Ultrasound can reveal free fluid in the abdominal cavity looking like anechoic zones in different parts of the abdominal cavity.

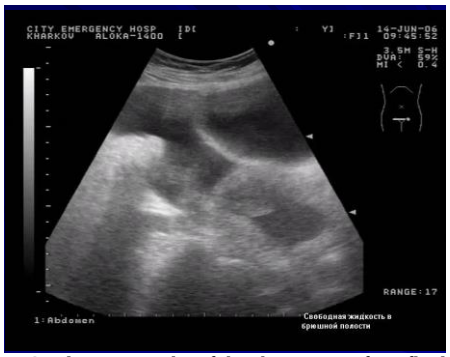


Fig. 13. Ultrasonography of the chest cavity: free fluid in the abdominal cavity. Large anechoic zone in the uterorectal, vesicouterine and interloop spaces

Findings interpretation. ***Pancreatic injuries*** can occur in the form of concussion (without violating the integrity of the organ) or with partial or complete rupture. At rupture, there is a disorder of the contour, shape and structure with the signs of acute pancreatitis. At concussion, expressed edematous infiltrative manifestations in the form of increased size, zones of diffuse echogenicity increase are seen.

Of great importance is ultrasound diagnosis in the ***diseases of the heart and blood vessels***. Echography of vascular hemodynamic

parameters is based on Doppler effect. Doppler ultrasonography is based on qualitative and quantitative evaluation of Doppler spectrum in the frequency shift.

At atherosclerosis of the major limb arteries, plaques up to 1 mm can be detected. It is possible to determine their structure, degree of lumen stenosis, type and velocity of the blood flow (Fig. 14).

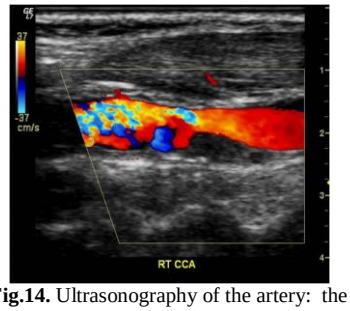


Fig.14. Ultrasonography of the artery: the lumen is narrowed due to an "unstable" atherosclerotic plaque

The study of the veins of the lower extremities records the blood flow in horizontal and vertical positions with the use of functional tests. Normally, the lumen of large veins is anechoic, and when the blood flow is decelerated, it is hypoechoic. The signs of pathology of the veins of the lower extremities are thickening of the wall **over 3 mm, absence of the diameter change at tension** and respiration, increased lumen at occlusion, visualization of the thrombus, changes in the valve (thickening, decreased mobility). Color Doppler mode demonstrates absence of blood flow at functional tests, absence of phase, blood flow deceleration at distal compression, reflux at proximal compression.

Ultrasound signs of ***thyroid pathology*** in terms of echography can be divided into diffuse (hyperplasia, thyroiditis, diffuse toxic goiter) and focal (cysts, nodular goiter, adenomas, cancer). Diffuse thyroid hyperplasia is characterized by increased volume, isoechoic structure, homogeneous fine-medium-grained structure, increase in the number of parenchymal vessels in color Doppler mode. Ultrasound signs of thyroiditis include thyroid enlargement, diffuse echogenicity reduction, appearance of hypoechoic regions of various shapes and sizes. Nodular goiter is characterized by the presence of round hyperechoic areas, the presence of a hypoechoic rim. Follicular adenomas are visualized as iso-, hyper- or hypoehoic formations. Most adenomas have a thick hypoechoic rim corresponding to the histological capsule, swelling of the parenchyma or capsule vessels.

In patients with heart diseases, echocardiography allows to identify the heart valves and their activities; to recognize the condition of the heart walls; to evaluate the valves movement; to measure the thickness of the walls, the dimensions of the chambers; to conduct Doppler echocardiography to reveal valve regurgitation, narrowing on the way of the blood flow.

Methods of computed tomography

Computed tomography (CT) *is a method for obtaining high-precision information with the use of computer systems. The founders of CT are a mathematician Cormack, who theoretically substantiated the possibility of obtaining information and constructing CT images, and an engineer Hounsfield, who implemented the idea in practice.*

Currently, there are four generations of computed tomographs. In the recent years, spiral computer tomography and electron beam CT have been used. The information obtained by CT is absolutely objective and is measured in units of computed tomography (Hounsfield unit).

In surgery, diagnosis of diseases of the lungs, abdominal organs is the most common area of CT use.

When investigating the organs of the chest, it is possible to differentiate the structures of the mediastinum and the roots of the lungs, to study subtle deviations in the structure of the "air" lung tissue (to detect bullets, tender shadows of fibrosis, etc.).

In diseases of the abdominal cavity, the internal structure of the parenchymal organs, such as the liver, pancreas, spleen, kidneys, etc., has become a "revelation" at intravital investigation of the patient because is not visualized at diagnostic laparotomy.

CT is based on the ability of various organs and tissues (both healthy and pathologically altered) to absorb X-rays. In turn, attenuation of X-rays are fixed by special sensors and can be represented as graphic "pictures".

The capabilities of invasive interventions under CT control have not been fully investigated, the technique is of particular value at operations on the pelvic bones and organs, on the spine, etc.

With the advent of spiral CT with a scan interval of 1 second, computed tomography with bolus contrast enhancement can be considered a method of visualization of blood vessels, including arterial blood vessels. The diagnostic information from transverse sections of the patient's body can be presented like multi-dimensional or voluminous (three-dimensional) reconstructions, which allow to assess visually the complexity of anatomical relationships.

Fig.15. A device for gas-jet dissection

Particularly effective for three-dimensional reconstruction are workstation programs allowing semiautomatic "grinding" of transverse sections, removing structures with similar density, but which are not consciously related to the object being investigated, such as Easy Vision workstation, Philips.

Computer technologies have advanced so far that now the subject of the diagnostic process is the so-called "virtual" endoscopy, in which one can move inside a reconstructed object with an exact coordinate fixation on transverse sections, which is especially important when investigating the portions of the intestine, bronchi, ducts, which are located behind a pathological narrowing, passage through which with a real endoscope is impossible, and only a "virtual" one can do it.

Directions of clinical use of CT are constantly increasing. For example, many scientists consider "low-dose" CT as an alternative to fluoroscopy in the diagnosis of lung diseases, etc.

The specialists from V.T.Zaitsev Institute of General and Emergency Surgery, Kharkiv, proposed a new method and developed an appropriate apparatus for separation of hepatic parenchyma with minimal traumatization of cells in the zone to be resected. For this purpose, pressurized carbon dioxide is used.

The appearance of the original device for the gas-jet dissection of the liver, called "Pneumojet", is presented in Fig. 15

Robotic Surgery

The history of robotic surgery begins in 1999 with the development of da Vinci robots. This robot is equipped with microinstruments, much smaller than standard laparoscopic instruments, as well as a miniature video camera that produces a color three-dimensional image of the operation in real time. The movements of the surgeon are precisely reproduced by the robot as delicate movements of microinstruments, able to move in all directions. With their help, the operation is much more accurate, keeping intact the finest plexuses of the nerves and blood vessels.

Clinical benefits of robotic surgery:

1) improvement of modern laparoscopic technologies;
2) a method for converting open surgery to a minimally invasive one;
3) development of new mini-invasive surgical operations.

The availability of da Vinci units in the world has increased from 180 in 2002 to 1200 in 2008, and 80% of robots are localized in the United States. The annual number of robotic operations is growing. In 2009, their number in the world was over 180,000.

Da Vinci surgical spectrum is rather wide. It performs operations in urology, gynecology, surgery, vascular surgery. In thoracic surgery, it successfully operates on the lungs, performs cardiac surgery.

Effects for the patient:

1) less traumatic operation;
2) decreased blood loss;
3) good cosmetic effect;
4) minimum risk of infection;
5) reduction of the hospital stay;
6) rapid recovery and return to normal life.

Effects for the doctor:

1) the surgeon should not physically perform actions that often require a large amount of time;

2) he is free from the negative effects - trembling hands, fatigue.

In addition, there are a number of economic effects: shortening the hospital stay, cost reduction, reducing the number of complications.

The most common routine operations in urology are prostatectomy, nephrectomy, cystectomy, adrenalectomy, orchidectomy, kidney removal from a living donor for transplantation; in gynecology - hysterectomy, removal of dermoid cyst, adnexectomy, salpingectomy, reanastomosis of the fallopian tubes, myomectomy, ablation of the endometrium.

In surgery, robotic technologies are used to perform resection of the intestine, hemicolectomy, total colectomy, rectopexy, thyroidectomy, distal pancreatectomy, partial hepatectomy, cholecystectomy, Nissen fundoplication, plastic surgery of paraesophageal hernia, esophagectomy.

CHAPTER I. SYSTEMIC SURGICAL PATHOLOGY

I.1 PURULENT SURGICAL INFECTION. ACUTE DISEASES OF THE SKIN AND SOFT TISSUES

*Ac**ute skin and soft tissue diseases** are purulent infections characterized by inflammation of a different location and nature caused by non-specific purulent coccus microflora - staphylococci, streptococci, gonococci, pneumococci, which are cultured in a pure form or in association with each other, often - in association with E. coli and Pseudomonas aeruginosa.*

Nonspecific purulent infection occupies one of the main places in surgery and determines the nature of many diseases and complications. Patients with purulent-inflammatory diseases make up about a third of all surgical patients.

In the structure of primary visits to outpatient surgeons, the proportion of patients with surgical infections of soft tissues reaches 70%. Such a high percentage is associated with the aging of the mankind, deterioration of the environment, uncontrolled use of antibiotics and other drugs. Acute purulent diseases of the skin and soft tissues often develop in patients with diabetes mellitus and obesity, with hormonal reorganization, immunity weakening, microtraumas, etc.

Despite the progress in modern pathogenetic treatment of patients with acute purulent diseases of the skin and soft tissues and the introduction of new diagnostic methods into the practical health care, the group of patients with this infection in the recent 30 years has not had the tendency to reduction. At present, to treat this group of patients immunocorrection, antibiotic therapy, detoxification methods, enteral nutrition, and others methods are used as well as new ways to treat purulent wounds.

In conditions of general allergy of the population against a background of reducing the immunoreactivity and unsatisfactory environmental situation, increasing the aggressiveness and resistance of microorganisms to antibacterial agents, increased risk of injuries and purulent diseases in both peace and war time (including catastrophe surgery), new information on the methods of treatment of acute purulent diseases of the skin and soft tissues is extremely necessary for future physicians, including those specializing in surgery and non-surgical specialties.

In their clinical course, these diseases, as a rule, pass two stages - *infiltrative* and *abscess stage* (abscess formation). *Complication stage* is possible.

Furuncle

***Furuncle is** purulent necrotic inflammation of the hair bulb and its sebaceous glands.*

This is caused by *Staphylococcus aureus*, less frequently by *Staphylococcus albus*. Multiple lesions are termed furunculosis.

The factors that lead to furuncle development include poor personal, domestic and industrial hygiene, microtraumas, reduced organism reactivity, associated predisposing diseases. Furuncles are usually located on the forearm, the back surface of the palm, on the back of the neck, the inner surface of the thighs, on the buttocks, on the skin of the face, in the external ear canal, at the nasal opening, and others. The most dangerous is the location of furuncles in certain areas of the face, when severe (even fatal) complications can develop. Furuncles do not develop on the skin deprived of hair.

Clinical presentation. The symptoms can be *local and general.* The disease begins with a slight chill and a slight increase in body temperature. A small pustule with a small inflammatory infiltrate shaped as a nodule is formed around the hair. During this period, the patient feels slight itching and tingling. By the end of the 1^{st} -2^{nd} day inflammatory infiltrate is formed, which projects like a cone over the skin level. The skin over it becomes red and painful at touch. At the top of the infiltrate there is a small accumulation of pus with a black dot (necrosis) in the center (in the future the pustule breaks and dries). On day 3-7 the infiltrate undergoes purulent melting and necrotic tissues in the form of a rod together with hair remnants are discharged with the pus. The purulent wound is cleared, covered with granulation tissue and heals. After the pus discharge, the edema and infiltration around the wound gradually decrease, the pain disappears. In the place of inflammation there is a small whitish, slightly drawn scar.

Furuncle may be *single* or appear in different amounts on a delimited body part - *local furunculosis*. Sometimes the appearance of furuncles in the form of multiple rashes in different areas of the trunk and extremities persists with short remissions for several years - *general chronic, recurrent furunculosis*.

Furuncles in the area of the face (lips, forehead), as well as in the scrotum, are accompanied by significant edema of the surrounding tissues, which is explained by the friability of subcutaneous tissue in these areas.

Furuncles and furunculosis should be ***differentiated*** from hydradenitis, anthrax, pseudotuberculosis of newborns and some infectious granulomas (tuberculosis, actinomycosis, syphilis).

The ***complications*** of furuncle include lymphangitis, regional lymphadenitis, abscess, phlegmon, thrombophlebitis, sepsis, purulent arthritis. A complication of a furuncle, distant purulent metastases, is observed in pararenal tissue, bone and (less) in the lungs. For example, furuncle may precede purulent perinephritis and paranephritis or hematogenous osteomyelitis.

***Face is the most dangerous location of furuncles*:** the region of the upper lip, nasolabial folds, the alae of the nose, in the supra- and infraorbital areas, in which development of dangerous *complications such as progressive thrombophlebitis of the veins of the face, thrombosis of the cavernous sinus, purulent meningitis and arachnoiditis, brain abscesses, sepsis are possible*. This is due to the topographic and anatomical features of the specified areas of the face. The infection with progressive thrombophlebitis extends through the facial veins (anterior facial vein, orbital vein pterygoid venous plexus, venous network of the oval foramen, cavernous venous sinus, upper orbital vein, anastomoses of venous sinuses of the dura mater, leading to their thrombosis, creating a threat of development of severe purulent basal meningitis and other complications, often fatal.

In this form, facial swelling develops rapidly, dense painful veins can be palpated, the general condition of the patient deteriorates, body temperature reaches a high level (40-41° C), rigidity of the occipital muscles, visual disorders (visual chiasma involvement) may be expressed. Untimely or inadequate treatment may result in severe complications.

Treatment is divided into *general and local*.

At uncomplicated furuncle the treatment is administered on an *outpatient basis*. Furuncles, which are located on the face, with rapid progression of the process called are "malignant" and even uncomplicated require inpatient treatment even from the very beginning.

In the *infiltration stage* of a single furuncle conservative treatment is used: antibiotic therapy according to indications (in case of dangerous localization), stimulation of body resistance, local treatment (ointment bandages, UHF, UVI, dry heat).

Local treatment of furuncles consists in a careful skin care around the inflammation center: rubbing with 70% alcohol, 2% salicylic alcohol solution or lubricating 1-3% alcohol solution of methylene blue, brilliant green, etc. the hair around the infiltration is carefully cut off. Moist dressings in the form of compresses should not be used because they contribute to infection spread and formation of multiple furuncles. At furuncles of the body, neck and extremities it is worth using adhesive bandages that protect the skin in the area of inflammation from mechanical effects (friction). After care of the skin it is necessary to put an antiseptic bandage with an ointment containing

sulfanilamides or antibiotics. A good effect is produced by ichthyol. When the furuncles are located on the limb, immobilization of the latter is recommended. Sometimes local infiltrative antibiotics are indicated. For this purpose, penicillin + streptomycin, chloramphenicol, oleandomycin and sigmamycin and others are used. The antibiotic is dissolved in 100 ml of 0.25% solution of novocaine or in 100 ml of isotonic saline solution. Infiltrations are repeated 2-3 times. With facial location and the presence of general symptoms, the patients are recommended bed rest and general treatment with antibiotics - preferably chloramphenicol or erythromycin. At cavernous thrombosis intraarterial administration of antibiotics (cephalosporins) in the carotid artery is indicated.

In the stage of *suppuration* of the furuncle, an operation under local anesthesia is performed: either furuncle opening with treatment and drainage of the cavity, or complete excision of the furuncle within healthy tissues with primary suture (when indicated).

When treating furuncles and furunculosis, it is also advisable to use nonspecific stimulating therapy, blood components transfusion, vitamins B, autovaccine, physiotherapy and climate therapy.

The ***prognosis*** of uncomplicated furuncle is quite favorable.

Carbuncle

Carbuncle *is simultaneous purulent necrotic inflammation of several hair follicles and adjacent sebaceous glands with formation of large cutaneous and subcutaneous necrosis and development of diffusion inflammatory infiltration with a tendency to spread in depth.*

This is caused by *Staphylococcus aureus*, less frequently streptococcus, *E. coli*, Proteus and enterococcus. The contributing factors include poor personal, domestic and industrial hygiene, microtraumas, reduced reactivity of the organism, concomitant diseases (diabetes, etc.).

The most typical location of the carbuncle is the back surface of the neck, the skin between the shoulder blades, the skin of the face, the lumbar region, the lower limbs. Facial location is especially dangerous due to the possibility of thrombophlebitis of the facial veins, meningitis and sepsis.

Clinical presentation of carbuncle consists of general and local symptoms. *General ones* include significant intoxication (temperature elevation up to 40° C, nausea, vomiting, loss of appetite, severe headache, insomnia, occasionally delirium and unconscious condition). The general phenomena are strongly expressed in the carbuncles located on the face, at large-sized carbuncles, as well as in patients with diabetes mellitus, obesity and in persons with endocrine disorders. *Local symptoms* include presence of inflammatory infiltrates with several purulent cores on the surface, edema, hyperemia, skin tension, purulent discharge, dysfunction of the limb at the respective location.

Complications of carbuncle include lymphangitis, regional lymphadenitis, abscess, phlegmon, thrombophlebitis, sepsis, purulent meningitis.

The ***treatment*** consists of the following steps:

1) hospitalization;
2) conservative treatment in the infiltration stage;
3) detoxification therapy;
4) anti-inflammatory therapy;
5) pain relief;
6) local treatment;
7) general strengthening therapy;
8) surgical treatment in the stage of abscess.

Tactics of carbuncle treatment in the *infiltration stage*, as a rule, should be ***conservative***. The patient is prescribed bed rest, immobilization of the limb, at facial location it is forbidden to speak, liquid food is recommended. Antibiotics are administered parenterally, taking into account the microflora sensitivity. Prior to the establishment of the pathogen and its sensitivity to treatment, high doses of cephalosporins are administered intravenously. At conservative treatment emulsions or ointments containing antibiotics should be used locally every day. In carbuncles in the infiltration stage, it is also necessary to use proteolytic enzymes in the form of powder (trypsin). Along with the treatment of carbuncle in patients with diabetes mellitus, adequate treatment for diabetes should be performed.

If the conservative treatment is unsuccessful for 2-3 days and abscess forms (progressive edema, temperature elevation up to 40° C, appearance of necrosis, enlargement of the inflammation area), urgent surgical treatment is indicated.

The operative intervention is carried out under anesthesia. *At dissection, treatment and drainage* of the carbuncle, *cross-shaped or H-shaped incisions* of the infiltrated tissue through the entire thickness of the necrosis to viable tissues are used, all necrotic tissues are excised and purulent tissues are removed. The wound is thoroughly washed with 3% hydrogen peroxide solution or antiseptic (dioxidine, Chlorophyllin, etc.) solution and antibacterial bandage with ointments on a hydrophilic basis with pronounced dehydrating effect (Laevomycolum, Levosin, etc.) or with hypertonic (10%) solution of sodium chloride is placed. The bandages are changed daily, on day 5-7, ointments on an indifferent basis, combining antimicrobial activity with anti-inflammatory action are started (Oxyzonum, Oxycort (Poland), Gioxyson (Germany), Geocorton (Germany), Locacorten (Germany) and those stimulating granulation growth (vinylin - Shostakovsky balsam, polymerolum, vulnosan are started, e.g. Kalanchoe ointment, sea buckthorn oil, juice and oil of rosehip, methyluracilum ointment, Ectericid, Combutec Algipor, Solkoseryl, Combined drug Levometoxid ointment).

Radical surgery (removal of carbuncle within healthy tissues with drainage of the wound and application of sparse sutures is possible.

Simultaneously with local treatment are administered massive antibiotic therapy (with definition of the microflora sensitivity), detoxification, infusion therapy (Neohaemodes, Rheopolyglucinum, polyglucinum, Rheomacrodex, saline solutions, glucose solutions, etc.), general, symptomatic therapy (blood sugar correction in patients with diabetes mellitus, etc.).

The ***prognosis*** of uncomplicated carbuncle is generally favorable, but in emaciated elderly patients, patients with diabetes mellitus, with facial location of the inflammatory process (the ways of infection spread are described in the section "Furuncle") carbuncle can endanger the life.

Abscess

Abscess *is an inflammatory formation containing pus and separated by a connective tissue capsule from the surrounding tissues.*

Causes of abscesses:

1) penetration into the tissues of pyogenic microorganisms (abrasions, pricks, wounds, non-compliance with the rules of asepsis and antiseptics at operations);

2) acute purulent inflammation of the skin and subcutaneous tissue (at furunculosis, carbuncle, phlegmon, lymphadenitis, etc.);

3) hematoma suppuration (traumatic and spontaneous);

4) metastatic abscesses at septicopyemia;

5) penetration into the tissues of substances that cause necrosis (turpentine, kerosene, etc.);

6) complication of various diseases (for example, at acute appendicitis, cholecystitis, pneumonia, etc.).

The pathogens are staphylococci, streptococci, rarely pseudomonas aeruginosa and E. coli, proteus.

Acute abscesses and chronic abscesses, as well as "*cold abscesses"* - swelling of tuberculosis origin - can be distinguished.

Clinical presentation. *Acute abscess* is characterized by all signs of inflammation: the area of the abscess is swollen and hyperemic, presence of fluctuation or fluid wave. Fluctuation may be absent when the abscess is located in the depth of the tissues and the wall of the abscess cavity is thick.

In *chronic abscess*, the signs of acute inflammation may be absent. The diagnosis is based on swelling, slight pain, symptom of fluctuation and findings of abscess cavity puncture.

In case of "*cold abscess*" or swelling of tuberculous origin, the presence of the main focus of tuberculosis, slow development of inflammatory events are characteristic.

Abscess should be ***differentiated*** from hematoma, aneurysm, vascular tumors, malignant neoplasms, cysts.

The ***outcome*** of abscess may be different. At a favorable course, the abscess can resorb or empty out, after which self-healing begins.

At an unfavorable course of abscess (more often without surgical treatment), *complications* may develop:

1) lymphadenitis, lymphangitis, thrombophlebitis;

2) phlegmons of soft tissues;

3) sepsis;

4) abscess rupture into any cavity (for example, in the pleural, abdominal or in the cavity of the joint, etc.) with development of severe secondary complications - acute pyopneumothorax, acute purulent peritonitis, etc.

Treatment. Before purulent cavity formation, in the *infiltrative stage*, conservative treatment is administered, using at the same time local or parenteral administration of antibiotics, dissolving therapy. On abscess formation (*stage of abscess*) urgent surgical treatment under general anesthesia is indicated.

The following methods of ***surgical interventions*** are used.

1. *Dissection and drainage of the abscess* (*open method*). The direction of the cuts on dissection must correspond to the direction of the skin folds, and on the limbs they are determined by the places of joint flexion. The position and size of the dissections should provide a good outflow of the pus. Drainage of the abscess cavity after its opening and treatment is made using glove or glove-tube drainage. Earlier, after the surgery in the first phase of the wound process, gauze bandages were used with a hypertonic solution of salt and antiseptic solution. At present, bandages with suction and antibacterial ointments (Laevomycolum, Levosin, etc.) are applied.

2. *Removal of the abscess together with the connective tissue capsule with flow drainage of the wound and sparse sutures (closed method).*

When treating the dissected abscess using an open method, the following principles should be observed.

1. Prevention of secondary infection of the wound (accurate following the rules of antiseptics and asepsis).

2. Removal of necrotic tissue.

3. Application of ointments with dehydrating (suction) effect, hypertonic solutions, enzymes for cleansing the wound.

4. Application of secondary sutures at granulating purified wound.

With the signs of intoxication, larger abscesses, concomitant pathologies (severe somatic diseases, endogenous disorders, obesity, etc.), it is expedient to use antibiotic therapy (with mandatory determining the microflora sensitivity to antibiotics) in combination with detoxification and symptomatic therapy, endocrine disorders treatment and the use of physiotherapy.

Phlegmon

Soft tissue phlegmon *is acute, generalized unlimited inflammation of cellular spaces.*

By *location*, there are subcutaneous, intermuscular, subfascial, intramuscular, mediastinal, retroperitoneal, pelvic tissue, and other phlegmons. Phlegmon often develops in the subcutaneous tissue due to weak resistance of the latter to the infection, frequent trauma and possibility of infection. At certain locations, the phlegmon has a special name. Thus, inflammation of the pararenal fat is called paranephritis, paraintestinal fat - paracolitis, the fat around the rectum - paraproctitis.

By the nature of the exudate there are serous, purulent, purulent-hemorrhagic and putrefactive forms of phlegmon.

Causes of phlegmon development:

1) progression of local purulent processes of the soft tissues (carbuncle, abscess, etc.);

2) inadequate treatment of purulent processes of the soft tissues;

3) injuries.

Phlegmons can be caused by staphylococci, streptococci, E. coli, pneumococci, salmonella.

Clinical presentation is characterized by general and local symptoms. The general ones include: fever to 40° C, chills, headache, general malaise. The temperature is more often constant. Of

the *local symptoms*, the most characteristic are rapid appearance and spread of painful swelling, redness of the skin over it, increased skin temperature, pain, dysfunction of the affected part of the body. The leukocyte count is high and neutrophilosis is pronounced. With the disease progression, the painful swelling is softened and a sign of fluctuation appears.

When the phlegmon involves the surrounding tissue, various ***complications*** may occur - secondary purulent arthritis, tendovaginitis and other purulent lesions; facial phlegmon may be complicated by progressive thrombophlebitis of the facial veins and purulent meningitis.

Treatment. To treat soft tissue phlegmons, ***only surgical methods of treatmen***t are used. In doing so, the following principles should be adhered:

1. Treatment should be administered only on the in-patient basis.
2. Bed rest is obligatory.
3. Immobilization of the affected limb.
4. Use of a large amount of liquid, dairy and vegetable diet.
5. Intramuscular injections of large doses of antibiotics, sulfanilamide drugs.
6. Application of detoxification, general strengthening and symptomatic therapy.

At surgical treatment of soft tissue phlegmons (main method of treatment), dissection and phlegmon drainage with the adherence to the above principles are used. Soft tissue phlegmon is dissected with one, or more often with several, parallel incisions of the skin and subcutaneous tissue (incisions in their position and size should provide a good outflow of purulent contents, their direction should correspond to the direction of the skin folds and determined by the places of flexion of the joints). The phlegmon opening should be done under general anesthesia. After dissection of the skin and subcutaneous tissue, careful treatment of the wound cavity with 3% hydrogen peroxide solution and furacilinum is required. The next stage of the operation is drainage of the wound with glove, glove-tube or tube drainages with prior irrigation using solutions of proteolytic enzymes. To close the wound, gauze bandages with adsorbent antibacterial ointments (see above) or hypertonic saline solution are used.

In the postoperative period, the following principles should be adhered to:

1. Immobilization of the affected area of the tissue.
2. Daily dressing, treatment of purulent wounds.
3. Antibiotic therapy taking into account the flora sensitivity and antibiotic tolerance.
4. Desintoxication, restoration and symptomatic therapy.
5. Correction of endocrine disorders.

Prognosis of a delimited form of subcutaneous phlegmon is favorable. With its severe complications and facial location of the phlegmon, the prognosis is unfavorable; the result depends on the treatment timeliness and adequacy.

Phlegmon of the neck *is an acute purulent inflammation of the soft-tissue spaces of the neck.*

Etiopathogenesis. Phlegmons of the neck can be caused by external or internal (trachea, esophagus) wounds (penetrating wounds of the neck with direct infection), purulent processes of the skin, pustular processes in the area of the scalp and other infectious lesions (abrasions fissures) in the region of the ears, cheeks, lower lip and chin, on the mucous membrane of the mouth, at the bottom of the oral cavity, in the jaws and teeth, purulent lymphadenitis, tonsillitis, purulent processes of the salivary glands, osteomyelitis of the skull base, cervical vertebrae, etc. Sometimes the cause of deep phlegmons is damage to the wall of the esophagus or trachea by foreign objects with subsequent development of infection in their around them. This is especially true for the throat and esophagus at the neck level, the damage of which with swallowed foreign objects (needles, studs, and other sharp objects) are not uncommon. Phlegmons of the neck may occur as a complication of iatrogenic esophagus injury (instrumental at esophagoscopy and gastroscopy, trachea intubation, probing, esophagus bougienage, surgical procedures). They may also occur as sepsis metastases. The location depends on the source of the infection.

Pathogens: in patients with odontogenic phlegmons, aerobes (78.8%) are represented by Staphylococcus aureus and Staphylococcus epidermidis, E. coli, hemolytic streptococcus, enterococcus, proteus and diplococcus; anaerobes (21.2%) are represented by gram-negative

(bacteroids, veillonella) and gram-positive (peptostreptococci, eubacteria) bacteria. Monoculture shows staphylococcus aureus (47.6%) and Staphylococcus epidermidis (33.3%), streptococci (19.1%), veillonellas, peptostreptococci and eubacteria.

Diseases caused by associations of aerobic and anaerobic microorganisms are characterized by severe course, severity of general and local clinical symptoms and signs.

Purulent exudate in patients with acute odontogenic purulent-inflammatory processes of soft tissues is represented only by blood cells. In acute odontogenic inflammatory processes, the "own" pus is formed and its formation is not associated with the mechanical "breakthrough" (spread) from the tooth socket to the bone marrow of the mandible and the related soft tissue. Purulent-inflammatory diseases of the soft tissues of the maxillofacial area occur against a background of temporary immunodeficiency - reduced content of lysozyme in saliva and blood, which is not eliminated by traditional medical treatment and requires immunocorrection therapy.

A number of classifications of ***purulent-inflammatory processes of the neck*** have been proposed, depending on location, clinical course, source of infection, etc.

Classification of neck phlegmons (V.I. Struchkov, 1984), depending on the location of purulent process location (pus accumulation) and lesion depth:

1) in the genian area;
2) in the submandibular region;
3) superficially along the anterior and posterior edges of the sternocleidomastoid muscle;
4) along the length of this muscle in its circumference in the space between the superficial and deep plates of the second cervical fascia;
5) in the inner edge of the sternocleidomastoid muscle;
6) superficially without a certain location in the region of the anterior and lateral surfaces of the neck under the surface muscle of the neck;
7) over the manubrium of sternum – triangle-shaped;
8) in front of the trachea (spread of the abscess to the anterior mediastinum is possible)
9) behind the trachea and esophagus (downward spread is possible to the posterior mediastinum and upward to the retropharyngeal space);
10) in the lateral cervical triangle, limited by the edges of the sternocleidomastoid and trapezoid muscles and the collarbone; these accumulations are usually edemas from deep adenophlegmons, located below the lower edge of the sternocleidomastoid muscle;
11) in the region of the anterior surface of the neck, corresponding to the location of the thyroid gland at thyroiditis, strumitis;
12) in the region of the lateral surface of the neck, at suppuration of bronchogenic cysts;
13) deep phlegmons between the occipital muscles in the back of the neck (arising as a consequence of osteomyelitis of cervical vertebrae or occipital bone).

Classification of abscesses and phlegmons of the maxillofacial region and neck depending on the source of infection (Yu.I. Bernadsky, 1985):

1) odontogenic – caused by gangrenous teeth and their roots;
2) intraosseous - the result of periostitis, osteomyelitis, problematic eruption of wisdom teeth, sinusitis, cysts, etc.;
3) gingival - arise as a result of periodontitis, gingivitis;
4) mucostomatogenic - arise as a result of stomatitis, glossitis;
5) salivary - arise as a result of sialodochitis, sialadenitis;
6) tonsillar-pharyngeal;
7) rhinogenic;
8) otogenic.

Topographic anatomical classification of odontogenic abscesses and phlegmons of the maxillofacial region and neck (G.A. Vasiliev, T.P. Robustova, 1981).

1. Paramandibular abscesses and phlegmons: a) in tissues adjacent to the mandible; b) superficial (submandibular, genian, parotid-masticatory areas) and deep (pterygoid-mandibular and parapharyngeal spaces, sublingual area, bottom of the oral cavity); c) in the tissues adjacent to the

upper jaw - superficial (hypoorbital and buccal areas) and deep (infratemporal fossa and pterygomaxillary fossa).

2. Abscesses and phlegmons of the regions adjacent to the mandibular region, where the purulent process extends (jugal and temporal areas, orbit, retromandibular region, neck), abscesses and phlegmons of the tongue.

Classification of phlegmons of the maxillofacial area and neck according to the course severity and the process spread:

Group 1 (mild) - phlegmons are located in one anatomical region;

Group 2 (medium) - phlegmons are located in two or more anatomical areas;

Group 3 (severe) - phlegmons are located in the soft tissues of the bottom of the oral cavity, neck, half of the face, as well as combination of phlegmon of the temporal region, infratemporal fossa and pterygomaxillary fossa).

Clinical presentation and diagnosis. Clinical presentation and diagnosis of neck phlegmon depend on location and spread of the process. Clinical manifestations are divided into local and general. ***Local manifestations*** of superficial phlegmon include swelling of soft tissues, hyperemia of the skin; diffuse, dense and painful infiltration; fluctuation development.

Deep submandibular phlegmon (phlegmon of the bottom of the oral cavity, Ludwig's angina) begins with rapid diffusion edema of the bottom of the oral cavity and submandibular region, expressed signs of general inflammation, sharp increase in pain aggravated by chewing and swallowing, marked salivation, locked jaw, difficult breathing. Deep phlegmons of the neck with a small amount of dull exudate are dangerous in contrast to the large encapsulated superficial abscesses. Therefore, the following *indirect signs* play a major role in the diagnosis of deep phlegmon: collateral edema, painful limitation of muscular movements, forced position of the neck, and others. Swelling and redness with deep phlegmons should not be expected. Radiating pains are of neurological character. An important symptom at deep phlegmons of the neck is painful *stiffed neck.*

At adenophlegmon, development of the inflammatory process is slower and is characterized by increase in tissue infiltration and formation of purulent exudate. If the onset of the inflammatory process is preceded by previous microbial sensitization of the organism to staphylococci, streptococcus, E. coli and other microorganisms, development of adenophlegmon occurs in a shorter term and is characterized by aggressiveness of the course, which complicates the differential diagnosis of adenophlegmon from odontogenic phlegmon.

The general manifestations of neck phlegmon include: expressed intoxication of the organism; leukocytosis, neutrophilic shift to the left, eosinopenia (aneosinophilia), high ESR (up to 55 mm/g), anemia that is not associated with iron deficiency in the blood serum. Reduced amount of red blood cells and hemoglobin concentration are due to inhibition of erythropoiesis rate under conditions of intoxication and others.

A characteristic feature of neck phlegmons located under the muscle along the vasculonervous bundle is that the patients avoid the slightest movements of the head and keep it slightly turned and inclined to the sick side. As a result, the pressure on inflammatory infiltrates placed under the muscle decreases.

Clinical manifestations of the diseases caused by anaerobic infection are characterized by severe course and absence of positive dynamics of the process, even with sufficient drainage of the inflammatory focus. The signs of intoxication grow rapidly, the patients develop tachycardia, microcirculation disorders. General manifestations of infection caused by asporogenic anaerobes include subicteric scleras, jaundice of the skin, anemia, significant leukocytosis, high ESR. The signs of anaerobe infection can include an unpleasant smell of exudates, a large amount of necrotized tissues in the purulent foci, dirty-gray color of the purulent contents with the presence of droplets of fat in it. Bacteroid multiplying is accompanied by production of gases, which facilitate easier invasion of the bacteria beyond the inflammatory focus. Increased pressure in the tissues of the pathological focus further affects the blood circulation, contributing to hypoxia and growth of anaerobic microorganisms. After surgery, a small amount of copious pus smelling unpleasantly is produced. The muscles look

like boiled meat. When melaninogenicus bacteroids participate in the process, the secretion from the wound is dark or brown. The use of antibacterial drugs is of low effect.

Purulent melting, which occurs with further progression of the process, is accompanied by changes in the configuration of the inflammatory infiltration, its outlines are smoothed out and become more vague. With the exit of the boil beyond the limits of the lymph nodes, further involvement is determined by the boundaries of the respective tissue space, as well as the adjacent spaces. If the boil is located in the tissue, surrounding the sheath of the carotid artery and jugular vein, it spreads relatively quickly along the vessels, especially in the presence of virulent infection. An example of this can be acute-purulent process, the source of which is carious teeth. In these cases, the upper group of cervical lymph nodes located under the upper end of the clavisternomastoid muscle on the jugular vein is initially affected. Hence, after melting of the lymphatic vessels, the boil spreads to the tissue of the vascular sheath and then to the subclavicular and axillary fossae.

Septic character of the process is characterized by a very large size of the inflammatory tumor, which occupies the entire half of the neck and extends below the limits of the clavisternomastoid muscle, and more rapid development of the inflammatory process.

A dangerous *complication of anaerobic infection* is ***septic shock***, which causes extremely high (50-70%) mortality (Yu.G. Shaposhnikov, 1984) and is characterized by development of septic phlebothrombosis not only near the infection focus, but also in remote areas - lungs, brain, liver, joints and other areas.

Features of phlegmon course in children. Children often develop adenophlegmons and rarely - odontogenic phlegmons. In some cases, phlegmons develop against a background of colds and acute otitis media. The clinical course of phlegmon in children is associated with the peculiarities the dentofacial system development. Imperfection of the immune system in young children contributes to more aggressive course of the inflammatory process.

Retropharyngeal abscesses (especially in children) are characterized by difficulty in breathing and sometimes asphyxiation.

Features of phlegmon course in elderly and old persons are similar to those in young people, but in the old ones phlegmons develop much later after the beginning of the previous disease, and melting of the inflammatory infiltrates occurs more slowly. The possibility of their spontaneous resorption is excluded, thus expecting tactics is not justified. Adenophlegmons in the elderly are rare. As a rule, they are similar to encapsulated abscesses. On dissection of such phlegmons, it is necessary to remove purulent-molten lymphoid tissue of the node.

Phlegmons of the neck are accompanied by common septic phenomena. But danger is possible not only with general complications. There is a danger of penetration of the acute purulent process in depth, especially after necrotic process of facsial tissue melting and infection penetration into the chest (into the anterior or posterior mediastinum).

Clinical presentation of the most common phlegmons of the neck. ***Phlegmon in the tissue under the lower jaw.*** This space is the most frequent location of neck adenophlegmon. They can be superficial and deep. The cause is dental caries, periodontitis, tonsillitis, ulcerative stomatitis, infected wounds of the face, and others.

Superficial submandibular phlegmon is adenophlegmon of the lymph nodes (separate or several) located below the lower jaw. The disease is characterized by inflammatory swelling, hyperemia, pain on palpation and often a chronic course. The pus does not spread, there is a tendency to burst.

Deep submandibular phlegmon is also called phlegmon of the floor of the oral cavity and Ludwig's disease. This is a severe disease that begins acutely, with high fever, chills, and malaise. It appears as diffuse swelling of the floor of the oral cavity and suprahyoid region, which are densely infiltrated and very painful. At the same time the skin does not get red, but most likely becomes pale with a blue tint. Locked jaw occurs early. Abundant salivation begins and difficulties on swallowing and breathing arise. Due to swelling of the uvula, there is a danger of dyspnea. The infection can spread to the mediastinum. The patients are intoxicated and their condition is severe. Mortality among the untreated patients is high (50%). The death occurs as a result of sepsis or dyspnea.

Genial phlegmon remains limited and is rapidly inflated. It is caused by fissures of the lips, as well as furuncles and eczema on the chin.

Phlegmons of medium spaces of cervical subcutaneous tissue. In this area, acute purulent processes are rare, and are mainly represented by lymphadenophlegmons of those lymph nodes in which the lymph from the larynx, trachea and the thyroid gland is accumulated. There is suppuration in the neighborhood (*per continuitatem*) at purulent thyroiditis and strumitis. Large accumulations of pus result in difficulty in breathing. Purulent cavities, located between the esophagus and the trachea and between the esophagus and *fascia praevertebralis*, are caused by inflammations of the retropharyngeal lymph nodes or osteomyelitis of the cervical vertebrae.

At esophagus rupture, phlegmons of the middle fascial cervical space develop instantly and spread freely to the mediastinum.

Phlegmons in the blood vessel sheath. By frequency these phlegmons occupy the second place after submandibular phlegmons. They originate from purulent inflammations of the deep lymph nodes located along the large vessels (the carotid artery, the internal jugular vein). They most often develop at tonsillitis and scarlet fever. This purulent process, known as "postnatal septic phlegmon of the neck", is severe, with a septic course of the disease. At cervical fascia rupture the pus spreads to the subclavian and axillary fossa. The complications of phlegmon in the vascular sheath include septic thrombosis of the internal jugular vein, septic erosion of the vessels, mediastinitis, swelling of the vocal system, sepsis. All above complications endanger the life of the patient.

Phlegmons of supraclavicular region develop in the supraclavicular subcutaneous tissue space and originate from lymphadenophlegmon or purulent processes in the facsial neck spaces. They are characterized by appearance of a painful tumor in the supraclavicular fossa between the sternocleidomastoid muscle and trapezoid muscle. Penetration to the vascular bed results in thrombosis and erosion.

Phlegmons on the back of the neck are much less common. They are caused by osteomyelitis in the cervical vertebrae and occipital bone.

Differential diagnosis. Phlegmon of the neck should be differentiated from tuberculous lymphadenitis, cold abscesses, actinomycosis, ligneous phlegmon, facial furuncles and carbuncles, erysipelas, acute inflammatory diseases of the salivary glands, suppurated cysts of the soft tissues. Odontogenic phlegmons can be differentiated from calculous and noncalculous sialoadenitis by the following: in sialoadenitis the pus is discharged from the ducts of the glands, there is a salivary calculus in the duct of large salivary glands, which can be palpated; radiography of the soft tissues of the floor of the oral cavity can detect salivary stones in the submandibular gland. Suppurated cysts of soft tissues can be differentiated from odontogenic phlegmon by the history of preceding soft-elastic formation; the puncture of the cyst shows liquid content; odontogenic source of inflammation is absent.

Differential diagnosis of *odontogenic* and *non-odontogenic* phlegmon. The clinical manifestations of non-odontogenic phlegmons of the maxillofacial area do not differ from those of odontogenic origin, which makes differential diagnosis difficult. Significant role in establishing the correct diagnosis is played by careful history taking. First of all, the duration of the inflammatory processes in the oral cavity should be established. The oral cavity is carefully inspected to investigate the condition of the teeth and the presence of various odontogenic foci of infection: pathological dentogingival pockets, fistulas on the alveolar sprout, pericoronitis, painful thickening of the alveolar sprout, infiltrates on the transitional fold, paradontitis, etc. Particular attention is paid to radiological examination of the jaws to detect pathological changes in the bones associated with dental diseases.

Treatment of patients with neck phlegmons should include all modern methods of combating acute purulent infection. *Algorithm of cervical phlegmon treatment:*

1) rest, both general, and in the area of inflammatory focus; the patient is offered bed rest;
2) injections of antibiotics (semisynthetic penicillins, cephalosporins, aminoglycosides);
3) local warming compresses and UHF-therapy;
4) local application of cold can be recommended only in the earliest stages of the disease, before the appearance of inflammatory infiltration;

5) in the stage of serous edema, diadynamophoresis with proteolytic enzymes is used;

6) immunocorrection therapy with lysozyme, trophosan and other immunostimulants is administered;

7) with the purulent process progression (ineffectiveness of conservative therapy) operative intervention guided by topographic-anatomical correlations of the organs and localization of the boil is recommended.

Surgical treatment of phlegmon. At surgical intervention, it is necessary to take into account the fact that adenophlegmons of the neck are always located under the fascia. Exceptions are those that occur in the lesion of the superficial lymph nodes in the region of the upper third of the sternocleidomastoid muscle. The incision must correspond to the area of the greatest fluctuation. Careful layered dissection of the tissues prevents the damage to important formations, primarily vessels.

All operations of phlegmon dissection should end with introduction of polychlorvynil drains (tubes) into the cavity of the boil. Tampons are used to stop capillary bleeding in the depths of the wound, and also protect the abscess cavity from premature collapse (before rejection of necrotic tissues and formation of granulations).

Subpectoral phlegmon (SP - phlegmona subpectoralis) is a severe purulent-phlegmonous inflammation of the friable connective tissue, located under large thoracic and small thoracic muscles.

The most common cause is hemolytic streptococcus.

Etiopathogenesis and pathological anatomy. SP begins as lymphadenophlegmon (in wartime the cause is a wound). Less commonly, the infection spreads from the supraclavicular or axillary region. The primary source of lymphadenophlegmon is located on the radius side of the forearm or humerus. The infection may be immediate as a result of injury or spreads metastatically. Phlegmonous inflammation begins with serous edematous infiltrate, in which the loose connective tissue becomes gelatinous. Locally there are limited purulent foci. Large purulent clusters do not form.

Clinical presentation and diagnosis. SP is characterized by the general phenomena of intoxication with a high fever, chills (semiconsciousness) with insignificant local focus (connection through the lymphatic system with lungs and pleura). At the initial stage of the inflammation, the local phenomena are unclear and manifest by pain in the shoulder region, slight abduction of the hand, and palpatory sensation in the subclavicular fossa, which is edematous and pasty at palpation. Thrombophlebitis of *v. sephalica* develops. Strong pain at passive movements in the shoulder joint is typical. If SP is not a complication, the diagnosis of subpectoral right or left adenophlegmon is made.

The condition should be differentiated from phlegmon of the anterior chest and anterior abdominal wall, purulent hidradenitis on the side of the lesion, pleurisy, lung abscess, purulent mediastenitis, erysipelas (develops post-operatively after incision, the etiopathogenesis is unclear).

Treatment is initiated as a ***conservative method*** with high doses of semisynthetic penicillins combined with aminoglycosides and metronidazole or protected penicillins with aminoglycosides and metronidazole; UHF and antiseptics are used topically, immunostimulants, vitamins and symptomatic treatment are administered; if necessary - drip infusion-therapy is indicated. After antibioticogram a certain antibiotic (antibiotics) is prescribed.

Surgical treatment consists in a wide opening of the phlegmon (if necessary several incisions and contraincisions-contrapertures) and drainage should be made. In the postoperative period, the entire range of conservative therapy is applied.

Prognosis. When the diagnosis is not established and treatment is not targeted, serious complications (including sepsis) occur even with small local manifestations of the infection (described in differential diagnosis section), and often lead to death. With timely and correct treatment, the prognosis is favorable.

Acute purulent hidradenitis

Acute purulent hidradenitis *is an acute purulent inflammation of sweat glands in the axillary, inguinal and perianal regions, as well as in the area of the nipples of the mammary glands in women.*

The causative agent is staphylococcus, often aureus. The contributing factors are sloppiness, obesity, intertrigo, scratches and diabetes mellitus. The causes include microtraumas during shaving and small lesions of the skin.

Etiopathogenesis. Staphylococcus aureus enters the duct of the sweat gland through the injury during shaving or any other minor skin damage, and the used antiperspirants prevent sweat discharge and washing out of the staphylococcus, contributing to development of microbial inflammation directly in the gland. From the infected sweat gland, the infection spreads to other sweat glands through the lymphatic routes. Staphylococci, getting into the sweat gland, begin to multiply actively, causing inflammation. In the gland, an inflammatory infiltrate containing multicellular leukocytes, lymphocytes, eosinophils, plasmocytes and a large number of staphylococci is formed. In the course of the inflammatory reaction, the infiltrate is subjected to purulent melting with destruction of the sweat gland.

Clinical presentation. At the beginning of the disease, a dense, painful palpable node develops in the depth of the subcutaneous tissue; the skin over the node is unchanged. 1-2 days later, the node enlarges up to 1-1.5 cm in the diameter, protruding above the surface of the skin in the form of a hemisphere. The skin above the node is initially hyperemic, and then becomes purple-red. The pain at this time increases, gaining a pronounced character, interfering with movements. After 7-10 days, an infiltrate is formed, with its melting a creamy (dense) pus bursts through a small opening in the skin. The infection simultaneously affects from five to seven sweat glands.

A massive infectious lesion causes swelling of the subcutaneous tissue. The skin of the axillary region sags down and on its surface several boils open resembling a nipple. Inadequate and untimely treatment results in melting of the subcutaneous tissue and merging of the boils and phlegmon development.

With a solitary lesion of the sweat gland, general symptoms are not observed. The reaction in the peripheral blood is absent or insignificant. At purulent lesion of several sweat glands, the signs of endogenous intoxication, such as severe malaise, are observed; pain syndrome limits the limb mobility on the affected side. The body temperature increases to subfebrile figures, chills may appear. The appetite worsens and sleep is disturbed. Leukocytosis, neutrophilosis with a shift to rod nuclear cells, increased ESR are observed in the peripheral blood.

The diagnosis of acute purulent hidradenitis is not difficult due to the typical location and characteristic distinct clinical presentation of the disease.

The disease should be ***differentiated*** from furuncles, lymphadenitis, axillary lymph node tuberculosis, actinomycosis and other deep mycoses. Unlike furuncles, purulent hydradenitis, does not form a primary pustule, the infiltrate is semispherical (not cone-shaped), purulent necrotic shaft is not formed. When the differential diagnosis is difficult, histological and microscopic investigations are necessary.

Treatment of acute purulent hidradenitis is aimed at cleansing of the purulent foci, the fight against the infection spread and prevention of the disease relapses. Solitary and recurrent purulent hidradenitis is in most cases treated on the ambulatory basis. Multiple involvement of sweat glands with the signs of intoxication require hospital stay, infusion of antistaphylococcus plasma, UV and laser irradiation of the blood, immunostimulants and courses of antibiotic therapy with semi-synthetic penicillins, 1^{st} and 2^{nd} generation cephalosporins, symptomatic treatment for relieving pain and sleep disorders.

Operative treatment is performed after the infiltrate softening with formation of an abscess. It consists in incision, cleansing and drainage of the purulent cavity. The operation is performed under general anesthesia. After careful treatment of the operating field with antiseptic solution, the skin over an abscess is incised with a small incision (in the direction of the skin folds), the cavity is washed with solutions of antiseptics, dried and drained with a rubber strip and bandaged with suction and antibacterial ointment on the water-soluble base (Laevomecolum, Levosin). In the presence of several purulent cavities, they are opened at the same time. In case of close location of purulent foci, their radical excision is performed within healthy tissues, with establishment of flow drainage and primary suturing. Bandages are changed daily.

Prognosis with timely and correct treatment is favorable.

Acute paraproctitis

Acute paraproctitis *is acute purulent inflammation the perirectal tissue.*

Paraproctitis occupies the 4th place after hemorrhoids, anal fissures, colitis among proctological diseases and accounts for 20-30% of all diseases of the rectum. Paraproctitis is a disease of adults.

This is caused by mixed microflora. Staphylococci and streptococci in combination with the E. coli (in 98% of all cases) are most often revealed in the culture. Such paraproctitis is called ordinary or banal. Specific infection (tuberculosis, syphilis, actinomycosis) is a causative agent in 1-2% of cases.

The *main routes* of infection into the pararectal tissue are the following:

1). Ducts of the anal glands (infection from the anal crypt, where 6-8 anal glands open, gets into the anal gland, and from there to the pararectal tissue).

2). Damage to the rectum mucosa. Microtrauma of the rectal mucosa can be caused by undigested food particles, dense lumps of feces, foreign objects (fish bone, fruit kernels, etc.).

3). Damage to the wall of the rectum with therapeutic manipulations (enemas, blockades, injections).

4). The presence of other diseases of the rectum (hemorrhoids, anal fissure, inflammatory lesions of the mucous membrane of the final segment of the rectum, ulcerative colitis, Crohn's disease, etc.).

5). Injury of the rectum during surgical operations, gunshot wound, accidents.

6). Hematogenous and lymphogenous ways of infection: at flu, tonsillitis, various purulent diseases of other areas.

7). Inflammatory lesions of the perirectal tissue due to diseases of the adjacent organs: limited pelvic peritonitis, diseases of the prostate gland, urethra, female reproductive organs, epithelial coccygeal disease.

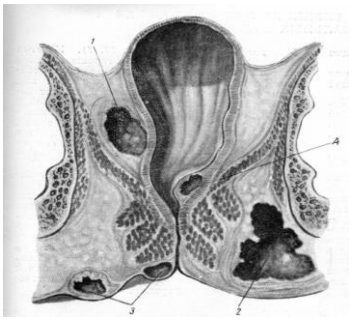


Fig. 16. Types of acute paraproctitis:
1 - pelvirectal; 2 - ischiorectal;
3 - subcutaneous;
4 – submucosal

Classification of acute paraproctitis.

By etiology:

1) non-specific;
2) specific;
3) post-traumatic.

By location (Fig. 16):

1) subcutaneous;
2) submucosal;
3) ischiorectal
4) pelvirectal;
5) retrorectal;
6) horseshoe-shaped.

Clinical presentation. The onset is usually acute. Chills, fever and rising pains in the rectum, perineum or in the pelvis appear following the prodromal period of malaise, weakness, headaches. These are the most common symptoms of the disease. The degree of severity of acute paraproctitis symptoms depends on the location of the process, the type of bacteria, their associations and the organism reactivity.

Subcutaneous paraproctitis is one of the most common forms of the disease (~ 50% of all types of paraproctitis). The patients report rapidly increasing pain in the perineum near the anus. Body temperature rises, reaching 38-39° C in the evening, chill appears. The pain is acute, pulsating, aggravated by movement, changes in the body position, tension of the abdominal press, with coughing, defecation; there is stool retention, and when the boil is located in front of the anus, dysuric disorders are present. The skin on the side of the involvement is hyperemic, there is swelling in the perianal region, the radial folds around the anus are smoothed. When the boil is located in the immediate vicinity to the anus the latter is deformed. The anal canal becomes slit-like, sometimes yawning. In

these cases, the patients may have gas incontinence, liquid stool, leakage of mucus. Palpation of the rectum demonstrates tenderness and fluctuation in the area of the boil.

Subcutaneous paraproctitis should be differentiated from ischiorectal paraproctitis. Subcutaneous paraproctitis is not located higher the pectineal line. With this form, the intestinal walls above the level of the anal canal are elastic, while in ischioretal one, digital examination reveals infiltration behind the intestine, the intestinal wall bulges.

Submucosal paraproctitis is the most mild form of paraproctitis (found in 1.9-6.3% of cases). The patients report pain in the rectum, aggravated during defecation; low-grade fever. Digital examination of the rectum determines rounded, tight-elastic formation, located under the mucous membrane over the pectineal line.

The diagnosis of submucosal paraproctitis is not difficult and is based on the complaints (dull pain in the rectum, subfebrile temperature) and data of digital examination of the rectum (boil bulging in the lumen of the intestine from the mucous membrane, flattening and infiltration of one of its walls).

Ischiorectal paraproctitis (35-47% of all cases of acute paraproctitis) develops gradually, beginning with deterioration of the general condition, appearance of occasional chills, weakness, sleep disorders. The signs of intoxication appear gradually: the temperature rises rapidly to 39-40° C, chills appear. Sharp movements and defecation cause feelings of weight and acute pulsating pains in the pelvis and the rectum. When located in the area of the prostate gland and urethra, dysuric disorders are observed. External signs of acute ischiorectal paraproctitis appear at the end of the first week. On the skin of the perineum, there is swelling and hyperemia; the tissues in this area become pastry-like, the pain in the pelvis becomes worse at palpation of the soft tissues of the perineum. Digital examination of the rectum reveals infiltration of the wall of the lower ampullar section and the anal canal above the pectineal line.

Pelvirectal paraproctitis (1.9-7.5% of the total) is the most rare and severe form of the disease. The initial stage of the disease is characterized by slowly elevating fever, periodic chills, headache, dragging pain in the joints, heaviness and uncertain pain in the pelvis or lower abdomen, radiating to the scrotum or bladder. Digital examination reveals tenderness of one of the walls of the medioampullar or inferoampullar rectum, infiltration of the intestinal wall. Rectoscopy shows hyperemic, velvety, with meshy vascular pattern mucous membranes adjacent to the infiltration. The duration of the first period averages from 1 to 3 weeks, although it can take up to several months. The late period begins with formation of an abscess in the area of infiltration of the pelviorectal tissue. With the abscess appearance, the disease takes an acute course: the dull pain in the rectum and pelvic area increases, this is accompanied by increased intoxication, hectic temperature, stool retention; abscess bursting into the rectum is possible. Digital examination of the ampulla determines thickening of the intestine, driving it back from the outside, bulging of an elastic, fluctuating tumor into the lumen, over which the mucous membrane of the intestine remains movable. The upper pole of the boil is usually not reached by the finger. Rectoscopy reveals a cleft-like or circular stricture of the lumen, the mucous membrane of the abscess is thinned, easy to bleed. X-ray of the pelvis, which should be performed after the obligatory preparation of the intestine with enema, reveals inclusions against a background of more or less intense shadow of the infiltration or abscess.

The condition should be differentiated from ischiorectal paraproctitis, suppurated perirectal cyst and abscess in Douglas space.

Retrorectal and horseshoe-shaped paraproctitis are variations of pelvirectal forms and occur in 1.7-2.8% of cases. The peculiarity of these forms is pronounced pain syndrome, which is noted from the very beginning of the disease. The pains are located in the rectum and the sacrum, aggravated by defecation and sitting. External signs manifest only in neglected cases, when the pus bursts into the pararectal tissue or through the skin of the perineum. Valuable diagnostic information is obtained at palpation of the sacrum and perineum, as well as digital examination of the rectum. Pressure on the coccyx sharply enhances the pain. Digital examination shows bulging in the back wall of the rectum, which, depending on the stage of the process and the terms that have passed since the onset of the

disease, may have different sizes and different densities, from dense to tightly elastic and always painful.

Differential diagnosis. Retrorectal paraproctitis should be differentiated from cystic, teratoid and tumor formations of the same location, chordomas and other tumors of the sacrum.

Treatment is operative; it should be performed urgently under anesthesia on an inpatient basis. Only in this case an adequate incision and drainage of the boil are possible.

Main stages of the *open method*: 1) opening and drainage of the boil; 2) elimination of the internal opening of the fistula, which connects the cavity of the boil with the rectum.

Two types of incision can be used, *radial and crescent*. Radial incision is more often performed in subcutaneous forms of acute paraproctitis, when the fistula passage is located inward from the sphincter. Crescent incision (Fig. 17) is performed at ischiorectal, pelvirectal and horseshoe-like forms of paraproctitis.

Operation technique. Under anesthesia, a rectal speculum is introduced into the lumen of the rectum and the anal canal and lower ampullar section of the intestine is inspected visually. Using fingers and by eye the boil limits are determined in the pararectal tissue and the intestinal wall; the inner opening of the fistula is looked for. If the location of the inner opening cannot be determined, the fistula is stained with methylene blue (the cavity of the boil is punctured with a thick needle, 2-3 ml of pus is sucked out with a syringe and 1-2 ml of 1% methylene blue solution with 1 ml of 3% hydrogen peroxide solution are injected through the same needle). The inner opening of the fistula is detected by a blue trace which is clearly visible when the anal canal is investigated with the help of the speculum.

In *acute subcutaneous paraproctitis*, the radial incision is used to open the abscess, the passage of the fistula is excised with the inner opening, the wound is washed with 3% hydrogen peroxide solution and tamponed with an ointment swab. A colonic tube is introduced into the rectum. In another variant (operation according to Ryzhikh and Bobrov) a crescent incision up to 5 cm in length, is made over the abscess stepping away 3 cm from the edge of the anus, the pus is evacuated. A grooved probe is introduced from the wound to the lumen of the intestine through the inner opening of the fistula. The fistula passage is incised along the probe. The mucous membrane with the involved crypt in the area of the inner opening of the fistula is excised. The wound is treated with hydrogen peroxide solution, 1% iodine solution, and a swab with ointment on a water-soluble base (Levosin, Laevomicolum) or Vyshnevsky ointment is introduced. A colonic tube is introduced into the rectum.

In *acute ischiorectal paraproctitis* a crescent incision (Fig.17) is used to open the boil.

The pus is evacuated. A probe or a tip of Bilroth's forceps is introduced to the intestine through the inner opening of the fistula from the side of the wound. Then the thickness of the muscular formations, located between the fistula passage, purulent cavity, the borders of the sphincter and the lumen of the intestine, is determined. If this is a sphincter fistula (a part of sphincter ani externum), the fistula passage is dissected along the probe or Bilroth's forceps. After this, the inner opening of the fistula is dissected in a wedge-shaped manner with the adjacent crypts, as well as the hanging over edges of the mucous membrane and the skin to prevent adhesion of the edges of the wound. The wound is treated with 3% hydrogen peroxide solution and 1% alcohol iodine solution and dried. A swab with ointment is introduced into the cavity. A colonic tube is introduced into the rectum. If after the boil dissection it turns out that the fistula passage is located outside the sphincter, that is, if between the fistula passage and the lumen of the intestine there is the entire thickness of the muscles of the sphincter ani externum, two methods of surgical treatment can be used – according to Ryzhykh and Bobrova or with ligature method.

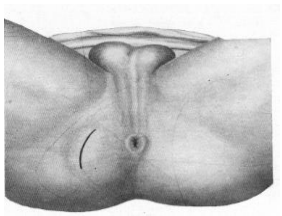


Fig. 17. Crescent incision at acute ischiorectal paraproctitis

The technique according to Ryzhykh and Bobrova consists in opening the boil using crescent incision, evacuation of the pus. Sphincterotomy is performed through the inner opening of the fistula, the mucous membrane is excised to the depth of 1 cm. The surgical intervention is accomplished in the typical manner.

At *ligature technique,* after boil opening and pus removal, crescent incision is continued to the middle line behind or in front of the anus, depending on the location of the inner opening of the fistula. Enclosing incision is used to excise the mucous membrane with the internal fistula. The inner opening of the fistula is carefully scraped with a sharp spoon and treated with 1% alcohol iodine solution. A thick silk ligature is introduced through the inner opening of the fistula and wound. It is placed along the middle line in front or behind the anal canal (depending on the location of the fistula) and tightened (Fig. 18). A colonic tube and ointment swab are introduced into the intestine. The wound of the perineum is tamponed with the aforementioned ointments.

In acute *pelvirectal paraproctitis* (the boil is connected with the rectum by fistula passage located outside the sphincter), two types of surgical interventions are used: according to Ryzhykh and Bobrova and ligature technique according to Marangos.

In *acute retrorectal paraproctitis,* ligature method is used, in *acute horseshoe-like paraproctitis* – two techniques: dissection of the fistula passage at its intrasphincter or transsphincter location and ligature - at extrasphincter location. During the surgery for horseshoe-like paraproctitis, it is imperative to conduct a thorough revision of the boil cavity and its edema.

It is possible ***to remove the boil within healthy tissues with adjustment of flow drainage of the wound and application of sparse sutures.***

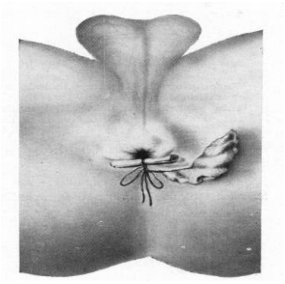


Fig. 18. Ligature technique of surgical treatment for acute paraproctitis

Postoperative management of the patients.

1. Diet. During the first 3 days the diet should be limited to waste-free products, and later - food that contains a minimal amount of waste-forming products. Spicy food is prohibited for the period of 3 months.

2. Regimen. Within 4 days movement in the ward is restricted to avoid an early urge to defecate.

3. Stool retention during the first 4 days after the operation. Drugs that delay defecation are prescribed (tincture of opium, 8 drops with ascorbic acid 0.35 3 times daily or norsulfazol 0.5 g 3 times a day with levomicetin 0.5 g 3 times a day.

4. The use of analgesics and narcotic drugs during the first week after surgical treatment.

5. Administration of symptomatic therapy by indicators.

6. Administration of infusion and detoxification therapy in case of intoxication.

7. Improvement of intestinal emptying. 4-5 days after the surgery 30 ml of Vaseline oil orally twice a day are prescribed to improve the wound healing. If in the morning of the next day there is no unaided stool, cleansing enema is recommended. After the act of defecation, general bath and dressing are recommended. With stool appearance, the following sequence of management: stool, bath, bandage, is required.

8. Antibiotic therapy. Before obtaining pus culture findings and determining bacteria sensitivity to antibiotics, injections of the 1st-2nd generation cephalosporins are prescribed (cephazolin, cephaloridine, cefuroxime, cefotetan, cefoxitin, cefamandole) in combination with aminoglycosides (gentamicin or amikacin).

9. Daily dressing. On the first day, the wound swab in the perineum wound is changed, colonic tube is removed and a tampon with Vishnevsky ointment is placed. On day 2, the tampon with Vishnevsky ointment in the wound of the perineum and the rectum is changed. From day 3 after surgery, the wound of the perineum is not tamponed. Suppositories are introduced to the intestinal lumen, loose tampons with antibacterial ointment, with 3-10% bee glue ointment, solution of proteolytic enzymes are introduced to the perineum wound.

Felon

Felon *is acute purulent inflammation of the finger tissues.*

Purulent diseases of the fingers and hand rank first in frequency among all purulent processes. Of all the primary patients seeking for surgical aid, those with felon and phlegmon of the hand make

up from 15 to 31%. The inflammatory process complicates 40% of small injuries of the hand, the economic losses associated with temporary disability many times exceed those from inflammatory diseases of another location and more often affect men of working age. In 60% of patients with this pathology there are complications, not related to the radicalness of surgical interventions. In 25% of patients finger mutilations follow repeated operations, they lead to disability in 8.0% of patients. The highest percentage of unsatisfactory treatment outcomes is noted at bone, tendon, articular, osteoarticular felons and pandactylitis, as well as combined and associated phlegmons of the hand. In 17-60% of patients with bone felon, phalanges are amputated. Pandactylitis treatment outcome is unsatisfactory in 60% of cases.

The main factors contributing to felon development:

1) microtrauma (abrasions, pricks, scratches, splinters, etc.);

2) occupation of the patient (persons working in the spheres where manual labor is used);

3) immunocompromised persons (avitaminosis, exhaustion, radiation sickness);

4) inadequate first medical aid (failure to provide first medical care at microtrauma of the finger).

The main purulent infection agents include:

- Staphylococcus
- streptococcus;
- E. coli;
- Proteus vulgaris;
- Pseudomonas aeruginosa

Classification of felon:

- subungual;
- periungual (paronychia);
- cutaneous;
- subcutaneous;
- tendinous;
- osseous;
- articular;
- osteoarticular;
- pandactylitis
- finger furuncle.

Any form of felon, as well as all purulent processes, is characterized by two stages of the inflammatory process: *serous-infiltrative and purulent.*

Cutaneous felon. The purulent process is intradermal. Acute cutaneous felon is characterized by the following clinical symptoms:

1) a throbbing bursting pain;

2) dysfunction of the finger: the finger is in a semi-flexed position, and attempts to straighten it are painful;

3) appearance in the center of the focus of a slight whitening of the skin against a background of its generalized hyperemia;

4) redness over the palmar surface of the finger;

5) fluctuation over the boil;

6) inflammatory infiltration of the finger.

Treatment is operative and consists in complete removal (possible without anesthesia) of the detached epidermis. After a careful toilet with 3% hydrogen peroxide solution and furacilin solution, ointment dressing is applied (levosin, laevomicolum, etc.). The limb is immobilized.

Subcutaneous felon. The process is located in the subcutaneous tissue. Its main symptoms are pulsating pain, swelling, limitation of the finger movement and temperature elevation. Neglected subcutaneous felon can lead to its transition to osseous, articular or tendinous. Treatment of *subcutaneous felon* is ***operative***, it consists in early surgical intervention and antibiotics administration.

Subungual felon is inflammation that develops under the nail after punctate wounds, splinters, subungual hematoma suppuration. Pus accumulation is clearly visible under the nail. Pressing the nail causes a sharp pain, the nail plate is shaking. Its fixation to the bed is lost, the nail is strongly attached only in the proximal section near the matrix. Swelling and hyperemia of the skin are not expressed. The main symptom is a throbbing bursting pain in the nail phalanx. The intensity of the pain increases with the inflammatory process development. Complete sequestration or surgical removal of the nail plate creates the necessary preconditions for recovery. Following wound epithelization, complete regeneration of the nail occurs 4 months later.

Paronychia. *Paronychia is inflammation of the nail fold that surrounds the base of the nail, accompanied by painful swelling around the nail fold and hyperemia of the surrounding tissues*. On examination, the attention is drawn to the hanging of the affected nail fold over the nail plate. Palpation of the swollen tissues of the back surface of the nail phalanx, where the inflammatory process is located, is rather painful. Due to the increasing inflammation, the patients quickly lose the ability to work, which makes them seek for the aid of a surgeon. At paronychia, purulent exudate accumulates in the thickness of the fold, lifts the epithelium and thins it considerably. The boil can sometimes burst spontaneously. The condition of the patient thus improves, the pain in the finger subsides. This circumstance gives rise to a delay with surgical intervention. The patients, and sometimes experienced surgeons, consider further surgical treatment inappropriate and use different antiseptic solutions and ointments on the area of the boil. Such measures extend the treatment period, because the purulent cavity cannot be sufficiently well emptied due to a small opening in the thinned skin. Despite some improvement, which is sometimes accompanied by the onset of wound epithelization, the inflammatory process persists. The pressure on the nail fold yields a droplet of pus. If conservative therapy is continued, the course of paronychia becomes sluggish, alternating periodically with increase in the inflammatory response, manifesting by increase in pain in the affected finger.

Treatment in the first days of the disease *in infiltration stage* is ***conservative***. It is advisable to use soda baths, alcohol compresses or repeated dubbing with iodine solution. The following procedure is used: the nail fold is lifted carefully bloodlessly with the scalpel tip; a needle is introduced under the fold and the tissues underneath are irrigated with 3% H_2O_2 solution and antiseptic solution, after which a gauze strip with a hydrophilic ointment is introduced, filling the space under the fold with it. Local treatment with the ointment is repeated for 2-3 days.

If 3-4-day conservative treatment fails and the stage of *abscessing develops*, ***surgery*** is indicated. The operation is carried out under conduction anesthesia according to Oberst-Lukashevich, using Klapp's technique: the nail fold is circumcised at a distance of several millimeters from its edge, slightly shifted in the proximal direction; the nail root is cut off with scissors; then a tampon with a hydrophilic ointment is brought to the place of operation, the entire root of the nail should be removed because leaving at least a small part of it delays recovery.

Tendon felon is tendovaginitis or inflammation of the tendon sheath developing as a result of direct exposure to microorganisms or as a result of displacement of the inflammatory process. At tendovaginitis, the exudate of the tendon sheath compresses the vessels, which promotes their thrombosis and, as a result, leads to necrosis of the tendon. The tendon sheaths if the 2^{nd}, 3^{rd}, and 4^{th} fingers begin from the base of the nail phalanges and end with blind pouches 1-2 cm proximal to the head of metacarpal bone, which on the skin corresponds to the distal palmar fold. The tendon sheaths of the 1^{st} and 5^{th} fingers also begin blindly, are combined with bursae of the palm - radial and ulnar. Both bursae go through the carpal canal and end in the lower third of the forearm - in Pirogov's space - in the tissue above the quadrate pronator muscle.

Symptoms such as deterioration of the general condition, pulsating pain throughout the fingers, uniform swelling of the tissues with smoothing of the interphalangeal fissure are characteristic. The finger looks like a sausage. An attempt to extend the finger leads to sharp increase in pain, while bending significantly reduces its severity. This symptom is one of the cardinal signs of tendon felon.

Tendon felon is treated surgically, which consists in opening of the sheath and excision of its necrotized areas, treatment and drainage. Delay with operation is extremely dangerous. The tendon,

deprived of blood supply due to compression of the mesotendinium vessels with exudate, dies quickly. Late interventions lead to elimination of inflammatory focus, but the flexure function of the finger will be irreversibly lost.

Purulent tendovaginitis of the 1st and 5th fingers often leads to ***radial and ulnar tendobursitis***. Sharp pain is located not only in the fingers, but also in the corresponding side of the hand. The finger swells and is half-bent, the movements are limited by the pain. Investigation with a button probe causes pain along the tendon sheath of the finger and the lower third of the forearm, there is edema of the back of hand, the temperature rises, there is headache, sometimes chills. In tendovaginitis of the 1st finger, the pus can burst into the muscles elevating this finger, into the forearm, into Pirogov's space, from where it can spread to the ulnar bursa and cause cross-phlegmon. In tendovaginitis of the 5th finger, similar development of the purulent process can occur through the radial bursa.

Treatment of purulent tendovaginitis of the 1st and 5th fingers, as well as purulent processes of the forearm should be ***exclusively operative***.

Bone felon. *Primary bone felon*, which develops after infected punctured wounds with the damage to the periosteum or bone, and *secondary bone felon*, which occurs as a complication of poorly treated subcutaneous felon are distinguished. This is observed if the operation has not provided a sufficient outflow of the purulent discharge, which is the result of small "preserving" cuts, which produce the conditions for infection spread in the depth of the tissues, to the bone phalanx of the finger. In such cases, after opening the subcutaneous felon following a brief period of imaginary improvement, diminishing of the edema and pain, rapid recovery does not occur.

In the early stages, the symptoms of bone felon are the same as those with subcutaneous one, but they are expressed more pronouncedly. A characteristic symptom of bone felon is clubbing of the nail phalanx; palpation produces sharp pain. On investigation with a button probe, the tenderness is not delimited, it is noted throughout the phalanx. The general condition of the patient is deteriorated, the temperature rises to 39-40° C, there is headache, chills. X-ray investigation demonstrates destructive changes in the phalanges by the end of the 2nd - beginning of the 3rd week from the disease onset. The *operation* should be done in the early stages, without waiting for obvious destructive changes revealed by X-rays. The operation consists in opening of the boil, careful revision of the affected bone, removal of its necrotized fragments or the entire phalanx, lavage and drainage of the cavity.

Articular whitlow. Articular whitlow is purulent inflammation of the interphalangeal or metacarpophalangeal joints. This occurs following injury of the interphalangeal or metacarpophalangeal areas of the finger from their dorsal surface, where the joints are covered only with a thin layer of soft tissues. Infection easily spreads into the articular cavity through the wound canal, thus creating conditions for development of infection and progression of the pathological process. The inflamed joint acquires a spindle-like shape, the posterior interphalangeal fissures become smoothed. An attempt to flex or extend the fingertips leads to sharp increase in pain in the affected joint. There is local temperature increase. Swelling and tissue hyperemia are most pronounced on the posterior surface of the finger. Joint puncture gives a small amount of turbid fluid. Involvement of the ligament, cartilage and bone apparatus of the finger results in pathological mobility and feeling of crepitation of the rough parts of the articular surfaces.

In the first stage of articular whitlow, the joint puncture is indicated every other day with evacuation of the contents and antibiotics administration into the joint cavity. Careful immobilization is mandatory. If pus is present, arthrotome is performed. The joint is opened with two parallel incisions and washed with antibiotics. a rubber strip is introduced to the subcutaneous tissue to prevent adhesions.

In the second stage of the disease, when X-ray detects destruction of the articular surfaces, their resection is performed. At articular whitlow of the 1st finger, it is very important to maintain the finger mobility.

Pandactylitis is purulent inflammation of all the tissues of the finger. The clinical presentation of the disease consists of a combination of all types of purulent lesion of the finger. The course of pandactylitis is severe, accompanied by pronounced intoxication (headache, fever), regional

lymphadenitis, cubital and axillary lymphadenitis. The changes in the peripheral blood are characteristic of acute purulent inflammation.

The cause of pandactylitis is a highly virulent infection that penetrates into the tissue of the finger as a result of injury. Pain gradually worsens and becomes intensive, painful, bursting. The swollen finger acquires blue-crimson color. The inflammatory process develops in the form of wet or dry necrosis. Purulent inflammation involves the bone, joint, tendon and soft tissues. Multiple fistulas, through which necrotic tissue, bone sequesters and necrotized tendons are discharged, appear. The finger takes the wrong, ugly shape. It is enlarged in volume, swollen, its skin becomes cyanotic, the movements are absent.

Irrational therapy in the presence of virulent infection creates conditions for the inflammatory process spread on the hand. The condition of patients worsens: body temperature rises, pain arises in the hand, swelling and tissue hyperemia increase and spread proximally. Only immediate surgical intervention followed by active, purposeful and complex therapy can stop the progression of the purulent-inflammatory process. *Treatment of pandactylitis is exclusively operative.* Surgical intervention consists in removal of the finger.

Treatment. Felon is characterized by two stages of the inflammatory process: *serous-infiltrative and purulent.*

I*n the serous-infiltrative stage*, ***conservative anti-inflammatory therapy*** is used.

When the serous-infiltrative stage passes into the ***purulent***, basic diagnostic features are observed:

1) pulsating bursting pain;

2) slight whitening of the skin against a background of its widespread hyperemia in the center of the focus;

3) fluctuation over the boil.

Urgent surgical treatment is indicated - opening, lavage and drainage of the boil.

General principles to be observed when treating felon:

1) early immobilization;

2) treatment with antibiotics;

3) sufficient opening and lavage of the purulent focus;

4) anesthesia at opening the boil:

a) according to Lukashevich-Oberst;

b) conducting-infiltration according to O.V. Usoltseva

c) general anesthesia;

5) adequate drainage.

The following requirements are applied to *surgical approach*.

1. Taking into account of topographic and anatomical features:

a) the direction of the large vessels, nerves;

b) location of the tendons.

2. Ensuring the adequacy of lavage and drainage of the boil.

3. Preservation of the finger function.

Lateral and hockey-*stick surgical* incisions are used (Fig. 19).

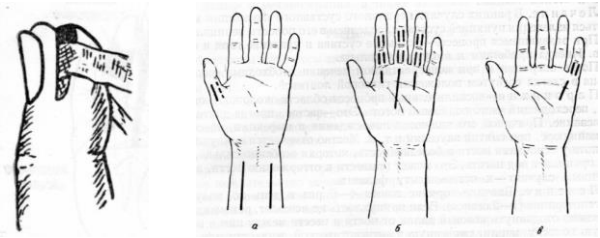


Fig. 19. Surgical access in the treatment of panaritium

The main directions of medical treatment:

1) antibiotic therapy;
2) detoxification therapy;
3) increase of organism resistance;
4) prevention of secondary infection (seroprevention when indicated).

Main directions of *local medical treatment*:

1) the use of antiseptics;
2) enzyme therapy;
3) creation of high local concentrations of antibiotics;
4) application of hyperosmolar solutions;
5) the use of agents stimulating wound healing.

Diagnostic and tactical errors are possible at felon treatment:

1) in establishing the correct diagnosis, the stage of the process and its volume;
2) in determining the terms of surgical treatment;
3) in the choice of the surgery volume;
4) in the choice of anesthesia methods;
5) due to the lack of an integrated approach to the postoperative period management;
6) due to underestimation of the concomitant pathology.

The following errors can be observed at *postoperative felon treatment:*

1) the use of gauze drains;
2) postoperative management not considering the phases of the wound process;
3) insufficient immobilization of the limbs;
4) errors of antibiotic therapy:
   a) the use of small and unreasonably high doses of drugs;
   b) prescription of antibiotics without taking into account the microbial flora sensitivity to them;
   c) the use of combinations of antibiotics without taking into account their interaction;
   d) underestimation of contraindications for antibiotic therapy;
5) mistakes of rehabilitation.

Main directions in ***felon prevention****:*

a) prevention of microtraumatism (safety rules);

b) prevention of infection at microtrauma (first medical aid in the first hours of the finger microtrauma).

Hand phlegmon

Hand phlegmon **(*Greek - phlegmon*)** *is an acute purulent inflammation of the tissue spaces of the hand.*

Pathogens. In recent years, powerful selective influence of antibacterial drugs have changed significantly the structure of acute purulent diseases of the hand. Staphylococci and gram-negative bacteria of *Enterobasteriaceae* family or a large group of so-called non-fermenting gram-negative bacteria occupied the first place. *Staphylococcus aureus* dominates in 69-90% of cases (less often in monoculture, more often in associations). In addition to *Staphylococcus aureus*, the association includes: *Staphylococcus epidermidis*, *Streptococcus pyogenes*, *Escherichia coli*, *Pneumococcus*, *Gonococcus*, *Salmonella thyphosa*. In some patients, purulent process on the hand becomes anaerobic non-clostridium phlegmon of the upper limb. The following clinically important groups of non-clclostridium anaerobes are distinguished: anaerobic nonspore-forming gram-negative bacilli (*Bacteroides et Fusobacterium*), anaerobic gram-positive cocci (*Peptococcus et Peptostreptococcus*), and gram-positive nonspore-forming bacilli (*Actinomyces, Propionibacterium, Eubacterium*). The main causes of hand phlegmon include trauma, microtrauma, wounds due to bites or clenched-fist injury (the latter is characterized by anaerobic and purulent infection).

The mechanism of hand phlegmon development (or pus spread) includes: - bursting of blind ends of the tendon sheaths of the 3rd and 4th fingers to the medial palmar space and the corresponding interdigital spaces, and from the interdigital spaces along the canals of the lumbrical muscles - into the deep portions of the palm and the posterior portion of the hand and vice versa; - along the tendon

sheath of the first and fifth fingers to the radial and ulnar bursae; - in length, directly from one tissue space (the palm or the back of the hand) to another, and further on the forearm as a result of destruction of fascia and aponeuroses forming these anatomical structures.

Classification of hand phlegmon. 1. Intermuscular phlegmon of the thenar (elevator of the 1st finger). 2. Intermuscular phlegmon of the hypothenar (elevator the 5th finger). 3. Interdigital (commissural) phlegmon (corn abscess). 4. Phlegmon of the medial palmar space (superficial or deep; or infratendinous and subtendinous, infra- and subaponeurosis). 5. Crossed or U-shaped phlegmon. 6. Subcutaneous phlegmon of the hand back (superficial as far as aponeurosis or aponeurosis is not damaged). 7. Subaponeurotic phlegmons of the hand back (in case of deep infection under aponeurosis, at stab wounds). 8. Furuncle, carbuncle of the hand back.

By infection spread:

1. *Combined* phlegmons of the hand - simultaneous affection of two or more tissue spaces.

2. *Associated* phlegmons of the hand (characterized by simultaneous presence of any phlegmon and any type of felon or at purulent pathology of the metacarpophalangeal joints).

3. *Total* phlegmons of the hand (the purulent process affects all tissue spaces).

By the inflammation stage.

1. *Initial or serous-infiltrative.*

2. *Purulent or purulent-necrotic.*

Clinical presentation and diagnosis of hand phlegmon

Diagnostic algorithm of hand phlegmon includes: - careful history taking (nature and remoteness of primary trauma or microtrauma, administered treatment, presence of comorbid condition); - evaluation of the results of objective examination (type of the affected finger, changes in the skin, location and severity of pain at point palpation with a button probe, presence of abnormal mobility in the joint or bone crepitation, etc.); - analysis of radiography findings.

Topical diagnosis of hand phlegmon is based primarily on revealing the signs of inflammatory changes in the area of the affected tissue spaces. Typically, local symptoms of hand phlegmon are accompanied by acute deterioration of the patient's health. The pain interferes with sleep, it is difficult to find a comfortable position for the affected limb. Diffuse purulent lesion of the tissue space has typical signs depending on the location. *Local signs* of the inflammatory process include swelling and hyperemia of the tissues, dysfunction of the hand, local temperature increase, pain on palpation. The degree of these symptoms varies and depends on the prevalence of inflammatory process, the pathogen virulence, protective organism response, its immunobiological reactivity, etc. Therefore, the clinical course of hand phlegmon is quite diverse: from simple clearly located forms of inflammation to large, prone to spreading purulent-necrotic processes accompanied by severe intoxication.

Phlegmon of abductor pollicis muscle is accompanied by acute edema of the thenar and the radial edge of the back surface of the hand. The thumb is slightly thickened and bent, the movements in it are painful. Sharp pain on palpation, tissue tension, and palatal skin folds are characteristic symptoms of inflammation of the thenar adipose tissue. Purulent exudate often extends along the edge of the first intramuscular muscle to the dorsal surface of the hand. In some cases there is purulent melting of the connective tissues septum separating the thenar cleft and the medial palmar space with infection of the latter and formation of purulent necrotic process in the middle palmar fossa.

Phlegmons of hypothenar are not accompanied by the phenomena of pronounced intoxication. Moderate swelling, hyperemia and tissue tension, pain at palpation are typical. Movements of the 5th finger increase the pain. There is swelling of the hand along the medial margin.

Commissural phlegmon is located in the distal part of the palm. The infection enters through the fissures of the coarse, callus skin in the area of the fetlock joint of the palm. Hence the other name of such phlegmon, corn abscesses. The inflammatory focus is formed, as a rule, in the commissural spaces of the 2nd-4th fingers. Phlegmons are accompanied by significant pain, swelling of the distal part of both surfaces of the hand. The fingers adjacent to the purulent focus are slightly separated and bent in interphalangeal joints. Extension is painful due to the tension of the inflamed palmar aponeurosis. Proliferation of the pus through the oval fissures of aponeurosis from the palmar to the back surface of the hand is possible. In addition, involvement by the process of the tendon of the deep

finger flexor located closely to the purulent-necrotic focus is also possible. The infection may also spread proximally through the channels of the lumbrical muscles. In these cases, the inflammation of the medial palmar space joints to the main focus.

Phlegmons of the medial palmar space are characterized by tension and tenderness of the palmar surface of the hand with edema of the back side. The fingers are slightly bent, their movements, especially extension, are sharply painful. In this type of phlegmon, the purulent exudate accumulates between the palmar aponeurosis and a thin fascial plate that covers the tendons of the fingers - subaponeurotic phlegmon of the medial palmar space. The purulent process between the fascia lining the interosseal muscles from the palmar side and the back surface of the long flexor tendons leads to formation of subtendinous phlegmon. It is very difficult to differentiate these diseases. The location of the purulent exudate and its distribution can be assessed correctly only during surgical intervention. Therefore, it is more appropriate to call the purulent-inflammatory foci in the central part of the palm *phlegmons of the medial palmar space*. The inflammatory process of this location is accompanied by elevation in the body temperature, headaches, changes in the peripheral white blood. The central part of the palm bulges. The skin with the smoothed folds is tense, fluctuation cannot be determined. Palpation of the inflammation focus produces severe pain. Edema of the hand back is significant; the 2^{nd} - 5^{th} fingers are bent in interphalangeal joints. An attempt of active or passive extension leads to tension of the infiltrated palmar aponeurosis and, as a result, to the pain aggravation. Late and inadequate treatment is complicated by the pus bursting into the thenar fissure, as well as proliferation of the accumulated pus by the canals of the lumbrical muscles to the back of the hand.

Crossed or U-shaped phlegmon is the most severe form of purulent inflammation of the hand. This is a combined injury of the palm bursae - ulnar and radial. The disease is a consequence (complication) of purulent tendovaginitis of the 1^{st} or 5^{th} finger. Under favorable conditions of the infection, the purulent exudate spreads to the bursa of the radial or ulnar side of the hand. Weak nonspecific immunobiological capabilities of the organism as well as late, inadequate treatment contribute to generalization of the infection and its transition from the proximal part of the palm to the opposite side. There is purulent inflammation of both bursae. Bursting and rapid spread of the pus are easier in case of direct coupling of the bursae in the metacarpal canal. Clinical observations indicate that the cause of crossed phlegmons is radial tenobursitis.

U-shaped phlegmons are accompanied by severe intoxication, elevated body temperature, headache, weakness. The hand is swollen blue-crimson, its palpation is extremely painful. The fingers are slightly bent to the palm, active movements in them are absent. An attempt of passive extension considerably aggravates the pain. Palpation reveals the most pronounced pain in the projection of the flexor tendons of the 1^{st} and 5^{th} fingers and in the proximal portion of the hand, that is, at the location of the blind ends of the ulnar and radial bursae. Bursting of the pus into Pirogov's space results in generalized pain and edema in the distal part of the forearm. The danger of U-shaped phlegmon consists in the fact that the purulent-inflammatory process can affect all facsial spaces of the hand: the medial palmar space at pus bursting from the ulnar or radial bursae, thenar fissure or hypothenar at purulent tenobursitis of the 1^{st} or 5^{th} fingers. Later the pus invades the back surface of the hand through the channels of the lumbrical muscles, creating a large purulent necrotic center. A real threat of the inflammatory process generalization is created. Even with the most favorable course of U-shaped phlegmon, in the late postoperative period the function of the hand is significantly reduced.

Subcutaneous phlegmon of the hand back is considered one of the mildest forms of purulent inflammation of the hand fatty tissue. Swelling and hyperemia of tissues are generalized, the boundaries of the purulent focus are difficult to establish. Careful palpation of the tissues reveals the focus of the tissue purulent softening.

Subaponeurotic phlegmons of the hand back develop as a result of infection invasion to the deep areas under the aponeurosis at stab wounds. Compared with the previous type of inflammation, the manifestations of subaponeurotic phlegmons are more distinct. Dense infiltration is determined, it is accompanied by edema and hyperemia of the hand back. In some cases, purulent lesions of the hand back may be secondary because the lymph vessels of the palmar surface carry lymph to the hand back, and therefore, at purulent lesions of the palmar surface the infection can be carried to its back surface.

Advance of the purulent exudate from the palm to the back may also occur through the canals of the lumbrical muscles and in these cases, to the hand back, which accompanies the inflammation of the palmar surface. Skin hyperemia develops and generalized pain occurs at palpation of the hand back.

Furuncle and carbuncle of the hand. On the back surface of the hand and fingers there is a significant amount of hair follicles, therefore furuncle or carbuncle can develop on the hand. There is no fundamental difference in the occurrence, development and course of furuncle (carbuncle) of the hand and inflammation of the hair follicles of any other location. Swelling, hyperemia and sharp pain at palpation, presence of necrotic content are symptoms of furuncle. At carbuncle, these symptoms are more pronounced, there are several necrotic rods, the general condition of the patient is aggravated, often characterized by intoxication (headache, weakness, elevated body temperature), regional lymphadenitis and lymphangitis.

Treatment of hand phlegmon. The main goal of the treatment is considered to be complete and steady control of the inflammatory phenomena with minimization of functional and esthetic negative consequences, and in some cases, the risk of fatal outcome.

All patients with phlegmons should be hospitalized. The diagnosis of acute purulent disease of the hand is an absolute indication for surgical treatment. Any attempt of conservative treatment is dangerous due to the risk of chromizing the process and its spread to deeper structures. Operative intervention and postoperative period, until the control of acute inflammatory phenomena, should be conducted in a hospital setting.

Surgical treatment. Preoperative preparation consists in washing the affected hand with warm water and soap. 30-40 minutes before the operation intramuscular administration of a wide-spectrum antibiotic is necessary, which limits the infection spread and promotes a more favorable postoperative course.

Treatment of purulent diseases of the hand consists of certain stages, the failure to fulfill which will complicate the course of the inflammatory process. To perform surgical interventions on the hand the method of choice is blockade of the radial, ulnar and medial nerves at the level of the radioulnar joint. Anesthesia is performed with 2% lidocaine solution or 1.5% trimecaine solution. The proximal spread of the inflammatory process to the area of the wrist joint excludes the possibility of using this method, and in these cases, good anesthesia is achieved with intravenous regional anesthesia under the tourniquet, axillary anesthesia, or blockade according to Kulenkampf. In case of contraindications, general anesthesia is performed.

Operative intervention in patients with purulent-inflammatory diseases of the hand should be performed at complete circulation control of the surgical field, which allows to determine the boundaries of necrosis and to differentiate the anatomical structures, to orient in the process spread, to perform radical necrectomy and to preserve important anatomical structures. When the process involves the main phalanx or the hand, the tourniquet is applied on the forearm.

The choice of approach to the purulent focus is an important aspect of surgical treatment. Skin incision should be carried out on less significant "non-working" surfaces of the fingers and hand. A scalpel should be used to cut only the skin; all further manipulations on the tissues are performed by "pushing" the tissues under visual control. After pus evacuation and revision, necrectomy is performed, its adequacy depends to a large extent on the surgeon's experience. Even small areas of non-viable tissues left after necrectomy can be the cause of prolonged course of the disease and repeated operations. At the same time, excessive radicalism in this complex anatomical region can cause irreversible anatomical and esthetic defects.

In operations on the hand, as well as on the fingers, eye instruments (scalpel, sharp scissors) should be used. This allows adequate incisions, to treat the viable tissues with care, to make it easy to manipulate in the wound, to completely remove necrotic tissues, to provide conditions for rapid healing of the wound and to restore the function of the organ.

Main stages of the operation for hand phlegmon

1. The task of the surgeon is to preserve the function of the hand. Before incision on the hand it is necessary to think about the zone and character of the scar, to what extent it will affect the hand function. The incision is made taking into account Langer's lines, which correspond to natural skin

folds. Large longitudinal incisions are not acceptable. The surgical approach should be as short and preserving as possible. A scalpel is only used to cut the skin. All further manipulations on the tissues are performed with the use of forceps and hooks, which allows visualization and preserving all important functional structures (vessels, nerves, tendons). The presence of a surgeon assistant is obligatory.

2. The next stage of the operation is thorough necrectomy, in which the purulent focus is to be excised as a primary surgical treatment. During necrectomy, the vessels and nerves are skeletonized. The affected tendon is not resected if it is possible to remove individual necrotic fibers. Necrectomy of the bone and articular structures should include removal of only sequestered sites. Interventions on the joints in purulent arthritis or osteoarthritis should be performed in the postoperative period in distraction mode, which is often provided by traction with a modified Kirschner needle or using a special device.

3. After necrectomy and hemostasis, each tissue space is drained with a separate perforated polychlorvynil tube, which is fixed to the skin by a separate suture. After the intervention on the joints and tendon sheaths, these structures require additional drainage. The wounds are treated with antiseptic, vacuumed and treated with low frequency ultrasound in antibiotic solution.

4. Radical necrectomy and adequate drainage of the residual purulent cavity allows to complete the operation by applying primary sutures to the wound. Wounds are sutured with atraumatic 3\0-5\0 threads. In severe lesions, the use of micro-irrigators and partial wound suturing are supplemented by gauze bandages soaked with ointment on a hydrophilic base.

5. If there is no possibility of one-time suturing of the skin defect, it is necessary to use the adequate type of skin plastics. In cases of bare tendons or bones, it is possible to use non-free skin plastics of Italian type - crossed or with a flap on vasculonervous stalk. It is better to cover granular defects with free cutaneous graft. All plastic operations are performed after control of acute purulent inflammation, but at the earliest possible terms.

6. Proper immobilization with the of measures preventing the skin maceration. Terms of immobilization of the operated hand are limited to the relief of acute inflammatory processes (phenomena).

7. Within the postoperative period, along with the regular treatment of wounds, antibacterial and anti-inflammatory therapy, physical therapy procedures, medical physical training are administered during the wound dressing. Early active workup of the finger and hand movements (after removing drainage and sutures) contribute to a more complete restoration of the hand function.

Treatment of some forms of hand phlegmon

Treatment of interdigital (commissural) phlegmon. When the purulent process affects one web space on the palmar surface of the hand, arciform incision according to Bannel is performed at the level of heads of metacarpals. Contralateral incision is done on the hand back, in the projection of the corresponding space. The wounds are interconnected and drained through the perforated microirrigator with placement of primary sutures. When two or three spaces are involved, one arciform incision of the skin is performed on the palmar side of the hand parallel to the distal transverse fold. On the hand back separate incisions are performed similar to those at the lesion of one web space, but their number corresponds to the number of the spaces affected by the purulent process. All posterior wounds are connected with an incision on the palmar surface. A microirrigator is placed introduced trough each web space, and one more tube is laid at the bottom of the palmar wound in a transverse direction.

Treatment of phlegmon in the thenar area. Surgical approach is an arciform incision measuring up to 4 cm, conducted parallel to the skin fold of the thenar and somewhat outward away from it. Precautions should be taken when performing the proximal part of the incision, in the so-called "forbidden zone", where the medial nerve motor branch of the median nerve goes to the thumb muscles. Damage to it leads to loss of mobility in the finger. At the hand back in the zone of the 1st web space, a contralateral arciform incision is made. When performing necrectomy and treatment of the wound, the cavity is drained with two perforated tubes, one of which is placed along the inner edge of the thenar area, and the other - along the main incision on the palmar side of the hand.

Treatment of hypothenar phlegmon. Linear- arciform incision is done along the inner edge of hypothenar muscle elevation. The posterior contralateral incision corresponds to the outer edge of the 5th metacarpal bone. After completing the main manipulations in the purulent focus, the wounds are interconnected. Drainage is done with two tubes, one of which placed along the inner edge of the facsial bed of the hypothenar, and the other along the main incision.

Treatment of subaponeurotic palmar phlegmon. The following are the best approaches:

1) Banell arciform incision, made from the 2nd web space at the level of the distal transverse fold parallel and mediad the fold of the thenar to the distal edge of the wrist (a fragment of this approach may be used);

2) arciform incisions, parallel to the distal or proximal transverse palmar crease (Zoltan).

Treatment of phlegmon of the medial palmar space. To open the phlegmon of the medial palmar space, the method of choice is Zoltan modified approach. The incision starts from the 4th web space parallel to the distal transverse skin fold to the 2nd web space, then to the proximal transverse fold, from which proximally along the thenar fold to the "forbidden zone". Mobilization of the formed piece together with the tissue (to preserve its blood supply) provides approach to almost all tissue spaces of the palmar surface, which creates conditions for complete and extensive necrectomy.

In the presence of a large wound in the zone of the planned incision (after a primary injury or surgery in other treatment facilities), the risk of ischemia and subsequent necrosis increases. In these cases, incision similar to that described above, but mirrored with respect to the longitudinal axis of the hand (on the opposite side of the hand, where there is no wound defect), is performed.

In case of significant skin damage in the central part of the palm, an arciform medial incision is performed along the axial line of the hand starting from the 2nd web space and ending with the proximal edge of the projection of the flexor muscle holder. retractor of flexor muscles.

Regardless of the selected approach (skin dissection), dissection of the palmar aponeurosis is carried out in the longitudinal direction and necrectomy is performed with the movement inward the tissues. Revision of the flexor tendons and subtendinous (deep) space is necessary for assessing their condition and identifying possible suppurated edemas.

Following necrectomy, drainage is performed with three or four microirrigators: two or three tubes (depending on the process dissemination) are placed under the palmar aponeurosis, further - under the transverse ligament of the palm and brought out through additional punctures at the level of the distal fold of the region of the wrist joint and in two or three (corresponding to the number of drainages) web spaces. One more microirrigator is passed under the flexor tendons in a transverse direction and brought out through additional punctures. After drains placement, integrity of palmar aponeurosis (atraumatic suture material 3/0 - 4/0) is restored.

Treatment of phlegmon in the hand back. The phlegmon is opened with several arciform small (up to 3.0 cm) incisions along Langer's lines along the perimeter of the purulent cavity. The portals of entry are subject to surgical treatment and can be used as one of the approaches. To drain the formed cavity, two microirrigators are placed along the lateral and medial edges in the longitudinal direction; they are brought out through additional punctures. Primary sutures are indicated only with full confidence in the tissue viability. In case of skin defects after necretomy or in case of skin ischemia on the hand back, the wound is loosely filled with gauze strips with ointment on a water-soluble base.

Treatment of U-shaped phlegmon of the hand and Pirogov-Paroni space. Operative intervention in U-like phlegmon begins with unilateral longitudinal lateral incisions along the "non-working" surfaces of the middle phalanx of the 5th finger and the main phalanx of the 1st finger, from which the corresponding tendon sheathes are opened; longitudinal lateral incisions in the lower third of the forearm are made to open Pirogov-Paroni space. Perforated 1.0 mm microirrigators are passed with the help of the guide line from a set for subclavian vein catheterization through the open lumen of the tendon sheaths of the 1st and 5th fingers in the proximal direction. Their ends are placed in Pirogov-Paroni space.

The next stage of the operation is incisions in the area of the thenar and the hypothenar similar to those in isolated phlegmons of the above spaces. Revision of the flexor tendons of the 1st and 5th fingers and their sheaths is possible all over.

After washing the sheaths with antiseptic solution, necrectomy in all wounds, vacuuming and ultrasonic treatment, each of the purulent process of tissue spaces (thenar, hypothenar, and Pirogov-Paroni) is drained with perforated in the middle part polychlorvynil drainage tubes.

Treatment of combined phlegmon. To dissect several spaces on the palmar surface, Zoltan modified approach is used. In case of damage to the medial palmar space and the thenar area, the incision is made parallel or along the distal skin fold of the palm using arciform continuation along the thenar border into the proximal parts of the hand to the wrist level. With damage to the medial palmar space and hypothenar area, similar approach is used, but it is rotated by 180 ° around the longitudinal axis of the palm. Simultaneous suppurative process of one or several web spaces does not require additional incisions. The existing purulent edemas on the hand back are opened using several arciform incisions in accordance with Langer's lines. These approaches are contraindicated at significant wound defects in the area of the medial palmar space due to the risk of necrosis of the mobilized cutaneous-subcutaneous tissue. In these cases, a T-shaped incision is used, its transverse portion carried out in parallel or along the distal fold of the palm, and the longitudinal one - from its middle portion in an arciform manner arcuate through the present wound to the level of the wrist. This approach because of its longitudinal part is less physiological than the described above, but when used in patients with primary wounds in the center of the palmar surface, the risk of skin necrosis is reduced almost to zero.

When the purulent process involves Pirogov-Paroni space, each of the above approaches should be extended to the level of the distal skin fold of the region of the wrist joint, then - along the fold to the radius edge of the lower third of the forearm, and is completed with its longitudinal incision to open the phlegmon of Pirogov's space.

In hand phlegmons with the pus spread to the forearm tissue above the quadrate pronator, arciform Canavel approach extended on the forearm is appropriate.

Adequate drainage of the postoperative residual cavities on the palm is usually promoted by two or three perforated tubes, placed along the edges of the corresponding cellular spaces. The involved interdigital spaces and the hand back are always drained separately.

When the radical character of necrectomy is assured, primary sutures are placed. The tissue remaining in the wounds are diffusely soaked with pus ("honeycombs"), and areas of the skin of doubtful viability are contraindications to wound suturing. In these cases, the remainder tissues are covered with gauze strips impregnated with ointment on a water-soluble base.

When treating total phlegmons (simultaneous involvement of all tissue spaces of the hand), the above approaches are used. Total phlegmons are characterized by rapid development of skin necrosis on the hand back, which is diagnosed, as a rule, when the patient is admitted to the hospital. In these cases, an arciform incision is applied through the necrosis zone with the excision of the latter.

The peculiarity of surgical treatment of total phlegmon (wide involvement, diffuse purulent imbibing of the tissue, absence of clear boundaries of necrosis, and unfavorable therapeutic background) consists in impossibility of one-time radical necrectomy during the first operation. This determines completion of the first stage of the operation – primary suture is not placed of the wound. All tissue spaces are loosely tamponed with gauze strips soaked with ointment on a water-soluble base. On the following days, dressings and staged necrectomy under anesthesia are performed in the operating room. Treatment of total phlegmon ends with control of acute inflammation and the wound closure by secondary sutures or skin plastics (Italian).

Treatment of combined hand phlegmon (CHP). Surgical approach at CHP should provide revision of the finger structures and involved tissue spaces, preserving the integrity of the vasculonervous bundles and minimizing functional damage. Therefore, in CHP, two variants of surgical approach regardless of the type of the phlegmon are used. When the process is located on the dorsal surfaces of the fingers and the hand, the incision is performed on the lateral neutral line of the affected finger with arched transition to the hand back. When the palmar surface of the finger and the hand is involved, the incision along the lateral neutral line of the respective finger is considered optimal, but it is performed with an arched transition to the area of the corresponding finger-palmar elevation; the affected tissue spaces on the palm are exposed with S-shaped extension of the existing

palmar incision in the proximal direction. Purulent edemas on the hand back are opened by arciform incisions along Langer lines. The existing purulent wounds are excised economically, possibly involving them to the main approach. After necrectomy and treatment of the focus, all involved anatomical structures and tissue spaces are drained with thin perforated polychlorvynil tubes.

An adequate drainage is achieved using the drainage-irrigation algorithm, namely:

1) minimum number of drainages should provide drainage of residual cavities both on the fingers and on the hand;

2) bursae and tendon sheaths should be drained separately if their integrity is preserved;

3) in cases of destruction of the sheath or bursa it is enough to install one or two drainages in the subcutaneous tissue - to place them along the "bared" tendons;

4) joint cavities are drained separately after intervention for osteoarthritis or arthritis (in the interphalangeal joints, the microirrigators are placed transversely, and in the fetlock joints - sagittally).

The algorithm of postoperative management of patients with CHP:

1) at CHP with joint involvement, postoperative period is managed in distraction mode (with impossibility of imposing a distraction apparatus in conditions of phlegmonous inflammation of the soft tissues, a needle structure or device for distraction of the fetlock joints is used);

2) primary suture is placed only on the viable tissues;

3) large wounds (up to 1.5 cm in width and over) require secondary sutures (small open wounds - up to 1.5 cm in length and up to 0.5 cm width, heal rapidly by second intention);

4) in large wound defects, inflammation control is followed by different variants of skin plastics;

5) open wound managing is considered a method of choice (it provides better conditions for treatment and drainage, and allows visual control of the wound process course).

Methods of prevention *of purulent diseases of fingers and hand* include reduction of industrial and domestic injuries; - complete treatment of skin injuries and timely primary surgical treatment of the wound.

Prognosis. Timely and complete treatment of purulent diseases of the fingers and the hand promotes a favorable life prognosis (however, the death rate in anaerobic phlegmons of the upper extremities exceed 20%).

Purulent mastitis

Mastitis *is an acute breast inflammation.*

Lactation mastitis (LM) occupies one of the leading places (26-67%) among postpartum purulent inflammatory complications and develops in 2.4-18.0% of new mothers. It frequently occurs after the first childbirth, after the second one it develops in 20% of cases, after the third one – in solitary cases. LM makes 95% of all acute inflammatory diseases of the breast. It occupies one of the leading places among postpartum purulent inflammatory complications. Non-lactation mastitis is rare. Ages of 15-50 are more common. The disease is not associated with lactation. The infection enters the breast tissue through the ducts or the damaged skin. Hematogenic and lymphogenic infection is also possible. Main causative agent is *Staphilococcus aureus* as a monoculture or associated with other pathogenic microorganisms (E. coli, streptococci, ptoteus). Chronic mastitis may occur at any age.

In the majority of cases it develops as a consequence of acute purulent inflammation in the breast or injections to the breast tissue as a dense infiltration with small abscesses separated by a fibrous thick-walled capsule. Less frequent cause of chronic mastitis is breast abscess bursting through the skin with fistula formation. In chronic mastitis, pus culture demonstrates polyresistant to antibiotics *Staphylococcus aureus or E. coli.* Newborn mastitis develops on week 2-3 of life both in girls and boys against a background of physiological swelling of the breast due to mother’s steroid hormones (estrogens) in the infant’s blood.

The infection enters through the damaged skin or with the blood. The disease is often preceded by intertrigo, various purulent inflammatory processes of the skin and deep tissues.

Depending on the functional state of the breast and the peculiarities of the inflammatory process, *lactation and non-lactation mastitis* are distinguished.

Mastitis classification

I. *Depending of the origin*:
1. Lactation
2. Non-lactation

II. *Depending on the disease course*
1. Acute
2. Chronic

III. Depending on the inflammation character
1.Non-purulent:
- serous
- infiltrative

2. Purulent:
- abscessing
- infiltration-abscessing
- phlegmonous
- gangrenous

IV. Depending on the affected side
1. Left
2. Right
3. Bilateral

V. *Depending on the boil location in the breast (*Fig. 20*)*
1. subareaolar
2. intraductal
3. intramammary
4. retromammary

VI. Depending on the process dissemination:
1. Limited (1 quadrant)
2. Diffuse (2-3 quadrants)
3. Total (4 quadrants).

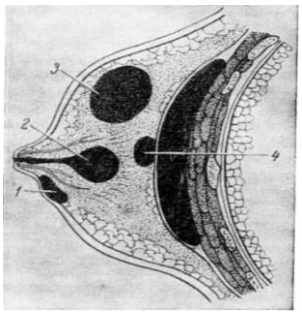


Fig. 20. Types of acute mastitis:
1. subareaolar; 2. intraductal;
3. intramammary; 4. retromammary

Main etiological factors.

1. Microinjury of the nipples (fissures and excoriation, damage to the breast skin, frequently developing in breast-feeding mothers after the first childbirth).

2. Lactostasis (milk congestion in the breast):

1) *objective causes*:
- lack of movement or nipple fissures;
- mastopathy;
- breast tissue scarring after injury or surgery;
- thin long and curved ducts;
- other congenital and developed changes in the breast which violate milk outflow;

2) *subjective causes:*
- violations of breastfeeding regimen
- insufficient or irregular milk expression after feeding, violations of the expression technique.

The breast can be infected through endogenic or exogenous routes, exogenous being more frequent. The infection enters through the nipple fissures (50% of cases), abrasions, eczemas, small wounds which occur at breast-feeding. The source of infection is disputable, but it is considered that the most common source of infection is the newborn, who passes the infection to the mother at feeding. Endogenic infections are frequently lymphogenic, but sometimes may be galactogenic or hematogenic.

In 85% of cases the disease is preceded by lactostasis. in the majority of patients it is not longer than 3-4 days. Combination of lactostasis and infection with pyogenic microorganisms is the main cause of mastitis development and progression. Lactostasis in this case triggers the process.

At incomplete milk expression, a considerable number of microorganisms remain in the ducts, they cause lactic fermentation, milk folding and ductal epithelium damage. Saur milk occludes the ducts causing lactostasis.

The amount of microflora which continues to develop in the closed space reaches the "critical level" and causes inflammation. Venous blood and lymph outflow are affected simultaneously. The edema of the interstitial tissue enlarges, compresses the ducts of the adjacent lobes resulting in lactostasis progression and inflammation.

15% of patients with purulent mastitis have nipple fissures which are caused by discrepancy between the redundant negative pressure in the oral cavity of the child and elasticity of the nipple tissue. Nipple fissures are caused by frequent and continuous contact of the nipple with the milk-wetted bra causing irritation and maceration of the skin, lack of movement and nipple erection; violations in the time of feeding. The above leads to breast dysfunction; the women have to refuse from feeding with this breast and thorough expression. Therefore, with the purpose to prevent mastitis it is necessary to observe a definite rhythm of feeding and expression.

Lactation mastitis development is also influenced by toxicosis of the first of second half of pregnancy, anemia, nephropathy, miscarriage danger.

A definite role in LM pathogenesis is played by the organism sensitization to drugs, staphylococcus, autoimmune reactions to organ-specific antigens (mild and breast tissue). A definite role in LM development is played by disorders of kallikrein-kinin system.

Staphylococcus aureus plays the main role in mastitis development. It is demonstrated in 97% of cases in blood and milk samples. These strains are characterized by pronounced pathogenicity and resistance to many antibacterial drugs. Such components of staphylococcus aureus as protein A and teichoic acid produce considerable immunodepressive effect. In the rest of cases, mastitis can be caused by epidermal staphylococcus, e. coli, streptococcus, enterococcus, proteus, pseudomonas aeruginosa.

The risk group of LM includes the women:

1) With the history of pyoseptic diseases;
2) With mastopathy
3) With breast and nipple development anomalies
4) With history of breast injury or surgery
5) Prone to fissures of the skin
6) With pathological premenstrual syndrome accompanied by diffuse enlargement and tenderness of the breast in the 2nd phase of the menstrual cycle;
7) who were administered oxytocin or prostaglandin due to weak labor activity (in this group the milk comes late in large amounts)
8) With pathology of pregnancy, delivery and postpartum period.

LM development is also influenced by the following:

1. Decreased immunological reactivity of the organism. Protein- and carbohydrate poor diet reduces the organism resistance to infections. The daily diet of the pregnant should include about 60-70% of animal proteins. To increase immune activity, vitamins A, C, D are necessary. The pregnant and feeding mothers require good rest and walk in the fresh air (2-3 hours a day, including before sleep), 10 hours of sleep a day. Smoking and alcohol are incompatible with pregnancy and postpartum period. A favorable psychoemotional atmosphere is necessary, because it also influences the state of the immune system.

2. Violation of the personal hygiene. Pregnant and breast-feeding need a warm shower not less than 2 times a day (in the morning and in the evening) and have to change underwear. The breasts require a special care. In pregnancy period it is necessary to wash them with water and rub with a clean bath towel. This promotes tempering and increases resistance of the nipples to mechanical injuries, which can develop at breast-feeding. Daily 15-20 min air bathes for the breasts are necessary in the second half of pregnancy: in summer under the sun near an open window, in winter – combined with small doses of UVI.

3. Redundant negative pressure in the oral cavity of the child during feeding is the basic factor of nipple fissure development. To prevent this complication, a tender pressing with two fingers of the newborn cheeks near the mouth angles is recommended. It is necessary to observe feeding technique and not to keep the infant near the breast for a long time. If the infant sucks drowsily and slowly, short breaks are necessary. After feeding, the breasts should be washed with warm water without soap, dried with a soft towel and left open for 10-15 min. It is necessary to place sterile gauze between the bra and areole, which is changed on wetting with milk. Lotions, creams and other remedies should not be used for hygiene of the breast and body.

4. Creation of cracked nipples during feeding. To treat cracks successfully, it is necessary, first of all, to temporarily stop breast-feeding, to ensure that there is no prolonged contact with of the milk with the crack. The milk is expressed with hands in sterile dishes, the baby is fed from a bottle, through a teat, in which a small hole is made with a warmed-up sewing needle. If the hole is large, the child will refuse to take the breast. To treat the nipples, sea buckthorn or rosehip oil, solcoseril ointment (applied to the affected area on a sterile gauze napkin) are used.

Prevention of lactostasis. To prevent lactostasis the following measures are recommended.

1. Special medical supervision is subject to: all primapara; women with pregnancy or childbirth pathology; women with anatomical changes in the breast.

2. Tight bandage of the breast, which is used to control lactation is forbidden (tight banding is extremely dangerous, as milk production continues for some time and there is always lactostasis, and disturbance of blood circulation in the breast leads to development of severe purulent forms of mastitis).

3. The bra should be made from cotton fabric (synthetic linen irritates the nipples and can lead to cracks). The bra should support well, but not squeeze the breast. It should be washed daily (separate from other linen) and worn after hot ironing.

4. The physiological mechanisms that stimulate the lactation should be taken into account. Early breast-feeding (in the first 30 minutes after birth) activates prolactin release to the blood and stimulates milk production. It is possible to use circular douche on the breast 20 minutes before feeding. To follow the correct technology of milk expression (manual method is most effective in lactostasis prevention). Particular attention should be paid to milk expression from the external quadrants of the gland, where lactostasis and purulent inflammation occur more frequently.

Differences in the course of inflammatory process in mastitis from that in acute suppurative surgical infection of another location is associated with postpartum increase in the functional activity and the characteristics of the breast anatomical structure.

Characteristics of the breast anatomy: lobar structure, a large number of natural cavities (alveoli and sinuses), a wide network of milk and lymphatic ducts, excess of fatty tissue.

In acute mastitis ***two stages of the inflammatory process*** are distinguished: *non-purulent* (*serous and infiltrative forms*) and *purulent (abscess, infiltrative-abscess, phlegmonous and gangrenous forms).*

Acute inflammatory process begins with accumulation of serous exudate in the intercellular spaces and leukocyte infiltration. At this stage, the process is still reversible. However, inflammation is poorly restricted and has a tendency to spread to the adjacent breast areas. Serous and infiltrative LM quickly becomes purulent with simultaneous involvement of new parts of the gland tissue. Purulent inflammatory process is more often intramammary, with involvement of two or more quadrants, the course is often protracted with frequent relapses. Infiltrative-abscess and phlegmonous are the most common forms of purulent mastitis.

In 10% of cases, LM has a latent course due to prolonged antibiotic therapy in abscess or infiltrative-abscess forms.

In some cases, breast gangrene develops as a local manifestation of the organism autosensitization to organ-specific antigens (milk and inflamed gland tissue). In this case the inflammatory process proceeds especially malignantly, with a large skin necrosis and a rapid spread to the tissue spaces of the chest.

Purulent mastitis is always accompanied by regional lymphadenitis.

Clinical presentation of acute purulent mastitis (PM) depends on the form of the inflammatory process. As indicated above, the following forms are distinguished:

1) serous (initial);
2) infiltrative;
3) abscess;
4) infiltrative-abscess;
5) phlegmonous;
6) gangrenous.

Serous (initial) form is widespread in surgical practice. This form is characterized by inflammatory exudate formation without any focal changes in the tissues of the gland. The disease begins acutely with pain, a sense of heaviness in the breast, chills, fever to 38° C and above. On examination the breast is enlarged, there is a slight skin hyperemia in the inflammation zone. Palpation in the area of hyperemia is painful. The quantity of expressed milk is reduced. Blood count shows moderate leukocytosis and increased ESR. Microscopy demonstrates visible clusters of leukocytes around blood vessels. With a favorable course of the disease, the serous form may be abortive; with inadequate and ineffective treatment, this form progresses with development of the subsequent phases and complications.

Infiltrative form of mastitis is continuation of the first one and may be its short manifestation. It usually proceeds in an aseptic variant, and in case of inadequate treatment it turns into various purulent complications. The patients present with the same complaints as in serous form, the above symptoms persist, but in the tissues of the gland painful infiltrate without clear boundaries, areas of softening and fluctuations is determined. The body high temperature and chills, both in serous and infiltrative forms, are caused by lactostasis, in which the milk that has pyrogenic effect, is absorbed into the blood through the damaged milk ducts. Desensitizing therapy and lactostasis control reduce the body temperature to 37.5° C in the majority of patients. If the treatment is absent or is inadequate, serous and infiltrative forms of mastitis become purulent in 3-4 days.

A*bscess form* is characterized by appearance of softening and melting focus with formation of a delimited purulent cavity. With this form the condition of the patient gets worse, general and local symptoms become more pronounced, intoxication progresses; the body temperature rises above 38° C; swelling and hyperemia of the breast skin increase. On examination, a sharply painful infiltrate (abscess), delimited by a pyogenic capsule can be palpated in the breast; in 50% of patients it occupies more than one quadrant; in 60% the abscess is located inside the breast, rarely under the areola or skin; in 99% fluctuation sign is positive; there is often a softening area in the center of the infiltrate.

The course of infiltrative-abscessing form of mastitis is more severe than abscess form. This is characterized by increase in the body temperature to 38° C and above, severe hyperemia, edema, pain and tenderness on palpation; there is a dense infiltrate consisting of a number of small abscesses of different sizes of honey-comb type (thus fluctuation sign is positive in 5% of cases) in the tissues of the gland. In 50% the infiltrate takes no more than two quadrants of the gland and is located inside the breast.

Phlegmonous form is characterized by deterioration of the general condition and marked signs of intoxication. The pain in the breast intensifies, weakness increases, appetite decreases, pallor of the skin is observed, the body temperature ranges from 38° C (in 80% of patients) to more than 39° C (in 20%). On examination, the breast is sharply enlarged, swollen, hyperemia of the skin is pronounced, sometimes with a cyanotic tinge; the nipple is often retracted. The gland is tense to palpation, sharply painful, the tissue is pasteous; in 70% of patients, fluctuation sign is positive. In 60% of patients, the inflammatory process involves 3-4 quadrants. Blood count shows elevated white blood cells count, reduced blood hemoglobin, shift in the blood formulas to the left. Urinalysis demonstrates albuminuria, granular cylinders.

In *gangrenous form*, the condition of the patients is extremely severe, there is a wide necrosis of the skin and deep tissues. This form is more commonly observed in patients who are late with medical care. The purulent process proceeds with rapid melting of tissues and involvement of the tissue spaces of the chest and is accompanied by a pronounced systemic inflammatory reaction. Most

patients have fever above 39° C. General and local symptoms of the disease are pronounced, fluctuation is determined in 100% of cases.

The inflammatory process involves all breast quadrants. On visual examination the breast skin is cyanotic-crimson, the epidermis is sometimes exfoliated with formation of bubbles filled with hemorrhagic fluid, there are foci of necrosis. Blood test shows significant leukocytosis, HB is reduced to 80-90 g/l. urinalysis demonstrates protein and elevated red blood cell amount; in 75% of cases there are hyaline and granular cylinders.

Diagnosis. To make the diagnosis of acute purulent mastitis (postpartum), complaints, disease and life history are considered, general and local clinical symptoms and functional state of the glands are evaluated, common laboratory and instrumental methods of investigation are used.

The first signs of the disease may appear on the first week after childbirth, but in the majority of cases 3-4 days before mastitis there is lactostasis. The typical complaints are headache, pain and feeling of heaviness in the breast, decreased lactation, weakness, chills, fever.

With the purpose of early diagnosis the following is used: 1) bacteriological examination of the milk from both breasts (qualitative and quantitative assessment of microbial bodies in 1 ml of milk); 2) cytological examination of the milk (counting of formed blood elements as markers of inflammation); 3) determining the milk pH, reductase activity; 4) breast ultrasound (evaluates the state of the glandular structures, the nature and location of the inflammatory process); 5) breast ultrasonography with simultaneous puncture of the infiltration with a needle with a wide lumen for obtaining the contents (this method has value only in abscess mastitis).

Differential diagnosis. Mastitis should be differentiated from lactostasis. In lactostasis, as well as in mastitis, the body temperature is elevated, the breast is enlarged, palpation is painful in all divisions, but *swelling and hyperemia of the skin are always absent*. If only lactostasis is present, milk expression relieves the pain, small-sized painless lobules with clear contours and fine-grained structure are palpated. With prolonged lactostasis over three days, purulent mastitis develops. If lactostasis is combined with mastitis, after milk expression, dense painful infiltrate is still palpated, the fine-grained structure of the lobules is not determined, the body temperature remains high, and the state of the patient does not improve.

Treatment of patients with mastitis should be started with the appearance of the first complaints of pain in the swollen breast.

Choice of medical tactics. Non-purulent forms of mastitis (serous, infiltrative) are treated conservatively, in purulent forms (abscess, infiltrative-abscess, phlegmonous, gangrene), surgery is performed on the in-patient basis.

Criteria for conservative treatment of mastitis:

1) the condition of the patient is satisfactory;
2) the disease duration does not exceed 3 days;
3) body temperature is below 37.5° C;
4) local symptoms of purulent inflammation are absent;
5) palpated infiltration is moderately painful, takes no more than one quadrant of the breast;
6) blood count is within the normal ranges.

At ***conservative treatment***, the following requirements are fulfilled.

1. Immobilization of the breast.

2. Compulsory milk expression or pumping (milk expression from both breasts every 3-4 hours - 8 times a day). First of all, the milk is expressed out of the healthy gland, then - from the involved; intramuscular injection of 2.0 ml of drotaverine (no-spa) 20 minutes prior to milk expression from the involved breast (3 times a day, for 3 days at regular intervals), and 5 minutes before expression - 0.5 ml of oxytocin, which improves milk flow, are recommended.

3. Antibiotic therapy under the flora sensitivity control (in staphylococcal infections, it is advisable to prescribe first-generation cephalosporins (cefazolin, cephaloridine); when staphylococci are associated with escherichia, klebsiella or proteus - second generation (cefuroxime, cefotetan, cefoxitin, cefamandol), with secondary infection – 3rd and 4th generation antibiotics (3rd generation:

cefotaxim, ceftriaxone, ceftazidime, cefoperazone, cefodizim, cefoperazone/sulbactam, 4th generation: cefepim, cefpirome).

4. Daily retromammary novocaine blockades in combination with broad-spectrum antibiotics in a half-daily dose.

5. Application of physical therapy: (ultraviolet irradiation, sollux, UHF therapy or ultrasound therapy, etc., physical treatment should be used at positive dynamics in a day after starting conservative therapy).

6. Desensitizing therapy (intravenous administration of antihistamines 2-3 times a day).

7. Semi-alcoholic bandages on the breast (do not apply any ointment warming compresses).

8. Systemic therapy, symptomatic therapy and vitamin therapy (vitamins B and C).

Technique for retromammary novocaine blockade. Place of performing: dressing room, operating room. Position of the patient: on the back on the operating table or dressing table. Treatment of the surgical field, the surgeon's hands, wearing sterile masks, gowns, gloves.

If the patient is infected or suspected of being infected (HIV infection, hepatitis B, etc.), personal protective equipment must be used: a gown, a waterproof apron, a hat, hospital footwear, safety glasses and masks, or protective covers that cover the entire face; gloves; microtraumas on the hands are treated with 3% iodine and glued with a plaster; *the gloves are checked for durability*.

The breast is pulled forward by hand and a long needle is inserted into the posterior fascia of the breast into the fatty tissue, to the area of the areola projection at its base, infiltrating the tissue with a 0.25% novocaine solution, parallel with the chest,. Then 100.0-150.0 ml of 0.25% novocaine solution with antibiotics are injected with a syringe through this needle, while controlling the position of the needle in the retromammary space: a) the sharp end of the needle remains fixed; b) when injecting the solution, infiltration of the breast tissue should not be demonstrated; c) the breast should be evenly elevated with the solution that fills the retromammary space.

The blockade relieves the pains and widens the milk ducts, which facilitates expression; a high concentration of antibiotic in the breast is achieved. The milk can be expressed 20-30 minutes. after the blockade.

In the absence of positive dynamics during two or three days of conservative treatment, indicating transition of the inflammation into the *purulent stage*, ***surgical intervention*** is indicated. There are 2 types of operations:

1) dissection, treatment and drainage of the boil *("open" method*);

2) removal of the boil within the limits of healthy tissues with adjustment of flow drainage and primary sutures *("closed" method*).

The following methods of anesthesia (O.O. Khizhniak) are used.

1. Local anesthesia:

- superficial (or topical);
- infiltration;
- regional;
- conductor (blockade of nerves or their plexi);
- epidural (from *dura mater*);
- spinal.

2. Narcosis (or general anesthesia):

- intravenous with independent breathing;
- inhalation mask with independent breathing;
- intubation (or endotracheal) with mechanical ventilation;
- intravenous with mechanical ventilation;
- inhalation with mechanical ventilation;
- combined: IV + inhalation anesthetics with mechanical ventilation.

Basic principles of surgical treatment of acute purulent lactation mastitis:

a) the choice of adequate approach to the purulent focus, taking into account the need to preserve the function and appearance of the breast;

b) radical surgical treatment of the purulent focus;

c) adequate drainage, including using a drainage-irrigation system;

d) closure of the wound with a primary suture, and at contraindications - secondary sutures and application of dermoplasty;

e) prolonged dripping irrigation of the wound after the surgery with solutions of antiseptics through a drainage-washing system.

Open surgery for acute purulent mastitis includes the ***following stages.***

1. *Dissection and treatment of the boil.* A *radial incision* (7-10 cm in length, short of 2-3 cm to the areola), paraareolar, along the lower edge of the breast – according to Bardenheuer (at retromammary disease). The finger is inserted into the abscess cavity to divide the existing cords and intersections. In the presence of a boil in the upper and lower quadrants of the breast, the incision can be done in the lower quadrant and the boil located in the upper quadrant of the breast is emptied from the incision. If it is difficult to empty two boils from one incision, it is necessary to make a second radial counteropening.

2. *Draining the boil cavity.* After the incision and thorough treatment of the cavity, it is drained with open drainage, using rubber strips of rubber surgical gloves, which are installed at the lowest (in lying position) section of purulent focus. Drainages are removed (gradually with pulling) 6-7 days or more after the complete cleansing of the wound cavity; reduction of exudation and appearance of granulations.

In the postoperative period, wound treatment should be administered in phases: in hydration phase, bandages with antibacterial ointments on hydrophilic base are used, in dehydration phase - bandages with antibacterial ointments on indifferent base. Along with this it is worthwhile to administer general treatment with antibiotics, vitamins; physical therapy.

During ***complete surgical removal of the boil with suturing and flow drainage of the wound*** (the type of sectoral resection of the breast), *flow drainage systems* are used which allow long-term rinsing of the postoperative wound in the postoperative period (until the complete wound cavity is cleansed) with solutions of antiseptics for mechanical removal of purulent discharge, microbial flora and small necrotized tissue areas.

Flow drainages can be *closed, semi-closed and open.*

The drainage-irrigation system (Fig. 21) consists of separate, polychlorvynil tubes of various calibers with openings on the sides. A tube with an internal diameter of 0.2 cm serves for constant drip irrigation of the cavity with antiseptic solutions. A tube of a larger diameter (0.4 - 0.6 cm) promotes outflow of the irrigation fluid.

The technique of drainage-irrigation system application. An irrigator tube with an internal diameter of 0.2 cm is conducted through the upper pole of the cavity and its two ends are brought outwards through maximally distant from each other punctures in the skin. In its middle portion of the irrigator tube, which is located within the cavity, small openings are made so that the solution of the antiseptic uniformly fills the lumen of the irrigator throughout. In this case cavity irrigation will occur from all openings, which improves the quality of washing. At large openings, the solution flows mainly through the first outermost opening. In the postoperative period, a dropper with antiseptic solution is connected to the both ends of the irrigator tube for continuous dripping.

The drainage tube with a diameter of 0.4-0.6 cm for the outflow of the irrigation fluid is brought through a puncture in the skin at the lower pole of the cavity and placed on its bottom. Openings for better outflow are made on the lateral surfaces of the tube. The diameter of these openings should not exceed the diameter of the tube itself in order to avoid the drainage obturation. The irrigator and drainage are fixed to the skin with silk sutures. The number of drainage tubes depends on the shape and volume of the inflammatory process.

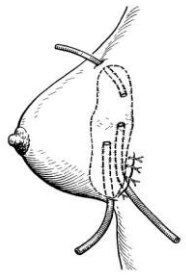


Fig. 21. Flow drainage at "closed" method of surgical treatment of acute mastitis

In phlegmonous form of mastitis or total involvement of the gland by the purulent process, two, and in particularly severe cases, three drainage tubes should be used for irrigation fluid outflow. The esthetic result does not depend on the number of drainages, because after their removal, the remaining scars are small.

The drainage-irrigation system is removed within 5 to 12 days after the operation. Indications for removal:

a) control of the inflammation in the breast and absence of pus, fibrin and necrotic particles in irrigation fluid;

b) the presence of a residual cavity not exceeding 5.0 ml.

The closure of wounds with primary suture after radical surgical removal of the boil with the use of drainage-irrigation system can shorten the healing period, improve the functional and esthetic results of treatment.

Contraindications to primary sutures include an anaerobic component of the infection and a large skin defect, when it is impossible to draw together the edges of the wound without tension.

Plegmonous-gangrenous and purulent-putrid mastitis, which usually occur with severe septic conditions and threaten the life of the woman, require breast removal - mastectomy. The latter is also necessary in complicated with deep necrosis erysepelatous breast inflammation. In those cases when necrotic centers in erysepelatous inflammation affect only the skin, it is necessary to wait for their demarcation, and then perform necrectomy.

Of great importance in preventing mastitis are rational ***preventive measures***:

1. Systemic detection and rehabilitation of pathogenic staphylococci carriers among pregnant and medical personnel.
2. Planned active immunization of pregnant women with staphylococcal anatoxin.
3. Preparation of breasts for breast-feeding the child.
4. Accurate observance of sanitary-hygienic and anti-epidemic rules in maternity hospitals.
5. Urgent isolation of the detected patients with postpartum mastitis in separate wards.
6. Strict adherence to the personal hygiene as well as the rules and techniques of breast-feeding and milk expression.
7. Sanitary and educational work on postpartum mastitis prevention.

General principles of diagnosis and treatment of patients with acute purulent diseases of the skin and soft tissues

I. History

1. Data on a possible illness or traumatic injury.
2. Reduced immunoreactivity due to external or internal factors.
3. Data on primary or secondary damage to the skin or subcutaneous tissue, muscle and bone formations.

II. Clinical presentation. Distinguishing *three phases of the course of inflammatory diseases of soft tissues:*

1) infiltration;
2) suppuration;
3) resolution of the pathological process with:

a) recovery;

b) progression with complications development.

The phase of infiltration is characterized by pain at the site of the injury, local edema and hyperemia, impaired function of the affected area (especially characteristic of the limbs), moderate hyperthermia to 37.5 ° C.

The phase of suppuration is characterized by bursting, pulsating unbearable pain at the site of the process; local edema, hyperemia and hyperthermia over the suppuration zone; positive symptoms of "fluctuations", "spasticity" and pastosity of the skin over the boil, impaired function of the affected area and adjacent anatomical areas; febrile and hectic temperature (over 38° C); intoxication (nausea, vomiting, headache); clinical blood tests demonstrate leukocytosis, shifting the formula to the left, urinalysis shows proteinuria, leukocyturia.

The *phase of resolution with recovery* is characterized by disappearance of the pathological signs.

The phase of progression with development of complications is characterized by spread of local pain to the adjacent anatomical areas, development of lymphangitis, lymphangitis, thrombophlebitis, metastatic infiltrates and abscesses; development of phlegmon and sepsis; hectic and septic type of the temperature curve; progression of intoxication with development of multiple organ failure syndrome (hepatic-renal, cardiovascular, respiratory, cerebral). Blood test demonstrates progressive increase in leukocytosis, shifting the formula to the left. Urinalysis shows pronounced proteinuria, leukocyturia, cylindruria. biochemical analysis of blood shows hypoproteinemia, increase in the amount of urea, creatinine, bilirubin, transaminases (hepatic-renal insufficiency). Coagulogram shows hypercoagulation (reduced blood coagulation time, increase in the amount of fibrinogen, appearance and increase of fibrinogen B).

In the clinical status there are signs of lesions of remote topographic anatomical structures and organs (meningitis, abscesses of the abdominal cavity and retroperitoneal tissue, etc.).

III. Tactics: in the phase of infiltration conservative treatment is indicated, in the phase of suppuration - urgent surgical treatment; in the phase of resolution with the result in recovery - complex treatment, mostly conservative; in the phase of progression with the result in complications, complex treatment is indicated - surgical (in case of secondary purulent foci), intensive infusion and antibacterial therapy, symptomatic and general treatment.

IV. Conservative treatment in the phase of infiltration.

1. Fighting the infection: - antibiotic therapy (cephalosporins - 2-6 g/day, aminoglycosides - 240 mg/day, semisynthetic penicillins - 4-6 g/day, oxazolidinones - 1200 mg/day, etc.) administered with the account of the microflora sensitivity and individual tolerance orally and parenterally depending on the purulent process severity and the presence of complications; - sulfanilamides (sulfaten, norsulfazol, etc.); - derivatives of 8-oxyquinoline and chinocalins (nitroxolin, dioxidine, etc.); - metronidazole drugs (trichopolum, metragil, etc.); - external application of ultraviolet irradiation, antiseptics.

2. *Fighting against inflammation*: anti-inflammatory drugs (analgin, aspirin, etc.); low-intensity laser irradiation; ointment with anti-inflammatory local action.

3. *Detoxification therapy*: drinking plenty of fluid; infusion therapy (when indicated).

4. *Increased organism reactivity*: vitamin therapy; immunomodulators; biostimulants

5. Symptomatic treatment and correction of disorders caused by concomitant diseases (diabetes mellitus, etc.).

V. Surgical treatment.

1. Types of anesthesia:

1) local infiltration anesthesia according to O.O. Vyshnevsky (infiltration of the skin and subcutaneous structures using the method of "creeping infiltrate" with 0.25% novocaine solution with addition in some cases of antibiotic, for example, aminoglycosine (gentamicin - 80 mg, canamycin - 0.5 g, monomycin - 1.0);

2) conduction anesthesia:

a) according to Oberst-Lukashevych - administration of 5-8 ml of 1% novocaine solution near the base of the finger above the superimposed ligature for blockade of the digital nerves;

b) according to Usoltseva - administration of 20-40 ml of 0.5% novocaine solution to the projection of the head of the radial or ulnar bone for pain relief respective to the radial and ulnar nerves;

c) according to Kulenkampf - blockade of shoulder plexus with 1% lidocaine solution - 30-38 ml 1 cm above the middle of the clavicle outside the subclavian artery, perpendicular to the apical processes of the 1^{st} and 2^{nd} thoracic vertebrae;

d) according to Voyno-Yasenetsky - blockade of sciatic nerves with 0.25% novocaine solution 40-60 ml, which is introduced below and outside the ischial tuberosity, in the projection of the intersection of the lines through the great trochanter- horizontal and along the outer edge of the ischial tuberosity, - vertical;

3) intravenous anesthesia;
4) neuroleptanalgesia;
5) mask anesthesia;
6) endotracheal anesthesia.

The type of anesthesia is determined individually, taking into account the location of the boil, the presence of complications, the degree of operative risk and data on the drug tolerance.

2. *Types of operations*:

1) opening, treatment and drainage of the boil;
2) excision of the boil within healthy tissues with flow drainage and suturing;
3) puncture method (used as a temporary surgical manipulation, has limited indications).

3. Types of sutures:

1) primary blind suture - is imposed after the excision of the boil with a pyogenic capsule against a background of flow drainage;

2) primary tardy suture - is imposed on day 3-5 after the operation of removing the boil in cases of inflammatory infiltration of the tissue surrounding the boil;

3) secondary early – is imposed on the purified granulating wound within the period from 7 to 5 days after the abscess dissection;

4) secondary late – is imposed on the scarring wound, in terms of 16 and more days after the abscess opening with excision of the scar tissue.

VI. Postoperative management:

1) immobilization of the affected area;
2) high-calorie meals;
3) detoxification treatment;
4) antibiotic therapy in the presence of complications and signs of the process generalization;
5) immunocorrection (immunomodulation, immunostimulation when indicated);
6) symptomatic therapy;
7) local treatment of purulent wounds, aimed at the following measures:

a) fight against infection (antiseptics, antibiotics, drugs with decontaminant action - derivatives of nitrofurans);

b) cleansing of the wound from non-viable tissues ("chemical necrectomy": the use of substances with dehydrating action, proteolytic enzymes, sorbents, multicomponent ointments on a water-soluble basis);

c) stimulation of regenerative processes in the wound after its cleansing from necrotic elements and appearance of granulations (ointments on a fatty basis, anti-inflammatory drugs, biostimulants and antimicrobial drugs).

I.2. SYSTEMIC INFLAMMATORY RESPONSE SYNDROME IN SURGICAL PATIENTS

The term ***"Systemic Inflammatory Response Syndrome" (SIRS),*** which was adopted in 1991 at Consensus Conference of American Society of Thoracic Surgeons and Emergency Physicians devoted to sepsis definition, and later accepted by the majority of countries, including Ukraine, means *severe reaction of the body to inflammation, which is accompanied by disorder of functions of a number of organs and systems, and is caused by infection, severe trauma, burns and other factors damaging the cells of the organ tissues.*

SIRS is the beginning of a continuously evolving pathological process and is a reflection of the superfluous, insufficiently controlled by the immune system, secretion of inflammation mediators in response to severe stimuli of both bacterial and non-bacterial nature.

SIRS becomes dangerous for a patient's life in cases when excessive production of inflammation mediators and disturbance of balance between pro- and anti-inflammatory mediators affect the control function of the immune system. In these cases, SIRS may be complicated by sepsis, septic shock, multiple organ dysfunction syndrome (MODS).

Clinicians should learn to diagnose this syndrome in the very early stages of its development in order to find out its cause, to apply the necessary measures to prevent its development and to

administer adequate treatment before the development of severe dysfunction in the lungs, heart, liver, kidneys, etc.

However, the definition of SIRS as a condition with a significant increase in inflammatory mediators blood concentrations is simplified. The nature of the biological response to the damage is determined not only by SIRS degree, but also, first of all, by the changes in the balance of secretion and the release of pro- and anti-inflammatory cytokines in the blood. In this regard, the term "compensatory anti-inflammatory response" (CARS) was introduced. At present, septic process is considered as a dynamic interaction between SIRS and CARS.

As at the present stage of medicine development the methods of direct anti-mediator effect are viewed with discretion, treatment of septic state is carried out with the help of conventional components of intensive care, which have already confirmed their relevance: adequate antimicrobial chemotherapy, full-scale infusion-transfusion therapy, timely support or compensation organs and systems dysfunction.

Despite the measures of prevention and the use of combinations of modern antibiotics, infectious complications, caused both nosocomial and community-based pathogens, increase in frequency.

History, criteria, current provisions of SIRS. In 1991, consensus conference of American Society of Thoracic Surgeons and Emergency Physicians devoted to definition of sepsis introduced a new concept - *Systemic Inflammatory Response Syndrome (SIRS).* The terms CSRI (syndrome of systemic response to inflammation) and SIR (systemic inflammatory response) are used in the literature of the CIS countries and are similar to the term SIRS. CSRI, SIR and SIRS determine one and the same concept, that is clinical and laboratory manifestation of a generalized form of inflammatory reaction.

The conference (1991) adopted a number of criteria and provisions of SIRS:

1) tachycardia> 90 beats per minute;

2) tachypnea > 20 per 1 min. or Pa CO_2 - 32 mm Hg against a background of mechanical ventilation;

3) temperature> 38.0° C or <36.0° C;

4) leukocyte count in the peripheral blood> 12×10^9/l or $<4\times10^9$/l or the number of immature forms> 10%;

5) the diagnosis of SIRS is made only in those cases when the infection is detected and two or more of the above criteria (signs) are detected;

6) the definite difference of SIRS from sepsis consists in the following: - in the initial stages of inflammatory process at SIRS, the infectious component may be absent; in case of sepsis there should be a generalized intravascular infection characterized by bacteremia.

In the initial stages of generalized inflammation (SIRS), at excessive activation of polypeptide and other mediators, they form a cytokine network. Later, generalized inflammation progresses, protective function of the local inflammatory focus is lost and mechanisms of systemic alteration are activated.

Cytokine network is a complex of functionally linked cells, which consists of polymorphonuclear leukocytes, monocytes/macrophages and lymphocytes secreting cytokines and other inflammatory mediators (tissue inflammatory mediators, lymphokines and other biologically active substances), as well as the cells (this the group includes endotheliocytes) of any functional specialty responsible for the action of activating agents.

SIRS development depends not only on activation of the cytokine network, but also on inadequate functioning of the sentinel system of cascade proteolysis of blood plasma. As a corresponding reaction of the organism it can develop in any pathology and disease, being universal and non-specific response.

Current SIRS provisions.

1. Mechanisms of SIRS development (Kozlov V.K.) are triggered by the influence of initiating factors (trauma, ischemia, infection), later its degree increases by staged activation of cells, including monocytes/macrophages, neutrophils, lymphocytes, platelets, endotheliocytes. These cells produce

both cytokines and other activating mediators and collectively form a network of interrelated functional units - cytokine network. Its excessive activation results in generalization of inflammation with loss of protective function of the local inflammatory focus. Simultaneously the effects of systemic alteration increase; the syndrome is formed as a systemic reaction of the organism to extraordinary effects. Such effects may be infection or injury of any origin.

2. SIRS is a mandatory component of sepsis, which clinically proves penetration of pathogen toxins, cytokines and other systemic inflammation mediators into the blood. Development of this syndrome states the fact of loss of relative autonomy by the infectious focus.

3. At the initial stages of SIRS development, the infectious component may be absent.

4. SIRS may have non-infectious origin.

5. SIRS development depends not only on the cytokine network activation, but also on the inadequate functioning of the sentinel system of blood plasma cascade proteolysis.

6. SIRS, as the corresponding reaction of the organism, can develop at any pathology or disease.

7. The syndrome is universal and non-specific.

8. The presence of SIRS symptoms indicates a high predisposition of the organism to sepsis.

9. Sepsis and SIRS are not synonymous.

10. SIRS symptoms are present:

a) in all without exception infectious diseases occurring cyclically (benign);

b) in a number of non-infectious diseases (acute destructive pancreatitis, severe trauma of any origin, severe allergic disorders, diseases that are caused by organ ischemia (for example, in CAD, myocardial infarction, etc.);

c) in all infectious diseases caused by specific pathogens characterized by generalized forms of the infectious process (typhus, typhoid fever, leptospirosis, infectious mononucleosis, generalized viremia);

d) in inflammatory diseases of the trachea, bronchi, lungs and pleura.

11. Appearance of SIRS symptoms, presence of severe trauma (including burns), severe forms of infectious complications is a direct indication for cytokine therapy to prevent sepsis development.

Mechanisms of SIRS development. Among surgical diseases a significant place is occupied by acute inflammatory diseases of the abdominal and chest organs, soft tissues of the body. The achievements of molecular biology allowed to revise the ideas about the nature of inflammation and regulation of the immune response to it. The universal mechanism that determines physiological and pathological processes in the body is intercellular relationships. The primary role in intercellular interactions regulation is played by a group of protein molecules called the cytokine system.

The corresponding reaction of the body to inflammation, regardless of the location of the inflammatory process, develops in accordance with the general regularities that are inherent to each acute inflammation. The inflammatory process and the corresponding response to it develop with the participation of numerous inflammation mediators, including the cytokine system, according to the same regularities, as both in infection penetration, and in the case of injury, foci of tissue necrosis, burns and other factors. Clinical manifestations of acute inflammatory diseases, along with the general symptoms of inflammation, have specific symptoms that allow differentiating one disease from another.

The reaction of the organism to inflammation, in which the functions of the vital systems of the organism are not impaired, is called ***local***.

In phlegmon or gangrene of the affected organ, the inflammatory symptoms become more pronounced and signs of the function impairment of the vital systems in the form of significant tachycardia, tachypnea, hyperthermia (or hypothermia), high leukocytosis (or leukopenia in combination with hypothermia) begin to manifest. This reaction of the organism can be characterized as severe inflammation, which takes the systemic nature and proceeds as a severe general disease of inflammatory nature, involving almost all systems of the organism. Such inflammation is called general systemic inflammation or systemic inflammatory response syndrome - SIRS.

Inflammation is adaptive reaction of the body, aimed at destroying the agent that caused the inflammation, and restoration of the damaged tissue. The inflammatory process, developing with the obligatory participation of inflammation mediators, may be accompanied mainly by a local reaction with typical and local manifestations of the disease and a moderate, obscure, general reaction of the organs and systems of the organism. Local reaction protects the body, freeing it from the pathogenic factors, discriminating "foes" and "friends", which contributes to healing.

Inflammation mediators, without which development of inflammation is impossible, are the following active chemical compounds:

1) cytokines (proinflammatory and anti-inflammatory);
2) interferons;
3) eicosanoids;
4) reactive oxygen species;
5) blood plasma complement;
6) biologically active substances and stress hormones (histamine, serotonin, catecholamine, cortisol, vasopressin, prostaglandins, growth hormone);
7) thrombocyte activation factor;
8) nitrogen monoxide (NO) and others.

Inflammation and immunity function in close interaction, their function is to purify the internal environment of the organism both from foreign elements, and from damaged, modified own tissues with subsequent rejection and elimination of the damage consequences. Normally functioning control mechanisms of the immune system interfere with the uncontrolled release of cytokines and other inflammatory mediators and provide an adequate local response to the inflammatory process.

The organism can respond to inflammation with both local, and general reaction, which is called SIRS.

Local reaction to inflammation or local inflammation *is a local process caused by damage or destruction of tissues and is aimed at preventing the progression of tissue damage, dehydration of the body and subsequent destruction of natural barriers by limiting both the damaging agent and the zone of damage to the tissues of the body.*

This process is characterized by cascade activation of complement systems, coagulation and anticoagulation, kallikrein-kinin systems, as well as cellular elements (endotheliocytes, leukocytes, monocytes, macrophages, mast cells, etc.).

The *criteria of systemic inflammatory reactions* characterizing the body's response to local tissue destruction, are: ESR, C-reactive protein, systemic temperature, leukocyte intoxication index, and other indicators with varying sensitivity and specificity.

The size of the focus of damage, the features of the damaging agent, the state of the macroorganism are the main factors that determine severity degree and the nature of local and systemic manifestations of inflammatory response. However, there are a number of common patterns that are inherent in all forms of damage and damaging agents.

In case of damage to tissue structures, *five different mechanisms* involved in induction and development of the inflammatory response are activated. Interacting with each other, they lead to formation of morphological signs of inflammation.

A*ctivation of coagulation system* is a leading trigger of inflammation. The biological content of this activation consists in achieving local hemostasis. At the same time, Hageman factor activated during the biological effect of the coagulation system becomes the central element of subsequent SIRS development.

Thrombocyte link of hemostasis performs a biological function, i.e. stops bleeding (similar to the components of the coagulation system). A number of substances (thromboxane A_2 and prostaglandins) released during platelet activation, due to their vasoactive properties, play an important role in development of the inflammatory response.

Mast cells after activation with factor XII and products of thrombocyte activation, stimulate release of histamine and other vasoactive elements that affect smooth muscles, relaxing the latter, provide vasodilatation of the microvascular bed. Vasodilatation of the microvessels leads to increase

in the vascular wall permeability, increase in the total blood flow through the zone of vasodilatation with simultaneous reduction in blood flow velocity.

Factor XII activates kallikrein-kinin system, providing conversion of prekallikrein to kallikrein, a catalyst for synthesis of bradykinin, the action of which is also accompanied by vasodilation and increased vascular permeability.

Activation of the complement system (CS), which follows both in the classical and the alternative ways, leads to creation of conditions for lysis of cellular structures of microorganisms. In addition, activated components of CS have important vasoactive and chemoattractant properties. Infection and the effect of other damaging factors cause activation of the CS, which in turn promotes synthesis of C-reactive proteins (C3, C5), stimulates production of thrombocyte activation factor and formation of opsonins that take part in the process of phagocytosis and chemotaxis.

Involvement of activators by the inflammatory process promotes summary effect: increases microvascular permeability and volume of the blood flow in the microvascular bed, decreases the blood flow velocity and forms soft tissue edema.

The products of the inflammatory cascade induction metabolism and enzymes released by their action form a wide range of chemoattractant cytokines - chemokines. The main feature of these low molecular weight proteins is expressed specificity of action for each population, and sometimes the subpopulation of leukocytes. Owing to this, selective accumulation of leukocytes (neutrophils, monocytes, eosinophils) occurs in the damage focus. This is the ***first phase of inflammation - induction phase. T***he biological content of the action of inflammation activators at this stage is preparation of transition to the second phase of inflammation - the ***phase of active phagocytosis,*** an essential role in which is played by endothelium cells.

The endothelium lining the vessels (with a body weight of 70 kg its area is 700 m^2 and its mass is 1.5 kg), regulates the process of metabolism of substances dissolved in the blood plasma, as well as the cell structures between the lumen of the vascular bed and the intercellular space. The highly reactive NO molecule radical is continuously synthesized in small amounts from L-arginine by the enzyme NO-synthase in the endothelium. The action of NO consists in the following biological effects: relaxation of smooth muscles of the vascular wall and inhibiting adhesion of platelets and leukocytes within the lumen of the vascular bed. These effects allow the lumen to be maintained in the dilated (expanded) state and prevent the stasis of formed elements. Due to the short half-life of NO, the latter affects only the cells closest to it and the cells that synthesized it.

The damage to the endothelial barrier leads to activation of endothelial cells, monocytes and vascular smooth muscle cells, which produce a soluble form of NO synthase. The latter provides synthesis of large amounts of NO, formation of which is limited only by the presence and amount of L-arginine and oxygen. These biological effects promote maximum dilatation of intact vessels, which leads to rapid movement of leukocytes and platelets into the area of injury. The produced NO contributes to the death of microorganisms.

Phagocytosis phase begins from the moment when the concentration of chemokines reaches the critical level required to create an appropriate concentration of leukocytes. The main task of inflammatory phagocytic response is removal of microorganisms and limitation of the inflammation. In this period transient bacteriemia may appear. Microorganisms that occurred in the blood, are destroyed by neutrophilic leukocytes, macrophages that freely circulate in the blood, and Kupffer cells that act as macrophages.

The most important role in removal of microorganisms and other foreign substances, as well as production of cytokines and various mediators of inflammation, is played by *activated macrophages*, which both freely circulate in the blood and are resident, fixed in the liver, spleen, lungs and other organs. The resident macrophages include Kupffer cells, which make up 70% of all macrophages of the body. They play a major role in removal of microorganisms in the event of transient or persistent bacteremia; products of decay of proteins and xenogeneous substances. Simultaneously with complement activation, activation of neutrophils and macrophages occurs. Components of CS, C3a and C5a, play the role of expressed attractants and stimulants for polymorphonuclear leukocytes. Other chemotaxis activators are most often TNF-α, IL-1, IL-8, leukotrienes and thrombocyte-activating

factor. As a result of release during this activation of granulocyte-macrophage colony stimulating factor and macrophage colony stimulating factor acting as hormones, myelopoiesis is intensified and circulating leukocytes are activated. In these conditions, circulating polymorphonuclear leukocytes can damage distant tissue areas that are not involved in the inflammatory process.

Chemoattractants gradient, directed from the center of the affected area to the periphery, determines the direction of leukocyte migration. Division of the connections between endothelial cells as a result of microvascular vasodilatation promotes the exposure of receptors that recognize neutrophils; they begin to move to the site of injury. Swelling of soft tissues not only creates the fluid channels necessary for the movement of leukocytes to the injury site, but also provides continuous opsonization, facilitating identification of target cells for phagocytosis. The process of phagocytosis begins with the arrival of the polymorphonuclear leukocytes to the lesion site..

Monocytes are another very important participant in the second phase of inflammation. During formation of an inflammatory reaction, which can last several days, monocytes reach the place of damage (the first activated monocytes appear at the lesion focus within 24 hours after the onset of the inflammatory reaction), where they differentiate into two different subpopulations: one is designed for destruction of microorganisms, and the other is for phagocytosis of necrotic tissues.

Activated macrophages carry out transcription of antigens (bacteria, endotoxins, etc.). With this mechanism, macrophages deliver antigens to lymphocytes, promote their activation and proliferation. Activated T-lymphocytes acquire much larger cytotoxic and cytolytic properties, dramatically increase production of cytokines. B-lymphocytes begin to produce specific antibodies. Due to lymphocyte activation, production of cytokines and other inflammatory mediators sharply increases, and hypercytonemia develops. Involvement of activated macrophages in the developing inflammation is the borderline between the local and systemic response to inflammation. The interaction of macrophages with T-lymphocytes and "natural killer" cells by cytokines provides the necessary conditions for bacteria destruction and endotoxin neutralization, limiting inflammation, and prevention of the infection generalization.

An important role in protecting the body from infection is played by natural *killer cells* (NK cells). They are produced in the bone marrow and represent a subpopulation of large granular lymphocytes that, unlike T-killers, can break down bacteria and target cells without prior sensitization. These cells, as well as macrophages, remove foreign particles and microorganisms from the blood, provide adequate production of inflammatory mediators and local protection against infection, maintain a balance between proinflammatory and anti-inflammatory mediators. Thus, large granular lymphocytes (NK-cells) impede microcirculation disorders and damage to the parenchymal organs.

Of great importance for regulation of acute inflammation by means of TNF are protein molecules, known as "*nuclear factor-Kappa B*" which play an important role in development of systemic immune response syndrome and multiple organ dysfunction syndrome. For therapeutic purposes, it is possible to limit activation of this factor, which will reduce production of inflammation mediators and can create a beneficial effect, reducing the tissue damage by inflammation mediators, simultaneously reducing the risk of developing organ dysfunction.

Endothelial cells play a significant role in SIRS development. Endothelial cells are the connecting link between the parenchymal organs cells and circulating platelets, macrophages, neutrophils, cytokines and their soluble receptors, therefore endothelium of the microcirculatory bed very quickly reacts both to the changes in the blood concentration of inflammation mediators, and to their maintenance beyond the vascular bed. In response to the damage, endothelial cells produce nitric oxide, endothelin, platelet activation factor, cytokines and other mediators. Endothelial cells are at the center of all reactions that develop at inflammation. It is these cells, that acquire the ability to "direct" leukocytes to the damage site after stimulation with cytokines. Activated leukocytes, located in the vascular bed, perform rotary movements along the surface of the endothelium of the microcirculatory bed, resulting in the marginal standing of the leukocytes. On the surface of the leukocytes, platelets and endothelium cells, adhesive molecules are formed. Blood cells begin to stick to the walls of venules, their movement stops. In the capillaries, microthrombi consisting of platelets, neutrophils and fibrin, are formed. As a result, initially in the inflammation focus, circulation in the microcirculatory

bed is impaired, permeability of capillaries sharply increases, there are typical signs of local inflammation. In severe aggression, hyperactivation of cells that produce cytokines and other inflammatory mediators occurs. The amount of cytokines and NO increases not only in the focus of inflammation, but also beyond its limits in the circulating blood. Due to the excess of cytokines and other mediators in the blood, the microcirculatory system of organs and tissues outside the primary inflammatory center is damaged to a greater or lesser extent. The function of the vital systems and organs is impaired, which leads to SIRS development. At the same time, against a background of local signs of inflammation, disorders the respiratory and cardiovascular systems, kidneys and liver develop, the inflammation proceeds as a severe general disease with involvement of all functional systems of the body in this process.

Anti-inflammatory mechanisms and their role in SIRS development. Anti-inflammatory mechanisms are triggered simultaneously with the beginning of the act of inflammation. They contain cytokines that possess a direct anti-inflammatory effect: IL-4, IL-10 and IL-13. Also, expression of receptor antagonists, for example, IL-1 receptor antagonist, is present. Distribution of soluble receptors of some cytokines may reduce their availability by binding receptors to target cells. Glucocorticoids and catecholamines also have a direct anti-inflammatory effect.

Mechanisms inflammatory response control are not fully understood. It is most likely that the key role in stopping the inflammatory reaction is played by reduction in the activity of the processes that caused it. It is known that polymorphonuclear leukocytes do not have a mechanism that would restore them after action in the inflammation focus. It is believed that apoptosis, programmed cell death, is a leading process to discontinue the activity of polymorphonuclear leukocytes. As soon as cytotoxic activity of monocytes and polymorphonuclear leukocytes decreases, the processes of reparative regeneration begin to predominate in the inflammation zone.

***Clinical significance of SIRS*.** The clinical manifestations of SIRS include 1) increase in body temperature above 38^oC or its decrease below 36^oC with anergy; 2) tachycardia - increase in the number of heart beats over 90 per minute; 3) tachypnoe - increase in the frequency of respiration over 20 per minute or decrease in $PaCO_2$ less than 32 mm Hg; 4) leukocytosis over 12×10^9 in 1 mm^3 or decrease in the number of leukocytes below 4×10^9 in 1 mm^3, or stab neutrophil shift by more than 10%.

The syndrome severity is determined by the number of existing signs of organ function abnormalities in the patient. In the presence of two of the four signs described above, the syndrome is assessed as light, with three signs - as average severity, with four - as severe. When detecting three or four signs of SIRS, the risk of progression of the disease and development of multiple organ failure, requiring special measures for correction, sharply increases.

Microorganisms, endotoxins, and local mediators of aseptic inflammation usually come from the primary focus of infection or foci of aseptic inflammation. In the absence of the primary focus of infection, microorganisms and endotoxins can enter the bloodstream from the intestine due to translocation, through the wall of the intestine into the bloodstream or from primarily sterile necrosis foci in acute pancreatitis. This is usually observed with pronounced dynamic or mechanical obstruction resulting from acute inflammatory diseases of the abdominal cavity. Mild SIRS is primarily a signal of excessive production of cytokines that are over-activated by macrophages and other cytokine-producing cells. If prevention and treatment of the underlying disease will not be started in time, SIRS will progress continuously, and the multiple organ dysfunction may turn into multiple organ failure, which is usually a manifestation of a generalized infection, i.e. sepsis.

Thus, *SIRS is the beginning of a pathologic process that is continuously evolving, which is a reflection of abundant, inadequately controlled by the immune system secretion of cytokines and other inflammatory mediators due to intercellular interactions in response to severe antigenic stimuli of both bacterial and non-bacterial nature*. SIRS resulting from a severe infection is not different from the reaction that occurs in response to aseptic inflammation in case of a massive trauma, acute pancreatitis, traumatic surgical interventions, organ transplantation, and large burns. This is due to the fact that development of this syndrome involves the same pathophysiological mechanisms and inflammatory mediators. Any medical institution is capable of definition and evaluation of SIRS

severity. This term is adopted by the international community of doctors of different specialties in most countries of the world.

The knowledge of SIRS pathogenesis will allow development of anti-cytokine therapy, prevention and treatment of complications. For these purposes monoclonal antibodies against cytokines, antibodies against the most active proinflammatory cytokines (IL-1, IL-6, TNF) are used. There are reports about good efficacy of plasma filtration through special columns that allow removal of excess cytokines from the blood. To suppress cytokine-producing function of leukocytes and to reduce blood concentration of cytokines, large doses of steroid hormones are used (but not always successfully).

A very important role in treatment of patients is played by timely and adequate treatment of the underlying disease, complex prevention and treatment of vital organ dysfunction. The frequency of SIRS in patients of intensive care units in surgical clinics reaches 50%. At the same time, in patients with high body temperature (this is one of the signs of the syndrome) treated in the intensive care unit, SIRS is observed in 95% of patients.

A cooperative study covering several medical centers in the United States has shown that of the total number of patients with SIRS only 26% developed sepsis and 4% - septic shock. Mortality increased depending on severity of the syndrome. In severe SIRS, it was 7%, in sepsis -16%, in septic shock - 46%. SIRS usually lasts for only a few days, but it can last for a longer time until reduction of the content of cytokines and NO in the blood, restoration of the balance between proinflammatory and anti-inflammatory cytokines, and until restoration of the function of the immune system to control cytokine production. With decreasing hypercytokinemia the symptoms can gradually reduce; in these cases, the risk of complications dramatically decreases and recovery can be expected on the next day.

In severe forms of the syndrome there is a direct correlation between the content of cytokines in the blood and severity of the patient's condition. Pro- and anti-inflammatory mediators can mutually reinforce their pathophysiological effect, creating an increasing immunological dissonance. It is under these conditions that the inflammatory mediators begin to produce a detrimental effect on the cells and tissues of the body. The complex interaction of cytokines and cytokinin-neutralizing molecules determines the clinical manifestations and the course of sepsis.

Even severe SIRS cannot be considered as sepsis, if the patient does not have a primary focus of infection (portal of entry)), bacteremia, confirmed by detecting bacteria in the blood in multiple cultures.

Sepsis as a clinical syndrome is difficult to determine. The consensus commission of American doctors defines *sepsis as a very severe form of SIRS in patients with a primary source of infection confirmed by blood culture, in the presence of the signs of CNS inhibition and multiple organ failure.*

We should not forget about the possibility of developing sepsis in the absence of the primary focus of infection. In such cases, microorganisms and endotoxins may appear in the blood as a result of translocation of intestinal bacteria and endotoxins into the bloodstream. Then the intestine becomes a source of infection, which was not taken into account when looking for the causes of bacteremia. Translocation of bacteria and endotoxins from the intestines into the bloodstream becomes possible in impairment of the barrier function of the intestinal mucosa due to ischemia of its walls at peritonitis, acute intestinal obstruction, shock, and other factors. In these conditions, the intestine becomes similar to a "not drained purulent cavity".

Multiple organ dysfunction (MOD) is a universal impairment of all organs and tissues by aggressive mediators of the critical state (aggressive mediators of inflammation) with a temporary overvelming of the symptoms of one or another insufficiency - cardiac, pulmonary, renal, etc. with simultaneous or sequential impairment of the vital systems of the organism.

According to *etiology*, two groups of MOD are distinguished. The first group includes MOD, which arose due to aggravation of a certain pathology, when one or more vital functions are damaged to the extent that their artificial replacement is required. The second group is iatrogenic MOD, caused by any pathology that arose in connection with medical actions - preventive, diagnostic, and therapeutic.

MOD pathogenesis has the following main mechanisms:

1) mediator (in autoimmune lesion);
2) microcirculatory and associated with it reperfusion;
3) infectious-septic;
4) phenomenon of double impact and other mechanisms.

The *mediator mechanism of* MOD development includes the function of the endothelium and functions of cytokines.

*Endothelial function*s:

1) endothelium actively changes permeability of the vascular wall, providing a passage of fluid containing substances from blood circulation into tissues and back from tissues into the blood circulation (this function is related to the active function of endothelium and is implemented through the system of mediators produced by the endothelial cell);

2) regulation of the lumen of the vessel that it lines (the mechanism - endothelium cells produce factors that narrow or expand the vessel, affecting the smooth muscle);

3) participation in coagulation, anticoagulation and fibrinolytic blood systems, in atherogenesis;

4) adhesion, aggregation and transformation of blood cells (leukocytes, platelets);

5) participation of endothelial cells in the inflammatory response, in development and spread of malignant tumors, in anaphylactic and other hyperimmune reactions (total - participation of endothelial cells in reactions of the immune-reactive system).

To provide the above functions, the endothelial cells have a host of specific receptors and secreted into the blood biologically active substances (endothelial relaxing and stimulating factors, nitric oxide, and many others).

At MOD development, a major role is played by synthesis of "*pathological*" NO, causing uncorrected or poorly corrected vasodilation, when conventional means of hemodynamic correction are poorly effective.

The clinical significance: to deactivate "pathological" NO, it is necessary to use an inhibitor of NO synthesis - methyl ester of L-arginine (especially in septic shock).

Microcirculatory and associated reperfusion mechanism of MOD development - a mechanism of development of hypovolemic vicious circle (consistently transient one to another by closed cycle that arose and led to MOD development, pathological disorders of the cardiovascular system - hypovolemia → reduction of cardiac output → rheology impairment → blood sequestration → hypovolemia).

The causes leading to the microcirculatory mechanism of MOD development include reduction of CBV against a background of external blood loss, blood sequestration, capillary leakage, etc. Against a background of CBV reduction, centralization of blood flow and reduction of microcirculation in the peripheral tissues occur, which leads to development of hypovolemic vicious circle.

However, the hypovolemic vicious circle may also occur at the mediator mechanism of MOD development, which begins with activation of the endothelial cell, which leads to the following mechanism - adhesion to the endothelium of various cells and structures, including platelet aggregation and adhesion of the type of vascular thrombocyte hemostasis.

In these reactions of body tissue ischemia, an active role is played by fibronectin, thromboxane, cytokines and eicosanoids (leukotrienes, epoxides). These reactions are counteracted by NO and prostacyclin. However, NO amount becomes insufficient for microcirculatory disorder elimination and development of "pathological" NO in large quantities, which ultimately leads to further blood flow deceleration and rheology disorders (effect of vasodilation) with the phenomena of aggregation and sequestration of blood, which, in turn, leads to organ tissue ischemia, and the latter results in MOD development.

Even major disorders of the organs function against a background of microcirculatory ischemia of the tissues will arise in the event that ischemia and blood flow in the tissues are restored after a period of time (during the period of microcirculation disorder, the tissues accumulate underoxidized products of metabolism). After microcirculation restoration in the tissues, mechanisms of reperfusion begin to act, against a background of which MOD occurs.

Changes in the tissues associated with ***reperfusion pathology***. After ischemia of organ tissue cells at reperfusion, further deterioration of tissue condition occurs, which is manifested in the appearance of three paradoxes: oxygen, calcium and ionic.

Oxygen paradox. Under ischemia conditions, enzyme systems of biological oxidation are damaged (F^{2+} - reduced iron - is accumulated, ATP is converted to AMP, then adenosine, inosine, hypoxanthine are formed). At reperfusion, tissue are damaged by oxygen radicals, when xanthine oxidase converts hypoxanthane into urates and oxygen radicals in the presence of O_2. Tissues damage at reperfusion occurs in the following sequence: at ischemia, ATP turns into AMP. After that adenosine and hypoxanthine and later xanthine oxidase converts hypoxanthin into urates and oxygen radicals in the presence of O_2, the urates interacting with H_2O_2 and O_2, form F^{3+}, the formation of which stimulates neutrophilosis; oxidants and toxins are formed, which also have their detrimental effect on the cells, causing tissue damage and death.

When inadequately large amounts of oxygen come to the tissue with enzyme systems of biological oxidation damaged by ischemia, this causes peroxide oxidation in the tissues, which damages the cell membrane and organelles of the protoplasm constructed from phospholipids and affects energy production (lung surfactant, which is a lipoprotein is affected). Protein peroxidation results in inactivation of numerous enzymes; peroxidation of carbohydrates leads to depolymerization of polysaccharides (peroxidation damages intercellular substance of the matrix).

Thus, peroxidation is both absolute and relative hyperoxia and occurs at reperfusion of tissues due to ischemia; normal metabolism and energy production are impaired, as they are components of the same process and operate in the presence of three components: metabolite transportation, work of enzyme systems and O_2 delivery.

Clinical significance. In clinical practice, restoration of blood circulation should be started with low concentrations of O_2, using iron chelators (deferoxamine), and classical antioxidants and xanthine oxidase inhibitors, allopurinol, etc.) should be used.

Calcium paradox. Pathological actions of calcium at reperfusion:

1) after ischemia when the blood flow is restored, calcium enters the cell, destroying the ribosomes, which impairs energy production (protein and ATP);

2) it activates formation of eicosanoids, resulting in increased microcirculation disorders, ischemia, impairment of membrane permeability, which is amplified by ionic paradox.

Clinical significance. In clinical practice, calcium antagonists should be used before and during blood flow restoration.

Ionic paradox consists in the following: after microcirculation and blood flow have been restored, the tissues begin to "attract" water, which is clinically demonstrated by swelling of tissues (in ischemia, tissues osmolarity increases by an average of 40-50 mosm/l; 1 mosm/l is equivalent to 19 mm Hg), and 40-50 mosm/l, respectively, - 760 - 950 mm Hg, therefore, after the blood flow restoration tissues begins to attract water; clinically it is expressed in tissue edema).

Clinical significance. In clinical practice, tissues reperfusion requires diuresis stimulation in order to reduce ischemic tissue edema.

In clinical practice, the following types of *ischemia and reperfusion* are observed:

1) at elimination of prolonged compression of soft tissues syndrome or removal of the tourniquet imposed for temporary hemostasis;

2) on melting of the viable intestine in strangulation intestinal obstruction;

3) when replacing blood loss or normalizing hemodynamics at hypovolemia of any origin;

4) at restoration of blood flow at embolus removal from the femoral artery at aortofemoral shunting;

5) during embolectomy or thrombolysis for pulmonary thromboembolism;

6) at elimination of coronary thrombosis in myocardial infarction or aortocoronary bypass.

All above pathologies without taking the necessary therapeutic and preventive measures can lead to MOD development.

Infectious-mechanical MOD development. Pathological changes in the intestinal wall lead to entrance of microbes to the bloodstream, after which SIRS and MOD develop.

The following pathological changes in the intestinal wall lead to entrance of microbes to the blood flow, SIRS and later MOD development (trigger pronounced cytokine mediatory chain reaction):

1) stress damage of the mucosa;

2) appearance of endotoxin of gram-negative microbes in the blood;

3) depletion of immune protection by stress and autoregulation functions impairment;

4) endotoxemia at dysbiosis (in intact intestine).

In turn, the following factors can result in *endotoxemia:*

1) the use of a large amount of antacid drugs leads to the changes in the intestinal flora towards gram-negative microbes that produce endotoxin;

2) the use of irrational antibiotic therapy (primarily gram-positive microorganisms are killed resulting in predominance of gram-negative microbes);

3) when the immune reactivity decreases, the endotoxin enters the bloodstream;

4) prolonged parenteral and hyperosmolar nutrition impair the intestinal flora and damage the mucosa, which leads to endotoxemia (absorption of endotoxins through the intestinal mucosa);

5) coprostasis in intestine paresis.

According to Marshall J.C., Christon N.U., Meakins J.L. (1993) "The digestive tract is a "non-drained abscess of multiple organ failure".

In clinical practice, prevention of SIRS and MOD consists in the following:

1) timely treatment of the large intestine;

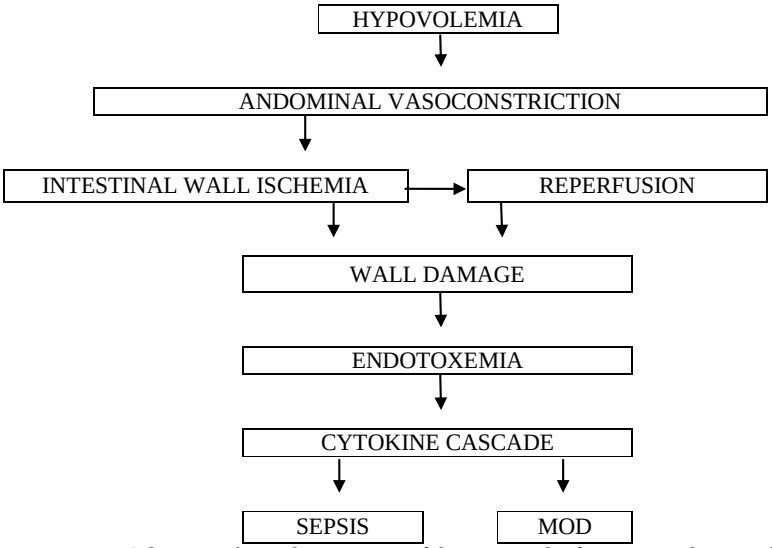
2) prevention and elimination of the large intestine paresis;

3) rational antibiotic therapy taking into account prevention of growth of gram-negative microbes;

4) the use of endosorbents of the endotoxin (enterosgel, etc.);

5) the use of early enteral nutrition with refusal, if possible, from antacids.

Scheme 1 shows the summary components of "intestinal-infectious" MOD mechanism.



Scheme 1. The total components of the "intestinal-infectious" mechanism of PON

The phenomenon of *double impact* against a background of autoimmune impairment, as one of MOD development mechanisms. The infection generalization (release of infectious factor from the focus to the bloodstream) is nothing more than a disorder of the inflammatory process autoregulation. The organism responds to the infection generalization with production of a cascade of cytokines, in the process of which a chain of interleukins is formed and stimulated, the main purpose of them is destruction of tissue cells damaged by ischemia, mechanical or other kind of trauma.

Under the conditions of impaired autoregulation, interleukins destroy not only damaged cells, but also healthy ones, and as a result, the organs are damaged in the form of acute autoimmune pathology.

Therefore, the mechanism of MOD development at double impact phenomenon against a background of autoimmune damage is characterized as destruction by the chain of cytokines not only injured, but also healthy cells, in which MOD development against a background of the phenomenon of double impact consists. MOD development by the mechanism of autoimmune impairment is one of the options of mediator (in this case, cytokine) mechanism of MOD development.

Clinical significance: to prevent the double impact phenomenon against autoimmune impairment, an immunomodulator Rondoleukin, inhibitor of MOD, SIRS, and sepsis, should be used.

Development of the double impact phenomenon against a background of iatrogeny, as one of the mechanisms of MOD development. Intensive therapy, administered at the signs or development of MOD consists in the use of infusions, respiratory support, intra- and extracorporal sorption, electrostimulation, polypharmacy, metabolic correction and other methods, causes a new stimulation of inflammatory cytokines production in high concentrations and leads to mediator aggression, which develops in the same tissues of organs, which are administered the above intensive therapy. In this case, iatrogeny is the administered intensive therapy.

In addition to the above-mentioned, MOD can develop after blood replacement, massive antibiotic therapy, normalization of hemodynamics, which will lead to reperfusion pathology.

***Clinical significance*:** using intensive therapy in MOD development, it is necessary to remember about the possible development of the double impact phenomenon against a background of iatrogeny and to take appropriate preventive measures.

Cytokines *are a family of biologically active peptides, which produce a hormone-like effect and provide interaction of the cells of the immune, hematopoietic, nervous and endocrine systems.*

By their nature, cytokines are average molecular weight proteins or glycoproteins (15-60 kD). Biological, physical and chemical stimuli can stimulate cytokine production.

Cytokines are proteins which are nonspecific in respect to antigens, that are produced mainly by activated cells of the immune system. Acting as mediators of the immune system, they regulate the strength and duration of the immune response and the inflammatory process, providing intercellular interactions, positive and negative immunoregulation, and are factors of growth and differentiation of lymphoid and other cells. Cytokines are not isolated peptides, but an integral system, the main components of which are the producer cells, the cytokine protein itself, the receptor that perceives it, and the target cell.

Interacting with each other by agonistic or antagonistic principle, they change the functional state of the target cells and form a cytokine network. Their action is realized on a network principle, that is, the information transmitted by the cell is contained not in one individual peptide, but in the most regulatory cytokines.

The peculiarity of cytokines is that they are secreted products of virtually all cells that take an active part in immune processes (lymphocytes, phagocytic, dendritic, mast cells, etc.), as well as other cells of the body (fibroblasts, endothelial cells, some somatic cells and etc.). Their biological role is extremely significant and varied; a number of cytokines have a pleiotropic property; on the other hand, several cytokines can produce similar effects. Cytokines take part in development of immune responses of cellular or humoral type, in transplantation and antitumour immunity, in induction of tolerance and in many other vital processes of the organism. In clinical medicine, the role of cytokines is considered predominantly in relation to immunomodulation, inflammation, wound healing, angiogenesis, neuroimmunodecrine interactions, bacterial and parasitic infections, malignant neoplasms, and regulation of hemopoiesis.

The whole family of cytokines has common properties:

1) they are synthesized in the process of implementing mechanisms of natural or specific immunity;

2) they exhibit their activity at very low concentrations (10^{-11} mol/l);

3) they serve as mediators of the immune and inflammatory reaction and have autocrine, paracrine and endocrine activity (paracrine effect is the effect of cytokines on the adjacent cells; autocrine effect is the effect of cytokines directly on the cell in which they were formed; endocrine or distal effect (general) is distant from the place of cytokine formation);

4) they act as growth factors and cell differentiation factors, thereby causing slow cellular reactions requiring synthesis of new proteins;

5) they form a regulatory network in which individual elements have a synergistic or antagonistic effect;

6) they have pleiotropic (semi-functional) activity and overlapping functions (pleiotropy is the effect of one substance on many objects, functions and properties).

Interleukins, monoclones and lymphokines are distinguished among cytokines. The specific name of the cytokine depends on which cells (leukocytes, monocytes, lymphocytes) predominantly synthesize the given cytokine, that is, cytokines are products of synthesis of leukocytes, monocytes and lymphocytes. For example, if cytokines are produced by leukocytes, they are called interleukins (IL), lymphocytes - lymphokines, monocytes - monokines. Part of the cytokines received the interleukin nomenclature and has a digital indication (from IL-1 to IL-16).

Other cytokines have left their original name due to their characteristic qualities and have the following literal designation: CSF (colony stimulating factors), OSM (ankostatin M), LTF (leukemia cell inhibitor), NGF (nerve growth factor), CNTF (ciliary neurotrophic factor), TNF (tumor necrosis factor).

Some lymphokines and monokines received interleukin nomenclature (systematization of cytokines according to the interleukin nomenclature was performed), not being derived from leukocytes, for example: - interleukin-4 (IL-4) is lymphokine because it is produced by T cells (T-lymphocytes), but at the same time it is included in the interleukin nomenclature and has the designation of interleukin-4.

Classification of cytokines. At present five main classes or families of cytokines are known, which are distinguished on the basis of the biological effect (function) or dominant effect on other cells:

1) *proinflammatory* cytokines (IL-1, IL-6, IL-8, IL-12, TRF-β transforming germinative factor) and *anti-inflammatory* (IL-4, IL-10, IL-11, IL-13 and etc.) having the biological effect of participation in the inflammatory reaction;

2) *tumor necrosis factor* (TNF) has a biological effect to influence the tumor process;

3) lymphocyte growth and differentiation factors (IL-7) have a biological effect to provide immune protection;

4) *colony stimulating factors* (CSF), which stimulate the growth of macrophage and granulocyte populations, and interleukins (IL-3, IL-5, IL-12) that participate in regulation of the growth and differentiation of individual cells;

5) factors that cause the *growth of mesenchymal cells,* have a biological effect to participate in regeneration of damaged tissues.

Major histocompatibility complex (MHC) has received this name because it is this cluster of genes that contains information about the proteins responsible for rejection reaction. Human MHC is located in the 6^{th} chromosome and consists of two classes: MHC I class and MHC II class.

Class I molecules are membrane glycoproteins consisting of a single polypeptide α-chain with a molecular weight of 45,000. The role of β-subunit is played by a non-covalently linked α-chain of a β2 –microglobulin molecule with a molecular weight of 12,000. The structural β2-microglobulin gene is located outside MNC, in another chromosome. α-chain consists of three extracellular domains (sites): hydrophobic, transmembrane, and short cytoplasmic. There are many allelic variants of the gene encoding α-chain of class I molecule, while the allelic polymorphism in β2-microglobulin appears only very weakly. As a result, the difference between separate individuals of the same biological species almost exclusively depend on α-chain polymorphism. A person has three loci encoding highly polymorphic α-chains of class I MHC molecules called HLA-A, HLA-B, and HLA-C.

Class II molecules are also membrane glycoproteins and consist of two homologous polypeptide chains with a molecular weight of 33,000-35,000 (heavy α-chain) and 27,000-29,000 (light β-chain), respectively. Each chain includes two extracellular domains (sites) that have a limited homology with the corresponding domains of α-chain of molecules of class I - immunoglobulin molecules and β2-microglobulin molecules. A person has three louses encoding class II antigens: HLA-DP, HLA-DQ and HLA-DR.

As well as in molecules of MHC I class, there are many allelic variants of class II antigens.

MHC produces other gene products. These molecules are called MHC class III proteins. These include three components of complement system: C_2 and C_4 proteins and factor B.

I.3. GENERALIZED PURULENT SURGICAL INFECTION (SEPSIS, SEPTIC SHOCK)

Over the recent decades, mortality in generalized forms of inflammation - severe sepsis and septic shock - has tended to increase. High rates of lethality make sepsis a topical medical problem. In its pathogenetic nature, sepsis is an immune failure, in which opportunistic flora becomes aggressive and causes generalized forms of infection.

Sepsis is annually diagnosed in 1,5 million patients in the world, in the USA in 750 thousand patients. There has been an increase in septic complications over the recent 20 years (US) from 82.7 to 240.4 cases per 100,000 inhabitants. More often sepsis complicates pulmonary, abdominal and urological infections. However, in 20-30% of clinical cases of diagnosed sepsis, the source of infection has not been detected.

To date, in the world (WHO) 500,000 patients die from sepsis every year (about 135,000 in the European Union, about 250,000 in the United States). In intensive care units of developed countries, approximately 30% of all cases of diagnosed sepsis end with death.

In the CIS countries, over the recent 5 years there has been an increase in mortality rates in surgical patients with severe forms of infectious complications, the mortality of patients with surgical sepsis is 65-89%, with pancreatogenic - 73%, in patients with septic endocarditis and myocarditis - about 70% , at development of purulent-septic complications after severe injuries - about 40%.

An increase in septic complications leads to an increase in the cost of treatment of surgical patients by 6 times. For example, in the US today about $ 17 billion are spent on the annual treatment of septic patients, in the EU countries - € 7.6 billion.

Current problems include new approaches to the diagnosis and treatment of sepsis. To solve these issues, the following principles should be complied with:

1. Recognition of the key role of immune disorders in the pathogenesis of purulent-septic complications, including sepsis.
2. Introduction to the list of criteria of multiple organ failure of indicators of immune disorders (laboratory signs of immunosuppression).
3. Application of modern drugs of substitution immunotherapy as a compulsory component of complex treatment.

Sepsis

This is a special form of the organism response to infectious agents.

Sepsis *is a non-specific infectious disease characterized by systemic inflammatory response syndrome that arises in the conditions of permanent or periodic invasion of microorganissms and their toxins from the focus of infection into the circulating blood, which leads to development of infectious MOD due to inability of the immune forces of the organism to limit the infection.*

Owing to publication of works on SIRS problem in 1991-2001, the recommendations of the consensus conference in Chicago (1991) were recognized as too broad and not sufficiently specific. At the recent conference in Washington,, 2001, devoted to development of a new approach to sepsis definition, the lack of complete identity between SIRS and sepsis was recognized; for practical medicine, it was proposed to use additional (as to SIRS) advanced criteria for diagnosis of sepsis. The latter consist of key and inflammatory changes, changes in hemodynamics, manifestations of organ dysfunction and indicators of tissue hypoperfusion.

Prior to the expanded criteria for the diagnosis of sepsis (until 2001), the diagnosis of "sepsis" was eligible in the presence of a focus of infection and two criteria. According to the decision of the 2001 conference (Washington), the diagnosis of "sepsis" is made in the presence of a focus of infection and the signs of organ dysfunction, which occur at least in one organ system in combination with reduction in tissue perfusion.

Thus, the diagnosis of "sepsis" can be made with the appearance of organ dysfunction at least in one organ system in a complex (in combination) with decreased tissue perfusion.

Classification of Sepsis (Consensus Conference, USA, 1991):

1) sepsis;
2) severe sepsis;
3) septic shock;
4) sepsis-induced hypotension;
5) multiple organ dysfunction syndrome;
6) refractory septic shock.

The current classification of sepsis used in all countries of the world is based primarily on clinical manifestations and was proposed at the Consensus Conference of the American College of Pulmonology and Society of Critical Care Medicine under the guidance of R. Bone - ACCP/SCCM in 1991.

1. ***Sepsis*** - the diagnosis of "sepsis" is made in the presence of two or more symptoms of SIRS in the proven infectious process (this includes verified bacteremia).

2. ***Severe sepsis*** - the diagnosis of "severe sepsis" is made in the presence of organ failure in the patient with sepsis (sepsis, combined with organ dysfunction, hypotension, tissue perfusion disorders, increased lactate concentration, oliguria, acute consciousness disorder).

3. ***Septic shock*** - severe sepsis with the signs of tissue and organ hypoperfusion and arterial hypotension, which is not eliminated by infusion therapy and requires catecholamines administration.

4. ***Sepsis-induced hypotension*** - reduction of systolic blood pressure below 90 mm Hg or by 40 mm Hg and more of "working" in persons with arterial hypertension in the absence of other causes. Hypotension is eliminated in a short time by infusion of solutions.

5. Multiple organ ***dysfunction syndrome (MOD syndrome***) - severe sepsis with dysfunction of two or more systems.

6. ***Refractory septic shock*** - persistent arterial hypotension, despite adequate infusion and the use of inotropic and vasopressor support.

Sepsis is also classified according to the following criteria: the pathogen nature, the focus location and the infection entry, the characteristics of the underlying disease that caused sepsis, as well as severity and duration of the course.

Sepsis classification

1. *The pathogen nature:*

a) bacterial-fungal (mixed, which means - double);
b) polybacterial;
c) sepsis caused by opportunistic pathogens: aerobes (gram-positive and gram-negative): staphylococcal, streptococcal, colibacilliary, pseudomonal;
d) sepsis caused by pathogenic microbes: mycobacterial, meningococcal, typhoid fever, plague.

2. *The focus location and the infection entry*: tonsilogenic; otogenic odontogenic; wound; gynecological.

Location sometimes suggests the etiology. If the entry is unknown, the sepsis is defined as cryptogenic.

3. *The characteristics of the underlying disease that caused sepsis*. Among all purulent-inflammatory diseases, dominating is surgical sepsis that occurs:

a) in case of infection generalization in victims of injuries and wounds;
b) as a complication of acute destructive pancreatitis;
c) as a postoperative complication (general surgical sepsis), after operations on the heart and vessels (angiogenic), on the abdominal organs (abdominal).

4. Depending on s*everity and duration of the course the following forms of sepsis are distinguished:*

In fulminant sepsis, the disease develops rapidly, in a few hours, in the most acute form a few days, in acute - the process may develop for several weeks.

Chronic sepsis is characterized by a sluggish course and the presence of obscure changes that are observed for months.

Recurrent sepsis is characterized by a change in the periods of exacerbation, when all symptoms get a vivid manifestation, and periods of remission with the absence of any noticeable clinical signs.

According to the decisions of the Consensus Conference, it is recommended not to use the terms that do not have a specific content load, such as "septicemia", "sepsis syndrome" and "refractory septic shock".

The diagnosis of organ failure is based on the agreed criteria, which formed the basis of SOFA scale (Sepsis Oriented Failure Assessment).

Clinical manifestations of sepsis. The clinical presentation of sepsis is extremely diverse and depends on the location of the primary focus and dysfunction of organs and systems. At the forefront may be the clinical presentation of severe polytrauma or burn, septic pneumonia or gastroduodenal bleeding, symptoms of peritonitis or pancreatic necrosis, toxic hepatitis, nephritis, or myocarditis.

Early symptoms include SIRS, primary focus, bacteremia.

The late symptoms of sepsis include signs that indicate the development of sub- or decompensation of the functions of various organs and systems due to intoxication or septic metastases and bacteremia or their combination.

The clinical manifestations that allow a physician to suspect sepsis development are divided into general and local.

Common symptoms include deterioration of the general condition, headache, confusion or even loss of consciousness, fever over 38° C, chills, weight loss, hemorrhagic rash. Dysfunction of parenchymal organs (kidneys, liver) with development of hepatitis and jaundice, enlargement of the spleen are typical. Gastrointestinal disorders include septic diarrhea, lack of appetite, nausea, vomiting.

Local signs include changes in the infected wound - pallor of tissues, edema, poor granulation, poor secretions that are dirty-cloudy, or putrefactive. At present, there are no objective criteria for the changes in the local infection focus, the presence of which reliably indicates generalization of the process in the body. However, in all cases of sepsis, the clinical presentation is confirmed by the changes in the focus. The type of septic wound can be characterized as follows: swelling of the tissues surrounding the wound significantly increases, there is a marked swelling of the edges of the wound itself, the bottom of which is covered with purulent-fibrinous coat of white or gray color, which is difficult to separate.

In some cases there is abundant purulent discharge, the smell of which one can suggest superiority of one or another microflora. Thus, the smell of foul meat is most characteristic of *Clostridium sporogenes; E. coli communis and Proteus vulgaris* smell ammonia and urine, pathogens of putrefying infection and bacteria of the fusiospirochetos group smell mold and putridity. Yellow and white pus is typical for staphylococcal infections, green for green streptococcus, dirty gray for intestinal bacilli and vulgar proteus, green and blue for Pseudomonas aeruginosa, etc.

The granulation in the wound is pale gray, bleed easily, which creates additional entries for re-infection. In the case of joining anaerobic infection or exacerbating the purulent process, a feeling of fullness appears in the wound.

Due to polymorphism of the general signs of the disease (jaundice, hemorrhagic rash, high body temperature, enlargement of the spleen, etc.), the differential diagnosis should made with typhus, tuberculosis, brucellosis, malaria, etc. The characteristic signs of sepsis (presence of local focal infection and characteristic local manifestations of the process in the purulent wounds) are important for the differential diagnosis. Complete and rapid elimination of the general and local manifestations of inflammation after opening of the purulent foci serves a distinctive feature of fever in abscess,

phlegmon, etc. In the absence of subsiding or disappearance of general and local manifestations of inflammation, the diagnosis of sepsis can be made for sure.

Sepsis immunopathogenesis *develops in three stages.*

1. Local production of cytokines in response to infection. The leading role is played by cytokines that control the processes of immune and inflammatory reactivity. Cytokines act in the inflammation focus and in the lymphoid organs, participating in protection of cells from pathogenic microorganisms.

2. Release of small amounts of cytokines into systemic circulation. The developing acute phase is controlled by proinflammatory mediators (IL-1, IL-6, IL-8, tumor necrosis factor-TNF) and anti-inflammatory cytokines (IL-4, IL-10, IL-13, etc.). The balance of relationships between pro- and anti-inflammatory cytokines normally provides wound healing, destruction of pathogenic microorganisms, support of homeostasis.

3. Generalization of inflammatory reaction. At pronounced inflammation or its systemic failure, certain cytokine species (TNF-a, IL-1, IL-6, IL-10, TGF-p, INF-γ) entering the systemic circulation accumulate there in the amount sufficient for impairment of permeability and functions of the capillary endothelium, onset of DVC syndrome, emergence of distant foci of systemic inflammation, MOD development. Within the first (initial period) of the inflammatory reaction generalization, which is called *period of hyperinflammation*, ultrahigh concentrations of proinflammatory cytokines, nitric oxide are released with development of shock and MOD. In this case, compensatory release of anti-inflammatory cytokines is observed. Their concentration gradually increases with a parallel reduction in proinflammatory cytokines. To designate this state, in 1996 R.C. Bone suggested the term CARS (compensatory antinflammatory response syndrome). At a balanced course, CARS suppresses systemic inflammatory reaction and leads to homeostasis recovery - *period of homeostasis recovery*. At pronounced or prolonged course, CARS causes development of immunosuppression, which is clinically manifested by chronization or dissemination of infection, repair process disturbance, endotoxicosis aggravation and formation of late polyorgan failure - *period of sepsis development.*

Of great importance in sepsis development is nitrogen monoxide (NO). High concentrations of NO cause decline in peripheral vascular tone, combined with reduction in vascular wall response to central and humoral stimuli.

SIRS development at sepsis leads to reduction in peripheral vascular tone, relative deficiency of total blood volume due to increased vascular permeability, sequestration of the liquid fraction of blood in the microcirculatory bed and early progressive myocardial dysfunction. Severe sepsis turns to septic shock.

In the initial period of shock, histamine, prostacyclin, bradykinin, released as a result of degranulation of TC or platelets are also involved in the pathogenesis of vascular insufficiency.

Thus, in case of microcirculation disorder and destabilization of central hemodynamics, circulatory, transport hypoxemia and hypoxia occur. "Centralization" syndrome develops, which leads to shunting the blood in the lungs and respiratory distress syndrome, which increases hypoxia.

Organ dysfunction in sepsis. In sepsis, dysfunction of liver, kidney, respiration and circulatory system occurs, syndrome of disseminated intravascular coagulation is formed.

Liver dysfunction in sepsis. Damage with the endotoxins of the liver sinusoids is accompanied by their sclerosis. Dystrophic changes in stellate reticulocytes and their destruction contribute to entry of microbes to the vascular bed; failure of cells in the reticuloendothelial system (RES) does not provide transformation of toxins, which leads to functional, and then to irreversible processes in hepatocytes with disorders of their detoxification functions.

In addition, endotoxins, on the one hand, stimulate phagocytic activity of leukocytes and reticulocytes, and on the other hand, microbial decomposition products block the ability of phagocytes to remove excess fibrin from the lumen of sinusoids. This may result in granular and fatty degeneration of hepatocytes. Clinically, this is manifested by development of hepatocellular insufficiency with impaired liver function.

The changes in the liver in sepsis are characterized as *ischemic hepatitis*. The main pathogenetic mechanisms of the parenchyma damage are associated with fixed liver macrophages or Kupffer cells. The sharp increase in the number of bacteria in the intestine, translocation of bacteria and toxins through the intestinal wall into the portal circulation, activate Kupffer cells and release of cytokines that affect RNA interleukins, TNF-a, leukotrienes, and B_4.

The cytokines activate the action of intercellular adhesive molecules and endothelial leukocyte adhesive molecules. The result of these pathogenetic mechanisms is migration of neutrophils to the cellular parenchyma. Neutrophils cause cell damage by releasing oxygen radicals, which is followed by lipid peroxidation in cell membranes.

The combination of the effect of activated Kupffer cells and neutrophils leads to microcirculatory disorders, microthrombosis and hypoxia. Main metabolic mechanisms in the liver are impaired: inhibition of adenylate cyclase, decreased gluconeogenesis, increased lipogenesis, reduced liver perfusion, hypoxia, lactatoacidosis, elevation of serum AlAT and AsAT.

This results in progressive cholestatic jaundice, hypoglycemia, encephalopathy, coagulation disorders, arrest of oxidative phosphorylation in mitochondria, membrane dysfunction and protein synthesis impairment, ion balance of hepatocytes.

Kidney dysfunction in sepsis. Kidney damage occurs as a result of the following:

1) hemodynamic disorders associated with necrobiosis and necrosis of the epithelium of the convoluted tubules in septic shock;

2) under the influence of endotoxins and activation of renin-angiotensin system and impairment of the organ blood flow followed by focal nephritis;

3) damage to the renal tubules with immune complexes and granulocytes.

The changes in the kidneys in sepsis are characterized as *acute tubular necrosis*. In the majority of patients, it leads to acute azotemia and causes severe disorders of water-salt metabolism. Its pathogenesis can be described as follows:

1) lack of extracellular fluid volume as a cause of hypovolemia;

2) influence of endogenous toxins on the kidneys often leads to pathological intensive fluid filtration from the lumen of the tubules to the renal interstitium, and then to the lumen of the microvessels through the damaged walls of the tubules;

3) pathological spasm of the afferent arterioles of the glomeruli;

4) inhibition of the local prostaglandin system of blood flow volume velocity regulation in the kidneys as a result of endotoxemia, which increases the spasm of the afferent arterioles.

Respiratory disorders in sepsis. At sepsis, the lungs are the first to be damaged. One of the leading factors in the pathogenesis of respiratory disorders is hypoxia of the pulmonary parenchyma due to its toxic damage; it is closely related to the disorders of the functional properties of the alveolar-capillary membrane. This leads to impairment of surfactant synthesis. Reducing surfactant amount decreases the surface tension of the alveoli and provokes their tendency to collapse.

At the beginning of the process development, destructive disorders dominate - the obstructive form of respiratory failure. Clinically, this is manifested by anxiety, tachypnea, stenotic breathing, cyanosis. In the catabolic phase of the process, respiratory failure is determined by damage to the lung parenchyma in the form of metastatic pyo- and pneumothorax.

The main pathogenetic factors of *respiratory distress syndrome* are direct and indirect damages of the vascular endothelium with biologically active substances (serotonin, histamine, prostaglandins), the influence of endotoxins, vasoactive peptides of microbial lysis, the action of which lead to leukocyte and platelet microembolism, which impair microcirculation in organs and tissues. This leads to disturbance of resistance in the pulmonary circulation and disorder of capillary permeability, which creates conditions for development of interstitial edema, consolidation of pulmonary parenchyma, development of respiratory distress syndrome in adults (*shock lungs*). Clinically, this is manifested by destructive pneumonia (bullous, disseminated form).

Disorders in the circulatory system. Hemodynamic disorders consist of three components:

1) systemic influence on the peripheral vasomotor tone and changes associated with this, the load on the myocardium;

2) changes in the myocardium functioning under the influence of neurohumoral factors of sepsis;

3) local effects of the infectious agent on the heart, including direct damage to the myocardium.

Based on the above, circulatory disorders in sepsis are determined by three main factors: changes in vasomotor tone and load on the myocardium, neurohumoral and endotoxic lesions. A complex of damaged endothelial cells, platelets, microemboli, elevated levels of NO, vascular tone mediators - prostacyclin, histamine, bradykinin, which cause redistribution of ions and disturbance of vascular sensitivity to endogenous vascular tone mediators, participate in this process. Additional factors that can enhance the vascular tone blockade are eicosanoids, decrease in the number of α-receptors in the vascular walls, and reduction of calcium in the contractile system of smooth muscles.

One of the main clinical manifestations of hemodynamic disorders in sepsis is hyperdynamics of the cardiovascular system, which is characterized by increased cardiac output and decreased vascular resistance.

Two types of hemodynamic disorders are distinguished: hyperdynamic and hypodynamic.

Hyperdynamic type of blood circulation is characterized by increase in comparison with the controls of minute heart rate (MHR) by 32%, systolic index (SI) by 35%, reduction of total peripheral vascular resistance (TPVR) by 65%.

In *hypodynamia*, reduction compared to the controls is observed in MHR by 50%, SI at 55% with an increase in TPVR by 54%.

Hyperdynamic or hypodynamic syndromic disorders are caused by the changes in the capillary bed. In hypodynamic syndrome, minute heart volume becomes low, and the peripheral resistance is high, precapillary vasoconstriction restricts the flow of oxygen into the cell, arterio-venous oxygen difference as well as acidosis increase. Hyperdynamic syndrome is characterized by increased minute heart volume and decreased peripheral vascular resistance. The arteriovenous oxygen difference decreases.

Hyperdynamic variant is observed in the early stage of sepsis, hypodynamic - in the later.

Septic shock

*The diagnosis of "**septic shock**" (turns to DVS-syndrome, which is the most common cause of fatal outcome) can be made in the presence of four key features: 1) clinical evidence of the presence of infection; 2) signs of systemic inflammatory response syndrome (≥2 SIRS criteria); 3) arterial hypotension, which is not compensated by infusion, or there is a need for the constant use of vasopressors to maintain blood pressure above the critical level; 4) clinical and laboratory signs (indicators) of organ hypoperfusion (hyperlactatemia mmol L, slow filling capillary syndrome, limb marbling).*

Clinical presentation of septic shock. The state of shock in septic patients develops due to inadequate perfusion of the internal organs, which is the result of acute circulatory failure. Sharp impairment of blood supply to the tissues is accompanied by development of tissue hypoxia. In this state, even intensive infusion therapy is not able to maintain blood pressure above the critical level; constant administration of vasopressor drugs is required.

Septic shock is diagnosed if hypotension cannot be controlled (BP_{syst} <90 mm Hg or it is 40 mm lower the baseline, or BP_{m}.< 60 mm Hg with absence of other hypotension causes).

The determinant for the diagnosis of "septic shock" is the fact of a previous infection and the presence of SIRS criteria. Other causes of hypotension that should be excluded are taking respective medications, generalized myocardial infarction, massive blood loss and severe traumatic damage to organs and tissues.

Clinical presentation of septic shock is characterized by persistent arterial hypotension, tachycardia, often arrhythmia, general retardation, paleness of the skin, sticky sweat. In the majority of patients, tachycardia alternates with bradycardia with arrhythmia and pronounced cardiac deficiency, oliguria that turns into anuria occurs.

Three types of septic shock are distinguished.

*Type 1 is **severe shock***, in which death occurs due to damage to the myocardium by the endotoxin.

Type 2 is ***"warm" shock,*** in which the level of pressure is within 70 mm Hg. Successful outcome is possible with a timely adequate therapy; "***Cold" shock*** is accompanied by uncontrolled low blood pressure, vegetative disorders, rapid decompensation development.

Type 3 is ***uncontrollable shock*** that contains the signs of the previous types and is incurable.

Sharp impairment of blood supply to the tissues and increasing tissue hypoxia form progressive multiple organ failure with multiple organ damage. First and foremost, respiratory distress syndrome and acute renal failure develop. Septic shock is also characterized by changes in the coagulating function of the blood, which leads to multiple hemorrhages, including organ localization. Septic shock and organ dysfunction are regarded as sepsis complications aggravating the patient's state, with a very unfavorable life prognosis.

Three phases can be traced in circulatory disorders at septic shock: hyperkinetic, hypokinetic, and terminal. In the initial *hyperkinetic phase*, the overall peripheral vascular resistance is reduced, and the values of cardiac output are normal or even slightly increased. In this case, arterial and venous pressure drops sharply. In the second *hypokinetic phase* reduction in both peripheral resistance and cardiac output is observed. In the third *terminal phase*, the phenomena of heart failure, hypoxia, acidosis, and disorders of the water-salt balance progress.

Generalized infectious complications in surgical patients can have a clinical course of septic shock from the very beginning. This is possible in the case when virulent pathogens massively enter the general blood flow, bypassing the natural barriers of non-specific macroorganism resistance. A similar clinical manifestation can also be observed in traumatic endo(auto)toxicosis and in case of massive death of gram-negative pathogens with release of large quantities of bacterial lipopolysaccharide, which functions as endotoxin with a potent hypotensive effect. If the patient is in a critical condition, originating from prevailing systemic microcirculation disorders, and later infection, severe sepsis may develop at once.

Diagnosis of septic shock and fulminant sepsis. The principles of diagnosis of septic shock and fulminant sepsis differ significantly from the diagnostic methods used in other forms of sepsis.

The diagnosis of septic state severity can be adequate when it is primarily based on the clinical presentation. This is due to: 1) the presence of clear clinical symptoms, which suggest severity of the patient's condition; 2) the need to diagnose these types of sepsis and start treatment no later than the first 6-8 hours after the clinical signs appearance, otherwise the treatment effectiveness will decrease.

Both forms can occur at any stage of the infectious process. The doctor should rely on the formal signs of septic shock according to the parameters proposed by R. Bone: septic shock = SIRS + MOD + hypotension or "severe sepsis" + hypotension, may not be sufficient for several reasons. First, these signs often coincide; second, due to the speed of development of the set of pathological reactions, it is difficult to diagnose them; third, the signs may appear against a background of a relatively good clinical state of the patient.

Differential diagnosis of septic (endotoxin) shock and syndrome of toxic shock.

The etiological factor of septic shock, as a rule, is gram-negative microbes. However, due to the prolonged existence of the purulent focus, the clinical presentation of septic shock may not depend on the type of the pathogen identified in the hemoculture.

Development of *septic (endotoxin, infectious-toxic) shock* manifests by primary microcirculation disorder, which can be estimated by the state of peripheral and central circulation. The following symptoms are typical: marble skin, collapse spots, drop of blood pressure, thread-like disappearing pulse, clear heart sounds; high body temperature, which rapidly drops to normal; the patient's euphoria is altered by inhibition. This condition is designated as a fulminant gram-negative (meningococcal, salmonellosis, escherichiosis, pseudomonas) sepsis.

Shock of infectious origin can develop without sepsis in bacterial (typhoid) or viral (influenza) infections, as well as entry of a large number of bacterial exotoxins to the organism. Shock may be induced by enterotoxin E of staphylococcus aureus or diphtheria toxin. In these cases, the shock is due to microbial toxins (toxin of toxic shock syndrome 1 and enterotoxin F), and autolysis products of the damaged tissues, which trigger a complex cascade of cytotoxic immune reactions with a predominant lesion of the vascular endothelium. Similar systemic reactions may also be due to the

infection with S. *pyogenes* and coagulase negative staphylococci producing exotoxins with the properties of superantigens. Circulatory disorders that increase with the influence of these factors, lead to a secondary increase in the intestinal barrier permeability and additional entry to the general circulation of endotoxins from the gastrointestinal tract. Toxic shock syndrome development is associated with wound infections, postoperative complications, mastitis, postpartum endometritis.

The pathogenesis of fulminant sepsis, caused by gram-positive microorganisms, more often staphylococcal microflora, is based on the primary cardiac impairment and reduction of its contractile ability (primary disturbance of central hemodynamics) due to the effects of staphylococcal exotoxin (cardiotropic poison). Fulminant sepsis is characterized by the triad of clinical symptoms:

1) a sharp elevation of the body temperature to 39-41˚C;

2) early development of acute left ventricular failure (cardiac asthma, pulmonary edema, expansion of the heart borders, dullness of the heart tones);

3) fear of death.

The tactic of treating patients with fulminant (gram-positive, usually staphylococcal) sepsis is aimed at eliminating heart failure and urgent nonspecific and specific detoxification (neutralization) of staphylococcal exotoxins.

Cell apoptosis

Cell apoptosis is considered a mechanism of cell damage.

Cell apoptosis *is an active process, different from necrobiosis, during which a program of self-destruction of a cell is implemented (programmed cell death).*

After the ligand-receptor interaction initiating apoptosis or minor damage to the cell membrane with superoxide radicals, it is disorganized; phosphatidylserine molecules appear on the surface. Then endonuclease, causing intercellular DNA breaks is activated. After the proteolytic cleavage of the molecule, the pro-enzymes pass into the active form and participate in the implementation of various pathways of apoptosis. With the increasing proteolysis of the cytoskeleton, fragmentation of the cytoplasm occurs. In the cell nucleus intercellular ligaments are destroyed and fragmentation occurs. In the final phase of apoptosis, apoptotic bodies are formed and undergo autophagocytosis. The cell passes away "without a scandal" and without leaving the trace.

The mechanism of programmed cell death is important for embryogenesis and morphogenesis. Normally, apoptosis limits the processes of cell proliferation and tissue growth, as well as participates in hormone-dependent involution. The launch of the apoptotic program is carried out by two main mechanisms:

1) binding of the ligands initiating apoptosis to the cytoplasmic cell membrane receptors;

2) by loss of apoptosis-suppressive activity in special proteins.

Apoptosis is a genetically determined process. The genes that implement the apoptosis program are activated by a receptor-mediated mechanism. The role of genetic inducers in the process of cell apoptosis, activated in response to a receptor signal, may be played by the genes Fas/APO-1, max, c-myc, ced-3, p53 b and others. Initiation of apoptotic processes in a cell can cause activation of long-lived cytoplasmic RNA. Suppressing the expression of some genes, such as the bcl-2 also causes apoptosis. The products of these genes substantially modify the biochemical processes in cells, for example, protein ARO-1 enhances formation of active free radicals, and the product of bcl-2 gene regulates transfer of calcium to the cytoplasm. Other products of the genes regulating apoptosis trigger neutral proteases of cytosol and can directly interact with nuclear DNA.

In generalized inflammatory reaction, activators of lymphocyte apoptosis are TNFα and glucocorticoids. Virtually all cytokines that are excessively produced in SIRS by activated mononuclear cells, including interleukins and interferons, can act as inducers of apoptosis in immunocompetent cells. Moreover, in cells of one type, one or another cytokine starts apoptosis, and in cells of another type inhibits it.

Circulating neutrophils when triggered by a generalized inflammatory reaction change their characteristics, deform and easily penetrate into the pulmonary capillaries, where under the action of chemokines (for example, IL-8 produced by alveolar macrophages) acquire the ability to migrate to the

lung tissue. In the lung tissue, when interacting with bacterial stimuli, neutrophils are activated and free radicals are released during the reparative explosion.

The limited intensity of neutrophil apoptosis allows them to participate in the pulmonary alteration for a long time and to amplify SIRS. Tissue-specific growth factors and hematopoietic cytokines act as apoptotic inhibitors for their target cells. continuation of neutrophil life in sepsis is associated with low intensity of their apoptosis due to the high activity of colony-stimulating factors of blood cells - G-CSF and GM-CSF. In parallel with the decrease in concentrations of these colony-stimulating factors, the intensity of neutrophil apoptosis is restored.

Active free radicals are also capable of initiating apoptosis. There is a lot in common in damaging effects between processes of necrobiosis and cell apoptosis. In response to minimal damage or damage that does not cause rapid development of deep hypoxia and severe deficiency of energy substrates, the cells are not in the state of necrobiosis, but may switch on a special biological program for self-destruction and react with apoptosis. With moderate damaging effects on the cell (criteria have not been defined yet) and the absence of hypoxia, there is reduction in the mitochondria transmembrane potential and generation of reactive oxygen species. If the cell antioxidant system does not compensate for the changes in the redox potential, the process already started in the cell progresses up to the cell death. In the absence of a pronounced energy deficiency and preservation of the cell genetic apparatus, the program of altruistic suicide - apoptosis - is implemented, and in the case of deep hypoxia and severe DNA damage, cell necrobiosis is initiated. Cell death as a result of apoptosis is not accompanied by leukocyte demarcation and perifocal inflammation, as implementation of programmed suicide helps to avoid production of significant amounts of damage mediators.

Examination of the patient with sepsis includes physical examination of organs and systems, assessment of the general condition, changes (location, extent of lesion) in the infection focus and the search for metastatic foci.

The modern methods of diagnosis include ultrasound, X-ray examination (including contrast), helical computed tomography, magnetic resonance imaging, angiography, etc.

Criteria for assessing sepsis severity. The patient's status is assessed using non-invasive and invasive methods.

Non-invasive methods. The state of hemodynamics is assessed by the level of blood pressure (BP) by the method of Riva-Rocci -Korotkoff with calculation of systolic (BP_{sis}) and diastolic (BP_d) indices, pulse and median-dynamic BP (BP_{med}), heart rate (HR), electrocardiography data (ECG), echocardiography, Doppler ultrasound, integral rheography of the body.

Cerebral hemodynamics is evaluated by the method of rheoencephalography, bioelectric activity of the cerebral cortex - by means of electroencephalography and computer brain mapping. The state of the respiratory system is assessed by severity and nature of dyspnea. Saturation of hemoglobin with oxygen (SaO_2) is evaluated by pulse oximetry, blood gases (PaO_2) - by transcutaneous monitoring. The body temperature is determined in degrees Celsius.

Invasive methods. The state of hemodynamics is estimated by the level of arterial pressure by the method of peripheral arteries cannulation, CVP - by cannulation of the upper vena cava. Full invasive monitoring is performed using Swan-Ganz catheter through the central or large peripheral vein. Depending on the location of the distal end of the catheter, right atrium pressure (RAP), right ventricle pressure (RVP), pulmonary artery pressure (PAP), including pulmonary capillary wedge pressure (PCWP) are measured.

Blood count is done with stained capillary blood smears; biochemical, immunological, bacteriological indices are assessed by studying venous blood; blood gases, indicators of the acid-base state - by studying arterial, venous and capillary blood.

Diuresis per unit of time is estimated using bladder catheterization.

Regional perfusion activity is evaluated clinically by organ function. Their functional activity may indicate myocardial ischemia, renal dysfunction, CNS dysfunction. Liver parenchyma damage is accompanied by increased activity of blood serum transaminases, lactate dehydrogenase and bilirubin

content, hypopalbuminemia, and decreased coagulation factors. Hypoperfusion of organs is sometimes manifested by stress ulcers and malabsorption syndrome.

The objective assessment of the patient's state is done using APACHE II (Acute Physiological Chronic Health Assessment) scale, which allows to assess acute physiological disorders and chronic health disorders, that is, it takes into account physiological effects of acute illness, premorbid condition, and the age of the patient. This scale is based on assessment of the physiological state of the patient (APS - Acute Physiological Score) according to 12 parameters. Quantitative indicators are calculated based on known and available clinical and laboratory data that correlate with morbidity and mortality. A score less than 10 indicates a relatively mild course of the disease, and over 20 - critical state of the patient.

The Multiple Organ Dysfunction Score (MODS) allows to evaluate severity of multiple organ damage, regardless of the cause, and to predict mortality based on the number of organs and systems involved in the pathological process. The state of 6 important systems is evaluated. For 5 of them, clinically tested integral indicators are used. To evaluate the blood circulation, it is suggested to use a specially adjusted integral hemodynamic parameter: "pressure adjusted heart rate (PAHR)", which is the derivative of heart rate and the ratio of CVP to BP_m.

Multiple organ disorder syndrome (MODS) is the presence of changes in the function of the internal organs in patients when homeostasis cannot be supported without external intervention. On the basis of laboratory and functional tests, disorders of organs function (lungs, heart, liver, kidneys, gastrointestinal tract, blood system, endocrine organs, and central nervous system) are determined. Severity of MODS is assessed using APACHE scale, simplified physiological scale (SAPS), or by specific scales to assess the organ function disorder:

1) MODS scale - dysfunction of several organs;

2) SOFA scale - assessment of organ failure in sepsis;

3) LODS scale - logical scale for assessing organ dysfunction.

These scales are based on assessment of organ dysfunction (whether present or not) and a stepwise assessment of organ function disorder.

The assessment of a patient's condition in MOD allows:

1) to conduct monitoring with the purpose of determining MOD severity;

2) to receive a final evaluation for a certain period of time in order to evaluate MOD severity;

3) to get a combined assessment taking into account lethality in order to determine the impact of MOD severity on mortality;

4) to obtain an overall assessment of MOD severity in patients in this institution in order to determine the quality of treatment.

Laboratory diagnosis of sepsis and immunosuppression. Laboratory findings are of considerable importance, they include blood count and blood biochemistry. In this case, anemia of varying severity should be expected (almost half of the patients may have hemoglobin content below 80 g/l). Thrombocytopenia is possible. As a rule, marked leukocytosis with a sharp shift to the left or lymphopenia is noted. Changes in neutrophils in sepsis consist of toxic grains, the appearance Doehle's bodies and vacuolization. Determining serum electrolytes, urea level, creatinine, liver parameters allow MOD monitoring.

Important prognostic tests are *markers of systemic inflammation* (increased blood levels of procalcitonin, C-reactive protein, and interleukins -1, 6, 8, 10 and tumor necrosis factor (TNF).

Diagnosis of immunosuppression in septic patients includes clinical signs of general immunosuppression and diagnosis of immunosuppression by laboratory methods.

The clinical sign of general immunosuppression is appearance of secondary septic centers and/or development of nosocomial pneumonia in patients with purulent-septic complications after adequately performed surgical rehabilitation and against a background of antibiotic therapy.

Diagnosis of immunosuppression by laboratory methods. There is a direct correlation between the degree of endo(auto)toxicosis and general immunosuppression. Therefore, laboratory markers of auto(endo)toxicosis can be used as indirect diagnostic signs of general immunosuppression:

1) appearance of toxic grains in neutrophils;

2) increase in leukocyte intoxication index (LII);

3) increase of blood plasma concentration of medium weight peptides.

Laboratory criteria for diagnosis of immunosuppression in sepsis include the following:

1) absolute lymphopenia with peripheral blood lymphocyte content $<1.2 \times 10^9$/l;

2) relative peripheral blood content of HLA-DR-positive monocytes <30%;

3) expressed immunosuppressive activity of the blood serum with suppressor activity index <0.8;

4) apoptotic lymphocytes content - 10%;

5) apoptotic neutrophils content > 18%;

6) cytokine imbalance (IL-1Ra/ NNFa) > 10.

These laboratory criteria, in combination with clinical signs, accurately determine the degree of endotoxicosis severity. Thus, the average degree corresponds to LII <3,0; severe - 3.0 <LII <6.0; extremely severe - LII> 6.0.

Immunosuppression is also characterized by a concomitant cytokine imbalance with a significant predominance of immunosuppressive cytokines, which can be considered an additional criterion for general immunosuppression.

Laboratory criteria of general immunosuppression are highly informative. However, if septic patients do not have lymphopenia, reliable diagnosis requires laboratory support at the level of modern immunological analysis, which involves mandatory use of:

1) culture method of blast-transforming mononuclear cells;

2) immunoenzyme methods for determining cytokine content in serum and culture media;

3) flow laser cytofluorometry.

Taken together, these methods allow to evaluate the processes of cell proliferation, cytokine production, severity of cytokine imbalance, as well as the processes of apoptosis in cells that provide mechanisms for immunoreactivity.

Algorithm of the main signs of significant immune dysfunction:

1) clinical manifestations of infectious syndrome (presence of two or more SIRS criteria);

2) lymphopenia (decreased ($\leq 1.2 \times 10^9$/l) absolute number of lymphocytes in the peripheral blood);

3) decreased (<20%) relative number of CD_3-positive lymphocytes in the peripheral blood.

Algorithm for assessing immune disorders severity:

1) determining of subpopulation imbalance of T-lymphocytes by means of estimating the absolute number of cells in subpopulations with the stenotypic markers of SD_4+ and CD_8+ cells and calculating t immunoregulatory index (ratio: CD_4+/CD_8+) (in sepsis, the number of cells in subpopulations decreases, which is accompanied by reduction (<1.0) of immunoregulatory index);

2) assessment of surrogate markers level of septic inflammation in the blood (C-reactive protein, IL-6, procalcitonin, CRP - in sepsis, blood content of these markers increases > 2 standard deviations from normal values);

3) assessment of bactericidal and phagocytosis ability of peripheral blood polymorphonuclear and mononuclear leukocytes (at later stages of septic process the values of the both functional parameters is significantly reduced);

4) evaluation of proliferative and other functional activity of peripheral blood mononuclear cells - mitogen-induced proliferation of lymphocytes, IL-2 production in culture of mononuclear cells in response to mitogen, HLA-DR expression level by circulating monocytes, and relative number of monocytes expressing activated marker A-DR (in sepsis, all of these functional characteristics are significantly reduced);

5) determining the number of apoptotic lymphocytes in the peripheral blood and assessment of the level of spontaneous and mitogen-induced lymphocytic apoptosis in vitro (in sepsis the number of apoptotic lymphocytes in the peripheral blood significantly increases as well as the level of lymphocyte activation apoptosis in the cell culture).

The above algorithm for assessing severity of immune disorders is recommended for practical use in patients with purulent-septic pathology. The same immune indices can be used in monitoring the therapy effectiveness.

Expressed lymphopenia, which is estimated in absolute amount of lymphocytes in peripheral blood, against a background of leukocytosis and stab neutrophil shifts, is an absolutely reliable laboratory indicator of severe immunosuppression in patients with purulent-septic pathology, indicating development of a life-threatening immune system failure.

A very important method of sepsis diagnosis is *microbiological investigation* (microscopy and cultures) of blood, urine, sputum, secretions from the wounds, pleural cavity, and also tissues of purulent focus.

It is necessary to carry out at least 4 blood samplings at intervals of at least 20 minutes from the peripheral vein (10-20 ml) prior to antibiotics administration. When administering antibiotic therapy, it should be interrupted for 24 hours, after which sampling is carried out.

It is well-known that even in the most severe patients, the frequency of positive results in culture does not exceed 50%.

Bacteria is not a mandatory attribute of sepsis. Its absence should not influence the diagnosis of sepsis in the presence of the above criteria of sepsis.

The presence of microorganisms in the blood in people without SIRS is considered transient bacteremia.

Table 1

Advanced criteria for sepsis diagnosis

Infection combined with the following changes
Key changes:
Fever (Sublingual temperature> 38^0 C)
Hypothermia (sublingual temperature < 36^0 C
Heart rate> 90 beats/min (> 2 standard deviations from age norm)
Tachypnea
Consciousness disorder
Edema or the need to achieve a positive water balance (> 20 ml/kg for 24 hours).
Hyperglycemia (> 7.7 mmol/L) in the absence of diabetes mellitus
Inflammatory changes:
Leukocytosis> 12×10^9/l
Leukopenia <4×10^9l
Shift of the cellular formula towards immature forms (> 10%) with normal leukocyte content
C-reactive protein> 2 standard deviations
Procalcitonin> 2 standard deviations
Hemodynamic changes*:*
Arterial hypotension: BP_{sys} < 90 mm Hg, BP_{mid}< 70 mm Hg.
Reduced BP_{syst} by over 40m m Hg (in adults)
Reduced $BP_{syst.}$ by 2 and more standard deviations from the age norm
Saturation SvO_2 > 70 %
Heart index > 3.5 l/min/m^2
Organ dysfunction manifestations:
Arterial hypoxemia - PaO_2/FiO_2 < 300
Acute oliguria < 0.5 ml/(kg×g)
Creatinine elevation over 44 μmol/l (0.5 mg %)
Coagulation disorders: APTT > 60 s or MNR > 1,5
Thrombocytopenia < 100×10^9/l
Hyperbilirubinemia > 70 mmol/l
Intestinal obstruction (absence of intestinal murmur)
Tissue hypoperfusion indicators:
Hyperlactatemia > 1 mmol/l
Decelerated capillary filling syndrome, limb marbling
Notes: $BP_{syst.}$ – systolic blood pressure, $BP_{m.}$ – mean blood pressure. In children and infants arterial hypotension is a late manifestation of shock; APTT – activated partial thromboplastin time; MNR -

international normalized ratio.

Table 2

The system for septic multiple organ failure assessment SOFA (J. L. Vincent, 1996)

Indicators	Severity degree				
	0	***1***	***2***	***3***	***4***
Respiratory system					
Respiratory index P_AO_2/FiO_2*	>400	<400	<300	<200 with respiratory support	<100 with respiratory support
Blood coagulation system					
Thrombocytes $\times 10^3$/ml	**>150**	<150	<100	<50	<20
Liver					
Bilirubin, mg/dl	<1.2	1.2-1.9	2.0–5.9	6.0-11.9	>12
Bilirubin, μmol/l	<20	20-32	33-101	102-204	>204
Cardiovascular system					
Hypotension	**Absent**	Mean BP <70 mm Hg	Dopamine < 5 or dobutamin at the same dose **	Dopamine > 5 or adrenalin <0.1 or noradrenalin <0.1**	Dopamine >15 or adrenalin > 0.1 or noradrenalin <0.1**
CNS					
Glasgow scale	15	13-14	10-12	6-9	<6
Kidneys					
Creatinine, mg/dl	<1.2	1.2-1.9	2.0–3.4	3.5–4.9	>5.0
Creatinine, μmol/l	<110	110-170	171-299	300-440	>400
Urination, ml/day				< 500	< 200

Note. FIO_2* - oxygen fracture (in the gas mixture). ** administration of sympathomimetics at least for an hour (μg/kg/min).

Table 3

Generalized organ dysfunction criteria

Hemostasis system dysfunction	Consumption coagulopathy: fibrinogen degradation products > 1/40: dimers > 2; prothrombin index < 70 %; thrombocytes< 100 (since 2001) – 150×10^9/l; fibrinogen < 2 g/l Dynamic changes: reduced thrombocytes > 50 %, increased prothrombin time > 20 %; since 2001 - APTT > 60 s.
Cardiovascular system dysfunction	Systolic pressure <90 mm Hg. or mean pressure <70 mm Hg, unregulated by fluid restoration for at least 1 hour (crystalloids 20-30 ml/kg for 30 min + dopamine> or = 5 μg/kg/min). Unexplainable by other causes acidosis (pH = 7.3) or baseline deficiency > or = 5.0 mmol/l + more than 1.5 time compared with the normal plasma lactate level elevation (>1 mmol/l (since 2001)
Acute RDSA (within ALIS - acute lung injury syndrome)	1) acute onset; 2) bilateral pulmonary infiltration (bilateral pulmonary infiltration on the x-ray film); 3) pulmonary artery wedge pressure < 18 mm Hg, necessity of artificial respiration with EEPI > 5 cm water; 4) hypoxemia, refractory before oxygen therapy. Difference between ALIS and RDSA in hypoxemia degree, which is expressed in the form of ratio of $PaO_{2/\text{to}}$ FiO_2; in ALIS PaO_2/FiO_2 < 300, in RDSA < 200 mm Hg.
Kidney dysfunction	Blood creatinine > 176 μmol/l or creatinine increase > 0.5 μmol/l (since 2001); urine sodium <40 mmol/l; diuresis rate < 0.5 ml/kg during 1 hour with adequate CBV
Liver dysfunction	Blood bilirubin > 70 μmol/l (since 2001), increased AsAT, AlAT or alkaline phosphatase 2 times and over the normal values

CNS dysfunction	< 15 points (Glasgow scale)
***Notes*:** RDSA (respiratory distress syndrome f adults) is the most severe form of ALIS (acute lung injury syndrome) In this case the patient has disorders of gas exchange with x-ray changes in the lungs not associated with heart failure and developed in the earliest time of the provoking injury. EEPI – end-expiratory pressure increase; PaO_2 – partial oxygen pressure; FiO_2 – exhaled air fraction	

Treatment of sepsis (main trends of intensive care). The question of sepsis treatment was urgent during the whole period of study of this pathological condition. The number of methods used to treat it is enormous. This can be explained by the heterogeneous nature of a septic process. Critical changes in the method of sepsis treatment occurred after approved definitions of sepsis, severe sepsis and septic shock have been accepted. This allowed different researchers to use the same language with the same concepts and terms. Another very important factor was an introduction of evidence-based medicine into clinical practice. Two of these circumstances allowed the development of scientifically substantiated recommendations for the treatment of sepsis, published in 2003 and entitled "Barcelona Declaration". It stated the creation of an international program such as "Serving Sepsis Campaign", a movement for effective sepsis treatment.

The main approaches in the treatment of severe sepsis and septic shock, which have the evidence base and are represented in the documents "Serving Sepsis Campaign", include:

- algorithm of infusion therapy;
- use of vasopressors;
- algorithm of inotropic therapy;
- use of small doses of steroids;
- use of recombinant activated protein C;
- algorithm for transfusion therapy;
- algorithm of AV in case of an acute lung injury / respiratory distress syndrome in adults (ALIS/RDSA);
- protocol of sedation and analgesia in patients with severe sepsis;
- protocol for glycemic control;
- protocol for the treatment of MOF;
- protocol of bicarbonate use;
- prophylaxis of deep vein thrombosis;
- prevention of stress ulcers.

According to the "Barcelona Declaration" – the only method, which received the substantiation for immunocorrection during sepsis, – is *passive immunosuppressive therapy*.

As one of the most important directions in the development of positive trends in the treatment of severe sepsis, an immuno-physiological approach can be mentioned. It is focused on the interaction of genetically determined mediators of an individual systemic inflammatory response, specifically, on the interaction of mediators in a single process which perform stimulating, inhibiting, ligating, adjuvant and sometimes a determinative action.

1. *Surgical sanitation of a nidus of infection.* Effective treatment of sepsis is impossible without surgical treatment of a nidus of infection. Detection of the primary site is a necessary condition for the correct diagnosis and successful treatment of sepsis of any genesis; necessary control of the source of infection.

Control of the source of an infectious process. Each patient with signs of severe sepsis should be thoroughly examined to identify a source of the infectious process and to carry out appropriate source control measures, which include three groups of surgical interventions: 1) drainage of the abscess cavity; 2) secondary surgical treatment; 3) removal of foreign objects that maintain (initiate) an infectious process.

It is proved that the surgery for necrotizing fasciitis in 24 hours and more after the diagnosis gives a reduction of mortality up to 70%, and an operation in a period of up to 24 hours – reduction of mortality to 13% (without surgery, mortality is approaching 100%). It should be noted that surgical intervention on elimination of a necrosis region refers to resuscitation measures, and the earlier the operation is made, the greater are the chances of the patient. Operative interventions, made in the late

period with a full-scaled picture of the DIC and multiple organ failure, did not lead to the reduction of mortality.

2. *Antimicrobial therapy*. The type of bacteremia in case of sepsis determines the choice of the scheme of antibiotic therapy (ABT). However, it should be noted that this type of treatment is used immediately upon admission to the hospital, when the type of a pathogen is still unknown. Therefore, during empirical administration of ABT, at the first stage it is necessary to prescribe antibiotics with a wide spectrum of action and always in combination.

It is worth pointing out that an optimal choice of empirical therapy for severe sepsis with MOF is carbapenems as drugs, having the widest spectrum of activity and showing the lowest level of resistance among healthcare-associated strains of gram-negative bacteria.

The alternative for them are sometimes cephalosporins of IIIrd-IVth generations protected by anti-pseudomonadal β-lactams (cefoperazone-sulbactam, piperacillin-tazobactam) and fluoroquinolones (ciprofloxacin). In some cases, additional administration of vancomycin, linzolid, and antifungal agents may be possible (fluconazole, amphotericin B). Intravenous administration of antibacterial agents is advisable, which is carried out before disappearance of the main symptoms of infection, which include stable normalization of body temperature, absence of signs of systemic inflammatory reaction, normalization of the gastrointestinal tract function, negative hemoculture, normalization of the leukocyte formula and the number of leukocytes in blood.

3. *Intensive therapy*, the main goals of which are:

1) improvement of oxygen transport in conditions of its increased consumption, characteristic for sepsis (hemodynamic and respiratory support);

2) nutritional support;

3) immunocorrection;

4) prophylaxis of deep vein thrombosis and thromboembolic complications, formation of stress-ulcers;

5) detoxification therapy.

Primary measures of intensive care to obtain the following parameters during the first 6 hours of intensive therapy: 1) central venous pressure (CVP) 8-12 mm of H2O; 2) average blood pressure ($BP_{average}$)> 65 mm Hg; 3) urine secretion > less than 0.5 ml per 1 kg of weight per hour; 4) saturation of hemoglobin of the central venous (lower hollow vein) or mixed venous blood with oxygen (Satv O_2)> 70%.

If Satv 70% cannot be achieved with CVP 8-12 mm H2O, then a transfusion of the erythrocytic mass up to the level of hematocrit 30% and more and administration of dobutamine in a dose up to 20 μg / kg of weight per min. is necessary.

Tasks of infusion therapy during sepsis include: correction of hypovolemia; redistribution of fluid between different sectors (intravascular, interstitial, intracellular); maintenance of optimal level of colloid-osmotic pressure of plasma, what leads to improvement of microcirculation; correction of hypoxia, etc.

In the conditions of the decrease of venous return and preload, caused by vasodilation, sequestration of circulating blood and extravasation of its components (capillary leak syndrome), associated with the inhibition of albumin synthesis in the liver, hypoalbuminemia and a decrease of the oncotic pressure of plasma occur.

Therefore, stabilization of hemodynamics with the help of a large volume infusion is accompanied by an increased fluid extravasation and formation of tissue edema what increase hypoxia and MOF. The expediency of the albumin use raises questions, because an increase of its concentration in plasma is temporary, and later its extravasation occurs, what increases interstitial edema.

Thus, *complex use of hydroxyethyl starch drugs is considered optimal for the administration of infusion therapy during sepsis* (starch molecules contribute to the decrease of the activation of endothelial cells and reduce the "capillary leak" of low molecular crystalloids).

Hemotransfusion is carried out at the concentration of hemoglobin less than 70 g / l, hematocrit less than 25%.

In the absence of a positive effect of infusion therapy, inotropic support is required.

To achieve relevant perfusion pressure, the drugs, which increase vascular tone (dopamine, epinephrine "adrenaline", norepinephrine "norepinephrine", phenylephrine "mezaton") are used.

These drugs also affect the synthesis of inflammatory mediators. Adrenaline, dopamine, norepinephrine and dobutane reduce the synthesis and secretion of TNF-a. Adrenaline also increases the content of IL-10, and dopamine - IL-6 (anti-inflammatory cytokines).

During refractory septic shock and ARDS in patients with sepsis, the method of administration of corticosteroids (within 5-10 days) is used. Hydrocortisone in a dose of 100 mg 3 times a day is administered as a continuous infusion of 0.18 mg / kg / h. It stabilizes hemodynamics and reduces mortality.

Therapeutic agents for hemodynamic support can be divided into three main groups: 1) infusion drugs; 2) vaso-suppressors; 3) inotropic therapy.

Infusion therapy belongs to the immediate measures for hemodynamic support and, primarily, cardiac output.

The aim of the infusion therapy in patients with arterial hypotension in critical condition, caused by sepsis: correction of CBV, stabilization and normalization of blood pressure, reactivation of adequate tissue perfusion, cellular metabolism, correction of homeostatic disturbances, decrease in the concentration of mediators of the sepsis cascade and toxic metabolites.

Infusion therapy is performed under control of the dynamics of arterial and central venous pressure, heart rate and minute diuresis. Patients, which do not respond to the administration of infusion media rather quickly, need invasive hemodynamic control to be adjusted. The volume of infusion is maintained in the way when the pulmonary capillary wedge pressure does not exceed the plasma colloid oncotic pressure (to avoid pulmonary edema) and is accompanied by an increase of the cardiac output. Monitoring of the adequacy of infusion also includes the parameters, characterizing gas exchange function of the lungs (PaO_2 and PaO_2/FiO_2) and the dynamics of the X-ray pattern of the lungs.

An average volume of infusion is usually about 30-40 ml / kg of body weight of a patient, but may reach 50-60 ml / kg or even more. Mainly, it depends on the amount of blood loss, duration of bleeding and compensatory capacity of the body.

The criteria for the infusion therapy efficiency are:

1) CVP 5 – 12 cm H_2O;
2) systolic blood pressure – more than 100 mm Hg;
3) $BP_{average}$ – more than 70 mm Hg;
4) diuresis – 0.5 ml / kg / h;
5) hematocrit – more than 30%;
6) saturation of arterial blood / hemoglobin with oxygen not lower than 92 mm Hg;
7) blood saturation in the upper vena cava - not less than 70%.

Qualitative composition of infusion media. The main purpose of intensive therapy is to restore adequate blood supply to the organs. At the initial stage of treatment, the renewal of plasma volume is more important than of globular one. Blood loss is not so much dangerous resulting in anemia but more in hypovolemia, what contributes to the development of hypoxia. Therefore, restoration of CBV deficiency should be started with infusion therapy, which includes drugs with pronounced volemic action (colloidal and crystalloid solutions).

Qualitative composition of an infusion program in patients with acute surgical pathology (septicemia, septic shock) should be determined by the peculiarities of homeostatic disorders: degree of hypovolemia, heart failure, phase of the DIC syndrome, presence of peripheral edema, level of albumin in the blood, presence and severity of acute lung injury.

Crystalloids. Crystalloid solutions are used as agents for compensation of CBV deficiency. The main component of them is sodium – the principal electrolyte, retained in the extracellular space fluid, moreover, 80% of it is located outside the blood stream. The main indication for the administration of crystalloids is the lack of interstitial fluid. Crystalloids have low molecular weight and therefore easily penetrate the vascular wall and fill the interstitial space. In this regard, crystalloids introduced into the body, disappear from the blood stream after 1-3 hours.

For saline solutions a distinction is made between simple and complex ones. The latter may be equilibrated or balanced.

1. *0.9% isotonic sodium chloride solution* belongs to simple saline solutions (its osmolality is 308 mOsmol / l; normal osmolarity of the plasma is 288 ± 5 mOsmol / l; maximum daily dose of the drug is 2000 ml).

2. *Jonosteril* is a balanced water-electrolyte solution, which consists of: sodium chloride, sodium acetate, potassium acetate. The pH of the drug is 5.0-7.0, and osmolality is 291 mOsmol/L. Indications: extracellular (isotonic) dehydration (loss of fluid during diarrhea, vomiting, fistulae, drainage and intestinal obstruction), as well as volume recovery during plasma losses and burns. Contraindications: edema, hypertonic dehydration and severe renal failure. Administration: IV with a dose of 3 ml / kg of body weight or 70 drops per minute, or 210 ml / hour with a body weight of 70 kg; maximum daily dosage – 40 ml / kg of body weight.

3. Ringer's lactate solution – a balanced combination drug containing sodium ions, sodium salts and calcium. The solution is isotonic toward the blood plasma. The solution is administered for the compensation of fluid deficiency, correction of metabolic acidosis, intoxications, etc. There are no significant advantages over isotonic sodium chloride solution.

4. *Lactasol* – lacto-saline solution of polyionic type with pH of 6,0-8,2 and lactasol osmolarity - 295 mOsmol/l. The solution can compensate water-electrolyte imbalance with the simultaneous correction of metabolic acidosis by increasing blood buffering capacity. Lactastol causes hemodilution and reduces blood viscosity, improves tissue perfusion and renal blood flow, enhances diuresis. In case of moderate acidosis a dose up to 400 ml of the solution is enough for administration. Maximum daily dose of lactosol is 2000 - 3000 ml.

5. *Acesol* – a complex saline solution, which consists of sodium acetate, sodium chloride, potassium salts. The solution has a weak alkaline reaction and is able to eliminate the deficiency of electrolytes and fluid volume, corrects the disturbances of ABS. In large dosages (more than 1500 ml) it has a detoxifying effect, improves renal blood flow, rheology of blood, stimulates diuresis. Development of hyperkalaemia is a side effect.

Colloids. Colloidal solutions are able to restore CVP quickly, increase colloidal osmotic blood pressure, normalize central and peripheral hemodynamics; have detoxification properties.

1. *Albumin 5% and 10% solution* is a natural colloid. It is indicated for hypoalbuminemia at a dose of less than 20 g/l, since an increase of colloid-osmotic pressure during an albumin infusion is transient in nature, and in conditions of increased capillary permeability albumin extravasation into interstitium occurs.

Albumin should not be administered for: thrombosis, chronic cirrhosis, chronic nephritis, protein gastroenteropathy, pancreatic enzymes deficiency, liver resection and paracentesis. Also, it should not be used for the elimination of edema. Albumin solutions should not be used as a source of protein during parenteral nutrition. For hypertension and heart failure, it is reasonable to administer albumin slowly at the concentration of 5%.

2. *Protein* is a drug similar to albumin. The basic mass of proteins consists of albumin (75-80%), what determines its therapeutic effect, as well as of stable α- and β-globulins (20%). Protein is 4.3-4.8% isotonic solution of donor blood proteins. According to its colloid-osmotic activity, the protein is close to the native plasma. The drug has all properties of native plasma for participation in the metabolism and its presence duration in the blood stream. It contains trivalent iron, which adds antiananemic properties to it. The drug does not affect blood coagulation system. A single dose of protein may be 400-500 ml, a daily one - up to 1000 ml. Introduced IV by drop infusion.

The drug is used for the correction of colloid-osmotic pressure, treatment of hypoproteinemia. In the treatment of the diseases, accompanied by anemia, it is administered as a transfusion course of 200-500 ml.

Infusion of protein is contraindicated in case of thromboembolic diseases, brain hemorrhage, hypertensive disease of grades 2, 3, cardiac decompensation. During administration of the protein, the risk of infection with the hepatitis virus and HIV infections (heating for 10 hours at + 600C) is excluded. However, the development of allergic reactions is possible.

3. *Plasma* is a natural colloid. Infusion is indicated for coagulopathy, which is preconditioned by a decrease of the coagulation potential of blood, with the aim of filling the plasma coagulation factors. Introduction of plasma is not appropriate to restore the volume of circulating blood in case of hypoproteinemia.

4. Preparations based on modified *gelatin* (synthetic colloid): Helofundine and Gelofusine - 500 ml (B. Braun Melsungen AG, Germany); Gelafusal H (Serum-Werk Bernburg AG, Germany).

5. Preparations based on *dextran* (synthetic colloid): longasteril 70 with electrolytes - 500.0 ml; longasteril 70 with sodium chloride - 500.0 ml, longasteril 40 with sodium chloride - 500.0 ml; longasteril 40 without sodium chloride - 500.0 ml; longasteril 40 with 20% sorbitol - 500.0 ml (fresenius); polyglucin 500.0 ml; rheopolyglucin -500.0 ml; reogluman -500.0 ml; rheomacrodex - 500.0 ml.

6. Preparations based on *hydroxyethyl starch* (Hydroxyethyl Starch - HES) – synthetic colloids. International names are distinguished by two groups of HES: hetastarch (HES 450/0.7) and pentastarch (HES 200/0,5). The first number is a molecular weight, the second is a degree of substitution. Numerous preparations of HES with different concentrations (from 3 to 10%), different average molecular weight (70, 130, 200, and 450 kD) and different degrees of substitution (0.4, 0.5, 0.6 and 0.7) have been developed. The degree of replacement of plasma volume, elimination half-life, influence on blood coagulation, oncotic pressure, blood rheology are the main differences between HES solutions.

This group of the drugs include: plazmasteril 6% (HES 450 / 0.7) – 250.0 and 500.0 ml; HAES-steril 3% (HES 200 / 0.5) – 500.0 ml; HAES-steril 6% (HES 200 / 0.5) – 250.0 and 500.0 ml; HAES-Steril 10% (HES 200 / 0.5) – 250.0 and 500.0 ml; HAES-steril 10% with low sodium chloride and 5% sorbitol – 500.0 ml (Fresenius, Germany); Infukoll 6% and 10% (HES 200 / 0.5) – 500.0 ml (Serumwerk Bernburg AG); Gecodes 6% (HES 450 / 0.7) (Yuria-Pharm, Ukraine) – 200.0 and 400.0 ml; Refortan 6% (HES / 200 / 0,5) – 500.0 ml; Stabisol (HE / 450 / 0.7) (Berlin-Chemie, Germany) – 500.0 ml.

Positive properties of HES:

1) they prevent the development of the syndrome of increased permeability of capillaries; in contrast to albumin and gelatin solutions, HES solutions have a positive effect in case of increased capillary permeability syndrome which develops during sepsis, respiratory distress syndrome in adults and others; HES molecules are capable to occlude pores in the capillary wall and reduce the level of a damage, associated with high permeability of the capillaries;

2) modulation of the action of circulating adhesive molecules. In contrast to the administration of albumin solutions, the administration of HES solutions to the patients with traumatic injuries reduces the number of circulating adhesive molecules;

3) expression of surface antigens of human peripheral blood monocytes;

4) administration of HES solutions is not accompanied by remarkable influence on the expression of surface antigens of immunological cells;

5) HES solutions do not cause additional activation of CS during the treatment of shock states of any etiology.

The main indications for the administration of HES drugs – hypovolemic state, essential CBV correction, disturbances of macro and microcirculation.

Contraindications for HES: a condition of hyperhydration, hypervolemia; decompensated heart failure; kidney damage with oliguria and / or anuria; intracranial hemorrhages; profound blood clotting disorders; significant thrombocytopenia; lack of fibrinogen; increased individual sensitivity to starch.

It is necessary to use starch preparations carefully in case of pulmonary edema.

7. *Derivatives of polyhydric alcohols.* The most widely used drugs on the basis of sorbitol and sodium lactate are sorbilact and rheosorbilact (Yuria Farm, Ukraine).

Rheosorbilact. Composition: 1 liter of solution contains sodium chloride 6g, potassium chloride 0,3 g, calcium chloride 0,1 g, magnesium chloride 0,2 g, sodium lactate 19,7 g, sorbitol 60g.

Sorbilact. Composition of sorbilact completely duplicates a composition of rheosorbilact, except sorbitol content, which is increased from 60 to 200 g in it. In this case, molar concentration of

sorbitol is increased from 330 to 1095 mmol, and total osmolarity of the solution makes from 0,9 to 1,7 osmol. Pharmacokinetics is identical to rheosorbilact. In addition to the action, provided by rheosorbilact, sorbilact has a more pronounced detoxification effect, more effective hemodynamics, enhances diuresis and stimulates intestinal motility.

For the purpose of restoration of microcirculation and normalization of blood rheological properties, the preparations with high rheological activity are used: refortan, rheopolyglukin, hreosorbilact, trental and others. These drugs quickly dissolve blood, improve its fluidity, attract fluid into the blood stream and improve capillary blood flow.

Complications of the infusion therapy in patients with acute surgical pathology: pulmonary edema and generalized peripheral edema.

The critical role in the occurrence of these complications is played by the volume of infusion, and not the choice of infusion medium (colloids or crystalloids).

4. Nutritive support. During MOF there is a prevalence of catabolism processes over the processes of anabolism due to the necessary compensation of increased energy needs (autocanibalism).

Nutritive support *is a compensation of energy, protein and electrolyte balances.*

Tasks of nutritional support:

1) prevention of translocation of microflora from the intestine;

2) prevention of the development of dysbiosis;

3) increase of the functional activity of enterocytes and protective properties of a mucous membrane;

4) reduction of the degree of endotoxicosis and risk of secondary infectious complications.

Criteria for the administration of nutrients:

- energy value of nutrition of a patient with severe pathology is 25-35 kcal / kg / day in the acute phase of the disease and 35-50 kcal / kg / day – in the phase of stable hypermetabolism;

- the administration of glucose in a dose less than 6 g / kg / day (30-70% of non-protein calories with a level of glycemia less than 6.1 mmol/l due to the risk of hyperglycemia development and activation of catabolism processes in a skeletal muscle; control of glucose concentration is made every 1-4 hours, depending on the clinical situation, it is necessary to maintain the level of glucose within 4,5-6,1 mmol/l (normoglycemia); at the level of glycemia more than 6,1 mmol insulin should be infused in a dose of 0,5 -1 units/h to support normoglycemia; a high level of glycemia and demand for insulin therapy is a factor of adverse outcome in patients with sepsis;

- the administration of lipids – 0.5-1 g/kg/day (15-20% non-protein calories); during parenteral administration of fat emulsions a scheduled around-the-clock mode of drug administration is used, priority is given to II generation fat emulsions of MCT/LCT type, which demonstrate a higher rate of utilization from the bloodstream and oxidation;

- administration of proteins – 1.2-2.0 g/ kg/ day (0.2-0.35 g of nitrogen/kg/day); careful control of nitrogen balance is required in this case; for effective synthesis of endogenous protein, it is important to maintain the metabolic ratio: non-protein calories/total nitrogen in the range of 1 g of nitrogen for 110-130 kcal;

- administration of vitamins – a standard daily set + vitamin K - 10 mg/day + vitamin B1 and B6 - 100 mg/day + vitamin A, C, E;

- administration of trace elements – standard daily set + zinc – 15 -20 mg/ day + 10 mg/l in case of liquid stool;

- administration of electrolytes – sodium, potassium, calcium according to balance calculations and concentration in plasma + phosphorus – 16 mmol / day + magnesium – 200 mg/day.

It should be mentioned that early beginning of nutritional support in the period of 24-36 hours is more effective than 3-4 days of intensive care.

Methods of nutritional supplementation: oral administration of enteral diets; enteral tube feeding diet; parenteral nutrition; parenteral + enteral tube feeding diet.

The choice of nutritional support method depends on the degree of the severity of nutritional deficiency and function of gastrointestinal tract.

Contraindications to nutritional support: refractory shock; environmental intolerance for nutritional support conduction; arterial hypoxemia which cannot be stopped; uncompensated hypovolemia; decompensated metabolic acidosis.

Enteral nutrition prevents translocation of microflora from the intestine, development of dysbiosis, increases functional activity of enterocytes and protective properties of the mucous membrane, reducing the degree of endotoxicosis and being a very important source of energy and plastic material at the same time.

5. *Immunocorrection.* There are several variants of the immune status of patients with intra-abdominal infection, depending on the level and degree of disturbances of the immune system.

The immunogram of the patients with a localized form of infection (type I) is characterized by a moderate decrease in the absolute number of T-lymphocytes (42.9±2.69) and a decrease in the relative number of B-lymphocytes (16.0±2.0%).

In patients with a common form of purulent infection of the abdominal cavity there is a significant decrease in the relative number of lymphocytes, absolute and relative number of T-lymphocytes (36.62±2,29); relative number of B-lymphocytes (13±1.8); the ability of T-lymphocytes for PGA-blast-transformation is inhibited ISB (13.28±1.1); the index of phagocytosis completeness decreases (0.58 ± 0.03).

Type II immunogram is characterized by a more significant decrease in the absolute number of lymphocytes, appearance of defective macrophages and a sharp increase in the number of T-suppressors.

Thus, *in patients with purulent infection of the abdominal cavity to the extent of its generalization, a condition of secondary immunodeficiency develops with the deprivation of all components of the immune system.* In addition to the implementation of general principles of surgical intervention and principles of the management of patients in the postoperative period, the measures for restoration of immune homeostasis acquire special significance.

Immunomodulators *are the drugs which have immunotropic activity and in therapeutic doses restore the function of the immune system.*

These drugs act predominantly on modified parameters, lowering the increased and increasing the decreased indicants of the immune system.

The drugs for central regulation of immunogenesis include thymus preparations.

Thymoptin contributes to the restoration of initially reduced indexes of cellular immunity, increased activity of neutrophils and phagocytosis, and elimination of sites of chronic infection.

Tactivin stimulates maturation, differentiation, functional activity and release of T-lymphocytes into circulation.

Thymalin has an ability to stimulate immunological reactivity of the body: regulates the amount of T- and B-lymphocytes, stimulates the response of cellular immunity; intensifies phagocytosis; stimulates regenerative processes.

Immunomodulators of bone marrow origin (myelopeptides). Stimulate various immune responses, repair disturbances in the processes of hemopoiesis, participate in the implementation of the functions of various subpopulations of lymphocytes. Preparations of this group contribute to the quick maturation of B-lymphocytes in the bone marrow, increase the number of cells, which produce antibodies; increase general resistance of the body. The important property of myelopeptides is an ability to stop pain. They have anti-stress action, stimulate B-system of immunity at the level of the secondary immune response and act on the red blood shoot of hematopoiesis, what allows to use them in case of anemia.

Cytokine preparations. At present, genetically engineered (recombinant) cytokine drugs deserve special attention as a means of sepsis treatment. They are administered for many severe and fatal diseases in life-saving cases. They include betaleucine (interleukin-1β), roncoleukinum (interleukin-2) and interferon preparations.

Betaleucine has immuno- and hemostimulating effect.

Interferon preparations are used for the correction of immunodeficy conditions of different etiologies. The use of rIFNγ drugs for the patients with trauma and with complications developed in

the post-traumatic period with a background of inhibited immunoreactivity by infectious complications, including sepsis, increased the level of expression of the activation marker HLA-DR on monocytes and their production of a number of cytokines with anti-inflammatory activity. The prophylactic administration of rIFNγ to the patients with critical injuries, when infectious complications have not yet developed, reduces the incidence of post-traumatic sepsis and the severity of an infectious process.

Roncoleukinum causes formation of lymphokinactivated killers. Stimulates cytolytic activity of natural killers and cytotoxic T-lymphocytes. Roncoleukinum is effectively used in the treatment of severe forms of infectious complications in wounded and injured surgical patients.

Roncoleukinum is a yeast recombinant human interleukin-2 (rIL-2), widely used as a means of immunotherapy in complex therapeutic regimens of the diseases of different etiologies. Most effectively, Roncoleukinum showed its worth in purulent-inflammatory diseases in surgery, bacterial and viral infectious diseases, as well as oncology.

Roncoleukinum is a modern biotechnological product, a genetically engineered analogue of the endogenous cytokine. The recombinant form of IL-2 is obtained from the cells of the producer – a strain of non-pathogenic baker's yeast of the *Saccharomyces cerevisiae* species, in the genetic device of which the human interleukin gene was incorporated. The active substance of Roncoleukinum is a single chain polypeptide of 133 amino acids with a molecular weight of 15.3 ± 0.2 kDa.

By contrast with *bacterial rIL-2 – Aldesleukin* (EU countries), *Proleukin* (USA), which do not have N-terminal alanine and in position 125 cysteine is replaced by serine (it is mutant), *yeast rIL-2 (Roncoleukinum, Russia)* is a complete structural analogue of the peptide component of human IL-2, differing from the endogenous cytokine only by the absence of a polysaccharide fragment. In the endogenous IL-2 molecule there is one region of glycosylation, the presence of which does not affect the function and affects only the duration of cytokine presence in circulation.

Indications for cytokine therapy by Roncoleukinum in patients of surgical profile. The main indication for cytokine therapy by Roncoleukinum is surgical sepsis of any etiology (abdominal, post-traumatic, wound, general surgical, burn, angiogenic). The drug should also be used in case of urological and obstetric-gynecologic sepsis. In case of a nidus of infection with the subsequent development of SIRS, the clinical basis for the administration of cytokine therapy is the manifestation of two or more symptoms of SIRS. Additional reason for cytokine therapy with Roncoleukinum is a laboratory-confirmed state of general immunosuppression, which is combined or followed by SIRS.

During the therapy by Roncoleukinum it is necessary to take into account the severity of endotoxicosis, which is accessed and calculated by the leukocyte index of intoxication (LII), which, in combination with the indicators of concentration of peptides of the average mass and clinical signs of intoxication, allows to accurately determine the degree of severity of endotoxicosis.

Synthetic immunotropic preparations. As drugs of thymic origin, these drugs specifically affect T-lymphocytes, activate their proliferation and synthesis of cytokines. They include: imufan, *levamisolum (decaris), methyluracil, polyoxidonium, licopid.*

Immunoglobulin preparations. In recent years, immunoglobulins have been widely used in clinical immunology for intravenous administration, the so-called intravenous immunoglobulins (IVIG).

Sandoglobulin (Novartis, Switzerland). Contains lgG (up to 95%), antibodies to cytomegalovirus. It is also effective during septic shock in adults and for chronic infections. Among other effective IVIG drugs there are *cytotect, intraglobulin F, octagam.*

Antistaphylococcal immunoglobulin is indicated for: purulent-septic processes, diseases of the musculoskeletal system (osteomyelitis) and other organs and systems.

Pentaglobulin – normal human immunoglobulin takes a special place among immunoglobulin preparations. It surpasses all other classes of immunoglobulins. Enhances phagocytosis of bacterial and viral antigens, neutralizes toxins and autoantibodies, activates and regulates complement systems.

6. Prevention of *thromboembolic complications, deep vein thrombosis and formation of gastrointestinal ulcers*. The release of inflammatory mediators during sepsis leads to the activation of the coagulation cascade and suppression of fibrinolysis. In this case there is hypercoagulation, DIC

syndrome and thromboembolic complications. Prophylaxis of deep vein thrombosis is also likely to affect the treatment results of the patients with sepsis. For this purpose, heparins and heparin drugs of low molecular weight (fraxiparine, fragmin, clexane) are used. The advantages of low molecular weight heparin preparations are: lower incidence of hemorrhagic complications, less pronounced effects on platelet function and possibility of single administration per day.

To prevent the onset of deep vein thrombosis in the patients with sepsis the following drugs are administered: *heparin* 5000 units sc 4 times a day; *Fraxiparine* (Nadroparin calcium) 0.3 ml sc q.d. for 5-7 days after surgery; *Clexane* (Enoxaparinum natrium) 0.2 ml q.d. for 7-10 days; *Fragmin* (Dalteparinum natrium) 2500 IU q.d. for 10 days.

High mortality rate in patients with streptococcal gastrointestinal stress ulceration bleedings of a high incidence (more than 50%) is reported. The use of *H2 blockers and proton pump inhibitors (PPI)* more than 2 times reduces the risk of their incidence. The efficacy of PPI is higher than the use of H2 blockers. However, it is necessary to remember about the phenomenon of nocturnal acid breakthrough during an isolated administration of PPI, and use them in combination with H2 blockers.

7. *Detoxication therapy*. Detoxication therapy includes methods of *extracorporeal detoxification: hemofiltration, hemodialysis, plasmapheresis and sorption methods.*

The most effective method of blood "purification" is *hemodiafiltration*, which combines two methods of mass transfer – diffusion and convection, and covers the entire range of substances that are removed during hemodialysis and hemofiltration. An additional contribution to the detoxification process is the sorption component of pathogens on the hemodiafilter membrane.

Also, methods of detoxification therapy include *"MARS" (molecular adsorbent recycle system), "artificial liver."* Designed for selective removal of low and medium molecular substances, it simulates the detoxification function of the liver. Three methods of mass transfer (diffusion, convection, adsorption) are used in the process. The main role belongs to albumin dialysis. Indications: liver failure.

Contraindications to the methods of extracorporeal detoxification: terminal state of a patient, external or internal bleeding, unsolved surgical pathology, nutritional dystrophy, hypovolemia, arterial hypotension. Age is not a contraindication.

During ***disability evaluation in patients*** with severe sepsis, and especially septic shock, it is necessary to stem from the following principles: in the absence of signs of SIRS and bacteremia, immune disturbances with depressive symptoms persist for a long time. Therefore, the following question is logical: which indexes of immunoreactivity can be used as criteria for treatment and whether full recovery is possible at all? Consequently, this category of patients should be considered lifelong immunocompromised.

Prophylactic medical examination. The patient should stay in a hospital until complete normalization of body temperature, laboratory findings, negative blood culture tests, absence of clinical manifestations.

After discharge from the hospital, a sick leave for 1-2 months is issued to a patient, and the latter stays under the care of a district therapist. The body temperature is measured daily in the morning and in the evening, and once a week – every 2-3 hours. The patient is examined by a physician once every 2 weeks, within this term CBC and UA are made. If a patient has no relapses of a septic process for 6 months, medical examinations are performed once in 6 months, and then twice a year. Control blood and urine tests within the first month are performed once every 10-14 days, and then once a month for the next 3-6 months.

The patient should stay under the care of a physician for 2-3 years after recovery.

After discharge from the hospital it is recommended to undergo preventive courses of antibacterial therapy for 2-3 weeks in 1, 3 and 6 months by the drugs, the administration of which was the most effective in the past.

Depending on the general condition and functions of vital systems, a patient may return to work or obtain the disability status. The disability group is assigned in accordance with general principles.

Prevention of sepsis. Such patients need long-term immunorehabilitation, including mediated immunotherapy drugs: vitamin-mineral complexes, plant immunoactive drugs and adaptogens, as well

as correcting drugs for intestinal dysbiosis. In the long term with prophylactic purposes, it is advisable to periodically administer the courses of soft immunocorrectors for the patients with sepsis: thymomimetic drugs, derivatives of bacterial muramyl dipeptides, as well as antioxidants.

I.4. GUIDELINES ON ANTIBIOTIC THERAPY FOR PREVENTION AND TREATMENT OF SURGICAL INFECTION

Antibacterial drugs in surgical practice are administered both for the prevention of postoperative purulent-septic complications, and for the treatment of infectious pathology of various localization. The frequency of postoperative purulent-septic complications, according to various researchers, makes from 0.29% to 30%. The risk of postoperative complications is minimal during "clean" operations (less than 2-5%) and maximal during the so-called "contaminated" operations (up to 30-40%). Differences in the represented data do not reduce the importance of the problem of these complications, which slow down the recovery of patients, increase mortality, duration of hospitalization and the cost of inpatient treatment. The use of antibacterial drugs for treatment is widely used in such areas of surgery as skin, soft tissues, bone and joint infections, in abdominal, thoracic and cardiovascular surgery, in urological and gynecological practice.

The main pathogens of surgical infection

The most common pathogens of postoperative wound infections are *S. aureus, E. coli, P. aeruginosa, Enterobacter spp., P. mirabilis, K. pneumoniae, Streptococcus spp., C. albicans, Citrobacter spp., S. marcescens, Candida spp.*

The spectrum of microorganisms is further determined by the type of surgical intervention, its duration, the term of a patient's stay in the hospital before surgery, local pattern of the microflora resistance to antimicrobial drugs.

Staphylococcus aureus holds a place among the most common pathogens of purulent infection. It often contaminates a surgical field during surgical interventions for upper and lower extremities, in the region of head and neck, as well as in thoracic and cardiovascular surgery. It is a frequent component of aerobic-anaerobic associations during operations of any localization. In infected "clean" and "clean-contamined" postoperative wounds in most cases penicillinase-forming staphylococci are found, which colonize the nasopharynx and skin and are resistant to benzylpenicillin and ampicillin. In case of identification of such strains, it is inappropriate to prescribe antibiotics for prophylaxis and purulent infection.

The main infectious organisms of postoperative purulent complications during operations for the upper portions of the intestine, liver, and urinary tract organs are staphylococci, enterococci and enterobacteria (*E. coli, Proteus spp., Klebsiella spp.,* etc.), against which cephalosporins of I-II generations, combined preparations of inhibitors of β-lactamase and penicillins, aminoglycosides, fluoroquinolones are active.

The main contaminating microorganism in colorectal surgery, in case of appendectomy, in operative gynecology is an association of aerobes and anaerobes, which requires the administration of cephalosporins of II-III generations, various combinations of antibiotics with anti-aerobic and anti-anaerobic activity.

Classification of antibacterial drugs

Antibacterial agents are classified on the basis of their chemical structure, mechanism of action, antimicrobial spectrum, type of action on the microbial cell (I.G. Bereznyakov, V.V. Strashnyy, 1997).

According to ***the chemical structure***, there are antibiotics (natural and semi-synthetic) and synthetic antibacterial agents.

I. ***Antibiotics.***

A. ***Beta-lactam antibiotics:***

- penicillins (natural, semi-synthetic, penicillinase resistant, aminopenicillins, combined preparations of aminopenicillins and beta-lactamase inhibitors, antipseudomonal penicillins, carboxypenicillins and ureidopenicillins, combined preparations of antipseudomonal penicillins and beta-lactamase inhibitors);
- cephalosporins (1st, 2nd, 3rd and 4th generations);

- carbacephems;
- oxalactams;
- monobactams;
- carbapenems.

B. *Antibiotics of other classes*: macrolides, azalides, glycopeptides, Laevomycetinum, ansamacrolids, tetracyclines, aminoglycosides, polymyxins, lincozamines, lipopeptide antibiotics, streptogramins, ketolides.

II. ***Synthetic antibacterial agents***: sulfanilamides, antituberculosis drugs, nitroimidazoles, derivatives of quinoxalin, derivatives of diaminopyrimidine, quinolones, fluoroquinolones, nitrofurans, 8-oxyquinoline derivatives, oxazolidinones, peptidyl deformylase inhibitors.

Based on ***the mechanism of action***, antibacterial agents are divided into *4 main groups*:

1) inhibitors of cell wall synthesis of microorganisms – penicillins, cephalosporins, vancomycin, phosphomycin;

2) drugs, that disrupt molecular organization and function of cytoplasmic membranes – polymyxins and some antifungal drugs;

3) antibiotics, that irreversibly suppress protein synthesis – aminoglycosides, reversibly – macrolides, tetracyclines, Laevomycetinum, lincozamides;

4) drugs, which interfere with the synthesis of nucleic acids – ansamacrolides, fluoroquinolones, sulfanilamides, trimethoprim, nitroimidazoles.

Based on the spectrum of antimicrobial action antibacterial drugs are divided into the following groups:

1) preparations that act predominantly on gram-positive and gram-negative cocci (staphylococci, streptococci, gonococci) and some gram-positive rods - natural and penicillinase resistant penicillins, Ist generation cephalosporins, macrolides, Vancomycin, lincozamides;

2) broad spectrum preparations, active against gram-positive and gram-negative microorganisms – aminoglycosides, tetracyclines, Laevomycetinum, aminopenicillins, antipseudomonal penicillins and cephalosporins of the second generation;

3) preparations with predominant activity toward gram-negative microbes – polymyxins, cephalosporins of IIIrd generation, fluoroquinolones;

4) anti-TB drugs, including antibiotics Streptomycin and Rifampicin;

5) antifungal drugs.

Antibiotics for prevention of surgical infection

Depending on the risk of postoperative infectious complications, all surgical operations are divided into 4 categories:

1. *"Clean"* operations are considered the operations, in which a lumen of the respiratory tract and the gastrointestinal tract is not dissected, there are no signs of inflammation in the operative area and the aseptic technique of an operation is not violated. The occurrence of pyogenesis during these operations is 1.5 - 5%.

2. *"Clean-contaminated operations"* are the operations in which the lumen of the gastrointestinal tract or respiratory tract is dissected, but their content does not get into an operated wound. The occurrence of pyogenesis in these operations makes 10%.

3. *"Contaminated operations"* are the operations during which the contents of the gastrointestinal tract or respiratory system flow out; operations for infected bile ducts and urinary tracts, as well as the operations involving the dissection of tissues with signs of acute inflammation, but without suppuration. During such interventions, occurrence of pyogenesis is about 20%.

4. *"Dirty-infected" (or primary-infected)* operations are made for purulent processes or perforations of hollow organs. The occurrence of postoperative suppurative complications exceeds 30%.

In addition to the degree of microbial contamination of a wound the development of wound infection in the postoperative period is influenced by the state of local and general immunity, nature of preoperative preparation, operative technique of a surgery, surgical tissue trauma, loss of blood, presence of foreign objects, virulence of microflora and resistance of bacteria to antimicrobial drugs.

At the present stage of the development of purulent surgery in the groups of contaminated and dirty-infected operations, the crucial task of a clinician is to minimize the number of these complications.

Principles of antibiotic prophylaxis.

1. Microbial contamination of a surgical wound is practically unavoidable, even in case of strictly followed aseptic and antiseptic techniques. By the end of an operation, 80-90% of wounds are inoculated with different microflora, most often with staphylococci.

2. During implementation of antibiotic prophylaxis, complete eradication of bacteria should not be tried to achieve. A significant reduction in their number already improves the functions of the immune system and prevents the development of purulent infection.

3. The effective concentration of the antimicrobial preparation in the surgical wound should be achieved before the operation and kept until its end.

4. Intravenous administration of antimicrobial drugs with a prophylactic purpose, as a rule, takes place in 30-40 minutes before surgery.

5. Further administration of antimicrobial drug for more than 24 hours after an operation does not lead to an increase of the efficacy of antibiotic prophylaxis. It is also important to have all available information about potential pathogens (their type, sensitivity to antibiotics).

Indications for preoperative antibiotic prophylaxis. Antibiotic prophylaxis in surgery (preoperative antibiotic prophylaxis) is the standard of medical care for many surgical interventions. The administration of antibiotics with a prophylactic purpose for surgical patients is recommended in two cases.

1. *Surgical intervention with a high risk of development of postoperative infectious complications:*

1) planned "conditionally contaminated operations" without using of implants and some "contaminated" operations;

2) all operations with the dissection of a hollow organ;

3) such "clean contaminated operations" as colon resection, anteroinferior and abdominoperineal resection of the rectum require additional preoperative prophylactic measures called "bowel preparations", which are designed to remove the content of the intestines (administration of a cleansing enema and laxatives in a day before the operation) and a decrease of the number of microorganisms living in it (administration of oral antibiotics with low systemic bioavailability – polymyxin, erythromycin, neomycin, bacitracin, metronidazole, etc.).

2. *"Clean" operations, during which uninfected tissues are dissected, hollow organs are not dissected and there are no signs of inflammation or infection. Indications for the preventive administration of antibiotics during "clean" operations are the following:*

1) any operations involving the implantation of vascular prosthesis (coronary artery bypass surgery, lower extremity surgery with venous prostheses) or joint prostheses;

2) any surgery, in which the development of surgical site infections (SSI) is associated with high risk for a patient (neurosurgery, heart, chest, mammary gland surgery).

Emergency "clean" operations and emergency cesarean operation, which is considered a "clean contaminated" operation, are also the indication for preoperative antibiotic prophylaxis.

Antibiotic prophylaxis is not indicated for scheduled and emergency operations, classified as "contaminated" and "dirty-infected" ones. In these cases, a course of antibiotic therapy is usually administered to patients due to the primary surgical infection which begins before a surgery and lasts during the postoperative period.

Criteria for selection of drugs for preoperative antibiotic prophylaxis. The activity spectrum of the antimicrobial drug should include the most frequent pathogens of postoperative infections, primarily staphylococci, as they cause 80% of the total number of postoperative suppurations. In addition, the spectrum of activity of the antimicrobial drug should cover other endogenous microorganisms, which contaminate the wound in case of disruption of the continuity of internal organs or mucous membranes.

Optimum regimens of antibiotic prophylaxis. For most "clean" and "clean-contaminated" operations – cephalosporin of the Ist (Cephazolin) or IInd (Cefuroxime) generations. For "contaminated" operations:

a) involving the organs of the gastrointestinal tract to the terminal ileum – Ist or IInd generations cephalosporin (Cefazolin or Cefuroxime);

b) involving the organs of the gastrointestinal tract below the distal segment of ileum, biliary tract, organs of the reproductive system, ENT organs – inhibitor-protected aminopenicillins (Amoxicillin / Clavulanate, Ampicillin / sulbactam) or cephalosporins of the Ist-IInd generation + anti-anaerobic drug (Metronidazole).

Cephalosporins of the IIIrd-IVth generations and carbapenems, aztreonam and fluoroquinolones, which have a higher cost, should not be used for preoperative antibiotic prophylaxis.

Dosage of antibiotics, routes and time of administration. In most cases, one full therapeutic dose of an antibiotic is administered for preoperative antibiotic prophylaxis. An antibiotic is administered intravenously. The first dose of an antimicrobial preparation should be introduced before the operation (30-60 minutes before incision), so that bactericidal concentrations were formed in the blood and tissues before the moment of initial skin incision. The optimal time of antibiotic administration with prophylactic purpose is an introductory anesthesia, that is, before the contamination of tissues by microorganisms. This allows to maintain the maximum serum and tissue concentrations during the whole operation and in most cases even a few hours after the closure of the wound. In case of massive bleeding or surgery duration of more than 3 hours, an antibiotic should be introduced repeatedly at intervals equal to 2-3 half-life periods. For scheduled operations, a time increase for prophylactic introduction of antibiotics for more than 24 hours is not indicated.

Prophylactic administration of antibiotics in surgical patients is ineffective in those situations, when there is a high probability of tissue recontamination in the postoperative period: in patients with tracheostomy and intubated patients, in patients with permanent urinary catheters, with central vascular catheters, in patients with the drainage of the pleural and abdominal cavity, in most patients with open wounds, including burn wounds. Despite the fact that these patients have a high risk of infection development, a surgeon should not continue antibiotic prophylaxis for more than 24 hours after a surgery. It is necessary to thoroughly monitor such patients and prescribe antimicrobial drugs only when an infection is confirmed by the results of a culture examination and evaluation of the sensitivity of an isolated agent to antibiotics. An increase in the duration of prophylactic antibiotic administration is unreasonable, and it is also a major factor, contributing to the emergence of antibiotic-resistant strains of microorganisms. It should be emphasized that preoperative antibiotic prophylaxis is a support, but not an alternative to surgical technique or the principles of aseptics and antiseptics, which should be strictly followed. The use of third-party materials and drainages, inaccurate tissue matching, tissue compression by overtightened sutures, presence of devitalized tissues, hematoma and seroma – all these increase the risk of infection development in the postoperative period.

Antibacterial therapy of surgical infections.

It is important to distinguish the situations, in which antibiotics are indicated for preventive purposes, from the situations, in which the course of antibiotic therapy is necessary. These situations require the administration of different drugs and different terms of antibiotic administration. The prophylactic administration of antibiotics in surgery is indicated only for preoperative antibiotic prophylaxis. Antibiotic prophylaxis, which starts immediately before a surgical intervention, should not be performed longer than 24 hours after surgery, since it is not intended to prevent the development of SSI, associated with tissue contamination in the postoperative period.

Long unjustified use of antibiotics for the purpose of prevention leads to the development of antibiotic resistance and may fade a clinical picture of surgical infection development, making it difficult to disclose a disease and administer proper treatment on time. In contrast to antibiotic prophylaxis, antimicrobial therapy of primary surgical infection starting, as a rule, in the preoperative period, lasts more than 24 hours after the operation and is focused on the treatment of a confirmed surgical infection. In case of primary surgical infection, the patients, who have the infection spread outside the primary nidus, should receive antibiotics for treatment, that is, for more than 24 hours. On

the contrary, in the patients with a focus of inflammation or infection, which may be radically removed during surgery (circumscribed local infection), only preoperative antibiotic prophylaxis is considered sufficient, that is, the administration of antibiotics is not longer than 24 hours. The presence of generalized infection, confirmed intraoperatively (detection of pus or infected peritoneal fluid during an operation) is an absolute indication for antibiotic therapy, since such patients are considered to be the patients with confirmed surgical infection.

Indications for antibiotic therapy in surgical patients. Systemic antimicrobial therapy is indicated to the patients with confirmed primary (i.e., which develops before operation) infection, and certain categories of patients with SSI. The decision to prescribe antibiotics for treatment of SSI is based on the clinical picture of infection and the type of SSI (according to the traditional classification). In most cases of the development of superficial SSI in the area of dissection without the signs of systemic inflammatory response, systemic antibiotic therapy is not required. On the contrary, in case of deep SSI of incision and SSI of an organ / cavity, as well as any SSI with systemic symptoms of infection, administration of antibiotics is required. It should be emphasized, that the primary measure in case of the SSI development, should be the collection of clinical material for culture examination. In this case, the main method of SSI treatment is wound drainage and / or sanitation of a site of infection. Antibacterial therapy is indicated as an addition to proper surgical intervention and only in case of systemic signs of infection.

Basic principles of antibacterial therapy for surgical infections. On administration of antibacterial therapy, each surgeon must strictly adhere to the following recommendations, which allow to treat patients effectively and to keep the development of antibiotic resistance under control:

1) use antibiotics with high activity to the isolated or probable infectious agent (narrow spectrum antibiotics should be used if possible);

2) systemic antibiotics should not be used topically;

3) antibiotics should be administered in appropriate doses, following an optimal route of administration and dosage regimen;

4) do not use new expensive antibiotics in the situations, in which traditional, cheaper drugs have similar efficacy.

Empirical and targeted (etiotropic) antibacterial therapy. It is necessary to distinguish targeted and empirical therapy. In cases, when a pathogen is not known, empirical antimicrobial therapy is chosen, based on the localization and type of infection, and also on the predictable spectrum of the most probable pathogens and their antibiotic sensitivity. After obtaining the information on an isolated pathogen and its sensitivity to antibiotics, it is possible to correct the therapy, prescribe the drug according to the results of the antibiogram, that is, to conduct targeted therapy.

Routes of administration of antibiotics. Appropriate methods of administration of antimicrobial drugs are parenteral (intramuscular, intravenous) and oral. Local administration of antibiotics is allowed only in exceptional cases. As soon as the patient's condition allows, the parenteral route of administration should be changed to the oral one (in case of its high bioavailability during ingestion). At the same time, oral administration of drugs cannot be advised to the patients with impaired consciousness, vomiting, dysphagia and is not recommended in case of dysfunction of the gastrointestinal tract, which may affect the bioavailability of a drug. Other ways of administration of antibiotics are inadequate, therefore should not be implemented.

Introduction of antibiotics directly into the abdominal or pleural cavity does not allow to achieve adequate tissue concentrations at the site of infection. An increase in the quantity of the administered antibiotic leads to the development of systemic adverse reactions.

Local administration of antibiotics in surgery. The administration of topical forms of antibiotics in certain cases can be effective in treatment of infected wounds. But:

1) combination of local and systemic antibacterial drugs is not more effective than the use of systemic antibiotics alone;

2) isolated administration of local antibiotics is often inferior in the effectiveness to the systemic antibiotic therapy.

During local antibacterial therapy, it is advisable to avoid antibiotics intended for systemic use. If it is impossible to avoid local administration of systemic antibiotics, it should be remembered that some of them will get to the bloodstream, what will increase the risk of development of systemic adverse reactions and contribute to the selection of resistant strains of microorganisms. Ideally, during local antibiotic therapy, it is advisable to administer only those antibiotics, which cannot be used systematically (e.g. Mupirocin, Silver sulfadiazine). *Except for burn wounds, local administration of systemic antimicrobial drugs (injections around wounds, introduction via drainage tubes, and irrigation of cavities during surgery) with the aim of prevention or treatment of SSI is an abuse in surgical practice and cannot be compared to parenteral administration of antibiotics by efficacy.* Local administration of systemic antibiotics in most cases does not allow to create optimal bactericidal concentrations of a drug in the nidus of infection and surrounding tissues, what leads to the formation of antibiotic resistance in microorganisms.

De-escalation therapy. Inadequate empiric therapy is a serious risk factor for the development of mortality in case of severe bacterial infections, especially nosocomial. Thus, appropriate initial therapy is an important factor, which determines an disease outcome in such patients.

De-escalation therapy *is a treatment strategy, based on the principle according to which an optimal treatment regimen for the patients with severe infections is empiric broad-spectrum antibiotic therapy or a combination of antibiotics, which cover all the most probable pathogens of the infection and overcome the most probable mechanisms of resistance.*

This approach is intended to avoid a high incidence of lethal outcomes, associated with the administration of inadequate antimicrobial therapy in patients with severe, especially nosocomial, infections. Examples of indications for de-escalation therapy are nosocomial pneumonia and severe post-operative infections.

De-escalation therapy is performed in two stages. *The first stage* involves an empiric administration of broad-spectrum antibiotics or a combination of antibiotics. The basic concept is that at the first suspicion of infection it is necessary to administer a broad-spectrum antibiotic, active to gram-negative and gram-positive microorganisms, including anaerobes (in some cases). It allows to reduce mortality, to prevent the development of organ failure and to reduce the period of a patient' stay in the hospital. Of course, for each hospital it is extremely important to have local microbiological data about the most common pathogens of infections and their sensitivity to antibiotics. *The second stage* is a de-escalation of antibiotic therapy proper and is targeted at the more effective treatment in regard to economy, as well as the maximum reduction of possible occurrence of resistant strains of microorganisms. In case of acquired information about the detected infectious agent and its antibiotic susceptibility, it is possible to switch to monotherapy using a direct-acting antibiotic, less toxic or less expensive, and, if necessary, to finish the course of antibacterial therapy. All this allows to avoid unnecessary expenses, unreasonably prolonged administration of antibiotics and their selective pressure, what leads to the development of antibiotic resistance and clinical inefficiency as a consequence of inadequate antibacterial therapy.

The main components of de-escalation approach are:

1) isolation of a pathogen and determination of its sensitivity to antimicrobial drugs;

2) assessment and modification of initial therapy based on the results of microbiological research;

3) assessment of the clinical efficacy of the initial therapy performed;

4) individualization of the duration of therapy, by reference to the characteristics of a patient and the dynamics of the clinical pattern of an infection.

Step antibiotic therapy in surgery. A modern and promising approach which allows to optimize antibiotic administration in the hospital is step therapy. The goal of this strategy is to reduce the cost of treatment associated with the use of antibiotics and reduce the terms of stay of patients in the hospital without influencing the efficacy of treatment and the quality of medical care.

Step therapy *is a two-step administration of antibiotics, when, to the extent of the improvement of a clinical condition of a hospitalized patient and the possibility of internal taking of drugs,*

intravenous / intramuscular administration of an initial antibiotic (s) is changed to the oral administration of the same or another antibiotic (s), equivalent by efficacy.

In case of treatment of hospitalized patients, administration of predominantly oral antimicrobial drugs should be kept. Exceptions include such situations as a critical condition of a patient, inability of taking drugs per os, absence of a similar oral antibiotic by efficacy. It has been shown, that if a patient can take in antibiotics and has no digestive tract disorders, then any differences in the results of the disease in case of administration of equivalent drugs, regardless of the method of administration (intravenous or oral), are not observed. When making a decision whether or not to perform step therapy, it is more important to consider not the route of administration, but the spectrum of activity of an antibiotic, its bioavailability and a degree of penetration into the tissue. In case of change from intravenous to an oral regimen of therapy, the total duration of an antibiotic course should not exceed the same in case of administration of only parenteral antibiotic therapy in this situation. Moreover, in many patients, with a significant clinical improvement of the condition, normalization of temperature and the number of leukocytes in the blood, antibiotics can be canceled without change to the oral regimen of therapy. Further administration of antibiotic therapy in this situation is not reasonable.

The duration of antibacterial therapy. Existing recommendations or evidence based data available in the literature should be used to determine an optimal duration of antimicrobial therapy for various surgical infections. In general, too short course of antibacterial therapy may be ineffective, but at the same time, unreasonable increase of the duration of therapy compromises the development of antibiotic resistance, increasing the risk of the development of adverse drug reactions, and also the cost of treatment. In case of administration of suboptimal doses for an unreasonably long period, the chance of formation of the resistance to antimicrobial drugs becomes even higher.

At present, there is much evidence that short courses of antibiotics, administered in adequate doses, are at least as effective as previously widely practiced prolonged (7-14 days) antibacterial therapy of surgical infections. Moreover, short courses of antibiotics reduce the cost of treatment, reduce the risk of adverse reactions, minimize the duration of antibiotic influence on bacteria and thus reduce selective pressure, which is one of the factors contributing to the development of antibiotic resistance.

Currently, there are ***two main approaches*** to the evaluation of the ***desirable duration of antimicrobial therapy in surgical patients***. The first is keeping to the treatment standards, by which the duration of a course of antibiotics is determined by the information, obtained during an initial surgical intervention.

Alternative approach means to determine a desirable duration of antimicrobial therapy based on the dynamics of the symptoms of an infection in a patient. The withdrawal of antibiotics in case of disappearance of clinical symptoms of infection is as effective as the courses of fixed-duration antibiotic therapy, and generally leads to the reduction of the duration of antibiotic administration. Thus, antibacterial therapy can be discontinued, if the symptoms of infection such as fever and / or leukocytosis disappear. *Persisting of clinical signs of infection till the end of a certain period, necessary for the evaluation of the effectiveness of therapy, is an indication for the additional diagnostic search for the infection nidus, and not for the prolongation of antimicrobial therapy. Antibacterial therapy of surgical infection without adequate surgical intervention is in most cases ineffective.* It should be emphasized that the most important primary measure in the treatment of surgical infections is the evaluation of the demand for surgery. An increase in the duration of the course of antibiotic therapy is a justified only in some patients, when it is not possible to achieve adequate control of the infection nidus, that is, when the primary focus of the infection cannot be or was not radically removed during an initial surgical intervention (osteomyelitis, pancreatic necrosis, cholangitis, diverticulitis). However, the final decision on the duration of antibiotic therapy is determined by the type of a surgical infection (patients with large infected burns, as a rule, require long courses of antimicrobial therapy, what is preconditioned by the course of the disease).

The use of combinations of antibiotics. In general, during the treatment of the surgical infection, monotherapy is more preferable than the combinations of antibiotics, apart from the need to use the synergistic effect of several antibiotics or an expanded spectrum of activity, which cannot be

achieved by the administration of a single drug. Monotherapy reduces the risk of drug interactions, frequency of mistakes in drug administration, inadequate dosaging, undesirable drug reactions and, as a rule, is cheaper than combined antibacterial therapy. The regimens of the combined antimicrobial therapy should be used as initial therapy only in the case of the tentative polymicrobial etiology of surgical infection, when antibiotics, which cover the entire spectrum of suspected pathogens, are not available and can be administered as monotherapy. One of such situations is the empiric therapy of surgical infection, caused by the association of gram-negative aerobes and anaerobes. Gram-negative organisms such as *Pseudomonas spp*. and *Acinetobacter spp*., are often the pathogens of nosocomial pneumonia in surgical patients, as well as the pathogens of intra-abdominal infections and severe skin and soft tissue infections. These microorganisms, as a rule, are characterized by multiple resistance to antibiotics and require administration of the therapy with antipseudomonal drugs, such as Ceftazidime, Cefepime, Imipenem, Meropenem, Ciprofloxacin. It should be kept in mind, that in case of administration of new antibiotics with "ultra-wide" spectrum of action (for example, carbapenems) which cover the entire spectrum of suspected pathogens, their combination with other antibiotics (cephalosporins, metronidazole) is not only inappropriate, but also increases the risk of development of antibiotic resistance, undesirable reactions and the cost of treatment. In practice, there is often a combination of antibiotics with such drugs as non-steroidal anti-inflammatory, antihistamine drugs. However, it should be remembered that the use of antihistamines does not prevent the body sensitization to antigens and products of vital activity of bacteria. Nonsteroidal anti-inflammatory drugs can mask a clinical picture of infections, as well as dramatically increase the probability of drug interactions.

Administration of antibiotics in ambulatory surgery. Most patients with surgical diseases, including surgical infections, may be treated in the ambulatory surgery centers or at the polyclinic. In general, there is no difference between the surgical care provided at these institutions and in hospitals. That is why the main commonly accepted principles of preoperative antibiotic prophylaxis and rational antimicrobial therapy remain the same for all surgical patients and do not depend on the place of treatment.

However, it is worth pointing out some features regarding the choice of antibiotics in outpatient surgery:

1) in ambulatory patients with surgical infection in comparison with hospitalized patients, antibiotic-resistant strains of microorganisms (MRSA, VRE, *P. aeruginosa*, etc.) are pathogens more rarely, therefore, less expensive traditional antimicrobial drugs can also be effectively used for the treatment of ambulatory surgical patients, as well as new antibiotics;

2) in the majority of patients with surgical infection treated outpatiently, administration of oral regimens of antimicrobial therapy is effective.

I.5. SHOCK AND POLYTRAUMA IN SURGICAL PATIENTS

Shock *is an acutely starting critical condition of the body with progressing insufficiency of life support system, caused by acute blood circulation insufficiency, acute respiratory failure, microcirculation disturbances and tissue hypoxia, what is manifested in dysfunction of all physiological systems.*

"Shock" in English means blow, strike, collapse. The term was introduced by a scientist and physician of Louis XV's army de Eran (XVIII century).

Shock in surgical patients is a common attribute of many different pathological conditions, which are common in surgical practice. Shock is not a diagnosis itself, because it always hides the disease, which caused it. However, in case of shock, decompensation of vital functions of the body takes place, and therefore it requires immediate diagnosis and specific intervention. As in case of many other conditions, rational treatment of shock is based on a deep understanding of its etiology and pathogenesis.

Traumatic, hemorrhagic and endotoxic shock types are common in the daily routine of a specialized surgical department.

The number of patients with severe trauma, complicated by traumatic shock, does not decrease every year, but, on the contrary, tends to continuously increase. According to WHO, the problem of traumas takes the 3rd place (after cardiovascular and cancer diseases).

Rapid growth of technological progress and infrastructure of modern society, mainly all types of transport, urbanization of cities has caused the emergence of a new medical problem for the last two decades – *severe polytrauma* (simultaneous severe damage of several organs and anatomical and functional systems in a patient).

The difficulty of the provision of medical care to the patient with shock is caused by multiple organ and system damages of vital functions, which are usually accompanied by traumatic shock.

Death from traumatic shock, especially in case of delayed provision of medical care, as well as in non-specialized hospitals, remains rather high. This fact raises the problem of the treatment of shock on the background of polytrauma to the level of primary goals of practical medicine.

The cause of *hemorrhagic shock* is an acute blood loss. The background of pathophysiology of the shock is consistent and does not depend on the localization of bleeding (internal or external) and its association with trauma or specific pathology, which caused it (gastrointestinal bleeding, aortic aneurysm rupture, etc.).

Endotoxic shock may complicate many acute surgical diseases as a result of the influence of endotoxins and toxic metabolic byproduct of pathogenic bacteria on cell membranes, blood coagulation components and complement system, what leads to increased coagulation, cell damage and blood flow disorders, especially at the level of microcirculation.

Classification of shock based on etiopathogenetic sign.

1. Shock as a result of damaging factors of the environment (pain, exogenous):
- *traumatic shock* as a result of mechanical trauma;
- *burn shock* in case of mechanical trauma, burn shock in case of thermal trauma;
- *electric shock.*

2. Shock caused by a sensory input in case of the diseases of internal organs (pain, endogenous):
- *cardiogenic shock* in case of myocardial infarction;
- *nephrogenic shock* in case of kidney diseases;
- *abdominal shock* during intestinal obstruction, liver colic.

3. Shock caused by humoral factors (similar to collapse by mechanism) is called *humoral*:
- post-hemotransfusion shock;
- hemolytic shock;
- insulin shock;
- *toxic* shock (bacterial, infectious and toxic shock and shock in case of traumatic toxicosis).

Classification of shock based on etiological sign.

1. *Hypovolemic shock*:
1) anaphylactic shock in case of allergy;
2) hemorrhagic shock in case of blood loss;
3) hypovolemic shock in case of dehydration (exsicosis);
4) traumatic shock in case of trauma;
5) burn shock in case of burns;
6) toxic shock in case of poisoning;
7) endotoxic shock in case of infection.

2. *Cardiogenic shock* in case of AMI, myocarditis, during the latter stages of circulatory failure in case of cardiosclerosis, cardiomyopathy.

3. *Arrhythmogenic shock* in case of tachyarrhythmia or bradyarrhythmia.

4. *Shock in case of electromechanical dissociation* EMD with PAWP, cardiac tamponade, pneumothorax with mediastinal shift of organs.

Classification of shock based on the severity of manifestation (except for terminal states):

Grade 1 - *light*, arterial pressure above 90 mm Hg;

Grade 2 - *moderate*, arterial pressure from 90 to 70 mm Hg;

Grade 3 - *heavy*, arterial pressure from 70 to 50 mm Hg;

Grade 4 - *extremely severe*, arterial pressure below 50 mm Hg.

Classification of shock based on the time of onset.

A. *Primary (early),* occurring at the moment of injury or shortly after injury.

B. *Secondary (late),* manifested several hours after injury, when neuro-reflexory disturbances are enforced by intoxication of the absorbed breakdown products, or, as a result, of discontinuation of analgesic drug action.

There are 2 phases of shock: *erectile* and *torpid*. Despite various reasons and some peculiarities of the pathogenesis of these types of shock, *vasodilatation* is the main cause of the development of any shock, and, consequently an increase in the volume of the blood stream, *hypovolemia*, decrease of the volume of circulating blood (CBV) due to various factors: blood loss, redistribution of fluid between the blood stream and tissues or non-compliance of normal blood volume to the enlarged blood stream capacity as a result of vasodilation. Emerging discrepancy between CBV and blood stream capacity leads to a decrease of the minute volume of cardiac blood and microcirculation disorder.

Traumatic shock

There are many definitions, concepts and classifications of traumatic shock, which reflect the main essence of a patient's condition to different extents.

In general, ***traumatic shock*** *is a pathological phase process, one of the early and main components of a traumatic disorder, which develops in the body in response to the influence of severe mechanical trauma and is characterized by profound disturbances of vital systems of the body (neuroendocrine, circulatory, respiratory systems, metabolism).*

Etiology and pathogenesis of traumatic shock. The etiology of shock is diverse. Shock may complicate severe burns, injuries, surgical operation, fractures, bruises, and so on. Pathogenesis of traumatic shock is always uniform - the cause of traumatic shock is excessive mechanical or physical injury. Nor toxemia, not anemia do not cause and cannot cause traumatic shock Orbeli L.A., Petrov I.R., Veselkin PI., Popov V.I., et.al.). From the very beginning, the cause of shock is the *pain*, which causes primary disturbances of the nervous system, blood circulation, endocrine glands, metabolism.

There are various **theories of pathogenesis** of traumatic shock.

1. ***The theory of hypocapnia***. As a result of damage, carbon dioxide content is reduced in the blood. Venous stasis develops, vascular tone decreases. Oxygen deficiency occurs, volume of circulating blood decreases. This theory narrows the pathogenesis of traumatic shock to chemical changes in the body, ignoring initial neural mechanisms.

2. ***The theory of plasma loss.*** The blood stasis in paralyzed capillaries leads to the release of plasma into the pericapillary space. Congestion, oxygen starvation of tissues, poisoning of an organism by decay products develop. A. Blalock (1942) considered that shock was "peripheral blood flow insufficiency." V.V. Pashutin in 1981 drew attention to the role of reduction of circulating blood volume during shock. N. Freeman (1938), emphasizing the importance of changes in the circulating blood volume, narrowed the pathogenesis of shock to a vicious circle: "... Shock is not an organism condition but a process. The essence of this process is to reduce the circulating blood volume caused by tissue asphyxiation, which, in turn, is determined by insufficient blood flow ...".

3. ***Toxemia theory.*** Products of decay of injured muscles poison the organism, paralysis of capillaries, which become very permeable to the plasma, develops, circulating blood volume decreases.

4. ***Vascular spasm theory.*** The cause of shock involves sudden and severe reflexory spasm of blood vessels. This spasm leads to circulatory failure even before the development of endocrine disorders (R. Lerish).

5. ***Neuroreflectory theory.*** The flow of pain impulses from the site of damage causes an initial disturbance of many parts of the central nervous system, including the hypothalamus and reticular formation (in the medulla oblongata, the pons region of brain – Pons Varoli, the Mesencephalon). This excitation may be short. It manifests itself in the increase of blood pressure, vasospasm, shortness of breath, increased activity of endocrine glands and metabolism. Excitation leads to the depletion of

energy reserves of the central nervous system. The torpid phase of traumatic shock develops. The torpid phase is not a manifestation of inhibition and adaptive response of the nervous system. On the contrary, it indicates the exhaustion of the body capacities in response. The organs and tissues of a patient lose normal trophic innervation, metabolic disharmony approaches. This leads to hypoxia, acidosis, insufficient nutrition of tissues and to a decreased excretion of metabolic toxins from the body. In paralyzed capillaries of muscles, a significant part of the blood pools. As a result, its amount that circulates through organs and tissues decreases. Cardiac minute volume is reduced, arterial pressure decreases, an ability to compensate haemorrhage is lost.

Emphasizing the importance of the pathogenesis of traumatic shock, it is necessary to point out once again that in case of a damage a powerful impulse flow induced by the action of a pathological stimulus goes from the affected region via afferent animal and vegetative fibers to the corresponding centers of the brain and spinal cord. In the region of cortex and subcortex an excitation concentrates, that changes to inhibition, as a rule. Prolonged occurrence of such inhibition focus can lead to depletion of nerve centers. In the torpid phase, inhibition of the protective type extends as a result of irradiation to other parts of the brain. Consequently, violation or even failure of controlling and regulatory functions of the central nervous system, and after that – violation of blood circulation, respiration, the function of endocrine glands and metabolism occur. These disorders, in turn, further aggravate the disturbances of the function of the nervous system, creating the so-called "chain reaction" and "vicious circles" that play a significant role in the development and result of traumatic shock. For example, as a result of painful irritations by reflectory way, an increase of adrenal secretion by the adrenal glands and the pituitrin - by the pituitary gland occur. The appearance of excessive amount of adrenaline in the body during the development of the erectile phase of shock as a result of its sympathergic action, is accompanied by narrowing of vessels, increased breakdown of glycogen in the liver and muscles, decreased glucose uptake, violation of oxidative processes and acidosis. These metabolic disorders interrupt functional status of tissues, function of the cardiovascular system, and in severe cases, can lead to irreversible changes.

Circulatory disturbances in case of a shock, which appear in a reflexive way, subsequently lead to oxygen starvation, due to which an already changed function of the nervous system is affected. Circulatory disorders also lead to plasmorrhea, deposition of blood and development of stasis. In turn, the stasis enhances hypoxia, the latter increases plasmorrhea and so on. The balance of vitamins changes, deficiency of vitamin C and B-complex vitamins occurs.

Thus, for pathogenesis of traumatic shock "chain reaction", characterized by increasing disturbances of the activities of many systems and organs, is typical. Circulatory and respiratory disturbances lead to metabolic disorders: reduction of main metabolism, anoxia, accumulation of lactic acid, hypoproteinemia, growth of non-protein nitrogen, hyperglycemia in the erectile phase, deficiency of ascorbic acid, decrease of redox processes, reduction of the chloride content, electrolyte metabolism disturbances. In case of soft tissue crushing, important role belongs to toxemia in connection with the accumulation of histamine-like products, as well as the activity of microbes, which rapidly develop in traumatized, nonviable tissues. A damaged organ, crushed limb is a source of irritation and intoxication, what contributes to the development of the phenomena of shock.

Clinical findings. Symptomology of traumatic shock is diverse and depends on the phase and degree, character and localization of trauma.

Motor and speech excitation with maintenance of consciousness are characteristic for the *erectile phase of shock*. Patients are loudly complaining of pain, underestimating severity of an injury. Pain reaction is sharply increased. The voice is confused, phrases are fragmentary, the look is anxious; increased sweating. The face and visible mucous membranes are more often pale, sometimes a face is hyperemic. The majority of patients show general hyperesthesia and hyperreflexia of the skin and tendon reflexes. The width of pupils is normal, their reaction to light is fast. Pulse rate is normal, but sometimes the pulse is slow, cordy, of satisfactory or even good volume. Blood pressure is normal or elevated up to 150-190 (maximum) and 100 (minimum). The erectile phase usually takes seconds or minutes and, as an exception, hours.

It is important to note that the sharper manifested disturbance in the erectile phase is, the more difficult the torpid phase usually proceeds and the harder the prognosis is.

The transition of traumatic shock from the erectile phase to the torpid one usually occurs quickly, within a few minutes. In some cases, it occurs gradually. In this case, depending on the extension of the processes of excitation and inhibition, there is one or another symptom complex: for example, motor excitement may be combined with low arterial pressure or general depression – with increased pressure.

The torpid phase is characterized by inhibition of the function of most systems of the affected organism. The classic description of the torpid phase of a shock belongs to M.I. Pirogov: "With a torn hand or foot, the rigid wounded is lying still at the dressing station: he does not scream, does not whimper, does not complain, does not accept anything, participates in nothing and does not require anything; his body is cold, his face is pale, like a corpse; his glance is rigid and is looking away; his pulse is thread-like, barely noticeable under the finger and with rapid intervals. The rigid wounded does not answer any questions at all, or only about himself in a whisper hardly heard; breathing is also barely noticeable. The wound and the skin are almost completely not sensitive; but if an ill nerve hanging from the wound is irritated by something, then the patient with one slight contraction of his own muscles shows the signs of sensation. Sometimes this condition subsides in several hours with the help of stimulants; sometimes it continues without changes until his death.

Anamnesis of the disease: an injury is reported. The course of the disease is acute, progressive.

The general condition of a patient, as a rule, varies from average severity to extremely severe condition. Severe traumatic shock is often caused by severe pain syndrome. The patients are agitated. Sometimes there is an impairment of consciousness, leading up to coma. The mind is inhibited, changing to depressive syndrome.

Appearance of the patient – pale or pale gray face, acrocyanosis, cold sticky sweat, cold limbs.

Decrease of temperature.

Objective status. Cardiovascular system – frequent weak pulse, decrease in arterial and venous pressure, collapsed subcutaneous veins. Respiratory apparatus – increased frequency and weakening of breathing. Abdominal cavity organ – characteristic features in the presence of injury of internal organs of the abdomen and retroperitoneal space. Musculoskeletal system – presence of bone carcass damage (for example, fracture of pelvic bones, fractures of tubular bones, separation and rupture of a distal part of a single limb, multiple fractures of the ribs).

The local status depends on the damaged anatomical-physiological area (APA) as a result of the injury.

In case of the most frequent damage – ***rib fractures***, an acute pain in the region of the fracture is observed during palpation, bony crepitus.

Flail chest (i.e., fractures along 2-3 anatomical lines accompanied by a violation of the integrity of the ribcage and pathological mobility of the chest) with development of the so-called "*paradoxical breathing*" is characterized by paradoxical movement of the chest – falling of the floating segment back during inhalation and its extrusion during exhalation.

In case of fracture of the sternum and ribs, a deformation in the fracture region is observed. By auscultation the breath is diminished on the damaged side.

In case of complications of the rib fracture by ***hemothorax*** – in the lower parts or above the entire surface of the lung on the injured side dullness of percussion sound, diminished breath sounds are observed; in case of ***pneumothorax*** – tympanitis, decreased breath sounds on the damaged side.

According to standard schemes, ***the plan for laboratory and instrumental examination*** of a patient with traumatic shock includes:

1. Clinical blood test.
2. Clinical urine test.
3. Estimation of the CBD.
4. Biochemical blood analysis.
5. Blood group and Rh affinity.
6. Coagulogram.

7. Plan radiography of the skull, pelvis, limbs, chest and abdominal cavity organs in two views.
8. ECG.
9. Bronchoscopy (in case of combined trauma by indications).
10. CVP measurement.
11. Ultrasound examination of the pleural and abdominal cavities.
12. Puncture of the pleural cavity.

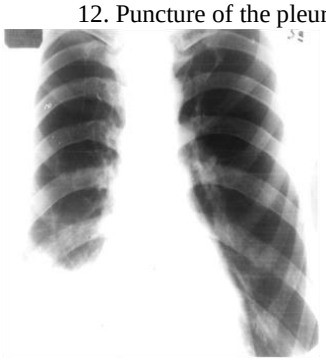


Fig. 22. Right-sided posttraumatic hemothorax

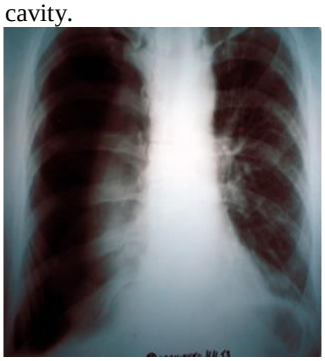


Fig. 23. Right-sided posttraumatic pneumothorax

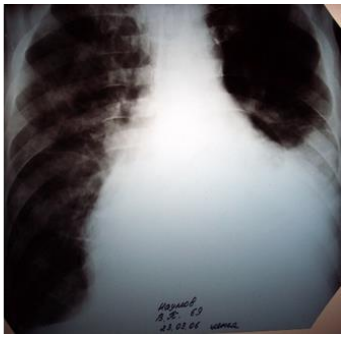


Fig. 24. Hemopericardium with cardiac tamponade

13. Diagnostic laparoscopy.
14. Diagnostic thoracoscopy.

Characteristic pathological changes.

1. Clinical blood test – in presence of signs of bleeding, anemia is possible (decrease of Hb, red blood cells), elevated ESR.
2. Clinical urinalysis – no changes are possible.
3. CBD is estimated in case of bleeding.
4. Biochemical blood test – possible increase of transaminases, C-reactive protein. For an abdominal trauma increased bilirubin, amylase is typical.
5. Coagulogram – no changes are possible, but in case of coagulopathy development, the changes are possible, typical for the syndrome of disseminated intravascular coagulation (DIC).
6. Plain X-ray of the chest organs in two views – fractures of the ribs, sternum, hemo- and pneumothorax, hemopericardium, cardiac tamponade may be observed.

The X-ray pattern of hemothorax is quite specific – it is characterized by the intensive homogeneous opacification on the injured side with a transverse upper contour (Damoiseau's line), the diaphragm-rib sinus is not visualized (Figure 22). Depending on the volume of intrapleural bleeding by P.S. Kupriyanov (1950) the following are distinguished: *small hemothorax* – opacification to the angle of the shoulder blade (up to 500.0 ml of blood); *average hemothorax* – to the level of the Vth rib anteriorly, the middle of a shoulder blade on the posterior surface (up to 1000,0 ml); *large hemothorax* – to the level of the IInd ribs anteriorly (1000,0-1500,0 ml); *total hemothorax* – complete opacification of the pleural cavity to the apex of a lung with displacement of the mediastinal organs to the healthy side (more than 1500.0 ml).

Pneumothorax is radiologically manifested by the presence of air in the pleural cavity and atelectasis, collapse (decrease in volume) of the lung (Fig. 23). Depending on the amount of free air in the pleural cavity and the degree of lung compression, it can be small, medium and total.

In case of hemopericardium with cardiac tamponade (Fig. 24), a change in the configuration of the heart and expansion of its borders are observed.

7. ECG – tachycardia, signs of myocardial hypoxia.
8. *Bronchoscopy* in case of combined trauma – red blood flow from a bronchus in case of a damaged lung. Tracheal and bronchial injuries can be seen.
9. *Measurement of CVP: a sharp decrease is observed during massive blood loss.*

10. Ultrasound examination of the pleural and abdominal cavities – in case of hemothorax or hemoperitoneum, fluid is found in the pleural and abdominal cavity on the side of the damage.

11. *Puncture of the pleural cavity* – serves as a diagnostic and therapeutic manipulation for a suspected hemopneumothorax.

12. Diagnostic laparoscopy and thoracoscopy (Fig. 25) are highly informative methods, they allow to specify the nature, localization of damage and to choose therapy management.

Differential diagnosis: is made on the principle of confirmation or exclusion of an injury of various organs and systems taking into account the involved anatomical and physiological area.

Treatment of patients with traumatic shock. *The staging principle of providing care to the sufferers from trauma* in an urban setting is carried out by emergency medical service teams and specialized surgical hospitals teams.

Stages of ***emergency medical care delivery*** for the patients with polytrauma, loss of blood and shock should be divided into a number of consequential periods, such as: *pre-hospital life support and diagnostic period, in-patient diagnostic, life support and surgical period* and in-patient post-shock period.

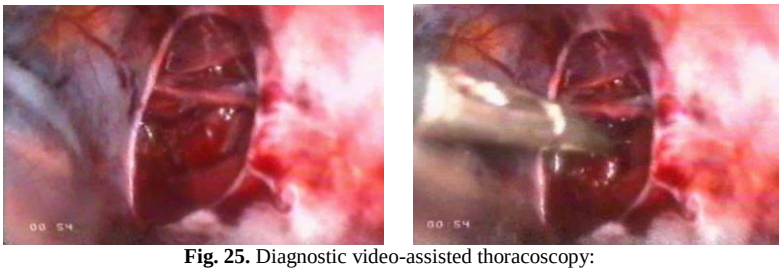


Fig. 25. Diagnostic video-assisted thoracoscopy:
a - bleeding from the intercostal artery; b - achievement of hemostasis by clipping of intercostal artery

First pre-hospital medical emergency treatment, which is time-constrained from the moment of the arrival of an ambulance team to the place of the event till the patient's transportation to the hospital ER (preferably a specialized one).

Main tasks of the stage:

1) to perform resuscitation diagnostics in the shortest time possible, first of all to determine which vital functions are violated;

2) to carry out transport immobilization in case of bone fractures of the limbs and temporary stopping of external bleeding (twisting of a tourniquet, etc.);

3) to provide neurovegetative protection of a patient suffering from pain by the administration of narcotic drugs (Promedol, Omnopon), other analgesics (Sodium oxybutyrate or Sodium thiopental) and sedative (Sibazon) drugs;

4) to start infusion therapy to support homeostasis (Refortan, Stabizol, Voluven, HyperHAES, etc.);

5) to carry out oxygen therapy in case of symptoms of acute respiratory insufficiency.

The choice of disease management in a hospital depends, first of all, on the phase of a shock, damage of one or another organ or system and is chosen individually. Rapid development of polysystem and multiple organ failure in case of traumatic shock constantly requires maximum reduction of the time for health care delivery at all stages.

At this stage, all patients, depending on nature, localization and degree of injury severity, can be divided into 4 groups:

Group I – the most severe injured patients with traumas, constituting immediate life-threat at the moment of initial examination (interruption of breathing or gross respiratory failure, cardiac arrest, arterial hypotension below 70 mm Hg, manifestation of external signs of profuse bleeding);

Group II – patients in dangerous conditions, but which do not threaten life at the moment. Most of such patients have penetrating wounds of the chest and abdominal cavity, bone fractures, closed injuries affected by unstable hemodynamics with signs of respiratory failure and blood loss;

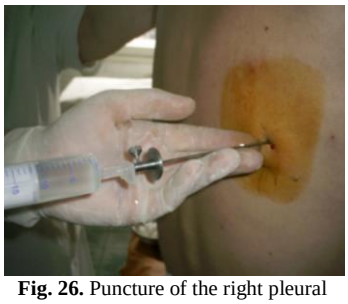


Fig. 26. Puncture of the right pleural cavity

Group III – patients with potentially dangerous injuries (with signs of injury of different body anatomical and physiological area, but stay in a relatively satisfactory condition);

Group IV – patients in a relatively satisfactory condition, having small damaged regions and wounds of safe localization of various regions of the body, dislocations, fractures of small bones under the influence of mechanical factors – road accidents, falling from height, a number of industrial injuries ...

Hospital phase includes measures (often carried out simultaneously in the conditions of an operating room):

1) urgent laboratory-instrumental diagnosis;

2) prolongation (if necessary) of resuscitation measures;

3) performance of urgent surgical interventions according to vital indications, depending on the nature of an injury;

4) delivery of pathogenetically grounded intensive infusion-transfusion therapy.

Puncture of the pleural cavity. Puncture of the pleural cavity serves as a diagnostic and therapeutic manipulation for suspected hemopneumothorax (Fig. 26).

To remove air, a puncture is performed under local anesthesia with a solution of Novocaine in the second intercostal space along the midclavicular line for a sitting patient. If, due to the critical condition the patient cannot sit, the puncture is performed in the position lying on the healthy side in the V – VIth intercostal space along the midaxillary line.

To remove blood, a puncture is performed in the VII-VIIthI intercostal space along the posterior axillary line with a patient sitting. To avoid damage of intercostal vessels, a puncture is performed in the upper border of the subjacent rib.

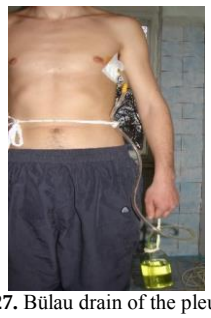


Fig. 27. Bülau drain of the pleural cavity

Fig. 28. Stabilization of the thoracic ribcage in case of "flail chest"

Analysis of the content of the pleural cavity – *Ruvelua-Gregoire test* is used to solve the problem on suppressed or ongoing bleeding into the pleural cavity and is evaluated as *positive (continuing bleeding)*, when the drained blood from the pleural cavity coagulates, and indicates *hemostasis* in the pleural cavity (*negative test*) when the blood, drained out of the pleural cavity, does not coagulate.

Methods of operative treatment and indications for them in the patients with traumatic shock. Surgical treatment depends on the damage of one or another organ or system, is chosen individually and focused at the restoration of the integrity of an organ and its functions.

The most commonly performed surgical operations: primary surgical management of wounds, suture of wounds of the liver and spleen (or splenectomy), suture of wounds of the stomach, small and large intestine (or their resection), suture of wounds of the urinary bladder, pancreas and kidney (or nephrectomy), drainage of the pleural cavity (Fig. 27), stabilization of the thoracic ribcage in case of "flail chest" with the help of special repositioning and stabilizing device for the fixation of a flail chest segment, developed at the Kharkiv Institute of General and Emergency Surgery of NAMS named

after V. T. Zaitsev (Fig.28), suture of wounds of lungs or their resection (recently more often by thoracoscopic intervention).

In each particular case, according to the indications, the main surgical intervention is combined with other operations – microcholecystostomy, intestinal intubation or entero- and colostomy, nephrostomy, drainage of the abdominal and pleural cavities, etc.

Pathogenetically grounded intensive care.

A. General guidelines.

1. Therapy of traumatic shock should be pathogenetic and differentiated depending on the phase and degree of shock, nature of an injury, individual characteristics of an organism, environmental conditions.

2. The complexity of pathogenesis and involvement in the pathological process of a large number of systems and organs lead to the necessary use of complex solutions.

3. The choice of treatment complex depends on the phase and degree of shock, taking into account compensation, stabilization or decompensation of the process.

4. Assessment of the degree and severity of shock during an initial examination is difficult. In this regard, in addition to the general condition of a patient, it is also necessary to take into account nature and severity of traumas during examination.

5. Therapy of shock should be performed taking into account individual characteristics of a patient's organism: age, gender, presence of somatic diseases and issues that aggravate or mask the development of shock (blood loss, fatigue, exposure to cold, fasting, alcohol intoxication, emotional and nervous commotion, and mental illness).

6. Due to the danger of the development of a secondary shock and lingering course of the torpid phase, anti-shock measures and patients' management should be prolonged.

B. Substantiation of the complex of therapy measures in case of shock. All measures in the complex of intensive therapy of traumatic shock can be divided into five groups:

1) focused on the normalization of disturbances of the nervous system;

2) focused on the control of blood circulation disturbances;

3) focused on the control of respiratory disturbances and elimination of hypoxia;

4) measures that normalize metabolic disorders;

5) focused on the elimination of endocrine disorders (many of the medications administered in case of shock have a combined effect, due to which this division is conditional).

Taking into account parallel development of pathological and compensatory adaptive changes in case of shock, it is advisable to distinguish the groups of drugs which have similar focus in the mechanism of action with adaptive and compensatory reactions of the organism. These reactions in response to trauma become active in a certain sequence. First adaptive reactions become active, then compensatory, passive protective and, finally, terminal mechanisms. Recovery from shock, if a process has not reached the so-called "irreversible changes" and inclusion of terminal mechanisms of sanogenesis occur in reverse order. Pharmacological preparations have an ability to influence various components of patho- and sanogenesis. These agents are represented in 4 groups: metabolic, stimulating, hypobiotic and inhibitory.

Stimulants are the drugs, which activate the functions of physiological systems and adaptation mechanisms that enhance metabolism. Stimulants include various *neurotransmitters, hormones and their derivatives* (for example, *hydrocortisone*, as well as its analogues – *prednisolone, dexamethasone*).

Stimulants are also the drugs that temporarily inhibit enzymes involved in the destruction of hormones and neurotransmitters. Thus, for example, *proserin* or *peganine hydrochloride* affect enzyme acetylcholinesterase, which destroys acetylcholine, systemic action is manifested in the activation of the cholinergic system. Stimulants include *diuretics, analeptics, cardiac products, immunostimulants*.

Hypobiotics are the drugs that, influencing the nervous system, produce effects characteristic for a torpid syndrome: analgesia, hypothermia, inhibition of mental, emotional and motor activity,

functions of visceral organs and metabolism. Hypobiotic agents include: *narcotic analgesics, neuroleptics, tranquilizers, anesthetics, hypnotic drugs, muscle relaxants, GABA-ergic agents.*

Inhibitors are the drugs that suppress exceedingly manifested active protective reactions of the body, and moreover they act directly on the organ or system, and not in a roundabout way through the nervous system of an organism (inhibitors differ from the hypobiotics by the latter). In general, inhibitors and hypobiotics are counterbalanced by stimulating agents. The inhibitors include: *diphenhydramine*, which blocks H1-histamine receptors, *heparin*, which inhibits the transformation of prothrombin into thrombin, as well as fibrinogen into fibrin, what is an anticlotting action, *enzymes*, which are the part of drugs of *Contrykal, Trasylol*, drugs belonging to the *group of antispasmatic drugs* (*No-Spa, Papaverine), antipyretics and non-narcotic analgesics*.

The following drugs have the most effective antishock properties in the ceiling doses: *Droperidol, hydrocortisone, prednisolone, diphenhydramine hydrochloride, Calypsol, Actovegin, novocaine, Diazepam, sodium oxybutyrate, morphine hydrochloride, Pituitrin, Contrykal, Hexenal, Gordox* (efficacy is shown in decreasing order).

Thus, it should be considered that during shock the drugs with hypobiotic action are the most effective, then with metabolic and inhibitory action, and at the last place – with stimulating action.

Treatment of the patients which underwent various surgical interventions for traumatic chest injuries is performed individually in the *postoperative period*. Different approach to the management of patients in the postoperative period is determined by the features of mechanical trauma, nature and degree of damage of the intrathoracic organ, thoracic structure, combination of injuries, severe shock, functional respiratory disorders, blood circulation, trauma of other vital organs, type of surgical intervention, age of a patient, complications, developed during an operation and others.

The results of surgical treatment of damaged intrathoracic organs showed that the prevention of postoperative complications should be started as early as in the operating room. Before extubation special attention is paid to the tracheo-bronchial tree lavage and restoration of proper independent breathing. Multiple lavage of the tracheobronchial tree during a surgery and before the removal of an advanced airway, especially in case of a lung injury, is performed with Metras catheters, introduced into the main and lobar bronchi.

If a surgery was performed for one of the intrathoracic organs, it is necessary to expand all lung portions on the operating table. Only after the restoration of proper independent breathing and gas exchange, which is controlled by the indices of blood gases, acid-base state, hemodynamics of large and small circles of blood circulation, absent rales in the lungs, an advanced airway can be removed.

Prolonged artificial ventilation of the lungs is administered to the patients with multiple chest trauma after an extensive operation for one of the intrathoracic organs with massive blood loss and severe cardiorespiratory disturbances, as well as to elderly people. Early restoration of independent breathing does not guarantee tissue oxygen regime and may lead to acute decompensation of cardiac activity. Such patients after thorough bronchial lavage with an intratracheal tube are transferred to the intensive care unit, where artificial ventilation continues. Prolonged AV is performed on the ground of the study of homeostasis indices, respiratory mechanics, pulmonary volumes.

If there are indications for a longer AV, tracheostomy is suggested. The immediate efficacy of surgical treatment of the organs of the chest and abdominal cavity depends on the active management of patients, therapeutic exercises and lavage of tracheobronchial tree.

It should be emphasized that postoperative bronchial sanation depends on the severity of a patient's condition, combination of injuries, postoperative complications (atelectasis, pneumonia), severity of the cough reflex, etc.

In the patients with poorly expressed cough reflex with critical general condition, sharply decreased cardiovascular parameters, the bronchial tree lavage with a puncture of the trachea is not always effective. Bronchial sanation in such difficult patients is performed by nasotracheal catheterization of bronchial tubes with Metras catheters or by bronchoscopy. However, the most effective sanitation bronchoscopy cannot be applied in patients with combined craniocerebral trauma and with fractures of the cervical spine as often as it is sometimes necessary.

Prevention of atelectasis, pneumonia, abscesses of the lungs, empyema of the pleura, pericarditis, thromboembolism of the pulmonary artery, suppuration are the issues of high priority in the postoperative period.

Prevention of atelectasis and pneumonia in the injured persons, especially with lung rupture, should be performed at all stages of surgical treatment. Before surgery – aspiration of the content of the trachea, bronchi, normalization of homeostasis. During the operation on the lungs – careful operation, considering of the anatomical features of the segments, separation of the lung along the entire length with compulsory transection of the pulmonary ligament, multiple aspiration of the content of tracheobronchial tree and expansion of all parts of the lung. In the nearest postoperative period – inhalation of enzymes, bronchodilators, active coughing up sputum or its aspiration with the creation of low positive pressure in the bronchial tree. The applied method of prevention and treatment of these complications greatly reduced the number of postoperative atelectases.

Prevention of pleural empyema is carried out by a balanced drainage of the pleural cavity. In case of the pleural empyema, drainage of the pleural cavity with active aspiration of the contents, its lavage with 1% solution of dioxidine and administration of antibiotics depending on the sensitivity of the microflora are performed.

Prevention of thrombosis and embolism should be carried out during an operation and in the postoperative period. In patients with increased coagulation, increased concentration of fibrinogen, especially fibrinogen B, administration of anticoagulants of direct action until stable normalization of coagulogram showings is necessary.

In case of severe traumas of the chest and stomach, emergency conditions may occur in the form of *total hemothorax, hemopneumothorax, cardiac tamponade, ruptures of the internal organs of the abdominal cavity, hemoperitoneum with the development of hemorrhagic shock*, in case of which an emergency thoracotomy or laparotomy is indicated to eliminate the source of bleeding.

Complications, which occur *in the postoperative period* with various injuries on the background of traumatic shock, are divided into *early* and *late* ones.

Early complications include: atelectasis of the lung, myocardial infarction, acute cardiovascular and acute cardiopulmonary insufficiency, myocarditis, peritonitis, acute adhesive intestinal obstruction.

Late complications include: pleurisy, purulent tracheobronchitis, pneumonia, pleural empyema, pericarditis, post-operative wound inflammation, abscesses of the abdominal cavity.

Examination of the patient's disability after serious trauma and traumatic shock. Observation of long-term results of treatment confirms that persons which suffered from serious injuries are not a socially unpromising group of patients. Work qualification is especially important for evaluation of the results of surgical treatment of such patients. On the average up to 80% of the operated persons return to previous work after surgical treatment of traumatic injuries of the organs of the chest, abdomen, pelvis and limbs, up to 15% of patients change to easier work, and 5% – must quit the job and get a disability status.

Hemorrhagic shock

Hemorrhagic shock *is a critical condition associated with an acute blood loss and resulting in the crisis of macro- and microcirculation, multiple organ and polysystem dysfunction syndrome.*

Hemorrhagic shock develops with acute blood loss of more than 20-30% of CBV. In clinical practice, per se, it is observed in case of suicidal attempts (slit wrists); ectopic pregnancy interrupted by a rupture of the fallopian tube; spontaneous rupture of the spleen; ulcer bleeding, etc. In most cases, pathogenesis of shock depends not only on the volume and time of blood loss, but also on the mechanism of its occurrence (volume and nature of an injury).

In *the pathogenesis of hemorrhagic shock*, it is necessary to distinguish the following links: acute blood loss, reduction of CBV, venous return and cardiac output lead to the activation of the sympathetic-adrenal system, what results in the spasm of blood vessels, arterioles and precapillary sphincters in various organs, including the brain and the heart. Redistribution of blood in the bloodstream, autohemodilution (fluid transfer to the bloodstream) occur on the background of

decreasing hydrostatic pressure. Cardiac output continues to decrease, persistent spasm of arterioles occurs, rheological properties of blood change (aggregation of erythrocytes "sludge" phenomenon).

At a later stage the peripheral vascular spasm causes the development of microcirculatory disturbances and leads to irreversible shock, which is subdivided into the following phases:

1) *the phase of vasoconstriction* with a decrease in blood flow in the capillaries;

2) *the phase of vasodilation* with the expansion of the vascular space and decrease in blood flow in the capillaries;

3) *the phase of disseminated intravascular coagulation (DIC*);

4) *the phase of irreversible shock.*

In response to the DIC, the fibrinolytic system is activated with lyzing of clots, and severe violations of hemocoagulation with possible occurrence of massive bleeding develop.

Not only an absolute value of blood loss, but also the body reaction to it from the adaptation reserve, which can be sharply reduced in case of different concomitant pathology, are of practical importance for the assessment of the severity of hemorrhagic shock.

Compensated, decompensated, reversible and irreversible hemorrhagic shock are distinguished by severity.

There are ***4 degrees of hemorrhagic shock.***

1st Degree. Deficiency of CBV up to 15%. BP is above 100 mm Hg, central venous pressure (CVP) within normal range. Insignificant pallor of the skin and increase of the pulse rate up to 80-90 beats / min, hemoglobin 90 g / l or more.

IInd Degree. Deficiency of CBV up to 30%. Moderately severe condition, weakness, dizziness, blackouts, nausea, retardation, skin pallor. Arterial hypotension up to 80-90 mm Hg, decrease of CVP (below 60 mm H_2O), tachycardia up to 110-120 beats / min, decrease of diuresis, hemoglobin up to 80 g / l or less.

IIIrd Degree. Deficiency of CBV is 30-40%. The condition is serious or critical, retardation, confused mental state, skin pallor, cyanosis. BP is below 60-70 mmHg. Tachycardia up to 130-140 beats / min, poor pulse volume. Oliguria.

IVth Degree. Deficiency of CBV is more than 40%. Extreme degree of inhibition of all vital functions: no consciousness, BP, CVP and the peripheral arterial pulse are not determined. Breathing is superficial, frequent. Hyporeflexia. Anuria.

Clinical manifestations. At an early stage, a patient may look relatively normal, but he has tachycardia at rest, and a decreased pulse pressure; orthostatic changes of pulse and blood pressure are also possible. With massive blood loss, the skin becomes cold and wet due to the blood outflow to vital organs. Young patients may not have any other manifestations of bleeding, despite the fact that the deficit of circulating blood volume reaches 25-30%. Below this limit or at an earlier stage of blood loss in more debilitated patients a sharp drop of blood pressure and cardiac output occur. Therefore, early detection of signs of hemorrhagic shock and its aggressive treatment to prevent decompensation are necessary.

The clinical picture of hemorrhagic shock is determined by the mechanisms, which lead to the deficiency of CBV, changes in the acid-base state (ABS) of blood and electrolyte balance, disturbances of peripheral circulation and DIC syndrome.

The symptom complex of clinical signs includes: weakness, dizziness, thirst, nausea, xerostomia, blackouts, skin pallor, cold clammy sweat, sharpened face features, tachycardia and weak pulse strength, low BP, shortness of breath, cyanosis.

In the course of hemorrhagic shock, there are 3 stages.

Stage I – *compensated, reversible shock* occurs with the blood loss of 15-25% CBV (up to 1300.0 ml of blood). Blood pressure at the same time slightly decreases, moderate tachycardia is observed.

Stage II – *decompensated reversible shock* is accompanied by loss of blood of 26-50% of CBV (1300.0-1800.0 ml of blood), there is a decrease in BP (systolic blood pressure below 100 mm Hg), tachycardia up to 140 per minute.

Stage III – *irreversible shock* occurs in case of acute blood loss of more than 50% of CBV (1800,0-2500,0 ml of blood), blood pressure is below 60 mm Hg or not determined, the pulse is more than 150 beats per minute.

In case of compensated shock, skin pallor, cold sweat, small and rapid pulse, arterial pressure within the normal range or slightly reduced, are observed, urinary excretion decreases.

In case of decompensated reversible shock, the skin and mucous membranes are cyanotic, a patient is obtunded, the pulse is small, rapid, arterial and central venous pressure decreases, oliguria is developing, the Shock Index (Allgöwer index) is increased, ECG shows a violation of myocardial supply.

In case of irreversible shock, there is absence of mind, arterial pressure is not determined, the skin is of marbled appearance, anuria, termination of urine production, is observed. The Shock Index is high. To assess the severity of hemorrhagic shock, the evaluation of CBV, volume of blood loss are important.

Clinical picture of hemorrhagic shock is defined by the mechanisms that lead to the deficiency of CBV, change of ABS of blood and electrolyte balance, to the violation of the peripheral circulation and DIC syndrome.

During the interview a patient presents with weakness, dizziness, thirst, nausea, dry mouth, blackouts, skin pallor, cold clammy sweat, sharpened face features, tachycardia and weak pulse strength, decreased BP, shortness of breath, cyanosis.

A case history depends on the causes resulting in acute blood loss. The course of the disease is acute, progressive.

Clinician-observed data. General condition of a patient is more often severe. Consciousness can be clear or impaired in different degrees. A forced position due to kinesalgia, febrile temperature are observed. A patient is adynamic. With fairly massive blood loss, the skin becomes cold and wet due to the outflow of blood to vital organs.

Cardiovascular events – tachycardia at rest and decrease of pulse pressure; possible orthostatic changes in pulse and blood pressure. In case of the deficiency of circulating blood volume of 25-30% a sharp drop in blood pressure and cardiac output are observed. Breathing organs – without specific characteristics, except for the cases when hemorrhagic shock is caused by pulmonary bleeding. Organs of the abdominal cavity – without characteristic features, except for the situation when hemorrhagic shock is caused by gastrointestinal bleeding.

Status localis. Depends on the type of the pathology in one or another system.

In accordance with the standard schemes, ***the plan for laboratory and instrumental examination*** of a patient with hemorrhagic shock (in case of any type of blood loss) includes:

I. *Compulsory in an urgent order:*

1) erythrocytes, Hb, Ht, platelets, fibrinogen;
2) diuresis;
3) CVP.

II *After stabilization of the condition:*

1) X-ray of the lungs;
2) FGDS;
3) ECG;
4) ABS and blood gases.

Differential diagnosis is performed between all other pathological processes, the symptomatology of which includes leading symptoms of bleeding.

Treatment of a patient with hemorrhagic shock.

First medical aid at the pre-hospital stage:

1) to perform resuscitation diagnosis for the detection of a possible source of bleeding;

2) to stop external bleeding temporarily (by twisting a tourniquet, etc.) in case of fractures of the limb bones and injuries of the vessels;

3) to start infusion therapy to stop bleeding and maintain CBV (hemostatics – 1% solution of calcium chloride, 5% solution of aminocaproic acid, fibrinogen, vitamin C, hemodynamic blood substitutes – albumin solution, Refortan, Stabisol, Voluven, HyperHAES, etc.);

4) to carry out oxygen therapy.

Choice of medical management at the hospital. The purpose of therapy measures for hemorrhagic shock is rapid restoration of circulating blood volume, accurate diagnosis and correction of a source of bleeding. Venous access provision is the main requirement to fill the circulating blood volume (CBV). To perform such measures, a patient can be placed in the Trendelenburg position and have antishock costume put on him. These two techniques help to move CBV from the periphery to the central circulation, create some "autohemotransfusion" and increase vascular resistance, what leads to an increase in systemic blood pressure and an increase of blood flow to vital organs.

The use of central intravenous lines is controversial, since measurement of central venous pressure (CVP) is not always informative or necessary in case of sequent blood loss. In addition, insertion of such lines is dangerous due to complications. On the other hand, in unstable patients, in patients with severe chest trauma and in patients with poor cardiovascular reserve, CVP monitoring can be useful for planning of reanimation measures.

The number of IV accesses placed depends on the volume of blood loss and the severity of a patient's condition. Due to the absence of a special calculation for the required number of IV lines, the following principle may be applied: it is better to have too many than too few. Venesection is usually reserved for the patients with very weak peripheral veins or for very serious patients with massive blood loss, which require maximum efforts during initial resuscitation.

The volume of intravenous fluid used in case of hemorrhagic shock depends on the amount of blood loss, as well as the presence of hemorrhage at the time of infusion therapy. Except for the cases of massive bleeding, in the beginning blood products are not usually necessary. The first measures are to take blood to determine its group and response to compatibility and start infusion therapy with crystalloids or colloids. Although there are certain contradictions in this regard, in most resuscitation centers Ringer's lactate solution or a common saline solution, instead of colloids, is used as an initial IV fluid. Considering that the effectiveness of these types of fluids, absence of additional risk of respiratory distress syndrome in adults (RDSA) and significant difference in cost, *the crystalloid solution for initial therapy is a solution of choice.*

The decision to *start blood transfusion* is taken on the basis of the general clinical picture, a patient's condition on admission and his reaction to the infusion of the crystalloid solution, as well as the control of bleeding. If a patient is dying at the time of hospitalization, it is necessary to start transfusion of the blood group O immediately. More often, hemotransfusion is started after the introduction of 2-4 liters of crystalloid solution, on the condition that the patient has the signs of hypovolemia or continuing bleeding. In such cases, transfusion of the same group blood is completely safe, furthermore, it can be obtained in 10-15 minutes.

Another factor, which defines the beginning of hemotransfusion is *hematocrit*. Since a certain time is required for interstitial fluid to get to the blood-stream, the hematocrit dilution is not initially observed and therefore cannot accurately represent the circulating blood volume. The patients with massive blood loss and low hematocrit on admission are an exception. And although this indicator of hematocrit is not a correct representation of the circulating blood volume, it serves as an indicator of severe bleeding and, consequently, of the demand for blood transfusion. Another function of the initial study of hematocrit is to establish its output value. Further evaluations of hematocrit can be used later for raw assessment of blood loss and may help to make decisions on hemotransfusion.

From perspective to other therapeutic measures in case of hemorrhagic shock, they come down to the control of vital signs of diuresis and hematocrit. Unstable patients may be consequentially administered more invasive monitoring with the placement of a CVP-catheter, intraarterial infusion lines and the Swan-Ganz catheter. The placement of the above mentioned systems may be postponed until the patient is transferred to the intensive care unit. Other measures include the blood test to support the output values of coagulation function, platelet count, electrolyte test, cardio monitoring and an administration of additional oxygen.

Pathogenetically grounded ***conservative therapy.***

1). *Restoration of CBV*: Polyglucin (Rheopolyglukin) – 400 ml, Gelofusine – 500 ml, Refortan – 500 ml, Stabisol – 500 ml, glucose – 10% solution, crystalloids. The volume of infusion therapy should be up to 200% of the volume of blood loss.

2). *Restoration of oxygen capacity of the blood*: erythrocyte mass (suspension) up to three days of storage. The main task is to provide adequate transport and oxygen consumption. With ineffective hemodynamics, normal hemoglobin values are not indicative of normal oxygen consumption and oxygenation of tissues.

3). *Protease inhibitors.*

4). *Membrane stabilizers:* prednisolone 300 mg, vitamin C - 500 mg, Troxevasin 5 ml, Etamsylate Na - 250-500 mg, Essentiale - 10 ml, tocopherol 2 ml, Cyto-Mack - 35 mg.

5). *Stimulation of diuresis and prophylaxis of acute renal failure*: Rheogluman - 400 ml, mannitol, Lasix in split portions to 200 mg in case of oligoanuria, Euphilin IV 240 mg.

6). Actovegin - 10-20 ml IV.

7). Antihistamines.

8). *Disaggregation drugs*: Trental up to 1000 mg in case of eliminated source of bleeding.

9) *AV*. Indications for AV in hemorrhagic shock:

- blood loss more than 30 ml / kg;
- coagulopathic bleeding;
- arterial hypotension more than 30 minutes;
- subsequent operations to stop bleeding;
- combined with gestosis – continued AV with blood loss more than 15 ml / kg;
- combination with another type of shock (anaphylactic, cardiogenic, hemotransfusion, septic).

Methods of surgical treatment. The main task of surgical interventions in case of hemorrhagic shock is *to stop bleeding*. The most commonly performed operations are: bleeding vessel ligation, suture of the organ wounds, excision of bleeding ulcers of the stomach and duodenum, splenectomy, nephrectomy, and others.

In the postoperative period, the treatment of acute posthemorrhagic anemia, including hemotransfusion with the aim of replacement, prevention and treatment of possible complications are carried out before everything else.

Endotoxic shock

Endotoxic shock *is a sudden massive attack of bacterial toxins on the body of the patient.*

Etiology and pathogenesis. Endotoxic shock develops in surgical patients on the background of toxic or terminal phases of generalized purulent peritonitis, pleural empyema, large intraperitoneal or pulmonary abscesses, extensive purulent-inflammatory diseases of soft tissues as a result of the influence of endotoxins and toxic products of the activity of pathogenic bacteria on the cell membranes, components of blood coagulation and complement system. All this leads to increased coagulation, cell damage and blood flow disorders, especially at the microcirculatory level.

The distinguishing characteristic of the pathogenesis of endotoxic shock is that blood circulation disturbances caused by bacterial toxins lead to the opening of arteriovenous shunts, and the blood avoids the capillary bed, going from arterioles to venules. Cell nutrition is disturbed by the reduction of capillary blood flow and the action of bacterial toxins directly on the cell, decreasing oxygen supply of cells.

Inadequate blood flow at the capillary level during shock leads to the changes in the metabolism of all organs and systems, what is manifested by the impairment of the function of the heart, lungs, liver, kidneys, nervous system. The degree of the insufficient organ function depends on the severity of the shock, and this determines its outcome.

Developed circulatory embarrassment, primarily microcirculation disturbances, leads to the ischemia of the liver and disruption of its functions, what increases hypoxia in severe stages of shock. Detoxification, protein-forming, glycogen-forming and other functions of the liver are disturbed. Disturbances of the main and regional blood flow, microcirculation in the kidneys cause violations of both filtration and concentration functions of kidneys with the development of oliguria, up to anuria.

This leads to the accumulation of nitrogenous waste in the body – urea, creatinine and other toxic products of metabolism.

Violations of microcirculation and hypoxia cause functional disorders of the adrenal cortex and decrease the synthesis of corticosteroids (glucocorticoids, mineralocorticoids, androgenic hormones), what aggravates impaired circulation and metabolism.

Disturbed blood circulation in the lungs causes violation of external respiration, decrease of alveolar metabolism, blood shunting, microthromboses, resulting in respiratory failure, what increases tissue hypoxia.

In considering the pathogenesis of endotoxic shock, it should be noted, that the complement system consists of at least 20 different self-propagating proteins and can be activated by at least any of two trigger factors. During activation, previously synthesized biologically active proteins are converted into humoral mediators of inflammation and tissue alteration. Activation of the complement occurs gradually, by an example of the blood coagulation cascade. The formation of a full complement leads to the lysis of the cell membranes of bacteria, erythrocytes and other tissues. Released during the activation of the complement, fragments of peptides activate other cellular and humoral effector systems.

Recent literature reports also show that endogenous cytokines, the main targets of which are white blood cells, endothelium and heart, are released under the influence of endotoxins and other bacterial products. Emerging inflammatory mediators and endogenous cytokines themselves affect the vasomotor tone, permeability of small vessels and aggregation of leukocytes and platelets. A reorganization in the terminal portion of the circulatory system occurs. As a result, there is loss of tonus of the vessels of resistance (arterial) and volume (venous). Blood can accumulate in the capillary bed, and plasma proteins transude into interstitial fluid. Stagnation of the circulation is observed in the venous system. As a result of the stimulation of b-receptors, arteriovenous shunts of the terminal portion of the blood flow are opened.

Meanwhile a significant importance in the pathogenesis of endotoxic shock is also given to the formation of nitrates in the body. During the inflammatory reaction in the body, macrophages play a key role in the formation of nitrates. Specific macrophage enzyme – NO synthase (macrophagal, which is localized in macrophages, myocardium and smooth muscles) converts arginine into nitric oxide (NO), from which nitrites and nitrates can later be formed. The main function of NO, which is synthesized by macrophages, is to provide their cytotoxic action. During the activation by bacterial endotoxins or T-lymphocytes, macrophages enhance the synthesis of NO synthase, which converts arginine into NO. Released from macrophages, NO quickly penetrates into bacteria and the cell dies. Thus, NO plays an important role in the immune protection of the body. In addition, NO helps to reduce the activity of border inflammatory cells, inhibits platelet aggregation and improves local blood circulation.

Pathogenic influence of the NO formation in the body during inflammation may be as follows. During inflammatory processes in the body active forms of oxygen, which are one of the important molecular targets for NO, can be formed. NO binds to oxygen, forming peroxynitrites, which many times predominate NO by the toxicity. Exactly they play an important role in many pathophysiological processes, including septic shock, as well as ischemic and ulcerative injuries of organs. Peroxynitrite causes damage to proteins and lipids of cell membranes, damages the vascular endothelium, increases platelet aggregation, participates in endotoxemia. NO itself, accumulated excessively in the cell, can cause DNA damage and have anti-inflammatory effect during endotoxic shock.

Table 4

Pathogenesis of endotoxic shock

Gram-negative microorganisms	Endotoxin	Tumor necrosis factor	Grampositive microorganisms
Pathologic lipopolysaccharide		Activation of polymorphic-nuclear leukocytes	
Activation of Factor XII		Enhancement of adhesion of endothelial cells	

	Formation of capillary thrombs	Synthesis of macrophage isoforms of NO synthase on the action of an infective agent with synthesis in the NO cell		
	Enhanced vascular permeability	Increased release of O_2 radicals	NO binds to active forms of O_2, forming peroxynitrites	Myocardial depressant factor
Depletion of the blood clotting system		Release of enzymes		
DIC syndrome	Blood extravasation	Activation of phospholipase of the cell membrane		
Hemorrhage and bleeding		Increase of arachidonic acid synthesis		Reduced emptying capacity and myocardial hypoxia
		Increase of PGE content; IL-1; IL-2		
		Bronchospasm		
	Acute respiratory failure		Centralization of blood circulation	
				Arterial hypotension
	Acute renal failure			
			Adhesion of platelets	
DEATH	Violation of tissue metabolism		Reduced peripheral resistance	Tissue hypoxia

Thus, at the initial stage of the development of the disease under the influence of endotoxins, primarily, the dilation of the walls of small vessels (mainly venules) occurs, and permeability of the vascular wall also significantly increases. As a result of the above, despite the absence of the absolute volume deficiency, venous return to the heart decreases (relative hypovolemia). In response, reflexory sympathetic phlebostenosis occurs. But the active phlebostenosis effectively reduces venous blood filling only in the case if the veins are well filled and stretched. If transmural pressure is low enough to bring the veins into the half-filled condition, even strong contractions of the smooth muscles of the veins slightly influence the amount of blood in them. In such situation, the narrowing of the veins may even slightly increase the local vein capacity, since it makes the wall more rigid, what results in a lumen becoming larger and more circular, despite the fact that the lumen of the circumference decreases. As a result of the decreased venous return, the activity of the sympathetic nervous system increases, what, in addition to the direct influence of endotoxins, leads to the constriction of pre- and postcapillary sphincters (stimulation of α-receptors). As a result, blood supply to tissues becomes insufficient, cardiac minute volume in this phase is usually normal or even increased (that is, CMV is normal or increased, HR increases, total peripheral resistance decreases and BP decreases). The arteriovenous oxygen difference and oxygen delivery to the periphery are decreased.

As the shock develops, *a vicious circle is formed.*

Precapillary arterial sphincters are more sensitive to the toxic effects (including acidosis), so their spasm quickly changes into paresis. Postcapillary (venular) sphincters are more resistant to metabolic disturbances and remain in the condition of tonic tension for a long time. Thus, the blood flowing to the capillary bed, is stagnated, and therefore tissue hypoxia increases, metabolic acidosis increases, plasma extravasation with increasing compression of capillaries occurs, what, together with the stagnation of blood in the venous bed, contributes to further reduction of venous return and an increase of *relative hypovolemia.*

As a result, the following cause-and-effect relationship occurs: stasis in the capillaries – visceral stagnation – water release – increased blood viscosity – aggregation of red and white blood cells, formation of red and white blood thrombi – depletion of coagulation factors and platelets as a result of disseminated intravascular coagulation – appearance of debilitating coagulopathy with a high tendency to hemorrhage.

In the affected areas, aerobic energy pathways change to anaerobic glycolysis. The change of oxygen metabolism to the glycolytic pathway significantly increases glucose consumption with simultaneous reduction in ATP release. This leads to the decrease of glucose level again. Biosynthesis of protein during shock is limited. This influences very quickly on the synthesis of those proteins, which have a short half-life, for example, coagulation factors. Thus, a disorder of blood coagulation increases even more.

During the shock, the release of potassium from cells starts. Metabolic acidosis occurs as a result of an increased production of lactate, as well as pyruvate, α-ketoglutarate and ketone bodies. Acidosis is partially compensated by increased breathing. As a result of an increased concentration of H + in the plasma, the following occurs: negative inotropic effect on the heart; decrease of the sensitivity of precapillary sphincters at the level of vasomotor reactions with the formation of edema; increase of catecholamine release; activation of the coagulation system as one of the causes of disseminated intravascular coagulation. Some substances that are formed during shock (including cytokines themselves), have a negative inotropic effect. Long-lasting action of these factors leads to cardiectasis and heart failure, thereby to the reduction of minute cardiac output.

RES is especially sensitive to the lack of oxygen. Toxic substances, for example, endotoxins of the intestine, stop being trapped by RES (mainly of liver) and with blood flow get into the heart and lungs. This mechanism is considered one of the main ones, which contributes to the change of the shock to the irreversible phase partly due to the development of irreversible collapse of peripheral vessels.

Table 5

Interrelation between macro- and microcirculation during shock

Reduction of blood volume, acute heart failure, disturbances of peripheral vessels			
	Coagulation		
Shockogenic mediators	Enhanced permeability	Edema, necrosis, release of enzymes	Reduced venous reflux
	Viscosity increase		
Injuiry of shockogenic organs	Vascular reactions	Severe coagulopathy	Low cardiac minute output
Lactic acidosis		Hypoxia	
Metabolic acidosis			Hypotension

Kidneys. Due to the efferent vessel contraction during shock, the pressure of glomerular filtration decreases, resulting in the development of oliguria (4-20 ml/g) or anuria (4 ml/g). The narrowing of renal vessels remains for a long time after normalization of blood pressure. Ischemia causes progressive tubular necrosis due to the glomerular and later tubular insufficiency with the formation of cylinders in the distal tubules. The sign of renal insufficiency is an increase of such compounds as urea and creatinine in blood, which are usually excreted in the urine.

Thus, as the shock develops, a vicious circle is formed, which leads to the following: stasis in the capillaries – visceral stagnation – water release – increased blood viscosity – aggregation of red and white blood cells, formation of red and white blood clots – depletion of coagulation factors and platelets as a result of disseminated intravascular coagulation – appearance of severe coagulopathy with a high predisposition to bleeding.

Thus, one of the main pathophysiological mechanisms in the development of endotoxic shock in case of purulent surgical diseases is the development of hypovolemia due to the sequestration of blood in the microcirculatory bloodstream and its discharge into the tissues due to the increased capillary permeability.

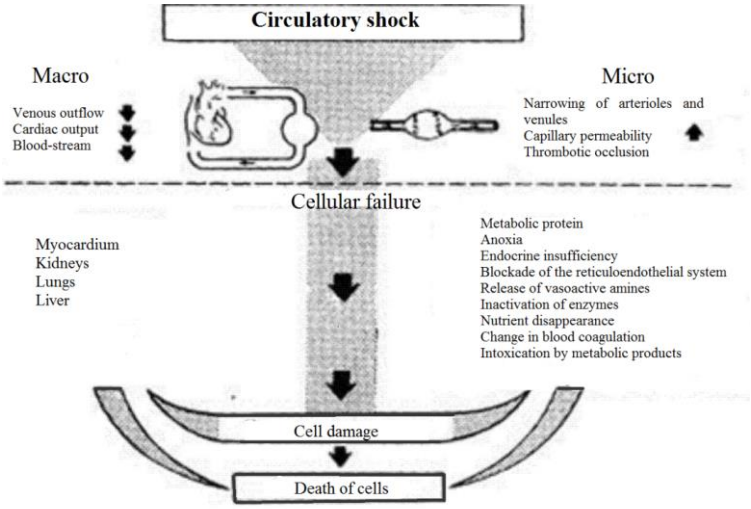


Fig. 29. Hemodynamic disorder and its association with general disorders during endotoxic shock

Clinical presentation of endotoxic shock develops against a background of increasing intoxication. The patient has a chilly sensation, followed by a sharp rise of temperature, often accompanied by nausea, vomiting, diarrhea and exhaustion.

Clinically, there are groups of symptoms that determine a degree of severity and prognosis.

Among the *early signs* of endotoxic shock there are hyperventilation, which causes respiratory alkalosis, and cerebral disturbances in the form of anxiety or total arrest. These first symptoms of shock do not often receive attention, what leads to delayed diagnosis and worsens the prognosis. As the disease develops, tachycardia, shortness of breath, arterial hypotension, and sometimes a tendency to hypertension increase, a pallor of extremities with acrocyanosis is observed. Skin is warm and dry ("*warm shock*").

During investigations in this period the following is observed: GPVR decrease and early signs of reduced ejection fraction (becomes normal on the 7-10th day), increase of cardiac output, heart rate, respiratory rate and PO_2 of mixed venous blood (due to arteriovenous discharge) may appear.

Table 6

Main signs of endotoxic shock

System	Pathology
CNS	Encephalopathy
GIT, liver	Gastric erosion Cholestatic jaundice
Blood	Early neutropenia, later neutrophilia DIC, especially in case of gram-negative bacteremia Thrombocytopenia in 50%
Kidneys	Proteinuria Focal proliferative glomerulonephritis Acute tubular necrosis
Metabolism	Hyper- or hypoglycemia Skeletal muscle proteolysis Hypertriglyceridemia Lactic acidosis
Respiratory organs	Respiratory alkalosis Increase of alveolar-arterial gradient PO_2 Respiratory muscle failure.

As the shock progresses, arterial hypotension develops and oliguria grows. During examination these patients show low values of central venous pressure (CVP), decreased circulating blood volume (CBV) and decreased cardiac output; increased general peripheral vascular resistance (GPVR), alveolar-arterial gradient PO_2, decrease of pH and PO_2 in arterial blood (i.e., hypodynamic reaction of systemic circulation) are observed; oliguria and lactic acidemia increase. Multiple organ failure occurs (acute heart and renal failure, respiratory distress syndrome, liver failure, DIC syndrome).

ECG may show changes, which are sometimes difficult to distinguish from those occurring during acute myocardial infarction. The prognosis is relatively favorable if proper therapy is started quickly.

As the shock develops, further decrease in blood pressure is observed, the skin color is gray, the limbs are cold. During investigation, increased parameters of CVP, significant metabolic acidosis and a very high concentration of lactic acid are observed. The prognosis is very unfavorable.

At a later stage, arterial pressure drops to 50-20 mm Hg and below, and often not is not determined. Some patients do not show temperature reaction and even hypothermia is observed. Patients have low CVP values, decreased CBV, cardiac output and severe lactic acidemia, what does not compensate respiratory alkalosis. Blood electrolytes are changing, tendency to hyponatremia and hypokalemia are observed. Changes in the ECG can be observed, which are sometimes difficult to distinguish from those registered in case of acute myocardial infarction. Coma occurs. Mortality with such manifestations reaches 100%.

Causes of death: multiple organ failure (50%), persistent arterial hypotension (40%), severe heart failure (10%).

Clinical presentation. A patient *presents* with chills, followed by a sharp rise in temperature, often nausea, vomiting, diarrhea, shortness of breath.

The history of the disease depends on the causes of intoxication. The course of the disease is progressive.

Clinician-observed data. General condition is usually heavy. The patient is adynamic, the level of consciousness is exhaustion. A pallor of extremities with acrocyanosis is observed. Skin is warm and dry ("warm shock"). Cardiovascular system manifestations: as the disease develops, tachycardia, arterial hypotension, and sometimes a tendency to hypertension increase. As the shock progresses, arterial hypotension increases. Low CVP values, decreased CBV, decreased cardiac output. Respiratory organs – shortness of breath, hyperventilation, causing respiratory alkalosis. The organs of the abdominal cavity – without characteristic features, except for the situation when the endotoxic shock is caused by peritonitis.

Status localis. Depends on the type of pathology in one or another system.

In accordance with the standard schemes, **the plan for *laboratory-instrumental examination*** of a patient with endotoxic shock includes:

1. Clinical blood test.
2. Clinical urine test.
3. Biochemical analysis of blood.
4. Coagulogram.
5. Blood electrolytes.
6. ECG.
7. Other instrumental methods depending on the type of pathology.

Characteristic pathological changes are manifested by pronounced changes in homeostasis parameters (decreased hemoglobin, a significant increase of leukocytosis and ESR, albuminuria, low total protein, increased urea and creatinine in blood, alkaline phosphatase, hypocoagulation, immunodeficiency, decreased CBV, etc.).

Differential diagnosis is made among the diseases in which endotoxic shock is possible.

Treatment of the patients with endotoxic shock should begin with the main surgical measure – removal or sanitation of a primary focus of infection, which led to the development of shock.

The most frequent ***surgical interventions*** are the following: elimination of the cause of generalized purulent peritonitis (appendectomy, cholecystectomy, excision of perforated ulcer,

resection of a necrotized region of the intestine with strangulated hernia, acute intestinal obstruction, etc.) with careful sanation and adequate drainage of the abdominal cavity and intestinal intubation, administration of guided laparostomy according to indications; lung resection with an abscess that does not respond to conservative therapy; excision of abscesses of soft tissues (including acute purulent mastitis, acute paraproctitis, etc.) with through drainage of a sutured wound; dissection and drainage of phlegmons of soft tissues (including phlegmons of retroperitoneal space, acute purulent paranephritis, etc.) and other surgical interventions.

The basis of rational treatment in case of endotoxic shock is the meticulous observation of a patient. Continuous recording of clinical data is quite useful. At the patient bedside, it is especially important to track some basic readings.

1. The state of the pulmonary blood flow (and preferably of the function of the left ventricle) is controlled using the Swan-Gants catheter. In case of its absence, it is necessary to measure central venous pressure (CVP). The introduction of a catheter into large veins or the right atrium allows to obtain accurate data on the relation between the condition of the right ventricle and the circulating blood volume, what makes it possible to regulate the volume of the introduced fluid. CVP above 140-160 cm H2O indicates some danger of the prolongated introduction of fluids and the threat of a sudden pulmonary edema.

2. Pulse pressure allows to estimate the stroke volume of the heart.

3. The narrowing of the skin vessels indicates the resistance of peripheral vessels, although it does not fully reflect the disruption of the blood flow in the kidneys, brain or intestines.

4. An hourly measurement of the volume of the excreted urine allows to control the level of blood flow in the internal organs and the degree of their perfusion. Usually this requires an introduction of a permanent urinary catheter.

Pathogenetically grounded ***conservative therapy***. Mandatory therapeutic measure for this category of patients is bed rest compliance (considering poor venous return, which may increase in a plantigrade position).

Infusion therapy is very important in the management of patients with endotoxic shock, along with active *antibiotic therapy*. Infusion therapy should be performed strictly in a differentiated way taking into account specific indications and under constant control of the general condition, level of hemodynamics, diuresis, indicators of CVP, as well as objective data on the lungs. Usually in patients with acute cardiovascular insufficiency, infusion is started with polyglucin, after a test for reactogenicity: during the first minute, the volume restoring solution (substitute) is introduced at a rate of 10-15 drops / min, then a 3-minute break is made to assess the general condition of the patient; 2nd minute – 20-30 drops / min, then 3 min break – evaluation of a patient's general condition. If the condition does not grow worse, IV drip infusion of the drug at the required rate is done, but not more than 60-80 drops per minute. Polyglucin is administered if BP level is less than 100 mm Hg. If the pressure is 100-110 mm Hg, it is possible to introduce reopolyglucin, which has not only hemodynamic and detoxification effect, but also improves microcirculation, eliminates the stasis in the capillaries, reduces adhesion and aggregation of platelets, which underlies its antithrombotic action. Typically, 400 ml of polyglucin and 400-600 ml of reopolyglucin are administered. If the effect is insufficient, plasma, albumin are used. In the absence of the above mentioned drugs 200 ml of 0.9% NaCl solution or Ringer's solution with lactate are administered IV for 5 minutes. In the absence of the effect 1-1,5 liters of solutions are injected additionally for 20 minutes. If symptoms of shock persist, invasive hemodynamic monitoring and injection of 2-4 liters of infusion solutions for 1 hour are indicated. In case of severe shock, as well as pulmonary edema, vasosuppressors are indicated.

Vasopressors. Dopamine – 5-20 μg/kg/min IV; in the absence of effect, norepinephrine 0.5-30 μg/min IV is added, reducing the dose of dopamine to the "renal" one (2-4 μg/kg/min) as possible.

Inotropic Agents. In case of low cardiac output, before dopamine or norepinephrine infusion, *dobutamine* 5-20 μg/kg/min is added.

Adverse reactions of the above drugs include ectopic rhythm disturbances, nausea and vomiting and sometimes tachycardia. They are usually negated when the dose is reduced.

The efficacy of corticosteroids for the resuscitation of patients from endotoxic shock has not been currently proven, although they may have some possible positive effect on biological membranes. In particular, there are references in the literature that the production of NO may slow down or disappear under the influence of glucocorticosteroids.

For hemostasis correction (including control of DIC syndrome) *heparin* is recommended in a daily dose of about 20,000 units, which is also an inhibitor of biologically active substances involved in inflammation.

During endotoxic shock *antibacterial therapy* should be carried out under the short program – single and daily doses of antibacterial drugs should be at least 2 times reduced. In case of concomitant diseases of the cardiovascular system bacteriostatic antibiotics may be administered.

The administration of *Curantyl* or *Isoptin (Finnoptin)* as a means of myocardial protection from hypoxia may be effective. Quite effective may be *Complamin* and *Trental*. For the patients who, in spite of elevated CVP or pressure in the lung vessels, have hypotension, *cardiac glycosides (Corglycone, strophanthin, digoxin or Izolanid*) may help if dobutamine is absent. The introduction of the drugs that have a positive effect on the *venous tone* (water-soluble camphor – *sulfocamacoquine, Cordiamin*, etc.) is also indicated. It is important to maintain uropoiesis in order to prevent renal tubular necrosis. After circulating blood volume is restored in case of stabilization of blood pressure, *diuretics* – 20-60 mg of *Lasix*, *Buphenox* or 200 ml of 10% *Mannitol* (in case of cardiac failure) are added if necessary.

In many patients with endotoxic shock, PO_2 of arterial blood is drastically reduced. In this regard, it is important to provide free breathing and oxygen supply for them through the nasal catheter, mask or tracheostoma from the beginning.

In case of normal or slightly reduced BP, but severe general intoxication and hyperthermia, infusion therapy is mostly detoxificative with administration of *Neohaemodes* (200-400 ml for 2 days). In case of major inflammatory process in the lungs (lung damage, 3 segments), the administration of *nonsteroidal anti-inflammatory drugs (Indometacin, Voltaren* 100-125 mg / day) is recommended.

Polytrauma

The problem of polytrauma is highlighted on the pages of periodical medical editions and various forums for several decades and the interest to it does not fade.

It is conditioned by an increase of the number of multiple and combined injuries, lack of effectiveness of preventive measures, disabilities, high levels of mortality. Due to the complexity of the problem, there are various methodological approaches to the organization of medical care to the patients.

The most adequate *definition of the concept "polytrauma"* is the one, proposed by Yu.G. Shaposhnikov et al. (1990) – ***"The presence of two or more damaged zones in one or more anatomical areas, besides one of the injuries or their combination poses a threat for the life and health of a patient and requires immediate actions of qualified or specialized medical care."***

Over the past decade, polytrauma has become one of the leading problems with the formation of a new branch of modern clinical medicine – ***surgery of severe injuries.***

This type of injuries is accompanied by intensive disorders of homeostasis systems, which determine the severity of the clinical course of a traumatic disorder and which preconditions high levels of disability and mortality.

Unsatisfactory results of polytrauma treatment are often associated with insufficient presentations of the pathogenesis of a traumatic disorder in this category of sufferers, what leads to the mistakes in determination of the scope, terms and reasonability of surgical interventions, sequence of reanimation measures, intensive and infusion-transfusion therapy.

Social and economic significance of the trauma for modern society is very high. In the economically developed countries, injuries and accidents take the third place among the causes of mortality and the second among the causes of disability and morbidity of the population with permanent and temporary incapacity for work. D.D. Tranky (1983) indicates that physical injuries are the main cause of the death in Americans between the ages of 1 and 38. So, at present in the United States 165 thousand deaths from injuries and twice as many cases of disabilities have been registered.

From all types of traumatism – home, industrial, sports, etc. – road traffic incidents result in the largest number of deaths and cause the most severe injuries.

The society does not remain indifferent to this "trauma epidemic", due to social-preventive measures, the number of injuries per 100 thousand of population does not increase, despite a sharp rise in the amount of vehicles. However, the severity of trauma increases.

Severe mechanical injuries among the causes of mortality are inferior only to tumors and cardio-vascular diseases, especially in persons younger than 45 years old. For the last 20-25 years, the structure of injuries has qualitatively changed as a result of the industrial development, car and train accident injuries, falls from a height have become particularly more frequent. They are the main causes of severe polytrauma, and in 15-40% of cases lead to the fatal outcome and in 12-15% – to the permanent disability.

High mortality in case of polytrauma is associated with the severity of damage of internal organs, severe open fractures and extremity avulsions, as well as with frequent – more than in half of the patients – early and late complications of trauma (traumatic shock, pneumonia, fat embolism, thromboembolic complications, sepsis, etc.)

The patients with polytrauma require long-term treatment, which is attributed to the multiplicity of skeletal injuries, enforced change in the treatment methods of each of them, as well as frequent local complications during the course of treatment.

Classification of polytrauma. *All types of multiple and combined injuries are divided into two major groups according to etiology: road traffic injuries and injuries due to falls from height.*

Road traffic polytrauma is observed predominantly in men (72,2%) of working age from 21 to 50 years old (71,2%). In a road traffic accident *according to the mechanism of injury*, 3 groups of patients are identified: 1) "pedestrians" hit by vehicles – 61.1%; 2) "drivers" and car passengers, involved in an accident – 30,4%; 3) "motorcyclists" and bikers, involved in a crash – 8.5%.

The people, who work for construction and repair of building walls, move carelessly during window cleaning, work on the balcony may suffer *in case of fall from height ("catatrauma").* Catatraumas differ with particular severity from the injuries received in a road accident, since there is almost always a combination of moderate and severe skeletal injuries with the damage of internal organs: rupture of parenchymal and hollow organs, mesenteric avulsion, injuries of internal organs with bone fragments.

Classification of polytrauma *according to the localization of damage* is necessary for the correct choice of method and the treatment stage, in the sequence of time it includes the following types of polytrauma.

Isolated trauma (another name – monotrauma) is a trauma of one internal organ within the boundaries of one cavity, trauma of one anatomical-functional formation of the musculoskeletal system. In case of magistral nerves or vessels of the extremities, the damage to a vessel or a nerve in one anatomical region without the damage to the organs of support and movement can be called their injury.

Multiple trauma is a damage to two and more internal organs in one cavity, injuries within two or more anatomical-functional formations of the musculoskeletal system.

Combined trauma is an injury of the internal organs in different regions, combined injury of the organs of the musculoskeletal system and major vessels and nerves. From the anatomical point of view, the ribs, as well as skeletal bones and skull base, should be considered the elements of the locomotor system. However, for practical surgery, the trauma of these formations is an inseparable part of the trauma of the organs contained in them. Therefore, fractures of the ribs, bones of the skull vault and the skull base, damage of the abdominal wall can be conventionally attributed to the category of internal organs trauma.

Multisystem trauma is the simultaneous presence of two and more etiologically diverse injuries in a patient.

Polytrauma is a combined concept, which includes multiple and multisystem injuries, having many similar features in etiology, clinical findings and treatment. The term "polytrauma" has become fully integrated in the work of medicine and ambulance staff, admission departments and resuscitation

units of hospitals. By analogy with a term "shock" it is a signal of severity, the reason for the initiation of emergency diagnostic and therapeutic measures.

Taking into account high risk of traumatism, the study of a traumatic disease has been established.

"***Traumatic disease*** *is a disturbance of the vital activity of an organism, resulting from the damage caused by excessive mechanical actions, which is an advanced complex of its functional disturbances, different in its various periods, and a collection of adaptive (adjustment) responses focused on the preservation of the life of an organism and restoration of functions and structures "(S.O. Seleznyov and G.S. Khudaiberenov, 1984).*

There are several periods of traumatic disorder (Table 7).

Table 7

Periods of traumatic disease and pathological processes, the most typical for them with its uncomplicated development

Periods	Pathological processes that appear at different stages of traumatic disease
1. Acute reaction to trauma (up to 2 days)	Acute blood loss, shock, toxicosis, direct damage of organs (primary), injury of the musculoskeletal system. fat embolism
II. Early manifestations (up to 14 days)	Acute disorders of the functions and systems: CNS, respiration, circulation, water-electrolyte balance, development of hepatic and renal failure.
III. Late manifestations (more than 14 days)	Development of dystrophic and sclerotic processes. Slowing down of the consolidation of fractures. Formation of false joints.
IV. Rehabilitation period	Partial or complete restoration of functions and structures of the organism

In-patients with injuries have the traumas of limbs most frequently (41.3%), after that the traumas of the head (32.5%) and chest (12.5%, of them the closed ones – 9.9%, wounds – 3.4%). They are the most severe and dangerous, as they cause violation of the functions of the heart and lungs.

In case of severe chest trauma in almost 30% of cases multiple bilateral bone fractures with the formation of a flail chest flap (unstable chest wall) are diagnosed, what complicates the lung ventilation greatly.

Thus, in case of polytrauma the lungs, heart, diaphragm and major respiratory tract are affected most of all, injuries of the esophagus, major intrathoracic vessels and thoracic ducts are rare.

Special aspects of the pathogenesis of polytrauma. In case of damage of any severity and localization a violation of the anatomical integrity of tissues or organs occurs, which result in the disturbances of their function. The extent and intensity of the functional pathophysiological disturbances directly depends on the anatomical severity of the injury, in other words, on the volume of damaged tissues, as well as localization of damage. In case of brain injury, for example, vital disturbances occur with a small (but determined!) area of damage, and in case of the trauma of locomotor organs – only with large in extent damage of soft tissues and bones.

From the moment of the influence of strong mechanical force leading to the rejection and death of tissue masses, such pathological processes as an excessive flow of algesic and vegetative impulsation, massive loss of blood from the damaged vessels of different size, time of preservation of disturbances of tissue perfusion, particularly manifested in the region of injury, and aggravated autointoxication, associated with this, appear and increase fast in the first period after the trauma.

Protective mechanisms during the polytrauma, especially severe combined bone and internal organs damages, are less perfect. This phenomenon accounts for more severe, "malignant" course of a series of polytraumas, than in the case of prognosed simple summarization of each separate injury. This effect, similar to the mutual action enhancement of some drugs during their combined administration, is well known as the "phenomenon of mutual load".

Specific intensity of disturbances of many physiological processes in case of severe polytrauma leads to the development of numerous general and local complications characteristic for this type of pathology.

Development and exacerbation of traumatic shock, change of hemostasis to hypocoagulation and, as a consequence, prolongation of bleeding, acute renal failure, fat embolia, pneumonia, thromboembolic and purulent-infectious complications leading up to sepsis, necrosis of tissues, regeneration disturbances – it is a hardly complete list of the problems that must be prognosed, should be prevented, and then must be efficiently treated.

The clinical picture of traumatic injuries of the internal organs of the thoracic and abdominal cavity in case of polytrauma is basically similar to the clinical picture of isolated injuries with some features, as indicated above.

Fundamentals of modern diagnosis and surgical management in case of polytrauma.

Diagnosis of polytrauma is started from the accident scene and is made in three stages:

1. *Investigatory, selective diagnosis* – detection of injuries and their consequences that threaten life at the moment and require resuscitation operations.

2. *Radical diagnosis* – determination of all possible injuries.

3. *Final diagnosis* – detection of the details of specific injuries, as well as detection of the injuries not detected earlier.

Diagnostic approach in case of polytrauma. *The structure of the diagnostic search in case of polytrauma* should be presented as follows:

1) initial determination of the character of APA injuries (law of "four cavities" priority);

2) detection of a dominant injury with an emphasis on the syndromes, which represent direct threat for life, determination of their influence on the severity of the patient's condition;

3) assessment of indications for resuscitation and emergency surgical intervention;

4) diagnosis of associated injuries, which aggravate the condition of a patient in the presence of a dominant injury.

Thus, diagnostic approach should be based on solving *general diagnostic problems* (detection of a dominant injury and the syndromes representing direct threat for life) and *individual issues* (detailed specification of competitive and associated injuries).

At the same time, universal tactical approach means following the principle of task priority in providing diagnosis, intensive care and surgical interventions according to top-down approach: from more severe and dangerous pathological processes and injuries to less dangerous.

For persons who have suffered from severe trauma, an examination should be made as soon as possible, but elaborately.

The examination begins from the *analysis of patient's complaints* (if he is bright-eyed).

General appearance of the injured, changes in the shape of the chest, level and character of abnormal movements, presence or absence of skin pallor, facial cyanosis, upper portion of the trunk, which can be seen at first view, already focus the physician on the injury of an internal organ.

During examination, a patient should be completely undressed, what is sometimes disregarded by physicians. The examination begins from the chest, going to the neck and the head. Then upper and lower limbs, pelvic ring bones are examined. The examination is finished with palpation, percussion and auscultation of the abdominal cavity. It is necessary to emphasize the importance of the examination of the patient's back, including the buttocks, and paying particular attention to any injuries of the spinal cord. These simple diagnostic techniques are important to determine pneumo-, hemo- and hemopneumothorax, hemorrhage into the mediastinum and pericardium, diaphragmatic injury and others.

Since the traumatism due to the road traffic accidents is of paramount importance by severity and the number of fatal cases, it is necessary to draw attention of physicians to the fact that, if an examination reveals a severe trauma of an intracranial organ, what requires urgent resuscitation measures, then the investigation of the circumstances of a road traffic accident, although they are extremely important, must be definitely obtained after urgent medical care delivery. If a patient's condition allows, it is desirable to obtain information about the circumstances of the injury from him, if it is not possible –from the witnesses of the accident.

The most important is the following information: which transport caused trauma and what part of the body was affected, average speed of transport, the position in which a patient was after injury, if

he was injured with a sharp or a blunt instrument, and which one exactly; if a patient fell after stroke, on which part of the body and on which item he fell down. Thus, on the basis of the mechanogenesis of trauma it is possible to characterize the extent of an injury in some degree, to evaluate its localization, to perform diagnosis of injuries and treatment in a targeted manner.

It is necessary to know the mechanogenesis of at least three most common types of traumatic accidents:

1) automobile-pedestrian accident;

2) injuries of drivers and passengers inside vehicles at the moment of their collision or crash with a stationary obstacle;

3) injuries of motorcyclists as a result of the collision with other vehicles.

In case of the collision of a vehicle and an adult pedestrian the hip, a region of the hip joint are the first to be injured. Due to the concussion of a human body there may occur a damage to the organs of the abdominal and chest cavities, but the greatest trauma of the cavity organs occurs in case of the vehicle collision when the organs are smashed by the car directly onto the place of their localization. Then, depending on the speed of the car and the position of a pedestrian, the latter falls down on the motor hood, hits himself on the iron parts what results in additional craniocerebral injury, or he is thrown aside or forward. At the moment of the blow on the road surface the injuries of the head, chest, upper extremities occur mainly.

In case of a truck-pedestrian accident, a trauma of different locatizations occurs – chest, lumbar region or upper abdomen. For this mechanogenesis severe wounds due to the great depth of a penetrating stroke are traditional. The most severe injuries are observed in case of vehicle running over a human body. A rupture with crushing of intrathoracic, intraperitoneal organs, multiple fractures of bones in the region of a wheel or caterpillar band rolling. As a rule, these victims die at the accident site or in the next hours and days after injury.

Both injuries of a driver and a passenger in the car in case of collision of vehicles with each other and rollover of a car have characteristic features. The force acting against the body of a driver and passengers at the moment of the collision tears them down from seats and throws forward on different parts of the frontal part of a car's cabin. A driver, due to the impact shock of the chest against a steering wheel, has fractures of the sternum, anterior parts of the ribs and various damages of the intrathoracic organs (injuries of the heart, lungs, aorta). At the same time, shinbones are injured. In passengers, the stroke of the head and trunk against the windshield and iron parts of the car causes craniocerebral trauma, trauma of the chest. Many years' experience in the treatment of the injured in traffic accidents, accumulated by V.F. Trubnikov and G.P. Istomin, showed that the most severe trauma of motorcyclists is craniocerebral one, but often the cause of a fatal outcome is a damage of the organs of the chest and abdominal cavity.

Thus, the knowledge of various variants of mechanogenesis of trauma occurence with morphological manifestations of injuries makes it possible to provide first aid to patients, make focused diagnosis and provide treatment.

Certain difficulties in case of polytrauma are caused by the *diagnostic formulation*, without which it is impossible to *arrange the injured persons into groups*, and which determines the choice of appropriate and effective emergency medical treatment (resuscitation and surgical) in this difficult and serious category of patients.

The diagnosis includes the following parts:

- dominant injuries, constituting a direct threat to life, which are fatal without treatment, and during treatment they are accompanied by mortality of more than 20%;
- less severe injuries – safe for life, but requiring inpatient treatment;
- other injuries, which require ambulatory treatment;
- complications of traumatic and nontraumatic genesis;
- associated diseases.

All patients with polytrauma are divided into four groups:

Group I – the injured in the terminal or extremely severe condition with the damage of two or more APA of heavy degree and severe polytrauma, the emergency care to whom is provided in an

intensive-care ward of the admission department and, depending on its results, further tactics is developed.

Group II – the injured with the damage of one APA, which are in severe or moderate severe condition, in combination with the damage of other region of moderate or heavy severity, requiring specialized care according to vital signs.

Group III – the patients in moderate severe condition with moderate or severe damages of two or more APA, requiring urgent professional and further specialized medical care.

Group IV – the patients in a relatively satisfactory condition of mild severity with the damage of one APA of moderate severity, in combination with the damage of other region of mild severity, or damages to several APAs of mild severity, requiring urgent professional qualified care.

Specific aspects of the surgical approach in case of polytrauma. *Surgical management of severe polytrauma and shock should be based on the principle of possible early operations in case of injuries of the organs of the chest, abdomen, retroperitoneum and skull. Operations in case of the musculoskeletal system injuries may be performed a little later.*

According to the nature of an injury, the time elapsed from the moment of a trauma, as well as the severity of a patient's condition, in some cases an operation is the beginning of resuscitation, in others – its continuation, and in third cases – its completion.

The choice of tactics depends on the urgency of operations.

Types of surgical interventions for polytrauma according to the degree and urgency categories.

The basis of the methodology of surgical treatment of polytrauma is formed on the one hand, by indications for surgical interventions, on the other hand – by the urgency of their performance, and also – by the state of the functions of the most important body systems, what is associated with the possibility of realization of the progressive concept of "surgical reanimation."

All surgical interventions are divided into:

1) primary, which are performed in case of damages of vital organs with the purpose of saving life, stabilization of basic functions of the organism, prevention of the development of severe complications and restoration of the functions of damaged organs and systems;

2) secondary, used in case of developed complications of polytrauma;

3) rehabilitation, representing a step-by-step recovery surgical treatment after resuscitation and stabilization of the condition of an injured person, as well as complex intensive care in the acute period of polytrauma.

An essential feature of surgical tactics in case of polytrauma is the demand of several surgical interventions for one and the same patient for a short period of time. Therefore, in each case it is advisable to set priority and order of such surgical interventions.

Primary surgical interventions according to the program purposes and urgency of performance are divided into three categories:

1) resuscitation;

2) urgent;

3) delayed.

Taking into account these circumstances, the following categories of urgency of surgical interventions in case of polytrauma are distinguished:

The first category of urgency includes surgical and resuscitation urgent operations in case of profuse bleeding on the background of the terminal state. In this situation, the mechanical component is eliminated by a resuscitation operation only, performed during the first 20 minutes from the moment the patient is brought to the operating theatre in combination with reanimation measures.

The second category of urgency includes operations performed during the first 30-60 minutes from the moment of admission with progressive internal bleeding on the background of unstable hemodynamic parameters after performance of main reanimation measures: catheterization of one or two central veins, correction of acidosis, intubation of the trachea with subsequent AV and introduction of a patient to general anesthesia.

The third category of urgency includes operations in case of extremity avulsion and crushing with temporarily stopped bleeding by a tourniquet, tamponade or tight dressing, as well as in case of

injuries of internal organs on the background of stabilized hemodynamics. Operations of this category are performed in 1-1.5 hours from the moment of a patient's admission to the clinic after catheterization of veins, high-volume stabilization of blood pressure with systolic index not lower than 90 mm Hg, as well as intubation of the trachea and AV.

The same group should include the operations which can be postponed for several hours after relative stabilization of vital functions of a patient's body and can be focused on the prevention of complications, creation of favorable conditions for healing wounds and maintenance of the functions of damaged organs.

According to the above mentioned classification the operations of the first category of urgency – resuscitation – are performed in clinic taking into account the phenomena of traumatic asphyxiation, acute massive progressive bleeding, tamponade in presence of a closed cardiac trauma and sudden cardiac arrest due to the circulatory disorders in the earliest terms regardless of the severity of a patient's condition, leading up to the state of clinical death.

These operations include:

1) drainage of the pleural cavity with hemopneumothorax;

2) mediastinotomy with tension mediastinal emphysema;

3) thoracotomy, which is performed in the absence of the effect of closed cardiac massage in two minutes after its arrest, with the damage of large mediastinal vessels with continued bleeding, as well as tamponade caused by a closed cardiac trauma;

4) laparotomy with continued massive intra-abdominal hemorrhage with injured parenchymal organs, great vessels of the abdominal cavity and retroperitoneal space.

Operations of the second category of urgency – urgent – are performed in case of injuries of vital organs and intensively developing disorders of the functions of the main life support systems. Such operations are performed in case of damage to the parenchymal and cavernous organs of the abdominal cavity, traumas of major vessels, open pneumothorax, crushing and avulsion of the extremities, as well as in case of progressive swelling of edema and dislocation as a result of compression of the brain or spinal cord.

Also, this group includes the operations performed for the patients with injuries that do not threaten life directly on admission: thoracotomy in case of stable collapse of the lungs, clotted hemothorax, operations for extraperitoneal damage of the intrapelvic organs; primary surgical treatment of open fractures, large and degloving tear-contused wounds of soft tissues of the trunk and extremities.

The third group includes delayed operations: primary surgical debridemen of the smaller surface area of a wound, osteosynthesis of closed fractures of the limb and pelvic bones, decompressive craniotomy. Indications for delayed surgical interventions are not set by a short-term threat to life, although refusal from them complicated the course of acute period of polytrauma.

Types of operations performed in case of polytrauma. Key elements of surgical treatment in case of polytrauma are characterized by high operational activity, which depends on the severity of injuries, localized in different APA, and increases with developing severity of damage in case of polytrauma.

Specific features of surgical management also include the demand to perform two or more operations for one patient. Often 2 or 3 surgical interventions are performed for one polytrauma patient.

Taking into account the demand to perform two or more surgical interventions for one polytrauma patient, the surgical approach, which provides performance of various types of surgical interventions, has been developed:

1) one-stage operations performed by one team of specialists;

2) stage-by-stage operations, as a rule, of different types, when in the course of one ongoing anesthetic-physiological therapy, without anesthesia recovery of a patient, after completion of the operation by one team, the next team of doctors performs surgery for other injuries (sometimes stage-by-stage operations are performed with a time gap as a patient's condition stabilizes – first resuscitation and only then urgent and delayed);

3) simultaneous operations, performed by several surgical teams at the same time and without hindering each other, which eliminate the consequences of trauma in different APA, mainly of the same type and

4) multistage operations of the palliative, resuscitation and reconstructive character.

Respecting polytrauma, the following simultaneous operations are distinguished:

a) absolute operations, refusal from which may lead to the development of a fatal outcome;

b) preventive operations, used to prevent the development of severe complications, which may later require a new surgical intervention;

c) diagnostic (explorative) operations performed in complex diagnostic situations, for example, to clarify a source of bleeding or damage to one of the vital organs of the chest or abdomen.

As a rule, only the same types of surgical interventions are performed simultaneously, what is substantiated by single indications.

In the absence of the conditions for the performance of simultaneous operations, it is necessary to perform different-type stage-by-stage surgical operations. Often, such operations are performed according to primary urgent and delayed indications, much more rarely resuscitation stage-by-stage operations are performed.

Thus, the proposed classification of surgical interventions, as well as the description of the types of polytrauma operations, allow to answer one of the most important questions concerning surgical tactics: same-type operations should be performed for indications simultaneously, and in the absence of them – on a stage-by-stage basis with the change of surgical teams without anesthesia recovery.

One-stage, but different-type surgical operations, should be also performed on a step-by-step basis, according to the compensation of vital functions and stabilization of the general condition of a patient.

In conclusion it is to be noted that at present time it is widely agreed that the treatment of such patients should be performed in specialized multifield hospitals, where it is possible to entirely support diagnostic and therapeutic processes.

Admission of a patient should be carried out not only in case of obvious signs of multiple and combined injuries. Difficulties during physical examination of a patient in the prehospital phase account for high percentage of undiagnosed injuries, reaching 12% even in the hospital phase. Therefore, hospitalization of the injured, who may have multiple, heavy and combined complications according to the mechanism of trauma, is quite logical.

The tasks of emergency care in the early prehospital phase are resuscitation correction of dysfunctions of vital organs, conduction of diagnostic measures within the minimum period of time to determine the dominant injury, priority time and volume of surgical interventions.

The volume and sequence of medical care in the hospital phase is determined by existing schemes taking into account the rational use of the "golden hour". There are difficulties in defining the concept of realization of the urgent operational guideline "according to vital indications". For example, a diagnosed hemoperitonium is subject to immediate surgical debridement, and a multifragmentary hip fracture in the upper third, the blood loss in case of which is up to 1500 ml, most often, is an object of delayed intervention.

The use of the "golden hour" is extremely important for surgical providing of the stability of the injuries of the locomotor system, especially such shockogenic localizations of fractures as the rib cage, hip, shin and pelvis. This allows to provide mobility of the injured, to reduce the number of local injuries maintaining the shock and prevents the development of many complications of local and general nature.

Catatrauma

Catatrauma (Ct) is a damage or injury resulting from fall from height.

Catatrauma is one of the most complex and difficult for diagnosis types of severe polytraumatic injuries. It occupies a special place in the structure of traumatism, what is specified, above all, by a large variety of mechanisms and circumstances of the emergence of numerous and different according to localization and severity morphofunctional injuries.

Currently, Ct takes the second place in the structure of trauma surgery, making up 40.0% of the all fatal injuries.

Difficulty in the diagnosis of this type of trauma is attributed to the fact that Ct differs greatly by the variety of fall types, mechanisms and circumstances of the occurrence of numerous and polymorphic injuries. Polymorphism of injuries, their severity and volume are determined by the height from which the fall occurs.

Most national and foreign authors note that in case of Ct severe damages almost always occur – severe skeletal trauma, damage to the internal organs of the abdominal cavity and chest, craniocerebral and spinal cord injuries.

In Ukraine, according to the statistics in 2015 about 40,000 people died as a result of injuries, and according to the medicolegal investigations 11-14 people from 100 injured die in case of Ct.

Classification of catatrauma and consequent injuries.

Understanding of the processes associated with the damage of tissues and organs in case of Ct is not possible without a scientific substantiation of the mechanism of trauma, depending on the specific case of falling and the way of land contact, as well as without unified formal terminology and Ct classification.

The injuries, occurring in case of Ct, are classified according to the time of their occurrence: – injuries at the initial stage of the fall – from the contact with the "separation surface" and from the impact of the objects which boost the acceleration to the body; – at the midline stage of the fall – from the contact with exposed objects (single, multiple contacts) and from the contact with the walls of a closed space; – at the final stage of the fall – during primary contact with the surface of touchdown: local (regional) and remote; in case of secondary contact with the surface of touchdown – also local (regional) and remote; during the body motion along the surface of touchdown – only local ones.

Depending on the height of the fall, the traumas are divided into injuries from falling on a flat area (from upright position) and injuries in case of a fall from height.

In general, all disturbances in the body in case of Ct are divided into local (direct effects of exposure to shock after impact) and remote deceleration (indirect action of impact in case of a sudden slowdown of the body after impact, what leads to a general concussion of the body).

In the first case fractures, contusions, injuries of tissues in the area of the body contact occur. If the amount of absorbed energy is sufficient, then there is an indirect shock action in the region distant from the point of an initial body contact. At the same time, the absorbed energy leads to deformation (bending, compression, twisting, extension, concussion) and hydraulic impact at different levels of structural organization of anatomical regions. Usually organs, vessels, mesentary, and epiploon are damaged.

Thus, a characteristic feature of traumatogenesis, which reveals biomechanical factors of the harmful action of Ct, is a multifactorial relationship, and at the heart of the obtained damages lies the effect of influence of absorbed energy, what causes many local and general disorders in the body of a patient with Ct.

In case of Ct the main rule of diagnosis is an application of the whole complex of diagnostic measures at the beginning of an examination for detection of possible injuries of the organs of the chest, abdomen and retroperitoneum, then of the skull, brain and spinal cord, and only after that the diagnosis of other injuries.

Clinical manifestations of Ct are characterized by high polymorphism and usually do not correspond to the nature and severity of the injuries, what is associated with the absence of pathognomonic symptoms in case of Ct and is caused by shock, the syndrome of comorbid aggravation, introduction of drugs at the prehospital stage and often alcohol intoxication. Difficulties in diagnosis are caused by polyfocal injuries, coma, cerebral compression, massive internal bleeding, large skeletal trauma, fatty embolism of the lung and brain vessels, what sharply restricts, and sometimes excludes the use of additional methods of examination of patients with Ct.

In case of Ct the organs of abdominal cavity and retroperitoneal space, including urinary system, are most often injured.

Due to the small informative value of clinical symptoms of the injuries of internal abdominal organs in case of Ct, priority should be given to instrumental diagnostics.

In terms of diagnostics, the following methods of investigation are used for Ct: X-ray, ultrasound, computer tomography, mini-invasive instrumental diagnosis – endoscopic, in particular, sanitation and diagnostic fibrobronchoscopy, laparocentesis and laparoscopy, angiography.

Specific features of surgical tactics in case of catatrauma. Depending on the nature of injuries in case of Ct and the demand for reanimation measures, the following types of interventions are performed: resuscitation, urgent and delayed. It should be emphasized that resuscitation laparotomy is made within the first 30 minutes after the patient's admission, focused on the elimination of profuse intraperitoneal bleeding and performed together with reanimation measures.

The designed surgical tactics can be represented in the following instructions:

1. In case of abdominal injury with simultaneous damage of hollow and parenchymal organs associated with acute massive blood loss, surgical interventions are included in the complex of resuscitation measures and are performed regardless of the severity of the condition of a patient with Ct.

2. In case of the injury of parenchymal organs, the priority should be given to the organ preserving surgeries, during which it is necessary to achieve stable hemostasis.

3. In case of the injuries of hollow organs, the volume of operations should be specified taking into account a number of risk factors: traumatic and hemorrhagic shock, acute massive blood loss, hypovolemia, endotoxicosis, as well as secondary immunodeficiency:

3.1. On admission within 6 hours from the moment of injury and in presence of no more than two of the above mentioned risk factors, one-stage application of enteroanastomosis with transnasal intubation of the small intestine should be performed.

3.2. After 6 hours from the moment of injury in presence of three or more risk factors, resection of the small intestine should be performed, which is supplemented by the formation of decompression enterostomy.

3.3. To form the anastamoses of descending colon in case of Ct with their extraperitonization and with intubation of the descending colon is recommended not later than 6 hours after the moment of trauma in presence of NO more than two risk factors. In other cases, they should be combined with colostomy.

3.4. In all cases of suture of ruptures of hollow organs additional sealing of the suture lines by epiploon or the mesenterium of the adjacent organs should be performed.

3.5. Use of minimally invasive endolaparoscopic interventions in case of catatrauma with injuries of the abdominal cavity organs.

3.6. Use of minimally invasive X-ray endovascular methods of hemostasis.

Thus, according to the literature on the problem of surgical treatment in case of Ct, there is no universal tactics. Some surgeons, being ardent supporters of active surgical intervention and basing on the practical experience prove their views with reason, others, on the contrary, following the expectant tactics also prove the correctness of their approaches. Therefore, there is probably a golden mean in the solution of this problem, which involves an individual approach in each particular case, at the same time not diminishing the benefits and neglecting the disadvantages of both active and expectant tactics of management of this category of patients. Moreover, in recent years, the latest surgical techniques are widely used both in diagnosis and in the treatment of this category of patients.

CHAPTER II. ACUTE SURGICAL DISEASES OF THE DIGESTIVE SYSTEM, PERITONEUM AND PERITONEAL CAVITY

II.1. CURRENT METHODS OF EXAMINATION IN SURGICAL DISEASES OF STOMACH AND INTESTINE

Different diseases of the gastrointestinal tract are among the most common diseases of the internal organs.

At present, up to 5% of the adult population suffer from peptic ulcer. The peak of the disease is observed at the age of 40-60 years; in urban dwellers morbidity is higher than in rural population; in

men, peptic ulcer develops more often and predominantly under the age of 50; duodenal ulcers predominate over gastric ones in a proportion of 3:1 (10:1 at young age). Relapses are observed in approximately 60% of patients during the first year after duodenal ulcer healing and in 80-90% during the first two years after healing. Mortality is due to bleeding, which is observed in 20-25% of patients, and perforation of gastric or duodenal wall with the development of peritonitis. Mortality in gastric wall perforation is 3 times higher than in duodenal wall perforation.

Many years of experience in the treatment of people with gastrointestinal diseases and the above-mentioned statistical data indicate that in some cases healthcare practitioners do not possess sufficient knowledge concerning the diagnostic features of many gastrointestinal diseases (functional disorders of the motor and secretory function of the stomach, symptomatic gastroduodenal ulcers, etc.).

The past decade was characterized by significant progress in gastroenterology. Pathogenesis of the stomach and duodenum has been studies in detail; various highly informative methods of investigation (X-ray, endoscopic, ultrasound) have been widely used in clinical practice, new methods of diagnosis have been elaborated and introduced into practice.

Functional and instrumental methods of examination in surgical diseases of the stomach

Current methods of examination of gastric secretion

Hydrochloric acid is released by parietal (delomorphous) cells of the body and fundal department of the stomach. Basolateral membrane of these cells contains histamine (H_2) and acetylcholine (M_3) receptors, whose excitation stimulates the formation of hydrochloric acid.

Gastric secretion (including hydrochloric acid) is regulated by central and peripheral mechanisms. The central mechanism of gastric secretion stimulation involves activation of the vagus with the release of the acetylcholine mediator. The latter, binds with the M_3 receptors of parietal cells and stimulates the production of hydrochloric acid. There is also an indirect acetylcholine stimulation of acid production, mediated by preganglionic vaginal activation involving M_1 receptors. Activation of *n. vagus* also promotes the secretion of gastrin and histamine in the mucous membrane of the antral department of the stomach.

Local mechanisms of gastric secretion stimulation act as follows: in response to food intake, leading to increased alkalinity of the contents of the stomach and the appearance of peptides, G-cells of the antral department produce hormone gastrin. The latter, acting on parietal cells, promotes the release of free calcium ions from the intracellular depot and, ultimately, causes hyperproduction of hydrochloric acid.

There are 3 phases of *gastric secretion*:

Phase I - **nerve-reflex**, which begins with *irritation of the vagus*, therefore it is called "vagal". ***Hypoglycemia*** is the most important among all the stimuli (visual, olfactory, and others); it occurs 3-4 hours after previous meal (or after a night's sleep), which causes the onset of the hungry state and leads to the beginning of phase I with the release of the so-called "inflammatory juice" (I.P. Pavlov) by the parietal cells of the gastric body. The peculiarity of the gastric secretion of this phase is that its volume is small, the hydrochloric acid content is low and its main purpose is to initiate the onset of the second powerful phase when it enters the antral department of the stomach.

Phase II of gastric secretion is called **neuro-endocrine** (or "*gastric*") and is initiated by *gastrin hormone* produced by the cells of the antral department of the stomach in response to their irritation with "inflammatory juice" and the food which a person begins to take. Gastrin through the blood begins to stimulate the release of gastric juice by the same parietal cells of the gastric body in sufficient quantities for digestion and rich in hydrochloric acid and enzymes.

Phase III of gastric secretion is called "***intestinal***" (not fully understood and is not investigated in practical medicine).

Currently, two basic methods are used to study gastric secretion and acidity: aspiration-titration and intragastric pH-metry.

Aspiration-titration methods. The essence of the methods is the aspiration of gastric contents by means of a thin probe into the test tubes, followed by the titration of the resulting content. It

determines the volume of secretion on an empty stomach and after stimulation in each portion, pepsinogen, total and free titration acidity, acid discharge and other parameters in order to study the functional state and potential capabilities of the secretory apparatus of the stomach.

One of the methods - according to Morzhatko's method, which determines "basal" (fasting) secretion, involves intravenous administration of insulin for stimulation of the 1st phase of gastric secretion, and hypodermic administration of histamine (pentagastrin) for the 2nd phase. Each phase is examined for an hour with aspiration of gastric juice every 15 minutes, that is, 12 samples (in test tubes) are received. In the laboratory, each sample is titrated and free HCl discharge is calculated in each of the 12 test tubes according to the formula: $D_{HCl} = A \times V / 1000$ mM/l (where D is HCl discharge, A is free HCl in the titration units and V is the volume of gastric juice in ml in each test tube), then the hourly discharge of hydrochloric acid in phases I and II is calculated by summing the resulting D_{HCl} in four test tubes of the corresponding phase. The obtained results are compared with the norm (phase I - 3-5 mM/l, phase II - up to 10-13 mM/l).

In practice, these methods proved to be insufficiently informative and difficult to standardize, due to which the method of ***pH-metry*** was proposed, which is based on determination of free hydrogen ions H^+ concentration directly in the stomach.

pH-metry. The principle of electrometric pH determination is that when immersed in a solution of antimony-calomel electrodes, chemical processes are accompanied by the release of electrical energy, the same as in the galvanic elements. The difference in potentials between the measurement electrode and the comparison electrode forms the electromotive force (EMF). The magnitude of the EMF depends on the activity of hydrogen ions in the electrolyte. This difference is small. To measure the EMF, a DC amplifier is used to which the display or recording device is connected.

Various variants of *pH-probes* with antimony-calomel electrodes are used in Ukraine. E.Yu. Linar's gastric probe is considered to be their basis. Acid-producing function of the stomach in our clinic is studied by a factory-supplied two-channel pH-probe of closed type. It has a common calomel electrode for body and antrum electrodes, located in antral olive; registration is carried out on AGM-10-01 device.

Patients stops taking food, antacids, atropine, ganglion blockers12 hours before the study. Under X-ray control, a two-channel pH probe is administered into the stomach to the level of its sinus. X-ray monitoring of the probe is currently the most accurate.

There are several implicit signs indicating the correct position of the probe in the stomach. Thus, in fractional probing, a satisfactory flow of gastric contents in most cases indicates that the end of the probe is in the antrum of the stomach. In pH-metry of different parts of the stomach, valuable data on this issue is often compared with the indications of the antral and intermedial (or body) pH-olive. In cases where, after the introduction of the probe, the values of pH-antral olive exceed the values of pH-intermedial (body) olive, it can be assumed that the probe is in the correct position. However, in some patients, there is no difference between pH values of the mentioned parts of the stomach and then the method is unacceptable. Thus, for the accurate study of the environment in different departments of the upper department of the gastrointestinal tract, the X-ray method of probe monitoring is currently indispensable.

During the study, each patient undergoes measurement of pH of the body and antral parts of the stomach with a 15-minute interval; values of intragastric pH are indicated on special forms. At the end of the study, changes in the pH of the body and the antral parts of the stomach are depicted graphically on these forms.

Patients sit in comfortable armchairs during the study. They are recommended not to swallow saliva, but to spit it into a special tube. This is necessary so that saliva does not artificially alter the intramuscular environment of the intestine.

Acid production in the body and antral parts of the stomach in phase II of gastric secretion is studied using *a histamine test* at the rate of 0.01 mg per 1 kg of the patient's weight, histamine hydrochloride is administered subcutaneously. *Medication vagotomy test* ("*atropine test*") is conducted

for preoperative study of the effectiveness of the planned vagotomy, and in the remote postoperative period to study the adequacy of the performed vagotomy.

The results of the acid-producing function of the stomach are interpreted in accordance with E. Yu. Linar (1968) and Yu.Ya. Ley (1976).

Characteristics of the acid-producing function of the stomach in the study of *basal pH*: pH = 1.5 and below (1.4; 1.3 and less) - *hyperacidity* (continuous acid formation); pH = 1.6-2.0 - *normocidity* (continuous acid formation of medium intensity); pH = 2.1-5.9 - hypoacidity (continuous acid formation); pH = 6.0 and above (6.1, 6.2 or more) - *anacidity*.

Characteristics of the acid-producing function of the stomach in the *use of dosed stimulation with histamine*: pH = 1.2 and less - *hyperacidic reaction*; pH = 1.21-2.0 - *normocidal reaction* (medium intensity); pH = 2.1-3.0 - *hypoacidic reaction*; pH = 3.11-5.0 - *lowered reaction*; pH 5.1 and above (5.2; 5.3 and more) - *anacidity*.

The effectiveness of *atropine test* is assessed by the degree of increase in the intraventricular pH as *high* (pH> 2.0), *average* (pH from 1.0 to 2.0), *low* (pH from 0.5 to 1.0), and *insignificant* or *negative* (pH to 0.5) or non-existent. According to the conducted research patients can be atropine-sensitive (A+ patients whose pH increased from 0.5 and lower, after administration of atropine, increased the) and atropine-insensitive (A-).

The method of prolonged continuous intracavity monitoring of pH (2009). This method is the most physiological and accurate, allowing to evaluate the effect of various factors affecting acid production (diet and medication, smoking).

The essence of the method: the 24-hour intragastric acidity profile is studied to detect the daily circadian rhythm of hydrochloric acid production and its significant change in patients with peptic ulcer disease, reflux esophagitis, gastritis and duodenitis (antropyloroduodenitis). Based on this study, it is possible in the majority of cases to differentiate autoimmune gastritis (up to achlorhydria as a result of parietal cell atrophy) from Helicobacter pylori-associated gastritis. The latter never manifests itself as achlorhydria, since in this form the atrophic process has a focal character, and in antral gastritis, antropylorobulbitis, peptic ulcer disease with the localization of ulcers in the pylorus and in the bulb of the duodenum, the acid-producing function is elevated, especially at night. In reflux esophagitis and insufficiency of the lower esophageal sphincter, this method detects regurgitation of acid contents of the stomach, and gives an opportunity to study the frequency and duration of reflux. This method allows us to assess the effects of various agents on the intra-esophageal, intragastric environment, depending on the dose, method of administration and the time of their administration.

The study is carried out using AGM-24 MP ("Gastroscan"-24) device – an acidogastometric automation complex (Russia). The AGM-24 MP device is designed for continuous recording of pH values in the esophagus, stomach and duodenum for 24 hours at a reading interval every 20 seconds. The study is carried out using a pH probe introduced transnasally and connected to a secondary converter (acidogastometer "AGM-24 MP"), with the subsequent transfer of an array of data for processing on a PC.

pH-probes used in this study comprise 2 mm measuring antimony electrodes fixed in a polymer tube of 2.0 mm in diameter and an external chlorine silver electrode for comparison. The probes can be used in three main ways: with one (G_1), two (G_2) and three (G_3) measuring electrodes. For patients of different age groups, the transducers are made with an inter-electrode distance of 50, 70, 90, 110 and 120 mm.

Preparation of the patient for examination, introduction of a pH-probe (under X-ray control) and interpretation of the results (basal, after dosed stimulation of histamine and atropine test) are similar to the traditional pH-metry.

Method of gastrin determination. Gastrin is determined in blood serum by enzyme method. The normal content of gastrin in plasma in adults is less than 100 pg/mg; average level is 14.5-47.5 pg/mg.

Gastrin exists in 4 main forms: gastrin-13, gastrin-17, gastrin-34 (containing 13, 17 and 34 amino acids in their molecules, respectively) and the unidentified "Big-Big"-gastrin.

Food is a physiological stimulant for the release of gastrin and it is also released: 1) under the action of reflex factors (stomach stretches when food enters); 2) under the influence of nerve stimuli; 3) under the influence of chemical factors, such as calcium and adrenaline.

Daily fluctuations in gastrin concentration in blood are as follows: minimal concentrations are observed between 3 and 7 o'clock in the morning, with the highest levels during the day. Basal gastrin levels increase significantly with age. In healthy people, food, especially protein, ingestion increases gastrin concentration in blood by 50-150% (the maximum level of gastrin in the blood is reached at the same time in 15-40 minutes.). The half-life of gastrin is 8 minutes. It is excreted from the blood by the kidneys, where it is cleaved after filtration and resorption. The secretion of gastrin is also stimulated by lowering the hydrochloric acid concentration. In norm, acid suppresses secretion of gastrin, when the stomach contains a sufficient amount of the latter. Not always hyperproduction of gastrin is accompanied by hypersecretion of acid in the stomach. Hypergastrinemia without hypersecretion of hydrochloric acid occurs in pernicious anemia, atrophic gastritis and vitiligo.

The most important clinical aspect of the use of *determining the level of gastrin* in plasma is the diagnosis and control of treatment of Zollinger-Ellison syndrome or gastrinomas. *Gastrinoma* is clinically characterized by hyperplasia of the mucous membrane of the stomach up to the formation of peptic ulcers. In this case ulcers are mostly postbulbular and can be observed in the extent of the esophagus to the small intestine. The disease is manifested by gastric hypersecretion, diarrhea, steatorrhea. The diagnosis of gastrinoma can be confirmed by high levels of serum gastrin in combination with clinical data. Morphologically, the substrate of *Zollinger-Ellison syndrome* is a gastrin producing tumor (ulcerogenic adenoma), usually of pancreas, the diagnosis can be confirmed by ultrasound or CT in combination with clinical data.

An increase in the level of gastrin in the blood can also be found in patients with gastric cancer. In patients with severe atrophic gastritis in the antrum of the stomach, the risk of developing stomach cancer is 90 times higher than that of people with normal mucous membrane. The study of gastrin-17 should be conducted after obtaining a positive result of the detection of antibodies to ***Helicobacter pylori***. For further differentiation of atrophic gastritis from nonatrophic one, it is necessary to determine the level of pepsinogen I.

Differential diagnosis of diseases, causing the increase of gastrin in blood, implies determination of gastrin after its stimulation by the introduction of calcium chloride. Calcium chloride is administered intravenously over a period of 4 hours. Blood samples are taken on an empty stomach and after 1, 2, 3 and 4 hours after the introduction of calcium chloride. In Zollinger-Ellison syndrome, in response to calcium administration, an inadequately high rise in gastrin concentration is observed, and in patients with atrophic gastritis, pernicious anemia, its level is reduced. Loading tests can be useful in determining the threshold values of gastrin concentration. About 90% of patients with Zollinger-Ellison's syndrome, having a threshold level of gastrin on an empty stomach, have an elevated level of gastrin in response to stimulation of secretin

X-ray methods for examination of the stomach

X-ray method is one of the main (along with fibrogastroscopy) in the diagnosis of diseases of the stomach in general and its ulcer disease in particular.

X-ray examination of the chest and abdominal organs. Studies begin with an observational X-ray examination of chest and abdominal organs. Target review helps determine the size of the liver and spleen, the height of the cupula of the diaphragm, the presence of free gas in the abdominal cavity, deformation of the spine, presence of calcifications and concretions, the size and shape of gastric air bubble, additional shadows on its background, position, size of gas bubbles and horizontal levels of fluid in the extent of small and large intestine.

Interpretation of the results of observational X-ray of chest and abdominal organs in perforated ulcers of the stomach and duodenum. In perforated ulcer (PU) of the stomach and duodenum, the radiological diagnosis mainly comprises the detection of *free gas in the abdominal cavity (pneumoperitoneum)*. Under the diaphragm, more often on the right when the patient is in a standing position or under the anterior and lateral abdominal wall in the patient's position on the back or side, there is sickle-shaped lucency (*Zhober's symptom*). This symptom is observed only in 70-75%

of patients. This is due to the difficulty of detecting a small amount of gas with small dimensions of the perforation opening, closure of the opening with gastric contents or adjacent organ, and also the presence of a number of patients with periprocess with a large number of adhesions (*covered perforation*). The presence of gas under the left cupula of the diaphragm is most often observed in perforation of a large curvature of the stomach and is interpreted as a "Yudin's sign".

The presence of gas under the diaphragm may be simulated by the interposition of the colon (Chilaiditi's symptom). To avoid mistakes, it is necessary to examine the patient in different positions. In the presence of free gas under the diaphragm after changing the position of the patient, the strip of gas is shifted accordingly, which is not observed in the intestinal interposition.

In suspected PU in the absence of symptoms of pneumoperitoneum special methods of examination are carried out: pneumogastrography, stomach studies with water-soluble contrast agents and the method of double contrast.

Pneumogastrography (*Hennelt's test*) implies administration of 1400-1600 cm^3 of oxygen or ordinary air through the probe to the stomach. The air is introduced with a probe (after insertion, the probe is closed with a clamp) or during gastroscopy through a gastroscope or duodenoscope. Passing through the perforation hole, the air accumulates under the liver or diaphragm, resulting in a characteristic radiological picture.

X-ray examination of the stomach with contrast. Exploration X-ray of the organs of the chest and abdominal cavity is followed by conventional method of contrasting the stomach with barium suspension in various projections. After the first ingestion of a contrasting substance, the folds of the mucous membrane of the stomach are examined by palpation with particular care, identifying their possible variants, transformation in time course and during peristalsis, as well as the modification of folds associated with changes in the position of the patient's body.

With the help of dosed compression and palpation at different degrees of filling of the stomach, it is necessary to consider such signs as rigidity and breakage of the folds of the mucous membrane, the symptom of single expanded fold, convergence, the presence of longstanding definable and indestructible stains in the background of the mucosal contour.

Examination of infants, persons with developmental abnormalities, as well as patients in the early postoperative period, sometimes involves artificial contrasts of the mucous membrane with iodine-containing substances (urotrast, verografin), at the rate of 20-40 ml per 60-100 ml of water. The filling with a barium suspension (in some cases, a double portion) allows to determine the shape, anatomical relationships, displacement of the stomach, its contours, the absence or presence of deformations, circulatory or marginal filling defects, the nature of the evacuation-motor function.

A compulsory condition is the poly-positional examination of the stomach, including orthoscopy (vertical position of the patient), trochoscopy (horizontal position of the patient in the back-front or anterior-posterior passage of the beam), laparoscopy (position of the patient on the side at the back and the anterior stroke of the beam, back and abdomen at the front of the beam).

The poly-positional examination in the above-mentioned provisions also provides a different degree of turns, bends, inclinations of the patient's body for the selection of optimal projections for the purpose of taking pictures.

The shape of the normal stomach in the X-ray image most often resembles a hook or a horn, although it can be very diverse depending on the tone of the stomach, the degree of its filling and the values of intra-abdominal pressure. Depending on the degree of the pathological process in gastric ulcer, its form undergoes changes from the insignificant to severe. The shape of the stomach is affected by penetration of the ulcer in the surrounding organs and perigastritis.

Cicatricial changes secondary to peptic ulcer can result in various deformations of the stomach. Normal peristalsis causes waves of average depth, manifested by 2-3 retractions along the large curvature, moving in the distal direction and giving a pronounced exhalation of the antral department.

Sufficiently active peristalsis of the stomach, not in all cases, can result in a complete emptying of the organ. Rare occurrence of small amplitude of peristaltic waves is observed in gastric hypotonia or stenosis of the pylorus in the stage of decompensation.

It is important to provide X-ray examination of the relief of the mucous membrane of the stomach. Normal mucus is characterized by the variability of the relief, which does not happen in pathological changes. In gastric ulcer in the presence of chronic gastritis, the relief of the mucous membrane radiographically appears to be significantly altered.

Impairment of the motor-evacuation function of the stomach in ulcer disease is not only causative, but also significantly affects the clinical manifestations of the disease. The condition of this function of the stomach is taken into account not only when choosing an optimal method of surgical treatment, but also serves as one of the criteria for evaluating its results in gastric ulcers.

The evacuation function of the stomach in gastric ulcer (without taking into account ulcer of pyloric portion) does not undergo significant changes and is often characterized as normal, although in some cases it may be delayed.

Nowadays X-ray diagnosis makes it possible to identify gastric ulcers in 95-97% of cases. X-ray signs of gastric ulcer are commonly divided into indirect and direct.

Indirect signs (changes in tone, evacuation, pain sensation, etc.) are not characteristic of ulcers and can occur with many other diseases.

The most commonly known and most prominent radiological sign of gastric ulcer is *ulcerative niche* (*Gaudek's symptom*). The latter is a defect of the inner surface of the stomach wall of varying size and depth, and often has a crater form, but may be sharpened or flat with a wide base. In penetration of the ulcer in the adjacent organs, the niche is located at a distance from the wall of the stomach and is usually connected to the gastric channel. The larger the "niche" and the farther it is from the gastric wall, the greater the extent of findings concerning the penetration. The relief of the mucous membrane around the ulcerous "niche" is often presented by *folds converging to it*. An inflammatory shaft is often determined around the ulcer, which makes the niche look deeper than it actually is. Also, radiologic signs of gastric ulcers include *retraction of the relief of large curvature ("index finger" sign)*. Gastric ulcer, as a rule, is solitary and is located on the small curvature or adjacent areas of the anterior or posterior gastric walls.

Summarizing the foregoing, gastric ulcer is characterized by the following reliable X-ray characteristics: the presence of a "niche", folds converging to it, an inflammatory shaft around the ulcer and retraction of the relief of large curvature ("index finger" sign).

Contrast radiography of the stomach allows to evaluate its *motor-evacuator function* to detect *stenosis of pylorus or gastrostasis* (complications in the postoperative period, more often after bilateral stem or bilateral selective vagotomy). For this purpose, X-ray Lindenbratten classification is used, in which:

Stage I of stenosis is a *compensation stage* in which there is a delay in the barium suspension in the stomach cavity for 6-12 hours;

Stage II is *subcompensation*, delay of contrast from 12 to 24 hours;

Stage III is *decompensation*, delay of more than 24 hours.

In the study of the stomach with water-soluble contrast agents, barium sulfate suspension is not used in suspected PU, as its penetration into the abdominal cavity causes the formation of dense, hardly soluble conglomerates. Water-soluble contrast agents (cardiotrast, gipak, urografin, gastrografin, etc.) are used in the amount of 40-60 ml. In small dimensions of the perforation opening and significant scarring, a minimal yield of contrast material may appear in the form of a small "tentacle" (*"tentacle" sign*).

In the *method of double contrast*, after preliminary removal of the gastric contents by a thin probe, 400-600 cm^3 of air and 40-60 ml of water-soluble contrast agent are introduced. This allows to detect not only free gas in the abdominal cavity, but also the output of contrast material outside the body at the perforation level.

Additional methods. *Pneumogastrography combined with pneumocolonography*. Air or a gaseous mixture is introduced into the lumen of the stomach, and using the Richardson cylinder, air is pumped into the large intestine. This method can provide valuable information on the size, shape and spread of the tumor, and also allows for a more accurate definition of elasticity or rigidity of the gastric wall than with basic X-ray techniques.

*Combination of double contrast of the stomach (*introduction of 30-50 ml of barium suspension) *with gas insufflation of the large intestine*. The described method helps to get information about air contours of the parts of the stomach adjacent to the large intestine. The most optimal positions are anterior direct, right and left anterior oblique in the vertical and horizontal position of the patient, as well as examination in lateral position.

Parietography (refers to the method of double contrast). This method involves introducing gas (oxygen or nitrous oxide) into the abdominal cavity, air or gaseous mixture into the lumen of the stomach. This study is conducted in tumors of the stomach, which require clarification of localization and extent of distribution within the body and beyond.

Triple contrast method. The final stage of parietography is triple contrast. In this method after double contrast poly-positional X-ray, and in some cases after parietotomography, patients swallow 2-3 gulps of barium suspension. Just as with double contrast, the patient should lie down on a couch and slowly turn to the back, to the left and to the right side, and then again X-ray examination is performed in the corresponding positions. The method improves conditions for studying the inner surface of the stomach. Triple contrast is combined with layered study.

Parietotomography is a tomography in combination with parietography. This method is used to receive a complete picture of the state of gastric walls in the region of small and large curvature. The depth of the tomographic layers depends on localization of the tumor in the stomach and the volume of the subject being studied. For tomography of the body of the stomach, X-ray tube is centered on the outer edge of the left direct abdominal muscle 1-2 cm above the navel. For tomography of the antrum, the tube is centered on the inner edge of the right direct abdominal muscle.

Examination of the stomach with the use of pharmacological agents. Studies involve administration of pharmacological agents, which include substances that enhance the tone and motility (morphine, aceclidine, proserin) or suppress it (atropine, metacin, buskopan, aeron, nitroglycerin). Morphine is administered subcutaneously in the amount of 1.0 ml of 1% solution or intravenously 0.5 - 0.25 ml of the same concentration. When administered intravenously, the drug gives a faster and more pronounced diagnostic effect with a minimum of side effects. Injection of morphine is followed by a study of the stomach filled with barium suspension in 10-15 minutes. The action of morphine leads to increased tone of the stomach, stimulation of peristalsis in areas which are likely affected by tumor. Indications for "*morphine test*" are the difficulties of differential diagnosis of infiltrative form of cancer, inflammation of the mucous membrane of the stomach. Aceclidine is used as a subcutaneous injection in a dose of 0.5-1 ml of 0.2% solution. Studies begin in 10-15 minutes.

Atropine, metacin, aeron, nitroglycerin and other cholinolytics are used in changes in the esophagogastric junction, antral and pre-pyloric divisions and the pylorus, various kinds of deformations of the stomach. Atropine is administered subcutaneously in the amount of 1-1.5 ml of 0.1% solution or intravenously in the amount of 0.5-0.75-1 ml of the same concentration. Metacin is used as a subcutaneous injection in a dose of 2-4 ml of 0.1% solution. Atropine and metacin are administered following a common X-ray examination. Immediately after the injection of the drug, the patient receives an additional portion of the contrast agent and the study is repeated in 10-15 minutes. Indications for the use of these drugs are determined during the X-ray examination of the stomach. For example, in a spasm of the pylorus or a cascade-like deformation of the stomach, the patient is given 2 tablets of aeron or 0.5 tablets of nitroglycerin under the tongue and after 10, 15 and 20 minutes after absorption the study continues.

X-ray examination with the use of pharmacological agents can be performed once or repeatedly. Multiple studies are required to create a cumulative effect of the agent, which causes functional changes in the body. For example, atropinization for 10-14 days in spasms of the esophagus, spasm of the pylorus, administration of No-Spa for the same purpose with further follow-up studies.

The use of pharmacological agents should be combined with conventional X-ray examination of the stomach and can be carried out with pneumogastrography (with a probe or without a probe), parietography, angiography. Indications for the use of a particular agents include difficulties of differential diagnosis of gastric cancer and non-tumor diseases, as well as tumors and tumor-like structures of adjacent organs.

Computed tomography provides gradual visualization of gastric wall and differentiates the structure of the tissue. Its role in the diagnosis of stomach diseases is universally recognized. The principle of this diagnostic method is that the source of X-rays, which rotates around the patient's body, conducts multiple studies from different points of the circle, transmits the data received to the detector, which are converted into electrical impulses and laid in the memory of the computer. The processed data reproduces a "slice" of the body or organ on the screen. The received information can be recorded and photographed at the same time. However, the high cost of equipment, the small number of devices, as well as the presence of radiation burden on the patient significantly limits its capabilities.

Angiography. The fact that blood is supplied to the stomach from 6 sources (left gastric, right gastric, gastro-duodenal, left gastromental, right gastromental, short gastric arteries), significantly complicates angiographic examination. Contrasting of these vessels is possible in selective catheterization of the abdominal artery or directly by the gastric arteries. Catheterization of the abdominal artery is carried out through the abdominal aorta using a special catheter. First, the catheter is punctured (by Seldinger) into the femoral artery, and then through the articular arteries in the abdominal aorta to the level of the diaphragm. The ostia of the abdominal artery are searched along the left anterior-lateral surface of the abdominal aorta at the level of the XII thoracic vertebra. After the introduction of the catheter in the mouth of the abdominal artery and some advance (1-1.5 cm), it is necessary to contrast blood vessels departing from the abdominal trunk, namely splenic, common hepatic and left gastric arteries.

Tri-iodine water-soluble agents (verografin, urotrast) are used to contrast vessels. Total amount of 35-40 ml of 60-80% solution of contrast material is injected at a rate of 10-15 ml per 1 second. The first photograph is taken two seconds after starting the introduction of contrast media, and then 3-4 photographs at intervals of 1 second and 3 more photographs with a gap of 2 seconds at the end of the series. Angiograms show arterial, capillary and venous phases of blood supply to the liver, spleen, pancreas and stomach.

Angiography of the stomach in pharmacological impact on the vessels of the abdominal cavity:

1) The patient is administered an antihistamine agent 30 minutes before the study and then subcutaneously, histamine in a dose of 0.04 ml per 1 kg of body weight. Then pneumoperitoneum is imposed and a gas-forming mixture is administered. Celiacography is performed 30 minutes after the administration of histamine;

2) 0.5 ml of 1% solution of histamine is injected intraarterially through a catheter inserted into the abdominal artery, and celiacography is performed in 1 minute. 300-350 ml of air is pumped into the lumen of the stomach in advance. X-ray picture shows vessels up to 4-5th branches, and the mucous membrane of the stomach is determined in the capillary phase.

Superselective angiography of the stomach. This type of angiography of the stomach involves specially designed "Cobra"-type catheter which can be used for catheterization of the left gastric, gastroduodenal and right gastromental artery. The dose of contrast substance in this form of angiography decreases to 10-15 ml, the serialization program remains the same.

Angiographic examination of the stomach is indicated in cases of difficulty in diagnosis and clarification of the extent of the spread of the tumor process on adjacent tissues and organs.

Radiocinematography. The essence of the method: the study of the patient begins with the conventional poly-positive television illumination, with radiography and at certain stages cinematography.

Algorithm of X-ray examination of the stomach

1. Fundus, cardiac part and subcardiac department. The first stage is radioscopy and radiography, pneumogastrography, double contrast, the use of pharmacological agents, radiocinematography. The photographs are made in anterior and posterior, right and left anterior oblique projections in the vertical and horizontal position of the patient, as well as in the lateral position on the left, right side and back. The second stage is parietography, tomography, triple contrast in the above-mentioned projections. The third stage is angiography in the direct and posterior projections.

2. Body, sinus and region of the angle. The first stage is radioscopy and radiography, radiocinematography combined with the use of pharmacological agents, pneumocolono- and pneumogastrography, double contrast in the anterior, posterior, direct, left, right anterior oblique, and lateral projections, as well as in the lateral position on the left and right sides. The second stage is parietography combined with a tomography, radiocinematography in the positions mentioned above. The third stage is angiography in the same projections.

3. Antral, pre-pyloric department and the pylorus. The first stage is radioscopy and radiography, double contrast, the use of pharmacological agents with photographs in the anterior, posterior direct, right anterior and posterior projections in the vertical and horizontal positions of the patient, in the lateral position on the left and right side. The second stage is parietography combined with tomography, radiocinematography in the positions mentioned above. The third stage is angiography in the same projections.

Differential X-ray diagnosis of gastric ulcers. Differential X-ray diagnosis of gastric ulcers is performed in malignant and benign tumors, rigid antral gastritis, aberrant pancreas - choristoma (additional lobe), bezoar, gastric sarcoma, gastric lymphogranulomatosis, gastric syphilis, gastric tuberculosis, Menetrier disease, diverticulum of the stomach, volvulus of the posterior wall of the stomach; deformation of the stomach, caused by pressure of enlarged adjacent organs, retroperitoneal inorganic tumors.

Gastroscopy of the stomach

Gastroscopy *is a visual examination of gastric cavity and various diagnostic and therapeutic manipulations conducted under the control of an opto-mechanical device, gastroscope.*

Gastroscopy currently takes one of leading places in the complex of diagnostic and therapeutic procedures in ***gastric ulcer***. This method of investigation allows to improve the diagnosis of ulcers of different localization, to conduct observations in time course in the process of their healing, to identify the associated lesions of the gastrointestinal mucosa (gastritis, erosion, etc.) and their relationship with the main process, as well as to conduct differential diagnosis of benign and malignant ulcers All these issues are especially important for surgeons in determining the indications for surgical treatment of patients and the choice of method of surgery.

Gastroscopy is intended to clarify the diagnosis of peptic ulcer, in suspected gastric tumors, degeneration of ulcer or polyp, for the diagnosis of acute gastroduodenal bleeding and perforations.

Gastroscopy is contraindicated in the general severe condition of the patient, in heart and lungs failure, stage III hypertonic disease, common atherosclerosis, aortic aneurysm, laryngeal and esophageal disorders, hemophilia.

Gastroscopy is performed according to the generally accepted method with patient lying on the left side. Premedication is carried out in 20-30 minutes before examination by the introduction of 2% promedol solution. Anesthesia of the pharynx is performed by spraying 0.5-1.0 ml 1% dicaine solution. Fibrogastroscopes of the Japanese company “Olympus” and other companies are used.

Interpretation of findings of visual gastroscopic examination. The endoscopic pattern of ***perforated ulcer*** is characterized by the absence of ulcer bottom, vertical white edges, and signs of acute inflammation around (ulcer limited by rigid callous edges, shaped like a cylinder or cut cone with a wide base, wrapped in the lumen of the organ, it can be filled with pieces of food and dirty-grey necrotic plaque).

During ***bleeding***, the bottom of the ulcer is filled with dark or fresh clots, from under which blood can flow. Edges more often bleed in acute ulcer. If the examination of the gastric walls is difficult due to the presence of blood or other contents in their lumen, then it is possible to rinse the stomach with cold water through the biopsy channel of the fibroscope. It is advisable to also purposefully wash the spot of the bleeding.

Mallory-Weiss syndrome often causes bleeding. Endoscopic examination defines longitudinally located disruption of the mucous membrane of the cardiac part of the stomach. The disruption is most often localized on the small curvature and on the posterior wall and begins immediately below the dentate line. The type of disruption depends on the time elapsed since its development. Immediately after the onset of bleeding, examination shows a rupture of the mucous

membrane of various sizes (from 0.5 to 2 cm) with sharp angles and edges is determined. The disruption is filled with clots of blood. The edges are torn, dazed with blood, of purple-red color. The disruption(s) is parallel to the folds. Endoscopy performed at later stages shows that the disruption has a shape of longitudinal superficial injury, filled with fibrin. The edges are moderately inflamed with pinpoint hemorrhages. At this stage, Mallory-Weiss syndrome differs from the ulcer by the absence of an infiltration shaft, convergence of folds and deformation of the surrounding mucous membrane. Examination at later stages shows an oval or linear region covered with a young epithelium in the region of the disruption.

During the ***exacerbation of the disease***, gastric ulcer is oval or round, covered with yellowish plaque due to fibrinous stratifications. On the periphery of the ulcer, there is a uniform roll, formed by a hyperemic edema of the mucous membrane. At the ***stage of healing***, inflammatory changes along the edges of the ulcer decrease, resulting in the smoothing of the roll and the ulcer becomes more superficial. The bottom is cleared of plaque. ***Scarring of ulcer*** is characterized by an area of hyperemic mucosa with the involvement of the gastric wall due to scar changes.

A typical ***chronic gastric ulcer*** looks like a tissue defect of oval, round or irregular shape with clear uniformly thickened edges, a smooth, fine-grained bottom covered with a yellowish-gray plaque.

Bleeding chronic ulcer can be filled with large clots that are not bleeding or a fluffy bright clot with blood leaking from under it. Washing the clots at the bottom of chronic ulcer shows one or more thrombosed or bleeding vessels.

In ***penetration of gastric ulcer*** to the adjacent organ, it is crateroid, which, in combination with the change in the mucus around and its immobility as a result of periprocess is a secondary endoscopic sign of penetration of ulcer.

In the course of gastric ulcer secondary to ***surface*** or ***atrophic*** gastritis, gastric ulcers are flat, and inflammatory changes in the mucous membrane around the ulcer are practically absent.

In ***flat ulcers***, located on the small curvature, there is a change in the relief of the mucous membrane due to scar deformation. At the same time, ulcer thickens, the bottom is covered with granulation, the inflammatory shaft of the mucous membrane decreases, granulation and epithelization develop from the edges of ulcer.

Symptomatic or hormonal ulcers are characterized by the correct circular form and number (always a few). They are located in groups; the bottom is flat, covered with a loose fibrinous plaque. The edges of symptomatic ulcers are flat, practically within the relief of the mucous membrane and of a round shape. Inflammatory changes on the edges of ulcers are almost absent; they are swollen secondary to mild hyperemia of the mucous membrane. Bleeding is observed from the edges of the ulcer.

Erosions of gastric mucosa can be *complete* and *incomplete*, can be localized separately, locally or diffusively throughout the surface. Incomplete erosions have the appearance of small (0.1-0.5 cm) polygonal surface defects in the mucous membrane, coated with pure white filament fibrin films or hematite brown crust, which are difficult to launder, exposing a bright bleeding granulation tissue or surface. Being located in groups, merging together, erosions form areas covered with ulcers, which are difficult to differentiate with surface ulcers. Erosions are located on the background of hemorrhages of the gastric mucosa, some of them have the form of pinpoint petechial hemorrhagic ulcers, covered with crust.

Complete erosions are located on the pillow-shaped, edematous inflamed basis, which rises above the level of the mucous membrane. “Streams of blood” are determined at the height of the bleeding, which extend from individual erosions in the form of threads or crust of brown hematin.

Reactive erosive gastritis secondary to acute chronic ulcer is often the primary source of bleeding. Histological study plays the main role in the differential diagnosis between ulcers and erosions.

Particular importance is given to gastroscopy in **early differential diagnosis of benign and malignant ulcers**. Unlike benign ulcers, **malignant ulcer** acquires an irregular shape. Its bottom becomes uneven, with roughness on the edges and polypoid expansions. There is a diffuse infiltration of the gastric wall at the site of the lesion.

Gastric cancer ranks third in terms of the frequency of bleeding. There are such varieties of gastric cancer as *exophytic* (growing to the lumen of the stomach), *endophytic* (infiltrating the wall) and *mixed* (exophytic-endophytic).

In exophytic growth, the tumor may have the appearance of a plaque, oblong polypoid growth or a mushroom-shaped form on a broad basis. The tumor is usually red or grayish-yellow. It has a hilly surface and is separated from the surrounding mucous membrane. In some cases, on the top of the tumor, an easily bleeding ulcer is formed, covered with plaque. *Exophytic forms of gastric cancer* include: *plaque form* (reflecting the early stage of the tumor), *polypoid* (mushroom-shaped, cabbage-shape) and *dish-shaped* (cup-shaped).

Endophytic cancer, located in the submucosal layer, is characterized by infiltrative growth. There are infiltrative ulcerative and diffuse forms. *In infiltrative ulcerative growth* an ulcer with uneven raised edges and hilly bottom is located on the background of focal thickening of the gastric wall. There is no clear peripheral shaft of the mucous membrane, which is observed in gastric ulcer. On the periphery of the cancer, the mucous membrane of the stomach is pale, with rigid edges, which break off at the edges of the ulcer and do not straighten out when the stomach is filled with air. The diffuse form of the tumor is characterized by the absence of clear boundaries. The infiltration of a whitish-gray color forms like a bulge. Its surface is uneven, the folds of the mucous membrane are rigid, not strained, peristaltic waves are absent in this place. The most common *submucosal* variant of tumor growth: the mucous membrane in the lesion zone bulges into the lumen of the stomach due to submucosal infiltration and thickening of the gastric wall. Layers of the mucous membrane are massive, enlarged, smoothed, with disrupted pattern. The color is gray-pink and dull. The walls of the stomach are rigid, immovable in instrumental palpation. This, as well as the absence of the usual lacquer gloss of the mucous membrane gives it inanimate appearance.

Flat-shaped form of gastric cancer is characterized by a meagerness of macroscopic manifestations, which creates great difficulty in diagnosing. Gastroscopy shows flat, smoothed, slightly degraded zone compared with the surrounding mucous membrane. The folds of the mucous membrane can converge to this zone. They end at the tumor site with some thickening. The area of tumor lesion is represented by smoothed folds (they may be absent). In the tumor zone, in some cases, swelling of the mucous membrane with a velvety surface may be observed. Instrumental palpation shows rigidity of tissues. Mucous membrane in the zone of lesion is pale or hyperemic, dull. Peristalsis here is slowed down or absent.

Mixed form of gastric cancer involves tumors characterized by a combination of exophytic and infiltrative components. Recently, it is believed that the mixed forms of cancer are a transient phase of transformation of exophytic cancer into infiltrative, and therefore, the phase of further progression of the tumor.

Benign non-epithelial tumors include: leiomyomas, fibromas, neurinomas and vascular tumors.

Leiomyomas. Endoscopic examination shows a single tumor bulging into the lumen of the stomach of a circular or semi-spherical shape with a smooth surface and equal contours. The boundaries of the tumor are determined visually quite clearly. The surface of the tumor may be slightly wavy (undulating). In small neoplasms, the mucous membrane smoothly passes from the wall of the stomach to the tumor, as if covering it. In tumors with a diameter of 4 and more centimeters, the mucous membrane stretches, thins and becomes whitish due to blood circulation disorders.

Fibromas. Spreading inside the wall and outside the stomach, fibromas cause bulging and deformation of the folds of the mucous membrane of the stomach. Due to the prevalence of the connective tissue in the tumor stroma, they have a dense consistency.

Neurinomas are more often situated inside the stomach (60%), less often in the subserous layer and rarely in muscle. They have all the endoscopic signs of benign non-epithelial tumors, dense consistency, and are ulcerative in about half of cases.

Vascular tumors. Developing in the *submucosal layer* of the gastric wall, these tumors, including ***hemangiomas*** (simple, cavernous, mixed), ***lymphangiomas***, as well as a special group of ***glomangiomas,*** have a largely similar endoscopic pattern, despite histological differences. They

usually represent a tumor node with clear equal contours, smooth or undulating surface, and sometimes have a lobar structure. The color of the mucous membrane above them changes from bright red to bluish, sometimes it is pale, depending on the depth of the tumor, the degree of development and filling of vessels with blood. The tumor base is wide. Mobility is limited: the movement is shifted along with the surrounding tissues. The mucous membrane of hemangiomas is often ulcerated. The diameter of the ulcers varies from 0.5 to 3 cm. The bottom is covered with necrotic plaque. In some cases, ulcers through the fistulous passage are associated with a cavity of degradation in an exogastrically located tumor node.

Intramural or exogastric tumors present significant difficulties for endoscopic examination. In such cases there is a picture of compression of the stomach from the outside. In compression from the outside: 1) the boundaries of the area of the gastric wall protruding to the lumen are fuzzy, in the form of a gentle slope; 2) there are no changes in the mucous membrane in the area of deformation; its color, mobility and other signs do not differ from those in other, unaltered parts of the stomach.

Malignant non-epithelial tumors of the stomach include ***sarcoma***. In sarcomas of the stomach, which ***bulge into the lumen of the organ***, are associated with endoscopic changes of the mucous membrane of the stomach. The tumor is located on a broad basis, it has a rounded or cone-shaped, irregular configuration that protrudes into the lumen of the stomach. Its surface is uneven and hilly. The mucous membrane above it is thinned, smoothed, whitish in the center and hyperemic on the periphery, loose, easily injured in instrumental palpation. On the top of the tumor node there are ulcers, necrosis. The shape of ulcers is irregular. Edges are uneven, raised and bulging.

Plaque-shaped sarcoma, unlike polypoid, has the appearance of one or more small (4-5 cm in diameter) protrusions on the mucous membrane. Such formations are on broad base and endoscopically resemble a thickened polyp. The peculiarity of the plaque-shaped sarcoma is the plurality of lesions with fine-point erosions. The color of the tumor is reddish-brown with white inclusions and sections of necrosis.

If the tumor has the form of individual granular thickening, then it visually resembles the focal hyperplasia of the mucous membrane of the stomach.

Infiltrative form of gastric sarcoma, in particular torous infiltrative, is characterized by the destruction of the mucous membrane, absence of folding and contour on a significant extent. It is usually represented by one or more submucous nodes that merge together. The visible boundary of a healthy and affected mucous membrane is practically absent.

Flat infiltrative form is characterized by some convexity of the mucous membrane in the affected area with an uneven, slightly bruised surface of a grayish-white color, has immovable, as if frozen appearance. Layers of the mucous membrane are hyperemic, loose, juicy, and easily traumatized and bleeding in instrumental palpation.

Gastric sarcoma should be differentiated from ulcer, ulcerative form of cancer, Menetrier's disease. Endoscopic differentiation of gastric sarcoma is based on the evaluation of the totality of changes in the gastric mucosa: 1) detection of one or more tumor formations in the stomach, determining the shape and nature of the growth of the tumor, its size; 2) assessment of the state of the mucous membrane of the stomach (severity of infiltration, remodeling of relief), detection and microscopic evaluation of the nature of erosive and ulcerative changes; 3) determination of functional disorders of the gastric wall in the area of the detected disorder.

Hematosarcoma is a rare type of gastric sarcoma which is difficult to diagnose, and it should be differentiated from ***leiomyosarcoma, neurosarcoma*** and ***fibrosarcoma***. The latter develop in deeper layers of the gastric wall and are prone to intramural or exogastric growth. Hematosarcoma may be represented by: nodes of a bulging form, nodes of a bulging form with infiltration of the submucosal layer and only one infiltration of the submucosal layer, which is represented by thickened, expanded folds of the mucous membrane that changes its relief. In the infiltrative form of the tumor, erosions and ulcers typical for this disorder, are superficial. Defects in the mucous membrane can be the same and in the case of a bulging form of tumor, the ulcers are large in size and have elevated edges of dense consistency.

Submucosal location of sarcoma is its principal difference from gastric cancer. However, if the mucous membrane is affected both in one and in other cases, then the differential diagnosis is complicated. Therefore, it should be taken into account:

1) in sarcoma ulcers are multiple, irregular, superficial, and in case of cancer, the ulcer is single;

2) in the sarcoma of the edge of the ulcer is sharp, clearly defined; there is no shaft surrounding the ulcer, observed in cancer;

3) in sarcoma mucous membrane is preserved to the very edge of the ulcer, which is not observed in cancer.

An important differential diagnostic test to determine the difference between hemato-sarcoma from infiltrative gastric cancer is the discrepancy between the severity of tumor manifestations and the functional capacity of the walls of the stomach. Benign ulcers differ from sarcomatous ones by greater depth, regular contours, more evident reactive changes along the edge of the ulcer (swelling, thickened edges, hyperemia, etc.). ***Gastrobiopsy*** is necessary to provide the final diagnosis in each individual case.

Laparoscopy for surgical diseases of the stomach

Revision of abdominal organs and diagnostic interventions using an endoscopic device, a laparoscope, is called laparoscopy (peritoneoscopy).

Laparoscopic examination of the stomach is mainly performed in cancer in order not only to diagnose, but also to *identify metastases and determine the issue of operability and spread of the process*. In hummocky tumor and loss of peristalsis of the stomach it is necessary to examine the lymph nodes in its ligaments, and the surface of the liver.

Normally, laparoscopy gives a possibility to examine the greater part of the anterior wall of the stomach, only the small curvature is covered by the left lobe of the liver. The surface of the stomach is smooth, yellow and white; pulsed vessels are clearly observed on the curvature, peristalsis is preserved. Gastrocolic ligament and transverse colon, which is located under the large omentum are well visualized.

Laparoscopy is used to determine the degree of involvement in the tumor process of the stomach itself and the presence of dissemination of tumor in the abdominal cavity. Evaluation of the degree of involvement in the pathological process of the stomach itself is based on the identification of direct and indirect signs of lesion.

Direct signs appear in cases where there is germination of a tumor of the serous membrane of the stomach with a characteristic laparoscopic pattern: tumor growth in the form of single or multiple nodes of irregular shape, whitish-gray color, dense texture. The gastric wall in this zone is indurated. Palpation with manipulator, biopsy forceps or the tip of the laparoscope shows rigidity and lack of elasticity of the wall. Serous membrane is dull, it becomes whitish-gray with multiple hemorrhages. In 50% of cases, germination of the serous membrane reveals whitish, semi-translucent miliary nodules, which are manifestations of cancerous lymphangiitis.

Indirect laparoscopic signs are detected in cases when the tumor does not sprout through the serous membrane of the stomach. In this case there are changes in the vascular pattern, pallor, or hyperemia of the serous membrane. Instrumental palpation shows a decrease in the elasticity of the wall in the area of lesion. Localization of the tumor on the posterior wall without its transition to large or small curvature is often associated with a deformation of the stomach or protrusion of its anterior wall. In some cases, induration of the omentum in the place of transition to the anterior wall of the stomach and the presence of dense enlarged lymph nodes in large or small omentum serve as indirect signs of gastric tumor.

In cirrhosis of the liver: the liver size is diminished; it is dense, its anterior edge is sharp; there are small nodules of yellow-green, light brown or pink color on the surface, separated by light strips of connective tissue.

Portal hypertension is characterized by enlargement of the veins of the anterior abdominal wall, omentum, stomach, mesentery, etc. The loops of the small intestine are bluish-pink and are usually bloated (due to flatulence). There is a clearly visualized large number of dilated blood vessels

in the adhesions of the organs with the abdominal wall. The spleen is significantly enlarged, dark purple. In the abdominal cavity there is a clear ascitic fluid with yellowish tint.

Primary liver cancer is divided into the following forms according to the appearance: infiltrating cancer, nodal cancer and cancerous cirrhosis. ***Infiltrating cancer*** usually affects part of the liver; its texture is dense, the surface is uneven, the capsule is thickened and has yellow-green color, sometimes with a brick tint. ***Nodal form of cancer*** is characterized by yellow-pink tumor nodes of different sizes on the surface of the liver. Nodes give the liver hummocky appearance**.** ***Cancer secondary to cirrhosis*** is characterized by external signs typical for cirrhosis, and typical cancer nodes.

Metastases of cancer in the liver. On the surface of the liver there are white (such as pearly spots), light yellow or yellowish-red nodes of round shape, of varying sizes, of a dense texture, containing small depressions in the center. Metastases are clearly visualized on the background of normal liver parenchyma. Metastatic nodes of **melanosarcoma** are round or oval, black, with depressions in the center.

Differential diagnosis between mechanical and parenchymal jaundice. An important feature is the color of the liver. In mechanical jaundice, the liver is green (stagnant bile), and in acute hepatitis, it is bright red. Filled, unemptied gallbladder also indicates some mechanical obstruction in the course of the common bile duct.

Carcinomatosis of the peritoneum is characterized by many small humps and larger nodules of different sizes scattered on the parietal and intramural peritoneum, as well as the presence of ascitic fluid of dark pink color.

In ***tuberculous peritonitis***, too, there are many humps that have military shape and the same size. Ascitic fluid is light, opalescent and is not hemorrhagic. Severe proliferative process leads to the formation of adhesions.

Ultrasonography in surgical diseases of the stomach

Ultrasonography of the stomach can be carried out both with and without filling with liquid.

Basic principles of ultrasonography of the stomach include:

1) preparation of the patient for examination;

2) selection of sensors taking into account the constitutional features of the patient;

3) the choice of the patient's position for obtaining a qualitative image of all parts of the stomach;

4) application of standard and intermediate scanning planes;

5) methodical techniques during the examination.

1. Ultrasonic examination of the stomach is carried out on apparatuses not lower than the middle class with color, energy Doppler modes with the help of complex sensors in the frequency range of 2-10 MHz (for patients with high body weight, the frequency range is 2-5 MHz, for thin - 5-10 MHz)

2. Ultrasound of the stomach is carried out strictly on an empty stomach without its filling (10-12 hours after the last meal) and continues with its filling of 500-1000 ml mineral still or boiled water. Patients with constipation are given a cleansing enema before the examination. Before the examination the patient should not smoke, drink and chew gum. It is necessary to discuss administration of medicines in advance.

3. The position of the patient during the examination depends on his constitutional features and the task of studying particular department of the stomach (antral department - on the back, the bottom and the body of the stomach - on the left side).

4. All types of ultrasound examination in two-dimensional mode provide three standard sections - sagittal, axial and frontal, as well as multiple intermediate ones. In order to obtain a qualitative image of the stomach, an intermediate cross-section is used.

5. During the study, a qualitative examination of the stomach is achieved by fan-like or swirling movements of the sensor at different stages of breathing, with abdominal tension for a few seconds. For better visualization of the abdominal part of the esophagus, the patient is advised to take additional portions of the fluid through the tubes under the control of ultrasound examination in real time.

Ultrasonography of *gastric ulcers*. The ultrasound symptom of the affected hollow organ in gastric ulcer has a different degree of severity, depending on the activity of the process. In the remission period, the lesion area has a regular shape, symmetrical appearance; the outer diameter of the affected area is less than 30 mm, moderate thickening of the wall (8-12 mm) in the area of the infiltration shaft around the ulcer lesion. The central part of the image of the corresponding cavity of the stomach, as a rule, is not narrowed and not fragmented. The length of the affected area determined by ultrasonography is greater than in radiography or endoscopy. This is due to the fact that the ultrasound records the reflection from the inflammatory shaft, and other methods not. In contrast of the stomach with fluid, the reliability of ultrasound diagnosis of gastric ulcers increases. The inflammatory shaft around the ulcerative lesion in ultrasonography has a reduced echogenicity, and the niche itself is somewhat elevated, since it is more often filled with echogenic content, consisting of fibrinoid-necrotic detritus, granulation tissue and microtubules of gas.

The echographic pattern of the affected area in gastric ulcer depends on the localization of the process.

In the first variant of the localization of the ulcer on the small curvature of the antral and the angle, middle and lower third of the body of the stomach, as well as on large curvature, the ulcerative infiltration does not protrude into the lumen of the stomach, its thickness gradually decreases, the edges of the niche are located to the periphery of the inflammatory shaft. This option comprises about 80% of all echographic types.

In the second echographic version (the ulcer is located on the anterior and posterior walls of the stomach, less frequently on the large curvature), the area of ulcerous infiltration rises above the level of the ulcerative lesion and protrudes into the cavity of the stomach; its size many times exceeds the size of the ulcerative lesion.

The third echographic type of ulcers of the stomach is characterized by the presence of a deep bottom (more than 10 mm) of a niche located at the level of the serous membrane or slightly higher, a small extension of the ulcerous infiltrative shaft, which is not more than three times the size of the ulcerative lesion. Such ulcers are located in the cardiac department, on the small curvature of the body, angle and antrum of the stomach, and accompanied by an enlargement of regional lymph nodes located perigastrally and / or in small omentum.

The fourth echographic type of ulcer has a tumor-like type and is characterized by asymmetry of the peripheral edges.

The fifth echographic type of ulcer is circular, is better diagnosed in the study on an empty stomach; is characterized by reduced echogenicity in the ulcerative infiltration and is always distinguished by the brightness of the symptom of the “affected organ”. Such ulcers are localized in the pyloric pdepartment of the stomach; on transverse sections of the pylorus infiltration is asymmetric: maximally thick near the niche and minimally on the opposite side.

Ultrasonography in *penetration of gastric ulcers*. It is very important to evaluate the depth of the ulcer and to diagnose the ulcer penetration. Penetration of the ulcer is diagnosed in the case of finding the bottom of the ulcer outside from the extrapolated outer contour of the gastric wall. Localization of the bottom of the ulcer allows to determine on the ecchogram the adjacent organ or structure with penetration. The echogenicity of the penetration channel is elevated, with hypoechogenic zone around it, which corresponds to inflammatory infiltration. Color Doppler mapping and energy Doppler sometimes determine gastric vessels near the bottom of the ulcer, which allows to some extent to assess the risk of stomach bleeding. Penetration of the ulcer is often accompanied by an increase in adjacent regional lymph nodes along the small or large curvature.

Ultrasonography in *perforated gastric ulcer.* Contrast examination of the stomach should not be performed in suspected perforation of the wall. Perforation of the gastric wall is associated with a small amount of free fluid in the abdominal cavity. In this case, the detection of gas between the liver and the diaphragm in the upright position of the patient may also indicate a perforation of the stomach wall.

Ultrasound in *polyps of the stomach*. Echographic polyps are visualized as steady preserved local formations of the mucous membrane protruding to the lumen of the gastric cavity. The form of

polyps on the echogram may be round, oval, oval-rounded, and irregular. Ultrasonography evaluates their size, length of the leg, surface and internal echo-structure. The minimum size of a polyp that can be detected in ultrasonography is about 4 mm. In typical cases, the structure of polyps has minimal heterogeneity, echogenicity is moderately elevated, the surface is smooth and the form is regular. In cases of marked heterogeneity of the structure, rough surface or absence of leg formation it has to be differentiated from a malignant tumor. During a peristaltic wave polyps on the leg resemble balls of ping-pong, they seem to "jump", are in a constant motion, limited by the length of the leg. Polyps are typically associated with a layered-structure of the gastric wall near the base of the polyp. The thickness of the wall of the stomach in the region of the base of the polyp varies within 6-8 mm.

Hyperplastic polyps have an equal surface and reduced echogenicity. ***Adenomatous polyps*** have irregular shape, uneven surface and increased echogenicity.

Polyps of a hemispherical or spherical shape on a broad basis must be differentiated from *leiomyomas*. The latter have a reduced echogenicity, a connection with the muscle layer and are delimited with the cavity of the stomach by echogenous submucosal layer. In the absence of malignant polyps, the regional lymph nodes of the stomach are usually not enlarged.

Color and energy Doppler study allows to determine the nature of vascularization of polyps for differentiation with malignant formations. Typically, vascularization of polyps is reduced, and there are isolated colored locuses.

Ultrasound in *malignant lesions of the stomach.* All variants of malignant tumor of the stomach ***(endophytic, exophytic and mixed growth)*** in ultrasound on an empty stomach appear as a symptom of the affected hollow organ, which often has an irregular geometric shape. Peristaltic waves in the affected wall of the stomach are not traced. The wall of the stomach in the area of the lesion has a reduced echogenicity, a heterogeneous structure, with the presence of hyperherogenic inclusions. On average, in each third case, there is organic stenosis of the pylorus.

There are *9 echographic variants of cancerous lesion of the stomach:* 1) circular - on a transverse section the tumor has a shape of a kidney, has a hypoechoic peripheral, hyperhogenous central part; 2) tumor infiltration has a thickness of up to 6 mm, without clear contours; 3) tumor infiltration has a thickness of up to 6 mm, with a rounded edge and a small length (up to 80 mm); 4) tumor infiltration with a rounded edge, thickness up to 20 mm, length more than 50 mm, more often with a large area of the ulcer; 5) externally resemples a chronic ulcer in the active stage, with a shallow (up to 10 mm) niche in the center of the image, rounded peripheral edge of tumor infiltration in the thickness of about 10 mm, the length of tumor infiltration 30-50 mm; 6) ulcer-like, but with a sharp or indeterminate peripheral margin, a length of tumor infiltration of 45-75 mm, a maximum thickness of up to 15 mm, a diameter of the ulcer lesion of up to 10 mm, the presence of a deep ulcer that reaches the serous membrane of the stomach; 7) erosive, noticeable thickening of the stomach wall is not observed, manifested by the absence of the mucous layer at the site of lesion of the gastric wall; 8) exophytic protrusion into the lumen of the stomach of an area of hypoechogenic infiltration with a flat surface; 9) polypoid, protrudes into the lumen of the stomach, has large dimensions (up to 200 mm), with areas of ulcer of the surface, thickening of the wall of the stomach on the basis of the tumor.

The main advantage of ultrasonography is the possibility of layer-level visualization of the gastric wall, which makes it possible to estimate the depth of tumor invasion, in particular muscular and serous layers. In germination of the tumor into the serous membrane, its outer contour becomes uneven, blurred and jagged.

Differential diagnosis of cancer and ulcers of the stomach is associated with a typical and reliable sign of the tumor nature of the ulcers of the stomach, namely the presence of a rounded, hanging peripheral edge of the ulcerous infiltrative shaft, the location of the lower point of the ulcerative lesion above the interpolated line of the inner contour of the gastric mucosa by 5-6 mm and more, a significant excess (8-10 times) of the size of the ulcerative lesion itself by the size of the ulcerative infiltration. Local thickening of the mucous layer with a decrease in echogenicity and a jagged edge can also indicate a malignant process. Gastric cancer can also be suspected in erosion with a continuous large length. In localization of the tumor in the upper part of the body, the bottom and the cardiac department, it is necessary to evaluate the condition of the wall of the abdominal part of the

esophagus. In germination of the tumor into the transverse colon, the symptom of the affected hollow organ is determined below the stomach, in accordance with the location of the intestine. Development of ascites in gastric cancer indicates the spread of tumor to the peritoneum. Relapses of tumor often result in pathologically enlarged lymph nodes and metastases to other organs. Cancer of the pylorus often leads to the development of stenosis of the pyloric canal.

When differentiating large polyps with a broad base and cancer, it is necessary to take into account thickening of the gastric wall in the region of the tumor base in its malignant nature.

Ultrasound in *duodenogastric reflux*. Duodenogastric reflux is diagnosed in about half of the cases of disorders of the pyloric department and it is 2 times more often common in women than in men. In ultrasonography, it is visualized in the form of a hyperechogenic beam moving from the pylorus toward the antrum of the stomach. In patients with reflux the filling of the stomach is evenly distributed, the tone is preserved; the peristalsis in most cases is intensified. In 30% of cases, duodenogastric reflux begins with the contrast of the stomach, while in others it occurs in 5-7 minutes.

Duodenogastric reflux is evaluated by the frequency and height of the beam column: with a frequency of up to one per minute, extending to the angle of the stomach, it is considered to be weak; 2-3 per minute and also reaches the angle of the stomach - moderate; more than 3 per minute and is located to the body of the stomach - severe.

Ultrasonography in *gastroesophageal reflux and esophagitis*. Qualitative visualization of the pyloric abdominal part of the esophagus shows a retrograde stream of stomach contents into the esophagus in the form of a column of hyperhogenic inclusions, namely acoustic shade bubbles. The image of the gastroesophageal reflux is better in the cinematographic mode, as well as in color Doppler. **Chronic esophagitis** in ultrasonography is manifested by uneven thickening of the mucosa, decreased echogenicity of the abdominal part of the esophagus.

Ultrasonography in *Menetrier's disease or hypertrophic gastritis.* Examination of the stomach without *filling with fluid* in the area of lesion shows a significant uneven thickening of its walls. In the transverse section, in similar lesions of hollow organs, the echogram shows that the central region of the formation has increased echogenicity, and peripheral region reduced echogenicity and resembles a slice of the kidney. Therefore, the circular lesion of the intestine or antral section of the stomach forms a "symptom of a false kidney" or "affected hollow organ", which is more typical for infiltrative tumors, but the term can also be used for chronic gastritis. On the background of the fluid, there are enlarged folds of the gastric wall due to hypertrophy of the mucous membrane and submucosal layers.

Functional and instrumental methods of examination in surgical diseases of the duodenum

Comprehensive examination of the duodenum

Under X-ray control, the patient is administered a probe for a comprehensive study of the stomach and duodenum. Its olives must be in the acid-forming, neutralizing zones of the stomach and in the duodenum, and the olive for collecting bile and pancreatic secretion at the level of the major papilla of the duodenum. pH-metry of the stomach is carried out in the way described above. The obtained data on pH in the stomach and duodenum can also be monitored for the evacuation of acidic contents from the stomach. Upon completion of examination of the stomach, the patient should lie on the right side. In this position, bile and pancreatic secretion are collected and examined by laboratory methods (microscopy, determination of enzymes, etc.).

X-ray diagnosis of surgical diseases of the duodenum

Basic techniques. In parallel with examination of the stomach, and in more detail after it, duodenum is examined by its artificial contrasting at different degrees of filling. Mandatory requirement is palpation, dosed compression and study of the shape, contours, relief of the upper, ascending and lower horizontal parts of the duodenum. The filling of the intestine can be short-term, so it is necessary to catch its anatomical-morphological and functional features and record them. If evacuation from the stomach is complicated due to spasm of the pylorus, then the following methods are used to evacuate the contents of the stomach (barium suspension):

1) artificially “push” the contrasting substance with the jerk movements of both hands simultaneously;

2) in 10-15 minutes carry out repeated examination after the initial one;

3) relieve spasm of the pylorus by injecting 1 ml of 0.1% atropine, 1-2% solution of dibasol or 2-3 tablets of aeron under the tongue. After oral or parenteral administration of the pharmacological agents, the study is continues after 15-20 minutes.

Additional techniques. *Duodenography in artificial hypotonia*. Duodenography is carried out in conditions of artificial reduction of the tone of the duodenum in order to prolong the filling of the lumen with a contrast agent: 1) by intravenous administration 1 ml 0.1% solution of atropine and 5-10 ml of 10% solution of calcium chloride and calcium gluconate; 2) by subcutaneous or intramuscular administration of 2-4 ml of 0.1% solution of metacin or 1 ml of 0.1% solution of atropine in combination with parenteral administration of 10-20 ml of 10% solution of novocaine; 3) by taking 2-3 aeron tablets under the tongue.

Probe duodenography in artificial hypotension. The patient swallows the probe with an olive up to a specific label with X-ray control. In 10-15 minutes the patient is put on a trochoscope and 200-250 ml of a liquid contrast medium is injected into the duodenum using Janet’s syringe through the probe, obtaining a uniform, compact filling. The study is carried out in different projections. To study pneumorelief of the mucous membrane and a more detailed study of the medial wall of the intestine, 200-250 cc of air is injected into it and radiography is repeated. After that, the probe is removed, the patient is transferred to the vertical position and continues to study the relief, position and contours of the intestine. The area of the large duodenal papilla is fixed on target roentgenograms.

Probeless duodenography (sparing) in conditions of artificial hypotonia. Hypotension of the duodenum is caused by two tablets of aeron under the tongue. After absorption of tablets in 15-20 minutes the duodenum is examined in its tight filling. X-ray is conducted in various projections. In case of superposition of the stomach and duodenum shadows the stomach is displaced by a compressor. In order to obtain a picture of pneumorelief of the duodenum in conditions of hypotension, the patient is administered a gas-forming mixture. The gas entering the lower part of the duodenum moves the contrast medium into the lower parts of the small intestine. Thus, it is possible to obtain a clear pneumorrelief of the mucous membrane, and the detected changes are fixed on the radiographs.

Angiography. The superior and inferior pancreatic-duodenal arteries take part in the blood supply of the duodenum, which are branches of the gastro-duodenal and superior mesenteric arteries. Indications for angiography are the clarification of the nature, localization, the extent of the tumor and the depth of germination of the organ and surrounding tissues, as well as differential diagnosis.

Interpretation of X-ray findings

X-ray diagnosis of *duodenal ulcer* has main (“niche”, inflammatory shaft and convergence of the folds of the inflammatory membrane) and auxiliary (enhanced motility and local spasm of the intestine, deformation of the organ wall, accelerated evacuation of the barrier mass from the stomach and its rapid passage along the duodenum and upper loops of the small intestine, etc.) radiological signs. *Symptom of “trefoil”* is a scar deformation of the duodenal bulb, which occurs as a result of multiple relapses with subsequent scarring.

Examination of the duodenal bulb in conditions of double contrast shows that the ulcers in it rarely reach out to the relief of small or large curvature, and in most cases are located on the anterior and posterior walls. Unlike lesion of the stomach, even acute ulcers of the bulb are almost always accompanied by its deformation and pronounced changes in its lining (broad and rigid ridges), and chronic ulcers are usually found on the background of uneven contours of the organ as a result of scar deformation and active periduodenitis. Duodenal ulcers, located distal to the bulb, in the acute phase are often accompanied by a narrowing of the corresponding department.

Malignant tumors of the duodenum and the large duodenal papilla

Malignant tumors of the duodenum located outside the papilla are more common than benign neoplasms. Tumors in the region of large duodenal papilla and ascending part of the intestine are slightly more common. **Ordinary X-ray examination of the stomach and duodenum** occasionally

reveals morphological changes in the area of the intestine outside the bulb in the form of narrowing of the lumen or atypical relief of the mucous membrane in the area of tumor location. There are the following types of impaired peristalsis: increased spastic peristalsis occurs when in hindered advancement of the contrast mass through the narrowed area of the intestine and, alternatively, sharply weakened peristalsis with the stagnation of the barium suspension in the pre-tumor zone of the duodenum and in the enlarged stomach.

Relaxation duodenography has a decisive factor in the diagnosis of duodenal cancer.

Exophytic form of cancer is characterized by a filling defect with uneven contours, deformation of the relief of the mucous membrane, rigidity of the walls of the duodenum. In some cases, the intestinal lumen in the area of cancerous lesion is somewhat expanded due to retroperitoneal, periduodenal proliferation of the tumor process. Functional impairments in exophytic forms of cancer are manifested by the absence of peristalsis in the tumor site, slowdown in the progression of the contrast medium in the intestine, and also by the above signs of functional impairment of the stomach.

Endophytic cancer of duodenum is radiologically manifested by asymmetric ring-like narrowing of the intestinal lumen in a limited area in the form of the so-called napkin ring. Infiltration of the intestinal wall is accompanied by the absence of peristalsis, rigidity and deformation of the relief of the mucous membrane. The suprasthenic expansion of the duodenum can be significantly expressed (the narrowed region reaches 5 cm in length and has uneven, underlined contours).

Sarcoma of the duodenum is much less common in cancerous tumors. The world literature has described just over 100 cases. These tumors affect the lower horizontal and ascending part of the intestine closer to the duodeno-jejunal fold and have the following radiological signs:

In the early stages of the disease, the tumor appears in the form of a small regional symptom of half-shadow in the area of Vater papilla. The marked stages of cancer of the large duodenal papilla appear to be large in size with a defect of filling with clear polycyclic contours.

- there are single or multiple defects in the mucous membrane with a diameter of 0.3-1.5-3.0 cm;

- the folds are deformed at the site of the injury, rigid, enlarged, irregularly located near the tumor; stenosis of the duodenum with its obstruction and with enlargement of the upper sections.

A cordlike band may be determined on the site of stenosis of the intestine. Barium mass lingers in the stomach and in the duodenum for a long time; the intestine is sharply obstructed and the barium suspension is ejected along with the vomiting masses.

Malignant tumors of the large duodenal papilla make up 5-18% of all tumors of the pancreatic-duodenal zone. In expansive growth, they are characterized by signs of exophytic neoplasms. Relaxation duodenography detects a tumor in the form of a defect of filling with a diameter greater than 1 cm, with equal, clear contours, localized on the medial wall of the lower part of the duodenum. In the early stages of the disease, the tumor has the form of a small regional symptom of half-shadow in the zone of location of the major papilla. The marked stages of cancer of the major duodenal papilla appear to be large in size with a defect of filling with clear polycyclic contours. The surface of the tumor of the major papilla can be impregnated with grains of contrasting substance. The shape of the filling defect may be wedge-shaped, crescent, spherical or lobulated. The mucous membrane of the tumor opposite to the wall of the duodenum is not destroyed by neoplasms, occasionally there is a deviation of the surrounding tumor of the unaffected folds of the mucous membrane along arcuate lines. Relaxation duodenography in advanced stages of the tumor of the major duodenal papilla shows changes in the entire duodenum and antral portion of the stomach as a result of the spread of the tumor process to the head and body of the pancreas.

Benign tumors of the duodenum and major duodenal papilla. The world literature describes about 350 cases of benign tumors of the duodenum. Benign tumors include adenomatous polyps: brunneroma, leiomyoma, lipoma, neurinoma, neuroma, hemangioma, and others. Benign tumors of the duodenum can be localized in the bulb or in any other part of the intestine. X-ray examination shows that these tumors have the form of expansive formations, which cause growth on the surface of the mucous membranes and give a clearly limited defect of filling in the lumen of the duodenum; in some cases there are niches on the surface of the defect, ulceration of the tumor, accompanied by bleeding.

Benign tumors have the form of limited nodal formations that grow into the lumen of the intestine or towards the abdominal cavity.

Polyps of the duodenum can be both single and multiple. Such groups of polyps are more common in the bulb or outside the bulb in the upper part of the duodenum. Multiple polyps in the bulb have the form of rounded filling defects with clear contours of 1-2 cm in the diameter and preserving the elasticity of the intestinal walls in the area of the polyps.

Differential diagnostics of tumors of the duodenum and of the major duodenal papilla should be performed with aberrant pancreas, ulcer of the duodenum outside the bulb, compression of the intestinal wall with an enlarged gallbladder or enlarged pancreas in chronic pancreatitis and cancer of the head of pancreas.

Aberrant pancreas in the form of a semicircle covers the lower part of the duodenum in the front, inside and outside on a section up to 4-5 cm long. The walls of the intestine at the site of compression remain elastic, the contours are smooth. Diagnosis can be crarified by the contrast of the ducts of the aberrant pancreas in endoscopic pancreatography.

Extrabulbar duodenal ulcer is more often localized in the upper horizontal part or in the upper half of the intestine. Penetrating extrabulbar ulcers are characterized by a deep niche with an inflammatory shaft and a spastic retraction of the opposite wall of the duodenum. The use of relaxation duodenography results in cessation of spastic contractions and it becomes apparent that it is a deep ulcer, not circular infiltration.

An algorithm for X-ray examination of the duodenum: 1) the first stage - the main radiological methods of investigation and images in the front straight and right front projection in the vertical, horizontal position of the patient, in the latero-position, anterior and posterior projection on the left side; 2) the second stage - relaxation duodenography (with and without probe) in the positions mentioned above, in conjunction with intravenous cholangiocholecystography; radiocinematography mainly in the upright position in the anterior straight, right anterior and oblique projections; 3) the third stage - angiography.

Duodenoscopy in surgical diseases of the duodenum

Duodenoscopy *is a visual examination of the cavity and mucosa of the duodenum with diagnostic manipulations using a fibroduodenoscope.*

Indications: duodenal bleeding, suspected perforative duodenal ulcer, foreign bodies, mechanical jaundice, suspected tumor of the major duodenal papilla, biliary duct stricture, cholecystopancreatitis, complicated diagnosis of duodenal ulcer, dyskinesia of the duodenum, etc.

Contraindications: acute cerebrovascular accidents, acute coronary circulatory disorders, preagonic and agonic conditions, phlegmonous esophagitis, chemical burns of the esophagus.

Method. The patients should lie on the left side, and for a thorough revision of the major duodenal papilla and biopsy taking on the abdomen or right half of the body.

Duodenal bleeding. Duodenal bleeding is most commonly associated with the following diseases:

1) acute duodenal ulcer;
2) chronic duodenal ulcer;
3) erosive duodenitis;
4) cancer of the duodenum;
5) cancer of the major duodenal papilla;
6) polyps and submucosal tumors of the duodenum;
7) diverticula of the duodenum.

Rarely, there are bleedings caused by cancer of the pancreas head and diseases of other organs and systems.

Acute duodenal ulcer is most often located in the bulb of the duodenum and has a characteristic endoscopic pattern. The bulb is of the usual shape and size, gets smoothed out fairly well. The ulcer is single in most cases, is on even mucosa without scar deformation (in some cases, against a background of limited or diffusive hyperemia of the mucous membrane), the ulcer has a regular round or oval shape in the size of 1 cm. The edges of the ulcer have an appearance of high,

smooth, swollen, hyperemic roller. The bottom of the ulcer is covered with whitish fibrin, blood clots and thrombus. Bleeding vessels in acute ulcer are, as a rule, not detected. Acute ulcer may be accompanied by an erosive bulbitis. In bleeding, the endoscopic pattern is characterized by the pallor and slurred color.

Chronic duodenal ulcer is localized on the background of scar deformation and wrinkling of the bulb and rarely reaches the size of the gastric ulcers. Chronic ulcers of the bulb have almost no rough, callous underlined edges. Most often mucous membrane is corroded in the area of scar deformation or "niche", which moves towards healthy mucous membrane and leaves behind a scar fold. In bleeding, the bulb is filled with red blood or a loose red clot. Reflux of bloody contents in the stomach is the sign of the localization of bleeding in the duodenum.

Erosive duodenitis is in most cases localized in the bulb and the initial postbulbary divisions of the duodenum. Erosions in the bulb are of large size and are fused against the background of hyperemic mucous membrane. Erosive lesions are often associated with acute or chronic duodenal ulcers.

Cancer of the duodenum is rare and has an endoscopic pattern that resembles the infiltration-ulcer form of gastric cancer. The tumor has a pineal form and protrudes into the lumen of the intestine, causing early deformation and narrowing of the lumen. In the initial stages the tumor is dense; mucous membrane is restrucrured, with impaired relief, inanimate grayish color. Instrumental palpation shows that the mucous membrane is inactive, rigid. Destructive changes in the form of ulcers and erosions develop in far-advanced stages. With the development of destructive changes, the tumor is a source of bleeding, covered with blood or clots, is easily bleeding when in contact with the fibroscope.

Cancer of the major duodenal papilla is very important as a cause of mechanical jaundice, but it is often a source of bleeding. The main endoscopic signs of cancer of the duodenal papilla: an increase in its size, changes in the relief and color of the mucous membrane, development of ulcer(s) on it; corroded initial department of the ampullary part, papillary hyperplasia of the mucous membrane of the initial part of the ampullary part and infiltration of opposite walls. In some cases, the mucous membrane of the initial department of the ampullary part of the major duodenal papilla acquires jelly consistency. The risk and incidence of bleeding increase significantly in the recurrence of cancer after papillectomy. Bleeding becomes prolonged and recurrent.

Relapse of cancer of the major duodenal papilla has the appearance of a flat, large ulcer-like defect of the mucous membrane. The bottom and edges are uneven. The edges look like a low pale roller.

Polyps and submucosal tumors of the duodenum are endoscopically similar to gastric ones. They are the cause of bleeding in cases of erosion and ulcers on them.

Diverticula of the duodenum are more often localized in the vertical section on the medial wall in the parapapillary region. They are the source of bleeding in erosions and ulcers on their surface and when they penetrate into adjacent organs. On X-ray examination with endoscopic control duodenal diverticulae often have the appearance of diverticulum pockets as a result of scar-ulcerative deformation of the bulb.

Foreign bodies of the duodenum, especially long and sharp, are fixed in the region of the upper and lower duodenal flexures. Diagnosis involves complex X-ray endoscopy.

Perforated ulcer of the duodenum is characterized by a deep cylindrical form due to the spastic shortening of its edges, which practically do not straighten out. The perforation is not always possible to detect and inspect visually. The perforation site has the form of a bottom defect with torn edges. In complicated detection of perforation site, the bottom of the ulcer is irrigated with sterile solutions and cautiously palpated with a catheter. In addition to the above signs there are indirect signs of perforation, namely: spastic contractions of the pylorobulbar division, vigorous peristalsis of the bulb and postbulbary divisions of the duodenum. The mucous membrane of the initial part of the duodenum is inflamed, swollen and is clearly hyperemic. Absolute sign of perforation is free gas in the abdominal cavity after duodenoscopy.

Differential endoscopic diagnosis involves endoscopic detection of urgent diseases directly in the upper gastrointestinal tract or indirect (side) signs of urgent diosrders of surrounding organs.

Acute pancreatitis is characterized by inflammatory changes of the mucous membrane of the stomach in the area of the posterior wall. Folds are dilated; thickened mucous membrane is hyperemic, edematous, with pinpoint intramucosal hemorrhages. In destructive processes in the pancreas the wall of the stomach is sharply rigid, protrudes into the lumen, the relief is "stiff". In purulent abscess, formed in the pancreas, the gastric wall is deformed in this area; the mucous membrane forms high, infiltrated, densely located folds. Ulcer or fistula of the abscess of the pancreas may open on this site, which itself protrudes into the cavity of the stomach or cavity of the duodenum, with a purulent-hemorrhagic discharge. In this situation, the fistulous passage should be used to receive suppurative content in order to detect microflora and antibiotic susceptibility, as well as for local treatment through an endoscope.

Acute cholecystitis is accompanied by uniform hyperemia and swelling of the mucous membrane in the lower parts of the stomach with pinpoint hemorrhages. Dyskinetic manifestations in the duodenum and reflux of duodenal contents in the stomach are typical. The astral department of the stomach is sometimes deformed due to perigastritis.

Laparoscopy in surgical diseases of the duodenum

Laparoscopy (peritoneoscopy) is a revision of the abdominal organs and diagnostic interventions using a laparoscope.

Indications. Laparoscopy is indicated in particularly difficult cases when physical, radiological and laboratory methods of examination do not give a possibility to conduct a differential diagnosis among a number of diseases: parenchymal and mechanical jaundice, cirrhosis and neoplasms of the liver, tuberculous and cancerous lesions of the omentum and peritoneum, various tumors and parasitic diseases of abdominal organs, tumors of the uterus and appendages, etc. The method is valuable for early detection of disorders of hollow and parenchymal organs. Under the control of a laparoscope it is possible to perform biopsy, liver puncture and splenoportography.

Contraindications include decompensated heart defects, hypertension, emphysema, bronchial asthma, acute inflammatory diseases of the abdominal organs, diaphragmatic hernia, pustular skin lesions.

Laparoscopic examination of *perforated gastric and duodenal ulcers* is characterized by a complex endoscopic pattern. This is due to the flow of gastro-duodenal contents in the abdominal cavity distant from the main pathological process. Inflammation in the abdominal cavity develops depending on the amount of volume of the gastro-duodenal contents, which entered into the department of the abdominal cavity. In this situation it is necessary, first of all, to exclude disorders of the appendix. In the area of the perforation of the ulcer, inflammatory-infiltrative changes and fibrin overlay are more evident than in the area of the right flank of the abdominal cavity. If it is difficult to determine the pathological focus in the abdominal cavity, it is necessary to conduct a joint gastro-laparoscopy (gastro-laparoscopy is contraindicated in persons with respiratory and cardiac insufficiency, severe concomitant diseases and severe adhesion process in the upper abdominal cavity).

Ultrasonography of the duodenum

Ultrasonography of non-tumor duodenal ulcer. The diagnosis of duodenal diseases is based on the knowledge of anatomy, physiology and the ability to receive images of the bulb and postbulbary department using various techniques. After receiving the image of the antral department of the stomach, the pylorus, duodenal bulb, it is necessary to monitor for several minutes to record the peristaltic waves and to better evaluate the layers of the wall. Normally, the wall of the duodenum has the same five-layer structure as the stomach. On the echogram, the first echogenic layer of the cavity corresponds to the border "fluid-wall-intestine", the second hypoechogenic one to mucous membrane with its own muscular plate, the third echogenic one to submucosal layer, the fourth hypoechogenic one to muscular membrane, the fifth echogenic one to serous membrane. After filling the intestine with fluid, it is possible to visualize the circular folds of the intestine. With passage of peristaltic wave the thickness of the wall increases and the layers are visualized better.

Ultrasonography of ulcer of the duodenal bulb involves direct visualization of the ulcer defect and infiltration around the ulcer. Ultrasound signs of duodenal ulcer are less evident than in gastric ulcer. The duodenal ulcer has the same echographic signs as in gastric ulcer, namely*: inflammatory*

shaft around the ulcer defect (has a decreased echogenicity), *a niche* (has increased echogenicity), and *infiltration around the ulcer* (has a decreased echogenicity).

Spasm is a frequent impairment of the motor and evacuation function of the duodenum. It can be an independent condition or indicate a peptic ulcer. In spasm lasting for several minutes, the state of the wall of the duodenum does not change, the peristaltic wave is not observed.

In ***deformation of the duodenal bulb*** the external shape of the organ does not change, but the layered structure of the wall is preserved.

Duodenitis is characterized by thickening and decreased echogenicity of the wall of the duodenum, with preserved integrity of each layer.

Erosions of the duodenal bulb are characterized by a discontinuity of the mucous layer, uneven, slight thickening, preserved differentiation of the layers, presence of depressions on the inner surface of the intestinal wall.

Color Doppler examination in the period of active inflammatory process shows increased blood flow.

Methods of examination in surgical diseases of the small and large intestine, rectum

X-ray methods of investigation of the small intestine

Significant length of the small intestine, variability of its position, mobility, accelerated passage and projection of individual loops create difficulties for a detailed study of this part of the digestive traact.

Basic X-ray techniques. Preparation of the patient: in the evening before and in the morning on the day of examination, the patient should not take food. The commonly used method of studying the small intestine has several options:

1) after examination of the stomach and duodenum, the patient undergoes radioscopy and radiography of the small intestine every 15 minutes before the transition of the contrast medium into the cecum;

2) in 30-40 minutes after examination of the stomach and duodenum, the patient receives the second portion of the contrast agent and lies on the right side; radioscopy is performed every 30 minutes in the vertical and horizontal position of the patient; lateral position is used to diagnose displacement of loops;

3) small intestine is examined in 2-3 hours after taking the contrast substance.

Accelerated advancement of the contrast agent is achieved by the following methods: 1) the patient drinks 200 ml of contrast medium (barium suspension) and the same volume of cooled saline solution; in 10 minutes, he drinks another 200 ml of cold (ice) saline; 2) after taking contrasting substance, the patient is offered to drink a glass of cold milk; 3) the patient swallows fractionally (in small portions) the contrast agent on “ice water”; 4) in order to increase the motility of the small intestine, the patient is subcutaneously administered 1 ml of 0.005% solution of proserin or takes 1-1.5 tablets of physostigmine (0.001) per os. The technique of artificial acceleration of the passage is recommended for the study of patency. It is not designed to study the relief of the mucous membrane, because the used agents and food ingredients lead to the accumulation of fluid in the lumen of the intestine and deformation of the folds of the mucous membrane.

In suspected obstruction, the patient is prescribed water-soluble contrast agents (gipak, verografin, urografin, etc.) in the amount of 20-40 ml diluted with 60-80 ml of physiological solution or glucose.

X-ray examination *in intestinal obstruction (level of the small intestine).* X-ray study (without the use of contrast agents) in intestinal obstruction detects gas and fluid levels in the loops of intestine, described by N. Kloiber (1919). X-ray examination should be performed by a radiologist. X-ray examination should be followed by obtaining radiograms (information on the picture and the ability to investigate the time course of the process). Any obstruction of the small intestine (adhesion, volvulus, obturation) is characterized by liquid levels with gas above them, elongated in the horizontal direction and located in the inflated loops of the small intestine in mesogastrium and small pelvis. The relief of the mucous membrane of the small intestine in obstruction is characterized by a transverse striatum resembling a spring (due to swollen and increased Kercking folds) - *Fermental’s symptom*.

To determine the type and location of obstruction the patient is offered to drink 150 ml of barium sulphate suspension and examination of the intestine is conducted in the follow-up with an interval of 1-1.5 hours. During the study, it is necessary to follow the advancement of contrast in the digestive tract (normally the contents of the stomach gets into the intestine in 4-5 hours). Detection of barium sulphate after a specified time in the large intestine eliminates obstruction of the small intestine. Accumulation of barium sulphate in the place of an obstacle in the area of the small intestine suggests obstruction of the small intestine.

Additional methods. *Relaxation (hypotonic) enterography or probeless enterography.* After roentgenologic examination of the stomach and the initial parts of the small intestine, the patients is administered an intravenous injection of 1-2 ml of 0.1% solution of atropine and 5-10 ml of 10% solution of calcium gluconate or calcium chloride. Within 10-15 minutes, the patient lies on the couch, periodically turning around the longitudinal axis of the body, taking a gas-forming mixture. Radioscopy and radiography are carried out in horizontal and vertical positions in the anterior, posterior direct projections and in lateral position. This method can be used to determine the elasticity of the walls, to study the lumen and the position of the intestine.

Probe enterography. The patient is prepared in the same way as for the usual examination of the digestive tract. The patient is administered a three-meter intestinal probe consisting of 2 paired tubes into the terminal loops of the ileum. The advancement of the probe through the small intestine is evaluated by the feeling of "dragging" it inside and discharge of chymus. The position of the main end of the probe is specified radiologically. When the probe gets into the terminal part of the ileum, the same agents are used as for relaxation duodenography. When hypotonia is achieved, the small intestine is gradually filled with a contrast medium with a Janet's syringe. Portions of 20-25 cm are sequentially investigated, for which 30-50 ml of contrast material are introduced and the relief of the mucous membrane is studied, and 100-200 cm^3 of air is injected to receive pneumatic relief. For tight filling of the colon with a contrast medium, 1 liter of barium suspension is used.

Retrograde hypotonic ileography. The patient is administered relaxants in the same way as in hypotonic duodenography (atropine). Contrast substance is administered into the colon with the help of an enema, seeking to open the ileocecal valve and maximizing the filling of the terminal portions of the small (iliac) intestine, which is conveniently investigated in the patient's horizontal position and in the lateral position. After emptying the colon from the contrast agent, air is injected to study pneumatic relief. Indications and contraindications are similar to those in duodenography in conditions of artificial hypotonia.

Radiocinematography. The principle of the method is described above.

Artificial pneumoperitoneum combined with pneumojejunoileography. This method is used to study the thickness of the walls of the small intestine.

Angiography. Arteries of the small intestine in the amount of 5-7 branch from the left surface of the trunk of the superior mesenteric artery.

Aarteries of the small intestine can be contrasted by catheterization of the trunk of the superior mesenteric artery and by direct probing of separate branches - ileac, colic, jejunal arteries (superselective angiography). Upper mesentericography is carried out by a special catheter, the distal end of which is modeled accordingly (curved at an angle of 90°). The catheter is inserted punchingly, according to Seldinger, into the femoral artery, and then pushed forward in the ascending direction through the arterial vessels in the aorta to the level of the upper edge of the first lumbar vertebra. With special manoever the distal curved end of the probe is inserted with the proximal end into the mouth of the superior mesenteric artery. The location of branching of the superior mesenteric artery is sought along the anterior wall of the aorta; the moment of insertion of the catheter tip into the necessary vessel is determined by its characteristic deviation from the axis of the aorta forward. 40-50 ml of contrast iodine agent is injected and images are taken in such a way as to register the arterial, capillary, parenchymatous, and venous phases of the intestinal blood flow. For this purpose, the first 2 images are taken at intervals of 0.5 second in 2 seconds from the beginning of the injection, the following 2 images at an interval of 1.5 second and the last 3 images at the end of the series at an interval of 3 seconds.

Superselective angiography. Superselective contrast of the separate branches of the superior mesenteric artery is performed with the **"Cobra"** type catheter. 1 ml of No-spa diluted in 10-15 ml of physiological saline is provisionally administered into the vascular bed (7-8 minutes prior to administration of a contrast medium). Contrast substance in a dose of 25-30 ml is administered for 2.5-3 seconds. Intravascular manipulations are facilitated by insertion into the lumen of a catheter of a special **"Cordis"** conductor with a long (7-8 cm) and soft distal end. Serial images are taken in 1.5-2 seconds from the beginning of the introduction of contrast agent according to the program, designed for registration of all phases of intestinal blood flow.

An algorithm for X-ray examination of the small intestine.

1. The first stage involves the basic radiological techniques with the help of barium suspension and in combination with "accelerators" of advancement. Optimal projections are direct anterior and posterior in the vertical and horizontal position of the patient.

2. The second stage includes probe and probeless ileography, probe relaxation enterography, pneumoperitoneum in lateral position on the back, right and left side, radiocinematography.

3. The third stage is angiography.

X-ray examination of the large intestine

Basic methods. The study of the colon in urgent practice is carried out both with and without the use of contrast agents.

When performing radioscopy of the patient's abdomen, the patient is taken behind the X-ray screen to determine with the help of exposure to radiation the presence of horizontal levels ("Kloiber's cups" symptom), gas-enlarged intestinal loops, indicating obstruction of the colon. If necessary, radiograms are taken.

Contrasting of large intestine by administrating the contrast medium can only be made conditionally in the section of the main methods. The method is used to determine gross anatomical and morphological changes (displacement, compression, obstruction) and functional state of the intestine.

Preparation of the patient for the study is not required. After receiving 200 ml of barium suspension, the study is carried out in 24-48 hours. In this case, the intense contrast shadow of the large intestine is determined which is filled in along its entire length or on a significant length. Contrast substance taken per os normally enters the cecum in 4-6 hours.

There are several options for the study of the cecum and ascending colon:

1) the first option - the patient takes a contrast agent in the amount of 200 grams in such a way as to trace its progress and to achieve the filling of the cecum and ascending colon;

2) the second option - the patient in the doctor's surgery drinks a cooled barium suspension (it is believed that the temperature factor contributes to accelerating the passage through the small intestine loops), the examination is conducted in the follow-up until the filling of the cecum and ascending colon;

3) the third option - after taking the usual portion of the barium suspension in 10-15 minutes, the patient drinks a glass of "ice water";

4) the fourth option - the patient takes the contrast agent in the amount necessary for the radiological examination of the stomach (200-400 g), after which in 10-15 minutes another 4 g of sorbit, dissolved in 100 ml of water (sorbit, causing hypertonic dyskinesia of the small intestine, contributes to the entrance of the contrast substance into the cecum, ascending, and in some cases, transverse colon in 15-30 minutes);

5) the fifth option - relaxation ileocecography;

6) the sixth option – in signs of obstructed administration of barium suspension (per os or retrograde) or water soluble iodine preparations (20-40 ml diluted with 40-60 ml of glucose), which are "accelerators" of the passage along the small and large intestines.

Any of the above options (optimally the fourth) is combined with the introduction of relaxants. Studies begin in 15-20 minutes after introduction.

Contrast irrigoscopy and irrigography (retrograde introduction of contrast agent through the anus). In case of suspicion of any disease of the large intestine, this method is obligatory.

Contraindications are massive bleeding, severe obstruction, perforation of the intestine, general severe condition.

Irrigography includes: 1) method of "tight filling", 2) method of "double contrast" and 3) method of "triple contrast". The first two are carried out obligatory, the third one according to indications.

Method of preparation of the large intestine for X-ray examination. The main condition for a successful study of the large intestine is the patient's preparation, which involves emptying the large intestine from the contents. In two to three days before the examination the patient should exclude products that contain a large amount of fiber causing gas formation (potatoes, beans, dark bread). In the course of the day, the patient is administered a laxative (30 ml of castor oil), in the evening before and in the morning 2 hours before the study a cleansing enema in the amount of 1000-1500 cm^3 with the addition of 0.5% tannin solution. The examination is carried out on an empty stomach, starting with an observational X-ray in the patient's upright position for the detection of horizontal levels of fluid, extended areas of the large intestine, gas accumulation, and calcifications in the abdominal cavity.

Method of introduction of barium suspension into the large intestine. The introduction of barium suspension in a quantity of 1.5 liters with the addition of 0.5% tannin solution is carried out using an enema in a patient's horizontal position. When filling the large intestine, the patient should lie in the position on the back, right, left side, in oblique projections, on the abdomen. If there is no need to examine the ileocecal valve and terminal department of the ileum, the administration of the contrast agent should be stopped at the moment when it reaches the hepatic angle.

X-ray examination of the large intestine involves a combination of radioscopy, X-ray palpation, compression and X-ray images.

To determine gross anatomical changes in the large intestine and its relationships with surrounding organs, it is necessary to take a revisional image in compact filling of the intestine (the technique of *"tight filling"*), and for a more accurate study of morphological changes, images of a smaller format ("spot"). This completes the first stage of the study.

Further, the patient is asked to evacuate the intestine from the contents and a method of *double contrast* is applied, which is based on the fact that after the large intestine is emptied from the contrast agent, it is filled with air using a *Richardson's cylinder* ("oxygen pad"), not "over-stretching" the intestine, after which X-ray is performed. The method is used to obtain a clearer idea of the internal contour of the large intestine, the elasticity of its walls and the state of the mucous membrane. The method is the final stage of X-ray examination.

According to indications, the third method of *"triple contrast"* is used. After conducting pneumoretoperoperoneum according to Rivas (up to 1000 cm^3 of air), images are taken on the background of "double contrast". The technique is used to determine the thickness of the posterior and lateral walls of the intestine, as well as germination of the tumor in adjacent organs and into retroperitoneal cellular tissue.

Contraindications to parietography of the large intestine and para-rectopneumography of the rectum are general severe condition, local inflammatory processes in the perianal area, severe varicose enlargement of hemorrhoidal veins.

Method of examination of the large intestine in insufficiency of rectal sphincter (M.U. Schniger, 1973). The author proposed a two-channel obturating catheter, the balloon end of which is lubricated with vaseline and injected at the desired height. After that, a balloon that fills the lumen of the intestine is blown, and a contrast medium is introduced through the second canal.

Method of artificial contrast of the rectum and sigmoid colon according to D.M. Abdurasulov involves two stages:

1) introduction of a thin rubber catheter to the level of rectosigmoid junction and filling of the rectum and sigmoid colon with a contrast agent to achieve half-tight filling of these departments of the intestine;

2) examination of relief of the mucous membrane after emptying from the barium suspension by natural defecation.

X-ray examination via colostomy is carried out in the same way as irrigography. The tip or rubber catheter is injected into colostomy. Contrast material fills the upright departments of the intestine. In radioscopy it is necessary to choose the optimal projection for taking images. Preparation of the intestine for this method is carried out according to the usual scheme.

Additional methods. Gas contrast methods. Their essence is to apply one, two or more contrast media.

Pneumocolonography. The introduction of air into the lumen using Richardson cylinder, connected by a rubber tube with an enema tip, followed by X-ray in the forward direct projection in a horizontal, vertical position or in a lateral position on the left and right side. The method is carried out in combination with pneumogastrography and tomography.

Double gas contrast (parietography). It is used to specify the ratio of the affected area of the large intestine with adjacent organs, tissues and the anterior abdominal wall, as well as for more reliable detection of relapses and metastases. Parietography involves introduction of oxygen into the abdominal cavity and air into the lumen of the intestine. The optimal projection for X-ray of the ascending and descending colon is a direct forward lateral position on the left and right side, for the transverse colon direct, anterior one in the vertical position of the patient. The study of the sigmoid colon is carried out in Trendelenburg's position.

Tomography. Imaging by sections is used to clarify the diagnosis, combining it with double contrast. Tomography of the rectum, descending, ascending colon and cecum is best done in the direct posterior projection in the horizontal position of the patient, transverse and sigmoid colon in the position of the patient on the abdomen with a turn in one of the oblique projections.

Computed tomography. This method is described above.

Pararectopneumography (parietography of the rectum) is used to clarify the spread of a tumor or inflammatory infiltrates beyond its walls into the cellular tissue adjacent to the rectum. The method involves introduction of gas into the pararectal cellular tissue, introduction of air and barium suspension in the anterior direct and lateral projections. Preparation of the patient is similar to the one with retrograde introduction of contrast agent. Pneumoperitoneum is introduced after emptying the bladder. The peculiarity of this technique is that examination is carried out immediately after the introduction of gas and that the amount of gas injected varies within 500-800 cm^3.

Triple contrast method. 50-60 ml of barium suspension and air are administered into the lumen of the intestine after introduction of pneumoperitoneum (500 - 800 cm^3 of air). The method is used to determine the thickness of the posterior and lateral walls of the intestine. Contraindications to parietography of the large intestine and para-rectopneumography of the rectum are general severe condition of patients, local inflammatory processes in the paraanal area, severe varicose enlargement of hemorrhoidal veins.

Tomography secondary to double contrast of the rectum is carried out in a direct posterior projection, with the central beam directed to the pubic symphysis. In this case, the optimal layers are those that are 4-5 cm ahead of the coccyx. When tomography is conducted in the lateral projection, it is necessary to distinguish the middle layer, which corresponds to the intergluteal area, and the layers located 1-2-3 cm up and down from the middle.

Methods of angiography. ***Superlelective angiography*** or ***upper mesentericography*** and separate contrast of its branches are used for the study of cecum, ascending and transverse colon (roughly right half of the large intestine). ***Superlelective angiography of the right and median colic arteries*** is carried out using methodical techniques similar to those with superselective angiography of arteries of the small intestine. ***Contrasting of the inferior mesenteric artery with Edman's green catheter.*** The mouth of the inferior mesenteric artery is searched along the anterior-left wall of the abdominal aorta at the level of the third lumbar vertebra. After the end of the catheter has penetrated into the mouth of the vessel, it is inserted into the artery as deeply as possible with light indrawal. This is a preventive measure against the reflux of the contrast agent into the aortic lumen. The correct position of the catheter is controlled by the introduction of a test dose of contrast agent in the amount of 2 ml. Before administering the contrast medium into the artery, air is blown into the lumen of the intestine using Richardson cylinder. 20 ml of contrast medium is administered using a catheter,

automatically with a syringe at a rate of 6-9 ml/s. Serial radiography with a frequency of 2 images for 1 second is carried out according to the program, designed to receive the arterial, capillary and venous phase for contrast of the vascular system. The arterial phase lasts 3-3.5 seconds from the beginning of the introduction of contrast agents, the capillary phase - up to 6-7 seconds and venous - to 14-15 seconds. Altogether 6 radiograms are made in this period of time. Contrasting of the median and inferior rectal arteries is not common due to their weak development in comparison with the superior rectal artery.

An indication for angiographic examination is the difficulty of diagnosis and differential diagnosis of tumors, tumor-like formations of the large intestine, determination of the extent of the pathological process, the study of the nature of branching, the level of branching of the superior and inferior mesenteric arteries, and the state of the regional vessel.

Contraindications for angiographic examination are: general severe condition of the patient, cardiovascular diseases in the stage of decompensation, infectious diseases, intolerance to iodized agents.

An algorithm for X-ray examination of the large intestine

The first stage is the retrograde introduction of contrast agents, radiography before and after emptying, double contrast (barium suspension and gas, water soluble iodine substance and gas) in combination with pneumogastrography in the anterior direct projection, anterior and posterior oblique projections, in the horizontal and vertical position of the patient, as well as lateral position on the right and left side; left anterior projections, lateral projection on the right side in the posterior-anterior beam of rays.

The second stage involves parietography in lateral position on the left and right side, on the back, in the horizontal and vertical position of the patient, in the anterior and posterior projections, lymphography.

An algorithm for X-ray examination of the rectum

1. The first stage is radiography in direct, oblique, anterior projections with semi-tight (tight) contrast or after emptying, double contrast (barium suspension and air, water-soluble iodine substance and air (in the same projections)).

2. The second stage depending on the localization of the process: a) on the posterior wall - para-rectopneumography in combination with tomography in the left or right laateral projections, angiography of the inferior mesenteric artery, penile phlebography; b) on the anterior wall - pericystography in combination with double contrast of ampulla, penile phlebography in the anterior direct and lateral projections, vesiculography, angiography of the inferior mesenteric artery; c) on the lateral surface - pararectopneumography in combination with tomography in the posterior direct projection, penile phlebography, angiography of the inferior mesenteric artery.

Interpretation of X-ray findings in surgical diseases of the small and large intestine, including the rectum

In intestinal obstruction (level of the large intestine). Mechanical obstruction of the large intestine is better detected by irrigoscopy (the amount of contrasting fluid should be at least 1 liter). *Volvulus of the sigmoid colon* is characterized by the symptom of a "corkscrew", which is a zigzag impregnation of barium sulfate in the place of the twisted intestine. *In the presence of formations obturating the lumen of the colon*, the administered contrast medium does not pass beyond the place of the barrier, and in the case of a malignant tumor, there can be corroded or uneven edges of the intestine. In *invagination*, barium sulfate covers the head of invaginate in the form of a claw or half-moon; sagittal placement of invaginates is characterized by enlightenment in the surrounding shadow (*a symptom of a "socket"*). *Functional intestinal obstruction* develops secondary to disorders of intestinal motility. Radiologically this is manifested in the absence of passage of barium sulfate through the digestive tract.

Crohn's disease (terminal ileitis) is associated with spasm and swelling of the intestinal wall, "*symptom of a string*" (a shadow of contrasting substance in the narrowed area of the ileum) and a porous pattern in the absence of stenosis.

In non-tumor (inflammatory) diseases of the large intestine. The most commonly diagnosed pathologies of non-tumor origin of the large intestine include the following diseases: nonspecific ulcerative colitis (NUC), Hirschsprung's disease, diverticulosis of the large intestine

In X-ray examination of NUC by the method of double contrast (*irrigography*), the following features are determined: rapid filling and accelerated evacuation (of barium suspension) from the affected area; reduction of intestinal lumen; lack of gaustration and contour; development of densely located niches and a double contour; in the chronic course: the affected part of the intestine is shortened, narrowed and rigid, there are many border and central filling defects, contours are flat, gaustration is absent.

X-ray examination of the affected intestine in Hirschsprung's disease shows narrowing in the rectum or rectosigmoid division and enlargement of the upper parts of the large intestine.

In irrigography, diverticula of the large intestine are defined as rounded depot of barium suspension that extends beyond the intestine; in double contrast, the tightly filled diverticula are clearly visible on the background of the mucosal relief; diverticulitis is characterized by typical radiological symptom, such as the presence of sharp projections resembling the teeth of the saw; spasm of the intestine and narrowing of the affected area; there is a gradual transition from the unchanged department to the affected area of the large intestine; intraperitoneal abscess is defined in the form of a clearly separated filling defect with the relief of the mucous membrane preserved above it; fistulas are associated with narrow passages located outside the lumen of the intestine and combined with its lumen.

In **tumors of jejunum and ilem.** ***Malignant tumors of the jejunum and ileum*** make up 3% of all malignant tumors of the gastrointestinal tract. Among malignant tumors, cancer and sarcoma occur more often than other tumors. *Exophytic type of tumor growth* is characterized by a rounded form of filling defects with deformation of the folds of the mucous membrane and rigidity of the intestinal walls. In the area of localization of exophytic tumors of the jejunum or ileum, peristalsis of the intestinal wall is not traced while its mobility is preserved. Radiological sign of progression of tumor growth is almost complete immobility of the affected loops of the small intestine. *Endophytic, infiltrating type* of growth is manifested by circular narrowing of the lumen of the jejunum or ileum in a limited area. The infiltration zone is asymmetric in relation to the intestinal axis with a disruption and destruction of the folds of the mucous membrane at the border of the narrowing. Impairment of the intestinal motility leads to the formation of parietal enlargement of the proximally located intestinal loops.

Jejunal and ileal sarcomas develop in persons under the age of 40. *Exinointestinal sarcomas* radiologically form a boundary defect of the filling or broad tumor ulcers. *Endointestinal sarcomas* are characterized by a filling defect in the intestinal lumen; contours of the defect are clear and even. The intestinal lumen may be stretched by a tumor node that exceeds the diameter of the unchanged part of the intestine. ***Lymphosarcoma*** is characterized by an enlargement of the lumen of the intestine with a marginal filling defect against the background of mucosal relief, enlargement of the folds of the mucous membrane, absence of peristalsis in the area of impairment. ***Infiltrative forms of sarcoma*** can be *nodular-polypoid* or *ulcerative-infiltrative*. Diagnosis of infiltrating forms of tumors is the most complicated. *Nodular and polypoid infiltrative* lesions radiologically represent multiple regional or central small defects of filling, characterized by enlargement of the intestinal lumen, impairment of peristalsis, rigidity of folds of the mucous membrane, which take a steady transverse direction. ***Ulcerative-infiltrative lesions*** are manifested by the alternation of constricted intestinal regions due to infiltration by sarcomatous tissue with sites of enlargement. Ulcerative niches in sarcomas of the small intestine have a large length and diameter, which often exceeds 2-3 times the usual lumen of the unaffected segment of the small intestine. *Functional signs of malignant tumors of the small intestine*: slowing down of the progression of the contrast suspension along the small intestine, delayed filling of the small intestine and slowing down of emptying of the jejunum; stenotic tumors located in the initial loops of the small intestine are characterized by enlargement of the lumen of the duodenum, antiperistalsis and reverse delivery of the contrast suspension into the stomach.

Benign tumors of the jejunum and ileum. Radiological signs of benign tumors of the jejunum and ileum are determined by the type of tumor growth - external or internal. *Submucosal tumors* are characterized by round or oblong filling defects with clear contours and usually do not cause deformation of the adjacent mucous membrane. *Tumors developing from the subserous layer* of the jejunum or ileum grow in the direction of the abdominal cavity pushing the intestine aside. *Hypotonic enterography* is used to diagnose impairment of the small intestine.

Tumors of the mesentery of the small intestine. Passage of barium suspension in large tumors of the mesentery is characterized by displacement of loops of the small intestine; free space at the location of the tumor without contrasted loops of the small intestine, which arcuately cover the palpated tumor. Arteriography of the superior mesenteric artery shows displacement of the vessels in the direction of the tumor. Histologically, tumors of the mesentery can be malignant (sarcoma, cancer) and benign (lipoma, fibroma, leiomyoma, neurinoma). The complications that cause tumors of the mesentery of the small intestine include: compression or germination of the tumor into the mesenteric intestinal vessels, infarctions and necrosis of the intestine with severe consequences.

Differential diagnosis of tumors of the jejunum and ileum should be done with Crohn's disease, tuberculous lesion of the intestine, lymphogranulomatosis of the small intestine, Boeck's sarcoid (deformation of the relief of the mucous membrane, multiple defects of filling, unevenness of contours); lipomatosis developing in the area of ileocecal valve; prolapse of the mucous membrane of the ileum into the cecum and enlarged folds that make up a valve - valve hypertrophy, with inflammatory changes and cicatricial deformations, determined by the pathology of the appendix and postoperative adhesions.

In malignant tumors of the large intestine. Radiological examination is the most reliable method for diagnosis of cancer of the large intestine. The clinical course of cancer of the large intestine depends on the localization of the process, stage, extent of the tumor, the general state of the patient. The duration of the latent period of the disease is from a few months to a year. Cancer of the large intestine is divided into the following forms:

1) *exophytic*, which includes polypoid and saucer-like cancer;

2) *endophytic*, consisting of ulcerative, ulcerative-infiltrative and diffuse-infiltrative cancer;

3) *mixed form* (exophytic-endophytic).

Cancer of the large intestine is diagnosed with the help of a double contrast technique, which reveals important details that are not clearly or nit fully detected when the intestine is tightly filled. Both methods (retrograde introduction of contrast agent and double contrast) help with high precision to diagnose cancer of the the large intestine based on the following radiological signs: a defect of filling (central or marginal) with uneven, corroded contours, transformation of the mucosal relief at the level of the lesion, "amputation" of the intestine, disruption of folds at the edge of the tumor, the presence of an additional shadow in the intestinal lumen against the background of gas, the presence of ulcers in the center or periphery of the tumor, violation of patency, restriction of mobility.

Cancer of the right half of the intestine is characterized by the presence of a hilly tumor in the lumen, with uneven corroded edges, the presence of ulcer, and restructuring of the folds of the mucous membrane around. The tumor of the left half of the colon in most cases circulates the intestine, which is due to infiltrative growth of the tumor; pathological area with uneven, corroded edges appears circularly narrowed; proximal intestinal lumen is enlarged. In later stages of the development of the tumor of the left half of the colon, barium suspension does not enter retrogradely the upper departments of the colon; there is a picture of chronic, subacute or acute intestinal obstruction.

Colon cancer with a predominance of *exophytic growth* on the background of gas appears as a shadow of irregular or oval form with a hilly surface that protrudes into the intestinal lumen; areas of the ulcer on the surface of the tumor are filled with contrasting substance. Colon cancer with predominance of *endophytic component* in double contrast is characterized by the presence of a circular narrowing of the lumen of the intestine, uneven contours and the presence of tumor in the intestinal lumen, rigidity of contours.

Tumors of the large intestine give *direct and indirect angiographic signs of malignant degeneration*. The direct signs include: hypervascularization of tumor vessels; the nature of

vascularization depends on the macroscopic type of tumor. Exophytic malignant tumors have increased blood supply and a distinct pathological vascular network. Colon cancer with predominance of endophytic component is represented on angiograms by single pathological vessels. In severe cases of tumor hypervascularisation, the newly created vascular network is represented by chaotically located, short, convoluted vessels, which sometimes blindly end, with the presence of constrictions and sack-like enlargements.

Sarcoma of the large intestine is a rare disease that occurs at a young age. The clinical picture is similar to that in cancer, except that there are no signs of obstruction in sarcoma. Sarcoma of the large intestine is characterized by marked expansive growth and leads to a sharp dilation in the intestinal lumen and has the following radiological signs: uneven and rigid contour, presence of filling defect, smoothness of gaustrum, absence of peristalsis.

In suspected ***rectal cancer***, the method of retrograde administration of a contrast medium is used. The indicated method allows: to confirm the presence of a tumor, to clarify the form of tumor growth and its extent, to find out the presence of synchronous tumors in the proximal departments of the large intestine, to study the anatomical variants of the sigmoid colon and the transition of the process to the surrounding tissues.

Anorectal cancer (*exophytic form*) is characterized by uneven and rough contours in a limited area, reconstruction of the relief of the mucous membrane, presence of a "ledge", "steps" at the level of the upper border of the tumor and healthy tissue.

C***ancer of the ampullary department*** (*exophytic growth*) is characterized by a deformation of the intestine, asymmetry of the walls, deformation of the contour, its disruption at the level of the upper and lower poles of the tumor, presence of tumor conglomerate on the relief, a site of ulcer in the tumor area. If endophytic component predominates, then in these cases there is a circular narrowing of the lumen of the intestine with a significant enlargement of the department lying above (suprastenotic enlargement), rigidity of the contour, hovering in the form of "visors" of unaffected walls above and below the tumor. Examination of pneumorelief shows a picture similar to that seen in cancer in other parts of the large intestine.

Artificial gas contrast of pararectal cellular tissue (**pararectopneumography**) is used to clarify the findings obtained following examination with a finger, which helps to obtain additional information that reflects the localization, form, spread of the tumor to the outer surface of the rectum wall or to the depth of the pelvic tissue. Signs indicative of tumor infiltration in pelvic tissue are characterized by a discontinuity or a lack of gas strip at a certain level. In germination of a tumor into the own fascia of the rectum and its spread to the parietal cellular tissue, gas is traced on the radiograms in the form of a continuous strip, indicating the remoteness of tumor infiltration from the walls of the pelvis. The diagnostic value of pararectopneumography is significantly increased when combined with a tomographic (CT) study, which gives a clearer picture of the extent, size and form of tumor infiltration. Septography, proctocystography and vasovesiculography are used to obtain the full amount of diagnostic information on the extent of the spread of tumor to neighboring organs.

Septography involves single infusion of gas into the bladder, vagina and rectum, determining the connection of the rectum tumor with the bladder and vagina on the basis of such radiological symptoms as uneven contour of the bladder, as well as the contour of the rectum adjacent to it, the absence of air layer between the walls of these organs.

Proctocystography is simultaneous artificial contrast of the rectum with barium suspension and the bladder with iodine agents, making it possible to assess the transition from the rectum to the wall of the bladder based on the detection of symptoms of unevenness, harshness and deformation of contours. Vasovesiculography (introduction of a contrast agent into the seminal ducts and seminal vesicles) helps to detect compression of seminal ducts in the pelvic-bladder area, their displacement, deformation, amputation, epansion, which is a sign of the spread of rectum tumors on them.

Colonoscopy in surgical diseases of the colon and rectum

Colonoscopy *is an endoscopic method for examining the cavity and the mucous membrane of the colon using a colonofibroscope (colonoscope).*

Indications and contraindications. Colonoscopy supplements X-ray and laboratory findings and is shown in repeated intestinal bleeding, in cases of suspected tumor of the large intestine, the presence of polyps in it, chronic ulcers, non-specific inflammatory processes. Contraindications: severe general condition of the patient, severe cardiovascular damage, bronchial asthma, ascites, acute enterocolitis, etc.

Colonoscopy in some surgical diseases

In ***catarrhal colitis*** the mucous membrane of the large intestine is loose, edematous, of intense red color. On separate sites it is covered with white plaques and mucus. Pattern of vessels is not traced.

In ***atrophic colitis*** mucous membrane is thinned, pale with matte hue and the folds are smooth. The network of blood vessels is well visible.

In ***non-specific ulcerative colitis*** (NUC) pathological process often begins in the distal part of the large intestine (in the rectum and sigmoid colon) and extends proximally. Less likely lesion immediately captures the entire large intestine and can spread to the terminal department of the ileum. NUC develops cyclically: periods of exacerbation alternate with remissions. Therefore there are acute, subacute and chronic phases of the disease.

Colonoscopy in the initial stage of NUC shows that the mucous membrane in the distal part of the large intestine is swollen, hyperemic, covered with hemorrhages in the form of petechia and larger hemorrhages. There is a large number of erosions and small, easily bleeding ulcers, sometimes covered with dark-gray or brown plaques. Vascular network is poorly visible. Mucous membrane is rigid. In the later stage of the disease ulcers enlarge, often becoming linear. The islets of intensely red mucous membrane protrude into the lumen in the form of fringed pseudopolyps. Most severe, progressive forms of acute NUC are characterized by almost complete destruction of the mucous membrane. The folding is lost, intestinal wall becomes fragile and can perforate in injection of excessive air into the intestine and manipulation of the colonoscope.

In the chronic phase NUC is very often associated with deformation of the large intestine. The latter is sharply shortened, its lumen is unevenly narrowed, the intestine loses elasticity, and when air is blown into it, becomes poorly pliable. If chronic NUC is associated with destructive changes, colonoscopy shows large ulcers, covered with whitish-yellow fibrinous films. The edges of the ulcers are represented by a hyperemic swollen mucous membrane. Its preserved areas and focal granulations protrude into the lumen of the intestine, forming multiple pseudopolyps. The internal surface of the intestine looks bumpy.

Pseudopolyps are quite characteristic of the chronic stage of NUC and have the most diverse form (finger-shaped, mushroom-shaped, coral-shaped), varying in size and color.

In those cases of chronic NUC, when reparative processes prevail, ulcers lose fibrinous films and are filled with pink granulations with delicate edges of the epithelium along their periphery. On separate sites ulcers heal and form islets of atrophic mucous membrane. They alternate with pseudopolypous formations.

Due to large destructive changes and deformation of the large intestine, colonoscopy in NUC should be carried out very carefully. Usually total colonoscopy can be performed only in the stage of remission of the disease. During the exacerbation of the pathological process, it is limited to examining only a limited area of the intestine.

Colonoscopy in tumors of the large intestine. Polyps are the most common benign tumors of the large intestine. They can be singular and multiple.

A ***single polyp*** usually has a rounded form, located on a stalk or broad base. The color does not differ from the surrounding mucous membrane and only when inflamed it becomes intense-red and bleeding. The size of the polyp varies from a few millimeters to 2-3 cm or more.

Less common is ***villous polyp*** on the stalk or on a broad base, and sometimes “sliding” on the surface of the mucous membrane. Villous polyp is bigger, has a lobular structure, is of bright red color.

Multiple polyps of the large intestine always give increased caution to the doctor as a precancerous disease.

Cancer of the large intestine has three main types: polypoid, ulcerative and infiltrative. In colonoscopy, *polypoid cancer* looks like a polyp, but differs from the latter by a more vivid color of

the mucous membrane, dense texture and presence of an easily bleeding ulcer, which is often covered with fibrinous purulent plaques. *Ulcerative cancer* of the large intestine looks like a crateroid-shaped deepening with corroded edges and rough bottom. The mucous membrane surrounding the tumor is rigid and paler. The tumor bleeds easily. *Infiltrative cancer* is localized in the submucosal layer. Externally it manifests itself by focal induration of the intestinal wall with the formation of bumps. Its mucous membrane is pale, with small hemorrhages and erosions. As the tumor grows, the lumen of the intestine in the site of the lesion is narrowed.

In ***stenosing tumors of the large intestine*** colonoscopy is complicated, since the semi-solid contents of the intestines can hide polyps and tumors and complicate their detection.

Rectoromanoscopy in surgical diseases of the rectum

Rectoromanoscopy is a visual examination of the mucous membrane and cavity of the rectum and sigmoid colon with a number of diagnostic and therapeutic manipulations under the control of an opto-mechanical device - a ***rectoromanoscope****.*

Indications and contraindications. Rectoromanoscopy is indicated in suspected neoplasms, polyps, ulcers (of various etiologies), chronic inflammatory process in the rectum and distal division of the sigmoid colon. Rectoromanoscopy is indicated in all cases of repeated intestinal bleeding, including in chronic hemorrhoids. The method is valuable for the detection of chronic dysentery and amebiasis. Contraindications are general severe condition of the patient, presence of acute inflammatory and purulent lesions of the anus, and scarring of the rectum.

Rectomorroscopy in some diseases.

In ***chronic proctitis***, the mucous membrane of the rectum is fluffy, sometimes hyperemic. There are single lumps of mucus on its surface.

In ***acute proctitis***, the mucous membrane acquires a bright red color, becomes swollen, with pinpoint hemorrhages. On some parts of the wall there are islets of mucus, pus, fibrin. Mucous membrane is typically easily injured (bleeds) during the study.

In ***ulcerative proctitis*** the mucous membrane at a great distance is swollen, dense and hyperemic. On certain areas of the wall there are ulcers with mucous-purulent and fibrinous plaques. Later, ulcers are covered with granulations, which often take the form of small polypous enlargements.

Amoebic dysentery is characterized by focal lesions. Rectoromanoscopy shows ulcers of varying sizes with corroded or, conversely, with raised edges, covered with white necrotic plaques. On the periphery ulcers are surrounded by wide rims of reactive hyperemia with pinpoint hemorrhages. On the rest of the site, between ulcers, the mucous membrane has a regular appearance that distinguishes amoebic dysentery from ***bacillary*** one. The latter is always characterized by severe diffuse changes (edema, hyperemia).

In ***internal hemorrhoids*** there are several (2-5, rarely more) varicose enlarged venous nodes with bluish tint 4-5 cm in the middle of the anus. They are more often located on the right and left hemispheres of the lumen of the rectum. Bleeding nodes are of bright red color. Sometimes there is a thin trickle of blood, discharged when the patient is makes an exertion. Catarrhal proctitis can accompany hemorrhoids.

Polyps of the rectum and sigmoid colon (single and multiple - intestinal polyposis) can be located at different distances from the anus.

A ***solitary polyp*** has a form of round or oval formation on a narrow stalk or on a broad basis. The size of the polyp varies from several millimeters to 1-2 cm or more. Its mucous membrane is pink, smooth or granular. In the case of inflammation or ulceration, the polyp acquires a cherry or crimson-cyanotic color and begins to bleed. Examination determines the shape and size of polyps, their number, depth of placement, the limits of the spread of pathological process in the region of the rectum or sigmoid colon. The polyps should always raise oncologic alertness, because polyps are characterized by malignancy.

Cancer of the rectum. A low-level tumor is usually diagnosed with a finger, a rectal speculum and an anoscope.

Deeper location of tumors (deeper than 10 cm from the anus) requires rectoromanoscopy. There are *exophytic form of cancer* (the tumor grows into the lumen of the intestine), *endophytic*

(infiltrative growth in the submucosal layer) and *mixed* (exophytic-endophytic). Therefore, the tumor may have the form of a node with disintegration in the center, focal induration of the intestinal wall or a bleeding ulcer located on a dense base. In tumor lesion of the intestine, its lumen is circularly narrowed.

In ***exophytic growth*** the tumor is dark red, the edges are uneven and seemingly deformed, hanging over the surrounding mucous membrane. The basis of the tumor and its edges are dense. There are dark clots of blood, less commonly fibrinous-purulent layers, on the surface. The tumor bleeds easily.

Endophytic (diffuse) cancer that grows under the mucous membrane manifests itself as a rigidity of the intestinal wall. Such a tumor becomes ulcerative later than in exophytic growth.

During rectoromanoscopy it is necessary to conduct a thorough, consistent examination of the rectum, especially its anterior wall, with straightening of all folds, so as not to miss a small tumor hidden behind the folds of the mucous membrane.

Laparoscopic examination of the intestine in certain diseases

Laparoscopic examination in ***Crohn's disease*** provides valuable diagnostic information on the possible surgical treatment. The study shows severe inflammatory changes in the intestine: edema and hyperemia of the serous membrane, presence of hemorrhages and fibrinous layers on it (perforation may be located in this area); peristalsis is absent or may be weak, but appears and intensifies in instrumental "palpation"; pressing the wall of the intestine results in formation of fossae on it; there is effusion in the abdominal cavity, which is taken for study.

Laparoscopic examination in ***acute intestinal obstruction***. The risk of laparoscopy should not exceed the diagnostic values in the clinical picture of acute intestinal obstruction. *Dynamic intestinal obstruction* differs from the mechanical by moderate paresis of the entire intestine, cyanosis of the individual loops, poor peristalsis or its absence, lack of changes in the peritoneum, organs and effusion. *Spastic intestinal obstruction* is characterized by the absence of organic lesions, the ability to trace how the "wave" of spasm passes through the intestine slowly and barely visible. In different types of mechanical intestinal obstruction during laparoscopy, it is necessary to carefully evaluate the general changes in the abdominal organs by panoramic examination, to determine the place of the obstacle, and then purposefully examine it. During laparoscopy, it is not always possible to examine the obstacle, since it is covered with bloated intestinal loops. In such cases, it is necessary to evaluate the indirect signs of obstruction, the nature of morphological changes in the intestine and to detect effusion in the abdominal cavity. Indirect signs allow to differentiate mechanical intestinal obstruction from the dynamic one.

II.2. CURRENT METHODS OF EXAMINATION IN SURGICAL DISEASES OF THE LIVER, EXTRAHEPATIC BILE DUCTS AND PANCREAS

Diseases of the digestive system are as common as cardiovascular diseases. Diseases of the hepatopancreatoduodenal area currently rank as one of the most common causes of disability (mostly affecting people aged 45 to 60) and mortality. Their social significance is increasing everywhere in the world. Diagnosis and treatment of diseases of the organs of the hepatopancreatoduodenal area (HPDA) are becoming more and more relevant to the problem of surgery. This is due to an increase in the incidence and an increase in the number of patients with advanced tumors, high postoperative lethality, which, even after palliative operations, varies from 13 to 59%.

Modern medicine has at its disposal great opportunities for a detailed study of the structure and functioning of organs and systems, rapid and accurate diagnosis of any deviations from the norm or diseases. Methods of laboratory diagnosis to a greater extent reflect problems at the cellular and subclinical level, but at the same time allow assessing "disruptions" in a particular organ. Instrumental diagnostic methods are used, among other techniques, to see what happens in the body. Only a comprehensive approach to the problem can yield high treatment outcomes.

Biochemical studies in liver diseases

They occupy an important place in the diagnosis of diseases of the liver, bile ducts and pancreas. Results of the study are not strictly specific. Not allowing the exact diagnosis in all cases,

they provide an opportunity to confirm the impairment of these organs, to assess their functional status and to assess the severity of the pathological process.

Among the numerous biochemical tests that characterize the functional state of the liver, the most important are the studies assessing pigment exchange, excretory and neutralizing liver function, protein, fat and carbohydrate metabolism, as well as the activity of enzymes.

Impaired pigment exchange. The evaluation of the nature of the disturbances of pigment exchange is based on the results of the study of bilirubin and its fractions in blood serum and bilirubin and its metabolites in urine and feces.

Determination of bilirubin in serum. In clinical practice, various methods for determining bilirubin and its fractions in serum are used. The most common of these is the Jendrassic-Grof method. It is based on the interaction of bilirubin with diazotiated sulfanilic acid to form azo-pigments. In this case, the *direct (which has already broken the bond with albumin), also known as bound bilirubin* (bilirubin-glucuronide) gives a quick *"direct" reaction* with diazo-reactive, whereas the reaction of *indirect* (still connected with albumin) and *free* (not connected with glucuronide) bilirubin proceeds much slower. To accelerate it, various substances-accelerators that break the ligament of bilirubin with albumin, transfer it from the spleen to the liver, for example caffeine (***Jendrassik-Kleghorn-Grof method***), which release bilirubin from protein complexes ("indirect" reaction), are used. As a result of interaction with diazotilated sulfanilic acid, bilirubin forms colored compounds. Measurement is carried out using a photometer.

Normally, blood serum contains *8.5-20.5 µmol/l of total bilirubin*, with about 75% of bilirubin (up to 16.5 µmol/l) in the proportion of "indirect" free bilirubin. It is also necessary o determine the *coefficient, the ratio of the total bilirubin to the "direct", which normally should not exceed 0.25.*

Determination of bilirubin in urine. Different qualitative methods for detecting bilirubin in urine are based on the transformation of this substance under the action of oxidizing agents in biliverdin, which has green color or bilirubipurins (red color). ***Rozin's test***. 1% alcohol solution of iodine is carefully drawn along the wall into a test tube containing 4-5 ml of urine. A green ring is formed on the border of urine and iodine solution in presence of bilirubin in urine. Solutions of trichloroacetic acid (***Foushet's test***), diazotiated sulfanilic acid (***Gottfried's test***) and other oxidizing agents are also used as oxidants. Normally, the minimum amounts of bound (direct) bilirubin are discharged with urine, which are not determined by described qualitative reactions.

Determination of urobiline in urine is also based on the formation of colored pink or red compounds in interaction of urobiline with HCl, copper sulfate, or Ehrlich reagent (paradimethylaminobenzolaldehyde). ***Neubauer's test.*** 3-4 drops of Ehrlich reagent are added to 3-4 ml of fresh urine. The coloration of urine in red indicates a diagnostically significant increase in urobiline concentration in urine. Normally, there are traces of urobilin in urine (no more than 5-6 mg per day), which are not determined by the usual qualitative reactions.

Interpretation of results. Evaluation of the disturbances of pigment exchange is often crucial in the differential diagnosis of jaundice - parenchymal, mechanical and hemolytic. In case of damage to the liver parenchyma, in ***parenchymal*** or ***hepatic jaundice*** in patients with hepatitis, cirrhosis, cancer and other diseases of the liver, there is damage to hepatocytes and violation of the capture of free bilirubin by the liver cell (I) and its binding to glucuronic acid (II), which leads to increase in *"indirect" free bilirubin* in the blood (Bi); impairment of discharge of bilirubin-glucuronide (*"direct" bound bilirubin*) from liver cells to the biliary capillaries caused by inflammation, destruction, necrosis and decreased hepatocyte membrane permeability, leading to regurgitation of bile back to sinusoids and systemic circulation and, therefore, to increase in *"direct" bound bilirubin* (Bd) in blood. Finally, dysfunction of hepatocytes is also accompanied by the loss of the ability of liver cells to capture and metabolize urobilinogen absorbed in the intestine (*mesobilinogen*), which in large quantities is released to systemic circulation and is excreted in urine as urobiline.

Thus, *the content of both free "indirect" and bound "direct" bilirubin in blood is increased in* ***parenchymal jaundice****. The latter, being a well-dissolved in water compound, easily passes the renal barrier and appears in urine, causing its dark coloration ("the color of beer"). Urobilin*

(mesobilinogen) is also present in urine in large quantities. In feces, the content of stercobilinogen may be slightly reduced due to disturbance of bile secretion by hepatocytes.

Mechanical (obstructive) jaundice develops due to obstruction of extrahepatic bile ducts by stone or due to compression of the common bile duct by tumor (cancer of the pancreas head, cancer metastases to the lymph nodes of the hepatic porta). As a result, *the release of bile into the intestine is blocked and, accordingly,* ***no urobilinogen (mesobilinogen) and stercobilinogen are formed****. In this regard, urobilin in urine and stercobilinogen in feces are completely absent (acholic feces). In blood, the level of bound "direct" bilirubin increases significantly, since its formation in the liver cell is not disturbed for a long time. Accordingly, there is a large amount of "direct" bound bilirubin in urine and the urine acquires a dark color ("the color of beer").*

Hemolytic jaundice is characterized by the formation of a large amount of free "indirect" bilirubin in the RES, which is not completely metabolized in the liver, although the function of hepatocytes is not disturbed and they work with increased load. As a result, the content of *free "indirect" bilirubin*, which does not pass the renal barrier and does not enter into urine, increases in blood. Since the amount of *bound "direct" bilirubin* released by the liver in the intestine and, consequently, stercobilinogen, significantly increases, the level of *stercobilinogen (urobilin),* which enters systemic circulation from hemorrhoidal veins, is significantly elevated in urine.

The main laboratory findings in parenchymatous, mechanical and hemolytic jaundice are presented in Table 8.

Table 8

Basic laboratory findings in jaundice of different origin

Labarotory findings	**Type of jaundice**		
	Parenchymal	**Mechanical**	**Hemolytic**
Bilirubin in blood	Indirect and direct are increased	Direct is increased	Indirect is increased
Bilirubin in urine	Present	Present	Present
Urobilin in urine	Present (mesobilinogen)	Absent	Present (stercobilinogen)
Stercobilin in feces	Present, but may be reduced	Absent	Present

It should be borne in mind that sometimes in the clinical practice the nature of the disturbances of pigment exchange may differ significantly from the above laboratory criteria for various types of jaundice. Thus, in severe damage of the liver parenchyma, especially in combination with severe cholestasis, the release of bound "direct" bilirubin into the intestine and, consequently, formation of metabolites of bilirubin (mesobilinogen, stercobilinogen), is sharply reduced. This leads to a significant reduction in the content of stercobilin in feces (acholic feces) and urobilin (mesobilinogen) in urine. Along with the sharp increase in blood levels of bound "direct" bilirubin it may resemble the disturbances of pigment exchange that are characteristic of mechanical jaundice.

Such a situation often arises in acute viral hepatitis (Botkin's disease) and other diseases. Characteristically, with restoration of the liver function and reduction of cholestasis in urine, such patients are found to have urobilin and stercobilin in feces. Correspondingly, the indicators of "direct" and "indirect" bilirubin in blood are aligned.

Often, in the relatively long course of diseases accompanied by blockage of the bile duct and the development of mechanical jaundice (cholelithiasis, cancer of the pancreas head), the liver parenchyma gets involved in the process for the second time, which leads to a partial violation of the capture and binding of indirect bilirubin and to an increase its serum content (along with "direct" bilirubin, which is characteristic for mechanical jaundice). At the same time, other laboratory parameters characteristic of lesions of liver parenchyma also change (see below).

Finally, it should be borne in mind that in partial obstruction of the bile duct, for example, by a "valve" stone, the picture of the mechanical jaundice may be incomplete.

Impairment of protein metabolism. The liver is known to be the only place of synthesis of albumins, as well as fibrinogen, prothrombin and some other factors of blood clotting. In addition, the liver plays a leading role in the formation of α-globulins, a significant proportion of β-globulins, heparin, and enzymes. Synthesis of γ-globulins is carried out by plasma (Kupffer) liver cells. Finally, it should be borne in mind that the liver is a place for natural metabolism of peptides and amino acids

coming there from other organs (for example, abdominal organs), which is an important *detoxification function of the liver*.

Therefore, it is not surprising that any impairment of the liver parenchyma is accompanied by more or less severe violations of protein metabolism, including the processes of protein synthesis and the cleavage of amino acids to the formation of ammonia and urea. The severity of protein defects depends on the severity and duration of the disease, as well as on the activity of the pathological process in the liver.

There are several types of impairment of protein metabolism, pathogenically associated with the presence of disorders of protein-synthetic, detoxification function of the liver, immune inflammation syndrome, etc.

Hypoalbuminemia, which is often combined with a decrease in the content of prothrombin, fibrinogen and other factors of blood coagulation, is one of characteristic laboratory signs of acute and chronic *liver failure*, including the one developing without signs of liver encephalopathy (syndrome of minor liver failure).

Hypergammaglobulinemia with an increase in the content of immunoglobulins (IgA, IgG, IgM) and changes in sediment samples (see below) is an important criterion of immune inflammation syndrome in patients with acute and chronic hepatitis, liver cirrhosis and other diseases.

The content of *α-globulins* in diseases of the liver and biliary system can be normal, elevated or reduced (in severe damage to the liver parenchyma). Significant increase in *α2-globulin* content is sometimes observed in metastatic liver damage. The level of *β-globulins* may be increased in all diseases that are accompanied by intra-or extrahepatic *cholestasis*.

A *decrease in the content of prothrombin* and other factors of blood coagulation (proconvertin, proaccelerin) is due to a reduction in the protein-synthetic function of the liver and is the cause of the development of *hemorrhagic syndrome*. It should be remembered, however, that synthesis of prothrombin is carried out in the liver with the participation of fat-soluble vitamin K. Therefore, hypoprothrombinemia can be caused not only the impairment of the parenchyma of the liver, but also by all pathological processes that lead to a disruption of vitamin K absorption in the blood (mechanical jaundice, intestinal disease, etc.).

The content of *total protein* in the blood, despite significant dysproteinemia, inherent in many diseases of the liver, may long remain normal. More severe liver damage is accompanied by an increase in total protein in the blood, mainly due to the high content of γ-globulins (even against severe hypoalbuminemia). A decrease in total protein in the blood, along with a decrease in albumin content, indicates a severe violation of *protein-synthesizing of the liver* and usually indicates an unfavorable prognosis of the disease.

The evaluation of the content of *ammonia and urea* in blood has an important diagnostic value for characterization of the processes of deamination of amino acids and neutralization of ammonia formed in the intestine in breakdown of food proteins. Violation of the processes of deamination of amino acids (primarily aromatic and sulfur-containing amino acids - tryptophan, phenylalanine, tyrosine, tauric acid, methionine, cysteine) and formation of urea in severe liver damage leads to an increase in the total content of amino acids in the blood and the level of residual nitrogen. At the same time, concentration of urea in the blood decreases, reflecting a decrease in the *urine-forming function of the liver*.

It should nevertheless be borne in mind that in clinical practice patients with severe liver diseases (usually in the terminal stage of hepatic failure) are found to have an increase in the content of urea, which is usually due to the associated renal injury and the development of *hepatic renal failure*. Especially persuasive such a combination of liver diseases with a violation of nitrogen-dependent function of the kidneys becomes in a simultaneous increase in the concentration of other nitrogen-containing compounds (creatinine, uric acid, indican, etc.). In more rare cases, moderate increases in urea may be due to a sharp *increase in protein catabolism*, for example, in acute liver atrophy or acute viral hepatitis, etc.

The reasons for increasing the amount of ammonia in the blood are: 1) violation of the neutralizing and urinogenic function of the liver, and 2) the development of portacaval bypass, leading

to the flow of ammonia and other toxic substances into the systemic blood flow, avoiding the liver. *Hyperammonemia* is characteristic for the terminal stage of hepatic failure and is usually preceded by the development of severe hepatic coma.

Sedimentation (flocculation) tests are based on determining the stability of the colloidal system of serum proteins, which varies with dysproteinemia of various origin, primarily in lesions of the liver parenchyma. Sedimentary samples, which have become widespread in clinical practice, include thymol, sublimate tests, Veltman's test, and others.

Thymol test. The test is based on photocolorimetric determination of the degree of cloudiness of blood serum after the addition of a saturated thymol solution. 0.1 ml of blood serum is added to 6 ml of thymol-veronal buffer solution. After 30 minutes of sedimentation, optical density of the solution is measured, comparing it with the optical density of the thymol-veronal buffer. Normally, the degree of serum clouding corresponds to 0-4 IU.

Thymol test is a particularly reliable and sensitive indicator of *activity of the inflammatory process in the liver*. The test becomes positive, above all, with an increase in the content of γ- and β-globulins, as well as the inhibitory capacity of serum β-lipoproteins. The test gives an opportunity to quantify the time course of the pathological inflammatory process in the liver. It increases, for example, in the early days of the jaundice period of acute hepatitis, as well as in the prolonged and chronic course of the disease. In obstructive jaundice, the test is in most cases negative. It should, however, be remembered that thymol test (as well as the change in the content of γ-globulins) is not strictly specific for liver damage: the test may be increased in nephrotic syndrome, systemic connective tissue diseases, and other diseases. In addition, alimentary hyperlipidemia significantly affects the results of thymol test.

Sublimate test is based on the interaction of serum proteins with mercury chloride sublimate, resulting in a colloidal solution of mercury salts. Violation of dispersion of serum protein fractions causes precipitation of coarse particles. 0.1% solution of sublimate is added to 0.5 ml of non-homogenized blood serum diluted with 1 ml of isotonic sodium chloride solution, to a persistent cloudiness, when a newspaper text can not be read through the vertical layer of the liquid. Sample results are determined in the number of milliliters of 0.1% solution of sublimate, required for serum titration (normally 1.6-2.2 ml). Violation of dispersion of serum protein fractions causes increased precipitate of coarse particles, due to which titration requires a smaller amount of 0.1% solution of sublimate (less than 1.6 ml). Such a sample is considered to be positive.

Sublimate test is less sensitive than thymol test to the disturbances of protein metabolism caused by various diseases of the liver and does not directly correlate with the severity of the damage to hepatocytes. Positive sublimate test is detected during a prolonged course of the process, its transition to a chronic form and, especially, in the development of liver cirrhosis.

Disturbance of carbohydrate metabolism. The liver is known to be involved in numerous reactions of intermediate carbohydrate metabolism (galactose and fructose transformation into glucose, synthesis and disintegration of glycogen, gluconeogenesis, glucose oxidation, glucuronic acid formation, etc.). In disturbed liver function, its ability to perform these reactions decreases, which leads to a decrease in oxidative phosphorylation, a deficiency of macroergic compounds and promotes anaerobic glycolysis, accumulation of acid metabolites in cells and subsequent damage to hepatocytes.

The level of glucose in the blood usually changes only in very severe liver damage. Therefore, various *functional loading tests* are usually used to *evaluate the carbohydrate metabolism disturbances* caused by liver damage.

The most common of these is *galactose loading test*. Galactose is known to enter the body with milk sugar. In the liver it is transformed into glucose-1-phosphate. In impaired liver function, the body's ability to use galactose is reduced. In galactose loading test the patient is offered to drink 40 g of galactose, dissolved in 200 ml of water, followed by determination of its content in the blood and urine. In a healthy person, no more than 3 g of galactose is released by the kidneys, and this release is carried out for no more than 4 hours. The maximum increase in the amount of galactose in the blood is observed in 30-40 minutes after administration and does not exceed 15% of the baseline level. Restoration of galactose concentration in blood takes place within 2 hours after it is consumed. *The*

level of maximum post-loading elevation of galactose in the blood and the duration of its decrease to the baseline values, as well as the total amount of galactose released from the urine, increase in impaired liver function. It should be remembered that the results of galactose loading tests are influenced by the absorption and excretory functions of the kidneys.

Abnormal fat metabolism. The liver plays a leading role in the metabolism of lipids, primarily neutral fats, fatty acids, phospholipids and cholesterol.

The content of *cholesterol* and esters of cholesterol in the blood has the most practical value for assessing the function of the liver and the biliary system. The level of cholesterol is usually reduced in severe liver damage and *hepatic insufficiency*. In this case, the content of ether-binding cholesterol has a special significance. In liver damage, the proportion of ether-binding cholesterol to total cholesterol decreases below 0.7-0.6.

On the contrary, in *impaired biliary excretion* in any part of the biliary system (intrahepatic cholestasis, concretions in the common bile or liver duct, pancreatic cancer with constriction of choledochus, tumor metastases in the liver hilum, hepatoma, etc.), there is an increase in the content of cholesterol in blood, which is usually combined with an increase in the activity of alkaline phosphatase (see below).

The content of phospholipids in blood is reduced in many diseases of the liver.

Abnormal mineral metabolism. The content of serum iron and copper has the most diagnostic value for diseases of the liver.

Iron is known to be present in the liver mainly in the form of three compounds:

1) *ferritin*, which is the main reserve metalloprotein used by the bone marrow in the synthesis of hemoglobin;

2) *hemosiderin*, which is formed in the process of decomposition of hemoglobin and accumulates in the liver with increased hemolysis;

3) *transferrin* is a transport protein that carries iron from the liver to the bone marrow.

The content of iron in serum can be sharply increased in acute hepatitis and, to a lesser extent, in chronic hepatitis and liver cirrhosis. In obstructive jaundice, the level of serum iron changes little or decreases.

On the contrary, the content of *copper*, which is present in the blood in the form of ceruloplasmin enzyme, is significantly increased in obstructive jaundice and changes little in hepatitis.

Abnormal enzyme metabolism. A change in the content of enzymes in blood in liver, biliary and pancreatic diseases is quite sensitive, albeit a non-specific biochemical test.

Aminotransferase. Determination of the activity of aminotransferases (ALT and AST) is of greatest practical importance for the early diagnosis of *cytolytic syndrome*. The activity of enzymes increases as early as at pre-jaundice stage, and sometimes even in the incubation period of acute hepatitis. In contrast to the changes in these enzymes in patients with acute myocardial infarction, in the damage of liver parenchyma, the content of ALT in blood plasma is generally higher than that of AST. De Ritis ratio decreases below 1.3-1.0, reaching 0.55-0.65. Exceptions include cases of alcoholic liver damage (alcoholic hepatitis, cirrhosis of the liver), which are characterized by a more significant increase in ASAT activity (de Ritis ratio increases by more than 1.3-2.0).

In severe liver damage, massive necrosis of hepatocytes, AST can be released from damaged mitochondria of these cells, with serum ASAT values gaining special diagnostic and predictive value. As a consequence, the AST / ALT ratio, which is called **de Ritis ratio**, is often used in the clinical practice, in addition to the direct AST and ALT values. Normally, its value is 1.3 ± 0.4. The clinical value of AST/ALT in serum has been extensively studied in various liver diseases. The results of the assessment of current publications show that in the case of uncomplicated viral hepatitis or non-alcoholic damage of the liver, the AST/ALT ratio, as a rule, does not exceed 1 or decreases to 0.6-0.8. On the contrary, an increase in the ratio by more than 1.4 (due to increased AST activity) is observed in cirrhosis, severe alcoholic and toxic liver damage with the destruction of most of the liver cells or in other diseases, for example, in Wilson's disease, the AST/ALT ratio may exceed 4 (Pratt DS, Kaplan MM, 2000; Paul T. Giboney, 2005).

Aldolase. Aldolase (fructose-1,6-diphosphate-aldolase) activity increases in many pathological conditions, accompanied by damage and cell destruction. Type B aldolase isoenzyme is the most specific in damage of hepatocytes, since it is contained only in the liver and is not normally found in blood.

γ-glutamyltranspeptidase (GGTP). Increase in the activity of γ-glutamyltranspeptidase (γ-glutamyltransferase) is one of the most sensitive biochemical tests indicating liver and biliary disorders. In the latter cases (*obturation* or *compression* of the common biliary or liver duct, intrahepatic *cholestasis*, biliary cirrhosis of the liver), the activity of this enzyme increases especially significantly. In diseases of the liver parenchyma the test is positive in 90% of cases. In normal GGTP activity, the likelihood of liver disease is very small. It should be remembered that the activity of GGTP increases in prolonged abuse of alcohol, which is mainly due to the toxic effects of ethanol on the liver. Narcotic drugs also increase GGTP activity.

Alkaline phosphatase belongs to a number of so-called secreted enzymes and is found in practically all organs, especially in the liver, bone tissue, and intestine. The most significant increase in the activity of alkaline phosphatase in the plasma is due to the blockage of the natural way of excretion of this enzyme, *obturation of the biliary tract* during blockage or compression of the common bile duct, intrahepatic cholestasis, biliary cirrhosis of the liver, cholangitis, especially accompanied by obstructive jaundice. In case of damage to the liver parenchyma, an increase in the activity of alkaline phosphatase in blood plasma is not so significant, although it is observed in most patients. It should be borne in mind that the enzyme activity often increases not only in diseases of the biliary system and the liver, but also in some bone diseases (rickets, Paget's disease, osteosarcoma, myeloma, bone fractures, etc.), as well as in the use of certain drugs with hepatotoxic action or enhancing cholestasis (barbiturates, agents with salicylic and nicotinic acids, indomethacin, etc.).

Lactate dehydrogenase. The activity of lactate dehydrogenase, especially its isoenzyme LDH5, is also often increased in diseases of the liver parenchyma (acute and chronic viral hepatitis, cirrhosis and liver cancer, etc.).

Thus, abnormalities of the liver and the biliary system are mostly characterized by an increase in the activity of blood plasma enzymes.

Cholinesterase. Unlike other enzymes, cholinesterase activity in diseases of the hepatobiliary system does not increase, but decreases. This is due to the fact that cholinesterase is synthesized in the liver and secreted into the bloodstream. An *impairment of the synthetic function of hepatocytes* is associated with a decrease in the production of this enzyme. In assessing the results of the study of cholinesterase in the blood, it should be remembered that the decrease in its level is also observed in administration of cytostatics, in various infections, fasting and by the end of the first day of acute myocardial infarction.

Examination of excretory and detoxifying liver function. Two tests are mainly used to study excretory liver function, which gives an idea of the general functional activity of hepatocytes.

Bromsulfalein test is among the most informative and sensitive methods of study of absorption and excretory function of the liver. After single intravenous administration of 5% sterile bromsulfalein solution at a rate of 5 mg per 1 kg of body weight, blood is collected from the veins of another arm and dye concentration is determined. In norm, no more than 5% of the administered dose remains in the blood 45 minutes after the introduction of dye. The rest of bromsulfalein is absorbed by the liver cells and is secreted into bile. The test is considered positive if, after 45 minutes, the color of the dye exceeds 6-12% of the baseline in the blood collected after 3 minutes. The modified bromsulfalein test method is also used, with prolonged intravenous administration of the dye and the assessment of the plasma clearance curve from bromsulfalein. The rate of clearance reflects the level of plasma flow in the liver, as well as the processes of absorption and release of dye by hepatocytes. Bromsulfalein test is one of the most sensitive methods for evaluating liver function. It becomes positive in any damage of the organ parenchyma (acute and chronic hepatitis, fatty hepatosis, liver cirrhosis, benign hyperbilirubinemia, etc.) even at the earliest stages of the disease and correlates well with the severity of the pathological process. It should be remembered that the test is also positive in intrahepatic

cholestasis and obstructive jaundice, which is associated with disturbance of bile outflow, as well as in congestive heart failure.

Indocyanine test is based on the same principle as bromsulfalein test. The solution of indocyanine is administered intravenously at a rate of 0.5 mg per 1 kg of body weight. In norm, no more than 4% of the administered dye remains in blood after 20 minutes. In impaired liver function, the amount of dye that is detected in the plasma after 20 minutes after administration is increased. The evaluation of the results of an indocyanin test is carried out in the same way as in bromsulfalein test. Positive results are revealed in minimal decrease of the function of hepatocytes.

Hippuric acid synthesis test (Quick's test) is used to evaluate the neutralizing function of the liver. For this test sodium benzoate is used, which, when administered intramuscularly or intravenously, binds in the liver with glycol, forming hippuric acid that is excreted from the body with urine. The test is conducted in the morning after a light breakfast and emptying of the bladder. The patient is given 4 g sodium benzoate, dissolved in 30 ml of water, and a half glass of water. After that, the urine is collected every hour for 4 hours, determining the content of hippuric acid. In health, hippuric acid is excreted in urine in the amount of 3.0-3.5 g, which is more than 60-65% of the maximum possible amount. In *impaired liver parenchyma*, the synthesis of hippuric acid and, consequently, its excretion with urine are reduced, which is regarded as a positive test. Quick's test is usually positive in acute and chronic hepatitis, liver cirrhosis, as well as in tumors and metastatic liver lesions, alveolar echinococcosis of the liver, obstructive jaundices, purulent cholangitis and multiple abscesses of the liver. At the same time, in limited lesions of the organ and uncomplicated diseases of the biliary tract and gall bladder, Quick's test gives normal results. It should also be remembered that the results of the test depend on the absorption of sodium benzoate and its release by the kidneys. Therefore, in many concomitant diseases of the stomach (peptic ulcer, gastritis), intestines (enteritis, colitis) and kidney, the results of the tests do not reflect the functional state of hepatocytes. Therefore, in recent years Quick's test is rarely used in clinical practice.

Biochemical syndromes. In analyzing the results of biochemical studies in patients with liver diseases, it is expedient to differentiate 4 laboratory syndromes, each of which to a certain extent corresponds to certain morphological and functional changes in the body. Usually in each case of the disease there is a combination of several biochemical syndromes.

Cytolytic syndrome, or hepatocyte integrity impairment syndrome, is caused by violation of the permeability of cell membranes, decay of membrane structures, necrosis of hepatocytes and release of enzymes into plasma. Syndrome of cytolysis occurs in viral, medicinal, toxic hepatitis and other acute liver damage, liver cirrhosis, chronic active hepatitis, as well as in fast-growing or prolonged obstructive jaundice. Diagnosis of cytolytic syndrome is based on the development of the following signs:

1) increase in the activity of enzymes in blood plasma (ALT, AST, LDH and its isoenzyme LDH5, aldolase, etc.);

2) hyperbilirubinemia (predominantly direct reaction);

3) increase of iron concentration in blood serum.

Syndrome of minor hepatocellular insufficiency (without hepatic encephalopathy) - a group of biochemical signs, indicating a significant decrease in various functions of the liver, primarily synthetic:

1) decrease in the activity of cholinesterase in blood plasma;

2) reduction of the content of prothrombin in blood serum;

3) hypoalbuminemia and (less often) hypoproteinemia;

4) decrease in the content of V and VII factors of blood coagulation;

5) reduction of cholesterol concentration;

6) hyperbilirubinemia (mainly due to an increase in free bilirubin).

Inflammatory (mesenchymal-inflammatory) syndrome is caused by the development of the so-called *immune inflammation* in the liver: sensitization of the immunocompetent tissue, activation of the reticulohistiocytic system, infiltration of portal ducts and intralobular stroma. The following criteria are used to diagnose this syndrome,:

1) increase in serum γ-globulins, often in combination with hypoproteinemia;

2) changes in protein-sediment test (thymol, sublimate tests);

3) appearance of nonspecific markers of inflammation (elevated ESR, increased seromucoid, appearance of C-reactive protein, etc.);

4) increased levels of IgG, IgM, IgA (see below);

5) increase of nonspecific antibodies in the blood (to DNA, smooth muscle fibers, mitochondria, microsomes);

6) changes in the reaction of blast lymphocyte transformation (BLT) (see below). The determination of these biochemical parameters can be used to diagnose the *activity of the pathological process in the liver*.

Cholestasis syndrome is caused by a violation of the biliary function of hepatocytes and bile ducts (*intrahepatic cholestasis*), as well as a violation of bile outflow from the hepatic and common bile ducts due to their obstruction (*extrahepatic cholestasis*). Both forms of cholestasis are characterized by the following biochemical changes:

1) increased activity of alkaline phosphatase, γ-glutamyltranspeptidase (GGTP) and some other excretory enzymes (leucine aminopeptidase, 5-nucleotidase, etc.);

2) hypercholesterolemia, often in combination with an increase in the content of phospholipids, β-lipoproteins, bile acids;

3) hyperbilirubinemia (mainly due to increased concentration of direct bilirubin).

Table 9 summarizes the main criteria for diagnosing the described biochemical syndromes.

Table 9

Basic criteria for the diagnosis of biochemical syndromes in liver diseases

Biochemical criteria	**Syndromes**			
	Cytolytic	**Minor hepatocellular insufficiency**	**Mesenchymal inflammatory**	**Cholestatic**
1	2	3	4	5
Total protein		Sometimes decreased	Often increased	
γ-globulins			Increased	
Albumins		Decreased		
Prothrombin		Decreased		
V and VII coagulation factors		Decreased		
ALT	Increased			
AST	Increased			
LDH and LDH_5	Increased			
Aldolase	Increased			
Cholinesterase		Decreased		
GGTP				Increased
Alkaline phosphatase				Increased
IgG, IgM, IgA			Increased	
Sedimentation tests			Changed	
Non-specific antibodies			Present in blood	
Cholesterol		Decreased		
Bilirubin	Increased (direct)	Increased (indirect)		Increased (direct)
Iron	Increased	Decreased (transferrin)		
Blast-transformation reaction			Changed	
ESR			Increased	

C-reactive protein			Positive	
Seromucoid			Increased	

Blood and urine tests for diseases of the pancreas

Changes manifested in general clinical and biochemical blood tests in diseases of the pancreas are very diverse. In patients with acute and chronic pancreatitis and pancreatic cancer, the most important are the changes in the activity of enzymes, number of leukocytes, leukocyte formula and ESR.

Enzymes. Table 10 shows normal values of blood serum α-amylase, urine and duodenal contents.

Table 10

Normal values of some pancreatic enzymes in serum, urine and duodenal contents

Enzymes	Blood serum	Urine	Duodenal contents
α-amylase	12-32 mg/(h×ml); 3,3–8.9 mg/(s×l)	Up to 160 mg/(h×ml)	1.7–4.4 g/(s×ll)
Lipase	0-28 mcmol/(min×l); 0-160 IU/l; 0–470 nM/(s×l)	—	
Trypsin	60–120 mcmol/(h×ml)	—	50–500 mcmol/ (ml×min)

An increase in the activity of enzymes in serum (amylase, lipase, trypsin, and trypsin inhibitors) is a leading laboratory symptom of ***acute pancreatitis***. The main cause of such hyperenzymemia in these cases is an abnormal outflow of pancreatic juice, due to inflammatory edema of the organ and compression of the pancreatic ducts. Preservation of secretory function of the pancreas is an important condition for hyperenzymemia.

In most cases, in acute pancreatitis, an increase in blood amylase activity is observed in the first hours or days after the onset of the disease. Hyperenzymemia persists for 1-3 days, and then the activity of the enzyme is reduced to normal. This emphasizes the need for the study in the early days of the disease to avoid diagnostic errors. It should be remembered that the level of urine amylases in acute pancreatitis does not always correspond to the increased activity of blood amylase: uric amylase levels can be normal, and blood amylase is sharply elevated. In addition, the normal or low activity of the enzyme in the serum does not exclude the diagnosis of acute pancreatitis, and, most often, the most severe clinical forms of the disease. In these latter cases, it is a question of sharply reduced functional capacity of the pancreas, due to pronounced necrotic changes in the organ (pancreatic necrosis, acute purulent pancreatitis). On the other hand, it should be borne in mind that an increase in the activity of amylase in serum and urine may occur not only in acute pancreatitis.

The most frequent causes of such, more often moderate, increase in amylase levels are (according to N. A. Skuya):

1) an obstacle to the outflow of pancreatic juice (cicatricial stenosis of the duodenal papilla, a stone in the major duodenal papilla, etc.);

2) exacerbation of chronic pancreatitis;

3) injury (wound) of the pancreas;

4) perforation of ulcers of the stomach or duodenum (due to absorption of the enzyme in the abdominal cavity);

5) obstruction of the intestine (absorption of the enzyme);

6) Oddi's sphincter spasm (dyskinesia of the biliary tract or duodenum, bile colic attack, morphine administration, etc.);

7) infectious parotitis;

8) ectopic pregnancy, perforation of tubes;

9) renal failure;

10) diabetic ketoacidosis.

The level of activity of pancreatic *lipase* in acute pancreatitis increases somewhat later than amylase activity, and remains high at about 10-12 days.

An increase in the activity of enzymes in the blood is observed in exacerbation of ***chronic pancreatitis***, although in 25% of patients with this disease the level of amylase and other enzymes in blood is normal or reduced. This is due to insufficient pancreatic function and (in some cases) lack of obstruction for the normal outflow of the gland secretion into the duodenum.

In ***pancreatic cancer***, the level of serum enzymes may be elevated, normal or decreased, due to the spread and localization of the tumor, as well as the presence of concomitant inflammatory and sclerotic processes in the pancreas.

Leucocytes and ESR. Changes in the number and composition of leukocytes are most common in acute hepatitis and exacerbation of chronic pancreatitis. Neutrophilic leukocytosis is common up to $10\text{-}20\times10^9$/l with a shift of the blood formula to rod neutrophils and metamyelocytes and an increase in ESR. The most pronounced changes are manifested in the severe and complicated course of acute pancreatitis (pancreatic necrosis, acute purulent pancreatitis).

Glucose. In acute and chronic pancreatitis and pancreatic cancer, in most cases, there are also significant changes in carbohydrate metabolism, in particular, *hyperglycemia* and *glucosuria*. In *acute pancreatitis*, these disorders are often temporary and due to involvement in the inflammatory process of the islets of Langerhans. Arrest of inflammation, as a rule, leads to the normalization of these indicators. In *chronic pancreatitis* and *pancreatic cancer*, identification of hyperglycemia and glucosuria often indicates the development of irreversible sclerotic changes in the pancreas and the development of secondary diabetes.

In patients with chronic pancreatitis, changes that occur in the so-called *double-glucose loading*, to a certain extent, may reflect the functional state of the pancreas (A.A. Shelagurov). The appearance of a two-hump curve or an excess of glucose levels in the blood for more than three hours indicates a decrease in organ function.

Immunological studies in liver diseases

Immunological studies in liver diseases have recently been given special significance, since any damaging factor leading to a violation of the integrity of the hepatocyte membrane (virus, alcohol, toxic and medicinal substances) cause aa release of a *specific hepatic antigen*, which, in turn, leads to the corresponding changes in the systems of cellular and humoral immunity and development in the liver of the reaction of the increased sensitivity of the slowed-down type in the form of lymphoplasmocytic infiltration of portal paths and intralobular stroma (*immune inflammation syndrome*).

Assessment of *humoral immunity* in clinical practice involves the study of the content of immunoglobulins of different classes, embryospecific globulins and antibodies to DNA, smooth muscle and mitochondria, and *cellular immunity* is evaluated by the number of T- and B-lymphocytes and their populations, inhibition of the migration of leukocytes and blast-transformation reaction.

Assessment of humoral immunity

Serum immunoglobulins. Immunoglobulins are known to be specific antibodies and a group of serum proteins produced by plasmatic cells. Of the five currently studied classes of immunoglobulins, the most practical significance belongs to three classes: IgG, IgA and IgM. Their share accounts for about 85%, 10% and 5% of all serum immunoglobulins.

In diseases of the liver, the concentration of immunoglobulins of different classes increases, which is used mainly to characterize the activity of the *mesenchymal-inflammatory syndrome (immune inflammation syndrome)*. However, the change in the ratio of individual serum immunoglobulins in various liver lesions is not a very reliable differential diagnostic sign of these diseases. However, it should be kept in mind that chronic active hepatitis is most often accompanied by an increase in the content of IgG and (to a lesser extent) IgM, in the primary biliary cirrhosis of the liver there is a predominant increase in the concentration of IgM, and IgA and IgG in alcoholic cirrhosis.

Embryospecific serum globulins. The appearance of the so-called embryonic proteins (α- and β-fetoproteins) in the serum of adult patients suggests:

1) either a sharp increase in *regeneration of hepatocytes*, inherent in the norm only to embryos and fetuses;

2) or the presence of *primary hepatocellular liver cancer* producing embryospecific α-fetoprotein.

In the first case, even in application of modern high-sensitivity methods for the detection of α-fetoprotein (immunoautoradiography, aggregate hemagglutination, etc.), the concentration of this embryonic protein is very low (for example, in acute or chronic hepatitis, liver cirrhosis).

Detection of autoantibodies.

1. *Chronic autoimmune hepatitis* is associated with the appearance of serum antibodies to actin components of the smooth muscle fibers related to IgG.

2. *Acute viral hepatitis* and some other diseases are characterized by the appearance of serum antibodies to actin and myosin of smooth muscle fibers related to IgM.

3. *Primary biliary cirrhosis* of the liver is characterized by the appearance of mitochondrial antibodies in the serum.

4. Detection of antibodies to smooth muscle fibers and mitochondria is not purely specific for the lesion of the liver or biliary system, since these immunoglobulins can be detected in other diseases as well as in healthy individuals.

Markers of hepatitis viruses. Identification of markers of hepatitis viruses is one of the most common and in most cases a compulsory method for the study of patients with liver disease.

Acute viral hepatitis is known to be caused by hepatitis B virus in about 68% of cases, by hepatitis A virus in 12%, hepatitis C virus in 16% and in other cases by the delta (D), Epstein-Barr virus, herpes and cytomegalovirus (B. Ehrlich). At present, the most available method is identification of the markers of hepatitis B virus (HBsAg, etc.), as well as markers of hepatitis A and C. Various methods of clinical immunology are used for this purpose: agar gel precipitation, radioimmune, immunoassay and other methods. The last two methods are the most sensitive. It should be remembered that in the case of obtaining positive results when using one of the methods, they need to be confirmed by other, more sensitive methods.

Hepatitis B virus (serum) is known to have a complex structure and includes three antigens:

1) surface antigen (HBsAg), which forms the outer membrane of the virus;

2) infectious antigen (HBeAg);

3) nuclear or cordiform antigen (nucleocapsid), closely linked to the DNA polymerase of the virus (HBsAg). Markers of hepatitis B virus include all three antigens (HBsAg, HBeAg, HBcAg) and antibodies to each of them (anti-HBsAg, anti-HBeAg, anti-HBcAg).

The most widely used in clinical practice is the *surface antigen HBsAg* corresponding to the Australian antigen, first discovered in 1963 by Blumberg et al in the blood of Australian aboriginal patient with hepatitis B.

Viral hepatitis B. In the blood of patients with acute serum hepatitis B, HBsAg appears long before the clinical signs of the disease (in the incubation period) and persists for several weeks after recovery. Detection of HBsAg in the blood for more than 6 months indicates chronicity. Antibodies to the surface antigen B (*anti-HBsAg*) appear within 3-5 months of the onset of the disease and indicate the development of immunity to hepatitis B virus. Anti-HBsAg antibodies disappear from the blood for 6 years in 20% of patients with acute hepatitis B, and in general are absent in 15% of reconvalescents.

The HBeAg antigen is detected in the incubation period (along with HBsAg) and in the early days of the icteric period of the disease. After a while, HBeAg disappears from the bloodstream and antibodies to this antigen appear (anti-HBeAg). Thus, the presence of HBeAg in the serum indicates a disease, and anti-HBeAg suggests recovery.

The presence of HBeAg and anti-HBeAg in the blood serum indicates that blood and other biological materials (saliva, sperm, vaginal secretion, stool, urine) are significantly infectious, which can cause introduction of infection to people in close contact (including sexual ones) with the patient. Thus, families of HBeAg-positive carriers, as well as patients with detected antibodies to this antigen, are often found to have serological signs of hepatitis B (R. P. Perillo et al.).

Core antigen (HBcAg) is difficult to detect in serum in patients with hepatitis B, but their blood often contains anti-HBcAg antibodies, which appear after 1.5-2.0 months from the onset of the disease. Immunoglobulins IgG anti-HVsAg can circulate over the years in the blood, as a reliable marker for hepatitis B.

It should also be remembered that HBsAg is detected not only in liver abnormalities, but also in patients with nodular periarthritis, hemoblastosis, Down syndrome, and leprosy. There is an assumption about the etiological role of HBsAg in nodular periarthritis.

Viral hepatitis A. The markers of viral hepatitis A (epidemic) include hepatitis A antigen (HAV Ag), antibodies to hepatitis A virus of the IgG class (anti-HAV IgG) and IgM class (anti-HAV IgM). The latter are the main test for specific immunological diagnosis of hepatitis A. *Antigen HAV Ag* is detected in feces in 10-20 days after infection and usually remains there for a short time (no more than 1-2 weeks). *Anti-HAV IgG antibodies* appear in the initial stage of infection and remain in blood for life. *Anti-HAV IgM antibodies* appear in the serum at the beginning of the icteric period and remain for up to 6 months. Thus, detection of anti-HAV IgM antibodies indicates *acute* viral hepatitis A.

Viral hepatitis C. The markers of viral hepatitis C (parenteral) include RNA of virus C (HCV RNA) and antibodies to the virus (anti-HCV IgG). *Antigen HCV RNA* is detected in the acute period of the disease. *Antibodies to C virus (anti-HCV IgG)* are detected in both acute and chronic hepatitis C.

Viral hepatitis Delta. Viral hepatitis delta (D) is caused by a specific (defective) virus (HDV), which becomes viable and virulent only in the presence of another "helper virus" (hepatitis B virus), whose surface antigen (HBsAg) uses the HDV virus to form its own membrane. Infection occurs by parenteral route. Markers of viral hepatitis D are: the antigen of the virus (HDV Ag), the antibodies to IgM and IgG (anti-HDV IgM and anti-HDV IgG) and also the RNA of the virus (HDV RNA). The presence in blood of *antigens HDV RNA, HDV Ag and anti-HDV IgM antibodies* together with the antigen of viral hepatitis B (HBsAg) indicates the *activity of the process in the liver*. *Anti-HDV IgG antibodies* appear later (usually after 12 weeks from the time of infection) and are detected in the blood for many months after the disease.

Chronic hepatitis. In accordance with the current classification of chronic hepatitis, adopted by the World Congress of Gastroenterologists (Los Angeles, 1994), there are the following variants of the disease:

1) chronic viral hepatitis (type B, C, D and viral hepatitis of the unknown type);
2) autoimmune hepatitis;
3) chronic medication hepatitis;
4) cryptogenic (idiopathic) hepatitis.

Other chronic diseases of the liver are included in this group, the clinical and morphological picture of which is very similar to that in autoimmune and viral hepatitis (primary biliary cirrhosis of the liver, Wilson-Konovalov's disease, primary sclerosing cholangitis, α_1-antitrypsin deficiency in the liver).

The share of viral hepatitis accounts for about 80% of all chronic inflammatory diseases of the liver.

The main principle of the given classification of chronic hepatitis is the establishment of the *etiological factor* of the disease, as well as the *degree of activity of the pathological process*. Therefore, in the diagnosis of chronic hepatitis, critical importance is given to immunological studies, as well as to the study of morphological changes in the liver (histological examination of the liver biopsy).

Detection in blood serum of patients with chronic hepatitis of certain combinations of antigens and antibodies allows not only to establish the basic etiological factor of the disease, but also to form an idea about the phase of the pathological process: replication or integration.

Chronic autoimmune hepatitis is characterized by the appearance of tissue non-specific autoantibodies in the blood, which are fixed on hepatocyte membrane. As a result, the lymphocytes themselves attack the liver cells, causing their damage. Laboratory markers of chronic autoimmune hepatitis are:

1. The presence of *autoantibodies to smooth muscle* (SMA) and *antinuclear antibodies* (ANA) in the blood, which is characteristic of the first type of autoimmune hepatitis (in the past it was referred to as "autoimmune chronic active hepatitis", or "lupoid hepatitis").

2. Presence of serum *autoantibodies to hepatic renal microsomes* (ALKM-1), characteristic for the second type of autoimmune hepatitis.

3. Presence of serum antibodies to soluble *hepatic antigen* (SLA) and antibodies to the *hepatic pancreatic antigen* (LP).

4. Absence of serum markers of viral hepatitis B, C and D (HBsAg, HBV DNA, antibodies to viruses C, D, etc.).

Primary biliary cirrhosis of the liver is characterized by the destruction of interlobular bile ducts as a result of granulomatous inflammation. A specific immunological test for this disease is the detection of *anti-mitochondrial antibodies* (AMA) of certain subtypes (anti-M2, anti-M4, etc.) in serum. *Anti-M2 antibodies* are detected only in primary biliary cirrhosis of the liver.

Studies of cellular immunity

Studies of cellular immunity in patients with various liver diseases (chronic viral hepatitis, primary biliary cirrhosis of the liver, etc.) can clarify the involvement of T-lymphocytes in the pathogenesis of damage to hepatocytes and the development of intrahepatic cholestasis. Thus, current methods of studying cellular immunity have established a decrease in the number of T-lymphocytes in peripheral blood and their simultaneous concentration in the liver tissue.

Patients with chronic autoimmune hepatitis and primary biliary cirrhosis in the study of the *reaction of blast transformation of lymphocytes* (BTL) with phytohemagglutinin are found to have a suppressed non-specific reactivity of T-lymphocytes. At the same time, it can be accompanied by delayed hypersensitivity to the hepatic antigen, which reflects the involvement of T-lymphocytes in the development of immune inflammation. Moreover, the severity of BTL, as a rule, corresponds to the activity of the pathological process and the intensity of lymphoid and macrophage infiltration of the liver tissue. This test is used to diagnose the activity of immune inflammation (see above).

In the majority of patients with chronic liver disease the study of the *reaction of leukocyte migration inhibition* (RLMI) shows increased sensitivity of T-lymphocytes to hepatic antigen and microsomal fractions of the liver tissue and hepatic-specific lipoprotein. The latter is known to be a normal component of hepatocytic membrane. Therefore, inhibition of the migration of leukocytes in stimulation by hepatic-specific lipoprotein (or other liver antigen) may also be considered as an indicator of the ongoing cytotoxic action of T-lymphocytes on liver cells and indicates active immune inflammation.

Hepatitis B virus is known to be able to remain in the body in some cases for a long time, without causing the corresponding damage to the liver, which is considered as a virus carriage. When a patient is diagnosed with HBsAg, the clinician should decide whether it is an indicator of a disease (even indolent HBsAg-positive hepatitis) or a virus infection in a healthy person. Diagnosis of liver diseases in these cases is based on the evaluation of clinical picture and the results of complex laboratory and instrumental studies. In this case, the study of humoral and cellular immunity is of particular importance.

Thus, the study of cellular and humoral immunity can be used to clarify the etiology and pathogenesis of liver diseases, differential diagnosis, evaluation of the activity of the pathological process and the state of the immune system.

X-ray examination of the gallbladder and biliary ducts

Several methods of contrasting are used for radiographic examination of the gallbladder and biliary tract. The most informative are cholecystography, cholegraphy and endoscopic retrograde cholangiopancreatography (ERCP).

Cholecystography *is a method of X-ray examination of the gallbladder with administration of an oral hepatotropic X-ray contrast agent containing iodine*. Tablets or powder of contrast agent (e.g., bilitrastum, cholevid, iopagnost, biliminum, etc.) are given to the patient in the evening on the eve of the study. The agent is absorbed into the bloodstream in the intestine, is captured by the cells of the liver and enters with bile the gallbladder. If the latter preserves *concentration capacity*, the content of

contrast agent constantly increases in it and in 12-15 hours X-ray shows an intensive shadow of the gallbladder. To evaluate the *motor function*, the patient is given a special "breakfast" (more often egg yolk), which causes the contraction of the gallbladder.

Intravenous cholegraphy *is an x-ray examination of the biliary tract and bile ducts in intravenous administration of hepatotropic iodine-containing contrast agent of bilognostum,* to create a much higher concentration of the agent in blood and bile. Therefore, in 10-15 minutes after the introduction of the agent X-ray shows a contrast image of the *bile ducts*, and then the *gallbladder*. At the same time, serial radiography shows the whole process of filling the gallbladder: contrasting bile is originally located in its upper part, forming a "horizontal level" on the border with the "old" (unconcentrated) bile, then as if it flows around the latter, partially accumulating at the bottom of the gallbladder, and finally fills the entire gallbladder. In 2-4 hours after the intravenous administration of biliognostum, radiography shows uniform shadow of the gallbladder, completely filled with contrasting bile.

Cholangiography implies *introduction of an iodine-containing contrast medium directly into the biliary tract, for example, by means of puncture of the gallbladder*. It is performed, in particular, by:

1) percutaneous puncture of the bile ducts (through the anterior abdominal wall);
2) laparoscopic cholangiography (puncture of the bile ducts under control of a laparoscope);
3) surgical cholangiography (puncture is performed during surgery);
4) postoperative cholangiography (introduction of contrast substance through a drainage tube left in the biliary tract).

The method of endoscopic retrograde cholangiopancreatography (ERCPG) is described below.

Interpretation of findings. Normally, the gallbladder is located to the right of the median line. The length of the shadow of the gallbladder usually does not exceed 5-8 cm, and the diameter is 2.5-3.5 cm. The shadow is homogeneous, its contours are even and clear. After taking egg yolk, the size of the gallbladder is reduced. Cholegraphy and cholangiography additionally show the right and left hepatic ducts and the common bile duct. The width of the latter does not exceed 0.7 cm in cholegraphy or 1.5 cm in cholangiography.

Diseases of the gallbladder and biliary tract are commonly accompanied by the following radiological changes:

1) stones in the gallbladder and bile ducts;
2) "deactivated"gallbladder;
3) signs of acute and chronic cholecystitis;
4) dyskinesia of the biliary tract, etc.

Stones in the gallbladder or bile ducts containing lime salts can be detected by X-ray examination without additional contrasts. They form rounded ring-shaped shadows, since lime is deposited mainly in the outer layers of stone. When there are many stones in the gallbladder, their surface has a festoon shape.

In the absence of lime salt stones, they are detected only in additional contrasting of the biliary tract. In *intravenous cholegraphy* it is necessary to take into account several possible radiological signs:

1) a defect of filling, appears on the background of the shadow of the gallbladder or bile duct;
2) enlargement of the bile duct above the place of its blockage by stone;
3) disruption of the shadow of the bile duct due to its complete obstruction for the contrast of bile;
4) blockade of the gallbladder ("deactivated" gallbladder) due to blockage of the duct with stone.

"Deactivated" gallbladder. Often, oral or intravenous contrast (cholecystography and cholegraphy) does not provide a possibility to obtain a picture of the gallbladder - it does not contrast. These cases are referred to as "deactivated" gallbladder.

It should be borne in mind that there are two more clinical situations, in which it is not possible to detect not only the shadow of the gallbladder, but also intra-and extrahepatic bile ducts:

1) presence of *parenchymal jaundice* (a decrease in liver function, which contributes to a decreased elimination of contrasting substance by liver cells);

2) *disrupted absorption in the intestine* of the contrast agent administered *per os* (in cholecystography).

Acute cholecystitis. In patients with acute cholecystitis, in oral or intravenous contrast, the shadow of the gallbladder is usually not detected ("deactivated" gallbladder), while the shadow of the bile ducts is preserved. In *destructive cholecystitis* it is often possible to detect *indirect radiological signs:* limited bloating of the right half of the transverse colon and bloating of the small intestine loops with the formation of typical small "arches".

Chronic cholecystitis is radiologically manifested by a disturbance of concentration function of the gallbladder: intravenous cholegraphy shows inhibited or, conversely, sharply accelerated filling of the gallbladder. The filling of the gallbladder occurs from the bottom up, at a certain stage of contrast there is no clear three-layer shadow. There are also signs of a change in its motor function. The volume of the gallbladder increases, and its shadow becomes less intense. Often, thickening of the walls of the gallbladder is also observed.

Dyskinesia of the biliary tract is manifested by impaired realease of bile from the liver and gallbladder. There are two types of dyskinesia of the biliary tract. *Hypertonic dyskinesia* is characterized by an increase in the tone of the gallbladder and biliary duct, spasm of Oddi's or Lyutkens' sphincter, a non-rhythmic emptying of the gallbladder. After taking choleretic "breakfast" the gallbladder sharply and rapidly contracts (for the first 5-15 minutes to 75%, and within 1.5-2 hours up to 90% of the initial volume), and then for a long time loses its ability to be emptied, being in a state of hypertension. On the contrary, *hypotonic dyskinesia* is accompanied by a decrease in tone and motor activity of the gallbladder and bile ducts. There is an increase in the initial size of the gallbladder and delayed emptying: 15 minutes after taking choleretic "breakfast" the volume of the gallbladder decreases by 20-30% and is maintained for the next 3-4 hours.

X-ray examination of the liver

Ordinary X-ray examination of the abdominal cavity is not very informative in diseases of the liver. The essential diagnostic value belongs only the contrast of the esophagus and the stomach, which is used to detect the *enlarged veins of the esophagus and the cardiac part of the stomach* in portal hypertension syndrome.

Several methods of **selective angiography –** *X-ray examination of the liver with artificial contrasts of its vessels* (celiacography, splenoportography, hepatovenography, etc.) are used to diagnose vascular pathology of the liver, clarify the nature of focal liver formations and its developmental defects, especially when deciding on possible surgical treatment. Finally, **X-ray computed tomography of the abdominal cavity** has recently become an important method in examination of patients with liver diseases.

Celiacography is used primarily for the study of arterial blood flow of the liver and spleen. Femoral artery is punctured under local anesthesia and a catheter is introduced into the mouth of the abdominal artery under X-ray control. 65% solution of hypaque or 60% solution of urotrast is used as an X-ray contrast agent, which in the amount of 30-50 ml is injected into the abdominal artery. Within 20-22 seconds, a serial X-ray is taken to study three phases of blood flow: arterial, parenchymatous (capillary) and venous.

The results of celiacography can be used to diagnose liver diseases, primarily focal formations. The most commonly celiacography is used to reveal the following changes of angiographic picture:

1. *Areas of local depletion* or even absence of a *circular, oval or irregularly shaped vascular pattern* most characteristic of alveococcosis, echinococcosis and benign liver tumors (hemangiomas). Often, at the edges of such vascular areas, the vessels are compressed, having arc shape and clear contour levels.

2. *Areas of hypervascularization of the liver* due to the formation of new, proper tumor blood vessels and slowing blood circulation in this area (malignant neoplasms of the liver, liver metastases). Characteristic is the prolonged delay in contrasting substance in the lesion area, especially noticeable

in the parenchymal (capillary) phase of blood flow. It should be borne in mind that neoplasms and metastatic liver lesions may also occur in non-vascular areas.

3. *Aneurysm* of the hepatic or splenic arteries.

4. *Stenosis* of the abdominal trunk and its branches.

5. *Diffuse depletion* of the arterial vascular pattern of the liver, narrowing of the partial and segmental arteries, vascular flexuosity, signs of redistribution of blood flow from the trunk of the abdominal artery towards the spleen (narrowing of the extraorgan department of the hepatic artery, significant enlargement of the abdominal and splenic arteries, increase in the spleen contrast in the parenchymal phase of the blood flow). Such changes are often found in liver cirrhosis.

6. Significant *enlargement* of the abdominal, common bile, proper hepatic, splenic and left ventricular arteries, accompanied by diffuse intensification of the vascular pattern of the liver and spleen due to the enlargement of segmental arteries and their branches of order I and II, as well as increased intensity of contrast of the liver and spleen in the parenchymatous phase of blood flow with uneven droplet structure of the liver. Such angiographic picture is most common in portal hypertension syndrome in patients with cirrhosis of the liver. At the same time in the venous phase, the contraction of blood vessels in the region of the cardiac part of the stomach and the lower third of the esophagus may be contrasted, indicating the development of portocaval anastomosis.

Splenoportography is a method of studying portal blood flow by introducing a contrast agent directly into the spleen pulp during its puncture.

The spleen puncture is performed under local anesthesia in the IX-X intercostal space on the left in the posterior inguinal line. After insertion into the spleen of a contrast medium, a series of X-ray images at intervals of 1.5-2 seconds is performed.

It should be remembered that the most dangerous complication of splenoportography is bleeding into the abdominal cavity, so one of the contraindications to the study is a disruption of blood coagulation.

Splenoportography makes it possible to study in detail the features of blood flow in the spleen, portal vein and its intrahepatic branching, to determine the position, size, contours and permeability of these vessels. The network of collateral vessels, by which the contrast substance is discharged into the system of hollow veins (cirrhosis of the liver) begins to contrast in the presence of any barrier to the blood flow.

Cirrhosis of the liver is characterized by the enlargement of the portal vascular bed, although the vein passage is preserved. There is a deformation of intrahepatic branching of the portal vein and a decrease in the number of vessels of order III and IV.

In ***thrombosis of the spleen or portal vein***, they do not contrast, and numerous venous collaterals are determined in the region of the splenic hilum and (or) along the splenoportal trunk.

Thus, splenoportography is one of the most valuable methods of X-ray examination in portal hypertension syndrome.

Hepatovenography (liver phlebography) is a method of artificial contrast of hepatic veins.

The method is the most informative for the diagnosis of *Budd-Chiari syndrome* (hepatic vein thrombosis with a disruption of the outflow of blood from the liver and the development of severe portal hypertension).

Hepatovenenography involves percutaneous puncture and catheterization of the femoral vein. Under X-ray control, the catheter is introduced into the ileum, inferior vena cava and then into one of the hepatic veins. After administration of contrast media, several X-rays are taken at intervals of 0.75 seconds. Then, if necessary, the probe is transferred to another vein of the liver and the study is repeated.

Contraindications to percutaneous hepatovenography include severe condition of the patient, acute infectious diseases, mental disorders, increased sensitivity to iodine agents, as well as thrombophlebitis of pelvic veins and lower extremities, thrombosis of the inferior vena cava.

In ***Budd-Chiari disease***, hepatovenenography can determine the direct signs of thrombosis and occlusion of hepatic veins.

X-ray examination of the pancreas

Ordinary X-ray examination of the abdominal cavity, even with the use of additional techniques (pneumoperitoneum or pneumoretoperoperoneum involving the introduction of gas, respectively, into the abdominal cavity and into the retroperitoneal cellular tissue), rarely reveals the shadow of the pancreas. The exception is calcification of the organ, which is well manifested even on revision X-rays and, especially, on localized tomography.

Interesting are several *indirect x-ray signs* that are detected in acute and chronic pancreatitis and pancreatic cancer indicating an increase in organ size and the presence in some cases of concomitant changes in the small and large intestine, including intestinal obstruction.

So, ***acute pancreatitis***, which is accompanied by inflammation and edema of the pancreas, is characterized by:

1) angulation of the stomach and its protrusion forward and upward with enlarged pancreas;

2) reversal of the duodenum and smoothing of its internal contour, due to swelling of the head of the pancreas;

3) bloating of individual loops of the small intestine (*symptom of "recurrent loop"*);

4) bloating of the areas of the large intestine with irregular fluid levels in them, which are predominantly at the level of the second-fourth lumbar vertebra to the right or to the left of the spine, depending on the damage to the head or tail of the pancreas;

5) high standing of the left dome of the diaphragm and reduction of its excursion, sometimes accumulation of exudates in the left pleural cavity (A.A. Shelagurov);

6) darkening of the upper floor of the abdominal cavity and irregular contours of the liver, kidneys and large lumbar muscles;

7) regional spasm of the transverse colon in combination with severe bloating of the liver and spleen (*symptom of a "truncated loop"*) or with limited bloating of the ascending colon and liver with a clear limit of visible gas (*symptom of the "truncated intestine"*). The last two signs, as a rule, are manifested in severe forms of acute pancreatitis.

It should be remembered that each of the following indirect signs is not specific for acute pancreatitis. Diagnostic value belongs only to a combination of several radiological signs, as well as their correspondence to the clinical picture of the disease.

Chronic pancreatitis results in nonspecific radiological signs indicating compression and displacement of the stomach and duodenum and violation of their motor function. Particular importance is attributed to certain features that occur during **relaxation duodenography**, which is carried out in conditions of artificial hypotonia.

In the normal course of this study, the wall of the intestine, including its descending part, enveloping the head of the pancreas, forms an even, slightly wavy contour. Enlargement of the head of the pancreas is accompanied by its straightening, evenness of the contour, a defect from pressure on the intestinal wall. Diagnostic value belongs to the following radiological signs:

1) increase in the unfolding of the duodenal loop, "straightening" off the inner contour of the descending part of the intestine;

2) limited compression on the inner contour of the duodenum;

3) filling with barium of the ampulla of the major duodenal papilla, and sometimes common bile and Wirsung ducts;

4) an increase in the size of the major duodenal papilla;

5) deformation of the folds of the mucous membrane of the duodenum.

The listed X-ray signs are non-specific for chronic pancreatitis and can be detected in the event of an increase in head size in ***pancreatic cancer***. Cancer of the body of the pancreas causes displacement or germination of the lower horizontal part of the duodenum. Circulatory tumor infiltration disrupts the function of the duodenum and can cause long-term duodenostasis.

Significant changes in the x-ray pattern in pancreatic cancer are detected during **angiography**, which provides a direct image of the pancreas. These include a violation of the regularity of the vascular pattern, narrowing and inequality of the contour and displacement of blood vessels, development of the newly created vascular network, a delay of the X-ray contrast agent in the venous

phase (A.N. Mikhailov). **Splenoportography** detects the displacement and compression of the splenic vein and its blockage.

Endoscopic retrograde cholangiopancreatography (ERCPG) and computed tomography of the abdominal cavity organs are of great informative value in pancreatic cancer and chronic pancreatitis.

Endoscopic retrograde cholangiopancreatography (ERCPG) is one of the most important modern methods for diagnosing pancreatic biliary disorders. The method consists in *retrograde filling of the contrast agent of the bile ducts and pancreatic ducts under visual inspection through the endoscope*. At the same time, ERCPG makes it possible to study the state of the mucous membrane of the esophagus, the stomach, duodenum, as well as the major duodenal papilla and pancreatic ducts. If necessary, it is possible to carry out an objective biopsy of these organs with subsequent cytological and histological examination. In addition, the method can be used for endoscopic surgical removal of stenosis at the level of sphincter, removal of stones from ducts, etc.

In suspected ***choledocholithiasis and stenosis of the bile ducts*** ERCPG helps to establish the cause of obstructive jaundice: a common bile duct stone, stenosing papillitis, cancer of the major duodenal papilla, stenosis of the terminal portion of the common bile duct, primary sclerosing cholangitis. If mechanical jaundice is associated with stenosis of the major duodenal papilla or large stones in the common bile duct, an **emergency endoscopic papillosphincterotomy** and, if necessary, extraction of a common bile duct stone should be performed immediately after diagnostic ERCPG.

ERCPG is often performed to establish the true cause of clinical manifestations of the so-called ***postcholecystectomy syndrome*** (pancreatitis, cholangitis, choledocholithiasis, stenosing papillitis, etc.).

The method of conducting ERCPG is described in the special instructions on endoscopy, but the practitioner should be focused on the possibilities of this method, its indications and contraindications.

ERCPG is one of the most accurate methods for diagnosis of chronic pancreatitis, and in these cases, its informativity exceeds the possibility of ultrasound examination of the pancreas. The criteria for chronic pancreatitis during ERCPG are irregularities of the main duct and lateral branches, signs of duct obstruction, formation of cavities, filling defects and dilation of ducts. A more reliable diagnosis of chronic pancreatitis involves a combination of ERCPG with a functional test with secretin.

In ***pancreatic cancer***, ECPC reveals significant unevenness in the lumen and disturbances in the structure of the terminal portion of the pancreatic duct.

ERCPG is also useful as a preoperative examination of patients with ***pseudocysts of pancreas***. The method makes it possible to accurately determine the location of ducts, the degree of their obstruction, presence of pseudocysts that are connected and not connected with them.

ERCPG method is relatively safe, although it should be kept in mind that sometimes the study may be associated with such complications as acute pancreatitis, sepsis, anaphylactic shock following introduction of a contrast agent, etc. In this regard, it is recommended to take into account a number of contraindications.

ERCPG is *contraindicated* in:

1) acute pancreatitis;

2) acute myocardial infarction, stroke, hypertensive crisis, circulatory failure and other severe patients;

3) intolerance to iodine agents.

To reduce the risk of septicemia, patients undergoing ERCPG are administered antibiotics for prophylactic purposes 24 hours prior to the study and within 1-2 days after it (1.0 ampicillin 3-4 times a day or 80 mg gentamicin 3 times a day).

X-ray computed tomography (CT) in diseases of the liver and pancreas in most cases (although not always) has more informative value and resolution capacity than conventional X-ray methods, and is usually used in diagnostic cases, especially in the differential diagnosis of ***focal hepatic formations*** (*alveococcosis, echinococcosis, liver hemangioma, cyst, primary liver cancer, metastatic organ damage*, etc.) and pancreas (suspected cancer or cyst, etc.). In ***diffuse damage of the whole liver parenchyma*** (*cirrhosis, fatty hepatosis, hepatitis* and other diseases), a computer

tomography allows to better describe the uneven structure of the organ, as well as to make a quantitative idea about the density of pathological formations of the liver, which helps in the differential diagnosis of chronic hepatitis, liver cirrhosis and fatty liver disease.

For example, in ***fatty liver dystrophy***, its shadow becomes less intense than normal, and the so-called densitometric index that quantitatively reflects the density of the investigated organ is an average of 20 units HU. Against the background of parenchyma with reduced density, the intrahepatic vessels look like structures of high density, that is, there is a reverse, compared with the norm, ratio of density. It is believed that such a picture is pathognomonic for fatty hepatosis allowing to accurately differentiate this condition from hepatitis and liver cirrhosis (A.N. Mikhailov).

In ***hepatitis and cirrhosis of the liver***, computer tomography can detect an increase in the size of the liver, spleen, unevenness of the structure of the liver, signs of portal hypertension. Moderate reduction of the densitometric index to 30-50 units HU is also typical.

Visual and quantitative assessment of the density of the structures visualized by computed tomography involves the employment of the scale of linear attenuation coefficient, which is called the *Hounsfield scale* (its black-and-white spectral representation is clearly visible on the monitor). The range of scale units ("*Hounsfield units*"), which corresponds to the degree of attenuation of X-rays by anatomical structures of the body, is from -1024 to +3071, that is, 4096 numbers of attenuation. The average score on the Hounsfield scale (0 HU) corresponds to the density of water, the negative values of the scale correspond to air and fatty tissue, positive to soft tissues, bone tissue and a more dense substance (metal). In practice, the measured attenuation may vary slightly on different devices. Densitometric analysis is carried out using standard mathematical programs. The density of anatomical structures and pathologically altered tissues is measured in the "region of interest" (ROI) of rounded or, if necessary, arbitrary form. The larger the area of interest in relation to the size of the pathological formation, the more objective is the measurement of density. The average value of the densitometric indices (mean) and the standard deviation (SD) must be taken into account. It allows to divide all pathological formations into homogeneous and heterogeneous ones. Fibrous structures are considered to be homogeneous if the standard deviation does not exceed one third of the average value of their density. Usually, this figure should not exceed 10-15 HU.

Particular difficulty is the detection of ***cancer of the pancreas head*** due to insufficient restriction of this part of the gland from surrounding structures and imposition of their image on each other (A.N. Mikhailov). It is also necessary to take into account that the density of tumor tissue and parenchyma of the pancreas differ little from each other, which is reflected in the close values of the densitometric index of these tissues.

For better visualization of the head of the pancreas in suspected tumor, it is recommended to conduct the study in the patient's position on the side or abdomen with the additional intake of 3% water-soluble X-ray contrast agent for contrasting the small intestine loops.

In ***cancer of the major duodenal papilla***, the enlargement of the liver and total bile ducts, enlargement of the gallbladder, defect of filling in the lower part of the duodenum and other signs are detected.

It should be remembered that computed tomography does not replace, but only substantially complements other methods of X-ray examination of the liver and pancreas (selective angiography, contrast study of biliary tract and gall bladder, ERCPG, etc.).

X-ray computer tomography is not indicated for the diagnosis of such diseases and syndromes as:

1) acute inflammation of the gallbladder and biliary tract (acute cholecystitis, empyema of the gallbladder, cholangitis);

2) "deactivated" gallbladder;

3) hydropic gallbladder;

4) dyskinesia of the biliary tract;

5) violations of the concentration function of the gallbladder.

In these and in some other cases, the above-mentioned X-ray methods of investigation are more informative.

Radionuclide studies

Radionuclide methods are used to detect morphological changes in the liver and pancreas, as well as to evaluate the function of these organs. Depending on the purpose of the study, the following techniques are used in the clinical practice:

1) hepatography - to study the absorption and excretory function of the liver;
2) radioporthography - to study portal circulation;
3) radionuclide scan of the liver – to study portal blood circulation;
4) radionuclide scan of the pancreas.

The great advantage of radionuclide research methods is their complete safety for the patient: the radiation load in most cases is much lower than in the usual X-ray examination.

Hepathography is used to study the absorption and excretory function of the liver with the help of a radioactive agent that is injected into the vein. The study predominantly employs Bengali pink, labeled ^{131}I. About 95% of the agent is injected into the bloodstream, is absorbed by hepatocytes and excreted into the small intestine with bile.

Bengali pink, labeled ^{131}I, is given intravenously at a dose of 0.2 μCu per 1 kg body weight. To measure radioactive radiation over the area of the heart, liver and small intestine (in the navel region), three sensors are installed. This gives a possibility to register the curves of changes in the radioactivity of the blood, liver and small intestine. Continuous recording takes place within 60-90 minutes. The curve of changes in radioactivity recorded over the area of the liver, shows an ascending curvature hepatogram, which, after reaching the maximum radioactivity smoothly passes into the plateau, and then in the descending curvature. The ascending part of the curve reflects mainly the rate of blood flow in the liver and its absorption capacity, and the downward part - the excretory function of the liver and the rate of excretion of the agent into the small intestine. *Normally*, the half-life of the isotope absorption of the liver is 8-16 minutes, the output is 75 minutes; the maximum absorption occurs in 20-30 minutes after the introduction of the drug, and the duration of the plateau does not exceed 6-9 minutes. Significant changes in these indicators are detected in various diseases of the liver, accompanied by a decrease in absorption and excretory function of hepatocytes and (or) disturbance of bile outflow through the intrahepatic and extrahepatic biliary system.

Diffuse liver diseases (*acute and chronic hepatitis, cirrhosis of the liver, etc*.) are characterized by slowdown in both absorption and elimination of the radioactive isotope: the curves have a more smooth and long rise and descent, after which the maximum radioactivity occurs, and the duration of the hepatogram plateau increases. Patients with cholestatic hepatitis and primary biliary cirrhosis of the liver are most strongly affected by the excretory function of the liver.

In various variants of ***obstructive jaundice***, due to obturation of extrahepatic biliary tract, elongation of the descending curvature is observed in the hepatogram, whereas the ascending curvature and the time to reach maximum radioactivity are almost unchanged.

Radioportography. **Intravenous radioporthography** is a method of radionuclide study of the portal circulation by administering intravenous albumin of human serum, labeled $_{131}$I. Special sensors, installed above the area of the heart and liver, are used to register the corresponding curves of radioactivity changes. Portal circulation is characterized by calculating the so-called *cardioportal time*, the time from the peak of the activity curve, recorded over the area of the heart, until the maximum invrease in the activity curve over the liver. *Normally*, it is *23-29 seconds*. Significant prolongation of cardioportal time is observed in chronic hepatitis, liver cirrhosis and other diseases that are accompanied by portal circulatory disorders, as well as in the portal vein thrombosis.

The method of **intrasplenic radioporthography**, which a direct study of portal circulation in intrasplenic administration of radioactive ^{131}I-hippuran, is more informative, though more complicated. For this purpose, under the local anesthesia, it is necessary to make a puncture of the spleen and introduce a radioactive drug inside the spleen. The curvature changes in radioactivity are used to calculate the so-called *splenic-liver time*, which reflects the rate of blood flow in the spleen and portal veins, and which is *normally 2-6 seconds*, as well as the duration of the plateau of the activity curve above the liver, which characterizes the time of blood transfusion through the liver. In various

diseases, accompanied by impaired portal circulation, both spleen-portal time (more than 6 seconds), and the duration of the plateau of radioactivity over the area of the liver are significantly increased.

Finally, for the quantitative determination of blood flow in the liver, the **method of intravenous administration of colloidal gold ^{198}Au**, which is predominantly absorbed by the reticuloendothelial cells of the liver, is used. At the same time, the rate and nature of such absorption also depend on the state of portal circulation. *Normally*, the liver blood flow is *1.5-1.8 l / min* in men. In diffuse liver disease, the magnitude of this indicator is significantly reduced.

Radionuclide scanning of the liver is a rather informative method for assessing the size, shape and structure of the liver and spleen by examining the nature of the distribution of radionuclides in the tissues of these organs. The study predominantly involves administration of a solution of colloidal gold ^{198}Au, which is selectively absorbed by the reticuloendothelial cells of the liver after intravenous administration. Other radionuclide agents are also used (^{131}I-Bengali pink, ^{98m}Tc-colloid, etc.). After intravenous administration of the radioactive drug for 60-90 minutes, the time course of its accumulation in the liver and spleen is recorded, using special gamma-cameras that allow visualization of the diffusion of radionuclides in these organs.

Scanograms of normal liver show the maximum accumulation of radionuclide in the center of the right particle, less in the left lobe and on the periphery of the organ. Normal spleen is not visualized in the ^{198}Au study.

*In **focal lesions of the liver** (echinococcosis, abscess, primary and metastatic liver cancer, benign tumor, post-traumatic hematoma of the liver, etc.),* scanograms reveal defects in the accumulation of the agent. It should be remembered, however, that the permissive ability of radionuclide scanning to detect focal hepatic formations is lower than in computed tomography: only cells that are larger than 30-40 mm are well visualized. In these cases, the sensitivity of the method is 65-90%.

*In **diffuse liver lesions** (hepatitis, fatty hepatoses, cirrhosis of the liver)* scanograms show an increase (less often a decrease) in liver size, violation of its configuration, a decrease in the contrast of the image and, in some cases, uneven distribution of radionuclides.

In most cases, liver cirrhosis is characterized by an uneven increase in the right and left portions of the liver. Most often, the left part increases to a greater extent, and such a violation of the organ configuration persists even in a significant decrease in the overall size of the liver. In chronic hepatitis and fatty hepatosis, enlargement of the liver is also observed, but the form and configuration of the organ as a whole do not change.

In micronodular and coarde nodular cirrhosis of the liver there is a more or less uneven distribution of the isotope in the liver tissue. Radionuclide is almost completely absent from the periphery of the organ. Often, zones of dense hatching alternate with areas of almost complete absence.

In all forms of liver cirrhosis, there is also an increased accumulation of ^{198}Au in the spleen, which is uncharacteristic for patients with chronic hepatitis and fatty hepatosis.

Finally, in all forms of diffuse liver damage, the contrast of the image is reduced, due to a decrease in the absorption of isotopes by the cells of the liver, reflecting a decrease in its function.

Radionuclide scanning of the pancreas. Radionuclide scanning of the pancreas involves intravenous administration of *methionine, labeled with a radioactive isotope of selenium (Se^{75}).* Accumulation of the radioactive agent in the pancreatic tissue is recorded in 30 minutes after the introduction of Se75. The study is used to determine the shape and size of the gland, to detect a violation of its structure in the form of uneven distribution of radionuclide in the tissue of the organ, and indirectly assess the function of the pancreas by the rate of accumulation of the isotope and its excretion into the intestine in the pancreatic secretion.

In ***inflammatory and dystrophic lesions of the pancreas***, the studies show a significant reduction of absorption and accumulation of labeled methionine and a pattern of uneven distribution of radionuclide in the pancreatic tissue. In violations of the outflow of secretion by pancreatic duct there is a significant delay in the withdrawal of the agent from the pancreatic tissue. In ***focal lesions of the***

pancreas (cyst, cancer of the head or body) scanograms often reveal local defects in the accumulation of the agent.

Ultrasound examination

Modern ultrasound (US) technology allows to assess the shape, size and location of the abdominal organs, such as liver, gallbladder, pancreas, spleen, etc., with high diagnostic accuracy, to detect focal formations (liver cancer, pancreas, tumor metastases, abscesses, cysts, hematomas, adenomas, etc.), to evaluate the density and structure of the parenchyma of the liver and pancreas with their diffuse lesions, to diagnose even small amounts (100-200 ml) of free fluid in the abdominal cavity, to identify calculi in the bile ducts, to evaluate changes in the large vessels, bile ducts, and so on.

Over recent years, ultrasound has been widely used in clinical practice as a method that helps to select optimal access for conducting puncture biopsy of the liver, drainage of the abdominal cavity and other procedures.

Preparation of the patient. In 3 days before the examination the patient is advised to exclude from the diet milk, black bread, fruit and vegetables, sweet juices and other products that contribute to the formation of gas in the intestine. In predisposition to flatulence, enzyme aagents (festal, panzinorm, etc.) and adsorbents (activated charcoal, infusion of chamomile, etc.) should be prescribed. In the evening prior to the study and in the morning, immediately before the study the patient is administered two cleansing enemas. However, this procedure is not obligatory in the absence of flatulence. If emergency ultrasound examination is required, special preparation of the gastrointestinal tract is not performed. It should be remembered that ultrasound examination of the abdominal cavity should be carried out not earlier than 2 days after x-ray examination of the stomach with contrast or esophagogastroduodenoscopy, and 3-5 days after laparoscopy or pneumoperitoneum. The study ends with scanning of the abdominal cavity to detect free fluid.

Ultrasound examination of the liver

Normally, the contours of the liver are almost entirely clear and even. The liver has a homogeneous structure with uniformly distributed signals of the same intensity, with the image of echostructure (vessels, ligaments, ducts). Constantly, the lower hollow vein appears in the form of a strip-shaped echo-negative formation with a diameter of up to 15 mm.

The portal vein, after its formation, from the superior mesenteric and splenic veins, flows into the liver hilum, which appear at the transverse and sagittal position of the probe. Intrahepatic ducts are normally traceable, their lumen is enlarged from the periphery to the liver hilum. Unlike the veins, intrahepatic ducts are devoid of walls.

Thus, a normal ultrasound picture of the liver is characterized by the presence of small, non-intensive, located relatively far from each other echo-signals, leaving echo-negative spaces between them. Echo-signals are homogeneous in size and uniformly distributed throughout the liver. Portal vessels are traced along the periphery of the liver; echo-structure of their walls is more marked than the echo-structure of the surrounding parenchyma of the liver, the sound conductivity of the liver is completely preserved; sagittal size is 9-12 cm; the liver is elastic and has a smooth, clear contour.

Assessment and interpretation of the study. ***Diffuse liver disease***. The most common diffuse lesions of the liver include *hepatitis (acute and chronic), fatty dystrophy and liver cirrhosis*. The correct diagnosis in an ultrasound study depends on a number of objective and subjective reasons. The former include the type of device, its sensitivity, resolution, the presence of factors that impair the image (obesity, gas formation in the intestine, etc.). Of great importance are the experience of a specialist and the diligence of the study. The greatest difficulty is the diagnosis of *early stages* of fatty dystrophy and liver cirrhosis.

In ***acute and chronic hepatitis***, the echographic picture is very nonspecific. It is usually determined by an increase in the liver in one or both parts, round shape of its edges. Echo-structure is often normal, poorly echogenic. Only in prolonged course of the disease echo-structure of the liver becomes “striated” with alternation of areas of poor and high echogenicity. In some cases, in particular, with the development of portal hypertension, it is possible to detect enlargement of the spleen and the enlargement of the spleen and portal vein.

Fatty liver dystrophy (fatty hepatosis). The main echographic feature of fatty liver dystrophy is the intensification of echo-structure of the liver in the form of a uniform increase in the number and size of echo-signals. This is due to deposition of fat in the liver lobes, the distance between them and their size increase to such an extent that the ultrasound waves are reflected from them.

The important but less specific features include enlargement of the liver, an increase in the lower angle of the left part by more than 45°, irregularity of the contours of the liver and the impossibility of detecting the portal vein.

Echo-pattern of fatty liver dystrophy depends on the degree of involvement of the liver cells. At the first stage of the disease, the liver is slightly enlarged, the edges are rounded. The echo-structure has a striated pattern, the parenchyma is unevenly compacted in local foci. This is the so-called *"islet" type of liver damage* that occurs in hepatitis. At the second stage, the liver is much larger, the lower edge is rounded, the structure of the parenchyma is small, the liver is diffusely and evenly condensed. At the third stage of the disease, the liver is of considerable size due to an increase in both lobes. It has a rounded shape. The structure of parenchyma is of high density (echogenicity), portal vessels are not detected.

Liver cirrhosis. There are direct and indirect echographic signs of liver cirrhosis. The diagnosis of liver cirrhosis is considered to be reliable if 3 direct or 2 direct and 2 indirect signs of the disease are detected at ultrasound examination (Table 11).

Table 11

Direct and indirect echographi signs of liver cirrhosis

Direct signs	**Indirect signs**
Changes in the size of the liver	Enlargement of the spleen
Changes in echo-structure	Ascites
Irregularities of the liver contours	Enlargement of the portal and splenic veins
Round shape of the lower edge	
Reduced elasticity and sound conductivity	

In most cases, the size of the liver is enlarged, often mainly in the left lobe of the liver. In the final stage of the disease, with the predominance of atrophic processes, the size of the organ decreases. Characteristic feature is an evident *round shape of the lower edge of the liver and irregularity of its contours*.

The echo-structure of the liver is substantially enhanced by the appearance of more frequent and large echoe-signals, which is associated with a significant restructuring of its architectonics, which is typical of cirrhosis. At an atrophic stage, the number and size of echo-signals decrease.

Finally, important signs of cirrhosis include *reduced elasticity and sound conduction of the liver*.

Indirect echographic signs of cirrhosis are associated mainly with the development of portal hypertension syndrome. Enlargement of splenic vein by more than 10 mm and portal vein by more than 15 mm are considered as reliable signs of increased pressure in the *v. porta* system.

Enlargement of the spleen and intensificatkion of its echostructure are observed in 60-70% of cases of liver cirrhosis, although this sign is specific not only for portal hypertension.

Ascitic fluid in the abdominal cavity at ultrasound examination looks like an echo-negative structure, which is accumulated in the abdominal parts of the abdomen, in the small pelvis or (with small amounts of fluid) located around the liver. In these cases, it is expedient to conduct the study by changing the position of the patient's body (lying and standing).

"Congested" liver. In all cases of congestive heart failure there is an increase in the size of the liver and rounding of its edges. The pathognomonic sign of the "congested" liver is the enlargement of the inferior vena cava and hepatic veins, branching of the liver veins at an angle close to 90°C. Characteristically, the inferior vena cava loses the ability to change the diameter in breathing: it does not narrow at all in inspiration, or narrows very little.

Focal changes in the liver. Ultrasound examination of the liver in focal changes in the liver is more informative than in diffuse lesions. In this case there is a local decrease or intensification of echo-structure, diffuse or focal enlargement of the size of the liver and inequality of its contour with the appearance of convexity. Volumetric focal processes in the liver can cause compression of the bile ducts with the development of obstructive jaundice.

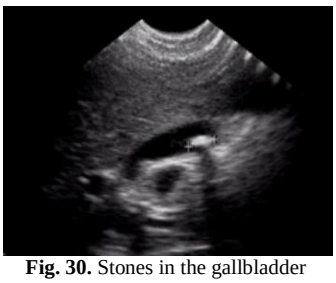


Fig. 30. Stones in the gallbladder and induration of its walls

The most common sign of cellular changes in the liver is a violation of the normal echo-structure of the liver. There are several types of focal disturbances of echo-structure.

1. Foci lacking echo-structure (liver cysts, hematoma, liver abscess, necrotized tumors).

2. Foci with reduced echo-structure (low-differentiated cancer metastases, sarcoma, malignant lymphoma, hepatocellular carcinoma, adenoma, hemangioma, abscess, hematoma, etc.).

3. Foci with intensified echo-structure (metastases of highly differentiated cancer, hepatoma, adenoma, hemangioma, scarring, foci of calcification).

4. Symptom of "target" – a reduction of echo-structure on the periphery of the focus and intensification in the center (malignant tumor of the liver).

Thus, the informativeness of ultrasound examination of the liver is quite high, especially in focal lesions of the organ. Nevertheless, it is necessary to take into account that there can be possible mistakenly positive and mistakenly negative conclusions. Therefore, the analysis and interpretation of the results of the study should take into account the clinical picture of the disease as a whole, as well as other laboratory and instrumental findings.

Ultrasound examination of the gallbladder and bile ducts

Ultrasound examination of the gallbladder and bile ducts has certain advantages over radiological examination (cholecystography, intravenous cholegraphy, etc.), since it completely eliminates exposure to radiation, is possible for children and pregnant women, as well as in the reduction of liver and kidney function.

The most common indications for the study of the gallbladder and bile duct are as follows:

1) acute and chronic cholecystitis;
2) cholelithiasis;
3) jaundice;
4) tumor;
5) dropsy and empyema of the gallbladder;
6) state after cholecystectomy or other operations on the biliary tract.

The study of the gallbladder involves evaluation of its condition, shape, size, respiratory mobility, the state of external and internal contours, wall thickness, wall structure, additional inclusion in the cavity of the gallbladder, evacuation function of the organ.

The study of *extrahepatic bile ducts* determines their position, diameter, wall state, and the presence of additional inclusions in the lumen.

Normally, the gallbladder appears as an echo-negative structure on the dorsal surface of the right lobe of the liver. The bottom of the gallbladder often extends from below the lower edge of the liver to 1.0-1.5 cm. Its length does not exceed 7-10 cm, and the width is 3-4 cm. The gallbladder has an elongated pear-shaped, oval or rounded form, clear and equal contour

Intrahepatic bile ducts are not detected in a healthy person. The diameter of the common hepatic duct does not exceed 3-5 mm, and the common bile duct is 4-6 mm.

Acute cholecystitis. Characteristic echographic signs of acute cholecystitis are thickening of the wall of the gallbladder by more than 4 mm. Its size may remain normal or even diminished, although a small increase in the gallbladder is more common.

Echo-structure of the gallbladder, primarily its internal contour, is usually reduced. In phlegmonous cholecystitis, the internal and external contours of the gallbladder are nondistinct. In concomittaant pericholecystitis the wall of the gallbladder has a double contour with an increase in the

echo-structure of the outer and a decrease in the echo-structure of the inner contour. A strip of fluid around the gallbladder indicates the presence of local peritonitis.

Chronic (non-calculous) cholecystitis. In the period of remission of chronic cholecystitis, the size of the gallbladder is reduced or normal. The most reliable signs are the thickening of the wall of the gallbladder with its simultaneous consolidation (intensified echo-structure) and clear contours. This distinguishes the echographic picture from that of acute cholecystitis.

Often the shape of the gallbladder changes: there are curves, wall contraction and more evident deformation of its walls.

At the same time it should be remembered that the diagnosis of chronic cholecystitis can not be made only on the basis of ultrasound findings: it is necessary to provide clinical confirmation.

Cholelithiasis. The problem of cholelithiasis has a leading place in the diseases of the gallbladder. Ultrasonic signs of cholelithiasis are divided into direct and indirect. The direct signs include intensified echo-signal corresponding to the location of the stone in the lumen of the gallbladder against the background of echo-negative structure of the bile. The size of the signal is slightly less than the true size of the stone (Fig. 30). When the patient is examined in a horizontal position, stones are located predominantly on the dorsal surface and in the neck of the gallbladder.

An important feature of cholelithiasis is the displacement of stones when changing the position of the body. In the upright position, the stones "roll" to the bottom of the gallbladder. The echo-structure, coming from a stone, the size of which exceeds 4 mm, always forms a shadow path after itself, an acoustic shadow that occurs as a result of the absorption of ultrasonic waves by a stone.

One of the *indirect signs* of cholelithiasis is an increase in its dimensions by more than 5 cm in diameter and up to 10 cm in length and also in the thickness of its wall and inequality of the contour.

An echographic pattern in the presence of stones in extrahepatic bile ducts resembles that of cholelithiasis. Stones larger than 3-4 mm in diameter give a stronger echo-signal. If the diameter of the stone exceeds 5 mm, its dorsal wall determines the acoustic shadow. Small stones size are usually not detected in ultrasonic passage. In these cases, an indirect sign of cholelithiasis is the dilation of the duct proximally to the place of its obstruction.

Differential diagnosis of obstructive and parenchymal jaundice. Echolocation appears to be one of the most informative and valuable methods of differential diagnostics of obstructive and parenchymal jaundice. It should be borne in mind that *one of the main echographic signs of obstructive jaundice is enlargement of the biliary tract*.

Differential diagnosis is based on the following principles.

1. If the intrahepatic and extrahepatic ducts are not dilated, and the size of the gallbladder is not increased, the obstructive cause of jaundice is doubtful. In these cases, it is most likely a consequence of diffuse liver damage (hepatitis, liver cirrhosis, etc.).

2. If the intrahepatic ducts are considerably dilated and the gallbladder and extrahepatic ducts are of normal size, the cause of jaundice should be seen in high obturation, for example, at the level of the common hepatic duct.

3. If the size of the extra- and intrahepatic bile ducts and the gallbladder is significantly increased and these dimensions do not change with the use of choleretic agents, the most probable cause of jaundice is obturation of the distal part of the common bile duct ("killed" stone, Oddi's sphincter cancer, carcinoma of pancreatic head, etc.). It should be borne in mind that the compression of the common bile duct in the tumor of the pancreas head is often accompanied by the Courvoisier's symptom (an increase in the size of the gallbladder secondary to obstructive jaundice). A stone in the common bile duct results in the dilation of the ducts but the gallbladder is often not enlarged.

Ultrasound examination of the pancreas

Echography enables to visualize the pancreas in different projections and assess its state in the progress of the pathological process, although due to the peculiarities of the anatomical structure and location of the pancreas, the study is associated with certain difficulties.

Indications for ultrasound examination of the pancreas are as follows:

1) any recurrent or persistent pain in the epigastric area;

2) palpated formation in the epigastric region or pain in palpation;

3) verified acute or chronic pancreatitis in order to detect complications in a timely manner (cysts, abscess, necrosis);

4) suspected cyst, abscess, hematoma, pancreatic cancer;

5) deformation of the posterior wall of the stomach during gastroscopy;

6) change in the shape and contours of the duodenal loop during X-ray examination.

Ultrasound examination of the pancreas begins in the patient's horizontal position with a sagittal scan. The sensor is installed longitudinally in the epigastric region to the left of the median line.

The examination of the pancreas is preceded by detection of large vessels of the abdominal cavity, aorta, inferior vena cava, splenic and portal veins, superior mesenteric arteries and veins, which serve as benchmarks for finding the pancreas, as well as the abdominal trunk. In this case, it is necessary to determine the position of vessels, their diameter, the external and internal contours, pulsation, changes in diameter on inspiration and on expiration, the presence of inclusions in the lumen.

The body of the pancreas adjoins the dorsal surface of the left lobe of the liver, and the head is attached to the duodenum. The examination of the pancreas is carried out at the height of maximum inhalation, when the left lobe of the liver lowers into the abdominal cavity. Studies continue with the transverse location of the ultrasound transducer, which gradually moves down to the visualization of the splenic vein. In transverse scanning, it is often possible to visualize the entire pancreas. If necessary, the study is carried out in other positions: at the position of the patient on the right and left side, in an upright position. In the study of the pancreas, it examines its position relative to the "vascular reference points" and the vertebral column, determining the shape, contours and size of the organ, the state of the pancreatic duct, the echo-structure of the gland, and identifying the presence of focal changes. The pancreas is located retroperitoneally across the posterior abdominal wall at the level of the first and second lumbar vertebrae. Although its shape may vary, the head is always the largest part of the gland. For practical purposes, it should be borne in mind that the size of the head more than 35 mm, the body over 25 mm and the tail more than 30 mm reliably indicate enlargement of the pancreas and an associated disorder. *Normally*, the echo-structure of the pancreas resembles the echo-structure of the liver in intensity. Small echoe-signals prevail, and they are evenly distributed throughout the gland. With age due to induration and fat deposits, echo-structure of the pancreas increases. Normally, the diameter of the Wirsung duct does not exceed 1.5-2 mm. After intravenous administration of secretin, its diameter increases to 2.5-5 mm.

In various pathological processes in the gland its echo-structure significantly changes. ***Acute pancreatitis*** is characterized by a significant decrease due to swelling of the gland, and ***chronic pancreatitis and cancer*** by an increase and heterogeneity (due to the development of fibrosis and cicatricial changes), commonly with enlargement of pancreatic duct (up to 2.5-3.5 mm). After the introduction of secretin, the diameter of its lumen almost does not change, which is an important diagnostic criterion for chronic pancreatitis.

Table 12 shows the direct and indirect signs of some of the most common diseases of the pancreas.

Table 12

Direct and indirect echographic signs of some diseases of the pancreas (M. M. Boger, S. A. Mordvov)

US-criterion	Changes in US-criterion	Diseases and syndromes
Direct signs		
Gland size	Diffuse enlargement	Swelling, inflammation
	Segmented enlargement	Tumor, cyst, abscess
Gland contour	Even	Norm
	Indistinct	Swelling
	Uneven	Tumor, chronic pancreatitis
	Smooth, bulging	Cyst
Gland echo-structure	Low density	Norm
	Increased	Chronic pancreatitis

	Decreased	Tumor, swelling
	Echo-negative	Cyst
Indirect signs		
Aorto-hepatic space	Increased	All cases of enlargement of the gland
Dorsal surface of the liver	Indention	Expansive process
Aorta and inferior vena cava	Displacement and indention	Expansive process
Diameter of Wirsung duct	Enlarged	Expansive process, chronic pancreatitis, reactive pancreatitis

Duodenal probing

Duodenal content is a mixture of bile with pancreatic, gastric and intestinal juices. The study of duodenal content is used to assess the state of the biliary system, as well as the function of the pancreas. Depending on the specific purposes, different methods of duodenal probing are used.

Assessment of the state of the bile-excreting system. At present, the method of *fractional duodenal probing*, which has significant advantages over the classical (three-phase) method, widespread in the past, is used. In fractional probing, duodenal contents are extracted every 5-10 minutes. This is made to graphically record its amount in time course and diagnose the type of bile secretion.

The procedure of duodenal probing. Duodenal probing is carried out using a thin rubber probe with metal or plastic olive at the end. It is better to use a two-lumen probe, with one channel for pumping gastric contents.

Studies are conducted on an empty stomach in the morning. The duodenal probe is inserted in the patient's sitting position (in the same manner as the thin gastric probe). When the label "40 cm" appears near the teeth, the probe is further advanced by 10-15 cm, a syringe is connected and gastric contents are aspirated. After that, the patient swallows the probe to the label "70 cm". Further studies continue in the patient's position on the right side;, a soft roll or pillow is put under the pelvis (in this position, it facilitates the passage of the probe to the sphincter and duodenum), and a warm water-bottle is put under the right hypochondrium. The outer end of the probe is lowered into a test tube, a tripod with test tubes is placed on a low bench near the head. In this position, the patient gradually (within 20-60 minutes) swallows the probe to a mark of 90 cm. As soon as the olive passes from the stomach into the duodenum, a yellow liquid begins to flow into the test tube - the duodenal contents, stained with bile.

There are **five phases of fractional duodenal probing**.

The first phase is a release of *duodenal contents* from the moment the probe enters the duodenum to the introduction of one of cholecystokinetic agents, *portion A*. This portion of the duodenal content is a mixture of bile, pancreatic, intestinal and, partly, gastric juices and does not have a large diagnostic value. The bile of portion A is collected for 10-20 minutes.

The second phase is a phase of *complete cessation of biliary excretion* due to *spasticity of Oddi's sphincter*, which occurs as a result of the administration of a cholecystokinetic agent (30-50 ml of warm 33% solution of magnesium sulfate through a probe or 75 units of cholecystokinin intravenously). Normally, the duration of the second phase does not exceed 4-6 minutes; its prolongation indicates an increase in the tone of Oddi's sphincter, and its shortening is a sign of hypotonia.

The third phase is a release of golden-yellow content of *extrahepatic bile ducts*, which lasts 3-4 minutes. The released bile also refers to the portion A (A_1).

The fourth phase is emptying of the gallbladder and a release of the thick *gallbladder bile* of dark yellow or brown color, *portion B*. This portion of bile is released as a result of contraction of the gallbladder arising from the action of cholecystokinetic agents, and simultaneous relaxation of Oddi's sphincter and the sphincter of the gallbladder. Portion B is 4-5 times more concentrated than hepatic bile, and contains a significant amount of bile acids, cholesterol and bilirubin. Realease of gallbladder bile (normally about 30-60 ml) lasts 20-30 minutes. If the gallbladder reflex is absent within 20-30 minutes after the introduction of magnesium sulfate, which in some cases may occur even in healthy people, it is necessary to introduce *antispasmodics* (30 ml of 20% solution of novocaine through a

probe or 0.5 ml of 0.1% solution of atropine subcutaneously), and in the absence of their effect, to repeat administration of cholecystokinin. If, after the administration of novocaine or atropine, dark gallbladder bile begins to appear, this indicates spasm of the sphincter; the absence of gallbladder reflex, even after administration of antispasmodics and cholecystokinin (repeated), suggests the presence of *organic barrier* (blockage of the duct with a stone) or *nonfunctioning gallbladder* (shrinkage, gallbladder cancer, etc.).

The fifth phase - after the cessation of the release of dark gallbladder bile through the probe, the bile of golden yellow color is released again, *portion C*. It is also collected in tubes for 30 minutes at 10-minute intervals.

Thus, fractional duodenal probing is used for indirect determination of the important features of bile secretion, gallbladder capacity, the presence of functional and organic bile duct disorders. In addition, all three portions of bile (A, B, C) undergo microscopic, and if necessary, bacteriological examination. The most diagnostic value belongs to the study of gallbladder bile (portion B).

Chemical study of bile includes determination of the concentration of bilirubin, cholesterol and bile acids, as well as calculation of the so-called cholatocholesteric index. Typically, different colorimetric methods are used to determine the concentration of these substances, although there are other and more precise ways of their quantitative determination (chromatographic, fluorometric, luminescent, etc.). Normal values of the most important bile ingredients are presented in Table 13.

Table 13

The content of bile acids and cholesterol in B and C portions of bile of healthy people

Substance	Bile portions	
	B	C
Bilirubin, mmol/l	3.4–6.8	0.17–0.34
Bile acids, mmol/l	31–84	9.9–16.7
Cholesterol, mmol/l	2.6–10.3	1.0–2.1
Cholatocholesterol ratio	9.0 ± 2.2	9.1 ± 2.3

The most important practical method is the assessment of *cholatocholesterol ratio*, the ratio of the concentration of bile acids and cholesterol in the bile. Bile acids (cholic, chenodesoxycholic, etc.) are the end products of cholesterol metabolism. In bile, they are present in the form of sodium salts, which prevent the loss of cholesterol in the precipitate and the formation of gallstones. Reduced bile acid in bile is observed in secretory insufficiency of hepatic cells (hepatitis), complicated course of acute and chronic cholecystitis and pancreatitis. These pathological processes are usually accompanied by an increase in cholesterol concentrations in bile. As a result, cholatocholesterol ratio may decrease significantly. Significant *decrease in cholatocholesterol ratio* (below 9.0) is observed in secretory insufficiency of the liver cells (for example, in viral hepatitis), acute and chronic cholecystitis and pancreatitis, and indicates a predisposition to the formation of stones in the gallbladder and biliary ducts.

Microscopic examination of bile is carried out immediately after the receipt of the material, as the cellular elements in the bile rapidly collapse under the action of bile acids and salts of fatty acids (leukocytes in the bile are destroyed, for example, within 5-10 minutes).

For microscopic examination, bile is taken without the admixture of gastric and intestinal juice (usually, portions of B and C are examined). The bile is poured into Petri dishes, removing drops of mucus and placed on a slide glass, preparing several specimens. Another portion of bile is centrifuged and native specimens are also prepared from the precipitation. The total number of specimens should be at least ten.

Microscopic examination of bile can show: 1) cellular elements (leukocytes, epithelium); 2) crystalline formations; 3) parasites.

Leukocytes The diagnostic value of finding leukocytes in bile specimens is insignificant. This is primarily due to the complexity of identifying these blood elements that are destroyed in bile for 5-10 minutes after receiving a portion of bile. Often, changed and rounded nuclei of the intestinal epithelium are mistakenly regarded as leukocytes. Finally, leukocytes found in the bile may have different origin (from the duodenal ulcer, oral cavity, gallbladder and stomach).

Detection of **epithelial cells** has more diagnostic value, but only in cases where the epithelium is sufficiently preserved in order to identify its origin: *cholecystitis* is characterized by identification of high prismatic muscular cells, cholangitis by small prismatic cells of the hepatic pathways or high prismatic epithelial cells of the common bile duct, pathological processes in the *duodenum* by large cylindrical cells with cuticle and villi.

Crystals of cholesterol can be found in bile even in healthy people. They look like thin, colorless, quadrangular plates with a breakable angle. An increase in cholesterol crystals indicates a change in the colloidal stability of the bile.

Microlytes are dark, large or multifaceted formations that consist of calcium salts, mucus and a small amount of cholesterol. Normally, microlites are not observed; their detection in the bile, as a rule, indicates a violation of the colloidal stability of the bile. This pathological process is often associated with the detection of even smalerl (microscopic) grains of different sizes and colors, which are commonly referred to as "sand".

Calcium bilirubinates are amorphous small grains of golden-yellow or brown color. Often they are found in the bile in combination with large amount of cholesterol crystals.

Crystals of fatty acids have the appearance of thin needles. Their appearance indicates a decrease in the solubility of fatty acids, caused by the inflammatory process, and a disturbance of colloidal stability of the bile.

Parasites. Duodenal contents can contain *vegetative forms* of some *parasites* (most often *lambliae*) and *eggs of helminths* (*opisthorchiasis, fasciolosis, dicroceliasis, stronhyloidosis, trichostrongyloidosis*, etc.). Their detection in different portions of bile testifies to the presence of helminthic invasion of the liver, gall bladder or duodenum.

A large number of cholesterol crystals, microlittes and "sand", calcium crystals, bilirubinates and fatty acid crystals in the duodenal content indicates a disturbance of colloidal stability of the bile and possible gallstone disease.

For **bacteriological examination**, bile is collected in sterile tubes and sent to a bacteriological laboratory. Normal bile is sterile. In different disorders cultures of bile on special nutritional media can have growth of *E. coli, streptococci, staphylococci* and other bacteria. However, the interpretation of these results is always complicated, since it is difficult to establish the origin of the cultured flora: from the oral cavity, intestine or biliary tract. Therefore, bacteriological examination of bile has only a relative diagnostic value.

It should be emphasized that in recent years the attitude of physicians to the results obtained in the study of duodenal content has changed significantly, also due to the widespread introduction of such highly informative methods of investigation as ultrasound, computer tomography, etc. in the clinical practice. *The real value of duodenal probing is currently limited to the detection of parasites, signs of disturbance of colloidal stability of bile, and also the detection of some functional disorders of bile duct.*

Study of pancreas function

The study includes:

1) determination of enzymes in blood and urine (see above);

2) determination of basal and stimulated secretion of bicarbonates and enzymes in duodenal contents;

3) study of feces on the content of fat, nitrogen, muscle fibers, etc.

Determination of pancreatic enzymes and bicarbonates in duodenal contents is indicated *in chronic diseases of the pancreas* or in symptoms characteristic of the **insufficiency of its external secretion function** (chronic diarrhea, steatorrhea, etc.). Studies are conducted on an empty stomach in the morning. To receive adequate results 5 days before the test the patient should stop taking enzyme agents. It is better to use a double probe that has openings at the level of the stomach and duodenum. Permanent aspiration during the study of gastric contents is needed to obtain a cleaner pancreatic juice. After introducing the probe, it is advisable to monitor its position radiologically.

Basal secretion of bicarbonates and pancreatic enzymes is investigated by aspirating pancreatic juice through a duodenal probe for 1 hour at 15-minute intervals. After that intravenous drip for 1 hour

is used to introduce the most adequate stimulator of pancreatic secretion - secretin in a dose of 1 U per 1 kg of body weight. Secretion stimulated by secretin is studied for two hours (aspirates are also taken at 15-minute intervals). Bicarbonates, α-amylase, lipase, trypsin are determined in each portion of pancreatic juice.

Interpretation of findings. Many diseases of the pancreas associated with the *insufficiency of its external secretion* are accompanied by a decrease in basal pancreatic secretion and a decrease in the content of enzymes and bicarbonates - *edematous form of acute pancreatitis, chronic pancreatitis with the development of atrophic processes in the gland, pancreatic cancer, which is especially accompanied by obstructive pancreatic ducts*.

The content of pancreatic enzymes in duodenal contents is often diminished also in diseases that are accompanied by *secondary changes in the function of the pancreas - in diabetes mellitus, cholelithiasis, cholecystitis, hepatitis,* and others.

At the same time, it should be borne in mind that the level of enzymes in duodenal contents is extremely variable both in norm and in disorders. Therefore, when evaluating the external secretion function of the pancreas, it is better to focus not so much on their absolute value in the basal phase of secretion, but in the time course during the phase of stimulated secretion. In a healthy person, in fractional duodenal probing, after the introduction of secretin, the concentration of enzymes and bicarbonates is reduced, but by the end of the first hour or during the second hour of the study, it returns to normal or even exceeds the baseline.

The criteria for *reduced external secretion of the pancreas* are:

1) reduction of concentration of bicarbonates (below 50 mmol/l) and enzymes in the basal phase of secretion;

2) sharper than in a healthy person decrease in their concentration in duodenal contents after the introduction of secretin;

3) within two hours of stimulated secretion studies, the content of enzymes and bicarbonates does not return to the baseline level.

Evaluating the results of fractional duodenal probing, it should be remembered that about 20% of patients with normal findings actually suffer from some diseases of the pancreas with a violation of its external secretion function. This indicates a lack of informativeness of the described method.

Coprologic examination in diseases of the pancreas. The study of feces in patients with pancreatic diseases has a definite, albeit very limited, value for the detection of insufficiency of the external secretion function of the pancreas.

Pancreatic enzymes are known to take an active part in the digestion and absorption of fat and proteins in the small intestine. In impaired pancreatic secretion feces often change appearance: they become grayish, aquire rotten smell, contain a large amount of fat, and the volume of excrement increases significantly (*polyfecalia*). Impaired digestion of fat is called *steatorrhea* ("fat" feces), and insufficient digestion of muscle fibers is called *creatorrhea*.

The most common causes of ***steatorrhea*** are:

1) pancreatic dysfunction, in particular, insufficient secretion of lipase enzyme;

2) impaired formation and release of bile;

3) various abnormal processes in the mucous membrane of the small intestine and other causes.

Steatorrhea, which developed as a result of *functional pancreatic insufficiency*, is characterized by an increase in the content of neutral fats and salts of fatty acids, the amount of fatty acids usually does not change.

The most common causes of ***creatorrhea*** are insufficiency of the external secretion function of the pancreas, achylia and some other pathological processes. Normally, microscopic examination detects few muscle fibers in feces, and in the process of digestion, they almost completely get rid of striated appearance, their ends become rounded. In diseases of the pancreas, a large number of undigested muscle fibers can be observed in feces; microscopy reveals a well-preserved transverse striation of muscle fibers, the ends of which remain sharp.

As a result of comparatively low sensitivity and specificity, the study of feces, which itself has a very limited diagnostic value, must be compared with the general presentation of the disease and the results of other methods of research (blood enzymes, urine, duodenal contents, ultrasound, etc.).

Laparocentesis

Puncture of the abdominal cavity (laparocentesis) and removal of the intraperitoneal fluid (ascites) are used for diagnostic and therapeutic purposes. *Indications* for therapeutic paracentesis are difficulty breathing, impaired cardiac activity or severe discomfort and abdominal pain associated with the presence of tense ascites.

The procedure for laparocentesis is *contraindicated*:

1) in impaired blood coagulation and platelet-vascular hemostasis;

2) in intestinal obstruction;

3) during pregnancy;

4) in patients with severe diseases of the cardiovascular system.

Technique of laparocentesis. Laparocentesis, as well as pleural puncture, is performed under local anesthesia more often with the help of a *trocar*. The patient, if allowed by his condition, sits on a chair. In this position, puncture usually occurs on the midline of the abdomen below the navel, although other accesses are possible. The puncture site should not be located in the area of the previous puncture, be contaminated or infected. The bladder should be drained in advance.

The puncture site is treated with an antiseptic solution (for example, an alcoholic solution of iodine) and coated with sterile material. After local anesthesia of the abdominal wall, a trocar is inserted into the abdominal cavity and a stylet is taken from it. Ascitic fluid under pressure comes out of the abdominal cavity. Some of it is collected in a sterile container (20-30 ml), it is advisable to pull the abdominal wall with a large towel or sheet to prevent collapse, which can develop as a result of rapid evacuation of the fluid and vascular vagal reflex. At the end of the paracentesis procedure, the trocar is removed and a sterile bandage is applied to the puncture site.

The abdominal cavity can also be punctured using a catheter. For this a large diameter needle is used to perform a puncture of the abdominal wall, after which a normal intravenous catheter is introduced through the needle. When ascitic fluid begins to flow freely from the abdominal cavity, the needle is removed.

Such a technique of removing ascites is particularly preferable if paracentesis should be performed in the horizontal position of the patient, lying on the back. This puncture is performed at an acute angle to the abdominal wall:

1) on the midline of the abdomen below the navel;

2) laterally from the rectus abdominal muscles

3) in the lower right quadrant of the abdomen directly above the anterior iliac crest.

When carrying out the procedure for laparocentesis, it is necessary to remember about possible complications, which include:

1) arterial hypotension or collapse, especially with the rapid removal of large quantities of ascitic fluid;

2) perforation of the intestine;

3) bleeding (usually stops spontaneously);

4) perforation of the bladder;

5) continuing leakage of fluid from the puncture site.

The latter is not so rarely found in clinical practice and with insufficient adherence to the rules of antiseptic can lead to the development of peritonitis. Therefore, in the case of a fluid flow from the abdominal cavity that lasts for several days, it is expedient to consult a surgeon and, if necessary, apply an “eight”-shaped suture to the place of puncture.

It should also be remembered that when a large amount of ascitic fluid is removed, the body loses a significant amount of protein, which in some cases can provoke a sharp deterioration of the state up to the development of hepatic coma.

The most common causes of ascites are:

1) cirrhosis of the liver;

2) congestive insufficiency of blood circulation;

3) peritoneal carcinomatosis.

Puncture biopsy of the liver

It is used not only to get histological confirmation of a diagnosis (which itself is important), but in many cases makes it possible to make an overview of the morphological variant of the disease and its etiology, which can be crucial in the prescription of adequate therapy and in prognosis of the disease.

There are several methods of *puncture biopsy of the liver*.

1. *Percutaneous (blind) biopsy* is the most simple and widespread method of obtaining a histological material, which is advisable to use mainly to clarify the morphological picture of diffuse liver lesions (chronic hepatitis, fatty hepatosis, liver cirrhosis, hemochromatosis, amyloidosis of the liver, etc.).

2. *Laparoscopic liver biopsy*, a method of focused biopsy of the liver, carried out under direct laparoscopic control. The method gives the most valuable results in the morphological diagnosis of focal lesions of the liver (abscesses, parasitic cysts, hepatoma and metastatic liver lesions, hemangiomas, etc.), as well as in the differential diagnosis of chronic hepatitis and liver cirrhosis.

3. *Open liver biopsy* is used by surgeons during laparotomy or surgical intervention on the organs of the abdominal cavity.

Percutaneous puncture biopsy of the liver is preceded by a thorough clinical and laboratory examination of the patient, first of all for the exclusion of diseases, the presence of which is a contraindication for the study.

Puncture biopsy of the liver is *contraindicated*:

1) in impairments of platelet-vascular hemostasis and blood coagulation system (implies any clinical and / or laboratory manifestations of hemorrhagic diathesis);

2) in obstructive jaundice or any suspected obstruction of extrahepatic biliary tract;

3) in any purulent processes in the liver (abscess, purulent cholangitis, echinococcosis of the liver), in the right pleural cavity (purulent pleurisy, pleural empyema), on the skin at the point of puncture (pyoderma);

4) in a comatose state of the patient;

5) in the presence of congestive liver in patients with heart failure, constrictive pericarditis or hepatic venous thrombosis.

Technique of the study. Puncture biopsy of the liver is carried out in the position of the patient on the back. The skin at the point of the puncture (usually in the IX-X intercostal space between the anterior and middle arterial lines) is treated with an antiseptic solution. After this, a 2% solution of novocaine is used for local anesthesia of the skin, subcutaneous adipose tissue and liver capsules. The puncture is made by a stylet, inserting it to a depth of 2-4 mm. Then, through the stylet, a puncture needle (Menghini needle) is inserted, connected to a 10-gram syringe containing 4-6 ml of isotonic sodium chloride solution. The needle is drawn to the liver capsule and 2 ml of isotonic sodium chloride solution is released to push out a piece of fatty tissue from the needle. The needle is injected into the liver parenchyma and using a piston syringe pieces of tissue of the organ are aspirated. At the end of the procedure, a sterile sticker is put on the place of the puncture and an ice bag is put. The patient should keep bed regime for 24 hours.

In adequate technique of puncture biopsy of the liver and strict consideration of all contraindications, the number of *complications* during this procedure is relatively small. The most dangerous of them include:

1) bleeding;

2) development of pleural shock;

3) damage to neighboring organs;

4) infection of the pleural or abdominal cavities.

It should be remembered that patients often experience pain at the site of the puncture, in the epigastric region, in the right shoulder and in the right supraclavicular region after puncture biopsy of the liver. As a rule, these symptoms are not dangerous and after a while disappear spontaneously.

Interpretation of findings. When evaluating the results of puncture biopsy of the liver, it should be borne in mind that the picture of intravital morphological changes in the liver is not always and not fully consistent with the individual clinical manifestations of the disease and the data of other laboratory and instrumental methods of investigation, including the results of functional loading tests. The latter, for example, are negative in half of patients with verified fatty hepatosis, in 1/4 patients with chronic hepatitis and in 13% of patients with compensated cirrhosis of the liver (H. Kalk). Even in decompensated cirrhosis of the liver there are cases where biochemical studies show normal results (see above). This once again proves that the morphological picture of liver damage is decisive in assessing the severity of the disease, the activity of the pathological process and the differential diagnosis of various diseases, similar in presentation. Of course, we are talking about cases where it is possible to conduct a qualitative puncture biopsy of the liver.

As noted above, the most important method of percutaneous puncture biopsy of the liver is to characterize *diffuse liver diseases* (hepatitis, liver cirrhosis, fatty hepatosis, amyloidosis, hemochromatosis, as well as secondary lesions of liver parenchyma in systemic lupus erythematosus and other diseases). Of practical interest is the possibility of puncture biopsy of the liver in the differential diagnosis of acute viral and acute alcoholic hepatitis, chronic hepatitis, fatty hepatosis and various variants of liver cirrhosis.

Acute hepatitis. Morphological study of punctate of the liver is used for reliable differentiation of acute viral and acute alcoholic hepatitis (Table 14).

Table 14

Morphological signs of acute viral and alcoholic hepatitis

Morphological signs	Viral hepatitis	Alcoholic hepatitis
Morphology of hepatocytes	Polymorphism	Monomorphism
The nature of dystrophic changes	Predominance of protein degeneration	Predominance of fat degeneration
Location of necrosis sites	Periportal	Centrolobular
Hepatocytes in cytoplasm	Councilman bodies (in coagulation necrosis of hepatocytes)	Alcohol hyaline (Mallory bodies)
Cell proliferation in inflammatory infiltration	Stellate endothelium	Polynuclear leukocytes

Chronic hepatitis. The main morphological feature of *chronic autoimmune active hepatitis* is intense inflammatory infiltration in the area of dilated portal tracts with moderate or sharply expressed piecemeal necrosis and the development of fibrosis. Inflammatory, necrotic and fibrous changes penetrate deep into the hepatic lobe. Other manifestations can include moderate or severe dystrophic changes in hepatocytes and signs of increased regenerative processes with the appearance of large hepatocytes with large nuclei, scattered in the liver parenchyma or forming specific islets - regenerates.

Fatty hepatosis is morphologically characterized by the presence of severe fatty dystrophy of the liver cells, as well as the development in the portal tract of fibrous tissue, often with the formation of strains (septa) and violation of the lumbar structure. There is also a proliferation of Kupffer cells and regeneration of the hepatic tissue (in the transition of the pathological process to liver cirrhosis).

Cirrhosis of the liver. Morphological criteria for liver cirrhosis are severe dystrophy and necrosis of the liver cells, which are accompanied by nodular regeneration, diffuse enlargement of connective tissue, and a profound restructuring of the liver architectonics. In the *micronodular form of cirrhosis* ("portal cirrhosis") liver punctates contain a large number of identical nodules with a diameter of less than 3 mm. *Macronodular form of cirrhosis* ("post-necrotic cirrhosis") is characterized by the development of nodes of different sizes, the diameter of which is much greater than 3 mm. Some nodes reach 3 cm. In mixed form ("micromacronodular cirrhosis"), the number of small and large nodes is approximately the same.

Primary biliary cirrhosis of the liver, above all, is characterized by a significant expansion of the bile ducts with greenish-brown bile cylinders. The cytoplasm of hepatocytes is stained with greenish bile. Subsequently, proliferation of the endothelium of the bile ducts is observed with destructive changes in hepatocytes in the parenchyma of the liver and their necrosis.

It should be borne in mind that identifying the form of liver cirrhosis by the results of a puncture biopsy is a rather difficult task. More reliable for diagnosis is the study of focused biopsy of the liver, obtained during laparoscopy.

Of practical interest are morphological signs of progression (activity) of the cirrhotic process, which, according to S.D. Podimova, includes:

1) severe inflammatory reaction around foci of hepatocytes death, blurred border between parenchyma and connective tissue layers due to penetration of cell infiltrates and collagen fibers in the parenchyma;

2) increased vascularization and inflammatory infiltration of connective tissue with abundant development of bile ducts;

3) active regeneration of parenchyma.

Laparoscopy

In diseases of the hepatobiliary system and the pancreas, laparoscopy is the most informative in recognizing *focal lesions* of these organs (nodal form of primary liver cancer, pancreatic cancer, metastatic liver damage, echinococcosis, liver abscess, etc.). In these cases, a visual review of the abdominal organs should be combined with their focused biopsy.

Laparoscopy is successfully used to diagnose *gallbladder diseases* (cancer, empyema, dropsy of the gallbladder), as well as to determine the level of biliary obstruction in obstructivee jaundice.

Thus, in low biliary obstruction (cancer of pancreatic head, cancer of Vater ampulla), the gallbladder is often enlarged, tense, and dilated (classic Courvoisier's symptom). If the compression site of the common bile or hepatic duct is located above the mouth of the duct (for example, in the metastatic lesion of the lymph nodes of the liver hilum), laparoscopy, in most cases, shows that the gallbladder is drooped. In obturation of the common bile duct by stone, the gallbladder is often wrinkled, with cicatricial changes in the wall. However, the latter two cases, may be characterized not only by a typical decrease in the size of the gallbladder, but also by its enlargement due to dropsy or empyema.

It should also be remembered that, if necessary, laparoscopy can be used to conduct an X-ray contrast study of the gallbladder and biliary tract (*laparoscopic cholangiography*), as well as endoscopic surgery on the biliary tract (*for example, laparoscopic cholecystectomy*).

II.3. ACUTE ABDOMEN SYNDROME

Acute abdomen *is a clinical syndrome that develops in injuries and acute diseases of the abdominal cavity and retroperitoneal space and requiring emergency surgical care.*

Relevance of the problem of tactics and strategy of the treatment in the presence of *acute abdominal pain* (AAP) in the patient is in no doubt. This is due to the fact that AAP is one of the most common reasons for emergency hospitalization. Thus, in the United States, 5-10 million people with acute abdominal pain are treated annually. At the same time, 40% of these cases are found to have *acute abdomen syndrome (AAS)*. Follow-up examination shows that the pain disappears within a few hours in 25% of patients. These are patients with "functional pain" or *"non-specific abdominal pain (NSAP)"*. In some cases, only a clear sequence of actions stops the development of severe complications. Knowledge of the algorithms of managing patients with AAP at different stages of medical care is mandatory for any clinician. Each doctor should have clear, reasoned answers to the question: what sequence of actions in acute abdominal pain syndrome is, which diagnostic and therapeutic measures should be carried out in this situation in out-patient settings, in a polyclinic, in a surgical hospital.

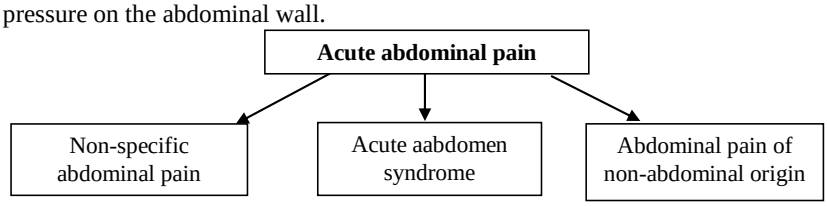
Acute abdomen syndrome

Acute abdomen syndrome (AAS) *is a clinical symptom complex manifested by pain and symptoms that are mainly localized in the abdomen, is characterized by limited or diffuse signs of peritoneal irritation, endangers the life of the patient and requires emergency medical care.*

Most often, there is confusion in the concepts of AAP and AAS. AAP is a more general concept, which includes three structural units: AAS, NSAP, and "pseudo-acute abdomen" - acute abdominal pain of non-abdominal origin (Scheme 2).

In practice, the diagnosis of AAS is used as a preliminary one due to lack of time and diagnostic capabilities to determine the exact cause of the disease.

The main symptom of AAS is the ***pain*** that the patient feels spontaneously, increased in percussion or pressure on the abdominal wall.



Scheme 2. Structure of acute abdominal pain

Acute abdomen is characterized by the following *basic signs*: *abdominal pain of different nature and intensity, tension of abdominal wall muscle, intestinal motility disorders*. These and some secondary features are found in various combinations in various pathological conditions caused by acute inflammatory processes of the abdominal organs, bleeding in the abdominal cavity, local disorders of the blood circulation, or intestinal obstruction.

Presentation of the acute abdomen can be imitated by pseudo-abdominal syndrome, in which abdominal pain is caused by diseases of the abdominal cavity that do not require emergency surgical treatment (for example, gastritis, colitis) or diseases of the organs located outside the abdominal cavity (for example, myocardial infarction, acute pneumonia, pyelonephritis). These diseases, although they may be accompanied by a number of signs of acute abdomen, are subject mainly to conservative treatment.

The main reasons for the development of AAS:

1. Acute non-specific inflammatory diseases of the digestive system. The most common are acute inflammatory processes in the appendix, gallbladder, pancreas.

2. Perforations of the hollow organ, most often as a result of various diseases or injuries of the abdominal organs causing peritonitis.

3. Internal bleeding in the abdominal cavity and retroperitoneal space developing spontaneously (e.g., uterine tube rupture during tubal pregnancy or the abdominal aortic dissection) or as a result of trauma (traumatic rupture of the liver, spleen, mesenteric veins, etc.).

4. Intestinal obstruction, which occurs as a result of volvulus, nodular formation, intestinal strangulation in the internal or external hernia, obturation, invagination, compression of the intestine with adhesions.

5. Acute violations of mesenteric circulation (arterial and venous), resulting in intestinal infarction.

6. Acute inflammatory processes and impaired circulation of the internal hollow organs (acute adnexitis, torsion of the pedicle of tumor or ovarian cyst, necrosis of the myomatous uterine node or ovarian tumor, etc.).

Clinical symptomatology. The leading symptom is *pain*, localized or spread throughout the abdomen, usually increases when moving. In case of major and severe disorders leading to the onset of AAS, for example, traumatic ruptures of the abdominal cavity, widespread pancreatic necrosis, pain syndrome is severe and may be accompanied by the development of shock. In young children, especially in hypotrophy, in patients with senile age, in exhausted patients, when the body reactivity and severe intoxication are reduced, the pain is minor.

A frequent symptom of AAS is *vomiting*, which may occur in the first hours and even minutes of the disease. Sometimes there is a persistent painful hiccup, which is usually associated with irritation of the diaphragmatic nerve. The so-called *phrenikus symptom* (a sharp pain in pressure on the area between the pedicles of the sternoclavicular-papillary muscle) usually occurs as a result of irritation of the nerve endings of the diaphragmatic nerve by the fluid exudate released with the contents of the gastrointestinal tract or blood. The same origin has the so-called *symptom of the tilting doll*, a sharp increase in pain in the abdomen when the patient is trying to take a horizontal position, due to which he remains in a sitting or semi-sitting position.

AAS is often accompanied by *impaired passage of the contents of the intestine*. Retention of feces and gases is associated with obstructive or dynamic intestinal obstruction. Less common is *loose stool* (at the beginning of the disease in intestinal invagination, acute appendicitis in pelvic placement of the appendix). An important symptom of AAS is the change in the nature of feces. Thus, admixture of blood is often observed in intestinal invagination and acute violations of mesenteric circulation.

Diagnosis is based on assessment of presentation, history and the course of the disease. Of great importance may be indications of past abdominal pain attacks, information about past diseases and operations, results of previous diagnostic and therapeutic measures. Important information can be obtained by examination and observation of the patient. So, generaalized peritonitis and a massive bleeding in the abdominal cavity are characterized by a motionless position of the patient in a certain position (often on the side with the feet brought to the abdomen), since the slightest movement causes a sharp pain.

In a number of pathological processes accompanied by recurrent cramping abdominal pains (for example, in acute pancreatitis, some forms of intestinal obstruction), the patient may groan, scream, rage and be agitated. In cases of massive intra-abdominal bleeding, perforation of the hollow organ (for example, perforated ulcer of the stomach) the patient may have a sharp pallor of the skin and mucous membranes.

In severe, advanced forms of disease with presenting signs of hypertension, the patient has indifferent expression of the face, indrawn cheeks, sunken eyes, pale gray skin covered with drops of cold sweat, the so-called *mask of Hippocrates*.

During the examination it is necessary to pay attention to the *shape of the abdomen and the form of the anterior abdominal wall*: the indrawn abdomen of the boat-like form is most often observed during perforation of the hollow organ, bloated and asymmetric in intestinal obstruction. A white, yellow or brown plaque is formed on the surface of the tongue shortly after the onset of the disease. With increasing intoxication and dehydration, it becomes rough and dry "like a brush".

An important step for assessing the severity of the patient's condition is to study *pulse and blood pressure*. Reflex bradycardia is observed in the first hours after the contents of the gastrointestinal tract, urine or blood enter the abdominal cavity. As peritonitis develops, the pulse rate increases, its filling decreases. Severe tachycardia is characteristic for intrahepatic bleeding, in which, as a rule, blood pressure decreases rapidly until the development of a collapse.

Palpation of the abdomen in AAS begins with the surface-investigatory one. Deep palpation should be carried out with caution, as it can cause a sharp painful reaction and protective tension of the abdominal muscles, which will not let get a clear idea of the state of the abdominal cavity. Frequent symptom, observed in palpation, is the *pain of the entire anterior abdominal wall or its various areas*. (Figure 31).

Palpation of the abdomen helps to determine the *tension of the muscles of the anterior abdominal wall* (*defense musculaire*) and an increase in abdominal pain in rapid removal of the palpable hand from the abdominal wall after a slight pressure is a *positive symptom of peritoneal irritation (Blumberg's sign).*

Pain intensification is noted in the sliding, tangent motion of the hand on the abdominal wall and in tapping with fingers. This and some other symptoms (especially positive Blumberg's sign) are considered to be signs of *irritation of the peritoneum*, that is *peritonitis*; their detection has a leading role in the diagnosis and decision on the need for emergency surgical intervention.

In perforation or rupture of the hollow organ, muscle tension can involve the entire abdominal wall and be very sharp *("flattened stomach").*

In limited peritonitis (for example, in the pelvic cavity, the cavity of the omental sac), in the weaak and exhausted patients, as well as in those who are in a state of intoxication or alcohol intoxication, the muscle tension of the anterior abdominal wall may be completely absent or insignificant It is also not detected in intra-abdominal bleeding, torsion of ovarian cyst, in the initial stage of acute intestinal obstruction up to the development of peritonitis, in the elderly and in patients with stretched atrophied muscles of the anterior abdominal wall (for example, in women who gave

birth to many children). In children detection of tension in the anterior abdominal muscles requires abdominal examination during natural or medication sleep.

An important element of palpation of the abdominal wall is an inspection of places of possible *outbreak of hernia* (umbilical and inguinal rings, the region of the femoral canal, postoperative scars, etc.).

Percussion of the abdomen can reveal disappearance of hepatic dullness, which is characteristic for perforation of the hollow organ, the presence of free fluid in the abdominal cavity, which is observed in peritonitis, intrahepatic bleeding. Intestinal obstruction is associated with pronounced tympanitis due to accumulation of gases in the intestine.

Auscultation of the abdomen provides an opportunity to evaluate the nature of intestinal motility (lack of intestinal noises or their amplification), to detect the symptoms of *"splashing noise", "the sound of a falling drop"*, which can indicate obstruction of the intestine.

Rectal study can show pathological processes in the distal rectum (for example, a tumor) and para-rectal cellular tissue, detect a sharp pain in the anterior wall of the rectum, indicating an accumulation of the inflammatory fluid in the pelvic cavity (*Kulenkampf's symptom*), a decrease in the tone of the anal sphincter and dilation of the empty ampulla of the rectum (*Obukhov hospital symptom*) are characteristic features of intestinal obstruction.

Vaginal study, including instrumental, is used to assess the state of internal genital organs and conduct a differential diagnosis with acute diseases of the abdominal cavity organs. Pain in a pendular displacement of the cervix (Promptov's symptom) indicates involvement of the internal genital organs.

Laboratory studies. Thus, increased leukocytosis with the shift of the formula to the left can be detected in the blood in inflammatory diseases, a decrease in hemoglobin levels, the number of red blood cells, and hematocrit in intraabdominal bleeding. The urinary sediment study is used for differential diagnosis of urinary tract diseases. If necessary, additional methods of investigation, for example, determination of biochemical parameters of blood, central venous pressure, volume of circulating blood, indicators of acid-base balance, water-electrolyte exchange, etc., are used.

***Instrumental studies*.** *X-ray examination* begins with an observational X-ray examination of the chest and abdominal cavity, and then X-ray of the abdominal cavity is performed in the vertical, horizontal position of the patient and in the lateral position. The examination of a patient in severe condition begins with observational examination of the chest and abdominal cavities in the horizontal position of the patient, on which it is necessary to get an image of both halves of the diaphragm, lateral canals of the abdominal cavity and the area of the small pelvis.

X-ray diagnosis of acute abdominal organs diseases is based on the detection of a complex of X-ray symptoms that directly or indirectly indicate the localization and nature of the disorder. Signs that directly indicate acute abdominal distension, accompanied by perforation or intestinal obstruction, and damage to hollow organs include: *free gas in the abdominal cavity (Zhober's symptom)* or in the retroperitoneal space; free fluid in the abdominal cavity; accumulation of gas with a horizontal level of fluid in the intestinal loops with the formation of characteristic *Kloiber's cups*.

Gas in the retroperitoneal space is visible on X-rays in perforation of ulcer or rupture of the duodenum, the right half of the large intestine, as well as pancreatic necrosis and phlegmon of retroperitoneal fiber.

Indirect X-ray symptoms, indicating the presence of acute inflammation in the abdominal cavity, include reflexive *bloating of the intestine*.

In cases where according to the survey radiography it is not possible to clarify the diagnosis, X-ray contrast studies of the stomach and duodenum are used: gas is administered through a probe into the stomach for the diagnosis of covered perforated gastroduodenal ulcers (*Hennelt test*) or into the intestine (mainly in the diagnosis of intestinal invagination in children, sometimes it is possible to eliminate obstruction). The main symptoms of AAS, which can be detected by these methods, are the output of the X-ray contrast agent and gas in perforated ulcers of the stomach and duodenum in the abdominal cavity, protrusion of the stomach forward in acute pancreatitis.

More advanced X-ray methods of investigation, namely *celiacography*, *mesentericography*, and others are used according to special indications.

Endoscopic methods of examination play an important role in the diagnosis of a number of diseases that are the cause of AAS. They are used to detect ulcer, tumor, diverticulum of different parts of the gastrointestinal tract, to establish the nature of the complications, and in some cases to carry out therapeutic procedures, for example, to remove a stone stuck in the major duodenal papilla or to carry out a recanalization in tumor of the large intestine that causes obstruction.

In complicated diagnosis, laparocentesis is used to detect blood or pathological fluid in the abdominal cavity.

A special place in the diagnosis of AAS belongs to ***laparoscopy***, which is, as a rule, indicted in uncertain diagnosis. Laparoscopy makes it possible to detect exudates or blood in the abdominal cavity, other direct or indirect signs of acute disease of the abdominal cavity and small pelvis.

Ultrasound (US) examination can detect even small amounts of fluid in the abdominal cavity, inflammatory infiltrates, ovarian cysts, enlargement of the fallopian tube in the presence of a fetal egg in it, gallstones, bile duct enlargement in choledocholithiasis, and others.

Diagnostic laparotomy is performed if a comprehensive clinical examination fails to diagnose or exclude acute illness or damage to the abdominal organs.

The differential diagnostic characteristics of some injuries, diseases and pathological conditions resulting in presenting signs of AAS are presented in Table 15.

Table 15

Differential diagnostic characteristics of some injuries, diseases and abnormal conditions resulting in presenting signs of AAS

Injury, disease, abnormal condition	**Characteristic history data**	**Clinical signs**				**Laboratory and X-ray findings**	
		Pain	**Vomiting**	**General condition, body temperature and patient behavior, stool**	**Abdominal, vaginal and rectal examination findings**	**Blood and urine parameters**	**Results of an observational X-ray examination of the abdominal cavity**
1	2	3	4	5	6	7	8
Closed abdominal injury with damage to the hollow organ	Blow into the abdomen, fall from height and other injuries	Occurs acutely throughout the abdomen, gradually increasing, constant, intensified with movement	Frequent, in rupture of the stomach, duodenum, with an admixture of blood	Severe. Painful facial expression, cold sweat, pulse is accelerated, body temperature gradually rises. Stool retention	Abdomen is indrawn, plate-like tension of the muscles of the anterior abdominal wall, sharp tenderness on palpation, marked Blumberg's sign. On percussion: absence of dullness over the liver, dull sound in flat places. Weakening of intestinal noises on auscultation of the abdomen. Hanging and tenderness of the anterior wall of the rectum, pain and hanging of the vaginal vault	Gradually increasing leukocytosis in blood with a shift of the leukocyte formula to the left. Urine without changes, blood in urine in bladder rupture	Free gas under the diaphragm, high standing of the diaphragm and limitation of its mobility
Closed abdomina	Injuries to the	Develops suddenly,	Develops rarely	Severe. Pallor, cold	The abdomen is slightly bloated, soft	A gradual decrease in	High standing and limitation

l injury with damage to parenchymal organs.	abdomen, especially secondary to malaria, liver cirrhosis, echinococcosis, etc.	without clear localization, radiates to the chest and shoulder blades. A characteristic reduction of pain in sitting position – "tilting doll symptom"		sweat. Patient is restless, pulse is accelerated, blood pressure is low. Body temperature in the first day is normal. Stool is without changes.	or moderately tense on palpation, positive Blumberg's sign. On percussion: dull sound in flat places. Intestinal noises are preserved. Hanging and pain in the anterior wall of the rectum, vaginal vault	hematocrit, the number of red blood cells and the level of hemoglobin. Urine is without changes.	of mobility of both diaphragm domes. Free fluid in the flat areas of the abdominal cavity, which moves in motion
Acute appendicitis	Gradual displacement of pain from epi- or mesogastrium to the right ileac region.	Constant, moderate, in the right ileac region, intensifies in movement and cough.	Once-twice, without bringing relief. Nausea is almost constant	Satisfactory, body temperature is normal or low-grade. Retention of stool, defecation may be single and loose.	Muscular tension and tenderness in the right ileac region, which increases in the position of the patient on the left, positive Blumberg's sign. Sometimes with pain in the right wall of the rectum and the right vaginal vault	Moderate leukocytosis. Shifting of leukocyte formula to the left	Without abnormalities
Acute cholecystitis	Attacks of acute cholecystitis, cholelithiasis in history	Intensive, constant in the right hypochondrium, radiates to the right shoulder and shoulder blade.	Multiple, sometimes with an admixture of bile, brings relief	Moderate severity, the patient is restless. Body temperature is elevated. Retention of stool	Sharp pain in the right hypochondrium, including when tapping on the right costal arch. The gallbladder may be palpable	Considerable leukocytosis, shifting of leukocyte formula to the left. ESR is increased.	Occasional X-ray contrast gallstones
Acute pancreatitis	Attacks of acute pancreatitis, considerable intake of alcohol or spicy fatty food, diseases of the biliary tract in	Gradually increasing to severe in the epigastric region or stabbing.	Recurrent, uncontrollable, painful, abundant, without relief.	Severe. The patient is restless, the skin is pale. Body temperature is normal, later it is elevated. At first, the pulse is infrequent, then more frequent.	The abdomen is bloated, soft and tender on palpation, pain in the epigastric region, pain in the lumbar regions. Vague Blumberg's sign. On auscultation: peristaltic noises of the intestine are weakened or absent	Moderate leukocytosis. Increase in amylase levels in blood and urine. ESR is increased	High standing and limitation of mobility of both domes of the diaphragm, transverse colon is bloated with air

	history			Retention of stool			
Perforated ulcer of the stomach and duodenum.	Ulcer of the stomach or duodenum in history.	Suddenly occurring, extremely intense, permanent, in the epigastric region or generalized.	Not typical	Severe, patient's position is forced (with feet raised to the abdomen), the patient tries not to move. Bradycardia in the first hours, later increasing tachycardia. Body temperature in the beginning is not elevated	The abdomen is indrawn, sharp tension of abdominal wall muscle. Pain all over the abdomen on palpation. Sharp Blumberg's sign. On percussion: hepatic dullness is absent, peristaltic noises are not auscultated.	Moderate leukocytosis, shift of leukocyte formula to the left.	Free gas under the diaphragm dome, high standing and limitation of mobility of both domes of theh diaphragm
Intestinal obstruction	Inflammatory processes, tumors, surgical interventions on the abdominal organs.	Cramping, very intense, throughout the abdomen.	Multiple, which brings temporary relief	Severe, the patient is restless. Tachycardia. Body temperature is not elevated. Prolonged retention of stool and gases	Abdomen is evenly or assymetrically bloated and soft; there is no muscle tension and Blumberg's sign at the beginning of the disease. On auscultation of the abdomen intestinal noises are uneven and intense. "Splash noise" on auscultation. Gaping of rectal sphincter in rectal examination	Moderate leukocytosis, shift of the formula to the left, ESR is increased	Bloated intestinal loops with fluid levels in them
Thrombosis and embolism of mesenteric blood vessels	Cardiovascular diseases. Disrupted cardiac rhythm	Sudden, constant, very intense throughout the abdomen	Multiple, does not bring relief	Severe. The patient is restless. Pallor, cold sweat, acrocyanosis. Accelerated and arrhythmic pulse. Blood pressure is low. Body temperature is not	The abdomen is soft, bloated, moderately painful in all divisions. Intestinal noises are weakened, then absent. Peritoneal symptoms are vague. Blood in stool in rectal examination	An increase in hemoglobin content, marked leukocytosis, shift of leukocyte formula to the left, ESR is increased	Accumulation of gas in the intestine

				elevated. At first stool has an admixture of blood, followed by retention of stool and gas			
Disrupted ectopic pregnancy	Disorders of the menstrual cycle, bloody discharge from the vagina	Sudden, intense, over pubic symphysis or generalized, radiating to the lumbar region, rectum	Rarely	Severe, marked weakness, unconscious condition. Skin and mucous membranes are pale. Frequent, weak pulse, low blood pressure, normal body temperature.	The abdomen is soft, painful over the pibic symphysis or in the ileac regions, on percussion: dull sound in flat areas. Vague Blumberg's sign. Bloody discharge from the enlarged soft uterus, hanging of the posterior vaginal vault	A decrease in hematocrit, hemoglobin level, erythrocyte count	Radiographic findings are without abnormalities
Ovarian rupture, torsion of the ovarian cyst	Previously diagnosed ovarian cyst	Sudden, intense in the lower abdomen, radiates to the lumbar region and the inner surface of the thigh	Not typical	Satisfactory, moderate tachycardia, normal body temperature	The abdomen is soft, painful in the lower regions with muscular tension in the same areas. Blumberg's sign may be positive. Sharp tenderness in displacement of the cervix and on palpation of the posterior vaginal vault. Sharply painful rounded elastic formation to the right or left of the uterus.	As a rule, without deviations from the norm.	Same

Therapeutic approach. A patient with suspected acute abdomen should immediately be hospitalized to an emergence care surgical hospital. Transportation of patients in a severe condition should be fast, safe and cautious. It is necessary to provide for the possibility of medical treatment aimed at stabilizing hemodynamic disorders at the prehospital stage.

The nature of therapeutic activities in AAS depends, first of all, on the underlying disease, which necessitates confirmation of the diagnosis, carrying out special additional studies. Before final diagnosis, the decision on the need for emergency surgery and, accordingly, determination of all therapeutic approaches, it is prohibited to administer liquid and food, and is advisable not to use analgesics, hypnotics, neuroleptics, tranquilizers, laxatives, enemas.

In some inflammatory processes that do not endanger the development of peritonitis, it is becessary to conduct complex conservative treatment, however, ***cases of AAS with signs of peritonitis***

secondary to perforation of hollow organs, intestinal obstruction, acute inflammatory diseases of the abdominal cavity, strangulated hernia, etc. ***require emergency surgical intervention for the treatment of life-threatening conditions, the nature of which depends on the disorder***.

It is carried out immediately after hospitalization or it is preceded by short-term intensive infusive preoperative preparation (within 1-2 hours) to correct hemodynamic, water-electrolyte and other disorders. Patients in a very serious condition with ongoing internal bleeding undergo surgical intervention immediately after admission to the in-patient department, along with resuscitation procedures. In complex cases, the nature and volume of therapeutic measures is determined by a consensus of specialists (surgeon, critical care physician, therapist, etc.).

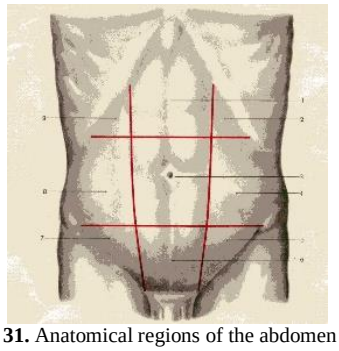


Fig. 31. Anatomical regions of the abdomen:
1 - epigastric;
2,9 – left and right hypochondrium;
3 – umbilical;
4,8 – left and right lateral;
5,7 – left and right ileo-inguinal;
6 - pubic

Prognosis depends on the nature and severity of the underlying disease, as well as on the time elapsed from the onset of the disease until hospitalization, patient's age and concomitant illnesses. The prognosis is unfavorable in advanced generalized forms of peritonitis, intestinal obstruction and thrombosis of mesenteric vessels with severe necrosis, especially in the elderly and senile patients.

The faster the patient with AAS is hospitalized, the earlier the exact diagnosis is made and the adequate urgent surgical treatment is performed, the lower the mortality and the better the immediate and long-term results.

Acute abdomen *is a clinical term, which means a serious condition of a patient with acute abdominal diseases, requiring urgent surgical intervention.*

The term can only be used as part of a preliminary diagnosis when referred to a hospital.

Inflammatory diseases of the abdominal organs include such diseases as acute appendicitis, acute cholecystitis, acute pancreatitis, perforated ulcer, acute intestinal obstruction, strangulated hernia, etc. *The main manifestation of these diseases, that is, the* ***leading clinical syndrome****, is* ***abdominal pain***, localization and the nature of which differs depending on the type of pathological process. Another manifestation of acute inflammation of the abdominal cavity is *intoxication*, which increases with increasing organ destruction and inflammation of the peritoneum. Features of inflammatory diseases of the abdominal cavity include progressive course of the disease, the possibility of various complications (peritonitis, intraperitoneal and organ abscesses, etc.), which complicate the patient's condition and worsen the prognosis of the disease. Patients with inflammatory diseases of the abdominal cavity require an urgent qualified surgical examination, consultations of adjacent specialists, the use of a complex of modern methods of examination and the provision of qualified, often specialized, differentiated emergency care.

In Ukraine, 80% of the diseases of the abdominal organs include acute appendicitis, acute cholecystitis, and in recent years the number of diseases of acute pancreatitis has increased.

The incidence of acute appendicitis (AA) is 20.7 per 10 thousand people with fluctuations from 13.8 to 31.1 per 10 thousand people in different regions of the country; surgical activity is 99.6%, and postoperative lethality is 0.05% (from 0 to 0.13% by region).

In the second place among acute pathologies of the abdominal cavity is the incidence of acute pancreatitis (AP): 6.7-6.95 per 10 thousand people (from 2.8 to 12.4 in different regions); surgical activity in AP is 10.6-11.0% (4.9-21.7% in different regions), and postoperative lethality reaches an average of 14.1-14.66%

The incidence of acute cholecystitis (AC) is 6.25 per 10 thousand people (from 1.48 to 10.8 in different regions) and ranks third among all acute surgical diseases of the abdominal cavity. Surgical

activity in AC is 57.5-58.0% (from 36.3 to 90.5 by regions), postoperative lethality varies from 0.28 to 0.32%, and at late hospitalization is up to 46.5%.

The incidence of perforated gastroduodenal ulcer (PU) ranges from 1.08 to 2.57 per 10,000 people in different regions; postoperative lethality ranges from 0.64 to 5.64%

The incidence of acute intestinal obstruction (AIO) is more than 9% of all acute diseases of the abdominal cavity, and mortality is up to 25%.

All internal organs of the abdomen are located in the ***abdominal cavity***. Its borders are in front and on the sides: the anterior and lateral walls of the abdomen, behind: the lumbar region, on the top: the diaphragm, below: the conditional plane along the line separating the cavities of the large and small pelvis.

The four conditional lines on the anterior abdominal wall divide the stomach into 9 anatomical regions (Figure 31).

II.3.1. ACUTE APPENDICITIS

Classification of acute appendicitis (AA). V.I. Kolesov (1972) classification of acute appendicitis is adopted and used in clinical practice.

1. *Simple* (superficial).
2. *Destructive*:

a) phlegmonous;
b) gangrenous;
c) perforated;
d) complicated
- appendicular infiltration,
- appendicular abscess
- appendicular peritonitis;

e) other complications (pylephlebitis, sepsis, etc.).

Presentation. In history taking with *complaints of the underlying disease*: pain in the right ileac area, which occurs suddenly, is permanent and has no characteristic irradiation; nausea, sometimes a single vomiting, retention of stool or loose stool; sub-febrile temperature.

Disease history: a sharp beginning without apparent reason, a gradual increase in pain, possible previous attacks. It is necessary to specify the time of the disease, whether the patient sought medical advice and where.

The general condition at the beginning of the disease is often slightly disturbed and remains relatively satisfactory. Behavior is calm, the patients lie on the back or right side.

Cardiovascular system: the pulse is accelerated at the beginning, correspondingly to the increase of temperature to subfebrile. Development of destructive forms and complications with severe intoxication is associated with tachycardia that does not correspond to an increase in temperature. Blood pressure is normal. Tones of the heart are without abnormalities. *Respiratory organs* are withoiut abnormalities.

Local status. *Examination of the abdomen* shows a lag in the anterior abdominal wall in the right ileac region when breathing. *Palpation of the abdomen* determines ***three main signs of AA,*** namely local pain and muscle tension in the right ileac region, positive Blumberg's sign. ***Positive pathognomonic "appendicular" signs***: *Rovsing', Voskresensky's ("sliding" or "shirt"), Sitkovsky's, Bartomier-Mikhelson's, Razdolsky's, Obraztsov's, Ostrovsky's, Krimov's (inguinoscrotal), Krimov-Dumbadze's (peritoneo-umbilical), Chugaev's ("string of appendicitis"), Larock's and other signs. Percussion and auscultation* of the abdomen may show no abnormalities, there may be some decrease in peristaltic intestinal noise in the right ileac region.

It should be remembered that clinical examination of patients with suspected acute appendicitis requires rectal and vaginal studies, in which the pain of the pelvic peritoneum and pelvic organs is determined (especially in the case of pelvic location of the appendix)

In order to avoid diagnostic errors in ***atypical forms of AA***, it is necessary to remember the features of presentation and to identify a number of specific signs, caused by ***different location of the appendix*** (Fig. 32)

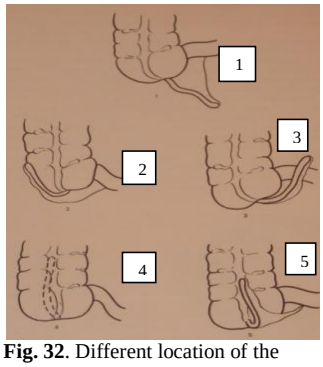


Fig. 32. Different location of the appendix:
1 – pelvic; 2 – anterior;
3 – medial; 4 – retrocecal;
5 - subhepatic

In retrocecal location of the appendix. In addition to pain in the right half of the abdomen (and often in the absence of it), the patient notes pain in the right lumbar region, sometimes radiating to the right thigh, difficulty in walking, increased pain in the abdomen. Walking may be characteristically difficult: the patient "draws" the right leg, without bending it in the hip joint (except for the contraction of *m. iliopsoas*, which is in contact with inflamed appendic, resulting in exacerbation of pain). The abdomen is soft, often moderately painful in the right ileac region, without tension of the muscles and with negative *Blumberg's sign*. At the same time, there is severe pain in the right lumbar region (*Yaure-Rozanov's sign*) or over the the right iliac crest and the tension of the muscles of the right lateral wall of the abdomen. *Gabai's, Varlamov's, Cope 1, and Cope 2* signs are positive. *Pasternatsky's sign* is positive, urine analysis shows elevated levels of erythrocytes and leukocytes, protein (associated with the transition of the inflammatory process to the abdominal cell wall with the involvement of the right ureter and renal pelvis). It is often incorrectly diagnosed with renal colic, acute pyelitis or pyelonephritis.

In pelvic location of the appendix, when it is located in the small pelvis, the pains are located above the right inguinal ligament or above the pubis, the same place is the location of pain, muscular tension, and Blumber's sign. Rovsing's, Voskresensky's and other signs are vague or absent. Transition of inflammation to the organs of the small pelvis (bladder, rectum, uterus with adnexa) may be accompanied by accelerated urination, loose stool. In such cases, *rectal* examination (a sharp pain in the anterior wall of the rectum) and *vaginal* in women are particularly important and informative. It should be differentiated from acute cystitis, dysentery, acute adnexitis, endometritis.

In high "subhepatic" location of the appendix, when both pain, and tenderness, and muscle tension with positive Blumberg's sign are localized in the right hypochondrium, classical "appendicular" symptoms are absent, and "bladder" ones (Ortner's, Ker's) can be weakly positive. It should be differentiated form acute cholecystitis. In these difficult cases, it is necessary to use the differential *Dupuis de Frenel's sign* and to expand the volume of additional methods of examination (biochemical blood assay, urgent ultrasound of the abdominal cavity, etc.).

Medial (mesoceliac) location of the appendix, when its apex is directed to the navel, and it itself is located between the loops of the small intestine is accompanied by a marked impairment of the general condition, generalized pain and tenderness, soft abdomen, marked bloating of the abdomen (due to early development of functional intestinal obstruction associated with the transition of the inflammatory process to the intestinal loops and the root of the mesentery of the small intestine with the development of paresis of the intestine). An erroneous diagnosis of acute intestinal obstruction, enteritis, and acute appendicitis is thought to occur only with the appearance of intraperitoneal infiltrate, sometimes with signs of abscess (hyperemia of the abdominal skin in the periumbilical area, a symptom of fluctuation), muscle tension and symptoms of peritoneal irritation. *Specific Gorn's, Gurevich's signs* can help in diagnosis.

It is necessary to remember about frequent diagnostic errors in "atypical" forms of AA that develop in ***young children (up to 5-7 years)*** due to a number of anatomical and physiological peculiarities of the child's body (underdevelopment of the large omentum, reduction of plastic properties of the peritoneum and resistance of the child's body to an infection that leads to rapid progression of inflammation with the development of destructive forms of peritonitis), with increased reactivity of the child's body and the tendency to generalization of any pathological process manifested by fever, vomiting, severe signs of intoxication, tachycardia, and frequent loose stool (even in simple

forms of AA). All of the above is complicated by the difficulties of physical examination of a small child, when only the sight of a doctor in a white gown causes a sharp negative reaction of the child, vigorous crying, leading to tension of muscles of the anterior abdominal wall. A false diagnosis of acute colibacillary infection or acute dysentery is made and the child is hospitalized to an infectious hospital, and referred to a surgical department with severe appendicular peritonitis. Specific *"children's" symptoms* can help in diagnosis, namely forced position on the back or right side with the drawn legs and arm on the right ileac region, the *symptoms of "drawing the leg"* and *"pushing the hand"*, when the child begins to actively push the surgeon's hand in palpation of the abdomen in the right ileac region.

In the elderly (from 60 to 74 years) and senile (from 75 to 89 years) patients, who currently make up more than a third of surgical patients at in-patient departments, the main difference in the presentation as compared to younger patients is not so much in "atypical" signs but in the *smoothness and vagueness of the same symptoms* that occur in all age groups (V.I. Kolesov, 1972): the disease does not start as acutely as in young, the pains are less pronounced (even in destructive forms), they are first localized all over the abdomen, and then - in the right ileac region, it is often accompanied by vomiting, bloating, retention of stool and gas. An important sign is pain on percussion of the right ileac region (*Rozdolsky's sign*), tension of the anterior abdominal wall muscles is vague or absent (due to the weakening of reflexes, muscle flaccidity, excessive development of the subcutaneous fat of the abdomen). General reaction to inflammation is weakened: the temperature is often normal, normal or slightly elevated leukocytosis, but, as a rule, there is a *shift of leukocytic formula to the left*. It should be remembered that in the elderly, presentation of AA is aggravated, and the diagnosis is complicated by the presence of concomitant, often severe diseases (diabetes, hypertension, emphysema, etc.).

In women in the second half of pregnancy, diagnosis of AA is difficult due to the displacement of the cecum and the appendix upwards by the enlarged uterus. It is necessary to conduct a differential diagnosis of AA with acute diseases of the gallbladder and pancreas (acute cholecystitis and pancreatitis), the right kidney (pyelitis of pregnant), with acute obstetric-gynecological disorders (miscarriage, acute right adnexitis), etc. *The combination of 3 signs:*

1) sudden onset of the disease,

2) pain in the right half of the abdomen;

3) pain in palpation of the abdomen to the right, are considered to be indications for obligatory hospitalization of pregnant to the surgical department for examination, supervision and diagnosis of AA in any period of pregnancy for urgent surgical treatment.

In accordance with the standard schemes, the ***plan for laboratory and instrumental examination*** of a patient with suspected AA includes:

1) clinical blood test;

2) clinical analysis of urine;

3) determination of blood type and Rh factor;

4) mandatory inspectional radioscopy (radiography) of the abdominal cavity and chest;

5) compulsory manual examination of the rectum and vaginal examination in women;

6) in patients over 40 years or by indications, determination of blood glucose, coagulogram, ECG, ultrasound examination of the abdominal cavity;

7) by indications the patients are examined by adjacent specialists;

8) in cases of complicated diagnosis, laparoscopy is used to detect direct and indirect signs of AA.

Characteristic abnormal changes may appear: leukocytosis, shift of leukocytic formula to the left; destructive and complicated forms with signs of severe intoxication are accompanied by "toxic nephropathy" symptoms (protein, minor leukocytosis and erythrocyturia, etc.); "direct" (inflamed changes in the appendix often with fibrinous stratifications) or "indirect" laparoscopic signs (greater omentum with adhesions in the area of the dome of the cecum, peritoneal cloudy effusion, hyperemia of the visceral and parietal peritoneum in the ileac region).

Differential diagnosis in suspected AA should be performed with acute surgical diseases, infectious, acute surgical diseases of the digestive organs (acute gastritis, enterocolitis, exacerbation of

peptic ulcer, food poisoning, acute dysentery, perforated ulcer of the stomach and duodenum, acute intestinal obstruction - invagination, Crohn's disease, acute typhlitis and perforation of colorectal cancer, etc.), with other diseases of the abdominal cavity (acute cholecystitis, pancreatitis, strangulated hernia, etc.), with acute gynecological diseases (acute right-sided adnexitis, torsion of the pedicle and rupture of the right ovarian cyst, ectopic pregnancy), with acute urological diseases (renal colic in acute pyelonephritis, cholelithiasis, hydronephrosis, acute cystitis), with abdominal form of myocardial infarction, etc.

Differential diagnosis of AA with ***acute right-sided adnexitis***. Feature of presenting complaints and history data (purulent discharge from the vagina, recent abortion, treatment of gonorrhea, etc.). *Shilovtsev's sign*: in the position of lying on the back, the doctor reveals a painful point in the right ileac region and, without lifting his hands, suggests the patient to turn to the left side; if the pain at the found point disappears and is shifted more medially, this indicates adnexitis, but if the localization of the point of maximum pain remains unchanged (not shifted), then most likely, the patient has AA (which is explained by the greater mobility of uterine adnexa (correspondingly, their greater displacement) in comparison with the mobility of the appendix). *Zhendrinsky's sign*: in the position of lying on the back, the doctor reveals a painful point in the right ileac region and, without lifting his hand, suggests the patient to sit in bed - an increase in pain indicates appendicitis, a decrease means acute salpingo-oophoritis. *In vaginal examination*: positive *Promptov's sign*, an increase in abdominal pain in pendulum movements of the uterus and positive *Pozner's sign*, an increased pain in the abdomen with the displacement of the uterus upward suggests acute adnexitis. Of great importance for diagnosis is the ***puncture of the posterior vaginal vault*** - the detection of cloudy effusion and thick odorless pus indicates pelvioperitonitis of gynecological origin.

Differential diagnosis of AA with ***ectopic pregnancy***. Presenting complaints include weakness, short-term loss of consciousness, vaginal bleeding), history data (delayed menstrual period), symptoms of intra-abdominal bleeding (pallor, tachycardia, hypotension, peritonitis symptoms, free fluid in the abdomen), vaginal and bimanual examination data, puncture of the posterior vaginal vault (blood).

Differential diagnosis of AA with ***acute urological disorders***. History data (cholelithiasis, attacks of renal colic, etc.), positive Borisov's chloriethyl test, Lorin-Epstein's novocaine blockade, chromocystoscopy (delayed or no secretion of blue-colored urine from the right ureter) indicate urological disorders.

Differential diagnosis of AA with ***perforated ulcer of the stomach and duodenum***. The following signs are typical for perforation: "stabbing" pain in the epigastrium, sudden onset of the disease, past "ulcer" history, absence of accelerated pulse and elevated body temperature in the first hours of the disease, "plate-like" abdomen, disappearance of liver dullness and presence of free gas in the abdominal cavity (*Spizharny's and Zhober's signs*), peritoneal signs, other pathognomonic symptoms of perforated ulcer (Eleker's, de Quervain's signs, auscultative Guiston's triad, etc.).

Differential diagnosis of AA with food poisoning. The pain is localized in the epigastrium or in the right half of the abdomen, the general condition worsens, vomiting appears. Signs of intoxication appear prior to abdominal pain, intoxication grows rapidly, vomiting is often multiple. There is no tension in the anterior abdominal wall muscles or Blumberg's sign.

Differential diagnosis of AA with ***acute intestinal obstruction***. Cramping abdominal pain, recurrent vomiting, bloating of the abdomen, retention of stool and gas; history data (adhesive peritoneal disease, Savitsky's syndrome of "small signs", etc.); absence of peritoneal signs and increase in body temperature at the beginning of the disease; positive *Val's, Kivul's, Sklyarov's ("splashing noise"), Spasokukotsky's ("the noise of a falling drop") signs*, intense at initial stages, and then sharply weakened peristalsis of the intestine and "dead abdomen" sign; peritoneal signs, positive *Kulenkampf's sign, "Obukhov hospital" sign*; X-ray examination shows fluid levels with gas above them in the stretched intestinal loops ("Kloyber's cups").

Differential diagnosis of AA with ***acute cholecystitis***. Characteristic complaints of pain in the right hypochondrium radiating to the right shoulder or right shoulder blade, fever, dyspeptic disorders (recurrent vomiting often with an admixture of bile); history data: acute onset usually after faults in the diet. Symptoms include pain and possible muscle tension in the right hypochondrium, other

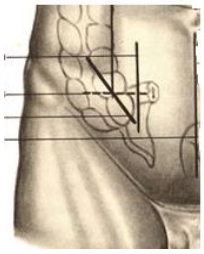


Fig. 33. Accesses to the appendix

pathognomonic symptoms of acute cholecystitis (Kerr's, Ortner's, Murphy, Mussie-Georgievsky's, etc.). Ultrasound examination often shows stones in the gallbladder and thickening of its walls.

Differential diagnosis of AA with ***acute pancreatitis***. History data: acute pancreatitis usually begins after a fault in the diet with a severe pain in the upper abdomen radiating the back, often "protective" in nature, accompanied by multiple vomiting, which does not bring relief. General condition worsens, toxicity increases. Positive Gobiet's, Cullen's, Mondor's, Halsted's, Korte's, Koch's, Voskresensky's, Mayo Robson's signs, etc. Amylase levels in the blood and urine are increased. X-ray examination shows inflated transverse colon, intestinal pneumatosis. Ultrasound examination detects an increase and a decrease in echogenicity of the pancreas.

Differential diagnosis of AA with ***pleuropneumonia***. History data: severe general condition, high fever, chills. Localization of pain syndrome and the strength of possible muscle tension varies with every hour. Auscultation determines weakened breath, crepitation, moist rattles. X-ray shows limited movement of the dome of the diaphragm, infiltration of the lung tissue, and sometimes fluid in the sinus.

Treatment of patients with AA. *General provisions*.

1. Patients with suspected AA require urgent hospitalization to a surgical hospital.

2. ***Diagnosed AA is an absolute indication for an urgent operation regardless of the form of the disease, the age of the patient, the period from the onset of the disease.***

3. Exclusions are patients with a dense, clearly separated appendicular infiltrate (at the same time progressive and abscessing infiltration is subject to surgery).

4. Pregnancy, even in the second half, is not a contraindication to surgery.

5. For patients with severe concomitant diseases (acute myocardial infarction, decompensation of blood circulation, severe pneumonia, etc.) the possibility of surgical intervention, which may be more dangerous than AA itself, is decided by the case conference of physicians in each case individually.

In complicated diagnosis, a follow-up observation is conducted for 2-3 hours and a thorough examination with the use of modern methods is performed; urgent laparoscopy is widely used. If during this period the symptoms persists and the diagnosis of AA is not eliminated, operation is necessary.

Surgical treatment of acute appendicitis.

Anesthesia. In uncomplicated AA adult patients are administered local anesthesia. In the case of intolerance to local anesthetics, in children, in patients with AA, complicated diffuse or generalized peritonitis, in technical difficulties during the operation, general anesthesia, mainly endotracheal anesthesia with controlled respiration, neuroleptanalgesia is administered.

Operational accesses (Figure 33). The most commonly used oblique-variable section of Volkovich-Dyakonov, which runs perpendicularly to the line connecting the navel and the front upper axis of the right iliac bone, through McBurney's point, located on the boundary of the outer and middle third of the indicated line. In diffuse peritonitis, the use of lower midline laparotomy is indicated, in generalized peritonitis wide laparotomy according to Petrov (from the xiphoid process to the pubis). There are accesses according to Lennander (para-rectal), Shprengel (transverse), and others.

Types of appendectomy: classical, retrograde, atypical. Recently, laparoscopic appendectomy has been used.

Completion of operation. Blind layered suture is used in simple and phlegmonous form of AA. In gangrenous form, the glove-tube drainage through the main wound is used. Drainage is also required in complicated forms of AA. There may be indications for the use of *gauze tampons*:

1) with hemostatic purpose in insufficient hemostasis;

2) to distinguish the location of the operation site from the rest of the abdominal cavity (for example, when detecting a dense appendicular infiltrate which cannot be eliminated and the only indication is to drain the abdominal cavity, in possibility of abscess in the postoperative period (the patient is weakened, sugar diabetes, etc.).

Postoperative period. After surgery, bed rest should be prescribed for 12-24 hours, cold and weight for wound for 2 hours, painkillers for 1-2 days. Drinking and liquid food can be allowed in 8-12 hours, transferring the patient gradually (every 2 days) to the diet 1a, 1b and 1. Change the bandage on the first day. Sutures are removed in uneventful postoperative course on the 5^{th} day, in elderly and weakened patients stitches are removed in 7-8 days. It is necessary to conduct prevention of possible postoperative complications. Before discharge from the hospital it is necessary to conduct a clinical blood test. After appendectomy in absence of complications, working capacity is restored 3-4 weeks after surgery.

Classification of complications after appendectomy (I.M. Matiashin et al., 1974).

Complications in the abdominal wall (in the wound):

I. *Early:*

1) infiltration of postoperative wound;
2) suppuration;
3) bleeding;
4) hematoma;
5) eventration.

II *Late:*

1) ligature fistulae;
2) inflammatory "tumors" of the anterior abdominal wall;
3) postoperative ventral hernia;
4) development of colloid scars;
5) development of neurinoma.

Complications in the abdominal cavity.

I. *Early:*

1) intra-abdominal bleeding;
2) peritonitis;
3) infiltrates of the anterior abdominal wall;
4) abscesses of the abdominal cavity;
5) phlegmon of retroperitoneal cellular tissue;
6) acute intestinal obstruction secondary to peritoneal commissures;
7) acute postoperative pancreatitis.

II *Late:*

1) inflammation of stump;
2) infiltrates and abscesses of the abdominal cavity;
3) inflammatory tumors;
4) intestinal fistulae;
5) peritoneal commissures
6) relapse of pain after appendectomy.

General complications of appendectomy: in the cardiovascular, respiratory or urinary system.

Measures that contribute to timely diagnosis and prevention of postoperative complications include daily physical, laboratory, and, if necessary, X-ray control, early uplift, exercise therapy, respiratory gymnastics, chest massage, consultations of adjacent specialists.

In absence of timely surgical intervention AA, as a rule, leads to the development of dangerous complications - appendicular infiltrate, appendicular abscess, appendicular peritonitis (topic II.11).

II.3.2. PERFORATED ULCER OF THE STOMACH AND DUODENUM

Classification.

I. *By etiology.*
1. Perforation of peptic or duodenal ulcer.
2. Perforation of cancer ulcers (ulcus-tumor).
3. Perforation in local disturbance of blood circulation in the gastric wall (atherosclerosis, hypertonic disease).
4. Perforation in parasite damage to the wall.
II. *By presentation*
1. Typical form of perforated ulcers.
2. Atypical form of perforated ulcers.
III. *By pathological data.*
1. Perforation of acute ulcers.
2. Perforation of chronic ulcers.
IV. *By topographic and anatomical localization.*
1. Perforation of gastric ulcers.
2. Perforation of duodenal ulcers (anterior and posterior wall).

Clinical signs. There are three periods of clinical course of PU:

I - pain or shock (up to 6-8 hours);

II - the period of "imaginary well-being" or imaginary improvement (up to 8-12 hours);

III - the period of progressive peritonitis and toxemia (after 12 hours)

The following ***4 main symptoms*** are characteristic of PU:

1) *"stabbing" pain in the epigastrium;*

2) *ulcerative history;*

3) *"plate-like stomach";*

4) *symptoms of spontaneous pneumoperitoneum - free gas in the abdominal cavity (Spizharny's and Zhober's signs).*

Presentation: Acute sudden, permanent "stabbing" pain in the epigastrium (Dieulafoy's sign), often radiating to the region of the shoulder blade or collarbone (Oelecker's sign), sometimes with a single vomiting.

History. Peptic ulcer is more common in the period of exacerbation. If the patient has not been examined before, it is possible to identify *ulcerative history*: pain in the epigastric area after eating, hunger, night pain, heartburn, taking soda to remove burning pain behind the sternum, seasonal pain aggravation (in the spring and autumn).

*On examination: **In the 1st period***: forced position, often on the right side with legs brought to the abdomen; pallor of the skin; suffering facial expression; pulse in the first hours is not accelerated (*Grekov's sign*), possible bradycardia; the stomach is indrawn, does not take part in breathing, on palpation: stomach is sharply painful in the epigastrium, sharply tense ("plate-like stomach"), positive *Blumberg's sign*; on percussion: disappearance of hepatic dullness (*Spizharny's sign*).

In the 2nd period: fever, nausea and vomiting in abdominal pain intensity. On examination: sharp pain, muscle tension and positive Shchotkina-*Blumberg's sign* in the right epigastrium, over the pubis; *De Quervain's sign* - pain, tenderness, muscle tension and positive Blumberg's sign in the right ileac region; dull percussion sound in flattened abdominal areas (due to peritoneal exudates); weakening of the intestinal peristalsis; on rectal examination: sharp pain in the upper wall of the rectum (due to inflammation of the peritoneum of the Douglas area, *Kulenkampf's sign, or "Douglas' cry"*). In localization of PU in the region of the posterior wall of the duodenum, subcutaneous emphysema in the navel region can be detected as a result of the spread of gas along the circular ligament of the liver (*Vigyiazo's sign*).

In the 3rd period: severe condition, signs of intoxication and dehydration: high fever, multiple vomiting, thirst, *"Hippocrates' face", "tongue as a brush",* low blood pressure and severe tachycardia; the abdomen is bloated, does not participate in breathing, painful and tense in all departments with positive Blumberg's sign; on percussion: dullness in flat places; auscultative *Guiston's triad* - heart tones, peritoneal friction noise and the "silver bell" in the epigastrium on the

Fig. 34. Radiological Zhober's sign in perforated ulcer , asickle-like strip of gas in the abdominal cavity under the right dome of the diaphragm

background of the absence of intestinal peristalsis ("dead abdomen" or "grave silence" sign); sharply positive Kulenkampf's sign in the study of the rectum

Diagnosis. The main method is ***inspectional radioscopy and radiography of the abdominal cavity***, positive *Zhober's sign* in all periods, a sickle-like strip of free gas between the right diaphragm dome and the liver (Figure 34).

In the **atypical form of PU** (perforation of the ulcer of the posterior wall of the stomach or "covered" perforation), *two main symptoms*, namely the "plate-like abdomen" and spontaneous pneumoperitoneum (Spizharny's and Zhober's signs) *are absent*, and presentation resembles that of acute pancreatitis (intense pains in the epigastrium). This is due to the fact that at the beginning of the disease, in perforation of ulcer of the posterior wall of the stomach, the contents of the stomach and air accumulate in the omental bag (*bursa omentalis*), the parietal peritoneum for some time remains intact, and in "covered" perforation the release of gastric contents into the abdominal cavity is short and very insignificant. But in PU the patient more often has peptic ulcer in history (although it is not always possible to identify a patient with "ulcer history"). In acute pancreatitis most often patients have history of cholelithiasis with attacks of acute cholecystitis, or alcoholism, the beginning of the disease the patient most often associates with disbalanced diet or drinking alcohol; Gobiet's, Korte's, Mayo Robson's signs are positive, biochemical blood assay shows an increase in blood amylase, which is not characteristic of PU.

Hennelt's test (*pneumogastrography*) plays a critical role in the diagnosis of atypical form of PU, when inspectional radioscopy of the abdominal cavity in suspected PU does not determine spontaneous pneumoperitoneum sign (Zhober's sign). A thick gastric probe is used to administer 1.5-2.0 liters of air into the stomach and after 15-20 minutes, an X-ray examination is repeated, in which in most cases a characteristic sickle-like strip of free gas appears below the right diaphragm dome.

In a negative result of the test, diagnostic laparoscopy (if possible) is used, which, starting with the second stage of PU, detects signs of purulent peritonitis with a *positive Neymark test* (with iodine), and in acute pancreatitis, the hemorrhagic nature of the peritoneal exudate with high amylase content, plaques of steatoenecrosis on parietal and visceral peritoneum.

Differential diagnosis *is performed with acute cholecystitis, pancreatitis, intestinal obstruction, and also with an abdominal form of myocardial infarction.*

Treatment. A patient with suspected PU should urgently be hospitalized to a surgical department. In the hospital, the patient should undergo a thorough clinical, laboratory, X-ray, if necessary, endocopic examination, and after confirmation of PU, an ***urgent operation for this life-threatening condition*** should be performed, since the latter is currently the main method of treating perforated ulcers.

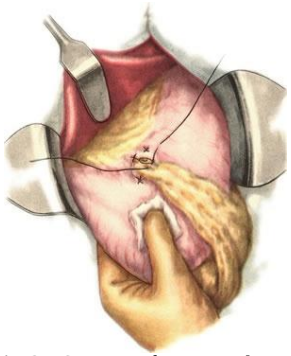


Fig. 35. Suturing of PU according to Oppel-Polikarpov

Operative treatment. There are ***two groups of surgical interventions*** *in PU*:

1) *palliative operations*, aimed at eliminating the causes of peritonitis and rehabilitation of the abdominal cavity - suturing of the perforation or its plastic closure with the pedicle of the omentum, sanation and drainage of the abdominal cavity;

2) *radical operations* aimed not only to eliminate the causes of peritonitis and rehabilitation of the abdominal cavity, but also to cure the patient from peptic ulcer - the primary classical resection of 2/3 of the stomach (currently rarely used) and organ-preserving operations - excision of PU with gastro- or duodenoplasty, is combined with various types of vagotomy and drainage of the stomach by operation on indications

The most widespreaad ***palliative operations*** in our country is *suturing of PU according to M.M. Ostrovsky* (1958), which implies suturing of ulcer in the lumbar direction in relation to the longitudinal axis of the affected organ by two-row sutures. The first row of sutures is applied through all layers of the organ wall; the second is a serous-muscle suture, with suturing of the strand of the septum (for additional peritoneal repair).

Plastic closure of the perforated opening with the "pedicle of the omentum" according to Oppel-Polikarpov is used in severe infiltration of the gastric wall around the perforated opening or its location in a narrow pre- or post-pyloric area (Figure 35). D.P. Chukhriyenko's (1958) method implies circle-wise suture, I.I. Neimark's (1958) method uses single-row suture with non-absorbent threads and without involvement of the mucous membrane.

The operation of choice is excision of PU in the limits of healthy tissues, with further reliable stitching of the defect by two-row sutures.

An important stage of the operation is careful rehabilitation of the abdominal cavity by drying (in the local and diffuse form of peritonitis) or multiple irrigation with antiseptic solutions with their aspiration (in diffuse peritonitis) and further adequate drainage of the abdominal cavity, depending on the distribution of peritonitis (drainage with one, two, three or four sleeve tubes). In generalized (diffuse) purulent peritonitis, a thorough lavage of the abdominal cavity is made by antiseptic solutions, intubation of the small intestine and drainage of the abdominal cavity according to Petrov. In the postoperative period, peritoneal dialysis is used.

As for ***radical operations*** indicated in the first stage of the disease in the absence of diffuse peritonitis, young and middle age patients without severe concomitant diseases most frequently undergo *organ-preserving operation of PU excision with pyloroplasty according to Judd-Horsley* (Figure 36), *in combination with selective or stem vagotomy*, Also, a thorough rehabilitation of the abdominal cavity and adequate drainage are required.

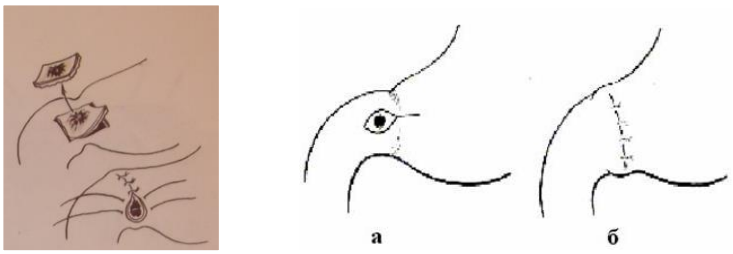


Fig. 36. Excision of ulcer with plastic repair according to Judd-Horsley:
a) excision of ulcer; b) pyloroplasty

Indications ***for primary resection of the stomach in PU*** are as follows: a short period (no more than 6 hours) from the moment of perforation, absence of signs of generalized peritonitis, prolonged course of peptic ulcer with stenosis of the pylorus, ulceration of the stomach, condition of the patient, allowing this traumatic intervention and high qualification of the surgeon. At present, primary resection of the stomach is mainly performed only in perforation of gastric ulcer, as well as in suspected malignancy of the ulcer.

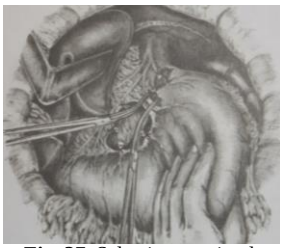


Fig. 37. Selective proximal vagotomy (Holly)

The operation of choice is the excision of perforation opening with gastro- or duodenoplasty and selective proximal vagotomy (SPV, Figure 37). PU is excised in the limits of healthy tissues with suturing of the defect with two-row sutures. The first row of sutures is superimposed through all layers with nodes inside, and the second is serous-muscle. After this, a thorough rehabilitation of the abdominal cavity is performed and SPV or other type of vagotomy (selective or stem) is carried out, depending on the severity of the patient's condition, his age and concomitant pathology, when the speediest end of surgery is of crucial importance.

It is necessary to finish the operation sleeve tube drainage

drawn out through a separate counter puncture in the right hypochondrium, according to indications (in diffuse peritonitis) and in the right ileac region.

Treatment measures ***in the postoperative period***, in the first place, should be aimed at combating peritonitis and prevention of purulent-inflammatory complications: antibiotic therapy, detoxification therapy (forced diuresis), correction of protein and electrolyte balance, etc.

II.3.3. ACUTE CHOLECYSTITIS AND ITS COMPLICATIONS

Classification. Classifications of acute cholecystitis are quite numerous. In practice, the classification of **B. A. Korolev, D. L. Pikovsky** (1971) is used.

I. *Acute cholecystitis:*

1. Catarrhal. 2. Flegmonous. 3. Gangrenous. 4. Perforated.

II. *Acute obstructive cholecystitis.*

III. *Acute cholecystopancreatitis.*

V. T. Zaitsev's (1979) classification is used in clinical practice.

Cholelithiasis

Acute form	***Chronic form***
1st stage (cholecystolithiasis)	
Acute cholecystitis:	Chronic cholecystitis:
Cataarrhal	*Primary chronic*
Flegmonous	*Recurrent*
Gangrenous	*Complicaated (dropsy, empyema,*
Perforated	*sclerosis)*
2nd stage (cholecysto- choledocholithiasis, stenosing papillitis)	
Acute cholecystitis with obstructive jaundice	Choledocholithiasis with obstructive jaundice
Acute cholangitis with obstructive jaundice	Chronic sclerosing cholangitis with obstructive jaundice
3rd stage (cholecysto-pancreatitis, cholecysto-hepatitis, biliodigestive fistulas, etc)	
Acute cholecystopancreatitis	Chronic cholecystopancreatitis
Cholangic abscesses of the liver	Chronic cholecystohepatitis
Acute cholecystohepatitis	Biliary cirrhosis of the liver
Acute cholangiohepatitis	Biliodidgestive fistulas

Presentation. Acute cholecystitis more often begins as an exacerbation of chronic, sometimes against the background of false well-being.

Hepatic colic occurs suddenly, more often at night, manifested by sharp cramping pains in the right hypochondrium, radiating to the right shoulder, shoulder blade, waist, right side of the neck. The pain is accompanied by nausea, vomiting, which does not bring relief. The pain is persistent (up to two weeks). The intensity of pain over time is reduced, it becomes constant, with a feeling of heaviness in the epigastric region, chills, fever to 38°C or higher, weakness, dryness, bitterness in the mouth. An attack of cholecystitis is provoked, as a rule, by a fault in the diet, physical or emotional overloading.

Careful study of the history of the disease is of paramount importance for proper diagnosis. The history should include data on the onset of the disease, the main symptoms of the course of the disease and the treatment at prehospital stage. For the correct assessment of the patient's condition, it is necessary to identify previous and concomitant diseases. It is important to study gynecological history in women. Many patients have family history of the disease. There can be possible indications of previous jaundice, sometimes recurrent, or previously detected stones in the gallbladder.

On examination. Restless behavior of the patient is typical for hepatic colic; pallor of the skin, suffering face, yellow skin and sclera in complicated forms of cholelithiasis.

Local status. ***Examination of the abdomen*** should involve assessment of its shape, participation in breathing, its skin and presence of bloating. *Palpation* should determine localization of pain, as a rule, it develops in the right hypochondrium, accompanied by tension of muscles. Extensive muscle tension develops on lated stages due to progression of the disease and the development of peritonitis. *Percussion* identifies drum belly. Diffuse muscle tension and dullness in flattened

abdominal areas occur later in connection with the progression of the disease and the development of peritonitis. *Auscultation* shows intensified peristaltic noises at the beginning of the disease, but in progression of the inflammatory process and development of destructive changes in the wall of the gallbladder, peristaltic noises are significantly reduced.

Kerr's, Zakharyin's, Murphy's, Mussi-Georgievsky's, Lyakhovitsky's, Grekov-Ortner's and Blumberg's signs are *pathognomonic signs* typical for acute cholecystitis.

In accordance with standard schemes, **laboratory-instrumental examination plan and possible pathological changes** diagnostic measures include exporative imaging of the abdominal organs (intestinal pneumatosis); clinical blood assay (increased ESR, neutrophilic leukocytosis with a shift to the left; leukopenia with lymphocytopenia and neutrophilosis with a shift to the left is possible secondary to intoxication); clinical urine analysis; biochemical blood test for the content of the total protein (usually normal with a decrease in alpha/globulin coefficient), total bilirubin (more commonly normal) and its fractions, alkaline phosphatase, amylase (increases in involvement of the pancreas), transaminases (more commonly normal). The findings are considered in combination with clinical data.

Ultrasound examination is the main method used to identify the nature of the pathological process in the biliary tract, as it specifies the size of the gallbladder, the thickness of its wall, the presence of stones (Figure 38.). As a rule, all this is enough to establish the diagnosis and determine the further surgical approach.

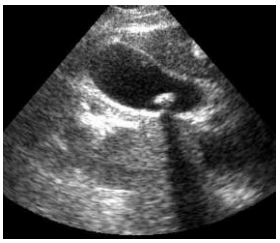


Fig. 38. US examination: stones

It should be differentiated from exacerbation of peptic ulcer, perforated ulcer of the duodenum, acute pancreatitis, acute intestinal obstruction, acute appendicitis, right-sided renal colic and pleuropneumonia.

Treatment. Petrov's therapeutic approach is used, nnamely trial conservative therapy for 48 hours in absence of peritonitis. *Indications for urgent surgical intervention are*:

1) absence of a positive effect from trial conservative therapy;

2) acute cholecystitis with signs of peritonitis;

3) acute cholecystitis, complicated by cholangitis, pancreatitis, obstructive jaundice, hepatic insufficiency;

4) destructive form of the inflammatory process of the gallbladder;

5) progression of complications.

Surgery on the biliary tract involves endotracheal anesthesia with muscle relaxants.

The operation of choice is laparoscopic cholecystectomy (LCE). LCE consists of three stages:

1) operational access (insertion of tools into the abdominal cavity);

2) imposition of pneumoperitoneum, direct separation of the gallbladder and adhesions or infiltrate, identification, clipping (Figure 39) and intersection of the gallbladder and the cystic artery, separation of the gallbladder from the liver;

3) removal of the gallbladder from the abdominal cavity.

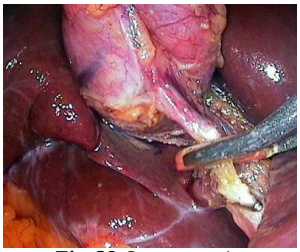


Fig. 39. Laparoscopic cholecystectomy: clipping bubble duct

Operation should end according to the operating situation. If destruction of the gallbladder is accompanied by signs of pericholecystitis and a large amount of effusion, drainage should be carried out from three points (subdiaphragmatic space, subhepatic, right ileac region). In wide choledochus and obstructive jaundice the operation should be completed by drainage of choledochus according to Halsted laparoscopically.

In patients with complicated forms of cholelithiasis, the first stage of treatment is endoscopic papillosphincterotomy, which eliminates stenosing papillitis, choledocholithiasis, jaundice, cholangitis.

Postoperative period. Therapeutic measures in the first three

days after the operation are aimed at supporting hemodynamics, adequate ventilation of the lungs, protein supplementation, pain reduction, combatting microflora, correction of water-electrolyte balance, anti-enzyme therapy. In short, the same intense conservative therapy is performed, as before surgery. It is mandatory to aadminister anticoagulants, especially in the elderly. Removal of drainage is carried out in 1-3 days, stitches are removed in 6-7 days after surgery. Peristalsis is restored in a day or two.

There is the following ***classification of LCE complications*** in clinical practice.

I. *Intraoperative:* emphysema of the anterior abdominal wall, omentum; bleeding from the arteries of the gallbladder, its bed, omentum; damage to the wall of the gallbladder; damage to the biliary ducts; damage to the diaphragm.

II. *Early postoperative complications (3-4 days after surgery):* leakage of bile (bed, stump of the duct, damage to the duct); bleeding from the bed.

III. *Late postoperative complications (5-10 days):*

1) external biliary fistula;
2) posttraumatic stricture of choledochus;
3) residual choledocholithiasis;
4) prolonged intestinal paresis;
5) suppuration or infiltration around the wound;
6) symptoms of complicated carboxyperitoneum;
7) postoperative hernia.

II.3.4. ACUTE PANCREATITIS AND ITS COMPLICATIONS

Acute pancreatitis (AP) *is an enzymatic lesion of the pancreas (P), which is an aseptic inflammation of the demarcation type, based on necrobiosis of pancreatocytes and enzymatic autoaggression followed by necrosis accompanied by secondary purulent infection.*

This process is autocatalytic and often ends with autolysis and necrosis of the organ. The incidence of AP in Ukraine is 102 per 100 thousand of population. Mortality in AP is 7-15% total, 30-40% in destructive forms.

Classification of acute pancreatitis (O. O. Shalimov, 1990).

1. *By morphological changes:*

- edematous pancreatitis: (serous, serous-hemorrhagic);
- necrotic pancreatitis (pancreatic necrosis) - hemorrhagic, fatty, mixed (each of them - small-focal, large-focal, subtotal, total);

Purulent pancreatitis: Primary purulent, secondary purulent and exacerbation of chronic purulent pancreatitis.

2. *By severity:*

- mild;
- moderate;
- severe;
- extremely severe.

3. *By presentation:*

- regressive;
- progressive;
- recurrent.

4. *By complications:*

- local complications;
- intra-abdominal complications;
- extra-abdominal complications.

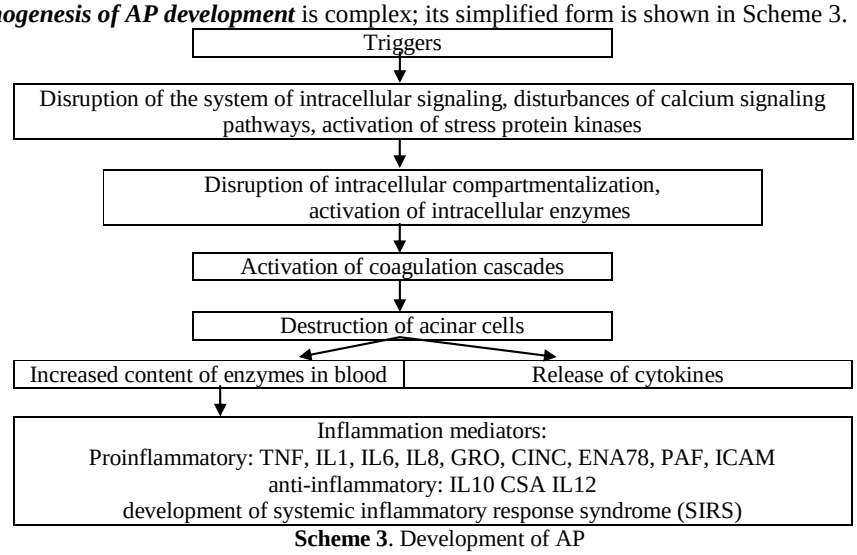
At present, classification of AP proposed by ***H.G. Beger*** (1991) is used all over the world, which was adopted by the participants of the ***international symposium on AP in Atlanta*** (1992):

1. Interstitial (edematous) pancreatitis.
2. Pancreonecrosis:

- non-infected;
- infected.

3. Abscess of pancreas
4. False cyst of the pancreas.

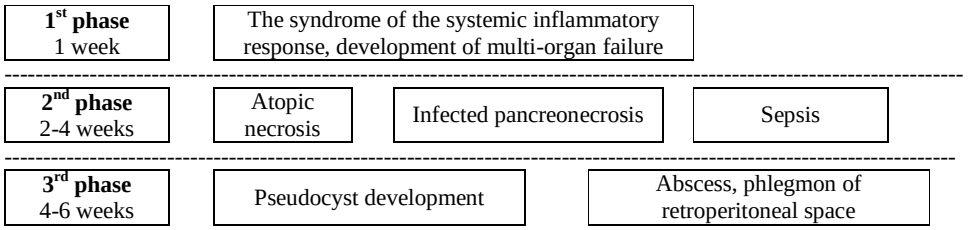
Pathogenesis of AP development is complex; its simplified form is shown in Scheme 3.



Scheme 3. Development of AP

The course of severe AP is divided into three phases, taking into account the time elapsed since the onset of the attack (Scheme 4):

Presentation. Patients *present with* intense pain in the epigastric region, in the left or right hypochondrium, sometimes stabbing, radiating to the back, lumbar region, with nausea, repeated vomiting (in severe cases multiple, which does not bring relief), bloating.



Scheme 4. Phases of AP development

The *history* suggests association of the disease with faults in the diet (fatty, fried, spicy food), gallstone disease, alcohol intake, trauma in the area of the pancreas.

Objective clinical manifestations depend on the severity of the course and duration of the disease:

1) general severe condition, suffering face, patient is moaning due to pain;

2) pallor of the skin, in severe course there are positive *"skin" Cullen's* (jaundice of the skin in the area of navel), *Halsted's* (cyanosis of the abdomen), *Grey-Turner's* (cyanosis of the lateral abdominal wall), *Mondor's* (purple spots on the skin of the face and trunk) signs; sometimes yellow skin and sclera;

3) tachypnea;

4) tachycardia.

Local status. The tongue is dry, covered with white plaque. The abdomen is bloated, and at the beginning of the disease only in its upper part, Gobiet's sign (due to paresis of the transverse colon), and then the whole evenly; at the beginning of the disease the abdomen is soft, painful in the epigastrium, left and right hypochondria without signs of peritoneal irritation. ***Pathognomonic***

Korte's, Voskresensky's, Koch's, Mayo-Robson's signs are positive. On percussion: belly drum in the upper abdomen, on auscultation: wak intestinal peristalsis.

In progression of the disease there is an increase in intoxication with impairment of hemodynamics (tachycardia, hypotension), abdominal muscle tension and positive signs of peritoneal irritation (*Blumberg's, Kulenkampf's signs*), dull percussion sound in the flat abdominal areas due to the development of peritonitis, possible development of reactive left-sided exudative pleurisy. In severe course, mental state may be disturbed due to intoxication (anxiety, psychosis, delirium).

In accordance with the standard schemes, a ***plan for a laboratory-instrumental examination*** of *patients with AP in basic diagnosis of severe AP includes*:

1) measurement of blood pressure, heart rate, temperature and diuresis in 1-4 hours;

2) explorative radioscopy of the chest and abdominal cavity (possible development of reactive left-sided exudative pleurisy, intestinal pneumatosis);

3) ultrasound of the abdominal organs (possible presence of stones in the gallbladder, thickening of its walls, enlargement of the pancreas and decrease in its echogenic density due to edema);

4) clinical and biochemical blood tests, determination of blood serum electrolytes, activity of liver enzymes and enzymes of the pancreas (amylase increase).

Laparoscopy (Figure 40) and laparocentesis (a study of parietal fluid for the presence of pancreatic enzymes) are also used. In a doubtful case, when diagnosing AP, a widespread use of CT with contrast enhancement is required.

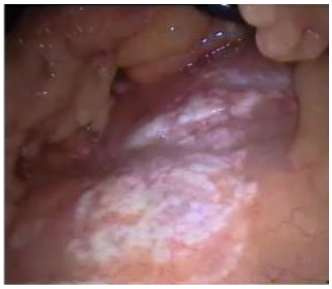


Fig. 40. Laparoscopy: steatonecrotic plaques on the visceral peritoneum in acute pancreatitis

Diagnostic criteria for severe AP:

1) presentation;
2) increased levels of serum amylase in blood or urine;
3) temperature higher than 38°C;
4) heart rate more than 90 beats / min;
5) leukocytosis more than 12×10^9 / l or less than 4×10^9 / l;
6) RR greater than 20 / min or PaO_2 less than 4.3 kPa;
7) ultrasound findings; in doubtful cases, CT with a contrast enhancement is performed.

Table 16

Prognostic criteria for AP severity according to Ranson scale

Prognostic criteria	
On admission	***In 48 hours after admission***
Age> 55 years Blood glucose> 11 mmol / L Leucocytosis> 16000 mm^3 LDH> 400 IU / l AST> 250 IU / L	A decrease in hematocrit level by more than 10% Plasma calcium <2 mmol / l. Baseline deficiency> 5 mEq / l An increase in urea> 1.8 mmol / l RaO2 <60 mm Hg BE> 4 mmol / l Liquid deficit> 6 liters
Prognosis according to Ranson scale	
Number of points	***Mortality, %***
0-2	<1
2-3	15
3-4	40
>6	100

Glasgow criteria (Imrie), simplified Ranson criteria: Ht> 47%; albumin <32 g / l; serum nitrogen> 10 mmol / l; calcium <2 mmol / l; leukocytosis> 16 × 109 / l; lactate dehydrogenase> 600 mmol / l; AsAT, AlAT> 100 micrograms; sugar> 10 mmol / l; blood urea> 16 mmol / l; the presence of three or more criteria within 48 hours after the onset of an attack indicates severe pancreatitis.

APACHE II (1985) and SARS (1993) scales are also used for an objective assessment of AP severity.

The level of C-reactive protein is also an important prognostic factor: its level in plasma above 150 mg / l indicates the presence of severe pancreatitis.

In 1990, the modified system of the CT-index of AP severity (CTSI) was introduced, which combines measurements of the volume of necrosis of the pancreas and the original CT-system (includes the calculation of peri-pancreatic inflammation and fluid-containing formations).

Patients with biliary pancreatitis should undergo ultrasound and endoscopic retrograde cholangiopancreatography (ERCP) for clarification of diagnosis.

AP should be differentiated from perforated ulcer of the stomach and duodenum, acute intestinal obstruction, destructive cholecystitis, AA, mesenteric blood vessel thrombosis, myocardial infarction, food intoxication, etc. By way of logical reasoning it is necessary to identify general signs for these diseases and AP, and signs excluding data of the disease, but patognomonic for AP.

Treatment of acute pancreatitis. *Choice of therapeutic approach. Interstitial, hemorrhagic (without signs of peritonitis) pancreatitis and non-life-threatening conditions in limited pancreonecrosis, involve conservative treatment:*

1) inhibitors of a proton pump (PPI) - counterlock, etc.;
2) statins - somatostatin (sandostatin);
3) the use of antiproteases - contrykal, proxylan, etc.;
4) NSAIDs - paracetamol, xefocam, ketanov, etc.;
5) antibiotic therapy - tienam, meronem, fluoroquinolones (avelox, loxof, cifran);
6) detoxification therapy.

Patients with *severe pancreatitis* should undergo treatment in a resuscitation unit. They must be administered:

1) monitoring of blood pressure and CVP;
2) mechanical ventilation (if necessary);
3) therapy with inotropic agents to support cardiac output;
4) adequate infusion therapy;
5) hemofiltration or hemodialysis in acute renal failure;
6) enteral probe nutrition, possibly supplemented by parenteral administration of therapeutic agents.

Infected pancreatic necrosis should be managed **surgically** by removal of necrotized pancreatic tissue.

Indications: infected pancreonecrosis; severe sterile pancreatic necrosis with no positive time course within 2 weeks after the start of adequate treatment.

Methods of surgical treatment of pancreonecrosis:

- open necrectomy with drainage of the sac of omentum;
- programmable relaparotomy or laparostomy – keeping abdominal cavity open; minimally invasive necrectomy with videoscopic retroperitoneal access; laparoscopic drainage of the abdominal cavity (flank and sac of omentum).

Long-term effects of pancreonecrosis: pseudocysts; exocrine insufficiency; diabetes; bile duct strictures; development of chronic pancreatitis (fibrosis, calcinosis, polycystic ovary).

Complications of AP: 1. Peripancreatic infiltration. 2. Purulent parapancreatitis. 3. Enzymatic (abacterial) peritonitis. 4. Purulent peritonitis. 5. Abscess of the sac of omentum. 6. Abscesses of the abdominal cavity. 7. Phlegmon of retroperitoneal cellular tissue. 8. Pseudo-cysts. 9. Obstructive jaundice. 10. Internal and external fistulas. 11. Gastrointestinal stenosis. 12. Thoracic purulent complications. 13. Purulent mediastinitis. 14. Sepsis. 15. Erosive bleeding.

II.3.5. STRANGULATED HERNIA

The most frequent and severe complication of abdominal hernia is its ***strangulation***. Strangulated hernia (SH) rank 4th (4.3-4.8%) after acute appendicitis, acute cholecystitis and acute pancreatitis. By the number of operated patients, strangulated hernias also rank 4th. Postoperative mortality remains high (7.2-12%) and in recent years there has been no downward trend.

Classification of hernias.

By etiology: congenital and acquired.

By anatomical features: inguinal, femoral, umbilical, abdominal white line, spigelian line, xiphoid, lumbar, gluteal, perineal, diaphragmatic, in the internal pockets, sliding hernia of the cecum and bladder (Figure 41).

By presentation: free or reducible (*reponibilis*), non-reducible (*irreponibilis*), strangulated (*incarserato*).

By the type of strangulation: elastic, fecal, retrograde (double loop hernia, Fig. 42), parietal (Richter's hernia), Littre's hernia (strangulation in hernia sac of Meckel's diverticulum).

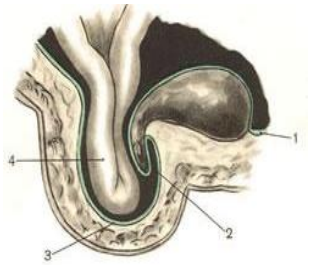


Fig. 41. Sliding hernia of the bladder: 1 – peritoneum; 2 – bladder; 3 – hernia sac; 4 – small intestine

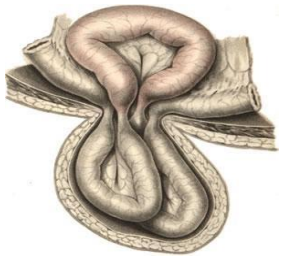


Fig. 42. Retrograde strangulation: W-shaped location of the loop of the small intestine in the hernia sac and abdominal cavity

Clinical symptoms. *The patients present with* acute sudden pain in the area of hernial protrusion, which appears most often at the moment of physical stress (running, lifting of heavy things, jumping, defecation), which does not subside. Since the onset of pain, the protrusion does not disappear horizontally (as it was before). History suggests that the patient suffers from a hernia.

Local status. Hernial protrusion does not disappear and does not change outlines when changing the patient's position. *On palpation* it is sharply painful, especially in the area of hernial orifices and tense. *Cough push sign* is negative. On percussion marked *drum belly* in the early stage, *dull percussion sound* in the late stage (due to inflammatory exudate). In strangulation of the small intestine there are signs of *high intestinal obstruction*, and in strangulation of the colon of *low intestinal obstruction*. Therefore, a strangulated hernia is often accompanied by vomiting (does not bring relief), bloating, retention of stool and gases. The involvement of the bladder in sliding inguinal hernia is accompanied by an accelerated and painful urination.

There are ***three periods*** of presentation in strangulation of intestine:

1) less than 2 hours after strangulation, absence of necrosis of strangulated intestinal loops and intoxication signs;

2) 2 to 8 hours after strangulation, necrosis of the intestinal loop, signs of intestinal obstruction, but without peritonitis;

3) 9 hours and more, peritonitis and intoxication.

The assistance provided to the patient at the ***pre-hospital stage*** should be reduced to a faster delivery of the patient to a surgical hospital. It is strictly forbidden to forcibly correct hernial protrusion, which can provoke hemorrhages into the intestine and mesentery, thrombosis of vessels, rupture of mesentery, perforation of the intestine. In addition, ***false*** repositioning of hernia may occur. In ***anapproved repositioning*** of the strangulated hernia at the preoperative stage, it is necessary to adhere to ***expectant approach***: mandatory hospitalization, supervision (possible development of symptoms of peritonitis in repositioned non-viable intestine), according to indications, control of the passage of aqueous suspension of barium sulphate. However, in the presence of abdominal pain, tachycardia, high leukocytosis, vomiting, history data suggestive of prolonged restriction or the presence of peritoneal signs the patient must be ***urgently operated*** on due to life-threatening condition, using a median incision. If the repositioning occurred during anesthesia, incision, or at other stages of the operation, it is desirable to detect and inspect the affected organ through the herniolaparotomatous wound.

The standard volume of examination recommended for patients with this disorder includes the following:

1) clinical blood test;
2) clinical urinanalysis;
3) coagulogram;
4) determination of blood type and blood Rh factor;
5) exploration radioscopy of the chest and abdominal cavity.

If the patient is over 45 years old or there are history data in favor of concomitant pathology (most often cardiovascular disorders), then it is necessary to make ECG, to consult the adjacent specialists (general practitioner, neuropathologist, etc.).

Differential diagnosis. Oblique inguinal hernias that do not descend into the scrotum should be differentiated with specific (tuberculosis, actinomycosis) and nonspecific lymphadenitis, syphilitic gumma, tumor metastasis (of the rectum, uterus, prostate gland), lymphogranulomatosis.

Oblique inguinal hernias that descend into the scrotum should be differentiated from *dropsy of testicles*, as well as from *dropsy of the spermatic cord*. In dropsy, on palpation of the spermatic cord near the outer opening of the inguinal canal, it is possible to freely join the fingers near its upper pole, feeling the seminal duct between them, whereas fingers can not be joined in inguinoscrotal hernia. On percussion in SH: drum belly, in dropsy of testicles: dullness. Differential diagnosis is also carried out by the method of diaphanoscopy.

The difference between dropsy of the spermatic cord from dropsy of the testicles is that the in the former disorder dropsical cavity empties at night and fills again when the patient receives a vertical position.

The same pattern can be in *orchitis and epididymitis.* However, acute epididymitis begins with severe pain in the testicle area; the pain radiates along the spermatic cord and is accompanied by chills, fever. In this case, there are no signs of intestinal obstruction, although in high temperature there may be vomiting.

In addition, inguinal hernias should be differentiated from the *dilation of the veins of the spermatic cord (varicocele)*, which is predominantly to the left, where the spermatic vein enters the renal vein at the right angle. Examination shows nodes of varicose veins interwoven with each other along the spermatic cord.

Femoral hernias should be differentiated from *enlarged lymph nodes* in various diseases, *metastases of malignant tumors* in the lymph nodes of this region, *benign tumors* of this region, *varicose veins of the lower extremities*, *specific edematous abscesses, cysts* located under the inguinal bundle.

Strangulated umbilical hernia should be differentiated from *non-reducible umbilical hernia*, *inflammation of the umbilicus, metastasis of the tumor of the stomach in the navel.*

Treatment of SH. *Strangulated hernia is a disorder* requiring urgent surgery as a life-threatening condition. *The operation is performed not later than two hours after hospitalization, including preoperative preparation.*

Preoperative preparation is necessary for patients admitted in a severe condition due to the existing concomitant pathology or aadvanced strangulation. It should be as short as possible (not to exceed 2 hours) and should be aimed at correcting the shifts in the water-electrolyte and acid-base balance of the patient's body in the treatment of concomitant pathology. It should be noted that this preparation does not save the life of the patient, and only urgent surgical removal of strangulation, as the cause of pathological changes, allows to cure the patient.

Operative treatment is most often performed under local infiltration anesthesia according to O. O. Vishnevsky with 0.25% solution of novocaine (after a tolerance test). If necessary, laparotomy requires a combined balanced anesthesia with mechanical ventilation. Operative treatment for strangulated hernia is composed of several stages:

1) a projection incision of the skin, subcutaneous tissue, surface fascia over hernial protrusion;
2) separation of the hernial sac;
3) dissection of the hernial sac with the fixation of strangulated intestinal loops with a ahnd;

4) dissection of the strangulating ring, examination of the contents of the hernial sac with a careful assessment of the viability of the strangulated intestinal loops or other organs (omentum, bladder, appendages of the uterus) and their repositioning to the abdominal cavity making sure they are viable;

5) suturing and dressing of the neck of the hernial sac and cutting off its peripheral part;

6) plastic repair of the inguinal canal.

There are the following types of ***plastic repair of the inguinal canal:***

1) facsial-aponeurotic (Girard-Spasokukotsky's);

2) muscular-aponeurotic (Kimbarovsky's);

3) with the help of additional or synthetic materials (alloplasty);

4) combined (own and other person's tissues).

It is impossible, without dissecting the hernial sac, to cut the strangulating ring, as the contents slip into the abdominal cavity and can become the cause of peritonitis in protruding nonviable intestine. If there are several loops in the hernial sac, they should be removed for revision to exclude retrograde strangulation.

Signs of viability of the intestine: restoration of normal pink color, absence of strangulation furrows and dark spots that are seen through the serous membrane; preservation of pulse in mesenteric vessels; presence of peristalsis. ***Signs of non-viability*** are: dark color; dim serous membrane; absence of peristalsis; absence of pulse in mesenteric vessels; flaccid, infiltrated wall. Their presence is an indication for laparotomy. The volume of resection of the intestine is as follows: from the apparent margin of necrosis not less than 40-50 cm of the deferent part of intestine and 20-30 cm of the abducting part of intestine.

In ***parietal strangulation*** it is not necessary to resort to wedge-shaped resection of the strangulated area, and it is necessary to perform resection of the intestinal tube, retreating 10 cm in both sides of the border of the strangulation and necrosis.

The following signs help to *avoid wounding the bladder* in strangulation of sliding hernia: the proximity of the cystic tissue, the fleshyness of the wall, the trabecular structure of the muscular membrane, diffuse bleeding in its cutting, the urge to urinate when the urine bladder stretches, the lack of enlargement of the formation in the patient's tension. In accidental damaging of the walls of the bladder, it is sutured with two-row stitches without involvement of the mucous membrane.

Postoperative complications in patients with SH are the same as in patients with acute intestinal obstruction: peritonitis, intestinal necrosis, failure of stitches on the site of intestinal anastomoses, interintestinal abscess, postoperative obstruction, eventration, pulmonary artery thromboembolism, pneumonia, myocardial infarction, etc.

In order to prevent the development of infectious complications, antibacterial therapy is required. It is necessary to carefully protect tissues from unnecessary traumatism, to achieve a minimum tension in plastic hernial orificess, to conduct thorough hemostasis.

The primary tasks of the postoperative period are as follows: a) combatting intoxication and dehydration; b) prevention of infectious complications; c) restoration of gastrointestinal tract functions; d) prevention and treatment of complications of the respiratory and cardiovascular systems.

Laparoscopic hernioplasty with dissection of hernia using a posterior approach with laparoscopy is almost always associated with the implantation of a synthetic net prosthesis. The approach may be transabdominal and extraabdominal. Important disadvantages of laparoscopic operations include their high cost, the need for general anesthesia and expensive equipment.

II.3.6. ACUTE INTESTINAL OBSTRUCTION

Classification of acute intestinal obstruction (AIO) (O. O. Shalimov, 1977).

I. *According to the mechanism of development*.

1. *Dynamic* (functional): a) spastic; b) paralytic.

2. Mechanical:

a) strangulation (volvulus, node formation, internal strangulation);

b) obturative (obturation by a tumor, blockage by a foreign body, fecal or biliary stone, ascaritic ball, coprostasis);

c) mixed forms of strangulation and obstruction (invagination, adhesion obstruction).

3. *Vascular* (intestinal infarction):

a) mesenteric vein thrombosis;

b) thrombosis and embolism of arteries (currently isolated in a separate nosological form - acute violation of mesenteric circulation).

II *By origin*: congenital and acquired.

III *By presentation*: acute and chronic.

IV. *By level*: high and low, small and large intestine.

V. *By character*: partial and complete.

VI. *By periods* of AIO (from the moment of the disease):

a) early (from 2 to 12 hours);

b) intermediate (12-36 hours);

c) late (after 36 hours).

VII. *According to AIO stages*:

a) neuro-reflex;

b) compensation and organic changes;

c) terminal

Clinical manifestations. The patients *present with* abdominal pain - with or without clear localization, cramping or permanent, periodically increased; retention of gas and stool, abdominal distension, borborygmi, nausea and vomiting (in late stages of the disease). It is possible to detect a syndrome of small signs (if AIO is caused by a tumor): general weakness, decreased ability to work, loss of appetite, weight loss for several months or a year. In history: presence of similar attacks in the past, previous surgical intervention and injuries of the abdominal cavity.

The general condition of the patient, as a rule, is from moderate to extremely severe, with possible cyanosis or pallor of the skin.

Local status. The examination of the abdomen should be carried out strictly in the patient's horizontal position. It should begin with an examination of the typical places of the hernial orifices to exclude the presence of a strangulated hernia. An important diagnostic feature is the degree of abdominal distension: as a rule, lower level of AIO is associated with more bloating and later development of vomiting. It is necessary to pay attention to the configuration of the abdomen, its asymmetry (local bloating, *Val's sign* with drum belly over it on percussion – *Kivul's sign*), the presence of visible peristalsis of the intestine (Shlange's sign).

Palpation should identify the localization of the greatest pain; possible detection of induration (invaginate, tumor, foreign body, etc.); from the 2nd stage of the disease the *"noise of a splash" (Sklyarov's sign)* can be determined; possible tension of the muscles of the anterior abdominal wall and a positive symptom of irritation of the peritoneum (*Blumberg's sign*), dullness of the sloping areas of the abdomen with the development of peritonitis. Compulsory ***finger examination*** of the rectum shows reduction of the tone of the external sphincter of the rectum, its empty and bloated ampulla *(Obukhov hospital sign), Kulenkampf's sign*. Rectal and vaginal bimanual examination reveals pathological formations (tumors, cysts, invaginates), which are the cause of AIO. On auscultation, attention should be paid to enhancing intestinal motility during the onset of cramping pain, the presence of intestinal noises and, very often, heart tones on the abdominal wall (*Bailey's sign*). Percussion of the abdomen can determine high drum belly in the circumference of the navel, dillness in the localization of the invaginate or tumor, as well as dullness in the flat abdominal areas due to accumulation of effusion (with the development of peritonitis); absence of peristalsis in the stage of diffuse peritonitis, *Lotheissen's sign of "grave silence"*.

In order to clarify the preliminary diagnosis of AIO in accordance with the standard schemes, the ***plan of laboratory and instrumental examination includes and identifies possible pathological changes***:

1) *laboratory tests:* complete blood count and urinalysis, hematocrit, acid-alkaline state, electrolytes, sugar, blood biochemistry, coagulogram, characterized by high leukocytosis and blood condensation (due to hypovolemic link of pathogenesis), protein reduction;

2) *finger examination of the rectum*: detection of Obukhov hospital sign, Kulenkampf's sign, tumors;

3) *ECG and GP consultation*: possible concomitant cardiovascular diseases;

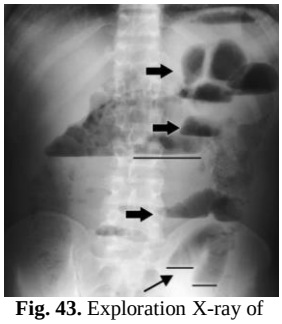


Fig. 43. Exploration X-ray of the abdomen: acute intestinal obstruction, Kloiber's cup sign

4) *X-ray examination*: exploration radiography of the abdominal cavity (if necessary in time course) shows presence of liquid levels with gas above them, *Kloiber's cup* (Figure 43), the location and shape of which can help to determine the level of obstruction, the study is conducted in the upright or in the lateral position of the patient; in some cases, X-rays are conducted with the oral administration of barium or water-soluble contrast agents; in suspected low intestinal obstruction: a study with contrast enema;

5) laparoscopy in case of suspected thrombosis of mesenteric vessels.

AIO should be differentiated from the following acute diseases: *acute appendicitis, perforated gastric or duodenal ulcer, acute cholecystitis, acute pancreatitis, torsion of an ovarian cyst, ectopic pregnancy, thromboembolism of mesenteric vessels, renal colic, abdominal form of myocardial infarction, food toxicoinfection.*

Treatment. When *choosing a medical approach*, it is necessary to remember that "the more the patient lives before surgery, the less he lives after surgery".

In case of functional intestinal obstruction, conservative therapy should be performed in a surgical hospital, which, as a rule, has a positive effect.

In *diagnosed mechanical obstruction* (but only in the absence of symptoms of peritonitis and hemodynamic disorders, that is, in the first stage of AIO) it is possible to conduct ***"trial" conservative therapy*** for up to three hours, aimed at eliminating obstruction, detoxification and correction of impaired homeostasis. It includes:

1) carrying out of an outflow from the stomach;

2) one of the most important measures is correctly performed siphon enema, the effectiveness of which is indicated by the cessation of cramping abdominal pains, reduction of its bloating, abundant release of gases and feces;

3) bilateral para-nephral novocaine blockade (if novocaine is tolerated);

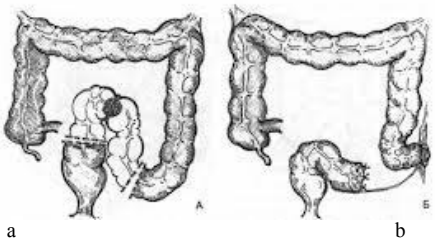
4) administration of IM proserin and IV hypertonic sodium chloride solution for stimulation of intestinal peristalsis;

5) conducting infusion detoxification and correction therapy;

6) symptomatic therapy.

It is allowed to repeat siphon enema (in its initial ineffectiveness) and administer proserin.

An urgent surgical intervention is performed for life-threatening condition in the absence of the effect of conservative therapy (clinical manifestations persist or increase).



a b

Fig. 44. Scheme of obstructive resection of the sigmoid colon according to Hartmann: a - the limits of resection; b - withdrawal of sigmoid stump in the form of colostomy

If the diagnosis is unclear, the doctor for 2 hours conducts differential diagnosis and “trial” conservative therapy. In the absence of a positive effect operative treatment is shown, and therapy is considered as preoperative preparation.

The operation is performed under endotracheal anesthesia by medial laparotomy. It should be noted that the scope of surgical intervention is determined by the revealed pathology, viability of the intestine, the severity of the patient's condition.

Variants of operations:

1) dissociation of adhesions causing obstruction;

2) disinvagination;

3) removal of the volvulus and nodular formation;

4) AIO caused by a tumor is managed by a two-moment radical operation in tumor operability (Hartmann's surgery for sigmoid cancer, Figure 44), right or left-sided hemicolectomy), in case of inoperability, palliative operations (one-or two-stem according to Maydle (Figure 45), colostomas proximal to tumors, bypass anastomoses, possible palliative resection of the intestine according to indications).

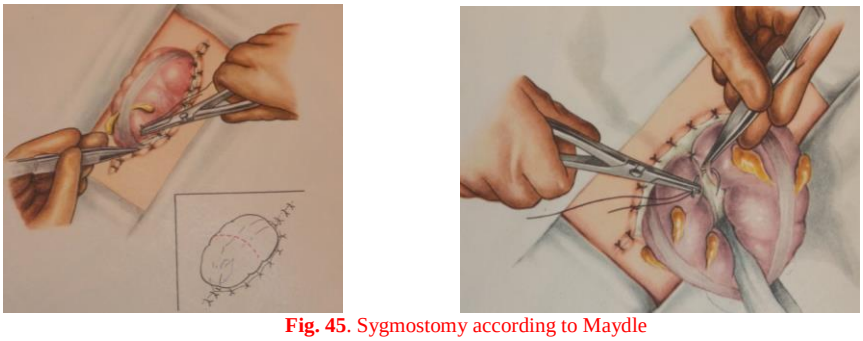


Fig. 45. Sygmostomy according to Maydle

In non-viability of the intestine, it is necessary to resect it within the limits of healthy tissues within 45-50 cm in the deferent direction and up to 30 cm in the abducting direction. It is necessary to provide nasogastric intubation of the intestine. Decompression of the large intestine is carried out transanally. Anastomosis may hide behind the peritoneum. The number of drains of the abdominal cavity depends on the severity of peritoneal events.

In the postoperative period: detoxification, antibacterial therapy, correction of water-electrolyte disbalance and acid-alkaline state, protein metabolism, prevention of complications in the respiratory and cardiovascular systems.

Separate forms of mechanical intestinal obstruction

Volvulus of the intestine is a distortion of some of its segment of 180-360 degrees or more, along with mesentery, around the axis, which goes from the base of the mesentery to the length of the corresponding area of the intestine. The incidence of volvulus of the small intestine is about 5%. Volvulus of the sigmoid colon is also common. In the early stages of the disease, the operation involves straightening of the volvulus. Apparent necrosis of the intestine requires resection of the intestine.

Nodular formations of the intestine: twisting of one loop of the intestine around the other with compression of their mesentery and a violation of blood circulation in both loops. The most crucial stage of the operation is the unbundling of a node, which is easy to perform only in the first hours of the disease. If the nodes do not resolve, it is necessary to perform a resection.

Invagination is penetration of the intestine section into the lumen of the adjacent area of the intestine. The incidence is about 15%. Therapeutic approach involves disinvagination by milking back. Non-viable intestine requires resection of the intestine with end-to-end anastomosis.

Adhesive disease is a disease that develops as a result of the adhesive process in the abdominal cavity after surgical interventions. Therapeutic approach involves separation of adhesions. Non-viable intestine requires its resection.

Internal strangulated hernias also cause the onset of intestinal obstruction. The exact diagnosis before the operation is impossible, with the exception of diaphragmatic hernias. Clinical manifestion comprises presenting signs of strangulation obstruction of the corresponding area of the intestine. Treatment is surgical.

Thromboembolism of the mesenteric vessels (acute violation of mesenteric circulation) occurs mainly in the elderly and senile, has much in common with mechanical intestinal obstruction. Immediately there is a lot of pain and vomiting, retention of stool and gases, bloating, expressed signs of intoxication. However, unlike mechanical intestinal obstruction, peristalsis is absent, after an enema, and sometimes spontaneously, there is bloody diarrhea. On palpation: moderate tension in the abdominal wall muscles, and in some cases, tumor-like formation with fuzzy borders. Diagnosis is complcated. Laparoscopy shows hemorrhagic nature of the peritoneal exudate and necrotized areas of the intestine.

II.4. CLINICAL FEATURES OF THE COURSE OF ACUTE ABDOMINAL DISEASES IN PREGNANT WOMEN

Acute diseases of abdominal organs present a vast group of abdominal organs diseases. They occur acutely and are life threatening; emergency surgery is the treatment of choice for the majority of the patients. The incidence of some diseases of this group in pregnant women is alike or exceeds the one in the nonpregnant state. The most frequent are the digestive tract organs' diseases (appendicitis makes 90% of cases).

There are some clinical features of the course of such diseases in pregnant women. Except for the threat for the mother, acute diseases of abdominal organs adversely affect the gestation and may cause premature termination of the pregnancy, miscarriage and premature delivery. Perinatal mortality is potentially probable to occur. Such complications are registered in 5-7 % of women with acute appendicitis, in 50-70 % – with intestinal obstruction. In case of peritonitis, the child mortality ammounts to 90%.

In case of any acute disease of abdominal cavity organs, the prognosis for mother and fetus considerably grows worse with the increase of the stage of pregnancy and in labor, which occurs due to the increased difficulty in making a diagnosis and, consequently, surgery delay.

The group of acute diseases of the organs of the abdominal cavity includes acute appendicitis, acute cholecystitis, acute pancreatitis, acute intestinal obstruction, perforated ulcer and incarcerated hernia. Moreover, the abdominal pain syndrome may be conditioned by pregnancy complications: threatening miscarriage, ectopic pregnancy, pyelitis gravidarum, etc.

The importance of correct treatment approaches in pregnant women is conditioned by the impact on both mother and the fetus, as well as, by the risk of complications, such as miscarriage, premature termination of the pregnancy, premature delivery.

There is standard classification of acute surgical abdominal diseases in pregnant women.

Classification of ectopic pregnancy. They include salpingocyesis (tubal pregnancy) ovariocyesis, *located in the rudimentary horn of the uterus,* ***intraperitoneal pregnancy. Salpingocyesis (tubal pregnancy)*** *is subdivided into* ***ampullar, isthmic, interstitial. Ovariocyesis*** *may develop* ***on the ovary surface*** and ***in the middle of the follicle. Intraperitoneal pregnancy*** *is subdivided into* ***primary****, when the implantation occurs in the parietal peritoneum, omentum or abdominal cavity organs, and* ***secondary*** *if gestational sac is attached in the abdominal cavity, after it has been discharged from the fallopian tube.*

Clinical features of examination of pregnant women with suspected acute surgical abdominal pathology.

Questioning of the patient gives the history of problems *(**complaints***) with the abdominal cavity organs, as well as with the female genital organs. *History taking* includes not only the development of acute abdominal disease, but also includes gynecological and obstetrical history.

On physical examination: the overall health state assessment of the pregnant woman with acute surgical abdominal pathology is done (consciousness, condition and severity); the condition of the cardio-vascular, respiratory and urinary systems are controlled.

The examination of the abdominal cavity organs (the stomach, intestine, liver, spleen, pancreas and the kidneys), when suspecting the acute surgical pathology, is done by way of general examination, palpation, percussion and auscultation of the abdomen to detect specific "pathognomonic" symptoms. It is described in the Chapter "*Local status*" or "*Locus morbi*".

Compulsory examination of the pelvic organs includes specific obstetrical examination of the pregnant (general examination of the pregnant, palpation and measuring of the belly, auscultation of the fetus heartbeat, vaginal examination).

The principal clinical syndrome *is pain in the abdomen.*

According to the standard scheme, ***the plan for the additional examination (laboratory and instrumental examination) of the pregnant women, who are suspected of an acute surgical abdominal pathology*** and are admitted to the emergency surgery department, includes and ditects definitive pathological changes:

1. Clinical blood analyses – leukocytosis and rise in ESR (erythrocyte sedimentation rate), low blood haemoglobin in case of the ectopic pregnancy.
2. Clinical urine analyses – possible signs of nephropathy.
3. Biochemical blood analyses: rise in bilirubin, LFTs, amylase in case of acute pancreatitis.
4. Coagulogram: distinctive hypercoagulability (shortening of the clotting time, high PT (prothrombin), fibrinogen A, fibrinogen B, fibrin, etc.).
5. Ultrasound examination of the abdominal cavity detects the changes, which are characteristic of specific pathologies.

It is compulsory to consult a gynecologist.

Differential diagnostics between acute surgical abdominal organs' diseases, gynecological and obstetrical pathologies and renal colic is to be made, taking into consideration clinical peculiarities of acute abdominal organs' diseases, which are presented below.

Peculiarities of the clinical picture, the differential diagnostics and treatment of the pregnant women with acute surgical abdominal diseases

Acute appendicitis

Course of the disease. Across the pregnancy, the conditions for acute appendicitis or chronic appendicitis exacerbation are being created. The hypotonicity of the uterus motor functional depression of gastro-intestinal tract and constipation may develop under the influence of progesterone. Due to the intestinal atony, the emptying of the latter is delayed. Simultaneously, the shift of secretory function of gastro-intestinal tract develops, which causes increased virulence of the intestinal flora. With pregnancy progression and the enlargement of the uterus, the location of the colon changes – it is shifted to the upper abdomen (including cecum and the cecal appendage). During the pregnancy, the local blood circulation considerably increases in the uterus. The increased blood flow to the pelvic organs may cause chronic appendicitis exacerbation.

The diagnostics is based on the same symptoms as those ones in the nonpregnant state: abruptness of the disease, initial epigastric pain and its shift to the right iliac region (Kocher symptom), single-shot vomit, tachycardia, rise in temperature; pathognomonic symptoms.

The most typical clinical pattern is observed in the first trimester of pregnancy. The disease diagnosis is complicated during the full-term pregnancy: due to the location change of the cecal appendage and overstretching of the abdominal wall, the clinical pattern becomes less clear. The form of the disease (simple or destructive) influences the clinical manifestations of acute appendicitis. The severity of the intoxication and high leukocytosis, with left deviation, may indicate a destructive form (phlegmonous, gangrenous and perforative).

The combination of *the three signs:*

1) abruptness of the disease;
2) pain in right abdomen;
3) pain on palpation of the abdomen on the right – are indicative of the mandatory hospital admission of the pregnant to the surgery department for examination and monitoring; on arriving at the diagnosis AA (acute appendicitis) in any gestational age, urgent surgery is indicated.

To confirm the diagnosis in doubtful cases, the patient is to undergo the follow-up control during 1.5-2 hours, with leukograms, biochemical blood tests and urine tests being repeated. The laboratory results are compared with the results of the clinical observation, body temperature, pulse rate, examination and palpation of the abdomen. Both surgeon and obstetrician-gynecologist are to carry out the monitoring. The pregnancy may be complicated with the threat of miscarriage and of the premature birth.

Surveillance of pregnancy and labor. On confirming the diagnosis of acute appendicitis, surgical treatment is to be indicated. The appendectomy should be performed under the endotracheal anesthesia and with the increased oxygenation. No form of appendicitis can be the ground for abortion at any gestational age. The Caesarean section is performed in some cases, when acute appendicitis is accompanied with an obstetrical pathology, which demands operative delivery; or when the size of the uterus is an obstacle for the main surgical operation. The therapy, which is aimed at preserving of the pregnancy (antispasmodics, tocolytics) and preventing of fetal hypoxia, is administered for the operative pregnant women. In cases of labor in the postoperative period, application of forceps is indicated to avoid physical exertion.

Acute cholesystitis

Acute cholesystitis occurs rather frequently in pregnant women. It is caused by hypercholesteremia that may develop across the pregnancy. Infection plays a significant role; gastro-intestinal tract disorders adversely affect the pregnancy. Gallstone formation and, consequently, cholelithiasis, are pathogenetically associated with cholecystitis. Family history with cholecystitis is reported.

The diagnostics of acute cholesystitis, like of the rest of abdominal organs' diseases during pregnancy, is associated with certain difficulties, which are conditioned by the change in the topographic anatomical relationships, and due to the pregnancy complications (late gestosis).

Clinical manifestations. The disease manifests itself with pains in the right hypochondrium, which radiate to the right shoulder blade, shoulder. Sometimes pains are of dull character and they are accompanied with nausea, vomiting and heartburn. On palpation, the gallbladder is tender. Percussion gives pain in the right costal arch (Ortner symptom); pain at Kera point (Kera symptom). There is a severe pain with interruption of inhalation during the palpation of the right hypochondrium with the left hand, placing the thumb under the gallbladder (Murphy's symptom). There is pain between the crura of the right sternocleidomastoid muscle (Myussi-Georgievsky's symptom).

Series of laboratory tests help to diagnose acute cholesystitis (general and biochemical indices of blood, urine), as well as the ultrasound examination of the gallbladder, liver and the bile ducts.

Differential diagnostics with cholesystitis in pregnancy is made, first of all, in case of pyelonephritis, urolithiasis; if jaundice develops – with viral hepatitis and gestosis. Pregnancy may be complicated by the miscarriage.

Surveillance of pregnancy and labor. The patients are admitted to the Department of Pregnant Women Pathology, the treatment is provided by the therapists and surgeons. The bed rest, hunger for 24-48 hours, antispasmodics (no-spa, Baralgin, Avisan, Papaverine), isotonic solution of sodium chloride and glucose, Neo- Haemodes and a complex of vitamins are administered. In case there is no effect of conservative treatment, a decidion should made as to the surgery. Indications for surgical treatment are complications of cholecystitis with peritonitis, cholangitis and obstructive jaundice. Surgery on the gallbladder and ducts is carried out according to the same rules as for the nonpregnant women; during the postoperative period, the treatment is to be prescribed, which is aimed at the preservence of the pregnancy. If the disease develops by the end of the pregnancy, the pelvigenital canal is to be prepared for further delivery, with the conservative treatment of cholecystitis being continued. In case of uncomplicated cholecystitis, the prognosis for the mother and fetus is generally satisfactory.

Acute pancreatitis

Pancreatitis takes the third place among the acute diseases of abdominal cavity, after the acute appendicitis and acute cholesystitis. In pregnant women, the disease develops more severely. Not only mechanical disorders, which prevent the outflow of pancreatic juice, contribute to the development of

pancreatitis, the hormonal changes, which are characteristic of pregnancy, contribute to its development as well. The background for the development of pancreatitis are infectious diseases, intoxication, stress, diseases of the liver and gastrointestinal tract, cholecystitis and gestosis. In pregnant women, edematous pancreatitis is most common, less frequent are pancreatonecrosis and purulent pancreatitis, which can affect certain parts of the gland, either most of it or the whole of it (focal, subtotal or total pancreatic necrosis). The severity of symptoms in pregnant women depends on the clinical form of the disease.

Clinical manifestations. Pregnant women complain of vomiting and severe pain in the epigastrium and in the left hypochondrium, as well as on the worsening of the general condition, poor appetite and insomnia. Eventually, the pain becomes sharp and the vomiting becomes painful and uncontrollable. The body temperature rises. Abdominal distention and intestinal paresis are registered. The onset of the disease is noted by the abdominal distention in the upper abdomen due to the paresis of the transverse colon (Gobie symptom).

In 40% of patients the jaundice of the skin and sclera develops. Sometimes there are neurological symptoms, headache and confusion. The tongue becomes dry and coated. Rash, hemorrhage, vasculitis may appear on the skin. Tachycardia and hypotension develop. Palpation of the abdomen gives pain in the epigastrium and in the left hypochondrium (in the projection of the pancreas), though the onset of the disease is not marked by muscle tension and symptoms of peritoneal irritation. Positive symptoms of Kerte, Kacha, Voskresenskogo, Mayo-Robson are noted.

Diagnostics of acute pancreatitis in pregnant women is complicated. It is essential to differentiate between acute pancreatitis and preeclampsia, premature detachment of normally situated placenta, urolithiasis, and acute cholecystitis. Laboratory tests help in the diagnosis.

Clinical analysis of blood detects high leukocytosis with left deviation. The bioassay of amylase in the blood and urine, which increases severalfold, is of greatest diagnostic value. The severity of the disease may be indicated by the development of hyperglycemia and hypocalcemia. Ultrasound examination of the pancreas and hemostasiogram analysis are compulsory. The results of laboratory tests are compared with the results of the clinical observation of the pregnant women.

Gestation and labor course. The disease causes a number of serious pregnancy abnormalities: miscarriage, premature delivery, premature detachment of a normally situated placenta, bleeding in the postnatal and early postnatal periods, fetal death in the womb.

Surveillance of pregnancy.Treatment of edematous pancreatitis begins with the abolishing of drinking and nutrition, the evacuation of gastric contents (through the nose with a thin probe), antispasmodics and analgesics are prescribed. Polyion solutions are injected (Ringer-Locke isotonic solution, lactasol, etc.) under the control of sodium, potassium, calcium, chlorine in the plasma to restore the water-electrolyte balance. To control intoxication Neo-Haemodez, Cocarboxylase, Ascorbic acid with 10% or 20% glucose solution (under the control of blood glucose) are indicated. The 4% sodium bicarbonate additive solution is introduced under the control of blood pH. The 1% solution of potassium chloride is indicated to remove the pancreatic edema. Reopolyglucin is used to improve the microcirculation and to restore CBV (circulating blood volume). To fight the infection, pregnant women are prescribed Penicillin antibiotics and Penicillin synthetic analogues (Ampicillin). In case of severe pancreatic necrosis, broad-spectrum antibiotics are prescribed. Bearing in mind the severity of the condition of the patients, the complex of vitamins and protein drugs are prescribed. It is compulsory that pancreatic enzyme inhibitors Trasilol, Contrycal, Gordox should be introduced. Infusion therapy is carried out under the control of hematocrit test and daily (hourly) diuresis, taking into account the loss of fluid with vomiting and of breathing. Every three hours, blood pressure and temperature are to be controlled. Antipyretic agents (Analgin, Paracetamol) are administered with the temperature, which rises above 38^0C. Intravenous infusion magnesium sulfate is prescribed. Favorable outcome is possible in acute serous pancreatitis; though a high percentage of maternal and perinatal mortality is registered with other forms of pancreatitis.

Surveillance of labor and of the postnatal period. If the edematous form of acute pancreatitis develops up to 12 weeks of pregnancy, it is desirable to terminate the pregnancy after the disease remits. In case acute edematous pancreatitis develops in the late stages of pregnancy, preparation of

the birth canal is to be prepared for further delivery, with the conservative treatment of cholecystitis being continued. If the disease occurs at any time during pregnancy and the pregnancy is under the threat of termination, the treatment aimed at maintenance of the pregnancy is inappropriate. Vaginal birth is carried out with thorough anesthesia, together with simultanuous intensive infusion therapy; the right ammount of antispasmodics is to be introduced. The second stage of labor ends with the application of the forceps. One of the most serious complications of childbirth is bleeding, which is associated with the blood coagulation values; therefore, the prevention of the bleeding is mandatory. Due to the severe intoxication of the parturient women, intra- and postnatal mortality of children increases. Childbirth gives little improvement as to the condition of the patients, so conditions for massive therapy are created. The spectrum of antibiotics, which are aimed at treatment of peritonitis or sepsis, gets wider. Indications for surgical treatment are purulent pancreatitis and peritonitis. The purpose of the surgery is to create an outflow of active enzymes from the pancreatic ducts, to impose a cholecystostatic discharge and to drain the small omentum, to create a canal (tampons, drains) for the passage of pancreas sequesters. To perform surgery of this kind with the underlying condition, it is necessary to empty the uterus.

On the other hand, the performance of two operations with the background of serious condition of the patients, affects the condition of the women even more badly. The postoperative period is severe; there is high percentage of complications and deaths.

Acute intestinal obstruction

Course of the disease. Acute intestinal obstruction (AIO) can occur in pregnant women and women in labor. The promoters are uterine synechiae, adhesions, chronic inflammatory processes, complicated postoperative period in the past, changes in the motor function of the gastrointestinal tract associated with pregnancy. In pregnant women, both mechanical and functional bowel obstruction occur, their frequency rate, during pregnancy and in the nonpregnant state, is approximately the same (88% and 12% respectively). Clinical manifestations of functional obstruction develop gradually, starting with the delay in the stool and due to the gas, distension of the colon, which subsequently spreads to the small intestine. Then "fecal" vomiting occurs and there is an increase in peritonitis events deelopment. Mechanical obstruction is subdivided into obstructive, strangulation, mixed and vascular (intestinal infarction).

Clinical manifestations of acute intestinal obstruction (AIO) are diverse, which is related to the dependence of the character of its manifestations on the level of the obstruction; it is either related to the absence or presence of compression of the vessels and nerves of the mesentery, the degree and the type; as well as to the time since the onset of the disease, and to the duration of pregnancy and the size of the ovum. The higher is the level of the obstruction, the more obvious are the general manifestations of the disease, vomiting and severe dehydration, but the less obvious are the bloating, flatulence; there is no stool retention.

The compression of the mesenteric vessels, which leads to acute disruption of the intestinal trophism (twisted bowel, nodulation, internal entrapment, less often and to a lesser extent during invagination and adhesive obstruction), causes necrosis, in a short time, with a subsequent burst. With the prevalence of compression of the veins, necrosis of the intestine develops after 1-2 hours from the onset of the disease, acute pains are noted due to the increasing swelling and imbibition of the intestine with blood; the clinical manifestations of shock and death may also occur. When the inflow and outflow of the blood into the mesentery is disturbed, the symptoms of acute intestinal obstruction (AIO) become obvious; though, rapidly progressing peritonitis, caused by the slight permeability of the intestinal wall and, sometimes, by its perforation, predominates.

In the initial period of the development of acute intestinal obstruction (AIO, which is the stage of the pain or the initial stage (2-12 hours), patients complain of a sudden pain in the abdomen (its distension is associated with gas and stool retention), and vomiting. The pains are of cramping charater (they occur with the increase of the intestinal noise), but they can be permanent as well and seizure-like (strangulation). Positive symptoms of Val, Kivul are detected.

In the second phase of the disease – ***the stage of false remission or the stage of compensation abilities*** (12-36 h) – hemodynamic disorders prevail. With to the weakening of intestinal contractions,

the pain decreases and its character changes: pain becomes permanent, the flatulence and vomiting increase.

There are signs of peritonitis, the impaired activity of parenchymal organs (the liver, kidney), increased saltwater imbalance (dehydration, hypochloremia, hyponatremia, hypokalemia, acid-base balance drift). Positive symptoms of Sklyarov ("splashing noise"), Spasokokukotsky ("falling drop noise"), Grekov ("Obukhov hospital") are registered, etc.

In the late period of the disease – ***the terminal stage or the stage of decompensation and complications*** ("*outcome period*"), the condition of the patients is extremely critical as a result of severe intoxication due to the development of diffuse peritonitis, severe hypovolemia and disorders of central and organ hemodynamics – high mortality rate of sick mothers and fetus is registered.

As a result of the maternal toxicity in pregnant women, women in labor and recently delivered women, it is difficult to diagnose diseases, especially in the second and third trimesters of pregnancy, since there are no typical symptoms of acute intestinal obstruction (AIO) on palpation and percussion of the abdomen in case of the full-term pregnancy, it is impossible to radiographically examine the gastrointestinal tract and determine the horizontal levels (Kloiber's cups).

AIO pain can be mistaken for the onset of the labor and patients are usually delivered to the obstetric hospitals; and, as a result, the time for surgical treatment is lost. Almost half of the pregnant women undergo surgery no later than 36 hours after the onset of the initial symptoms of acute intestinal obstruction.

Acute intestinal obstruction (AIO) can cause premature detachment of a normally situated placenta, and it can be mistaken for other acute abdominal pathologies or rupture of the uterus. As a result of all the complications, the outcome of pregnancy with an AIO is commonly unfavorable.

Surveillance of pregnancy and of labor. With acute intestinal obstruction (AIO), the management of pregnancy depends on the type of the intestinal obstruction. The conservative treatment methods are not only of therapeutic, but also of a diagnostic value.The conservative treatment can begin with the introduction of atropine sulfate and administration of siphon enema. Administration of motility agents to determine the nature of the obstruction is not justified. At the same time, the evacuation of gastric contents (through the nose, with a thin probe) is performed. Infusion therapy should be carried out in full scale and should precede the surgical treatment. The failure of the conservative measures during 2-3 hours qualifies for the surgery. The patients are to be operated on in an obstetric hospital, starting from the 28th week of pregnancy (being in the early stages, patients may stay in surgical departments). In case the diagnosis of acute mechanical obstruction is clear upon the admission to the hospital, the pregnant woman is immediately to be prepared for the surgery. The prevention of intrauterine hypoxia is mandatory.

The extension of surgical interference, which is associated with obstruction, is determined and performed by the surgeon. The abortion is indicated in cases of dynamic intestinal obstruction caused by the pregnancy itself, if the surgical treatment is noneffective.

In other patients, surgical interference in uterus should be avoided unless it is necessary to empty it for technical reasons or due to strict obstetric indications. When the Caesarean section is forced to be performed under peritonitis, this should be followed by emptying of the uterus and its surgical removal. Life saving of the patient with a serious illness should prevail over all other considerations.

Perforated ulcer

Perforated gastroduodenal ulcer occurs seldom in pregnant women. This disease is caused by a long ulcerative history, the lack of preventive antacid therapy, failure to follow a diet, and stress. The significant role belongs to Helicobacter pylori infection; gastrointestinal disorders have also an effect.

Clinical manifestations. A sharp ("stabbing") epigastric pain manifests the disease. Nausea, vomiting, heartburn are present. The pregnant woman's general condition is progressively getting worse. On palpation, a sharp pain and rigidity of the anterior abdominal wall muscles are present; *Shchetkin-Blumberg* symptom in the epigastrium is positive. Pathognomonic symptoms of PU (*Spizharny, de Querven, Kulenkampf, auscultative triad of Gusten*) are positive. Eventually, the pattern of diffuse peritonitis develops.

The diagnostics of perforated ulcers, as well as other acute diseases of the abdominal cavity, during pregnancy, is associated with the well-known difficulties, caused by change in the topographic-anatomical relationship, in addition to the pregnancy complications (late gestosis). The main diagnostic method is *x-ray examination* – the survey radiography of the abdomen (*Jobert's symptom*).

Surveillance of pregnancy and of labor. When confirming the diagnosis of the perforated ulcer in a pregnant woman, urgent surgical treatment is commonly indicated. The intervention should be carried out under the endotracheal anesthesia under the increased oxygenation. Prevention of intrauterine hypoxia is mandatory. The extension of surgical interference is determined and performed by the surgeon. As a rule, common suturing or excision of the perforated ulcer is performed and followed by gastro-or duodenoplasty; a thorough lavage and adequate drainage of the abdominal cavity hae to be performed. Caesarean section is administered in some cases when being combined with obstetric pathology that require operative delivery, or when the size of the uterus may prevent the main operation. When Caesarean section is forced under peritonitis, uterus emptying should be followed by its removal. Having been operated on, the pregnant women are prescribed therapy aimed at preserving pregnancy (antispasmodics, tocolytics) and prevention of the fetal hypoxia. In cases of onset of the labor in the postoperative period, application of forceps is indicated to avoid physical exertion.

For a more detailed description of clinical manifestations and definitive pathological changes in the additional methods of research, as well as treatment of acute surgical diseases of the abdominal organs, refer to Chapter “Acute Abdomen Syndrome”.

Ectopic pregnancy

Fallopian pregnancy. Clinical manifestations and diagnostics. Most often, the doctor has to deal with disturbed tubal pregnancy, which is characterised with various clinical manifestations.

Pregnancy disturbed due to the type of the rupture of the tube, usually does not presuppose diagnostic difficulties. It is recognized due to the following data: acute onset against the background of a good general condition, which for some women (not all!) is preceded by the delay in regular menses from one day to several weeks. Suddenly sharp pains occur in the lower abdomen to the right or left, which radiate to the anus, sub- and supraclavicular region, shoulder or scapula, to the hypochondrium. The pains are accompanied with nausea or vomiting, dizziness and loss of consciousness, sometimes with a loose stool. The general condition of the patient is getting progressively worse, up to the development of severe hemorrhagic shock. In some patients it takes several hours, in others – 20-30 minutes, depending on the speed of the bleeding.

Physical examination of the woman usually gives every reason to confirm the diagnosis of the internal bleeding. The patient often shows deferred reaction, and the signs of anxiety are produced more rarely. The skin and visible mucous membranes are pale, the extremities are cold, the respiration is frequent, superficial; tachycardia is noted, the pulse is weakly filled; low blood pressure is noted, the tongue is moist, not coated; the abdomen may be somewhat swollen; and rigidity of the anterior abdominal wall muscles is not present. On palpation, there is pain in the lower abdomen, especially on the affected side. Symptoms of peritoneal irritation are also detected here. On percussion, as a rule, the dullness of percussion sound in the sloping areas of the abdomen is observed.

When viewed with the help of the mirrors, different degrees of cyanosis or pallor of the mucous membrane of the vagina and the vaginal part of the cervix (exocervix) can be detected. There is no bleeding from the cervical canal, since the bleeding, which starts with the detachment of the decidual membrane, usually occurs later, in the postoperative period.

In some cases, if the doctor still has doubts about the correctness of the diagnosis, and the patient's condition remains relatively satisfactory, the recto-uterine pouch puncture (culdocentesis) through the posterior vaginal vault can be performed.

Internal rupture of the fetus container or ***tubal abortion*** (***aborted ectopic pregnancy)*** presents significant diagnostic difficulties. This version of the abortion is characterized by a slow course that lasts from several days to several weeks. Periodically renewed partial detachment of the ovum from the fetus container is accompanied by slight (20-30 ml) or moderate (100-200 ml) bleeding into the lumen and into the abdominal cavity, without creating a noticeable effect on the patient's general

condition. However, at any time the bleeding can become significant or abundant, which, of course, clarifies the clinical picture, but significantly worsens the patient's condition. Interruption of the pregnancy, which began as an internal rupture of the fetus container, may often cause an external rupture accompanied by an increased bleeding.

Clinical findings. Carefully collected history significantly facilitates the diagnostics. In the past, patients may have inflammatory processes of internal genital organs, abortions, appendectomy, suffer from infertility; they may use contraceptives and ovulation inducers or may have ectopic pregnancy.

The main symptoms of pregnancy, which is interrupted due to the internal rupture of the fetus container, are represented by the following triad:

1) delay of menstruation
2) abdominal pain
3) bloody vaginal discharge.

Unfortunately, this triad is not pathognomonic for the tubal abortion. These symptoms occur in many other gynecological and extragenital diseases, which makes the diagnostics much more difficult.

The guiding symptom *of the tubal abortion is pain.*

It may occur in almost all the patients. The causes of pain and its nature are diverse. Pain may occur as a result of the bleeding into the lumen. Blood can pour into the abdominal cavity and accumulate in the recto-uterine pouch or along the lateral channel of the corresponding side to spread to the upper sections of the abdominal cavity, and can irritate certain parts of the peritoneum. The bleeding can temporarily stop, and then recover with an unpredictable force and frequency. Different reasons for the bleeding clinically correspond to the comprehensive nature of the pain: paroxysmal, cramping with irradiation into the rectum, shoulder, shoulder blade, collarbone and hypochondrium. The attack of the pain may be accompanied with weakness, dizziness, darkening of vision, cold sweat, nausea, less commonly – by vomiting and, sometimes, – by loose stools.

In case the abdominal bleeding continues, the intensity of the pain increases, the general condition of the patient becomes worse and the doctor discovers the symptoms of the disease, similar to that of a ruptured tube. More often, however, the attacks of the pain cease completely.

The woman again feels quite healthy or she has a feeling of heaviness in the lower abdomen, she has a sensation of a foreign body, that puts pressure on the anus.

Complaints on the *bleeding from the genital tract* take the second place in the frequency rate (80%), they come several hours after the pain attack. They are caused by the separation of the decidual membrane, which is triggered by the fall in the amount of the sex hormones. The complaints have a persistent character. The amount of the blood loss is insignificant, more often it is miserable, the color is dark, it can be almost black or brown. In rare cases, scraps of decidual tissue come with blood.

The third symptom is *the delay in menstruation* (65%). This symptom is not determinative, since the bleeding due to abortion may begin before, at or after the next day of the expected menstruation and mask its absence. Moreover, termination of pregnancy can occur in the early stages, even before the possible onset of the next menstruation.

The results of physical examination depends largely on the time when it is carried out. If the patient is examined during or immediately after the pain attack, the clinical picture will be more obvious. At the time of the attack, the patient’s skin and mucous membranes are pale; moderate tachycardia in the presence of normal or slightly low blood pressure are present. The abdomen is soft, not swollen, there is lower abdominal pain on the side of the affected fallopian tube. The more or less obvious symptoms of peritoneal irritation are also determined in the absence of the abdominal wall muscles tension. The dull percussion tone is infrequent. If some time has passed after the attack, the patient may feel completely healthy, have the usual color of the skin and mucous membranes.

On examination of the vagina and cervix with the help of mirrors, loosening and cyanosis of the mucous membrane, typically occurring definitive bloody discharge from the cervical canal of the uterus can be detected. On bimanual examination, the external os is closed on palpation, the uterus is enlarged, as it corresponds to the expected gestational age (or it is less than the one of the expected gestational age). With a very early termination of the pregnancy, the uterus may not be enlarged. The

disturbed tubal pregnancy leads to a one-sided increase in the uterine adnexa. The shape of the palpable formation can be diverse: sausage-shaped or spindle-shaped with clear contours due to the formation of hematosalpinx; the shape is of an indefinite form without clear contours in case of peritubal hematoma. If an ectopic hematoma is formed, the parovarium formation is palpated in a single conglomerate with the uterus. Palpation of the formation is always painful, and mobility is rather limited. Vaginal vault may remain high. The displacement of the uterus to the pubis, in the presence of even a small amount of blood in the rectouterine space, causes sharp pain.

Differential diagnostics. A significant number of patients may have not the entire symptom complex, and the symptoms, which are present, are often devoid of typical characteristics. In this case, tubal abortion is masked by other gynecological and extragenital diseases: early-onset uterine miscarriage, ovarian apoplexy, acute inflammation of uterine adnexa, pelvioperitonitis, malnutrition of subserous uterus myoma, torsion of the ovarian tumor pedicle, appendicitis.

Differential diagnosis is based on the clinical course of the listed diseases and the use of additional methods of research.

The clinical picture of the onset of fetus miscarriage includes complaints on cramping or pulling pain in the lower abdomen, bright bleeding from the vagina after a delayed menstruation, there are no signs of internal bleeding; external os is open; the size of the uterus corresponds to the expected pregnancy (delayed menstruation). The degree of anemization corresponds to external bleeding.

The clinical picture of ovarian apoplexy and tubal abortion has many similarities, so their differential diagnostics is rather complicated. The absence of menstruation and other subjective and objective signs of pregnancy make it lean toward ovarian apoplexy, but this is very circumstantial.

The guiding symptom of acute inflammation of the uterine adnexa, *as well as disturbed ectopic pregnancy, is pain, but the characteristics of the pain are not the same.*

With the inflammatory process, pain increases gradually, and is accompanied with fever; there are no signs of internal bleeding. On vaginal examination the size of the uterus is normal, the adnexa are often enlarged on both sides, the vaults are high. The malnutrition of subserous uterus myoma causes a pain that occurs quite acutely, but without signs of internal bleeding. Uterine myoma is to be differentiated from ectopic hematoma with disturbed tubal pregnancy. Ectopic hematoma, along with the tube and the uterus, may represent a single conglomerate and is somewhat similar with uterine myoma. However, the borders of uterine myoma are clearer and it is usually mobile.

Acute onset is characteristic of ***torsion of the ovarian tumor pedicle***: pain in the right or left iliac regions, nausea, and vomiting. There are no signs of internal bleeding. Symptoms of peritoneal irritation may occur. The internal studies are quite specific: the size of the uterus is normal, round, elastic consistency, painful formation in the uterine adnexa, high vaginal vaults, normal vaginal discharge.

With ***appendicitis***, pain appears in the epigastric region, then descends to the right iliac; it is accompanied with nausea, and less often with vomiting or fever. Symptoms of internal bleeding are absent. There is no bleeding from the vagina. Pain, the abdominal wall muscles' tension, symptoms of peritoneal irritation in the right iliac region and positive "appendicular" symptoms are noted. Internal research shows no changes in uterus and uterine adnexa. There is a rather definitive picture of white blood: leukocytosis, neutrophilia with the left shift.

To clarify the diagnosis of tubal pregnancy, disturbed due to the type of the internal rupture of the fetus container, there are numerous ***additional methods of research***. The most informative and comprehensive are the following:

1) testing of presence of human chorial gonadotropin (HChG) or its three subunits in the blood serum or urine;

2) ultrasound scanning;

3) laparoscopy.

The *testing of HChG* helps to establish only the fact of the presence of the pregnancy without specification of its location, therefore it can be used for differential diagnosis with inflammatory

process in the uterine adnexa, ovarian apoplexy, endometriosis of the uterine adnexa and the similar diseases.

Ultrasound scanning is a common non-invasive method, in combination with the testing of human chorial gonadotropin (HChG), it can provide high diagnostic accuracy. The main signs of the tubal abortion, detected by the ultrasound examination, are the absence of the ovum in the uterus, enlarged uterine adnexa, and the presence of fluid in the recto-uterine pouch. The heart pulsation of the embryo during the ectopic pregnancy is rarely recorded.

The most informative method is ***laparoscopy****.*

Such well-known (to the gynecologists) method, as ***the puncture of the recto-uterine pouch of the abdominal cavity,*** which is performed through the posterior vaginal vault, has not lost its significance. The detetion of the dark liquid blood, which contains small clots, confirms the presence of the tubal pregnancy.

In many cases, to perform differential diagnostics, it is advisable ***to carry out the histological study of the endometrial smear (scraped off material).***

Progressive tubal pregnancy is rarely diagnosed. The reason is the lack of fair clinical symptoms. However, the use of the comprehensive research methods makes it possible to detect ectopic pregnancy before its termination. The early diagnostics contributes to timely adequate treatment, which may save not only the health, but also the reproductive function of the woman. Progressive tubal pregnancy is a short period condition (4-6 weeks, it rarely occurs longer than this period).

Clinical findings.There are practically no obvious symptoms which are characteristic of progressive ectopic pregnancy. When the menstruation is delayed, or it seems to a patient that there is something unusual with it. The signs that are characteristic of physiological or complicated uterine pregnancy may appear, such as food faddism, nausea, excessive salivation, vomiting, breast engorgement, sometimes, minor pains in the lower abdomen, which are not of a certain nature. The general condition of the patient is quite satisfactory. Specific studies in the early stages of the progressive tubal pregnancy are usually unable to produce the results, which may confirm the diagnosis. Cyanosis and maceration of the mucous membrane of the vagina and cervix are not significant. Due to the hyperplasia and hypertrophy of the muscular layer and the transformation of the mucous membrane into the decidual, the size of the uterus, as a rule, corresponds to the period of the menstruation delay. The increase in the uterus, however, is not accompanied with the change of its shape; it remains pear-shaped and somewhat oblate in the anteroposterior direction. The softening of the isthmus is not significantly marked. In some cases, it is possible to palpate the enlarged tube and to detect the pulsation of the vessels through the lateral vaults. It is much easier to suspect the progressive tubal pregnancy, if it exists for more than 8 weeks. It is from this time that the size of the uterus lags behind the expected duration of the gestation. The detection of thickening of the fallopian tube is expectant.

Diagnostics. The human chorial gonadotropin (HChG) test, ultrasound examination and laparoscopy can confirm the diagnosis.

Treatment. Now, there exists a universal point of view on the tubal pregnancy treatment: as soon as the diagnosis is established, the patient is to be operated on. The nature of the operation is governed by many factors: the location of the ovum, the severity of pathological changes in the affected and the opposite tubes, the amount of blood loss, as well as by the general condition of the patient, the patient's age and the patient's desire for pregnancy in future.

If abortion is accompanied by a heavy bleeding, the time factor plays a crucial role when providing the emergency care. The anesthesiologist, within a short time, is to take resuscitation measures, aimed at bringing the patient out of shock and to stabilize the patient's condition, and after the anesthetic care shoul be proceed to. By this time, the gynecologist should be ready for the surgery. *The operation of choice in such a situation is the removal of the fetus container, i.e. the fallopian tube.* Surgery is carried out in 3 stages:

1) celiotomy, hemostasis;

2) resuscitation;

3) the continuation of the operation.

Laparotomy can be carried out using any method the surgeon is good at. The hemostatic clamps are imposed on the uterine end of the tube and its mesentery. After this, the operation is temporarily stops until the anesthesiologist signals that continuation is possible. At this moment, the operating doctor can assist the anesthesiologist to perform resuscitation, providing him with the blood taken from the abdominal cavity. Infusion of the autoblood helps to quickly bring the patient out of shock, and this does not require prior blood group test and Rh phenotyping or blood compatibility testing. The operation can be continued only with the permission of the anesthesiologist. The pipe is to be cut off. The clamps on the uterine end of the tube and its mesentery are replaced with catgut or synthetic ligatures. Peritonization is usually carried out with the usage of a round uterine ligament. Then the remnants of the liquid blood and the clots are carefully removed under the continued full anesthesia. The abdominal wall is tightly sutured in layers.

In the absence of massive bleeding, indications for salpingectomy are significant pathological changes in the fallopian tube, as well as the age of the woman (35 years and older), who does not have the desire to maintain the reproductive function.

In other cases, it is necessary to strive to carry out a *"conservative" organ sparing surgery*, taking into account the following conditions: the patient's satisfactory condition at the time of surgery with compensated blood loss; the condition of the patient's health, which does not interfere with the carrying out of pregnancy and childbirth in the future; minimal changes in the uterine tube (the ideal condition is progressive pregnancy); the woman's desire to maintain the reproductive function; high professional skills of the surgeon.

"Conservative" operations are performed with the application of the microsurgical techniques. The most common of these are *salpingotomy and segmental resection of the isthmic part of the tube, with an end-to-end anastomosis.*

Laparoscopic surgery can be performed in case of a short duration of the progressive pregnancy, with the diameter of the fallopian tube not exceeding 4 cm; or in case of the disturbed pregnancy with an unsignificant damage to the tube and a moderate blood loss. The most common surgical intervention in these conditions is salpingotomy and salpingectomy.

Uncommon cases of ectopic pregnancy

Interstitial tubal pregnancy. It occurs highly rarely and it is interrupted rather late (at the 3rd-4th month) according to the type of the external rupture of the fetus container; it is accompanied with a heavy bleeding and is characterised with an obvious clinical picture.

The correct diagnosis is usually made during surgery, when the uterine deformity is detected due to the protrusion of one of its corners, high transposition of the ligamentous apparatus proximal to the lesion, in case of an obliquus location of the uterine fundus. The perforation may be of different size, but it has no communication with the uterus. The surgery includes – excision of the lateral angle of the uterus and imposing of two rows of separate stitches on the wound: muscular-muscular and serous-muscular. Peritonization is carried out with the involvement of the round uterine ligament.

Ovariocyesis. It occurs extraordinarily rarely. The ovum can implant on the germinal epithelium, which is sometimes associated with endometreosis, or it can develop incide the follicle. Progressive pregnancy, as a rule, is not diagnosed.Termination of the pregnancy is accompanied with bleeding of varying severity, which determines the peculiarities of the clinical picture. The topical diagnosis is possible only during *laparoscopy or laparotomy.* The following symptoms are characteristic of the ovarian pregnancy: an intact fallopian tube; the fetus container, which is the ovary, and which is connected with the uterus by means of the utero-ovarian ligament. *To treat the ovarian pregnancy surgery is performed, which initially starts with the ovarian resection and is proceeded with the removal of the parovarium.*

Pregnancy in the rudimentary (embryonic) horn of the uterus. It occurs very rarely. The rudimentary horn is marked by an underdeveloped muscle layer and by an inadequate mucosa. Progressive pregnancy is very rarely diagnosed. It can be suspected on the basis of unusual resuls of the internal gynecological examination: enlarged uterus (duration of gestation of more than 8 weeks

does not correspond to the delayed menstruation term) is rejected to the side; a tumor-like painless soft formation is detected on the opposite side, which is connected with the uterus by a thick pedicle.

Ultrasound examnaion or laparoscopy may be of great help here. The disturbed pregnancy occurs analogous to the type of the external rupture of the fetus container; it is accompanied with a heavy bleeding and it requires an *urgent surgical intervention*. *The surgery presupposes the removal of the rudimentary horn along with the adjacent fallopian tube.*

Abdominal pregnancy. Primary and secondary abdominal pregnancy occur extremely rarely. Progressive primary pregnancy is practically not diagnosed, its termination gives the clinical picture of an excited (tense) tubal pregnancy. With such a diagnosis, the patient is usually operated on. Secondary abdominal pregnancy occurs, as a rule, after the tubal abortion. Pregnancy can be carried to the advanced terms, which is an extraordinary threat to the life of the woman. At that, the fetus is rarely viable; malformation of the fetus is often detected. Secondary abdominal pregnancy can be suspected in women who had pains in the lower abdomen at an early term of the pregnancy, which is accompanied by a moderate bloody vaginal discharge. The woman typically complains on the painful movement of the fetus. On external examination of the patient the wrong position of the fetus can be detected, the small parts can clearly be felt. The fetus container does not contract, which can usually be determined on palpation. On the internal examination of the woman one should pay attention to the displacement of the cervix upwards and sidewards. In some cases, it is possible to palpate the uterus separately from the fetus. Ultrasound scanning does not detect the uterine wall around the fetal sac. The treatment of the abdominal pregnancy is always *only surgical*.

The nature of the surgery is extremely wide and unpredictable. It depends on the pregnancy terms and on the place of ovum implantation. In ***early stages*** of the aborted abdominal pregnancy, it is sufficient *to dissect the tissues of the bleeding area away* and to impose several stitches in. ***At the advanced pregnancy terms***, the placental villi penetrate deeply into the underlying tissue, so the placental site has to be removed together with the placenta: *removal or hysterectomy of the uterus is performed, uterine adnexa are removed, the bowel is resected and a part of the greater omentum is amputated*.

In the postoperative period, an individual management program is compiled for each patient, taking into account the general condition, the type of the ectopic pregnancy and the amount of the surgical intervention; this includs a set of the following measures:

1) the overall effect on the body due to the medication that improves non-specific body defenses that stimulate hematopoiesis and intensify anabolic processes;

2) a course of physiotherapy;

3) a short course of hydroturbations with a saved pipe.

(Habitual) miscarriage *is an inevitable miscarriage from its beginning up to 37 weeks*. If the miscarriage occurs more than 2 times, it is considered to be a *recurrent pregnancy loss.* Depending on the term of the pregnancy, it is subdivided into *spontaneous abortion* and *preterm birth*.

Spontaneous abortion (in domestic obstetrics) is considered to be the termination of pregnancy in the first 28 weeks of pregnancy. The mass of the fetus in these cases does not exceed 1000 g, and its height – 35 cm.

Preterm birth is the termination of the pregnancy from 29 weeks to 37 weeks. During this term, a premature baby is born with the weight of 1000–2500 grams and 35–45 cm tall.

Spontaneous abortion. The frequency of the spontaneous abortions (spontaneous miscarriages) reaches 15%, and preterm births – up to 7%.

Etiology. The causes of spontaneous miscarriages (spontaneous abortion) and preterm births are quite similar and extremely diverse. It often occurs that not one, but several causative factors lead to the termination of the pregnancy.

Despite the conventionality, these factors can be grouped as follows:

1) uterus pathology;

2) abnormality of the chromosom apparatus;

3) immunological disorders;

4) endocrine pathology;

5) infectious factors;
6) somatic diseases and intoxications;
7) psychogenic factors;
8) complicated pregnancy.

Clinical manifestations. Whithin the clinical course of the spontaneous miscarriage, ***the following stages, or forms***, are distinguished: *threatened miscarriage, starting miscarriage, abortion "in progress", complete and incomplete abortions.*

The threatened miscarriage is characterized by an increase in the contractile activity of the uterus muscles, but the ovum still communicates with the uterus. Clinically, this form of miscarriage is manifested by light gnawing pain in the lower abdomen and (or) in the sacrum. No bleeding is registered.

With the ***starting miscarriage***, the increased contractile activity of the myometrium leads to partial detachment of the ovum and insignificant bloody discharge from the cervical canal. The pain intensifies, sometimes acquiring the character of slight contractions. The miscarriage that began in the second trimester may manifest itself with a painful symptom with no bleeding. On vaginal examination, shortening of the uterine cervix and a small opening of the external os are noticed.

Further progression of the termination of pregnancy is referred to as ***abortion "in progress".*** The ovum loses its connection with the fetus container and descends into the lower part of the uterus or into the cervical canal. Abortion "in progress" is accompanied with severe cramping pains in the lower abdomen and a significant and profuse bleeding. In case of a rigid opening of the external os, the ovum can be completely expelled from the uterus into the cervical canal. The cervix is significantly increased in volume, and its body is reduced. This type of the abortion (abortion “in progress”) is also called *cervical abortion.*

If a part of the ovum has gone beyond the uterus, and the uterus contains only its remnants, such an abortion is called ***incomplete***. The guiding symptom of this stage of the abortion is *bleeding* of varying severity: from insufficient to heavy, which may develop hemorrhagic shock.

With ***complete*** abortion, the ovum is rejected entirely, only parts of the decidual membrane can remain in the uterus. This form of the abortion occurs extremely rarely, and if it occurs, it usually happens at the end of the second trimester.

The clinical manifestations of spontaneous abortion depend on the terms of the pregnancy, the form of the abortion, and the cause of the termination of the pregnancy.

In the first trimester, combination of pain syndrome and bloody discharge is typical for the miscarriage. In the second trimester, the initial manifestations of abortion are cramping pains in the lower abdomen, bleeding starts after the birth of the fetus.

The exception is the termination of pregnancy on the background of placental presentation (placenta previa), when bleeding becomes the guiding symptom, and it is abundant, as a rule.

Threatened miscarriage manifests by minor pains in the lower abdomen. A starting miscarriage is accompanied by increased pain, scanty bleeding is possible. An increase in the sharp cramping pain and abundant bleeding are characteristic of abortion "in progress".

The decrease in the pain, with the background of continuing bleeding of varying severity, is typical for the incomplete abortion. With the complete abortion, the pain subsides and the bleeding stops.

The specific clinical manifestations of spontaneous miscarriage develop due to the etiological factor, which caused it. Therefore, *the genetic factors* lead to miscarriage in the early terms of pregnancy. The abortion, which is caused by *istmicocervical insufficiency*, occurs in the second trimester of pregnancy; it starts with the discharge of amniotic fluid and ends with the rapid birth of the fetus on the background of weak painless contractions.

The termination of pregnancy with *antiphospholipid syndrome (APS)* is realized through the development of the chronic form of DIC syndrome (disseminated intravascular coagulation syndrome). Death of the embryo or fetus is often associated with decidual and/or placental vessels’ thrombosis. The formation of the syndrome of pregnancy, that does not develop, may occur. The history of women with antiphospholipid syndrome, may include a common miscarriage, intrauterine fetal death. Deep

and superficial veins' thrombophlebitis, arterial thrombosis in the cerebral, coronary, mesenteric vessels, as well as, in the retinal and glomerural arterioles are common with APS.

Abortions against the background of *androgenemia* in the early terms begin with bleeding, then a pain symptom joins; often, in such cases, syndrome of pregnancy, which does not develop, is formed; intrauterine fetal death may occur in later terms.

Death of the ovum with the further expulsion of it from the uterus can be observed in the presence of a *chronic and acute infection*, while bleeding is rarely heavy.

To diagnose spontaneous miscarriage is usually not problematic. It is based on the patient's complaints, the results of the general and gynecological examination, the results of colpocytologic, hormonal and ultrasound examinations.

Complaints presented by the patients with different degrees of the severity of spontaneous miscarriages, have already been described in details before.

The general condition of the patient may result from the fact of being pregnant and the ammount of blood loss due to the form of spontaneous abortion. With a threatened miscarriage and starting miscarriage, the condition of the women is usually satisfactory, in case early gestoses does not develop and if the miscarriage is not caused by a severe somatic pathology. With abortion "in progress", incomplete and complete abortion, the patient's condition depends on the duration, intensity and degree of the blood loss. Prolonged, insufficient bleeding may lead to anemea, the severity of which determines the condition of the woman. Acute blood loss can cause shock.

The results of the gynecological examination with threatened miscarriage indicate that the size of the uterus corresponds to the period of menstruation delay. The uterus contracts in respond to palpation. There are no structural changes in the cervix. With starting miscarriages, the cervix may be somewhat shortened with its external mouth being slightly open. Spasmodic body of the uterus that corresponds to the gestation period, the lower pole of the ovum, which is easily reached through the cervical canal, indicate the abortion "in progress". With incomplete abortion, the size of the uterus does not correspond to the terms of pregnancy (its size is less); at that, the cervical canal or external os are open.

Diagnostics. Laboratory and instrumental methods are used to make an early diagnosis and dynamic observation of the initial stages of the pregnancy termination.

Colpocitologic study helps to identify the threat of termination of pregnancy long before the onset of clinical symptoms. It is well-known that *the kariopicnotic index (KPI)* should not exceed 10% during the first 12 weeks of pregnancy, in 13-16 weeks it equals to 3-9%, in the later terms KPI is within the limits of 5%. An increase in KPI suggests a threatened abortion.

The detection of presence of *chorionic gonadotropic hormone, estradiol and progesterone* in plasma is significant when making a prognosis. Chorionic gonadotropic hormone in the serum of pregnant women in the first trimester makes 45000-200 000 IU / l, in the second trimester-70000-100 000 IU / l.

The detection of the ammount of 17-ketosteroids (17-KS) in the daily amount of urine in women with androgenemia is of significant diagnostic and prognostic value. If the amount of 17-Cs exceeds 42 μmol / l, the risk of spontaneous abortion is very high. Lupus anti-coagulant, antibodies to cardiolipin and thrombocytopenia are revealed in the venous blood of the pregnant women with APS (antiphospholipid syndrome).

The echographic signs of the threatened miscarriage in the early stages of pregnancy are: the location of the ovum in the lower parts of uterus, ill-defined contours, deformities, constriction of the ovum, local tension of the myometrium. From the end of the first trimester of pregnancy, with the threat of its termination, it is possible to identify areas of placental abruption, to measure the diameter of the isthmus, which should not exceed 5 mm.

Treatment. With the threat of spontaneous miscarriage, treatment should be carried out taking into account the duration of pregnancy, the stage of the clinical course and the cause of the disease. It is necessary to start the therapy as early as possible, because it is easier to carry on the pregnancy in the threatend miscarriage stage, it is more difficult to do this in starting miscarriage and it is impossible to be done in all the rest, which are coming after this. When prescribing a treatment and selecting a

dose of medication, in the first trimester of pregnancy, one should remember about their possible embryotoxic and teratogenic effects.

Unfortunately, it is not always possible to identify the cause of the termination of pregnancy, but it is always necessary to strive for this in order to succeed with the least effort.

Treatment of women with threatened and starting spontaneous miscarriage should be carried out only in a stationary. The treatment consists of the following: 1) a complete, balanced, vitamin-rich diet; 2) bed rest; 3) non-pharmacological affecting measures; 4) drugs that reduce psycho-emotional stress and relax the smooth muscles of the uterus.

The success of treating a threatened and starting miscarriage depends on immediacy and adequate remedies choice. Hospitalization of patients should be carried out at the first, even minor symptoms of the disease; since first minutes staying in hospital treatment should be carried out to the extent necessary and only when the desired effect is achieved the dosage of medicines can be gradually reduced and the range of means and methods of treatment be narrowed.

Preterm birth. Clinical manifestations. Due to the clinical manifestations, preterm labor is divided into *threatened preterm labor and started preterm labor*.

Threatened preterm labor is characterized with minor pain in the lower abdomen or lower back. Sometimes complaints are completely absent. Palpation of the uterus reveals the increased tone and excitability. Fetal heartbeat is not weak. On vaginal examination no changes in the cervix are registered.

In case of started preterm labor, the pains intensify and become cramping in nature. Vaginal examination shows a shortened cervix or cervical effacement. Often there is a discharge of amniotic fluid. The dilatation of cervix up to 4 cm indicates a latent phase of the first stage of labor, cervical dilatation of 4 cm and more indicates an active phase.

Diagnostics. The diagnosis of preterm birth is not difficult. It is based on the complaints of the pregnant woman and the results of external and internal obstetric research. The results of the clinical examination of the pregnant woman are confirmed by *hysterography*.

Management. The tactics of the preterm labor depends on the following factors:

1) the stage of the gestation course (threatened, started);
2) the terms of the pregnancy;
3) the condition of the mother (somatic diseases, late gestosis)
4) the fetus condition (fetal hypoxia, fetal malformations)
5) the state of the amniotic sac (whole, opened)
6) the degree of the cervical dilatation (up to 4 cm, more than 4 cm);
7) presence and intensity of bleeding
8) presence or absence of infection.

Treatment. Depending on the obstetric situation, *conservative management* or the *invasive therapeutic approach* are used.

Conservative management (prolongation of pregnancy) is indicated for threatened or started labor with the term of up to 36 weeks, with the whole amniotic sac, with the external os opened up to 4 cm, good condition of the fetus, in the absence of severe obstetric and somatic pathology, and signs of infection.

The complex of the treatment of threatened and latent phases of the started preterm labor includes:

1) bed rest;
2) a bland, vitamin-rich diet;
3) medication;
4) physiotherapy;
5) reflex and psychotherapy.

Pregnant women are prescribed sedative therapy – common valerian and motherwort, Tazepam, Diazepam (Sibazon, Seduxen). Antispasmodics drugs (Methacin, No-spa, Papaverine), antiprostaglandine (Indometacin), calcium antagonists (Isoptin) are used. A special role in eliminating of the threat of the termination of the pregnancy belongs to the drugs of ***tocolytic action*** – β-

adrenomimetics (partusisten, Bricanyl, alupent (metaproterenol)). The treatment starts with intravenous drip, after the desired effect they switch to the tablet formulation. Drug treatment is supported by *physiotherapy procedures* – magnesium electrophoresis, sinusoidal modulated current.

With the threat of abortion up to 34 weeks, it is necessary *to prevent respiratory distress syndrome* in the new-born. During 3 days, pregnant women take corticosteroids (Dexamethasone at a dose of 8 mg or Prednisolone – 60 mg), which contribute to the synthesis of surfactant and the maturation of the fetal lungs. A week later, the course of corticosteroids can be repeated.

Special attention should be given to a group of women with the threatened and started preterm labor when the cervix is dilated less than 4 cm against the background of the amniotic fluid discharge. In the absence of infection, good condition of the mother and fetus and the gestational age of 28-34 weeks, it is possible to prolong the pregnancy, if to strictly follow all the rules of aseptics and antiseptics (sterile pads, disinfection of the external genital organs, insertion of the vaginal suppositories or antibacterial tablets). It is necessary to strictly control the first signs of the infection of the birth canal, which may develop: thermometry, blood tests, bacteriological examination of the vaginal discharge. At the same time glucocorticoids are prescribed, the maturation of the fetus lungs is promoted. When the signs of infection are shown, labor induction is prescribed.

Invasive therapeutic approach is used for threatened and started labor with severe somatic diseases of the pregnant woman, severe gestosis, fetal hypoxia, fetus abnormality and death of the fetus, if signs of infection.

In case of the started preterm labor, the delivery is carried out through the birth canal under constant cardiomonitoring control. The premature labor requires special care. It is necessary to widely use antispasmodics, provide adequate pain relief therapy without narcotic drugs. The regulation of the labor, in case of any disturbance, should be carried out carefully. The weakness of the labor is to be controlled by the introduction of Prostaglandin or Oxytocin under the careful control of cardiotocography.

The preterm labor is often complicated by the *precipitated labor or oxytocia*, in these cases, tocolytics or magnesium sulfate are indicated. Prevention of hypoxia of the fetus is compulsory.

The expulsive stage of the labor is a great danger for a premature baby, therefore, to prevent fetus birth injuries, the expultion should be carried out very carefully, without protection of the perineum. To reduce the resistance of the pelvic floor muscles, a pudendal anesthesia or perineotomy is indicated.

In the follow-up period, measures should be taken to prevent bleeding.

Delivery with the ***Caesarean section*** in case of the preterm labor is performed when clinically indicated. The indications are as follows: placenta previa, premature detachment of a normally located placenta, eclampsia and the transverse position of the fetus. Other surgical indications in the interests of the fetus (complicated birth during pelvic presentation, aggravated obstetric history of the mother – stillbirth, miscarriage, infertility) are followed only if the neonatal resuscitation is available.

The child born prematurely has signs of immaturity, so the primary treatment and all therapeutic measures should be carried out in the incubator.

Prevention of the miscarriage and preterm birth. Premature birth is a serious problem for the practical obstetrics, they are dangerous due to serious complications for the mother and the fetus (newborn). The perinatal mortality in case of the preterm labor is 20 times higher than in the term birth, which is explained by the general physiological immaturity of the body of the newborn. The expectancy rate of the preterm birth is especially high in women, who are at a high risk.

All ***the risk factors for miscarriage*** are divided into 4 groups:

1) socio-biological factors (age, occupation, bad habits, living conditions);

2) obstetric and gynecological history (nature of the menstrual cycle, the consequences of previous pregnancies and childbirth, gynecological diseases, uterine malformations)

3) extragenital diseases (acute infections during pregnancy, heart defects, hypertension, kidney disease, diabetes mellitus);

4) complications of the present pregnancy (severe EPH-gestosis (edema, proteinuria, hypertension0, RBC sensitization, antiphospholipid syndrome, hydroamniosis, multiple pregnancy, placenta previa).

The more often the risk factors of miscarriage are combined, the higher is the chance of the preterm delivery.

Pregnant women, who have undergone acute surgical diseases of the abdominal organs, are to be managed at a polyclinic by a surgeon and have to be under regular medical check-up in the clinic for women at the place of residence in case of the maintenance of the pregnancy.

II.5. CLINICAL FEATURES OF THE COURSE OF ABDOMINAL ORGANS' ACUTE DISEASES IN THE ELDERLY PEOPLE

The problem of clinical features of surgical diseases, especially of acute diseases of the abdominal organs, in elderly people (from 60 to 75) and senile people (from 75 to 90 years) is relevant not only due to the fact that the number of such patients is increasing, (now they make one third of the surgery patients in hospitals), but also due to the fact that the clinical symptoms of these diseases are often "atypical", which makes it difficult to make a diagnose even in the hospital. They are the ground for emergency surgery. Therefore, all this does not make it possible to show noticeable success in the treatment of the elderly patients.

Aging *is a natural biological process that inevitably develops with age and is characterized by a gradual decrease in the adaptive abilities of the body and an increase in the probability of death.* ***Old age*** *is the final stage of the body's vital activity, the consequence of the aging process. The time of the onset of the old age is highly conventional; the idea of it changes with the extended life-span.*

The common signs of physiological aging, like change in the appearance, mental changes, performance decrement, physical activity decrease, etc., appear at the age of 60. According to the international classification of the population as to the age groups, the age from 60 to 75 years old is conventionally considered elderly people, and people of age after 75 are considered senile people.

The distinction is made between ***normal (or physiological) and presenilation aging***. There is a decrease in the level of biological processes' activity already at the age of 30-35. During physiological aging, the change in the basic physiological systems of the body is relatively steady, and the person remains physically and mentally active and remains being interested in the surrounding world until a very old age. Significant adaptive abilities of the body can provide a sufficiently high basic physiological functions activity rate, that is, practical health, for a long time. That is why aging, being a general biological process, should not be associated with a disease.

Presenilation occurs largely due to previous diseases, environmental factors, harmful habits, as well as body's regulatory systems burdens, and is characterized by an early development of age-related pathology. For example, the use of alcohol, smoking, improper feeding significantly reduce or arrest the adaptive abilities of the body, thereby they contribute to the development of the diseases, which are specifically attributed to the old age. Presenilation is the consequence of acceleration of the aging process.

Thus, elderly and old people of the same age can be a heterogeneous group due to their physical and mental health. The bodybuild of the man plays a significant role in this, mainly due to the heredity.

There is a certain pattern of the very nature of aging, which is selectively handed down from generation to generation. Thus, among the representatives of one family, age-related changes are most noticeable primarily in the cardiovascular system, in representatives of another family – in the endocrine system, and so on.

The slower aging of the women is associated with the features of the genetic apparatus (the average life expectancy of women in the developed countries is 4–11 years longer than that of the men).

The process of physiological aging begins in different tissues and organs not simultaneously and develops with different intensity. Aging presents gradual decrease in the cell viability of the body:

protein biosynthesis is changing, the activity of oxidative enzymes decreases, the number of mitochondria decreases, the function of cell membranes is disturbed.

Finally, the aging of the cells leads to their destruction and death. The cell death is not the same in different organs and parts of the same organ. The rate of cell aging is determined by its belonging to a functional system. Thus, changes in the connective tissue are manifested in all the components, the functionality of the muscle cells decreases with age. At the same time, some important adaptive reaction develops in other cells, thanks to which the optimal level of the vital activity of the body in general can be maintained for a long time.

Age shifts occurring in the body due to the aging of cells and tissues, cause significant changes in the regulation of the organs and systems' functions. The functions themselves gradually change, which, in turn, leads to the structural changes. For example, during the aging, the mass of the brain decreases, the convolutions become thinner, respectively, the grooves widen. However, along with the dystrophic changes in the brain cells, adaptation changes occur, which are differently expressed in different brain regions.

The main manifestations of human aging are associated with age-related changes in the function of the central nervous system. First of all, the mobility of excitation and inhibitory processes weakens. It is harder for the acquired reflexes to develop, the nature of the inborn reflexes changes. The activity of the analyzers is impaired, the sensitivity and sense of smell are reduced, visual acuity decreases and the ability of the eyes to accommodate decreases, the upper audibility limit decreases. There are changes in the regulation of the internal environment of the body, the failures occur more often.

There is a decrease in mental activity, in which perception becomes difficult, its capacity decreases, attention concentrating decreases as well, emotional instability develops. Anxiety, negative emotions (irritability, temper, tearfulness, uncertainty, greed, indifference) develop. Often anxiety is caused by the condition of health. Anxiety turns into fear, and is replaced by despondency and hopelessness. Such negative emotions dramatically reduce the vitality, which in turn increases the manifestation of the old age.

Knowledge of the peculiarities of the psyche of the elderly and senile people, understanding of the mechanism of mental changes caused by aging, should make the basis of medical staff approach to patients of older age groups and should be taken into account when providing the medical care. Sometimes it is not the somatic pathology, but negative social factors (changes in work and life stereotypes, family conflicts, etc.), which are the reason that an elderly person feels sick. Therefore, it is especially important to pay attention to the mental status of the patients in older age groups. Any stressful situation can cause a series of shifts in their condition of health, causing aggravation of latent pathological processes, and the development of serious conditions. A good advice, help in changing the lifestyle, improvment of the relationship with the close people are often the main factors that contribute to better health condition.

Age-related changes in the cardiovascular system are largely the main reason why an aging organism is limitated in its adaptive capacity. Over the age of 60, the heart mass is reduced. In elderly people and specifically in the old people, the heart rate at rest decreases. On moderate physical exertion, an increase in the heart rate, which is specifically attributed to the young people, usually does not occur in the elderly people, which, on the one hand, protects the heart from excessive tension, though on the other hand, it limits the blood supply to the body.

With a sharp increase in the heart rate, which is associated with heavy physical exertion, there is a mismatch betweenthe blood flow to the heart through the coronary vessels and a sharp increase of the processes in the heart, which leads to insufficient blood supply to the heart muscle.

Clinical features of the course of diseases in the elderly people

First, it is necessary to take into account the multiplicity of the pathology that is common for the elderly and senile age. As a rule, during the careful examination of patients of this age, pathological processes are found in various systems of the body, which creates additional difficulties in treatment and worsens the prognosis. They are often caused by various factors,which are usually closely related to the changes in the nature of the age. In people of old age from a variety of

pathologies they may have, more than 6/7 of the whole ammount are not easy to detect. Complaints of the patients are associated only with a part of this amount, while in order to properly heal, one must know the pathology of the elderly and the senile people. This requires a wide range of the doctor's knowledge, the doctor is to understand the clinical features of the course of many somatic diseases in people of elderly and senile age, as well as the peculiarities of emergency care, the doctor should know the symptoms of the main diseases.

The peple of elderly and senile age may have diseases that developed in their youth or in the adulthood. Mainly, this concerns some inflammatory or metabolic processes as well as stable disorders in the functioning of any organ with a long-term chronic course. Like in young people, they may develop acute conditions, including infectious diseases. But very often acute diseases may become chronic. Many common diseases can occur in the elderly people insidiously, with no clear clinical symptoms, but, at the same time, they may have a tendency to develop severe complications. Thus, infectious and inflammatory diseases often occur without a pronounced temperature, which can be explained by the reduced reactivity of the body. Pneumonia, tuberculosis and diabetes mellitus development is often of slow and hidden character.

Gastric ulcers are of a specific origine – they are asymptomatic and manifest suddenly, with gastrointestinal bleeding. Acute surgical diseases of the abdominal organs that require urgent surgery are sometimes paucisymptomatic. The process of neoplasms' development is slow.

These clinical features often make it difficult to timely diagnose and lead to late treatment, especially when the elderly patient himself cannot clearly draw the line between the health and the illness.

In contrast to the disease etiology in young people, it is usually hidden in the elderly patients, and it is often caused endogenically; it is the result of the disease climax, and is associated with a number of overlaping causes.

In elderly, and especially in the senile age, the morbidity pattern changes significantly due to a decrease in the number of acute diseases and an increase in the number of diseases associated with the progression of chronic pathological processes. The most common diseases are: the ischemic heart disease, arterial hypertension, vascular lesions of the brain, chronic nonspecific lung diseases, late stages of diabetes mellitus, neoplasms.

Clinical features and the course of a disease in people of older age groups do not alway make it possible for the doctor to use the common diagnostic schemes. Diagnostics, as well as treatment, in people of elderly and senile age (often from the age of 60), require approaches, different from those that are used for younger people. The combination of several diseases often makes it impossible to conduct a full examination. So, due to the concomitant diseases (for example, severe hypertension, cardiac arrhythmia, history of transmural myocardial infarction) it is sometimes not possible to carry out the necessary endoscopic examination of the digestive tract organs.The inability of the patient to keep the barium enema makes it impossible to carry out irrigography, etc. When observing the elderly patients, and when assessing their condition, it is necessary to take into account not only the degree of the deviation from the age norms, but also the irregularities of the stages and the tempo of the development of involution and degenerative-dystrophic changes that are observed in case of presenilation, should be accounted, as well as the development of new adaptation mechanisms in various systems of the body.

Cardio-vascular diseases

Ischemic heart disease. The cardiovascular system is usually the first to undergo changes. Angina pectoris and myocardial infarction discrimination in the elderly and especially senile people is difficult due to the peculiar course of ischemic heart disease. At this age, *the painless form* of ischemic heart disease is much more common. The equivalent of the pain is often the *paroxysmal dyspnea*. The absence of the pain complaints in some cases may be caused by the changes in the mental sphere.

The gastralgic form of myocardial infarction, which manifests itself in young people by the "abdominal cramps", in the olderly people, and especially the senile people, is usually characterized only with the discomfort in the epigastric region or in the lower abdomen, sometimes with the urge to urinate. In case of the localization of the pain in the chest, neck and especially in the shoulder joint, it

is necessary to take into account the possibility of impaired coronary circulation, which cannot be excluded even if nitroglycerin is ineffective. *Therefore, in the elderly and senile age, the results of ECG (!) is of a more diagnostic importance than in the middle-aged patients.*

The onset of acute myocardial infarction in people of older age groups is often stroke-like, which is mainly due to the age-related changes in the blood supply to the brain. When analyzing the signs of myocardial infarction, it should be borne in mind that the temperature response in elderly and especially senile patients is usually less pronounced or is often completely absent. They have significantly changed blood reaction: leukocytosis, increased ESR.

Myocardial infarction in elderly and old people is much more often complicated by ***cardiac rhythm disorders*** *(atrial fibrillation, extrasystole), acute cardiovascular insufficiency*. Heart failure (mainly of the left ventricular type) occurs more often, and in the post-infarction period cardiac decompensation may also develop.

Arterial hypertension. Severe arterial hypertension in people over the age of 60-65 is rarely observed, since patients with rapidly developing hypertension do not live to that age, they often develop either a stroke or myocardial infarction.

Hypertensive heart disease, which develops in late age periods, is relatively *paucisymptomatic*. There are complaints of general weakness, tinnitus, gait instability (caused primarily by impaired blood supply to the brain due to atherosclerosis) and very rarely – complaints of headaches that are typical for the hypertension. Hypertensive crises occur infrequently and they are less pronounced than in middle-aged people.

Antihypertensive therapy for the patients over 60-65 years old is prescribed, as a rule, with the blood pressure of above 160/65 mm Hg, and if there is shortness of breath or symptoms of coronary insufficiency. In case of severe arterial hypertension, the blood pressure should be lowered with the help of antihypertensive drugs very carefully, so as not to cause a sudden change in the blood supply to the vital systems of the body.

Heart rhythm disturbances that occur within the aging period, and especially if atherosclerosis develops, usually is manifested by general weakness, fatigue, anxiety, insecurity (particularly when walking) and other symptoms, which the patient himself and the people around him consider to be just a manifestation of the old age but not the signs of a heart disease.

During the treatment of arrhythmia in elderly and senile people, pharmacotherapy has its own features (administration of Novocainamide (Procainamide Hydrochloride) is not recommended due to the possibility of severe cardiac conduction disorder and a sharp decrease of the blood pressure, Quinidin and other medication should be carefully used).

Heart failure in the olderly and senile people usually develops gradually. Absence of clinically significant symptoms in people of older age groups can be deceitful and is often caused by the hypotension due to general weakness, reduced visual acuity, and damage to the musculoskeletal system. The development and clinical course of the heart failure in elderly people largely depends on the severity of cardiosclerosis, changes in the blood supply to the central nervous system, lungs, peripheral circulation, the state of the neuroregulatory apparatus and the endocrine system.

Signs of cerebral ischemia, due to a decrease in stroke volume, often occur much earlier than congestion in other organs and systems. Sleep disorders, general fatigue, dizziness, and noise in the ears indicate decrease in cerebral blood flow. Confusion, agitation and motor restlessness, aggravated at night and often accompanied by insomnia, may present the early symptoms of cerebral circulatory failure associated with low cardiac output.

The early manifestation of left ventricular insufficiency and stagnation may be a weak cough, which often occurs during exercise, when changing from vertical to horizontal position. *The signs of dyspnea on physical excurtion is considered to be one of the earliest symptoms of heart decompensation, which is under development.* Dyspnea, when being calm, especially paroxysmal, which occurs in patients at night, is always considered to be pathological. Right ventricular circulatory failure may not first manifest itself with obvious dyspeptic disorders, but with edema of the feet and legs as well. Though swelling of the feet and legs should be differentiated from the defeat of the veins or damage to the joints.

Respiratory tract diseases

There are also some peculiarities with respiratory tract diseases in people of older age groups. The older the patient is, the harder it is to recognise pneumonia. Temperature reaction in most patients is mild or absent. Stabbing chest pains and chills are also frequent. The clinical picture is dominated by symptoms of a general nature: loss of appetite, weakness and apathy. Many patients develop impaired orientation; there are some other manifestations of intoxication. The auscultation gives miserable and indistinct results, shortening of percussion sound and the intensity of pectoral fremitus may not be registered.

Diagnostics of pneumonia in older people should be based on the manifestations of common symptoms (cyanosis of the skin of the face and lips, accelerated shallow breathing, weakness, apathy), the presence of previous acute respiratory viral infections, and most importantly – results of X-ray examination and clinical blood analysis (leukocytosis, accelerated ESR). Reduced immunological reactivity, chronic bronchitis (especially in smokers), obstructive emphysema, changes in the vascular system of the lungs contribute to the lingering course of pneumonia, and development of the chronic form.

The choice of antibacterial therapy for pneumonia in people of elderly and senile age is difficult due to the decrease of the excretory function of the kidneys and drugs' metabolism in the liver. Toxic antibacterial drugs should be avoided, taking into account the poor tolerability of sulfanilamides by the elderly people.

Antibacterial therapy should be combined with respiratory analeptics, cardiac glycosides, if necessary – with antiarrhythmic and antianginal drugs. Broncholytic means, herbal medicine, vitamins, physical methods of treatment should be widely used.

Digestive tract diseases

Diseases of the gastrointestinal tract in the elderly and senile age are more often functional in nature as a result of the age changes in the digestive system. Gastric ulcer that occurs in old age (the so-called "senile ulcer"), in most cases is symptomatic and is most often predetermined by trophic disorders in the gastric mucosa. These disorders are associated with anti-sclerotic changes in the vascular system of the stomach, which leads to a decrease in its blood supply, which results in the decrease of the intensity of biochemical processes.

Although *aggravation of peptic ulcer in elderly patients occurs more often than in middle-aged people, and complications arise more often (rupters, bleeding).* At this age, the risk of *malignant degeneration of the stomach ulcers* increases. The frequency and intensity of exacerbation of the duodenal ulcer usually decreases with age. The frequency rate of gallstone disease (especially in women), pancreatitis, chronic cholitis, hemorrhoids increases with age.

Abdominal cavity organs are not sensitive to many stimuli that, when exposed to the skin, provoke severe pain. Paresis of the intestine, rupters or other damage to the internal organs of the abdominal cavity do not cause pain. The stimuli, to which visceral pain fibers are sensitive, are: expressed distention or damage of the intestinal wall.These can be: tension of the peritoneum (i.e., with tumor), distention of a hollow organ (e.g., in case of a biliary colic), or strong muscle contractions (e.g., in case of the intestinal obstruction). The nerve fibers, which are responsible for the pain in the hollow organs (the intestines, gallbladder and the bladder), are localized in the muscular layers. In the parenchymal organs (the liver, kidneys and the spleen) the nerve endings are inside their capsules and respond to its tension with an increase of the volume of the organ. The mesentery, the parietal peritoneum and the parietal layer of the posterior wall of the abdominal cavity are pain-sensitive, whereas the visceral peritoneum and the greater omentum – are not.

To feel the pain, the level of the increased tension should be high enough. The gradual increase in tension, e.g., like in case of tumor obstruction of the biliary tract, can procede for a long time without pain.

Inflammation and ischemia can also cause visceral pain, at that, inflammation can increase the sensitivity of nerve endings and reduce the threshold of sensitivity when other stimuli cause pain (as well as, during the development of the disease in its different stages). Many biologically active

substances (bradykinin, serotonin, histamine, prostaglandins, etc.) are involved in the mechanisms of the onset and progressing of the pain during inflammation.

The briefly listed pathophysiological mechanisms of the formation of the abdominal pain create a problem, which often leads to a late or, in the best case, delayed diagnosis. That is why it is always important and useful to refer to this problem anew.

Clinical manifestation of acute surgical diseases of abdominal cavity organs in elderly people

Acute appendicitis. In the elderly and senile age, destructive forms of acute appendicitis (AAp) predominate. This is caused, on the one hand, by the reduced reactivity of the body, and, on the other, by the atherosclerotic damage of vessels, which is the direct cause of the rapid disruption of blood supply and the development of necrosis and gangrene of the vermiform process. It is in people of advanced age that the so-called primary gangrenous appendicitis occurs, which develops immediately, omitting the stage of catarrhal and phlegmonous inflammation.

Clinical manifestations of the disease depend on the level of inflammatory changes in the vermiform process, but the clinical findings of acute appendicitis in elderly patients do not respond to pathological changes in the vermiform process more than in young people.

Symptom complex of Acute appendicitis (AAp) in patients of this group often has *an unclear picture*. In elderly people, often with destructive appendicitis, the overall situation remains satisfactory. The body temperature, even with the destructive appendicitis, increases moderately or remains normal. The pulse increases and does not always respond to the temperature.

Independent pains in the abdomen, even in the presence of the destructive forms of appendicitis, are moderate or weak, and are more often diffuse in nature, and are less often clearly localized in the right iliac region. As a result of the physiological increase in the pain threshold in the old age, patients often do not pay attention to the abdominal pain in the epigastric phase at the onset of the disease.

Nausea and vomiting occur more often than in middle-aged people, which is associated with the rapid development of the destructive process. Stool retention is not critical, since there is a physiological tendency for the delayed bowel evacuation at an older age.

On *objective examination of the abdomen* expressed flatulence much more commonly occurs in the elderly people than among the young people, especially with destructive forms of the disease, as a result of intestinal paresis and dynamic intestinal obstruction; they show only moderate pain in the right iliac region, even in case of the destructive appendicitis.

One of the most important and early symptoms of acute appendicitis(AAp) is when the *tension of the muscles of the abdominal wall* is insignificant or absent as a result of relaxation of the damaged muscles of the abdominal wall, due to the age; Schotkin-Blumberg symptom in half of all the cases is not obvious. "Appendicular" symptoms (*symptoms of Voskresensky, Vitkovsky,* etc.) are often questionable or negative, *the most permanent positive symptom is the one of Razdolsky*.

Table 17

Differential diagnosis of (RO) acute appendicitis with other diseases

Diseases	Similarities	Differences
Right-sided adnexitis	Lower abdominal pain on the right side, fever, leukocytosis	Pain is more obvious in the groin and above the womb. Abundant mucopurulent discharge from the vagina is observed. Gynecological history: often – previous abortion, body temperature response is often above 38°C. The most common examinations are: digital rectal investigation and finger vaginal examination.
Ovarian apoplexy or ectopic pregnancy	Acute abdominal pain, tachycardia, positive symptoms of peritoneal irritation	Pain occurs in the middle of the menstrual cycle or when it is disturbed. The pain is accompanied by dizziness and weakness. Pain radiates to the anus and sacrum. The abdomen is soft, but painful in the lower parts. Anemia and the lowered pressure are registered. The results of the vaginal and rectal examination: on

		finger (digital) examination – painful overhanging of the posterior vaginal vault or the anterior wall of the rectum, blood, puncture the posterior vaginal vault.
Right-sided renal colic	Pain in the right iliac region, vomiting, painful palpation in the right iliac region	The pain is acute and cramp-like, it radiates to the right inguinal region, genitals, right thigh. The abdomen is soft, painless, negative "appendicular" symptoms. The pulse, body temperature are normal, leukocytosis. The urine contains a large amount of fresh erythrocytes. A concrement in the right kidney or ureter is diagnosed by the ultrasound examination.

In the elderly and senile people, *appendicular infiltration occurs more often than in the middle-aged people*, which is characterized by a slow development. Patients often notice a tumor-like formation in the right iliac region a few days after the attack of non-intense pain, which makes us pay particular attention to the differential diagnosis between the appendicular infiltrate and the cecum tumor.

Due to the reduced reactivity of the body, an increase in the number of leukocytes in blood is not always observed even with severe forms of appendicitis, or they are slightly increased, within (10-12) $\times 10^9$ / l, but the *neutrophilic shift is usually obvious*.

All this makes it difficult to diagnose AAp in the elderly and senile patients. At the same time, it is very dangerous to underestimate the mild symptoms of AAp, which results in late surgical treatment in case of advanced destructive phenomena in the process.

Treatment. *The peculiarity of the clinical course of AAp in older people is that it is difficult to clearly recognize a clinical form of acute appendicitis before surgery. This suggests the need* ***for active surgical tactics****, moreover, the risk of appendectomy at old age is often exaggerated.*

When choosing the method of anesthesia, local anesthesia is preferred, especially in patients burdened with concomitant diseases of the respiratory and cardiovascular systems.

As blood circulation insufficiency and atherosclerosis are the common problems of the cardiovascular system of the elderly people, ECG is to be always performed and followed up by a therapist, who can prescribe cardiotropic drugs. Due to the fragility of the vessels, intravenous punctures and injections should be carried out carefully.

In people of elderly and senile age, emphysema and pulmonary pulmonary fibrosis are often observed. When preparing for an operation, the doctor should pay special attention to respiratory gymnastics, which is an important prophylactic measure of postoperative pneumonia, and the patient should avoid overcooling.

In elderly patients, certain changes occur in the liver and kidneys. In this regard, barbiturates are to be prescribed with great care. Any medication for people over 60 is prescribed in a reduced amount – $^1/_2$ -$^1/_3$ of the total dose for the adults. As for narcotic analgesics, it is better to introduce pantopon, promedol instead of morphine, since the latter causes suffocation.

In case the diagnos is indeterminate, *pararectal incision* is preferable, which, if necessary, can easily be continued up or down. In case of destructive appendicitis, it is advisable to leave microirrigator in the abdominal cavity to inject antibiotics in the postoperative period.

Acute cholecystitis, empyema and hydrocholecystis. Clinical manifestations. For acute cholecystitis (ACC) in elderly people, as well as in younger patients, localization of pain in the right upper abdomen (in the right hypochondrium), with or without symptoms of peritoneal irritation are typical; pain may radiate to the right supraclavicular region, under the right scapula, right shoulder; there are dyspeptic manifestations (nausea, vomiting).

On objective examination of the abdomen – the local pain is felt in the right hypochondrium with or without symptoms of peritoneal irritation; muscle tension is often absent; weakly positive “pathognomonic” symptoms of Kerr, Ortner, Murphy, Zakharyin, Lyakhovitsky, Myusi-Georgievsky. Subhepatic pervesicular infiltrate may be palpated.

In elderly patients, asymptomatic variants of the course of destructive forms of ACC are quite common.

With exacerbation of chronic cholecystitis and with blockage of the cystic duct that show no signs of inflammation, these symptoms may also be absent in the elderly people.

The same symptoms can also be demonstrated by the AAp (with a considerable length and "high subhepatic" placement of the (VP) vermiform process).

Diagnostics. Moderate leukocytosis with the left shift and an increase in ESR are detected.

The most reliable diagnostic technique in this situation is *ultrasound examination*, which reveals calculi in the gallbladder and shows the signs of its inflammation, as X-ray examination with acute and aggravating chronic processes is ineffective.

Treatment. Conservative therapy leads to subsidence of clinical manifestations of ACC in no more than 50% of patients.

Active surgical tactics for ACC allows us to shorten the time of hospital stay and improve the results of treatment. Early surgery, performed before the bacterial infection of the bile, prevents cholecystogenic changes in the liver and the development of decompensation of concomitant diseases. *Preference is given to mini-invasive intervention – laparoscopic cholecystectomy.* Postoperative mortality in early operations for ACC is not much different from those in the planned operations for chronic cholecystitis.

Diseases that begin with pain in the right hypochondrium and that are accompanied with the development of the obstructive jaundice:

a) acute and chronic calculous cholecystitis

b) choledocholithiasis;

c) acute or aggravating chronic pancreatitis;

d) progressive stenosis of the terminal portion of the common bile duct (stenosing papillitis or tubular stenosis of the common bile duct)

e) cancer of the gallbladder, common bile duct and pancreas;

f) liver diseases: acute and aggravating chronic hepatitis, cirrhosis, primary sclerosing cholangitis, primary and metastatic liver cancer.

The main symptom under consideration is ***obstructive jaundice****.*

Diagnostics. *Laboratory tests'* main objective is to detect ***alkaline phosphatase*** to confirm the suspicion of its mechanical nature.

Further research program that may clarify the nature and pathogenesis of jaundice should be structured as follows:

- *ultrasound examination*, which detects biliary hypertension – ductal and intrahepatic; often the level of the obstruction can also be determined; it can not only open the examination program, but also finish it;

- *endoscopy of the upper gastrointestinal tract* (descriptive signs of the damage to the pancreas, damage to MDP (Major duodenal papilla), tumors of the stomach and duodenum, parapapillary diverticulum with the sighns of diverticulitis can be obtained);

- *endoscopic retrograde cholepancreatography*, which allows not only to detect the signs of defects in the pancreatic, common and hepatic bile ducts (stones in them, other obturation pathology) that caused jaundice, but also to restore the natural passage of the bile into the duodenum due to *mini-invasive surgical procedure* that leads to relief of the biliary system – *endoscopic papillosphincterotomy (EPST);*

- *laparoscopy* has an advantage in the cases, when the previous methods have not specified the diagnosis and the clinical picture of the disease indicates rise in the acute process, so the operation becomes inevitable. With the help of laparoscopy, it is often possible not only to establish the level of obturation, but also to perform *minimally invasive surgeries*, that lead to the relief of the biliary system: *laparoscopic microcholecystostomy, peritoneal omental sac drainage*, etc. These *minimally invasive surgeries* are considered to be the first stage of radical operations aimed at eliminating the causes of the obstructive jaundice (and, sometimes, they may also be the final treatment).

Perforated ulcer. Clinical manifestations and diagnosis. Patients of more than 60 years old do not often have ulcerative history or it is short. When interviewing, you need to persistently look for the

information about the period that preceded the rupture: Was there a heartburn? Did the patient take soda or not?

The course of the disease and symptoms depend on the location of the rupture. The ruptured hole with the stomach ulcer is larger and is covered less often, than the ruptured hole with the duodenal ulcer.

X-ray examination is of great help in making a PU diagnosis in elderly people – it is *the plan radiography of the abdominal cavity*, which detects free gas in the form of a sickle under the right diaphragm dome above the liver, in the vertical position of the patient (*Jobert's spontaneous pneumoperitoneum symptom*).

Being in a serious condition, patients are examined when in lateroposition, on the side, when the free gas is located between the side wall of the abdomen and the internal organs (liver, spleen).

In the presence of pneumoperitoneum and characteristic symptoms, the diagnosis of perforated ulcer is undeniable. The absence of the radiological symptom does not always indicate the absence of perforation.

In such doubtful cases, especially *with atypical forms of the disease* that occur in the older people, *the pneumogastrography method* or *Hönelt's test* is often used (Nédelt, 1963): for example, when there is no air sickle under the dome of the diaphragm on the right survey film of the patient, who is in the horizontal position, from 1600 to 2000 ml of air is injected through the gastric tube, the X-ray photographs in the vertical position are to be repeated; as a result a sickle of air is detected.

When the free gas is not detected in the abdominal cavity after this test, either urgent endoscopy is performed, with insufflation of 200-300 ml of air into the crater of the ulcer and then the X-ray photographs are repeated, or the methods of *laparocentesis or laparoscopy* are used: turbid exudate, obtained during laparocentesis through the catheter, which has an acidic environment and gets blue colored when iodine is added (*Neimark's test*); or when the laparoscopy detects a perforation hole or fibrin deposits on the duodenum or on the stomach. All this supports the diagnosis of perforation ulcer.

Laboratory diagnosis of the perforated ulcers is of secondary importance. Clinical blood tests present leukocytosis, neutrophilia,left shift in leukocyte formula, accelerated ESR. With a severe course of the disease, the amount of hemoglobin decreases, the content of residual nitrogen, urea and creatinine increases. The electrolyte composition of the blood changes, the level of chlorides decreases, acidosis progresses. The urine tests detect the signs of kidney damage: protein, leukocytes, erythrocytes and hyaline casts.

Differential diagnostics of the perforated gastric and duodenal ulcers. In patients of elderly and senile age, perforated ulcers are difficult to differentiate from angina pectoris and myocardial infarction, acute cholecystitis, appendicitis, and intestinal obstruction.

With angina pectoris and myocardial infarction the abdominal pain is registered in the xiphoid process, but not throughout the epigastric region, unlike in case of the perforated ulcer; the patient does not complain of nausea and vomiting; pain in the upper abdomen is not pronounced; there is no tension, the symptoms of peritoneal irritation are negative.

Acute cholecystitis is more common in women, and perforated ulcer – in men. Severe pain in the right hypochondrium is characteristic of acute cholecystitis, the shock-like state is not observed, tension is less obvious, Shchotkin-Blumberg symptom is less obvious, and the temperature rises early. The patient, who suffers from the perforated ulcer, is immobile, though the one with acute cholecystitis – is restless. After vomiting, the patient with the perforated ulcer feels relief, which is not the case with cholecystitis. In acute cholecystitis, an increase in the gallbladder can be detected, which will be sharply painful on palpation.

With acute appendicitis, pain and tenderness in the epigastric region disappear, and 6 hours after the onset of the disease, the pain and tension of the muscles are felt only in the iliac region; in case of the perforated ulcer, muscles' tension and sharp pain at the onset of the disease are felt in the epigastrium. Six hours after the onset of the disease, they spread throughout the right abdomen (including *symptom de Kerven* in the right iliac region). The *Shchetkin-Blumberg symptom*, in acute

appendicitis, is determined only in the iliac region, in case with perforated ulcer – in the epigastrium and right abdomen.

Intestinal obstruction is specifically attributed to the cramping pains, which can be felt in any part of the abdomen (depending on the cause), with perforated ulcer – the pains are constant, sharp, at the beginning of the disease they are felt only in the epigastrium. In case of AIO (acute intestinal obstruction), the pathognomonic symptoms are determined: severe vomiting, when the disease is neglected, it gains the fecal character, there is stool and gas retention; abdominal distention occurs, it is characterized by asymmetry *(Valya's symptom), symptoms of Sklyarov's "splashing noise", Spasokukotsky's "falling drop" symptom, Loteisen's "dead stomach" symptom*, etc.

Treatment. The method of treatment of patients with perforated gastroduodenal ulcers is ***only surgical.*** *The purpose of the surgical intervention is to prevent the progression and treatment* of ***peritonitis*** *by the prevention of the communication of the stomach cavity with the abdominal cavity, careful sanitation and adequate drainage of the abdominal cavity should be carried out.*

About 40 methods, including their modifications, are used to treat perforated gastric and duodenal ulcers and are described in the world literature; while the surgical treatment of the duodenal and gastric ulcers must be differentiated. The time interval from the moment of perforation to the beginning of the surgical intervention is important.

The age of the patient and his health condition also play a certain role when *choosing a method of surgery* – the incidence of patient's risk during a surgery can be influenced by a concomitant somatic pathology and its degree.

More often *palliative urgent surgery is performed, such as: a common Ostrovsky's suturing of the perforated holes (punch-hole size), or elcoplasty with omentum pedicle, using Opel-Polikarpov method, with thorough sanitation and adequate drainage of the adominal cavity* in elderly patients with obvious comorbidity. This causes a severe general condition, even with timely admission to a hospital with local peritonitis, or, which is even worse – with diffuse or general peritonitis, as it aggravates the severity of the patient's general condition. With general peritonitis, in the presence of severe functional AIO (acute intestinal obstruction), *intubation of the small intestine* via apendico- or cecostoma, is also indicated (transnasal intubation is contraindicated to the elderly patients due to the high probability of the development of severe pneumonia and respiratory failure).

Elderly patients with PU (perforated ulcer) and with local peritonitis, if not being in a severe general condition, and who were timely reffered to a hospital, *may be urgently and radically operated on*. These are the surgeries, which are commonly performed: *ulcer excision with accompanying duodenal or gastroplasty, which are combined with selective vagotomy (bilateral selective vagotomy or Bourget technique*). And another is *stem vagotomy according to Exner* (which significantly reduces the duration of the operation, if compared with SPV – selective proximal vagotomy), with *one of the types of pyloroplasty, which are compulsory* (according to the method of Jada Horse and Finney and others) to prevent gastrostasis.

An important stage of the perforated ulcer operation is a *thorough lavage of the abdominal cavity* – aspiration and thorough swab in case of local and diffuse peritonitis, or adequate rinsing with antiseptic solutions, and by their aspiration with electric suction machine, with diffuse (general) peritonitis; which is followed by adequate drainage (by one glove-tube drainage – with local peritonitis, by three – with diffuse peritonitis, and four – with diffuse peritonitis). In case of a large amount of purulent exudate, treatment of diffuse purulent peritonitis is performed by lavage, intestinal intubation and Petrov's drainage of the abdominal cavity.

It should be remembered that patients of elderly and senile age, as well as the patients, who were referred to the hospital late, after the perforation, require the obligatory and appropriate short-term preoperative preparation, which is advisable to be carried out directly in the operating room.

Therapeutic measures in the postoperative period, should, first of all, be aimed at the control of peritonitis and prevention of pyoinflammatory complications: antibiotic therapy, deintoxication, forced diuresis, control of protein and electrolytic balance, etc.

Acute intestinal obstruction. Classification (A.A. Shalimov, 1977).

According to the mechanism of development.

1. *Dynamic* (functional - *due to disorders of the intestinal innervation*):
a) spastic;
b) paralytic.
2. *Mechanical*:
a) strangulation;
b) obturation;
3. *Mixed forms* of strangulation and obturation intestinal obstruction.
4. *Vascular* (intestinal infarction):
a) mesenteric venous thrombosis;
b) thrombosis and embolism of arteries (at present a *separate nosological form* is singled out – *acute disorder of the mesenteric circulation*).

Dynamic spastic AIO (Acute intestinal obstruction) develops with long-term chronic intoxication (lead, mercury, etc.), with diseases of the spinal cord (syringomyelia), with hysteria in women.

Dynamic paralytic AIO (Acute intestinal obstruction) develops with acute pancreatitis, with peritonitis, with closed abdominal trauma, with severe infections, after major surgeries, with treatment with ganglionic blocking agents, β- adrenoblockers in large doses, etc.

Mechanical AIO: *obturation obstruction* – obturation with a tumor, blockage with a foreign body, fecal stone or gallstone, ascaris, coprostasis; *strangulation obstruction* – adhesive obstruction, twisted bowel, closed-loop obstruction, constricted hernia; *mixed obstruction* – indigitation (often caused by bowel polyps, ulcerative scars, enterocleisis with foreign bodies – gallstones, bezoar, etc.).

Clinical manifestations. In the diagnosis of this group of patients, attention is driven to careful analysis of anamnestic data (including pharmaceutical history) and the general condition of the patient, especially of *vascular damages* in elderly and senile people, who suffer from heart and vascular diseases that lead to thrombosis and embolism of mesenteric vessels. An important diagnostic feature is fluid bloody excrements. This group of patients is the most prominent and practically understandable.

With obturation – the pain is cramping, with strangulation – the pain is cramping and constant (shock often occurs).

Special attention should be paid to *chronic (or partial)* intestinal obstruction, which is often an early symptom of *tumor obstruction* and requires very careful examination of the patient when colonoscopy is obligatory.

No less significant is the group with *paralytic drug obstruction*, and in this respect, the medical history is big importance, thus unnecessary surgical intervention may be avoided.

The diagnosis of intestinal obstruction is made on the basis of *anamnesis* (abdominal surgery, the "*small signs of Savitsky syndrome* "), *objective examination data* (abdominal distension, asymmetry, peristalsis visible to the eye, increased intestinal motility during the attacks of cramping pains, pathognomonic of Valya, Kivulya, Sklyarov, Spasokukotsky, Obukhovsky hospital symptoms, etc.) and *radiological data*.

Diagnostics. *X-ray diagnosis of acute intestinal obstruction*. As early as 6:00 after the onset of the disease, there are radiographic signs of intestinal obstruction. Pneumatosis of the small intestine is the initial symptom (normally gas is found only in the colon). Subsequently, fluid levels are determined in the intestines ("*Kloyber's bowls*"). The level of the fluid, which is localized only in the left hypochondrium, indicates a high level of obstruction. It is necessary to distinguish between *small and large intestines' levels*. With the *small intestine* levels, the horizontal dimensions prevail over the vertical ones. They are located in the centre of the abdomen and in the small pelvis; the folding of the mucous membrane (Frementhal's symptom) in the colon is visible. As to the *large intestine* levels, the vertical dimensions prevail over the horizontal ones, they are located peripherally in the abdomen; haustration is detected.

Radiocontrast studies with the supply of barium through the mouth are not appropriate in case of intestinal obstruction, this contributes to the complete obstruction of the narrowed segment of the intestine. Application of water-soluble contrast agents in case of obstruction is promoted by

sequestration of the fluid (all the opacifying agents are osmotically active), their use is possible only if they are administered through a nasointestinal probe with aspiration after the study.

An effective means of diagnosing colonic obstruction, and in most cases of its cause, is ***irrigoscopy***.

Colonoscopy is undesirable in case of colonic obstruction because it leads to the flow of air into the drive loop of the intestine and may contribute to the development of its perforation.

Treatment of acute intestinal obstruction begins with conservative therapy, which includes aspiration of gastric contents, siphon enema, bilateral perirenal blockade, intramuscular administration of prozerin, intravenous infusion of fluids. It should be remembered that only the clear introduction of crystalloid solutions may contribute to the sequestration of the fluid; it is necessary to introduce plasmoozinumenyh solutions and protein preparations in combination with crystalloids.

The effectiveness of the conservative treatment is determined by the clinical (disappearance of pain, a significant reduction in abdominal distention, discharge not only air, but also fecal masses with siphon enema) and x-ray data (disappearance of intestinal fluid levels).

If the pain and radiological signs of the disease remain unchanged, this indicates lack of the effect and dictates the need for ***urgent surgery***.

Principles of surgical treatment of acute intestinal obstruction.

1. Elimination of mechanical obstruction or creation of a by-pass for the intestinal contents. In case of the *small bowel obstruction*, it is necessary to strive to eliminate the cause of the obstruction (up to the bowel resection) with the application of the intestinal anastomosis. In case of the *colonic obstruction*, it is necessary to eliminate the cause of the obstruction, and, since the most common cause is obturating cancer, it is necessary to resect the intestine with the tumor in the indicated volume, when it is operable – to perform *radical surgery*. The imposition of the short-term inter-intestinal anastomoses after this makes it impossible to perform the stitches and may cause peritonitis; therefore, only for the right-sided localization of the colon tumor in young patients with unresponsive intestinal obstruction, one-stage right-sided hemicolectomy with ileotransveranastamosis is possible. In other cases, two- or three-stage surgeries are performed with low left-sided colic obstruction, (*Hartmann surgery technique*).

When the tumor that caused the AIO (acute intestinal obstruction) is inoperable in elderly patients, *a palliative operation is performed – the imposition of a permanent single or double-barrel colostomy (according to Maydl) proximal to the tumor.*

2. Removal of necrotic and suspected areas of the intestinal necrosis.

3. Unloading the dilated part of the intestine – this helps to restore the micro-circulation in the intestinal wall, and the tone of the muscle membrane and peristalsis. It is possible to achieve the unloading by nasogastric, gastro- or cecostomy intubation of the small intestine during the operation.

4. Before the operation, premedication with antibiotics is necessarily to be performed (daily dose of a broad-spectrum antibiotic is administered intravenously 30-40 minutes before the operation), which is advisable to be combined with metronidazole.

5. After the operation – detoxification therapy, correction of protein and water electrolyte disorders, stimulation of the motor-evacuation function of the gastrointestinal tract, prevention of thromboembolic are to be performed.

Hernia in the elderly people. Stomach hernias are extremely common in the elderly people. Every year more than 20 million operations are performed in the world, which makes from 10 to 21% of all surgical interventions. Every 3-5th inhabitant of the Earth is a potential hernia carrier. Now about 510-570 million people suffer from this disease. The peak incidence is observed in people older than 50 years. The number of these patients is 60-65% of the total number of hernias. This is due primarily to the increase in life expectancy and global aging of the population over the past decades.

Mortality in elderly patients after planned operations, especially for large hernias, is 7–20%, and after emergency operations it makes 25 – 40%. This is explained by the fact that the surgeons often do not take into account both the characteristics of the tissues of the abdominal wall (age-related morphological changes in the tissues) and the features of the aging body of the patient (the reserves of the cardiovascular and respiratory systems are sharply reduced). The situation is exacerbated in the

presence of a large number of comorbidities that aggravate the clinical course of the hernia. Concomitant diseases are observed in 86.6% of elderly and senile patients. They are hypertension, coronary heart disease, chronic bronchitis, varicose veins, diabetes, obesity, and others. More than 50% of these patients have two or more comorbidities.

Particularly difficult clinical situations arise with large hernias. They occur most often in patients of elderly and senile age. Even in the absence of pronounced complications, such as infringement or acute intestinal obstruction, the hernia itself leads patients to a profound disability. It is with such hernias that the patients have the greatest number of comorbidities, and the same patient often has 2-3 or more diseases.

Among patients with ***strangulated hernia***, the patients of elderly and senile age prevail. Complications and the highest mortality rate are most often observed in this group of patients. Postoperative complications after emergency interventions for hernias are also most often observed in patients of older age groups. Peritonitis, thromboembolism, pneumonia and other complications are associated with infection and the activity of vital systems. Mortality rate here reaches 45%. Currently, there are hundreds of different methods of surgical treatment of inguinal hernias and most of the methods have been used for many decades. But despite this, the effectiveness of treatment is far from ideal – in patients older than 50 years, the relapse rate reaches 15-30 %.

The achievement of the second half of the 20th century was the proclamation of *the principle of "tension free" plastic* to be the best treatment of inguinal hernias. This revived the extinct interest of surgeons in aloplasty and, largely due to the advances of polymer chemical technologies; this brought herniology to a new level of development. The principle of "tension free" plastic became the basis for the successful treatment of patients of elderly and senile age with inguinal and femoral hernia. Its compliance qualitatively changed the results of treatment of this most common category of patients: the frequency of hernia relapses decreased by an order of magnitude, the rate and usefulness of patient rehabilitation increased, the number of complications significantly decreased, hernioplasty was possible in those patients who had previously been denied surgical care for various reasons.

Acute pancreatitis in older people. Acute pancreatitis is among the most serious and dangerous diseases of the abdominal organs. Mortality rate in case of acute pancreatitis reaches 15-20% according to the World Congress of Gastroenterology. Among patients with acute pancreatitis, older people amount to 30-66%. The highest incidence of the disease occurs in people of 61-70 years of age. Among the patients, women predominate. All this suggests that with aging there is a number of factors that contribute to the development of this pathology.

The most common cause of acute pancreatitis in the elderly and old people is biliary tract disease. According to statistics, 60-80% of patients with acute pancreatitis, displayed a provisional or concomitant disease of the biliary tract.

The alimentary factor is of great importance in the development of this pathology in old age. Often the disease develops after an active consumption of proteins, especially fatty foods and alcohol. Substantial night food intake contributes to this.

These causes play a major role in the development of the disease in elderly people, but in the process of human aging, their role increases significantly. This is primarily *due to morphofunctional changes in the pancreas during aging*. It has been noted that age-related destruction of the ducts, blood and lymphatic vessels of the pancreas begins at the age period of after 30–40. In this regard, the development of periductal fibrosis, epithelial hyperplasia, up to the formation of mastoid processes directed inside the duct, obliteration of the ducts, decrease in the total mass of glandular parenchyma, and the development of adipose tissue deserve special attention. Up to 75-90 years of age, some of the particles are completely replaced by fatty tissue, and the total amount of functioning tissue sometimes decreases up to 30-40%. There is a definite sequence of morphological changes in the gland: first, vascular changes develop, then connective tissue and fatty tissue grows, after which atrophy of the gland developes.

In its turn, age-related changes in the structure of the secretory apparatus of the gland is one of the main reasons for the weakening of its exocrine function during aging.

Clinical manifestations. The most frequent symptom of the disease in older people is the *sudden pain attack in the epigastric region and in the left hypochondrium* when consuming large amounts of food, especially fatty or canned food. The pains are constricting, often engirdling.

At the same time, they can be so intense that some patients experience shock. The second most common symptom of the disease in elderly and senile people is *frequent, fierce vomiting in small portions ("in driblets")*, which, as a rule, does not improve the patient's condition. Vomiting is accompanied by pain in 80-90% of cases. The presence of bile in the emetic fluid indicates the patency of the common bile duct. In severe forms of acute pancreatitis, bloody vomiting may occur.

In elderly patients with acute pancreatitis, *intestinal paresis* is often observed. Despite pronounced pain, the abdomen is soft on palpation, there is only a slight muscle tension in the epigastric region. Such discrepancy is a characteristic sign of pancreatitis in older people. The tongue is usually dry and coated. Patients are restless. The skin of the face acquires a pale bluish tint, sometimes jaundice of the sclera and skin is noted. Breathing is rapid (28-30 per minute), pulse is rapid (100-140 beats per minute) and deficient, with low tension; blood pressure is often low.

However, in older and senile people, acute pancreatitis' *symptoms are, more often, not very pronounced and develop slowly*, if to be compared with younger people: slight epigastric pain are registered, they do not radiate to the back and lower back; there is no vomiting, the stomach is slightly swollen. Often there are pains in the region of the heart that are analogous to the picture of coronary insufficiency; sometimes the pain resembles renal or hepatic colic. In some cases, the pain is continuous, like the one with acute cholecystitis; sometimes it is colicky like, like with cholelithiasis or urolithiasis.

The more frequent *development of acute serous pancreatitis into necrotic and purulent* is characteristic of older people. The main criteria for such a change are: increased abdominal pain, signs of peritoneal irritation, body temperature rise up to 38–39 ° C, effusion into the abdominal and pleural cavities (the exudate contains pancreatic enzymes), increase of amylase level in the blood and urine, leukocytosis and increase in the neutrophilic left shift, the general condition worsens, intoxication is more pronounced, and, finally, shock may occur, which may often cause death. Besides, the temperature response in elderly patients is not very expressed, as well as the changes in the blood.

Quite often in elderly and senile patients, the relatively favorable course of acute pancreatitis is suddenly complicated by acute cardiovascular and hepato-renal insufficiency, cerebral coma, and intestinal obstruction.

Diagnostics. Laboratory research is of important diagnostic value in recognition of acute pancreatitis. One of the most persistent symptoms (in 75-90% of patients) is an increase of amylase level in blood (and urine) (more than 32 GM/DL), 2-4 hours after the onset of the attack. It is advisable to do the amylase test at the peak of the attack several times a day. However, in elderly and senile people, the amylase indices may be low with acute pancreatitis, and in case of pancreatic necrosis, when the patient's condition worsens, amylase indices in blood and urine may be within the normal limits or even low. This can be explained by the fact that the existing age-related decrease in the number of functioning pancreatic parenchyma is accompanied by destructive changes caused by the pathological process. This results in almost complete destruction of the acinar cells of the gland, which produce pancreatic enzymes. *Therefore, if hyperenzymemia and hyperenzymuria are not detected, this still does not exclude the presence of acute pancreatitis in older patients.*

During the blood study, *moderate leukocytosis with a neutrophilic left shift, eosinopenia, lymphopenia, monocytopenia, increased ESR, hypoproteinemia, and increase in the γ-globulin fraction* are detected. Often, *hyperglycemia and glycosuria* are detected in elderly patients, which is associated with the lack of endocrine function of the pancreas.

Differential diagnostics. Acute pancreatitis in elderly patients, is, first of all, to be differentiated with such diseases as myocardial infarction, acute cholecystitis, hepatic colic, perforated gastric ulcer, acute peritonitis, intestinal obstruction, acute appendicitis.

It should be emphasized that acute pancreatitis in older people is timely diagnosed in about 25% of cases due to its frequent atypism. Therefore, in case of any pain in the abdomen in the elderly and senile people, acute pancreatitis should be considered.

Treatment. Therapy for acute pancreatitis should be carried out in a hospital and begun as soon as possible. It is necessary to create a physiologically dormant gland state: strict bed rest, fasting for 3-5 days, ice bag on the stomach. During fasting, physiological solution with 5% of glucose is titrateed intravenously, but not more than 1500-2000 ml per day, to control intoxication and dehydration. Electrolyte metabolism correction is performed, as it is often disturbed. With frequent vomiting and expressed gastric flatulence, it is advisable to pump off its contents with a thin probe introduced through the nose.

The 0,1% atropine solution (0.75 ml) is introduced subcutaneously 2-3 times to relieve pain and to supress pancreatic exocrine function; it is advisable to administer no-spa, papaverine, bencyclane (galidor) and other antispasmodics in combination with promedol, dimedrol. It is not recommended to prescribe morphine, as it can cause spasm of sphincter of Oddi and increase pain. Bilateral perirenal procaine block (50-80 ml of 0.25-0.5% solution) and administration of novocaine (5-10 ml of 0.5% solution) in physiological solution intravenously have a good therapeutic effect.

An important factor in the treatment of acute pancreatitis is the administration of drugs that suppress the activity of pancreatic *enzymes (their inhibitors – Trasylol (aprotinin), Contrykal (aprotinin), Gordox (aprotinin)).* Durin the first 3-4 days 25000-50000 units of the drug are introduced intravenously by drop infusion in 200 ml of saline solution per day, then the dosage is reduced to 10,000-20000 units per day during the week.

In case of shock, intravenous drop infusion of of 5% glucose solution (1.5-2 l) have to be performed; caffeine, ephedrine, camphor, cordiamine are to be introduced subcutaneously; 200-250 ml of plasma and plasma substitutes are infused daily. Since the first days of the disease, calcium gluconate or calcium chloride are to be administered intravenously. It is advisable to introduce steroid hormones in severe course of the edematous phase of acute pancreatitis. Antibiotics are prescribed to control the infection. In order to prevent thrombosis during the first days of the disease anticoagulant therapy is commonly performed.

In elderly and senile patients, ***life-saving surgery for acute pancreatitis is only performed to control the development of complications*** (peritonitis, abscess, bleeding, perforation, complicated pseudocyst). This is due to the fact that in patients of this age the existing changes in the cardiovascular and respiratory systems, metabolic disturbances, etc., the adaptive ability of the body is very limitted, which may cause the development of severe complications in the postoperative period (cardiovascular insufficiency, pneumonia, thromboembolism and others).

Acute pancreatitis is much more severe in older and senile people, with considerable risk of mortality (70% of all the deaths from acute pancreatitis occur in people after 50 years of age). The most common causes of death are: the development of cardiac, hepatic and renal failure and intestinal obstruction. A significant percentage belongs to focal pneumonia, thromboembolic complications, diabetes mellitus.

Thus, in elderly people, *acute abdominal pain and fever* may occur in cases of acute pneumonia, basal pleurisy, acute pericarditis, rheumatic carditis, thyrotoxic crisis, nodular nonsuppurative panniculitis (Weber-Christian disease - skin-visceral, visceral, abdominal variants), acute mesadenitis, pyelonephritis, shingles, hemorrhagic vasculitis, and inflammatory diseases of the abdominal organs. Acute abdominal pain and a drop of blood pressure are observed in case of acute myocardial infarction, acute pancreatitis, artery dissection of the abdominal aorta, acute adrenal insufficiency, gastric ulcer perforation, acute disorder of the mesenteric circulation, etc.

Each symptom complex gives the opportunity to limit the range of nosological entities, which makes it possible to make the diagnosis; as each symptom complex includes acute diseases of the abdominal organs, the research program should be designed with the aim to either confirm or reject them. At the same time, in all the cases, after a generally accepted clinical study of instrumental methods to be applyed, ***the investigation should begin with ultrasound***, which should be carried out as follows:

- “exploratory examination - superficial” (upper, middle, lower abdomen); it has to be tested if there are any pathologies of a gall bladder and pancreas, free fluid in the abdominal cavity; the diameter of the vessels of the inferior vena cava, the aorta are to be determined; the pathology of the

kidneys (stones, including the ureter, carbuncle kidneys) has either to be detected or rejected, the pathology of the uterus, appendages have to be established, as well as some other rare changes;

- considering the results of the exploratory examination, a detailed study of the abnormal focus should be carried out; in certain clinical situations some investigations should be carried out to detect abscesses, subhepatic and suprahepatic infiltrates; in case of jaundice – to establish its nature (mechanical, parenchymatous;) in case of hepatomegaly – to define or to deny its "congestive" character.

The second, most logical method of the research (if the diagnosis is still not clear) is "***emergency laparoscopy"***. This method makes it possible to perform the differential diagnosis of acute appendicitis, acute cholecystitis, perforated gastroduodenal ulcer, acute pancreatitis, bowel infarction, acute diseases of the pelvic organs. At the same time, in case of any medical reasons, *the drainage of the abdominal cavity, omental bursa and cholecystectomy* can be performed.

These two instrumental studies, as a rule, are sufficient to confirm or reject the "acute abdomen" diagnosis, to determine the pseudoabdominal syndrom (PAS) diagnosis, largely determine its cause and choose the most rational way for further diagnostics (ECG, EchoCG; X-ray, CT, special laboratory tests, serological, morphological studies – in specialized hospitals).

II.6. ACUTE DISORDERS OF THE MESENTERIC CIRCULATION

Acute disorders of mesenteric circulation with the development of bowel infarction - is a relatively rare disease. Bowel infarction is most often caused by the blockage of the superior mesenteric artery and its branches, due to embolism (60-90%) or acute thrombosis (10-30%). The main source of embolism are blood clots in case of rheumatic heart disease and atherosclerotic vascular disease, myocardial infarction, endocarditis, less often – in case of aortic thrombi. Acute thrombosis usually develops with the background of pathologically changed arteries due to atherosclerosis, nonspecific aortoarteriitis, fibrous-muscular hyperplasia, periarteritis nodosa, diabetes mellitus. Acute obstruction of the mesenteric arteries often (in 40-50% of patients) is preceded by prodromal symptoms of chronic insufficiency of the mesenteric circulation. This explains the fact why intestinal infarction occurs predominantly in elderly patients (average age is over 50).

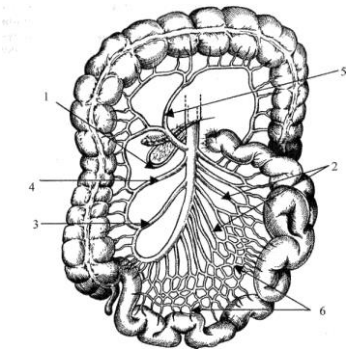
Acute occlusion of the mesenteric vessels often leads to intestinal infarction, as there is no time for collateral circulation to develop. The frequency and conditions for the development of bowel gangrene after the occlusion of the mesenteric vessels are not well understood, necrosis does not always develop. In the presence of the occlusion of only inferior mesenteric artery, bowel necrosis seldom occurs.

The intestinal infarction may be the result of not only acute arterial occlusion, but also – of venous thrombosis, a combination of arterial and venous occlusion, long spasm of the branches of the mesenteric artery. This pathology can develop in patients with severe heart failure, endotoxic shock, allergies, as a result of impaired microcirculation in the intestinal wall, and in young women – due to hormonal contraceptives**.**

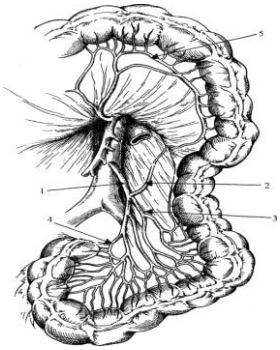
Acute intestinal ischemia causes progressive changes with underlying absence of collateral blood flow, which become irreversible after 2-4-6 hours. The viability of the bowel can be maintained in case the malperfusion disorders are eliminated during this period. However, even if the resection of the intestine is not indicated, the effects of ischemic infarction in the form of fibrosis with impaired absorption and motor-secretory functions of the intestine remain, presenting normal macroscopic picture.

The blood supply of the small and large intestine (colon) is carried out by ***the branches of the superior and inferior mesenteric arteries*** (Pictures 46, 47).

The causes of the disorders of visceral circulation can be varied. The nature and degree of the dyscirculation depend on the etiology of the damage of the visceral branches of the aorta. In this regard, several *classifications of acute mesenteric circulation disorders* were proposed.



Picture 46. Superior mesenteric artery and its branches:
1 - inferior pancreaticoduodenal;
2 - branches to the jejunum and ileum;
3 - iliocolic artery;
4 - right colic artery;
5 - middle colic artery;



Picture 47. Inferior mesenteric artery and its branches:
1 - inferior mesenteric artery;
2 - left colic artery;
3 - sigmoid arteries;
4 - superior rectal artery;
5 - Riolan arc

Classification of the disorders of mesenteric circulation.

I *According to the etiology*.

1). Occlusive: embolism of arteries, arterial thrombosis, venous thrombosis.

2). Non-occlusive

II. *According to the progression:*

1) with compensation;

2) with subcompensation;

3) with circulatory decompensation

In case of mesenteric circulation compensation, all the functions of the intestine are fully restored without any consequences. Recovery of patients occurs spontaneously or under the influence of conservative therapy. Subcompensation of the mesenteric circulation causes a number of bowel disorders associated with insufficient blood supply: abdominal angina, ulcers of the intestine, enteritis and colitis (often ulcerous). In the future, patients may suffer from various complications: bleeding, perforation, the intestinal wall phlegmon, intestinal stenosis. Mesenteric blood circulation decompensation leads to the formation of intestinal infarction, extensive purulent peritonitis, and severe abdominal surgical sepsis. It is with such a course of the disease the emergency abdominal surgery deals with most often.

Stages of acute mesenteric obstruction.

Stage I – the initial stage (duration up to 7 hours). The clinical picture presents a dominating *triad of symptoms*: abdominal pain, shock and diarrhea. There is a characteristic discrepancy between the severe general condition of the patient and relatively not very pronounced changes that are revealed during the examination of the abdomen: distension and moderate pain without symptoms of peritoneal irritation. On auscultation – impaired intestinal motility. The blood picture is not changed. On X-ray examination pneumatization and small-bowel wall thickening are detected.

Stage II (7-12 h). Severe pain in the abdomen, on palpation there is an increase in pain, but there are no peritoneal symptoms, the patient's condition gradually becomes worse. On finger rectum examination the bloody flux may appear. The blood examination may present an increase in leukocytosis, there are radiographic changes.

Stage III – the stage of bowel necrosis (after 12 h). There are the symptoms of diffuse peritonitis and paralytic intestinal obstruction, high level of leukocytosis; on x-ray examination of the abdominal organs – multiple fluid levels.

Clinical manifestations *of acute mesenteric arteries obstruction* are, in many ways, similar to the syndrome of acute abdomen of a different etiology. Acutely progressive course is very characteristic, especially in the initial period, and, at the same time, there is scanty objective data.

During the interview, the patient may complain on:

- the abdominal pain – the first and the main symptom, which occurs suddenly, the pain is severe, usually constant, in paraumbilical zone; then it spreads throughout the abdomen; it often radiates to the back; in a few hours the pain may disappear or decrease for a while (latent period);
- abdominal distention, during the first hours of the onset of the disease nausea, vomiting (sometimes with blood), pallor, sweating, Trelat's stool (bloody stool with mucus) occur.

Disease history. With severe development of acute abdomen syndrome in elderly and senile patients with heart defects, myocardial infarction, and severe atherosclerosis, it is necessary first to exclude the mesenteric arteries obstruction.

Objectively. The general condition, as a rule, is severe, there may be a suffering expression on the face, cold sweat, pallor with acrocyanosis, tachycardia, hypotension. The temperature rises only in some patients.

Status localis. On examination and palpation of the abdomen – detention of varying degrees, the lag when breathing. Pain is more often felt to the left of the navel. The protective tension of the abdominal muscles is very often absent, at first, but there is always a sharp sensitivity on superficial palpation of the abdomen. Tension (muscular defense) occurs in the later periods during the development of peritonitis. Peristalsis is usually sharply decreased.

After 3-12 (24) hours from the onset of the disease, on a slight pain releaf, paralytic intestinal obstruction develops, peristalsis fades away; there is vivid abdominal distention and general condition is severe; endotoxic shock occurs caused by a decrease in circulating fluid volume and intoxication. In the final stage of peritonitis which is specifically attributed to a common clinical picture of paralytic intestinal obstruction, accompanied with severe intoxication, and when the diagnosis of acute mesenteric obstruction is made as well as when laparotomy is often performed too late, the changes in the intestine are usually irreversible.

The clinical picture described above is typical for patients with acute obstruction in *the superior mesenteric artery* territory.

Acute occlusion of the *ventral trunk* (stem) is rarely observed and is specifically attributed to the acute pain in the upper abdomen, back, right hypochondrium, early nausea, and bloody vomiting. The disease must be differentiated from the perforated gastric ulcer, acute pancreatitis, cholecystitis, myocardial infarction, which occurs with the syndrome of pseudoabdominal angina.

Early and timely diagnostics of the disease is difficult to be made even in typical cases. Approximately 6% of patients are diagnosed in a timely manner. The difficulties in diagnosis, on the one hand, are caused by the rarity of the disease, and on the other hand, by a very short period from the onset of intestinal infarction – from the very first clinical symptoms up to the development of irreversible changes in the bowel.

In case of acute thrombosis of the mesenteric vessels, which develops with the background of chronic insufficiency of the mesenteric circulation, the development of clinical symptoms is more gradual. In this case, irreversible changes in the bowel occur at a later period, which creates favorable opportunities for the timely diagnosis and surgical treatment of the disease.

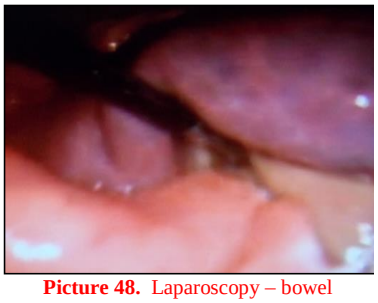
Laboratory and instrumental diagnostics. *High level of leukocytosis* ($15\text{-}20\times10^9$/l), which is determined in patients within a few hours from the onset of the disease with the background of severe pain syndrome and scanty objective data, is indicative and permanent.

X-ray examination at an early stage does not usually show pathological changes (unlike mechanical, strangulated intestinal obstruction, perforative ulcer), and in the late stage, there is a picture of paralytic intestinal obstruction, that is, non-pathognomonic signs that occur with any intestinal obstruction, regadless its etiology.

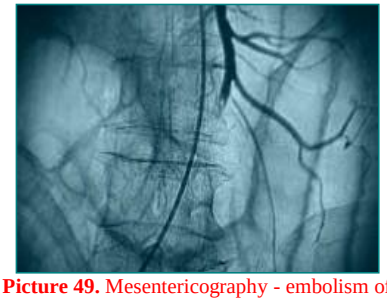
The modern methods of early diagnostics of intestinal infarction include *angiography and laparoscopy.*

During *laparoscopy* (Picture 48), the diagnosis of bowel infarction is made due to the change of the color of the intestine, presence of hemorrhagic effusion, mesentery edema. Only angiography makes it possible to make an accurate early diagnosis.

Abdominal aorto-arteriography and selective angiography (Picture 49), performed by way of retrograde aortic catheterization through the femoral artery or translumbar puncture aortography, make it possible to make a diagnosis in the first 5-6 hours. When the patient is in a serious condition, in a late stage of the disease, it is impractical to use angiography, so urgent laparotomy occurs to be more sufficient.



Picture 48. Laparoscopy – bowel infarction



Picture 49. Mesentericography - embolism of the superior mesenteric artery

During the differential diagnostics the following peculiarities must be considered.

1. The presence of ***possible reasons for embolism*** (atherosclerosis, cardiosclerosis, myocardial infarction, cardiac malformation, etc.).
2. Sudden onset of abdominal pain, usually severe, woth no certain localization.
3. The discrepancy between the severe painful abdominal syndrome, severe general condition (endotoxic shock) and scarce data on clinical and radiological examination.
4. High level of leukocytosis in the early stage of the disease.
5. Bloody stool and vomiting.

It is extremely difficult to differentiate ***acute arterial and venous thrombosis of the mesenteric vessels.*** Venous thrombosis of other localization, signs of portal hypertension, tumors of the pancreato-duodenal zone are indicative of *venous thrombosis*.

Diseases, which to be differentiated with ***acute abdominal ischemia***, are very diverse. Primarily, these are the diseases of the digestive tract organs, which are clinically manifested by abdominal pains and intestinal dysfunction. In this regard, it is necessary to conduct a thorough clinical examination of patients using modern methods of investigation.

There are several reasons for not timly diagnosis. The main reason is lack of doctors' apprehensive attitude to abdominal ischemia, as this pathology is very rare. Moreover, like any othe acute disease, abdominal ischemia has its own specific features of development, which are not quite typical in their clinical manifestations in the initial stages. This explains the history of the disease, as well as the fact that the correct diagnosis is made, as a rule, in the decompensation stage only.

It should be emphasized that the fact of acute abdominal ischemia does not exclude the possibility of a concomitant disease of the abdominal cavity. This circumstance requires careful analysis and critical evaluation of the clinical picture, as well as of the laboratory investigations.

Acute abdominal ischemia should be differentiated from ***acute pancreatitis***. Differential diagnostics presents certain difficulties here. Both pathologies are clinically manifested by pain in the epigastric region, similar to intestinal dysfunction. In acute pancreatitis, at the beginning of an attack, patients are restless, " dash " in bed, then prefer to lie on their backs and do not move actively in order to avoid an increase in pain. Pain, in most cases, is acute, belt-like, can often be controlled by antispasmodics. Drug use brings only temporary relief. The pain lasts much longer and does not at all coincide with the cycle of digestion; it is often accompanied by vomiting, which does not bring relief. There is specific irradiation of the pain, which spreads to the shoulder, shoulder girdle, costovertebral angle (Mayo-Robson's symptom) during pancreatitis exacerbation.

Tongue is furred and dry. The distention of the upper abdomen may be noted (*Gobiet symptom*), there is pain in the supraumbilical region *(Corte symptom*). As the inflammatory process spreads to the tissues that surround the pancreas and the peritoneum, pain occurs in the hypochondria; protective tension of the muscles of the anterior abdominal wall develop and the *Shchetkin-Blumberg symptom can be positive.*

On exacerbation of pancreatitis, peristalsis of the intestine is sluggish or not heard. In case of pronounced swelling of the gland, the pulsation of the abdominal aorta may not be detected (*Voskresensky symptom*). In case of chronic abdominal ischemia, intestinal peristalsis is usually vivid, and the pulsation of the aorta is usually unchanged.

The level of leukocytosis is high, amylase is increased, diastase and other laboratory data add to the clinical picture.

There are certain difficulties in differential diagnosis with ***acute appendicitis***.

As a rule, the disease begins with a sudden pain attack, sometimes immediately in the right iliac region, but, more often, first it occurs in the epigastric region and is localized in the right iliac fossa only a few hours later.

In case of a common form of inflammation, the pain attack retains its intensity for several days; in case of a destructive form, it is more pronounced at first and decreases as the destruction develops until it completely disappears for some time (necrosis of the receptor zone). Especially intense pains that cause the patient to "dash" in bed, are characteristic of empyema of the cecal appendage.

Non-destructive inflammation is characterized by the absence of a general reaction – normal temperature, no vomiting, etc. The destructive form, on the contrary, is accompanied by rise in the temperature of up to 38°C, vomiting that occurs after the onset of pain, stool retention (very often) or diarrhea (less often). An increase in leukocyte reaction and alkaline phosphatase activity in peripheral blood leukocytes are indicative.

In typical cases, right ileal fossa is painful on palpation, there is local muscle tension and symptom of peritoneal irritation is positive.

In some cases, the disease develops atypically. For example, an attack begins with indomitable vomiting, diarrhea, and abdominal pain without typical localization. The temperature may remain normal, but may rise above 38°C. On palpation of the anterior abdominal wall, the symptom of inflammation is expressed throughout, but not sharply. In other cases, a sharp painful attack is the only complaint of the patient.

The close and bonded reflex link of the cecal appendage and the cecum with the stomach and duodenum in some cases explains the similarity of symptoms with chronic abdominal ischemia, gastric ulcer, duodenal ulcer and other diseases of the abdominal cavity.

The atypical symptoms of acute appendicitis can be explained by the location of the vermiform process. For example, in case of the retroperitoneal location of the organ, abdominal symptoms are absent and the clinical picture is similar to renal colic. In case of the subhepatic arrangement of the vermiform process, the pain is felt in the right hypochondrium. However, without characteristic irradiation in the supraclavicular region, the symptoms of cholecystitis are questionable. In case of pelvic localization of the vermiform process, the pain is localized in the suprapubic area. There are the symptoms of pelvic organs damage – rectum (loose stool 2-4 times without pathological impurities), bladder (frequent urination, but without dysuric phenomena and without significant changes in urine analysis), the first plan symptoms in women are those of the genitals. We should not forget about the left-sided arrangement of the vermiform process.

The course depends on the form of the disease. Non-destructive inflammation is usually not dangerous for the patient. Pains persist for several hours or days and are reduced either spontaneously or under the influence of therapy. It is this form that has clinical symptoms that are similar to chronic abdominal ischemia.

Destructive forms of acute appendicitis are always dangerous, because the development of the pathological process often proceeds quickly and leads to various complications – the formation of infiltration, periappendicular abscess, peritonitis, pylephlebitis, sepsis, and in case of atypical location –retroperitoneal and paraspinal phlegmon.

The diagnosis is established on the basis of the history and clinic of the disease. The value of history is invaluable, and it is its underestimation that often leads to diagnostic errors. Severe pain, no matter how quickly it lasts, is followed by vomiting and other dyspeptic disorders and it should be regarded as a manifestation of acute destructive inflammation in the abdominal cavity.

With destructive forms of acute appendicitis, as well as in all doubtful cases, appendectomy is to be indicated.

In Crohn's disease, the clinical manifestations are caused by the localization of the damage in one or another part of the gastrointestinal tract. In most cases, the disease develops slowly, but acute, suddenly arising forms of terminal ileitis are also observed. The course is relapsing, unlike with acute mesenterial ischemia.

Crohn's disease is characterized by pain and dyspeptic syndrome. There are frequent stool disorders. As a rule, there are common symptoms, such as fever, weight loss, anemia, in severe cases – impaired absorption syndrome. On palpation through the anterior abdominal wall, a tumor-like formation can be determined at the site of the process localization.

Gastroenterocolitis is an inflammatory disease of the stomach, small and large intestines all together. The reason for it can be bacterial infection, food and drug allergies, as well as various intoxications.

The onset of the disease is usually acute, sudden, characterized by the gastrointestinal dyspepsia: belching, heartburn, heavy feeling in the epigastric region, spilled pain that is localized in the upper abdomen or around the navel, vomiting with food, diarrhea (sometimes after stool retention for 1-2 days). Along with this, there are also general symptoms of intoxication: fever, fatigue, confusion, cardiovascular disorders (up to collapse), headaches. In some cases, dyspeptic symptoms are at first mild or completely absent, and the leading sign is general intoxication. With a mild course of the disease and timely treatment, acute gastroenterocolitis disappears in a few days, whithout any consequences. In more severe cases, it lasts for 1-2 weeks, sometimes turns into a chronic recurrent form.

Typhlitis is the inflammation of the *cecum*. In this pathology, pain is observed in the right hypogastric (iliac) region with irradiation to the groin, leg, lower back, less often – in the right epigastric region, of diarrhea and constipation occurring in turns, spasm or dilatation of the cecum, pain on palpation. Often this is accompanied with duodenitis, chronic chole cystitis, tonsillitis and granular pharyngitis.

Transversitis is the inflammation of the *colon transversum* (often in combination with pancolitis, it occurs independently very rarely). Pain that is felt in the middle of the abdomen immediately after meal intake, borborigmi, diarrhea and constipation that occur in turns, occur typically, dysphagia (reflex disturbancy of swallowing) may also sometimes happen. The colon transversum is painful on palpation, spasmotic, swollen and dialted.

Proctosigmoiditis is the inflammation of the *rectum and sigmoid colon*. There are pains in the left hypogastric region and rectum, feeling of squeezing, overflow, often false desire to discharge gases. During periods of pronounced exacerbation mucus is found, sometimes with streaks of blood. Stool disturbance is more often manifested by constipation or insufficient, *insufficient fractional discharge syndrome* (loose, mushy or heterogeneous stool (3-4 times a day), which more often occur in the morning with short intervals or after each meal intake is scanty, as a result without feeling of total discharge). The sigmoid colon is spasticly reduced or swollen, and is painful on palpation. With this manifestation, the differential diagnosis of chronic abdominal ischemia is beyond doubt. Differential diagnosis with sigmoid and rectal tumors (rectoromanoscopy and colonoscopy, X-ray examination with contrast enema) is mportant.

Abdomenalgia are another pathological conditions that are characterized by abdominal pains. This group includes diseases that are not listed above, but which are to be differentiated with acute abdominal ischemia.

Fleeting episodes of abdominal pain occur in children in 12% of cases. Out of these, only 10% can demonstrate the organic basis for these abdomenalgias. Only in 30% of patients with psychogenic

disorders of the gastrointestinal tract the abdominal pain occurs as a leading symptom. The psychogenic nature of chronic pain is detected in 40% of patients.

It should be emphasized that, as a rule, abdomenalgias are characterized by multifactorial etiology and pathogenesis, the main manifestations of which are psychogenic, neurogenic, endocrine, metabolic and other mechanisms or the combination of them. Such pains are often are reflected by a vague term *"inorganic"* in the literature.

Abdominal pains in patients with the so-called ***solaritis*** and ***ganglioneuritis*** are usually closely associated with disorders of the affective sphere, experienced emotional stress or impaired vegetative regulation, rather than with infectious damages of the vegetative plexus and nodes. Somatic genesis of pain in these patients is usually excluded after thorough examination.

Different variants of abdominal pain are considered below.

Abdomenalgia of psychogenic nature. The connection between the dynamics of a number of parameters of the mental sphere, events that occurred in the patient's life with the onset of the disease events, the dynamics of the disease course and the manifestations of the clinical picture of the abdominal pains, are a strong argument in favor of the diagnosis of the abdominal pain, which demonstrates the psychogenic nature. Patients, as a rule, are aimed at finding the organic substrate of their disease for a long period (months, years). The possibility of the fact that the pain, which occurred due to social and psychological factors, seems unlikely for the patients most often. Moreover, the idea that stress, the emotional distress can provoke or exacerbate somatic sufferings is quite real and logical. In these cases, it is important to clarify the internal picture of the disease, history of life and stress experienced, as well as the life events, and to establish the causal mechanism of the psychogenic nature of the disease.

The characteristic feature of the abdominal pain, which occurs due to the psychogenic nature, is the presence of concomitant polysystemic permanent and paroxysmal vegetative manifestations. Abdomenalgia in the picture of the *vegetative crisis* is a fairly frequent clinical situation. At that, abdominal pain can occur as the first symptom or at the height of of the crisis. They are often accompanied with an increased intestinal motility.

Abdomenalgia due to mental diseases. Abdomenalgia ranks third among psychiatric patients who complain of pain. There are various descriptions of such pains. They are marked as "*abdominal psychalgia*" and emphasize such features of the disease as lack of connection between pain and topography of organs, variability of localization, intensity, nature of pain, unusual descriptions of "coloring" of the pain. Usually, dissociation is also noted between the description of the pain as "excessive", "unbearable" and a fairly satisfactory general condition of the patient, his mood, appetite, sleep and behavior, which are marked against other psychological disorders. The presented characteristics give ground to suspect in patients senesto-hypochondriac and depressive disorders of endogenous origin, in which abdominal pains are only a part of the manifestations of the clinical picture of the disease. The pathogenesis of pain is associated, in fact, with a mental illness, when "abdominal pain" is an overvalued, crazy idea and, which explains the patient's pathological behavior.

Abdominal migraine. Abdominal pain in case of the abdominal migraine is most common in children and adolescents, however, it often occurs in the adults. It is characterized by an intense, diffuse nature, but sometimes it can be localized in the navel, and be accompanied with nausea, vomiting, diarrhea, pallor and cold extremities. Concomitant vegetative manifestations can vary from a moderate to severe vegetative crises. The duration of pain varies from half an hour to several hours or even several days. There are various combinations of migraine cephalgia – simultaneous occurance of abdominal and cephalgic pain, the dominance of one of the forms during their simultaneous presence. The following factors should be taken into account in diagnostics: the association of abdominal pain with migraine-type headache, provoking and accompanying factors characteristic of migraine, young age, family history, therapeutic effect of anti-migraine drugs, an increase in the rate of linear blood flow in the abdominal aorta detected by the Doppler examination, especially with paroxysm.

Abdomenalgia with epilepsy. Abdominal pain can be a manifestation of a common attack with vegetative-visceral manifestations or manifestation of a vegetative-visceral aura. At that, it is characterized by the spread of pain and discomfort, sometimes in combination with nausea, from the

abdomen up to the head, after which there is a shutdown of consciousness and/or various partial disturbances; generalized seizure can occur as well.

It is noted that the abdominal pains in case of epilepsy are more characteristic and are manifested by their foci in the right hemisphere. This emphasizes their special role in the formation of algic and vegetative manifestations. Diagnostic criteria for abdominal pains of epileptic nature are paroxysmal and short duration (seconds) of the attack against the background of other manifestations of epilepsy, pronounced affective-vegetative manifestations, the presence of a series of epileptic phenomena in the attack itself, stunning after the attack, specific changes in the EEG.

Abdomenalgia in case of tetanus. A characteristic symptom of tetanus pain is its periodic, spasmodic and painful nature. Pain can be either paroxysmal or permanent. Patients complain of "colic", a feeling of contraction, squeezing, abdominal cramps.

In the diagnosis of the tetanus nature of abdomenalgia, the following factors are important: identification of paresthesia and musculo-tonic phenomena in the extremities (*the phenomenon of the hand of an obstetrician, pedal spasms or Carpopedal spasms*); symptoms of increased neuromuscular excitability (*Chvostecfc 's Sign, symptoms of Trousseau, Trousseau- Bonsdorff*) electromyogram changes (*doublets, triplets* when conducting an ischemic test with hyperventilation) when examining muscles in the first interdigital space; hypocalcemia, hypomagnesemia, hypophosphatemia.

Pains that are identical in nature, also occur with hyperventilation syndrome, for which tetanus disorders (increased neuromuscular excitability) are quite characteristic. The pathogenesis of abdominal pain in tetanus occur due to increased neuromuscular excitability, which is associated with the occurrence of muscle contractions and spasms in striated and smooth muscles, impaired mineral balance, and pronounced vegetative dysfunction.

Abdomenalgia in case of a periodic disease. The disease is characterized by recurrent attacks of acute pain in the abdomen and joints, accompanied by a rise in temperature up to 40-42°C. Similar states of health may last for several days. After that, they disappear, but after a while they appear again (hence the term "*periodic*").

Paroxysms of pain resemble the clinical picture of "*acute abdomen*." There is nausea, vomiting, diarrhea; on palpation of the abdomen there is sharp tension of the muscles of the anterior abdominal wall, the *Shchetkin-Blumberg symptom* is sharply positive. Taking into account the fact that fever and abdominal pain are also accompanied by an increase in ESR and leukocytosis, surgery is often indicated for these patients, and some of them are performed repeatedly. In such patients, the phenomenon of "*geographical abdomen*" is regestered, which is characterized by the presence of numerous postoperative scars on the abdomen.

The periodic disease affects patients of almost all nationalities; however, most often it occurs among the representatives of certain ethnic groups, mainly among the inhabitants of the Mediterranean region (the Jews, Arabs and Armenians).

Abdomenalgia in case of porphyria. Porphyrias are a large group of diseases of various etiologies that are based on the *disturbancy of porphyrin metabolism*. One of the variants of the disease, which is the most common one, is *intermittent porphyria*.

The leading symptom of this form of the disease is abdominal syndrome, which is characterized by recurrent abdominal pain that lasts from 2-3 hours to several days. The pain can be accompanied with vomiting, constipation and less often with diarrhea. Pathognomonic is the urination, which is of red colour ("*Burgundy wine" symptom*), the intensity of which depends on the severity of the disease. It is noted that the use of barbiturates (as a hypnotic drug) provokes exacerbation of the disease in these patients, which is manifested by the red colour of urine. Special analysis reveals *positive reaction to porphobilinogen in feces and uroporphyrin in the urine*. As the disease progresses, signs of *damage to the nervous system (polyneuropathy, radiculopathy)* are added.

Abdomenalgia of vertebro- and myogenic nature. Abdominal pain can result from the degenerative changes in the spine, spondylosis, tuberculosis, tumors or spinal injuries. The occurrence of abdominal pain is realized through the vegetative-irritative, radicular, viscerotomotor, myofascial mechanisms. Myofascial syndromes (damage of the rectus and oblique abdominal muscles) can form

not only against the background of vertebral pathology, but also as a result of prolonged muscular tension (rowing), trauma of the anterior abdominal wall and due to other non-vertebral causes.

Important characteristics of such pains are the ones related to the movement of the body, changes in intra-abdominal pressure, restriction of the movements. More often, they manifest as one-sided localization and as a combination with lumbar pain and pain in the back, which are of permanent character. In case of myofascial pains, muscles are painful on palpation and trigger zones are found. It should be noted that radicular syndromes at the thoracic level of the spine are rare, therefore, abdomenalgia of the radicular character is rare.

Abdomenalgia in case of organic damage of the brain and spinal cord. Currently, in neurological practice, abdominal pains can occur most often in case of a *myelotrophy*. With this disease, they manifest themselves as a so-called tabetic crisis, which is characterized by a sudden onset, sharp, cramping, "tearing" pains that quickly reach maximum severity.

The pain is more often localized in the epigastrium, but it may radiate to the left hypochondrium or to the lumbar region. While periodically increasing, the pain may persist for several days. Functional disorders of the gastrointestinal tract may be present.

Serological studies and analysis of neurological symptoms are of big importance for the diagnosis. The pathogenesis of these abdomenalgias is finally unclear.

Much less often abdomenalgia can be observed with *multiple sclerosis, syringomyelia and with brain tumors*. Acute abdominal pain is described and occurs in case of acute encephalitis, vascular damages of the nervous system, encephalopathy and other diseases. Abdomenalgia with tumors of the IV ventricle are characterized by high intensity, and are accompanied with spontaneous vomiting without prior nausea. Tumors localized in the temporal and superior parietal lobule of the brain can cause severe visceral, and more often, epigastric pains.

Abdomenalgia with gastrointestinal diseases of unknown etiology. In recent years, the fact that mental factors and autonomic dysfunction play an important role in the pathogenesis of the so-called inorganic diseases of the gastrointestinal tract has become clearer. At the same time, there are two situations when the abdomenalgic syndrome can be the main or one of the leading manifestations of the disease. These are *irritable bowel syndrome and non-ulcer dyspepsia syndrome*.

Irritable bowel syndrome is a chronic pathological condition characterized by the abdominal pain and bowel function disorders (diarrhea, constipation) without loss of appetite and weight loss. The duration of the disorder is no less than 3 months in the absence of organic changes in the gastrointestinal tract, which could cause the existing disorders. Pain syndrome is characterized by a variety of manifestations – from diffuse dull to acute, spastic, from persistent to paroxysmal abdominal pain. The duration of painful episodes is from several minutes to several hours. In 90% of cases, the pain is accompanied by dyspepsia (diarrhea or constipation). At the same time, 70-90% of patients with irritable bowel syndrome display change in the mental sphere in the form of anxiety and depressive disorders.

The syndrome of non-ulcer dyspepsia is manifested by abdominal pain, discomfort or nausea, which occurs periodically, lasts at least for a month, is not associated with physical activity and does not disappear within 5 minutes of recreation. Pain with dyspepsia is largely identical to the pain with irritable bowel syndrome. They are usually accompanied by a feeling of heaviness, abdominal fullness in the epigastric region after eating, they are also accompanied by gaseous or food eructation, an unpleasant metallic taste in the mouth and sometimes by loss of appetite. Besides, borborigmus, the feeling of "pouring" and the intensity of intestinal motility usually disturb the patients. Most often, they develop diarrhea, less often – constipation. However, it should be noted that such disorders, despite the fact that they cause the patient numerous sufferings, which cause asthenic and autonomic disorders, do not significantly affect the overall social activity of patients.

Thus, there is a number of diseases that are very similar to acute abdominal ischemia due to their clinical manifestations. In their diagnostics, in addition to the clinical picture, the onset of the disease is of great importance. *In all doubtful cases*, ***angiographic examination or computed tomography*** *with contrasting of the blood flow* is indicated.

Treatment. Only the agony state makes it necessary to abandon surgery in case of acute occlusion of the mesenteric arteries. Deliberate overdiagnosis of intestinal infarction, with immediate hospitalization in case of a dubious diagnosis, should be justified. ***If intestinal infarction is suspected, laparotomy is mandatory, even if it can only remain experiential.***

Treatment of acute obstruction of the mesenteric vessels is ***surgical***. *The purpose of the surgery* is to restore the blood supply in the occlusive vessel's territory, if possible, and to resect necrotic bowels. The choice of the method of operation is determined by the patient's condition, by the nature of occlusive vascular damage, the period from the onset of the disease and the state of the intestine.

In the absence of obvious signs of intestinal gangrene during the first 6 hours from the onset of the disease, the operation on the vessels is performed in order to eliminate the blockage. With the development of intestinal necrosis, it is advisable to combine intestinal resection with the surgery on the mesenteric vessels, if the patient's condition permits. The elimination of blockage and restoration of blood circulation prevent the progress of blood cloting, improve the postoperative course, and is an effective prevention of anastomosis.

With local infarctions and advanced collateral circulation, vascular surgery is not always advisable. In these cases, the resection of the affected intestine is performed.

In the presence of an intestinal infarction, ***supreme radicalism*** *is compulsory*. If the vessels surgery is not performed, the resection of the intestine should be carried out within the territory of the occluded vessels. In case of the severe occlusion of the main stem of mesenteric artery, radical resection is indicated, even if the area of intestinal necrosis is limited. However, resection of almost the entire small intestine and the right half of the large intestine is usually incompatible with life. In severe conditions, the patient's treatment is usually limited to resection of the intestine or exploring laparotomy.

II.7. LOCAL AND DIFFUSE PURULENT-INFLAMMATORY PROCESSES OF THE ABDOMINAL CAVITY, PERITONEUM AND RETROPERITONEAL SPACE (INFILTRATES, ABSCESSES, PERITONITIS)

Purulent inflammation of the visceral and parietal peritoneum – ***peritonitis*** **–** is one of the most severe complications of various diseases and damages of the abdominal organs, which is accompanied by local and severe general symptoms of the disease, by serious and often irreversible damages of the vital organs and systems. Therefore, diffuse purulent peritonitis (DPP), according to modern concepts, is characterized as a systemic inflammatory response of the body (SIRS) in reaction to the development of a purulent-necrotic process in the abdominal organs; and it is clinically manifested by endotoxicosis and multiple organs' dysfunction syndrome (MODS). Acute surgical diseases of the abdominal organs are complicated with generalized purulent peritonitis (GPP) in 15-30% of cases, with mortality rate, which ammounts to 25-30% of cases, and in case of the development of the syndrome of multiple organ failure (SMOF) – up to 80-90%.

The forms of ***circumscribed peritonitis***, which occurr as infectious complications of emergency surgeries on the abdominal organs, most often include intra-abdominal abscesses (from 0.01 to 2% of cases). Depending on the source of infection and topographic features, the sites of necrosis that are separated by the pyogenic membrane appear in typical places: in the subdiaphragmatic and subhepatic space, between the loops of the small intestine, around the cecum and in the pelvis.

Purulent-inflammatory diseases of the ***retroperitoneal cellular tissue*** (retroperitonitis, para-raephritis, paracolitis, paracystitis and parametritis) can develop primarily (by penetration of infection from distant suppurative foci or in trauma) and secondarily (due to metastasis, as a complication of inflammatory diseases of the retroperitoneal organs, or due to hematogenous or lymphogenous causes, or due to the migration of infection from neighboring organs – *per continuitatem*).

Intraabdominal abscesses

Abscesses (circumscribed peritonitis) occur in typical places in accordance with the source of infection and topographic features of the abdominal cavity. Due to destructive appendicitis, perforated gastric and duodenal ulcers, acute cholecystitis and pancreatitis, after various surgeries on abdominal organs and postponed generalized purulent peritonitis, ***abscesses*** are most often subdivided into: *ileac*

(appendicular), subdiaphragmatic and subhepatic, rectouterine pouch (pouch of Douglas) and interintestinal ***due to their localization*** **(Fig. 50).**

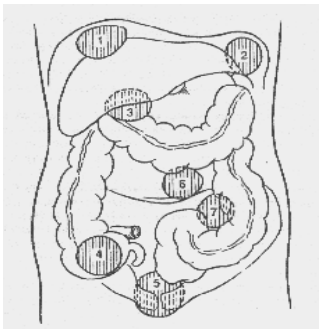


Fig. 50. Typical locations of abdominal abscesses:
1, 2 - right and left subphrenic; 3 - subhepatic;
4 - right ileal; 5 - pelvic;
6 - mizkishechny; 7 - abscess in mesentric root of the sigmoid colon

In case of appendicular (ileac) abscess, the pathological process can develop in two ways. In case the inflammatory process is controlled, the pain syndrome decreases in 2-3 days, the temperature decreases and the inflammatory tumor is palpable in the right iliac region does not tend to increase. With the progression of the process, the temperature becomes intermittent or hectic in nature, the pain in the right iliac region, dyspeptic phenomena increases. The pain increases with coughing, walking, rough riding. On examination, the tongue is wet and furred. The abdomen gets behind in the right lower quadrant when breathing; bulging can be observed. On palpation in this area, soreness, moderate muscle tension and slightly positive symptoms of peritoneal irritation can be detected. On deep palpation, sharply painful, immobile infiltration without fluctuations is determined.

Paralytic intestinal obstruction with fluoroscopic identified pneumatosis in the right half of the abdomen and some levels of fluid (Kloyber bowl) are well expressed. On rectal or vaginal examination there is pain, which aggravates on palpation of the lower pole of the formation.There is high and progressively increasing leukocytosis in the blood with a shift to the left. The increase in infiltration and its painfulness with the hectic nature of the temperature, require clarification by the ultrasound study of the localization and size of the abscess. The decision as to the emergency surgery should be made.

Subdiaphragmatic and subrenal abscesses, which are localized in the right and left subdiaphragmatic space between the diaphragm, liver and transverse colon, clinically manifest constant pain that is aggravated by taking a deep breath in the corresponding hypochondrium, which radiats more often to the back, scapula and the shoulder (via the phrenic nerve), hiccups and nausea. The body temperature is of intermittent character up to febrile. Pulse is accelerated up to 100-110 beats per minute. The patient is often in a forced position either on the back or on the side, sometimes in a sitting position. The tongue is dry and ferred with dirty gray bloom. The abdomen is moderately swollen, painful on palpation in the hypochondrium and bulged out, dilated intercostal spaces (Kryukov symptom). The skin in the strip of swelling and edema of the lateral surface of the lower half of the chest becomes yellowish and dry to the touch. Symptoms of peritoneal irritation are rarely determined. With percussion, depending on the degree of compression of the lung, we determine the lung sound up to absolute dullness (especially from the front) and, during respiratory movements, the dullness zone does not change; when the body position changes, the dull signal shifts. On auscultation, a type of weakened vesicular to bronchial respiration is determined; respiratory sounds at the border of the abscess disappear.

On percussion of gas-containing subdiaphragmatic abscesses (SDA), there are three types of different tones – a clear tone of the lung, gas tympanitis and pus dullness. Gas with SDA, when shifting due to the change in the position of the body, is always in its upper part (*Deve symptom)*. On auscultation in the abscess zone there is the sound of a falling drop, and with a rapid change in the position of the patient, there is a “splashing noise” of Hippocrates.

Subdiaphragmatic right anterior and posterior abscesses are, as a rule, difficult to be diagnosed and the course of the disease is severe. They are characterized by: pain in the right hypochondrium and in the lower parts of the chest on the right; *on an objective study* – bulging of the abdominal wall in the right hypochondrium, there is pain, enlargement and prolapse of the liver, *the percussion phenomenon of Berlot*, which determines 4 types of percussion sound, which successively replace each other: pulmonary sound, shortening of percussion sound, tympanitis and dullness that correspond to lung tissue, reactive exudate in the pleural cavity, gas and the level of fluid in the

abscess cavity; *on X-ray examination* – immobility and high position of the right dome of the diaphragm, hepatoptosis, there is gas with the fluid level below the diaphragm. The following positive *symptoms* are possible to occur: *Haubrich* (dullness and weakening of breathing in the lower parts of the right lung), *Kryukov* (painful palpation of the IX-XI ribs on the right), *Duchenne* ("paradoxical breathing" – the involvement of the epigastric region during inhalation and protrusion of it during exhalation), *Yaure* (balloting of the liver when the chest is tapped with the other hand), *Senator* (immobility of the spine when walking due to the rigidity of the back muscles), *Latten* (retracting of the intercostal spaces in the places of diaphragm attachment).

Blood tests show leukocytosis, neutrophilia, left shift of leukocyte blood test, an increase in ESR, anemia, dysproteinemia, and the appearance of C-reactive proteins, which remain during the antibiotic therapy.

According to fluoroscopic and radiographic reports, subdiaphragmatic abscesses (SDA) show thickening of the diaphragm up to 8-17 cm with a higher standing of the dome of the diaphragm and decrease in its mobility on the affected side; *"sensitive" (reactive) effusion* in the posterior-lateral parts of the pleural cavity. Below the diaphragm, radiolucent focus (gas-filled fundus) above the level of the liquid is displayed. ***Ultrasound examination*** (echography, sonography) makes it possible to diagnose SDA and estimate the dynamics of the development and the possibility of controlled puncture drainage of abscesses in 90-95% of cases. The study of the punctation and the detection of tyrosine, hematoidin and bile pigments in them, makes it possible to judge about the genesis of SDA.

The ***computed tomography (CT) and magnetic resonance imaging (MRI)***makes it possible to clearly visualize the location and size of SDA, to differentiate it from reactive pleurisy, hepato-and splenomegaly in 95-100% of cases.

In case of ***subhepatic abscess***, which is located between the liver and the stomach, clinical manifestations are more erased, and it is difficult to detect a gas-filled fundus under the liver with a level of fluid radiographically. Contrast studies of the gastrointestinal tract may reveal the displacement of the intestine or stomach by an infiltration. If the abscess is caused by the leakage of the anastomoses, it is possible for the contrast substance to enter the abscess cavity via the intestinal lumen. Ultrasound examination, computed tomography and magnetic resonance imaging make it possible to specify the size and location of the abscess, its relationship and communication with the adjacent organs, and to identify the best ways of incision and drainage.

Pelvic abscess (Douglas space) is mainly caused by perforated appendicitis, perforation of the diverticulum of the colon, purulent inflammation of the internal genitalia, less often by the residual abscess of diffuse forms of purulent peritonitis. In case of an abscess that occupies the rectouterine uterine or rectal-vesical-cystic space, as a rule, there are no peritoneal symptoms. The protective tension of the anterior abdominal wall muscles develop only with the accumulation of pus (1 l or more) and inflammation of the peritoneum.

Patients complain of a feeling of heaviness, pain in the lower abdomen, frequent and painful urination, an increase in stool frequency, or diarrhea with rectal tenesmus. ***With digital rectal or vaginal examination***, the overhang of the anterior or posterior wall of the rectum is determined, the sharp pain and pulpy infiltration with the softening zone in the center are determined. The blood test reveales signs of a purulent process. ***With rectal or vaginal ultrasound examination***, the size and extent of the abscess are clearly defined. Then after its transrectal or transvaginal puncture, purulent discharge is detected.

Inter-intestinal abscesses are often multiple, they are usually located in the upper (above the mesentery of the transverse colon) or lower floors of the abdominal cavity between the intestinal loops, mesentery, abdominal wall and epiploon. Often, they are the result of peritonitis with incomplete recovery, or infectious-inflammatory destruction and perforation of the hollow organ of the abdominal cavity. Diagnosis is difficult because of the moderate intensity of the dull nature of abdominal pain without clear localization, with periodic abdominal distension and an increase in temperature up to 38 ° C and above until the evening time.On examination, the abdomen is mostly soft, with local tension of the abdominal muscles, there is tenderness only in the region of the abscess. Sometimes the stomach is asymmetrical with ulcers that contact with the abdominal wall. On palpation, pathological formation,

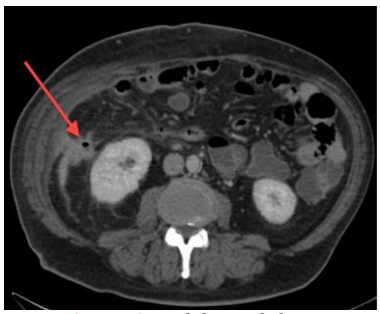


Fig. 51. CT - abdominal abscess (there is gas in the cavity)

moderately painful and immobile can be determined. The symptoms of intoxication may develop with abscesses. ***The survey fluoroscopy of the abdominal cavity*** and large-sized abscess detects the darkened focus, which is sometimes found with the level of liquid and gas, the phenomena of intestinal paresis and the impression of intestinal loops in contrast study.

The exact localization and size of the abscess is established by the ultrasound examination, CT (**Fig. 51**) and MRI.

The abscesses of the abdominal cavity are treated only surgically. In case of urgent indications – opening, sanation and adequate drainage of the abscess under general anesthesia are performed.

Pus removal and drainage are performed after separating the source of the infection from the rest of the abdominal cavity with the help of gauze tampons (to prevent infection of the intact parts of the abdominal cavity). The choice of the operative intervention depends on the exact localization of the abscess and the ability to drain it most efficiently, excluding infections of the pleural and abdominal cavities.

Treatment of appendicular abscess. The surgery is performed after the introduction of a day dose of broad-spectrum antibiotics and metronidazole, under general anesthesia, ***extra-abdominal route*** is preferable (***according to Pirogov***). After the pus removal and lavageof the cavity with antiseptics, appendectomy is performed, if possible; the drainage is carried out with double lumen drains for active aspiration of the contents, which is followed by the lavage in the postoperative period. It is better to aoid inserting tampons into the wound. In the postoperative period detoxification therapy is to be carried out, antibiotics (aminoglycosides) in combination with metronidazole are to be introduced. ***Prognosis and complications*** depend on timeliness and adequacy of the surgery.

In case of the anterior-superior SDA (subdiaphragmatic abscess), *the lateral extrapleural extraperitoneal rout according to A.V. Melnikov* is applied. In this case, the skin incision, 10–12 cm long, is performed along the X rib line, parallel to the supposed margin of the pleural sinus of cartilage and to the mid axillary line; the resection is carried out along IX and X ribs (8–10 cm long). Pleura is stripped up by blunt dissection, the diaphragm, which is dissected along the wound is exposed, the peritoneum is stripped off the abscess, the latter is punctured and, after collecting the pus, it is opened and drained.

With the localization of the abscess ***in the anterior section***, *the Clermont anterior subcostal extraperitoneal route* is applied. The incision starts with the lateral edge of the rectus abdominal muscle (up to 10 cm long) parallel to the costal arch. Pulling the costal arch up and forward, the surgeon slides his finger along the transverse fascia upward, peels it from the inner surface of the transverse muscle and the lower surface of the diaphragm. The abscess is opened in the fluctuation zone or along the dotting needle.

In case of posterior SDAs, *an extraperitoneal posterior rout is used below and parallel to the XII rib*, starting at three fingers' breadth above the paravertebral line to the axillary line. The tissues are cut until the transverse fascia; if necessary, the resection of the XII rib is performed. Then it is proceeded as with the above described anterior rout. When applying active flow- drawing drainage, the cavity closes on average for 20-27 days, that is, 10-20 days faster than with standard drainage.

Subhepatic abscesses are also revealed when *being incised along the costal arch*. Sometimes, under the control of the ultrasound examination or CT, abscesses are drained by percutaneous puncture with two tubal drains: for washing and aspiration, which is especially important in elderly and debilitated patients with concomitant pathology.

Pelvic abscesses *are opened through the rectum or vagina in women.* After cleaning of the intestine, emptying the bladder with a catheter and stretching the rectal sphincter according to Subbotin with spinal or epidural anesthesia, transrectal or transvaginal abscess puncture is performed in the zone of softening of the infiltrate. When receiving the pus under the visual control, by stretching

of the anus with a bivalve gynecological mirror, the ulcer is opened with the needle-shaped lance, with the scalpel limited in its cutting surface up to 1-1.5 cm; the blade is wrapped with a plaster. When pus appears, the wound canal is expanded with a soft curved clamp, and a drainage tube is inserted to wash and sanitize the abscess cavity. Nearby, a thick vapor tube is introduced to the rectum at a depth of 10-15 cm; it is manyfold perforated and is wrapped with a napkin, Vishnevsky ointment is to be applied in the area of contact of the tube with the rectal sphincter (usually during 48-72 hours).

Inter-intestinal abscesses are opened and drained using the rout, which depends on its location and the number of abscesses. It is important to remember that opening of the abscesses should be carried out after a preliminary delimitation of the free abdominal cavity by gauze napkins. With abscesses that are tightly adjacent to the anterior or lateral walls of the abdomen (flank), their puncture and flow-through drainage is possible under ultrasound or CT control, followed by active washing with antiseptic solutions and by the intestinal intubation to control postoperative paresis and enteric intoxication.

Purulent peritonitis

Diffuse purulent peritonitis (RGP) *is a secondary purulent inflammation of the visceral and parietal peritoneum, which is characterized as a systemic inflammatory respons syndrome (SIRS) in response to the development of a purulent-necrotic process in the abdominal cavity. It is clinically manifested by endotoxicosis and multiple organ dysfunction (MODS).*

Classification of peritonitis.

I. *According to the etiology peritonitis is distinguished into:*

1. *Primary peritonitis or "mixed bacterial peritonitis"* when the peritoneum is infected via hematogenous route and due to the extraperitoneal sources.

2. *Secondary peritonitis* – is a form of complicated intra-abdominal infection due to primary and postoperative destructive lesions of the abdominal organs.

3. *Tertiary peritonitis or "peritonitis without a manifesting source of infection"* – is a recurrent and persistent form, which develops against the background of suppressed mechanisms of local and systemic anti-infective protection with multi-organ dysfunction and without an obvious source during laparotomy.

II. *According to the nature of the exudate*:

1). Serous.

2). Serofibrinous.

3). Fibrinous-purulent.

4). Purulent.

III. *According to the extensiveness of the process*.

1. *Local peritonitis* – is localized in one anatomical region:

1) *circumscribed* – completely delimited by adhesions from the abdominal region (infiltration, abscess)

2) *non- circumscribed* – the process can progress in the future without delimiting adhesions.

2. *Diffuse peritonitis* – the process affects more than one anatomical region – the damage of two floors of the abdominal cavity (the third remains intact).

3. *Generalized peritonitis (common) peritonitis* – the pathological process occupies all the anatomical areas (all the three floors).

IV. *According to enteric insufficiency*.

Stage I – stage of compensation or normal postoperative paresis;

Stage II – stage of decompensation, or true functional obstruction of the intestine;

Stage III – the terminal stage or paralysis of the alimentary canal.

V. *According to the clinical course*.

1. *The compensation phase* – the pathological process is compensated by eliminating of a clearly identified source of peritonitis, adequate lavage of the abdominal cavity, infusion therapy, rational antibiotic therapy, by organ and metabolic support.

2. *The phase of decompensation* is a pathological process, which is manifested by severe general intoxication; it is compensated by eliminating the source of peritonitis, adequate lavage of the

abdominal cavity, extracorporeal detoxification, rational antibiotic therapy and infusion therapy, by organ and metabolic support together with the control of the paralytic ileus.

3. *The phase of multiorgan failure* – is charaterised by severe disturbancy of homeostasis and metabolism, dysfunction of the pulmonary and cardiovascular systems, hemostasis, paralysis of the digestive tract, severe dysfunction of the liver, kidneys and central nervous system.

Clinical manifestations. *During the first phase (compensation phase),* due to moderate endotoxicosis caused by the infectious-inflammatory process in the abdominal cavity, the following symptoms are clinically registered: excitation, consciousness is not disturbed, the tongue is dry, tachycardia (up to 100 beats per minute); moderate dyspnea is possible (up to 30 per minute), the body temperature is subfebrile. There is a moderate paresis of the digestive tract organs with normal diuresis. The intense pain of constant nature in the abdomen aggravate with changing of the patient's position. This makes them lie motionless on the side with their legs led to the stomach. Vomiting with large amounts of stagnant gastric contents does not bring relief. *On objective examination* of the abdomen: there is a protective lagging of the anterior abdominal wall during breathing. Tension and tenderness in the area of the peritonitis development center is observed –"plate-like stomach"; on percussion, there is severe tympanitis caused by the intestinal paresis; on percussion, there is a dull sound in the flank of the abdomen in areas, where the peritoneal exudate accumulates (when it amounts to 700-1000 ml).

On deep palpation of the anterior abdominal wall, the inflammatory infiltrate, intussusceptum, increased and destructively changed organ (gallbladder), tumor, the area of the most pronounced Shchetkina-Blumberg symptom are detected. On auscultation there is a weakened peristaltic noise with possible discharge of gas and stool.

In the second phase (decompensation phase), the "local" manifestations in the form of pain syndrome and tension of the abdominal wall are decreasing against the background of the developing true paralytic ileus of the intestine and the growing pronounced intoxication of the body. Clinically – excitement, euphoria or atony are observed in patients. Besides, there are adynamia, regurgitation and vomiting with brown liquid, stool retention and gas discharge, sharply pointed face features, sunken eyes, dry tongue, severe tachycardia (up to 120 beats per minute), moderate hypotension, shortness of breath (30-36 per minute), hyperthermia (more than 38^{o}C).

On objective examination of the abdomen, it is dramatically swollen with no peristalsis (or sharply weakened). *The Shchetkin-Blumberg symptom* is not so sharply expressed as in phase I.

During the third phase (multiorgan failure), due to severe disorders of goomeostasis and metabolism, the function of the pulmonary and cardiovascular systems, as well as the liver, kidneys, central nervous system is disturbed. There is also disturbance in the hemostasis which may be followed by the paralysis of the alimentary canal. The severity of the patient's condition manifests itself in impaired consciousness in the form of precoma or coma (confusion, delirium), pale skin with acrocyanosis, exacerbation of facial features (facies hippocratica), severe tachycardia (120-140 beats per minute and more). The shortness of breath (more 36 min), uncorrected hypotonia, indomitable vomiting, a large amount of gastric and intestinal contents with fecal odor are indicative.The abdomen is dramatically swollen with generalised pain, muscle tension and positive Shchetkin-Blumberg symptom in all the portions. The pain is dull in sloping areas of the abdomen; there is no peristalsis (the symptom of "final silence"), with positive Kulenkampf symptom.

The diagnostics of peritonitis is difficult in children and elderly people, primarily due to the difficulties with adequate contact and collecting of full history of the disease. In this case, peritonitis in children often proceeds as a hyperergic reaction, characterized by severe pain, high hyperthermia and leukocytosis, abrupt tension in the abdominal muscles. In elderly people, tension in the abdominal wall, hyperthermia and leukocytosis, as a rule, are weakly expressed due to a decrease in the body's reactivity to pain.

In case of postoperative peritonitis, which occurs due to anastomoses leakage, accumulation and suppuration of blood and exudate that develop on the background of the action of antibacterial and analgesic agents, the diagnosis causes particular difficulties. This is due to the fact that after the surgical operations on the abdominal organs in the early postoperative period, intestinal paresis,

tension of the abdominal muscles, Shchetkin-Blumberg symptom, hyperthermia, tachycardia and leukocytosis are often noted.It is possible to suspect the development of postoperative peritonitis when pain persists and there is an increase in the above mentioned clinical symptoms. The principles of diagnostics, regardless of age and cause, are the same as described for acute inflammatory and destructive diseases of the abdominal organs.

In accordance with standard schemes, there is ***the plan of laboratory and instrumental examination*** and *possible pathological changes*. In general, the analysis of blood shows leukocytosis, neutrophilia, accelerated ESR. The increase of markers of the indices of blood intoxication is moderately pronounced. Diagnosis of peritonitis is associated with the sequential use of non-invasive (ultrasound, review fluoroscopy and radiography of the chest and abdomen, computed tomography) and minimally invasive (laparocentesis, laparoscopy, according to indications – puncture of the accumulations of pus).

Survey fluoroscopy (radiography) detects accumulations of free gas under the right or left dome of the diaphragm (*Jobert's symptom*), which confirms the perforation of the hollow organ, with high standing of the diaphragm and restriction of its mobility, "reactive" pleurisy. All these indicate a pathological process in the upper abdominal cavity. Detection of free fluid in the abdominal cavity adjacent to the site of the inflammation, paretic gas-swolle enteric loops ("*enteric arches*"), round shaped formations with fluid level and gas above it in the intestinal loops (*Kloyber bowls*), the output of the contrast agent, introduced per os, outside the lumen of the organ of the alimentary canal – all these factors objectively confirm the diagnosis.

Ultrasound examination and CT are able to determine the accumulation and volume of the exudate, its localization, and the enteric contrasting – to detect the source of peritonitis.

During the second phase, leukocytosis with left neutrophilic shift is noted in the blood. Biochemical and special studies detect the decrease in the volume of circulating blood, protein, impaired water-electrolyte balance and acid-base status — hypokalemia and alkolosis. With decrease in diuresis — hyperkalemia and acidosis are determined.

During the third phase the blood test shows high leukocytosis, which is replaced by leukopenia due to secondary immunodeficiency. ECG detects signs of toxic myocarditis and electrolyte disorders. The blood test reveals critical breakdown of water and electrolyte balance, acid-base status and signs of disseminated intravascular coagulation.

To determine the nature of peritoneal exudate (blood, bile, enzymes, pus, etc.) in all doubtful cases, it is necessary to start with the most common studies: laparocentesis (puncture of the abdominal wall in classical Kalka points with te introduction of a catheter into the abdominal cavity) or fine-needle puncture of zones where the accumulation of fluids is most likely to happen (transrectally, transvaginally, etc.). If there the diagnosis is uncertain and the source of peritonitis is not determined, laparoscopy is indicated. It makes it possible to examine almost all the abdominal organs, to identify the source of peritonitis and the nature of the exudate, to evaluate not only the visceral, but also the parietal peritoneum. In the most severe diagnostic cases, microlaparotomy and diagnostic laparotomy are used.

Treatment of widespread purulent peritonitis (WPP) *includes emergency salvage operation by means of extensive laparotomy according to Petrov, removal of the focus of infection or drainage of the abscesses, aspiration-mechanical cleaning of the abdominal cavity, repeated lavage with antiseptic solutions during and after the surgery (peritoneal lavage), control of paralytic intestine by way of intubation of it with a probe, aspiration of gastric and intestinal contents and stimulation of intestinal motility, adequate draining of the abdominal cavity* (Fig. 52), *if necessary,* ***laparostomy*** *is used to perform programmed relaparotomies with the aim of repeated sanitization of the abdominal cavity.*

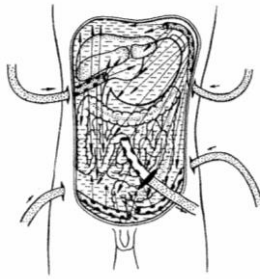


Fig. 52. The abdominal cavity drainage scheme with widespread purulent peritonitis

This main stage of treatment should take place against the background of massive antibacterial therapy and massive infusion

therapy, with correction of protein metabolism, disorders of water and electrolyte metabolism and acid-base status correction, maintenance of the functioning of pulmonary and cardiovascular systems, liver and kidneys is to be carried out.

The preoperative preparation as to the volume and intensity should be carried out taking into account the phase (stage) and peritonitis wideness, severity of intoxication and MOF (multiorgan failure) in order to achieve stabilization of homeostasis (blood pressure, central venous pressure and to increase diuresis) within 2 hours.

Emergency salvage surgery is performed under general anesthesia and artificial respiration, an extensive midline laparotomy is performed to eliminate the cause of peritonitis (excision and suturing of the perforated ulcer, appendectomy, cholecystectomy, bowel resection, etc.).

To prevent complications it is necessary: to perform peritoneal repair of the inflammatory-altered areas, to impose external intestinal fistulas, when suturing of the defects are impossible, to perform peritoneal repair of the suture line with large omentum (omentopexy), adhesive compositions and polymer films, which increase their tightness, to perform decompression of the anastatic chamber, or to insert the prosthetic stent.

The objective criteria for the viability of tissues and organs (that are clinically based on the presence of pulsation of the arteries, the coloration of the serous cover of the organ, the vasomotor reaction in the compromising zone, the brightness of the peritoneum and its hyperemia, as well as the instrumental transillumination and capillary blood flow assessment, impedancemetry, etc.) remain controversial.

Sanitization of the abdominal cavity incase of generalized peritonitis starts with lavage using 0.5% warm aqueous solution of chlorhexidine, miramistin, dioxidine, to which 3% solution of hydrogen peroxide in the ratio of 1:10, ozonized with saline at an ozone concentration of 5.0-7.0 mg / l or solutions of furatsilin 1: 5000, sodium hypochlorite in concentrations up to 1200 mg / l can be added. Thorough drainage with an electric suction pump and gauze napkins has to be done; removal of loose infected fibrin layers is mandatory .

The amount of the lavage fluid (from 5 to 10 liters) depends on the extention of peritonitis and the nature of its content. In addition to careful suboperative treatment of the abdominal cavity, sometimes with the use of ultrasound or irradiation by defocused radiation of the helium-neon laser, the need for *prolonged lavage* arises. The following methods are applied:

1) traditional (from 4 points according to Petrov) drainage of the abdominal cavity with a

blind suture of the laparotomy wound (in women, additional rout is through the posterior vaginal fornix)

2) peritoneal (through drainage or fractional) lavage;

3) staged laparostomy lavage (with programmable abdominal lavage)

4) controlled laparostomy.

Peritoneal lavage (dialysis) – through (8-10 l.) or fractional (2-3 l.) lavage of the abdominal cavity for 2-3 days with glove-tubular multiperforated drainages to remove the infected material (exudate, fibrin membranes) (Fig. 53).

1:5000 units of furatsilin solution are introduced via tubular drains (microirrigators) in the hypochondria, with the addition of aminoglycoside antibiotics and 500-1000 units of heparin per 1 liter of solution (prevention of the fibrin clots formation in dialysate), or ozonizing physiological salt solution (OPSS) with ozone concentration of 5-7 mg / l without any additives is used. The outflow of washing liquid is performed through the iliac tubular drainage; they are also used to inject the solution fractionally for 2-3 hours, which is followed by aspiration. The most optimal option is to establish the multiperforated tubular drainages subdiaphragmally on the right and on the left, subhepatically; and two lower tubular drainages are to be installed through the iliac area into the pelvic cavity, with the output through separate punctures of the anterior abdominal wall. After cleaning of the fluid coming from the abdominal cavity and separating

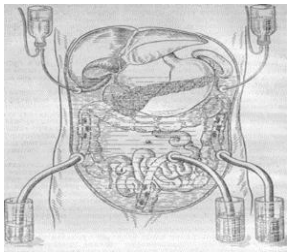


Fig. 53. Through drainage of the abdominal cavity

of the spongyly binding drainages from the free abdominal cavity, the prolonged lavage is stopped and the tubes are removed, only rubber tube drainages remain.

With widespread purulent peritonitis in the phase of multiple organ failure, anaerobic peritonitis the treatment is carried out with the usage of the method of *laparostomy*. After eliminating the source of peritonitis, simultaneous sanitization of the abdominal cavity and intestinal intubation, the floor of the intestinal loops and the omentum are placed on a sterile, perforated polyethylene film, which goes 1.5-2 cm beyond the wound edge.

Tubular micro-irrigator is installed over the film and is covered with spongy antiseptic wipes. Circular seams are overcasted on the wound (with wound edges drawn together up to 5-2 cm) through all the layers excluding the peritoneum, with round-the-clock slow (15-20 drops per minute) irrigation of the wound. Repeated revision of the abdominal cavity with mechanical cleaning of the loops of the intestine and organs, irrigation of it with the antiseptic solution (up to 15-20 liters) is carried out according to indications in 24-48 hours, in an amount of 2-5 sessions of stepwise lavage.

The programmed relaparotomy is very similar in its meaning and stages to laparostomy, with the only difference – the abdominal cavity is sutured with separate skin sutures or zippers overcasted into the wound edges. Repeated debridement of the abdominal cavity after removal of the skin sutures is carried out in 24-48 hours. At the same time, in case of anaerobic peritonitis, laparostomy is preferable, which provides better aeration of the surgical wound.

Decompression of the hyperinflate paretic intestine should be performed with the application of the intubation multi-perforated probe transnasally, trans-cecally or totally antegradely transrectally. The complete necessity of the intestinal decompression for 6–12 days with rinsing (enteric lavage with ozonizing physiological salt solution (OPSS)), administration of enterosorbents (Enterodez, belosorb), selective decontamination of pathogenic microflora (polymyxin M-B, Diflazon, fluconazole) and early enteral feeding, can be due to the pronounced intestinal wall hypoxy in peritonitis. It is characterized with the distubed secretory, motor, suction and digesting functions, and contributes to the colonization and translocation of pathogenic bacterial microflora.

Numerous *disturbances of homeostasis* and pathophysiological changes in the body of the patient, who was operated on for widespread purulent peritonitis, call for *the need for their correction* in the postoperative period with the help of infusion targeted treatments:

1) compensation of circulating blood volume (CBV) deficiency, correction of hypovolemia and normalization of central hemodynamics (crystalloid and colloid solutions, hemodilution agents – low-molecular saline dextran – reopolyglucin, gelatinol, albumin hemodez-Ringer in equal proportions of 25 ml / kg);

2) normalization of oxygen transport (ozonized saline, hypertonic solution of 10% sodium chloride, 4-6 ml / kg in combination with reopolyglukine – 1:1, hydroxyethyl starch preparations – with hematocrit level of at least 30%);

3) correction of dehydration and normalization of the electrolyte composition of blood (isotonic sodium chloride solution, Ringer's solution, chlorol salt, trisol, potassium chloride, etc.) – under the control of the level of electrolytes in blood plasma;

4) correction of hypoproteinemia (blood products - native and frozen plasma (OctaPlas), albumin);

5) neutralization of metabolic acidosis (4.5% sodium bicarbonate solution, lactosol);

6) maintaining of vascular tone and blood pressure against hypovolemia and hypotension (prednisone, hydrocortisone);

7) general and organospecific support (heart drugs, anti-enzyme drugs, hepatoprotectors, synthetic anabolic hormones, various vitamins);

8) antioxidant therapy (ozone therapy – ozonizing physiological salt solution (OPSS) and major autohemoozonotherapy – (MAHOT); vitamins E and C, pentoxifylline);

9) selective decontamination of the small intestine (intestinal intubation, enteric ozone therapy, enterosorption, polymyxin M-B).

In cases of severe intoxication in patients with widespread purulent peritonitis (WPP) in the postoperative period, ***extracorporeal detoxification methods*** are widely used: *hemosorption*,

plasmapheresis, ozone therapy, ultraviolet blood irradiation (UVIR) and laser blood irradiation (LBI), hardware hemodilution and hemodialysis.

The central position in the complex postoperative treatment of RGP patients is given to the deliberate *infection control*, which includes both local application of antibacterial therapy methods and intramuscular, intravenous, intraarterial and endolymphatic administration of antibiotics and antibacterial agents.

During antibiotic theraputic management, it is advisable to keep to the following rules:

1) when prescribing two or more antibiotics, the effect of their interaction, the maximum concentration in the body and elimination half-time should always be taken into account, the routes of administration of drugs are to be separated;

2) take into account the nephro-, hepato- and neurotoxicity of certain drugs must be taken into account;

3) the antibiotics have to be prescribed in accordance with the sensitivity of the manifesting microflora or, first of all, empirically, taking into account the frequency of occurrence and the maximum sensitivity of the microflora;

4) in order to avoid the formation of antibiotic resistance of the microflora, antibiotics must be replaced by the second-line drugs every 7-8 days;

5) antibiotics must be combined with antiseptic drugs (metronidazole, nitrofurans, sulfonamides) and physico-chemical methods (ozonotherapy, ultraviolet blood irradiation and laser blood irradiation, etc.), which enhance the effect of drugs of choice;

6) to prescribe prophylactically antifungal drugs must be prescribed prophylactically (diflucan, Diflazon, fluconazole, nizoral, etc.).

The complex implementation of measures ***aimed at supporting and enhancing of the body's natural immunoresistance*** is important in the treatment of widespread purulent peritonitis; this, as a rule, includes:

1) the use of pharmacological agent of natural and synthetic origin (levamisol-decaris, Diucifon, Salmosanum, bioglobin, polyanions, etc.);

2) administration of blood products and products of the immune system of animals (thymalin, T-activin, timoptin, timomodulin, human leukocyte immunoglobulin and gammaglobulins, etc.);

3) the use of anti-stress drugs to prevent secondary immunodeficiency (ganglioblockers, adrenoblockers, lithium oxybutyrate and sodium oxybutyrate);

4) physical-chemical methods of detoxification and immunostimulation (ozone therapy, ultraviolet and laser blood irradiation, membrane plasmapheresis, hemosorption).

The patients, who were operated on in the third phase of generalized (common) peritonitis (multiple organ failure), require prolonged inotropic ventilation (artificial pulmonary ventilation, often with positive pressure at the end of expiration, PEEP), organ and metabolic support, the use of methods of extracorporeal "replacement" of the function of internal organs (hemodialysis, hemofiltration, ozone therapy, membrane plasmapheresis, etc.). In case of the development of septic shock and respiratory distress syndrome, hemofiltration with ozone therapy becomes the method of choice.

In case of widespread purulent peritonitis, the mortality rate ammounts up to 25-30%, and in case of the development of multiple organ failure – up to 85-90% of cases. Therefore, it must be remembered that the prognosis of widespread purulent peritonitis *depends on the nature of the underlying disease that is caused by peritonitis, the timeliness and thoroughness of surgery, the adequacy of enteric and intraperitoneal detoxification, the complexity of the etiopathogenetically justified therapy.*

Pyoinflammatory processes in retroperitoneal tissue (*spatium retroperitoneale* – retroperitoneum)

Classification of inflammatory diseases of the retroperitoneum.

I. *According to the etiology.*

1. *Primary* – are of hematogenous origin, coming from the purulent focus located outside the retroperitoneal space.

2. *Secondary* (up to 80%) – the retroperitoneal organs are the source of infection.

II. *According to the charakter:*

1. *Acute (catarrhal, infiltrative, purulent).*

2. *Chronic (sclerosing and fibro-purulent).*

II. *According to the nature of the disease course*.

1. *Acute* (catarrhal, infiltrative, purulent).

2. *Chronic* (sclerosing and fibrous-purulent).

III. *According to the localization of the process.*

1. *Paranephritis* – lesions of the adrenal fiber: anterior, posterior, upper, lower and total.

2. *Retroperitonitis*.

3. *Paracolitis* – between the posterior surfaces of the ascending and descending colon and retroperitoneal fascia, medial to the root of the mesentery of the small intestine.

4. *Paracystitis*.

5. *Parametritis*.

Purulent-inflammatory diseases of the retroperitoneal space (RPS) may have *serous, purulent* and *putrefactive* nature. According to the above mentioned classification, and depending on ***the location of the damage***, *perinephritis, paracolitis and inflammation of the retroperitoneal tissue itself* are distinguished. Abscesses and phlegmons, which develop in the retroperitoneal tissue, most often arise as a complication of inflammatory diseases of retroperitoneal organs (*secondary*). Infection can penetrate by the way of hematogenous or lymphogenous route from distant purulent foci (*primary*).

The clinical picture of pyoinflammatory processes (PIP) is diverse, but *the signs of general intoxication prevail* – chills, high body temperature, anorexia, fatique, apathy, leukocytosis and left shift of blood leukocytes, there is increase in ESR. In severe cases, dysfunction of the cardiovascular system is observed.

With the formation and enlargement of the abscess in retroperitoneum, the pain occurs in the lumbar region or lateral parts of the abdomen; and in case of paracolitis – in the depth of the abdominal cavity. At the same time, changes in contours, bulging of the abdominal wall in the lumbar or epigastric areas, formation of infiltrate, muscle tension, etc. are detected. Retroperitoneal abscess is often accompanied by flexion contracture in the hip joint on the site of the leision.

Purulent inflammatory lesion of the odd-numbered median cellular space, which fascially covers the peritoneal part of the aorta, the inferior vena cava and the network of lymph nodes and vessels, is extremely rare and does not require surgical rehabilitation.

Severe ***complications*** of pyoinflammatory processes in retroperitoneum are as follows: rupture of the retroperitoneal abscess into the abdominal cavity with the subsequent development of peritonitis, the spread of the retroperitoneal phlegmon in the mediastinum with the development of mediastinitis, the occurrence of secondary osteomyelitis of the pelvic bones or ribs, intestinal fistula, paraproctitis, purulent inflowing into the gluteal region and the hip.

The diagnosis of pyoinflammatory processes in retroperitoneum is made on the basis of the clinical picture, as well as on the data of *ultrasound and radiological studies*. At the same time, on the survey images, the abrasion of the contours of the organs of the retroperitoneal space and the lumbar muscle are visible; and radiopaque examinations (excretory urography, irrigoradiography, etc.) make it possible to clarify the source of the inflammatory process.

CT and MRI with double contrasting, after the painful introduction of modern radiopaque substances (Iohexol, Iopamidol, Iopromid-Ultravist, Gadobutol-Gadovist, etc.) makes it possible to detect most accurately the presence, localization, and distribution of pyoinflammatory processes in the retroperitoneal space.

The convincing confirmation of the purulent process in the the retroperitoneal space is the pus received during *puncture under the ultrasound control*.

Treatment of the inflammatory processes in the retroperitoneal space ***in the early stages,*** in the absence of symptoms of purulent fusion of tissues, is ***conservative***, complex, antibacterial in compliance with the mentioned above principles; ***in the purulent stage***, ***the treatment is surgical*** – with the usage of lumbotomy that depends on the localization and spread of the abscess; the purulent

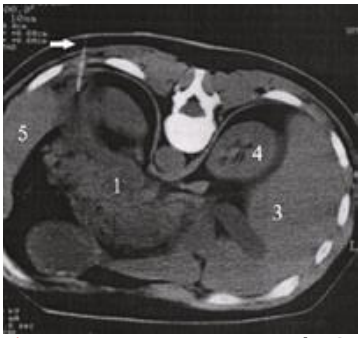


Fig. 54. Percutaneous puncture under CT navigation, puncture needle is indicated

contents are evacuated, the cause (stone) or source (necrotic sequesters, pyonephrosis etc.) of the secondary suppuration of the retroperitoneal tissue is removed, the cavity is drained by rubber tube drainages (at least 3-4); and, in case of increased bleeding, additionally, by cellophane-gauze tampons. The posterior angle of the wound, where the tampons and drainage tubes are removed, remains for up to 10 cm inaccessible.

In recent years, in case of limited abscesses of the retroperitoneal space, *percutaneous puncture of the abscesses and the drainage according to the Seldinger* method under the control of ultrasonography (ultrasound) or CT, has received a wider use. (Fig. 54). In some cases, this minimally invasive method allows to drain the combined abscesses of the organs and tissue space under the local anesthesia. Then, the repeated daily rinses via the drainage tubes with warm antiseptic solution and introduction of antibiotics makes it possible to achieve the elimination of the abscess with obliteration of the residual cavity.

Postoperative detoxification, antibacterial, immunostimulating therapy should be combined with lavage of the abscess cavity by the antiseptic and proteolytic solutions with active aspiration of purulent-necrotic masses. Further complex physiotherapy (diathermy, ozonid ointment, mud poultice applications, hydrotherapy, etc.), tonic and absorbable therapy (Lydase, aloe, vitamins, antioxidants, etc.) are aimed at preventing of the retroperitoneal fibrosis (*Ormond's disease*).

II.8. OBSTRUCTIVE JAUNDICE. LIVER FAILURE

Recently, there has been a significant increase in the hepatopancreatobiliary zone (HPBZ) organs diseases throughout the world. The number of patients of this group in surgical hospitals amounts for up to 25% of all the patients with diseases of the gastrointestinal tract. Among the liver surgical diseases and extrahepatic bile ducts diseases, the most severe are the ones accompanied by persistent obstruction of the common bile ducts with subsequent development of obstructive jaundice (OJ). The incidence of obstructive jaundice (OJ) in people with HPBZ, according to various authors, ranges from 12.0 to 45.2%. The problems with diagnostics and differential diagnostics of the causes of the biliary ducts obstruction have not lost their relevance at the present time.

The vast experience gained by foreign and domestic surgeons shows that obstructive jaundice is most oftenly caused by cholelithiasis (ChL) with choledocholithiasis (36.9%). There has been a tendency to an increase in patients suffering from obstructive jaundice of non-calculous origin, caused most oftenly by the stenosis of the major duodenal papilla (MDP) – 16-29%, pancreatitis – 5.4-27.4%, cicatricial stricture of extrahepatic biliary ducts – 5.3 -15%, parasitic liver diseases – 1.6-4%, as well as malignant neoplasms (cancer of the head of the pancreas, major duodenal papilla (MDP) cancer, bile ducts cancer).

The problem of treatment of patients with obstructive jaundice of various origins remains to be an urgent and largely unsolved problem. Decompression of the biliary system is one of the main goals and the most important component of therapeutic measures in these patients. In case of obstruction of the biliary system, cholangitis and liver failure, the surgical treatment is quite risky and is accompanied by high mortality rate. Surgical interventions in patients with obstructive jaundice that are performed under emergency conditions are accompanied by a large number of complications, and mortality rate amounts up to15-30%, which is 4 times higher than in cases when obstructive jaundice can be eliminated before the operation.

Portal hypertension (PH) remains one of the ill-studied syndromes of surgical hepatology. In most cases, doctors have to deal with patients in the stage of decompensation of the disease and the development of complications, accompanied by a high percentage of mortality and significant patient disability.If to take into consideration the social nature of the development of the pathology and its steady growth, these factors indicate a high relevance of this problem.

Obstructive jaundice

Obstructive l jaundice *is a life-threatening condition that develops as a result of the disturbancy of the natural passage of bile from the liver via the bile ducts (Fig. 55), further into the duodenum at any level, which is manifested by a complex of symptoms and occurs as a complication of diseases of the hepatopancreatoduodenal zone. Progression of jaundice results in the rapid development of liver failure, and in case of absence of any medical care the result is death.*

Clinical classification of obstructive jaundice.

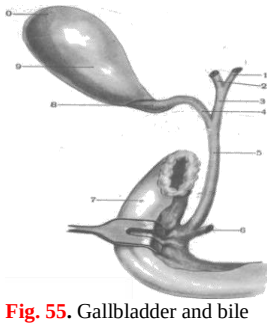


Fig. 55. Gallbladder and bile ducts: *1- ductus hepaticus sinister; 2 - ductus hepaticus dexter; 3 - ductus hepaticus communis; 4-ductus cysticus; 5 - ductus choledochus; 6 - ductus pancreaticuse*

I. According to the etiology.

1. *Benign*:

a) malformations (atresia, cysts of the common bile duct, duodenal diverticula, located near the major papilla of the duodenum, bile ducts' hypoplasia)

b) cholelithiasis (stones in the gall and hepatic ducts, Mirizzi syndrome, impacted stones of the major duodenal papilla);

c) inflammatory diseases (acute cholecystitis, cholangitis, chronic indurative or pseudotumor pancreatitis);

d) inflammatory and postoperative strictures and bile ducts' stenosis, inflammatory or cicatricial papilostenosis;

e) parasitic diseases of the liver and bile ducts.

2. *Malignant*: cancer of the head of the pancreas; cancer of the major duodenal papilla; cancer of the liver and common bile ducts; papilomatosis of bile ducts; cancer metastasis and porta hepatis lymphomas (hepatic hilar lymph nodes).

II. According to the time duration of the course (by the classification of E.V. Smirnov, 1974).

1. *Acute* – up to 10-14 days.
2. *Acute prolongated* – from 15 to 30 days.
3. *Chronic* – over 30 days.

III. According to the degree of outflow of bile.

1. *Complete*.
2. *Incomplete*, including *intermittent* (with valvular stones in the duct).

Clinical manifestations. When interviewing a patient, there are complaints on the main disease, the yellow colour of the skin, mucous membranes and sclera, discolored feces (in case of valvular stone the feces are either colored, or not coloured), the urine is dark, and the skin is itchy. The other symptoms such as pain, dyspepsia and weight loss are caused by the underlying disease, which caused the disturbancy in the outflow of the bile.

The earliest and most common symptom of obstructive jaundice ***with choledocholithiasis*** is pain.

Appetite disorders and dyspeptic symptoms are noted in later stages.

Pain is usually localized in the right hypochondrium or in the epigastric region. Its nature is varied: terebrant pain, gnawing pain, colic-like pain, sting pain, dull pain, moderate or strong pain, sometimes radiating to the back.

Along with the typical clinical picture of choledocholithiasis, the two forms of *atypical course* are distinguished:

1) choledocholithiasis that occurs with severe jaundice, but without the characteristic attacks of biliary colic;

2) choledocholithiasis, which is accompanied by jaundice, but without characteristic atacks of biliary colic, though, with pronounced common signs of inflammation – fever, chills and corresponding changes in blood; jaundice occurs 2 weeks after the temperature rises,; it is accompanied by chills. The time period of fever is usually short.

The jaundice ***of tumor nature*** is characterized by a long subfebrile condition, persistent loss of appetite, weight loss. An increase in body temperature is associated with the presence of cholangitis, or, which occurs more rarely, it is associated with the tumor destruction.

Pruritus is one of the fairly frequent symptoms. In case of obstructive jaundice, pruritus is a more persistent symptom and occurs with calculous jaundice in 50% of cases; in case of the cancer of pancreatic head it amounts up to 75% of cases. Pruritus in choledocholithiasis may occur before jaundice.

Complaints, which deal with other organs and systems, arise depending on the reasons that caused obstructive jaundice. Often in patients, who suffer from jaundice caused by gastro-intestinal diseases, abnormities of the general condition and dyspeptic disorders are detected. Before the jaundice occurs, the patients with obstructive jaundice caused by a malignant tumor of the hepatopancreatoduodenal zone, show general symptoms and signs, characteristic of tumors, the so called "slight signs syndrome" (general weakness, rapid fatigability, loss of appetite, weight loss), over a long period of time.

Table 18

Pathogenetic classification of jaundice

Types of jaundice, bilirubin fraction	**Cause-effect relations**
1	2
Suprahepatic jaundice (Cythemolytic icterus)	
Increased bilirubin production, indirect fraction (non-conjugated blood bilirubin)	
	Sickle cell anemia
Hemoglobinopathy	Deficiency of glucose-6-phosphate dehydrogenase
Fermentopathy	Spherocytosis
Erythrocyte structure disorder	Sideroblastic and B_{12}-deficient anemia
Ineffective erythropoiesis	Alcohol
Drugs and other chemical compounds	Mycoplasmosis, viral infections, sepsis
Infections	Blood transfusion
Incompatibility of blood groups and Rh factor	Artificial heart valves, hypothermia
Blood corpuscles destruction	Systemic lupus erythematosus, hemolytic anemia, hepatitis
Autoimmune chronic diseases	Leukemia
Intrahepatic jaundice	
Impaired consumption (capture) of bilirubin by the liver cells, indirect fraction	
Infection, intoxication, drugs	Posthepatitis, hyperbilirubinemia
Disruption of bilirubin conjugation	Gilbert's syndrome, Crigler-Nayar and hepatitis
Violation of excretion of bilirubin in the bile capillaries, direct hyperbilirubinemia – (intracellular cholestasis)	Syndromes of Dabin-Johnson, Rotor. Medicinal effects (anabolic steroids, aminazine, etc.) Benign Jaundice in Pregnant Women
Damage to liver cells (necrosis, inflammation, etc.), hyperbilirubinemia due to direct and indirect fraction	Hemochromatosis. α-1-antitrypsin deficiency. Wilson-Konovalov disease. Acute viral hepatitis, chronic hepatitis. Cytomegalovirus. Amebiasis. Leptospirosis. Infectious mononucleosis. Granulomatosis Primary liver cancer, metastatic liver cancer
Violation of the outflow of bile by extrahepatic bile ducts, direct hyperbilirubinemia (intrahepatic cholestasis)	Primary biliary cirrhosis. Sclerosing cholangitis. Medicinal cholestatic hepatitis
Subhepatic jaundice	
Disruption of the outflow of bile via the extrahepatic bile ducts	
Infectious diseases	Ascending cholangitis
Cholelithiasis	Choledocholithiasis
Trauma	Bile duct stricture
Malignant neoplasms	Cancer of the gallbladder, duodenal papilla.

	Pancreas cancer

The history of the disease must bear the following information:

1) to state the time when sclera jaundice, darkening of urine and stool discoloration appeared, as well as their connection with the pain attack;

2) if the patient applied for medical care and where he applied for the help to, or if the patient self-medicated, what medications were taken and what was the effect;

3) to state the way of previous examinations (diagnostics of calculous cholecystitis during previous examinations, laboratory tests data, the results of X-ray examination, instrumental methods of diagnosis);

4) if there is weight loss (characteristic of malignant tumors), lack of appetite (characteristic of viral hepatitis), fastidium; if the patient is an alcohol abuser (it is necessary to ask the patient's relatives and friends about it);

5) if there were any contacts with icteric patients; if the prodromal symptoms are present ("flu-like" condition, pain in the joints, dyspepsia, etc.) with hepatitis A (Botkin's disease);

6) if any injections were made to the patient (medicines, drugs); if blood transfusions, tattoos, dental procedures were performed; if he had occasional sexual contacts, trips abroad (perhaps he had malaria);

7)the information about previous surgical operations on the organs of the hepatopancreatoduodenal zone, the character of the surgeries, the information about the postoperative period;

8) if there are any symptoms of persistent hepatitis or cirrhosis of the liver.

The patient's past medical history should bear the information about the working conditions, occupational hazards, etc., ther should be obstetric and gynecological history of women. It is important to define the type of professional activity of the patient: whether he is in contact with hepatotoxic poisons, animals.

Objectively. When assessing the general condition of the patient with obstructive jaundice, fatique, irritability can be observed. The patient's general condition in case of choledocholithiasis depends on the severity of of the biliary colic attack. The condition worsens significantly with concomitant pancreatic necrosis. During the tumoral nature of the obstructive jaundice, the general condition is caused by the spread of the neoplastic process (the degree of tumor invasion into neighboring organs, the presence of distant metastases).

In case of obstructive jaundice, the skin and mucous membranes, depending on the etiology, are of orange-yellow, greenish-yellow or greenish-brown (greyly pale) colour. It is important to pay attention to the signs of scratching on the skin, which can occur with generalized skin itch, characteristic of cholestasis and hematomas in places of minor injuries. The signs of "vascular spiders" are characteristic of chronic liver damage. Patients may show xanthomas (subcutaneous cholesterol deposits) and xanthelasma (small formations of pale yellow color in the upper eyelid of about 6 mm, protruding above the skin surface due to the deposition of lipids).

The cardiac disorders are angina and bradycardia. Initial bradycardia in case of obstructive jaundice is caused by a reflex, which occurs during mechanical stretching of the intrahepatic and extrahepatic bile ducts, and further due to the action of bile acids on the vagus nerve tone.

Local status. On palpation of the abdomen in case of choledocholithiasis pain in the right hypochondrium is detected, with cholangitis form there is an increase in the liver and moderate pain. There may be positive Ortner symptoms (tapping on the edge of the palm along the right costal arch causes pain) and Murphysymptom (severe pain and involuntary breath-holding while inhaling are registered when the four fingers of the left hand are on the right costal arch and with the thumb pressing on the right hypochondrium towards the gall bladder). The gallbladder is usually not palpable due to its wrinkling, its increase is possible with edema and empyema.

With tumors of the pancreatic head against the background of jaundice, the gall bladder is considerably enlarged and painless (Courvoisier symptom) on palpation in the right subcostal area.

A particularly significant *increase in the liver* is observed with biliary tract tumors.

In case of cholangitis, *Charcot's triad* (fever, jaundice, pain in the right upper abdomen) is observed. In severe cases, sepsis can develop, during which the *Reynold pentad* may appear – Charcot's triad + hypotension and confusion, which indicates the development of biliary septic shock.

The leading clinical symptoms and syndromes*, characteristic of the obstructive jaundice, are abdominal pain and jaundice.*

If the cause of the obstructive jaundice is not corrected, the liver is gradually affected, the symptoms of liver failure join.

In accordance with the standard schemes, ***the plan of laboratory and instrumental examination*** of the patient with the obstructive jaundice in the surgical department includes and *identifies possible pathological changes.*

1. *Clinical blood test* – leukocytosis, accelerated erythrocyte sedimentation rate, left shift in the formula are observed in case of the purulent-inflammatory nature; incase of the malignant process anemia is likely to occur.

2. *Clinical urine test* – bilirubinuria is registered. In case of complete cholestasis, urobilin is absent in the urine, the signs of toxic nephropathy may be observed.

3. *Biochemical blood analysis*. The stage of cholestasis is defined by the amount of *total bilirubin and its fractions* circulating in the blood plasma (hyperbilirubinemia is noted with predominaying direct fraction – an increase in the rate of direct / total bilirubin). The markers of cholestasis are *alkaline phosphatase (AF)* and *γ-glutamyltranspeptidase (GGTP).* In case of cholestasis, the excretion of these enzymes into the bile is disturbed and their concentration in the serum increases. The activity of the pathological process in the liver (cytolysis) is assessed by the level of activity of *aspartate amino transferase* and *alanine transaminase* (AAT, ALT). AAT is a mitochondrial, and ALT is a cytoplasmic enzyme.

With a pronounced activity of the pathological process - cytolysis - the indicators of the activity of AAT and ALT are increased several times. In case of severe hepatocellular insufficiency, their activity may decrease sharply. The state of the synthetic function of the liver is determined by the amount of plasma proteins, *their fractions and prothrombin index (PTI).* All the albumin and 80% of globulins are synthesized in the liver. The decrease in the amount of plasma proteins (hypoproteinemia) is an indicator of the violation of the synthetic function of the liver. *Hypercholesterolemia, an increase in urea, the pathological lipoprotein X* may be present.

4. *Coagulogram*: characteristic hypocoagulation (lengthening of the clotting time, decrease in the prothrombin index) is present.

5. To verify the tumoral diseases of the liver and biliary tract, the definition of *tumor markers* - α-fetoprotein and carbohydrate antigen CA 19-9 is recommended. But it should be remembered that CA 19-9 is excreated exclusively with bile. Therefore, even a slight increase in the level of total bilirubin in the serum may be the cause of the increase in the marker level. Out of all the laboratory methods of research, the determination of *serological markers of viral hepatitis* is compulsory for each patient with jaundice.

6. After the clinical detection of jaundice and further laboratory confirmation, *instrumental appraisal of its nature* is necessary. A wide range of modern instrumental methods makes it possible to visualize the liver, bile ducts and surrounding tissues to determine the nature, cause and extent of the disorder in the bile outflow. The survey, first of all, begins with *non-invasive methods*, later moving on to *invasive*, but more diagnostically accurate. *Ultrasound examination methods* are used as a diagnostic screening.

In the presence of modern equipment, ultrasound examination makes it possible to differentiate obstructive jaundice from parenchymal in 80-97% of cases due to the characteristic echographic signs of biliary hypertension: expansion of the intrahepatic and extrahepatic bile ducts. In 90% of cases, ultrasound examination makes it possible to determin the level of the mechanical obstacle in the bile outflow. However, the diagnostic accuracy of this technique is 40-50%. On ultrasound examination, the bile ducts are dilated, the diameter of the common bile duct exceeds 0.8-1 cm. Concrements can be found in the gallbladder, but the accuracy of the determination of stones in the common bile duct is not high. In rare cases, diagnostic errors are possible, when a gallbladder tumor is interpreted as a

congestion of stones. With the tumor of the pancreatic head or pseudotumoral pancreatitis, the size of the organ increases. The focal lesions of the liver (metastases, hydatidomas) are detected.

7. On CT, all organs of the hepatoduodenal zone are visualized. CT is most effective in detecting choledocholithiasis, expansion of the intrahepatic and extrahepatic bile ducts being the signs of obstructive cholestasis, cysts and liver tumors (which size is 0.5 cm or more), acute pancreatitis, cystic pancreatic masses; and CT is less effective if it is necessary to differentiate chronic pancreatitis and cancer of the pancreas. The diagnostic capabilities of ultrasonography and CT are limited, so there is need in additional radiopaque examination of the bile ducts (endoscopic retrograde cholangiopancreatography (ERCPG), Percutaneous transhepatic cholangiography (PTCAG)) in order to clarify the diagnosis and determine the possibility of non-surgical jaundice treatment.

8. *Gastroduodenoscopy* is important both for the diagnosis of the main disease, which caused bile ducts obstruction, and for the evaluation of the accompanying changes in the stomach and duodenum. Gastroduodenoscopy is particularly effective in detection of the strangulated stone and major duodenal papilla cancer. Diagnosis of the strangulated stone is based on direct and indirect endoscopic signs of the disease. The disease is rarely diagnosed according to the direct endoscopic features, which include the visualization of the stone directly at the orifice of the papilla.

Most often, the diagnosis is made according to the indirect endoscopic signs: an increase in major duodenal papilla (MDP) in size up to 1-1.5 cm, its bulging into the lumen of the duodenum, and hemorrhages, hyperemia, edema, and erosion and fibrinous plaque in the papilla mucosa. In case of the strangulated stone, the orifice of the papilla either is open or is not differentiated. Endoscopic diagnosis of the cancer of major duodenal papilla usually does not cause difficulties if there is a polypous tumor with a bumpy or nodular surface of purple-red or crimson color with patches of ulceration. The tumor may be of different sizes; it thrusts out into the lumen of the intestine and occludes it. With the degradation of the tumor, contact bleeding develops. The diagnosis of the cancer of major duodenal papilla is confirmed by cytological examination of the biopsy material.

9. *Relaxation duodenography*. On X-ray examination, there are signs of indurative pancreatitis or malignant tumor of the pancreas, which grows into the duodenum – Frostberg symptom (deformation of the internal contour of the descending part of the duodenum in the form of the specular reflected digit 3). The duodenal diverticulum can be detected in major duodenal papilla zone.

10. *Endoscopic retrograde cholangiopancreatography (ERCPG)* is indicated if the results of ultrasound examination is questionable in cases when extrahepatic biliary system blockage is supposed. A fiberglass duodenoscope is used to cannulate the common bile duct and pancreatic duct. After the radiopaque substance is introduced into the duct, a series of shots is performed. The study is indispensable in the diagnosis of tumors and impacted gall stones in major duodenal papilla, of the level of obstruction of the bile ducts and in assessment of the anatomical and functional state of the biliopancreatic system. When using this technique, it is possible to diagnose small-sized pancreatic tumors. The cytological examination of the epithelium and the contents of the pancreatic duct is possible. Early diagnosis of the disease with the help of this first-priority radiocontrast method of research makes it possible to rationally solve the issues of treatment tactics, and, in addition, to reduce the time of examination of the patient.

11. Percutaneous transhepatic cholangiography (PTCAG) is indicated in cases when it is impossible to clarify the nature of the disease using the ERCPG method for a number of reasons (with biliary tract blockages in the portal fissure of the liver, etc.); if in patients with long-term obstruction of the bile ducts, the cholemic intoxication was expressed, it is advisable to combine a diagnostic study with intraductal therapeutic measures to eliminate it. At the same time the state of the intrahepatic ducts, the spread of their occlusion is determined. Under the local anesthesia, a long thin needle is inserted into one of the dilated intrahepatic ducts to introduce a contrast agent through the skin and liver tissue, under the ultrasound control.

12. In case of unclear nature of jaundice, in some cases it is advisable to use *diagnostic laparoscopy*. Differential diagnostics of jaundice is mainly based on the visual assessment of the color of the liver: its bright red color is a reliable sign of viral hepatitis, and green or greenish-brown liver color with smooth surface and expansion of the subcapsular bile ducts indicates obstructive jaundice. It

is difficult to establish the etiology of jaundice according to the endoscopic signs in the early stages of the disease, since it is known that the liver gains its characteristic color 2-3 weeks after the onset of the jaundice.

Laparoscopy is among the technically simple and relatively safe methods of research. The use of laparoscopy is contraindicated in case of extreme severity of the general condition of the patient, severe cardiac and pulmonary insufficiency, as well as in case of suspected massive adhesive adhesions in the abdominal cavity.

13. If pancreas disease is suspected, it is necessary to use *radionuclide scanning* in patients with obstructive jaundice of non-tumor nature. The method is used both to assess the degree of dysfunction of the pancreas, and to identify the focal lesions in it. Indications for scintigraphy are suspected indurative pancreatitis, as well as the impossibility to exclude cancer of the pancreatic head. Besides, scintigraphy is indicated in case of unclear nature of obstructive jaundice and if the patients, cannot undergo a radiopaque study of the bile ducts due to the severity of the general condition and because of the intolerance to iodine preparations.

Evaluation of the pancreas condition is carried out according to generally accepted criteria: location, shape, size and contour of the image of the gland, the nature and rate of accumulation of the radionuclide, the presence of zones with increased or decreased activity. The final conclusion about the nature of the lesion of the gland, according to the results of scintigraphy, should be done only after the results of multivariate analysis of the data obtained from clinical laboratory examinations and other diagnostic methods.

In case of suspected obstructive jaundice, ***the differential diagnostics*** should first be performed with parenchymal jaundice in viral hepatitis, as well as with obstructive jaundice of cholelithiasis and tumor nature (Table 10).

Treatment of patients with obstructive jaundice. *The choice of disease management.*

1. Obstructive jaundice should be eliminated within the first 10 days from the onset due to the threat of cholangitis and liver failure.

2. Treatment of patients with obstructive jaundice should be complex.

3. *Conservative measures* should not be long, should be carried out simultaneously with the examination of the patient and be regarded as pre-surgical preparation of the patient.

4. *Surgical treatment* of obstructive jaundice is performed as life-saving operation. Depending on the nature of the pathological process and the severity of the patient's condition, surgical treatment may be *radical* or *palliative.*

The goal of the ablative surgery is to eliminate the cause of cholestasis and biliary decompression, in case of palliative surgery only external or internal drainage of the bile ducts is performed.

In case of severe jaundice (rise of total bilirubin up to over 100 mmol/l), cholangitis, concomitant diseases in the stage of decompensation, *surgical treatment is carried out in two or several stages*. At the first stage, temporary external decompression of the biliary tract is necessary as a preparation for the main (second) stage of the treatment; in some cases, this procedure may be the final palliative treatment. At the second stage of the treatment the question of tactics for further management of the patient is resolved after stabilization of the patient's condition and clarification of the cause of obstructive jaundice.

Table 19

Differential diagnostics of jaundice

Differential character	Viral cholangiolitic hepatitis	Obstructive jaundice caused by a malignant tumor of the hepatopancreatoduodenal zone	Obstructive jaundice related to choledocholithiasis
Disease progression	Gradual, cyclically flowing, there is moderate intoxication at the fastigium stage being a manifestation of cytolysis syndrome	Progressive, intoxication may occur in the later stages	Acute, sudden, with a quick transition from health to illness
Pain	The feeling of heaviness in the right hypochondrium, epigastric region	Dull hurt, of varying intensity, occurring in the right hypochondrium or shingles. It may be absent at the initial stage.	Sharp acute pain, often short-term
Jaundice	From barely noticeable to pronounced with reverse development of the cyclic flow of the infectious process	Gradually progressive, gaining a greenish colour	Comes after the pain attack
Skin itch	Irregular, of varying intensity as cholestasis develops	The main symptom. Itching that increases and becomes worse at night. Scratching signs appear on the skin.	May appear as jaundice develops.
Dyspeptic disorders	Anorexia, nausea are the typical symptoms	Not present	Nausea ,vomiting
Body temperature	In most cases, is normal or subfebrile. May increase due to long-standing cholestasis with the development of inflammation in the biliary tract	Subfebrile , rarely normal. May be hectic due to the inflammation in the biliary tract or tumor metastasis	High or subfebrile. When the infection is activated, the temperature curve acquires a remitting or intermittent character.
Chills	Not present	Not present	Well-pronounced in case of cholangitis.
Acholia	Intermittent	Constant, as obturation increases	Short-term, at the fastigium of biliary colic, depending on the migration of the stone
Arthralgia	One of the typical symptoms	Is not specifically attributed to	Is not specifically attributed to
Bleeding	In severe and prolonged forms of the disease there is a tendency to cholemic bleeding, resulting from coagulation disorders. Hemorrhagic syndrome increases under the influence of surgical interventions	Hemorrhagic phenomena are accompanied by abundant blood loss, lesions of the Vater papilla are especially characteristic	May occur with long-lasted jaundice.
	Epidemic anamnes is mportant (blood	Before the development of jaundice, the general	The history includes calculous

Anamnesis	transfusions, parenteral manipulations in case the anti-epidemic regime is violated)	symptoms of tumors that last for a long time are: loss of appetite, fatigue, weight loss	cholecystitis with pain attacks, which are accompanied with jaundice
Prodromal period	The variants of the prodrom are: arthralgia, dyspeptic, asthenovegetative, catarrhal	Prodrom phenomena are not well-expressed	Prodrom phenomena are not well-expressed
Premorbid state	Within normal limits	Chronic diseases of the stomach, pancreas, biliary tract, genetic predisposition	The past history with pain attacks, dietary disorders, genetic predisposition
Age	Parenteral infection with viral hepatitis B, as well as A and B are observed in adults, the elderly people, as well as in children under one year of age.	More often after 40–50 years of age	More often after 20–30 years of age
General condition of the patient	Due to the severity of the disease and the severity of hepatonecrosis	In the presence of intense jaundice, the general condition is not disturbed very much: the patient's activity and appetite are sufficient	Depends on the severity of the liver colic attack. The condition worsens significantly with concomitant pancreatic necrosis.
Enlargement of the gall blader	In some cases, the gallbladder can be vivid on palpationin case of secondary sclerosis of the extrahepatic tracts and the development of obstructive syndrome	Increased painless gallbladder is an important diagnostic sign (Courvoisier symptom)	Often clearly palpable with obstruction of the cystic duct
Enlargement of the liver	Moderate equal increase, mild pain	Hepatomegaly, liver thickening, often lumpy surface	The liver is sharply enlarged according like with reactive hepatitis. When combined with choledocholithiasis accompanied with cholecystitis, and pancreatitis, signs of peritoneal irritation may appear.
Enlargement of the spleen	One of the symptoms of the disease	Is not specifically attributed to the disease, in some cases may be palpable.	Not enlarged
General blood analysis	The formula is normal, sometimes leukopenia is present. ESR is normal.	There is tendency to anemization. Neutrophilic leukocytosis, less often leukopenia may be detected. ESR is usually elevated.	Neutrophilic leukocytosis is present. ESR is significantly increased.
Blood bilirubin	Different degrees of increase in total bilirubin due to free and conjugated fractions. Bilirubin mono-glucuronide	Bilirubinemia increases consistently due to the conjugated fraction	Increased total bilirubin, mainly due to the conjugated fraction

	prevails.		
The activity of the enzymes: AAT, (ALT), ADH , 5-H, alkaline phosphatase	A marked increase in the activity of ALT, ADH, moderate 5-H, alkaline phosphatase	A considerable increase in the activity of 5-H, alkaline phosphatase, normal ALT indices	Moderate increase in the activity of 5-H and alkaline phosphatase
Dysproteinemia	Short-term moderate hypoalbuminemia, increase in the amount of α- and β-fractions of globulin	Progressive hypoproteinemia, reduction in albumin globulin ratio less than 1 with an increase in β-globulins	As a rule, proteinogram remains normal
Lipids	The level of phospholipids, β-lipoproteins and especially triglycerides increases	Significant increase in phospholipids, β-lipoproteins, moderate increase in triglycerides	Insignificant changes
Glucocorticoid test	Effective. As a rule, jaundice decreases when prescribing steroid drugs.	Ineffective. In some cases, leads to a decrease in jaundice due to anti-inflammatory action.	Ineffective
Keller's test	Ineffective	Ineffective (the prothrombin time after administration of 10 mg of vitamin K is normalized during the day)	Ineffective
Veltman Coagulation tape	Extension of the Veltman tape or the right shift	Shortening of the Veltman tape or the left shift	Normal indices with the long-term jaundice. There is tendency to shorten the tape or to the left shift
Thymol test	Positive test	Negative or elevated indices	Negative test
Serum cholesterol	Reduced or normal cholesterol levels with the decrease in the ester-conjugated level up to about 50%	Abruptly elevated	Normal or increased cholesterol in case of the normal ratio of total and ether- conjugated ones
Galactose load test (40g)	Positive test (release of less than 3 g of galactose in urine) (in case of severe forms of hepatitis)	Negative test	Negative test
Urine	Dark colour of the urine even before the skin and sclera gain the yellowish colour. Positive reaction to bile pigments, bile acids, urobilin.	Dark color with a greenish tint. A sharply positive reaction to the bile pigments and bile acids, negative reaction to urobilin.	Urine gains the color of dark beer. Positive reaction to the bile pigments and bile acids, in case of complete obturation – the reaction is negative for urobilin.
Coprological data	Positive reaction to stercobilin is not of a constant character. The reaction to the hidden blood is negative.	The reaction to stercobilin is negative, and as to hidden blood, as a rule, it is positive. The feces contain large amounts of undigested fat and muscle fibers.	There are large amounts of fatty acids in the feces. Neutral fat and latent blood are missing. In case of complete obturation, the reaction to stercobilin is negative.

Duodenal intubation	Duodenal juice is normally colored. It is possible to obtain three doses of duodenal contents in the presence of a positive reaction to stercobilin.	Duodenal juice is colorless; it does not contain pigments. The portions of "B" and "C" cannot be obtained. Sometimes blood and atypical cells are detected.	Duodenal juice is colorless, does not contain pigments. Portions of "B" and "C" cannot be obtained.
Accelerated chromatographic duodenal intubation	With chromatographic duodenal probing, a portion of "B" is of blue-green (malachite) color, portions "A" and "C" are of normal color	The same data are recieved as with the Einhorn probing.	The same data are recieved as with the Einhorn probing.
Plain radiography of the right hypochondrium	No diagnostic peculiarities are presented.	In very rare cases and in the presence of a large tumor, it is possible to obtain an image of a indurated head of the pancreas, displacement of the right kidney.	In 3-10% of cases the survey X-ray presents calculi in the gallbladder.
Radiography of the gastrointestinal tract	No specific findings	The deflation or expansion of the duodenum horseshoe and displacement of the duodenal arch, the deformation of the contours of the intestine and the pylorus. Defect on the medial contour in the descending part of the intestine in case of the tumor invasion, the symptoms of its narrowing and the displacement of the stomach upwards and to the left with large tumors.	No specific findings
Hypotonic duodenography	X-ray picture with hypotonia contains no deviations from the norm	The defects of filling, with ulcerous edges during tumor invasion into the mucous membrane in the projection of major duodenal papilla. An increase in the size of the major duodenal papilla up to 3 cm or more in case of tumor lesions. Deformity of the upper horizontal branch of the duodenum (Frostberg symptom)	When stones of the terminal part of the common bile duct are impacted, a rounded serration along the inner edge of the duodenum may be noted.
Endoscopic retrograde cholangiopancreatography	Normal picture of the biliary system. In case of the established diagnosis of viral hepatitis, this study is not indicated.	Endoscopic signs of the major duodenal papilla tumor are determined; incarcerated calculus, indirect signs of pancreatic head tumor. Major duodenal papilla cannulation fails with complete obstruction.	The endoscopic examination determins the swelling of the major duodenal papilla and its increase of more than 2 cm. With successful retrograde administration of a contrast agent, the X-ray picture of the choledocholithiasis is presented.
Ultrasonic investigation	Diffuse changes in the liver parenchyma, increase in the size of the	Drastic dialation of the intra- and extrahepatic bile ducts, enlargement of the gallbladder, tumor-	Drastic dialation of the intra- and extrahepatic bile ducts, the presence

	organ; there are no pathological changes as to the biliary system	like change in the pancreatic head	of a indurated echostructures in the cavity and in the lumen of the common bile duct
Scintigraphy of the liver with radioactive rose bengal (rose bengal ^{131}I)	A significant absorbing-excretory dysfunction of the liver, blood clearance reduction and slow down in the release of the administered drug from the liver	Maximal accumulation of the drug in the liver, the blood clearance almost does not change, the drug is excreted into the intestine in its maximum in 6-7 hours or more	The drug accumulates in the liver.It is not removed into the intestine in case of persistent or increasing jaundice
Laparoscopy	Cherry-colored liver, atonic gallbladder, it can be increased in its size	The liver is enlarged, it is of reddish brown, dark cherry or green colour. The gallbladder is stretched when the tumor is localized distal to the cystic duct. In case of tumors of the common bile duct and the gate of the liver, the gallbladder is distended. Sometimes metastases to the liver, cancer of the gallbladder are detected	Moderate enlargement of the liver, its color is greenish-yellow. Gall bladder adhesions with inflammatory changes in the wall are registered in most cases
Percutaneous perhepatic cholangiography (with long-standing, chronic jaundice — the method is dangerous)	The method of examination is complicated (dilatation of the intrahepatic bile ducts are not registered); in cases of success, the pathological changes are not detected	Drastic dialation of the intra- and extrahepatic bile ducts. The filling defect gives fuzzy contours or duct obturation	Drastic dialation of the bile ducts, half a month filling defects in the ducts in case of the presence of concrements

Principles of pathogenetically justified ***conservative therapy***. With obstructive jaundice, conservative therapy is prescribed for all the patients along with the examination regardless the diagnosis.

1. *Diet.* Table number 5a. The diet should ncessarily include dairy products (cottage cheese), vegetables, fruits, berries, juices. The content of easily digestible carbohydrates should not exceed the physiological norm. There should be plenty of water. Meals should be served in small portions and frequently. The food products should be either boiled or pureed.

2. *Detoxification*: infusion-transfusion therapy, hemodilution (neo-gemodez, reo-polyglukin, 5% glucose solution, 0.9% NaCl solution IV up to 2 l / day. The volume of transfusion is determined by the age of the patient and the state of the cardiovascular system), forced diuresis (20–40 mg of furosemide IV after infusion, mannitol), lympho-sorption, plasmapheresis, extracorporeal connection of the isolated liver, extracorporeal hemosorption, hyperbaric oxygenation.

3. *Antispasmodics* (papaverine hydrochloride 2 ml of 2% solution or no-shpa 2-4 ml of 2% solution, or phenicaberan 2 ml of 0.25% solution in IM 3 tid), painkillers (baralgin 5 ml / IV or IM 2-3 g / day).

4. To eliminate metabolic disorders, in particular hypoproteinemias and hypoalbuminiemias, *protein preparations* are used, the non-cleaved proteins (dried plasma, protein, albumin) the effective half-life in the body of which is 14-30 days are not choosen, but priority is given to amino acids, which are immediately consumed by the body to synthesize organ proteins. These drugs include casein hydrolyzate, Aminosol, Alvezin, Vamin, Aminon in the dose of 400-1000 ml. The deficiency of albumin must be started to be restored 3-4 days before the operation by transfusion of 10-20% solution in the amount of 100-150 ml per day and continue for 3-5 days after it.

5. To provide the patient with *energetic material*, as well as to stimulate the regenerative processes in the liver, to increase its antitoxic function and resistance to hypoxia of hepatocytes, it is recommended to administer concentrated glucose solutions in a volume of 500-1000 ml per day. To increase the efficiency of metabolism of intravenous glucose, it is necessary to add insulin, at that, its dose should be slightly higher than that of the standard, so that its metabolic effect is manifested. In addition, ATP, B vitamins complex, ascorbic acid and Mexidol are administered.

6. *Management of electrolyte disorders* is carried out by the introduction of isotonic solutions containing sodium, potassium, calcium, chlorine.

7. In connection with hypocoagulation existing in patients with breast cancer on the basis of the low level of blood coagulation factors, primarily of the prothrombin complex, *to normalize blood coagulation and prevent bleeding*; parenteral administration of vikasol 3 ml of 1% solution IM (administered during 4-5 days before surgery), 1% solution of calcium chloride IV, transfusion of frozen plasma.

8. *Control of the infection*. To stimulate nonspecific immunity, prodigiazon, imunofan or levamisole may be successfully used. Antibiotics that are actively excreted from the body with bile are ampicillin, gentamicin, cephalosporins, metronidazole, rifampicin, rimactane. In case of cholangitis tetracycline hydrochloride at 0.1 g IM 2-3 times / day or vibramycin (doxycycline hydrochloride) 0.1-0.2 g per 200-300 ml of 5% glucose solution or 0.9% NaCl solution IV by drop infusion (for 1-2 hours) 1 time / day are recommended.

9. When there is skin itch, cholestyramine is prescribed 4-8 g 2-3 times / day.

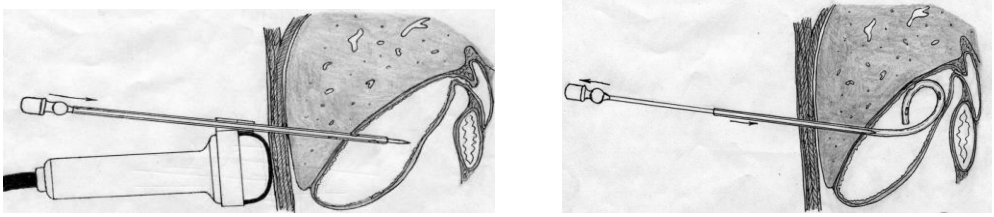


Fig. 56. Percutaneous puncture cholecystostomy
(Introduction of the drainage into the cavity of the gallbladder)

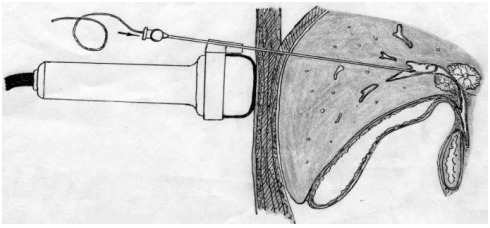


Fig. 57. Percutaneous puncture cholangiostomy
(Introduction of the drainage into the intrahepatic bile ducts)

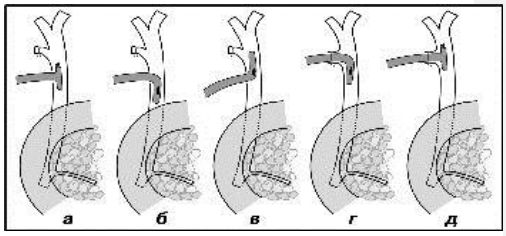


Fig. 58. Drainage of the choledochous duct using the method of: a - Kehr; b - Kerte; c - Vishnevsky; d - Holstead-Pikovsky; d - Abbe

Existing methods of surgical interventions and indications. The choice of the method of drainage of the bile ducts depends on the level of the blockage. *Methods of external drainage* of *the bile ducts are*: 1) in case of a low level blocking, if the patient has not previously undergone cholecystectomy, the cholecystostomy accompanied with ultrasound, CT or laparoscopic control (Fig. 56) are carried out 2) in case of high level blocking, the only decompression way of the bile ducts is percutaneous transhepatic cholangiography; (Fig. 57); 3) endoscopic nasobiliary drainage; 4) intraoperative (laparoscopic) - choledochostomy by Kehr, Curte, Vishnevsky, Holstead-Pikovsky, Abbe (Fig. 58); 5) hepaticostomy.

In case the external drainage is performed as a final palliative intervention, it must be supplemented with duodenostomy and subsequent external bypass surgery to return bile to the gastrointestinal tract.

Internal drainage methods (bilio-digestive bypass surgery):

- endoscopic papillosphincterotomy (EPST)
- different variants of endoprosthetics of the bile ducts (“indwelling drainage”; tantalic mesh prostheses)
- cholecystojejunostomy (Monastyrsky’s surgery) or cholecystoduodenostomy;
- choledochoduodenostomy (according to Yurash, Vinogradov, Flerken, Fenstrer)
- choledocho-, bihepatico- or hepaticojejunostomy on the defunctionalized loop by the Ru method, with the Sipl, Smith-Prader, and Vekler framed drainages.

*The choice of **an ablative surgery** method depends on the nature of the disease and the level of obstruction.*

1. *Cholecystocholedocholithiasis* – cholecystectomy, choledocholithtomy, choledochoscopy, external drainage of the choledochous duct. In the presence of II-III degree stenosis of major duodenal papilla (patency of less than 3 mm), intraoperative antegrade papillosphincterotomy or balloon dilatation, transduodenal papillosphincteroplasty, pre-or postoperative endoscopic papillosphincterotomy (EPST) is performed. In the presence of stenosis of within 20 mm choledochoduodenostomy is indicated. In case of jaundice caused by postoperative residual or recurrent calculi, EPCT is performed or, in the presence of external fistula – percutaneous lithoextraction is carried out under the control of choledochoscopy or fluoroscopy.

2. *Acute cholecystitis*, complicated by subhepatic infiltration, pancreatitis or cholangitis – cholecystectomy and external drainage methods.

3. *Benign diseases of the duodenum*, head of the *pancreas (parapapillary diverticulum, indurative pancreatitis, cysts)* – bilidigestive bypass surgery (cholecystoduodenostomy or colonic cholecystojejunostomy, with retroperitoneal access ahead of it, – Monastyrsky's surgery, which is complemented by inter-Brown's intestinal anastomosis; in case of pseudotumorous pancreatitis the patient can be completely cured due to the surgery).

Amputation of the major duodenal papilla (MDP) – is the removal of the distal part of papilla, the distal ends of the gall and pancreatic ducts are not removed. The surgery is indicated if it is proved that benign tumor is located in the distal papilla.

4. *Malignant neoplasms of the hepatopancreatoduodenal zone* – Papilectomy (Brunshvig's surgery), preserving (wedge-shaped) pancreatoduodenal resection, pancreatoduodenal resection, choledoch resection, hemihepatectomy, or palliative internal bilidigestion and external drainage operations.

In cases of tumor diseases, when ablative surgical treatment is not possible, ***minimally invasive interventional-radiological techniques*** are used – the intervention, carried out under the control of various types of radiation guidance – ultrasound and X-ray, which allows to improve the quality of life of patients. These include *balloon dilatation and stenting of the bile ducts*. Their aim is recanalization (i.e., restoration of the patency) of the blocked portion of the biliary system and implantation of remote or permanent tubular devices (drainages, stents) into the bile ducts, the devices are internal "struts", which ensure unhindered passage of the bile. The advantageous features of these interventions are as follows: they are well tolerated by the patients (the pain syndrome is absent after the operation, the patient is not limited in diet and physical activity), and they can be performed in aged and debilitated patients.

Postoperative rehabilitation of patients with obstructive jaundice, preventive services, diagnosis and treatment of possible postoperative complications.

1. *Regimen* depends on the amount of intervention. After the laparotomy access, bed rest is recommended up to 3-5 days, after minimally invasive interventions, active regimen is possible since the second day. Breathing exercises and physical therapy are performed.

2. *Diet*: clear liquid diet («0» diet) during 1-2 days. Then 20a diet – for 3-5 days, since the 6th day 5a diet is prescribed.

3. Mandatory components for obstructive jaundice in the postoperative period, in addition to the above mentioned, are the drugs that improve the functional state of hepatocytes and stimulate the process of their regeneration. These include the *hepatoprotectors* – Essentiale, Legalon, Carsil, Sirepar, glutamate and lipoic acid, methionine, Lipocaine, modern drugs - Heptral, Hepabene, Hepa-Merz and others. It should be remembered that these drugs should be administered after the elimination of cholestasis, not to cause failure in the adaptation of hepatocytes to the resulting changes in case of biliary hypertension and cholemia. Multicomponent therapy for obstructive jaundice should include vitamin therapy with B vitamins (B_1, B_6, B_{12}), A, C, E.

4. The drainage tubes are removed from the abdominal cavity in 3-5 days. The choledochous duct drainage is installed in the position of the siphon on the 9-10th day, and is removed 12-14 days after the control fistulocolangiography.

5. During the 3-5 days, detoxification, antibacterial, hepatotropic therapy should be continued.

Among the various ***complications of the postoperative period*** in case of obstructive jaundice, the two groups can be distinguished: *complications that are common for all the abdominal organs surgeries* (complications of the respiratory organs, cardiovascular system, infectious and inflammatory processes in the operation zones of the abdominal cavity, thrombosis and embolism) and *complications that are specific to the patients with obstructive jaundice* (hepato-renal failure, pancreatitis, cholemic hemorrhage, cholangitis). In patients with a short period of jaundice, *hypercoagulation* often develops before surgery, which requires

anticoagulant therapy in the postoperative period, whereas patients with a long-time jaundice are more likely to have *hypocoagulation*, which requires hemostatic drugs to be given to the patient. Both types of treatment should be carried out under the strict control over the blood coagulogram.

In cases when the hepatonephric insufficiency starts and develops, the following are prescribed:

1) bed rest;

2) strict diet, predominantly carbohydrate;

3) oxygen inhalation;

4) cleansing and siphon enemas and gastric lavage 2-3 times a day;

5) *hepatoprotectors* (Essentiale, Legalon, Carsil, Syrepar, Glutamic and lipoic acid, Methionine, Lipocaine, modern drugs – Heptral, Hepabene, Hepa-Merz and others; Glutamic acid intravenous infusion, 1 % solution up to 1000-1500 ml, are performed for blood ammonia binding);

6) administration of broad-spectrum antibiotics;

7) cardiac function and stable blood pressure support; maintenance of renal filtration at the proper level (intravenous infusion of polyglucin, neocompensant, etc.);

8) in severe cases, the use of steroid hormones (intravenous prednisone 20-40 mg per day) with simultaneous intramuscular administration of hydrocortisone (750-1250 mg per day) is recommended.

In case of an increase in ***renal insufficiency*** development, it is necessary to apply hemodialysis using the "Artificial kidney" apparatus.

Patients, who have suffered from the obstructive jaundice, should be under the follow-up monitoring. The patients should be examined by a family doctor and a surgeon twice a year, and even more often, if medically required. It is compulsory to monitor the clinical and biochemical blood indices. If necessary, the surgeon prescribes an ultrasound examination of the abdominal organs.

Portal hypertension

The syndrome of portal hypertension *(PH) develops with many diseases, a characteristic feature of which is the impaired outflow of blood from the veins of the portal system.*

The term "liver failure" is a collective concept that includes not only the impairment of one or several liver functions due to acute or chronic damage of the parenchyma, but also the impairment of the functional state of the vital organs, specifically the brain.

Classification of portal hypertension (PH) depending on the level of the block that caused the impaired porto-hepatic blood flow and PH (Saenko V.F., et al., 1997).

1. *Pre-hepatic blocking* – impaired patency of the portal vein or the branches that form it as a result of thrombosis, compression, cavernous transformation.

2. *Intrahepatic blocking* – impaired patency at the level of the liver due to the cirrhosis of the liver.

3. *Suprahepatic blocking* – impaired outflow of blood from the liver, as a result of hepatic veins thrombosis (Budd-Chiari syndrome).

4. *Mixed block* – violation of the patency of the extra-and intrahepatic portal vessels.

5. Improvement of the hepatopetal circulation in connection with the formation of arteriovenous fistulas in the spleen and liver with the transfer of pressure to the portal system.

Portal hypertension (PH) according to the level of pressure increase in the portal system.

1. *Portal hypertension of I degree – pressure of 250-400 mm WG.*

2. *Portal hypertension of II degree – pressure of 400-600 mm WG.*

3. *Portal hypertension of III degree – pressure of more than 600 mm WG.*

Endoscopic classification of the degree of varicosity of the veins of the esophagus and gastric cardia (N. Soehendra, K. Binmoeller 1997).

A. *Varicose veins of the esophagus*.

I degree – the diameter of varicose veins does not exceed 5 mm, they are elongated,and are located only in the lower third of the esophagus.

II degree – the diameter of varicose veins is 5-10 mm, they are sinuous, they are located in the middle third of the esophagus.

III degree - the diameter of varicose veins is more than 10 mm, they are strained, the walls are thin, the veins are located close to each other, red markers are visible on the surface of the veins.

B. *Varicose veins of the stomach*.

I degree – the diameter of the veins is not more than 5 mm, they are barely visible above the gastric mucosa.

II degree – the diameter of varicose veins is 5-10 mm, they are listed as having a solitary-polypoid character.

III degree – the diameter of varicose veins is more than 10 mm; they are thin-walled, the veins are listed as having a solitary-polypoid character and constitute a large conglomerate of nodes.

The classification of the liver cirrhosis (LC) is proposed by the World Hepatology Association (Acapulco, 1974) and WHO (1978).

A. *Morphological classification*:

1) micronodular liver cirrhosis (the node diameter varies from 1 to 3 mm);

2) macronodular cirrhosis (the node diameter exceeds 3 mm);

3) (non-complete) diffuse nodular cirrhosis;

4) mixed type (in which the nodes are of different size).

B. *Etiological classification*:

1) viral;

2) alcoholic;

3) medicinal;

4) secondary biliary;

5) congenital (hepatolenticular degeneration, hemochromatosis, α_1-antitrypsin deficiency, tyrosinosis, galactosemia, glycogenosis);

6) stagnant (circulatory failure);

7) disease and Budd-Chiari syndrome;

8) metabolic alimentary (application of enteroapocleisis, obesity, severe forms of diabetes mellitus);

9) liver cirrhosis of unknown etiology (cryptogenic, primary biliary, children's Indian type).

Badd-Chiari disease is the primary obliterating endoflebitis of the hepatic veins with thrombosis and their subsequent occlusion, as well as the abnormalities in the development of the hepatic veins, which cause the disturbances in the outflow of blood from the liver.

Budd-Chiari syndrome is a secondary disturbance in the outflow of blood from the liver in a number of pathological conditions that are not associated with changes in the liver vessels. At the core of it is stenosis or narrowing of the inferior vena cava at the level of the diaphragm, liver portion or of the suprahepatic portion, and of the hepatic veins. The main reason is thrombophlebitis, phlebothrombosis, perivascular edema and sclerosis, often in the background of caval hypertension. In 22% of cases, Budd-Chiari syndrome develops in case of the liver cirrhosis. There are acute and chronic forms of the disease.

Clinical manifestations of PH. Clinically, portal hypertension is divided *into **4 stages***.

Stage I – preclinical. The patients may complain of heaviness in the right hypochondrium, moderate flatulence and general malaise.

Stage II – pronounced clinical manifestations. Subjectively and objectively, the severity and pain in the upper abdomen, right hypochondrium, flatulence, dyspeptic disorders and hepato- and spleno-megaly are detected.

Stage III – pronounced clinical manifestations with the presence of all signs of portal hypertension, ascites, there is no pronounced bleeding.

Stage IV – the stage of complications. In such cases, the bleeding from the veins of the esophagus and stomach, bleeding from rectal veins, increased bleeding from the nose, gums (hemorrhagic diathesis) are observed. There is ascites, which is difficult to cure.

The underlying disease ***complaints*** – in accordance with the clinical stage of the disease.

Every third patient with PH has more or less pronounced clinical symptoms of encephalopathy – the syndrome that combines neurological and psycho-emotional disorders caused by metabolic disorders of the central nervous system, the latter are associated with *liver failure.*

Other organs and systems complaints. In varying degrees, unmotivated weakness, impaired productivity, fatigue, irritability, headache, insomnia are marked.

The *history of the disease* should contain the established presence of the following etiological factors of PH:

1) stress factors (mental, alimentary, toxic);

2) whether the patient suffered from hepatitis, liver cirrhosis (intrahepatic blocking);

3)inflammatory, post-traumatic, neoplastic diseases of the organs of hepatopancreatobiliary zone;

4) whether the patient was treated for alveococcosis or liver echinococcosis (intrahepatic or subhepatic blocking);

5) in case schistosomiasis is suspected, it is necessary to know if the patient visited the countries where the disease is particularly common. These include: Egypt, Greece, China, Portugal, Cyprus. It is important to remember that the most severe complication in case of schistosomiasis is periportal fibrosis with the development of portal hypertension, which can occur 10–15 years after being infected with *Schistosoma mansoni*;

6) congenital and acquired liver vascular anomaly;

7) congenital and acquired pathology of the inferior vena cava;

8) whether the patient was operated on for appendicitis – whether there were postoperative purulent complications (development of pylephlebitis);

9) childhood omphalitis or umbilical vein catheterization complications;

10) the age at which the symptoms of portal hypertension first appeared;

11) find out whether the patient was taking specific medications (*amiodarone, chlorpromazine, isoniazid, methotrexate, methyldopa, tolbutamide*), exposure to toxins (*arsenic, iron, copper*);

12) it is important to ask the patient whether he had fever and abdominal pain, as spontaneous bacterial peritonitis with PH may often be almost asymptomatic.

Patient's life history should contain the information about the patient's working conditions, occupational hazards, etc., in women – obstetric and gynecological history. It is important to find out the type of professional activity of the patient: whether he has any contacts with hepatotoxic poisons, animals.

Objectively. changes in the state of consciousness, intelligence, behavior, neuromuscular disorders, adynamia, lack of appetite are observed due to *encephalopathy*. A distinction is made between the *latent* stage, which can be distinguished according to the results of electroencephalogram and detection of ammonia in the blood, and four successive stages of clinical disorders:

1) *mild* – sleep disturbance, loss of attention, neurasthenia, euphoria, depression, irritability, minor tremor, changes in handwriting, etc.;

2) *moderate* – lethargy (timelessness, amnesia, lack of inhibition, fear, apathy; speech, hyporeflexia, torpor, ataxia)

3) *severe* – disorientation, stupor; deep amnesia, inability to count; inappropriate behavior, paranoia, rage; hyperreflexia, pathological reflexes, spasticity;

4) *coma* – lack of consciousness.

With the development of liver failure, ochrodermia and icteric mucous membranes, "spider veins" on the skin of the body, subcutaneous or submucosal hemorrhages, palmar erythema are observed.

Cardiac disorders in the patient with PH, angina and bradycardia may occur, with severe PH, there are symptoms of cardiac decompensation.

Local status. There are signs of collateral circulation: dilatation of the anterior abdominal wall (“arachnogastria”), dilated hemorrhoidal veins, ascites, paraumbilical hernia (Fig. 59).

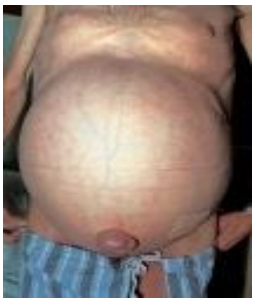


Fig. 59. “Arachnogastria”, ascites, paraumbilical hernia

There is hepatomegaly (or, on the contrary, liver size reduction), liver is painful on palpation, splenomegaly, skin hemorrhagic syndrome, edema, metabolic disorders and impaired microcirculation in the form of skin manifestations (pigmentation, purpura, spider veins) or bleeding from the nose, gums, gastrointestinal and subcutaneous bleeding.

Banti syndrome *(non-cirrhotic portal hypertension) isa leading symptom with PH.*

In accordance with standard schemes, the plan of laboratory and instrumental examination of the patient with PH at the surgical department includes:

1. *Clinical analysis of the blood*. The number of red blood cells, color indicator and ESR indirectly indicate the state of the liver functioning as a whole. In the later stages of the disease anemia and an increase in ESR are often observed. With hypersplenism, there is a decrease in leukocytes of less than 4×10^9/l, in platelets – less than 1×10^8/l, besides, pancytopenia can be observed as well. In patients with liver cirrhosis, there is a decrease in the number of platelets. Patients with hemochromatosis, usually present blood with high hemoglobin level in combination with low hemoglobin concentration in erythrocytes.

2. *Urine analysis*. In case of liver cirrhosis, it is important to determine the parameters which characterize the function of the kidneys (protein, white blood cells, red blood cells, creatinine, uric acid). This is important, since 57% of patients with cirrhosis and ascites present renal failure (clearance of endogenous creatinine is less than 32 ml/min with normal serum creatinine levels). Bilirubinuria is possible. With complete cholestasis, urobilin is not present in the urine, there are possible signs of toxic nephropathy. In patients with edematous ascitic syndrome, it is necessary to observe daily diuresis.

3. *Biochemical blood analysis*. With compensated liver cirrhosis, the activity of liver enzymes may be normal. A significant increase in ALT, AAT, γ-glutamyltransferase is observed in alcoholic hepatitis with further development of cirrhosis; and primary biliary cirrhosis produces a sharp increase in Alkaline phosphatase. Besides, in patients with liver cirrhosis, the total bilirubin increases and albumin decreases. The ammount of aminotransferase is always reduced in the terminal stage of cirrhosis (there are no functioning hepatocytes and no enzymes). There are indicators of unfavorable prognosis: bilirubin is above 300 μmol / l; albumin is below 20 g / l; the prothrombin index is less than 60%.

4. *Coagulogram*: characteristic hypocoagulation (lengthening of clotting time, reduction of prothrombin, of prothrombin index and fibrinogen).

5. *Determination of antibodies* to chronic hepatitis viruses. Antibodies to viruses that cause chronic hepatitis should be investigated, even if cirrhosis of the liver is directly related to the chronic alcohol intoxication.

The diagnosis of viral hepatitis B (HBV). The main marker is HBsAg, the HBV DNA. The presence of HBeAg indicates the activity of viral replication. When HBeAg disappears and the antibodies to it (anti-HBe) appear, this characterizes the termination of HBV replication and is interpreted as a state of partial seroconversion. There is a direct link between the activity of chronic viral hepatitis B and the presence of viral replication, and vice versa.

The diagnosis of viral hepatitis C (HCV). The main marker is the presence of antibodies to HCV (anti-HCV). The presence of a current infection is confirmed by the detection of the HCV RNA. Anti-HCV are detected during the recovery phase and are no longer detected 1-4 years after acute viral hepatitis. An increase in these indicators indicates chronic hepatitis.

Determination of the levels of IgA, IgM, IgG in serum. The elevated levels of serum IgA, IgM, IgG often manifest alcoholic lesions of the liver, primary biliary cirrhosis and autoimmune diseases, but they do not always change regularly during treatment, and, therefore, the results of these studies in some cases are difficult to assess.

6. To determine the functional state of the liver, the generally accepted score is the one presented by Child-Pugh and Mansour A. et al (1997), in which there are the following groups: A (5-6 points), B (7-9), C (10 or more) (Table 20).

Table 20

Criteria for assessment of the functional state of the liver
(Classification of the severity of liver disease according to Child-Pugh)

Indicant	1 point	2 points	3 points
Bilirubin (μmol / L)	<34	34-51	>51
Albumin (mg / l)	>35	28-35	<28
Ascites	Doesn't have	medication-based treatment	refractory
Encephalopathy	Doesn't have	grade 1-2	grade 3-4
INR (international normalized ratio)	<1.7	1,7-2,2	>2.2

Mansour A. et al, 1997; Propst A. et al, 1995

Indicant	Class A	Class B	Class C
Points	5-6	7-9	10-15
Viability index during 1 year	100 %	81 %	45 %
Viability index during 2 years	85 %	57 %	35 %
Lifespan	15-20 years	3-15 years	1-3 years
Mortality in abdominal surgery	10 %	30 %	82 %

7. In case of *liver failure*, the blood test presents hypoproteinemia, violation of the ratio of protein fractions with coarsely dispersed components (globulins) predominating, hyperbilirubinemia, decrease in the level of fibrinogen, prothrombin, cholesterol. The level of transaminases is increased. Thymol test, as a rule, shows an increase, sublimate test – significant reduction. Impaired water and electrolyte metabolism (a decrease in the level of potassium and sodium in the blood plasma) is detected quite early.

Instrumental methods of diagnosis

1. *Esophagogastroduodenoscopy* makes it possible to diagnose portal hypertensive gastropathy (characteristic mosaic pattern of the mucous membrane), esophageal varicose veins (visualized as sulcate filling defects in the lower esophagus), to evaluate the effectiveness of conservative and surgical treatment. Besides, when using this study, the severity of trophic changes in the mucous membrane of the esophagus and the walls of the veins can be determined, as well as the risk factors for bleeding (esophagus venous dilation, erosive esophagitis, telangiectasia and red markers: "red cherry" spots, hematocystic spots). When the varicose veins of the esophagus or stomach are detected, the diagnosis of portal hypertension is beyond doubt.

2. *Ultrasound examination of the abdominal organs*. The ultrasound examination of the abdominal organs can detect natural portocaval collaterals, esophageal varicose veins. Ultrasound examination makes it possible to determine the size and structure of the liver and spleen, the presence of ascitic fluid in the abdominal cavity, the diameter and increased

echogenicity of the portal vein, hepatic veins and inferior vena cava; sulcate splenic and superior mesenteric veins, to reveal the places of compression of the portal and inferior vena cava.

Ultrasonographic characteristics of portal blood flow disorders are determined by the level of its blockade. The *prehepatic blocking* is characteristic of: the distention of the inferior vena cava, negative test of dosed compression; dilatation of proper hepatic veins, intrahepatic anastomoses between the proper hepatic veins. Signs of an *intrahepatic blocking* are: an increase in liver echodensity, depletion of the intrahepatic portal pattern, an expansion of the stem and lobe branches of the portal vein. The *posthepatic blocking* is characterized by: localized narrowing of the veins of the portal system, absence of the stem of the portal vein, detection of an additional vascular network; additional echo-formation in the lumen of the portal system; compression of the veins of the portal system from the outside (if additional echostructures are detected outside the vessels).

3. *Ultrasonic Doppler examination of hepatic and portal veins*. Due to this method, it is possible to obtain the information about hemodynamics in the portal system and collaterals, which is under development. It also makes it possible to detect the changes in the direction of blood flow in the hepatic veins and the hepatic segment of the inferior vena cava (it may be absent, reverse or turbulent). It is also helpful in evaluation of the quantitative and spectral characteristics of the blood flow and estimation of the absolute values of blood volume in certain areas of the blood vessels.

4. *X-ray computed tomography*. This study provides information about the size, shape, condition of the liver, the density of the organ parenchyma. Visualization of the intrahepatic vessels of the liver depends on the ratio of their density to the density of the liver parenchyma. So, normally, the vascular stems of the liver are visualized in the form of oval and elongated formations; however, when the density of the liver decreases, the image of the vessels merges with the parenchyma.

5. A highly informative method in the diagnosis of varicose veins is multilayer *CT angiography*.

6. *Magnetic resonance imaging* makes it possible to obtain an image of the parenchymal abdominal organs, large vessels, retroperitoneal space. Due to this method, diseases of the liver and other organs can be diagnosed; there is a possibility to determine the level of blockade of the portal blood circulation and the severity of collateral blood flow, the condition of the abdominal veins of the liver and the presence of ascites; to evaluate the function of splenorenal anastomosis after surgical treatment.

7. *Hepatoscintigraphy* makes it possible to determine (according to the distribution of the pharmaceutical preparation) the absorbing-excretory function of the liver, its size and structure. The severity of cirrhotic changes in the liver can be estimated. Besides, with portal hypertension, radioisotope drugs accumulate not only in the liver, but also in the spleen.

8. *Transcaval hepatic venography* is performed to determine the patency of the hepatic veins and the hepatic segment of the inferior vena cava (with suspected Budd-Chiari syndrome); to evaluate the effectiveness of surgical therapy. Contrast medium is injected through the catheter, installed into the inferior vena cava to the orifices of the hepatic veins, or by transjugular catheterization of the hepatic veins.

9. *Portography*. Angiography is a highly informative method of research, which makes it possible to judge about the state of the splenoportal bed. With the help of serial images obtained during angiography, it is possible to study all phases of the blood flow in the liver – arterial, parenchymal and venous. The study of images of the venous phase makes it possible to assess the state of the portal system.

To visualize the portal vein and its ducts, two principal approaches are proposed and used: via the arteria and via the vein.

10. During *splenoportography*, a water-soluble contrast agent is injected into the spleen (verografin, hepac) and a series of shots are taken. In case the patient suffered from splenectomy previously, portography can be performed with cannulation of the umbilical vein. This method

makes it possible to identify the level of the blocking. In 1-2% of patients, bleeding from the spleen can occur and the phenomenon of false thrombosis of the portal vein can be recorded – "slipping" of the contrast agent through the collaterals before it reaches its portal vein. Splenoportography can be performed by direct puncture of the spleen. Currently, this method is not applied: catheterization of the inferior vena cava or hepatic veins is performed.

11. *Splenomanometry* is used to establish the form of portal hypertension, to determine the amount of pressure in the portal system and assess the state of the vascular bed. For this purpose, the puncture of the spleen is performed with delaying of breath in the VIII intercostal space along the mid-axillary line. When receiving blood from the needle, the Waldman apparatus is connected up to it and pressure is measured. In a healthy person, it is 150-200 mm WG. In case of portal hypertension, the pressure can reach more than 600 mm WG. The increase of the pressure up to more than 300-350 mm WG is a risk factor for variceal hemorrhage.

12. *Measurement of the level of hepatic vein wedge pressure in the portal vein system and its branches.* The value of the detected posthepatic vein wedge pressure (PVWP) corresponds to sinusoidal pressure. To determine the sinusoidal pressure, the cardiac catheter is introduced through the ulnar vein, the right heart portion and the inferior vena cava and into one of the hepatic veins until the wedged intrahepatic vein of a small diameter. The intra-splenic pressure (ISP) and the free portal pressure (FPP) are also measured. The intra-splenic pressure (ISP) is determined due to the puncture of the spleen and it displays presinusoidal pressure. The same pressure reflects free portal pressure (FPP), which is determined due to the catheterization of the mesentric vein or the main stem of the portal vein. The normal values of the intra-splenic pressure (ISP) and free portal pressure (FPP) equal 16-25 mm of mercury, posthepatic vein wedge pressure (PVWP) – 5.5 mm of mercury.

13. *Laparoscopy* is used in doubtful cases. It is important to identify the nature of pathological changes in the liver, biopsy has to be performed; the severity of splenomegaly and the degree of varicose veins of the portal system are to be determined; the possible ascites is to be verified.

14. *Liver biopsy*. During the study of liver biopsy specimens in patients with portal hypertension, various histological results can be obtained, which are determined by the disease that led to circulatory disorders in the portal vein system. If no pathological changes are detected in the biopsy specimens, it is necessary to presuppose that the patient has prehepatic blocking.

Differential diagnostics, in cases of suspected portal hypertension (PH) and taking into consideration the clinical findings and the existing complications, should be performed between:

a) the diseases of the blood system (Gaucher disease, Chauffard – Minkowski disease, Verlgof disease, chronic myeloid leukemia, spleen lymphocytomas);

b) gastroduodenal bleeding caused by peptic ulcer and other causes;

c) malignant neoplasms of various localization, accompanied by ascites;

d) decompensation of the cardiovascular system.

Treatment of patients with PH. *The choice of treatment tactics*. Treatment of patients with PH should include the following:

1) elimination of life-threatening complications;

2) portal pressure lowering;

3) correction of the dysfunction of the portal system organs: liver failure, immunoreactivity, collateral blood flow.

Principles of pathogenetically justified conservative therapy.

1. *Diet therapy*. In case of acute hepatic encephalopathy, it is necessary to reduce the amount of protein up to 20-30 g per day with a gradual increase in the amount and predominance of vegetable proteins. With chronic encephalopathy, the amount of protein in food is to amount to 60 g per day.

2. *Drug reduction of portal blood flow presupposes the use of drugs that affect the portal system blood flow*. The most common groups of drugs are:

1) *hormones* (reduce the arterial blood flow in the organs)

2) *Pituitrin* is administered intravenously at a dose of 20 U per 200 ml of 5-10% glucose solution during 50 minutes, in 30-40 minutes additionally 10 U of the preparation for 20 ml of glucose oad (pituitrin is effective in the narrowing of the arterioles of the abdominal organs cavities that causes a decrease in hepatic blood flow and a decrease in portal pressure up to 40-60 %);

3) *Vasopressin* (Terlipressin, Remestyp – synthetic vasopressin analogs) is administered intravenously 20 IU in 100 ml of 5% glucose solution during 10 minutes;

4) *Somatostatin* is administered in the dose of 25-50 μg / h in 5% glucose solution oad for 2-5 days. (affects the smooth muscles of the mesenteric vessels and increases the resistance in the arterioles of the internal organs, thereby reduces portal blood flow; in case of arterial hypertension, the drug is not recommended; synthetic analog of Somatostatin – Octreotide, Sandostatin);

5) *nitrates* (*Nitroglycerin, Nitroprusside, Isosorbide*) – are venous and arterial vasodilators, they reduce total peripheral resistance, and lead to the deposition of blood in peripheral vessels, reduce the flow of blood into the portal system; nitroglycerin is administered intravenously – 40-400 μg / min, it can also be administered under the tongue – 0.6 mg every 30 minutes (it reduces portal pressure by 30%, and is used alone or in combination with Pituitrin, Nitroglycerin in combination with Vasopressin can lead to significant decrease in portal pressure; to reduce the portal hypertension, *Isosorbide Dinitrate* (*Cardiket, Nitrosorbide), Isosorbide Mononitrate (Monomac, Olicard)* can also be administered;

6) *β-adrenergic blockers* (*Propranolol, Anaprilin, Obzidan*) are prescribed to reduce the effects of portal hypertension, 20-40 mg per day for a long time (months, years). When taking β-blockers the heart rate is expected to be reduced by 25% from baseline; these drugs can reduce portal pressure by about 40%, the effect is achieved by reducing cardiac output and reducing heart rate;

7) *diuretic therapy* – Spironolactone (Veroshpiron) is prescribed 200-400 mg per day in combination with Furosemide (Lasix) – 40-240 mg per day in condition of a salt-free diet; additional intravenous hemodesis and polyglucin are recommended with diuretics;

8) *reduction of hyperammonemia – Lactulose* (*Duphalac, Normase*) are taken in the form of syrup, 30 mg 3-5 times a day after meals until the laxative effect appears; the drug creates an acidic environment in the colon, binds; and reduces the formation of ammonia in the intestine, reduces aromatic amino acids with cerebrotoxic action; in case of constipation, cleansing enemas with of magnesium sulfate solution are helpful; *Ornithine-Aspartate* (20–40 g per day or 9–18 g orally) and *Ornithine-Ketoglutarate* are used to enhance the neutralization of ammonia in the liver; sodium benzoate (10 g / day) is used to bind ammonia in the blood;

9) *antibacterial therapy* - it must be remembered that all patients with cirrhosis of the liver and bleeding from varicose veins of the esophagus and stomach, have a high probability of developing bacterial infections, so prophylactic antibiotics can reduce this risk; before the pathogen is identified, Cefotaxime (Claforan) – IV 1.0 g 3 times / day – can be prescribed;

10) *decrease in the inhibitory processes in the central nervous system* – antagonist of benzodiazepine receptors, Flumazenil, a single dose of 0.4–1 mg, can be prescribed.

Management of liver failure is the most difficult problem in the treatment of patients with PH (portal hypertension). In case of acute liver failure, it is necessary to carry on an intensive infusion therapy and administer *10% glucose solution with insulin, vitamins* (B_1, B_6, B_{12}, C, Retinol, lipoic acid, etc.), *hepatoprotectors* (Essentiale, Legalon, Carsil, Sirepar, glutamic and lipoic acid, Methionine, Lipocaine, drugs of today – Heptral, Hepabene, Hepa-Merz, etc.), protein drugs, drugs to improve the rheological properties of blood, anti-oxidants. When the immunological indices fall, it is necessary to provide immunostimulants (Thymalinum, Thymogen (Oglufanidum), Immunal).

In case of autoimmune processes: chronic active hepatitis (autoimmune, caused by HBV, drug-induced, cryptogenic), primary biliary cirrhosis, cryptogenic liver cirrhosis – *immunosuppressants Pprednisolone, Azathioprine)* are to be prescribed. The initial dose of

Prednisolone is 30 mg per day, when the effect is reached, the dose is reduced up to 5, 10, 15 mg of Prednisone with the dosage of 10-20 mg combined with 50-100 mg of Azathioprine, then follows the maintenance doses: Prednisolone – 5-10 mg, Azathioprine – 25-50 mg.

Corticosteroids are administered to treat the patients with chronic hepatitis and if the autoantibodies and viral markers are detected, *antiviral drugs (Interferon*) are used in case of a viral infection.

Prevention of fibrosis is important in the treatment of patients with cirrhosis. In order to reduce collagen synthesis, Colchicine is used (1 mg per day, 5 times per week).

The treatment of *hepatic encephalopathy* should presuppose a combination of the following aspects: amelioration of risk factors, drug therapy, diet therapy. Amelioration of risk factors includes acute bleeding control, anemia correction, normalizing of electrolyte balance, depletion or catharsis.

Current approaches to operative interventions and surgical indications

The surgical indications for the patients with portal hypertension syndrome are: esophageal or cardiac varicose veins with or without bleeding, splenomegaly with hypersplenism, and ascites.

There are more than 200 operative interventions' approaches. They can be subdivided into a number of groups:

1) *correction of PH due to the collateral blood outflow*:

a) organ portocaval anastomoses (PCA), gastrorenopexy;

b) vascular veno-venous portocaval anastomoses:

- direct portocaval anastomoses (PCA) (Ekk, 1877);
- mesenteric-caval anastomoses (MCA) (Bogoraz, 1925);
- splenorenal anastomoses (SRA, Whipple, Blakemore, Lorl, 1945);
- distal splenorenal anastomoses (DSRA, Warren et al., 1974);

2) *reduction of the portal system blood supply*:

a) splenectomy;

b) ligation and endovascular occlusion of the arteries (more often – splenic);

3) *surgical operations aimed at intensification of the regenerative processes in the liver* (given that cirrhosis is a dual process - dystrophy with the development of connective tissue and intensified regeneration):

a) the autonomic nervous system surgeries – periarterial sympathectomy of the hepatic artery (Malle-Gui) in case of initial liver cirrhosis and chronic active hepatitis;

b) atypical marginal liver excision;

c) liver electrocoagulation (D. V. Usov);

d) laser irradiation with extraperitonization of the right lobe of the liver (A. V. Beresnev et al.)

4) surgical operations aimed at separation of the anastomotic connections of the veins of the esophagus from the veins of the portal system:

a) Tanner surgery and its modifications;

b) subcardiac gastrotomy with insertion and ligation of the veins of the cardia of the stomach and the lower third of the esophagus (operation of choice at the height of bleeding)

c) Crile procedure and its modifications;

5) *ascites surgeries*:

a) deflection of the ascitic fluid into the retroperitoneal tissue;

b)deflection of the ascitic fluid into the vein (according to Ryuot, A.E. Borisov, peritoneovenous bypass surgery with Levin valve)

c) external drainage of the thoracic duct – lymphovenous anastomoses;

d) Kalba-Oppel-Shalimov operation;

6) *the radical method* to treat PH is ***liver transplantation***, which allows to facilitate the course of portal hypertension, prevent the repeated bleeding, reduce the manifestations of ascites and encephalopathy; this operation is performed according to the Child-Pugh scale.

Current principles of treatment of PH based on surgical staging and its complications.Taking into consideraton the polysyndrom character of the disease manifestations, there is no unique method of surgical treatment as it cannot be based on one of the syndrom-complexes, according to which the disease is called.

The basic principle of treatment of PH and its complications is gradual elimination of disease syndromes using the method of complex conservative and surgical treatment, i.e..minimally invasive methods that are consistently applied and are pathogenetically substantiated with mutual force-induced action.

1) peritoneovenous bypass surgery (in case of ascites);

2) lymphatic draining operations (acute liver failure, ascites, cholestasis, bleeding);

3) endovascular chronic embolization of the hepatic arteries – EHA (ascites, bleeding, portal decompression, reduction of lymph formation in the liver);

4) endovascular embolization of the splenic artery - ESA (hypersplenism, an alternative to dangerous splenectomy), the combination of EHA and ESA (increased decompression and homeostasis-correcting effect);

5) balloon occlusion of the inferior vena cava (BOIVC) according to A.E. Borisov (reduction of caval hypertension and elimination of the hepatic postsynusoidal blocking – ascites, bleeding)

6) endovascular occlusion of varicose veins (VV) and endoscopic sclerotherapy – prevention and treatment of hemorrhages from varicose veins of the esophagus and stomach;

7) laparoscopic ascito-corrective and decompressional interventions;

8) endoscopic biliodecompressive interventions (microcholecystostomy, percutaneous subhepatic bile duct drainage – with hepatic and extrahepatic jaundice);

9) in case of high portal pressure (PP) and insufficient decompression at the previous stages – EHA or ESA and DSRA (distal splenorenal anastomosis), MPA (magnetic resonance angiography) can also be performed;

10) subcardiac gastrostomy; gastrorenopexy; azigo-caval anastomosis.

Treatment of splenomegaly and hypersplenism presupposes drug immunostimulation, administration of immunomodulators (Thymalin, Timogen, Immunal), the stimulation of hemopoiesis (implantation of hematopoietic cells of the fetal liver). In cases of pronounced impaired hemopoiesis, thrombocytopenia, surgical treatment of hypersplenism – splenectomy – is adminnistered. A less traumatic method of treatment is endovascular embolization of the splenic artery.

Treatment of ascites in patients with portal hypertension should include the following approaches: diet therapy, the use of diuretics, evacuation of ascitic fluid.

Diet therapy for patients with ascites is based on sodium restriction (no more than 20 mmol per day). *The use of diuretics – thiazide diuretic, furosemide, ethacrynic acid* – should be supplemented by the administration of potassium preparations. Doses of potassium-sparing diuretics (*Spironolactone, Amiloride, Triamteren*) should be selected individually, taking into account body weight, clinical test indicators, the stage of the disease. In cases of resistant ascites, *paracentesis* is performed, the indications for which are: tense ascites, combination of ascites with edema, prothrombin level of above 40%, serum bilirubin level of below 170 mmol / l, platelet count of more than 40,000 in 1 mm, creatinine level in blood of less than 3 mg %, daily excretion of sodium of more than 10 mmol. The procedure involves the removal of 4-5 liters of fluid per day with the administration of albumin IV. *Total paracentesis* presupposes a single total removal of fluid with the simultaneous administration of albumin, 6 g per 1 liter of fluid. Contraindications to total paracentesis is the terminal stage of the disease. *Peritoneum venous bypass* provides long-term constant reinfusion of ascitic fluid into the venous system. For this purpose, the method of Le Veen is applied ("peritoneal jugular shunt" surgery with the usage of the pressure valve). A positive effect in the treatment of ascites is observed with *endovascular embolization of the splenic artery*.

Postoperative complications:

1) *acute*: acute hepatic impairment, vascular orifice thrombosis, thrombohemorrhagic syndrome;

2) *subacute*: postoperative ascites;

3) *chronic*: encephalopathy.

Emergency medical assistance in case of PH complicated with bleeding from oesophageal varicosity and cardiac orifice varicosity. Bleeding from phlebectasia of the esophagus and stomach is characterized by massiveness and a tendency to relapse. To control the acute bleeding is a complex task. The application of *the Blackmore obturator probe and carrying out of conservative hemostatic therapy (ε-aminocaproic acid, Dicynone, Trasylol, Fibrinogen, Vicasol, calcium chloride solution, Gelatinol, Polyglucin, transfusions of erythromass and plasma*).

The obturator probe is recommended to be used for 2-3 days with an interval of 1 hour per day in order to prevent complications (formation of pressure sores in the wall and ruptures of the esophagus). The method of installment of the obturator probe is as follows: anesthesia of the nasopharynx is performed, the probe is inserted through the nose into the stomach; 200-250 ml of air is injected into the lower cylinder with the help of Janet's syringe, the probe is removed until tight; 200-400 ml of air is pumped up to into the upper cylinder.

The advanced direction of *local hemostasis* is the performance of endoscopic sclerotherapy. The method is applied and is effective at the height of bleeding, and it is performed to achieve sustainable hemostasis. For endoscopic sclerotherapy, the solution of Thrombovar, 96% alcohol, sodium morrhuate, ethanolamine, etc. are used. In case of acute bleeding, intravasal injection |of the drugs is effective. To prevent the relapse and to achieve stable hemostasis, paravasal administration of drugs and several courses of sclerotherapy (one time per week) are indicated. Lasting effect is observed when sclerotherapy is used together with the obturator probe. The following complications may occur during sclerotherapy: fever, chest pains, lung and pleura diseases, erosions, ulcers, strictures of the esophagus, esophageal perforation, bleeding from erosions, ulcers, in case of esophageal lesions – mediastinitis, aspiration.

An effective method to control the acute bleeding and to prevent the relapse is to use endovascular embolization of esophageal varices and the stomach or left gastric vein. For this purpose, percutaneous transhepatic entry, transumbilical approach. Embolization is performed using synthetic particles of different diameters or the Gianturco coil.

If the above-described methods are ineffective in controlling of bleeding and in case of its recurrence, *surgical interventions* are indicated "at the height of the bleeding", which are supposed to stop the bleeding. The most common are: the left gastric vein ligation, gastrotomy with the bleeding veins ligation, Tanner's operation (the intersection of the stomach in the subcardial portion, of vessels of the gastrocolic omentum and gastrohepatic omentum) or its modifications. One of the methods used in emergency cases is the intersection of the esophagus with the use of the suturing device.

To prevent bleeding surgical operations are performed, which are aimed at the separation of the portal and gastroesophageal blood flow and lowering of PH. To separate the gastroesophageal blood flow, Tanner and Walker operation (transsection of the esophagus with thoracotomic entry from the left side), Sugiura-Futagawa's operation (paraesophageal and paragastric devascularization from the thoracic and abdominal approaches) are used.

Patients with PH should be under follow-up care. Twice a year, and even more often, if indicated, (in case of a high risk of bleeding the repeated examinations are carried out every 6 months) should be examined by a family doctor and a surgeon. It is imperative to monitor the clinical and biochemical blood values.

If the condition of patients with liver cirrhosis is stable and the varicose veins are not large-sized, the repeated esophagogastroduodenoscopy, as well as the ultrasound examination of the abdominal organs, are performed every 1-2 years to assess the dynamics of the pathological process.

Drug and surgical treatment have a temporary effect, although sometimes the effect is quite long-termed. Patients usually die because of aggravation of symptoms of the liver disease and the complications. Favorable prognosis is observed in the subhepatic form of the syndrome. In case of timely operation – applying of vascular venous anastomoses (of one or another modification) – some patients return to work-related activity.

Prevention of portal hypertension involves the prevention and timely treatment of the diseases that provoke the development of this syndrome.

Liver failure

Acute and chronic liver failures are differentiated and the distinction is made between 3 stages.

stage I - initial (compensated)

stage II - pronounced (decompensated)

The terminal stage of liver failure may end with hepatic coma.

Depending on the cause, ***endo - and exogenous forms of liver failure*** are distinguished.

The clinical manifestations of liver failure are varied and are determined by the cause they are caused. They also depend on what functions of the liver are most impaired (protein-synthetic, chromogenic, detoxification, etc.).

The severity of liver failure is closely interrelated wi th *the intensity of jaundice, hyperammonemia*. The sources of the latter are food proteins, blood that pours into the lumen of the digestive tract (usually in case of bleeding from phlebectasia of the esophagus). Ammonia is formed from proteins under the influence of digestive juices and enzymes. In case of impaired liver detoxification function, ammonia is indestructible and enters the general bloodstream, creating a toxic effect on the brain. The severity of liver failure determines the severity of neuropsychiatric disorders. In the early stages, mental depression is observed, or, vice versa, euphoria develops, which are often interchangeable. Later, changes in the neurological status occur: dystaxia, speech disterbance, and mental slowness. During the final stage of the disease coma develops.

The diagnostics and treatment of liver failure are described above.

II.9. ACUTE GASTROINTESTINAL HEMORRHAGE SYNDROME (AGIHS)

All the forms of gastrointestinal bleeding, which are combined under the name "**Acute gastrointestinal hemorrhage syndrome (AGIHS)**", may occur being the result of various diseases. Most often these bleedings occur as a result of peptic ulcer disease (60-85%). However, it should be emphasized that gastrointestinal bleeding of non-ulcer etiology, especially related to portal hypertension, gastric cancer and erosive gastritis, is also often noted in surgical practice.

Gastrointestinal bleeding often occurs due to lesions of the vascular wall (impaired permeability, atherosclerotic changes, increased angiasthenia, varix dilatation, aneurysms),due to the defective coagulation properties of blood and its fibrinolytic activity.

In this regard, the diagnosis and treatment of acute gastrointestinal bleeding is one of the most difficult parts of emergency surgery.

Ulcer haemorrhage is observed in 10-15% of patients with gastric ulcer and duodenal ulcer.

Etiology of the acute gastrointestinal hemorrhage syndrome (AGIHS). All the diseases that may cause AGIH syndrome can be subdivided into two groups (***A. A. Shalimov***).

I. Gastric AGIH – related to the pathology of the digestive tract organs (DT) (gastric and duodenal ulcers, benign and malignant tumors of the stomach and esophagus, hemorrhagic gastritis and duodenitis, Mallory syndrome, peptic esophagitis, burns and foodstuffs, food and heart disease, food syndrome, Mallory-Weiss syndrome, peptic esophagitis, foreign bodies and esophageal injuries; hiatal hernia, etc.)

II. Agastric AGIH – related to the pathology of other organs and systems (portal hypertension, specifically liver cirrhosis, with the development of esophagus and cardiac gastric

phlebectasia, which are the source of bleeding), hemorrhagic diathesis, vitamin-deficiency disease, hemobilia, leukemia, etc.

Of the many reasons given for AGIH, *gastric and duodenal ulcers* are the most common – 55 to 85%; *esophagus and cardia phlebectasia* in case of portal hypertension cause AGIH in 5-18% of cases, *erosive lesions* of the mucous membrane of the esophagus, stomach and duodenum – in 9-16%, *cancers* of the alimentary canal – in 8-10%, *Mallory-Weiss syndrome* – in 2.5-7.5% of cases.

Acute gastrointestinal bleeding occurs from arteries, capillaries or veins, rarely from two or three vascular elements at the same time. Only due to traumatic wall damage, as is the case with Mallory-Weiss syndrome, the arteries, capillaries and veins bleed all together. In case of malignant tumor, mixed bleeding also occurs, becouse the vessels are destroyed randomly due to the tumor process, but in this case severe bleeding rarely occurs.

In case of peptic ulcer, the source of bleeding is most often the artery, less often the bleeding is of the arteriovenous nature. In case of esophagitis, gastritis, duodenitis, the bleeding is often of capillary nature, it occurs from diffuse surface inflammatory or degenerative sites.

Classification of AGIH (Acute gastrointestinal hemorrhages).

1. According to the frequency, the distinction is made between: *one time hemorrhages, relapsing hemorrhages and frequently relapsing hemorrhages.*

2. According to the severity of blood loss, AGIH are subdivided into three degrees (A. A. Shalimov, 1972): *I – mild (low-grade*) – blood loss of up to 20% of the volume of blood circulation (VBC), on average, the circulating blood deficiency (CBD) – of up to 1000 ml; *II – moderately severe* – blood loss is from 20 to 30% of VBC or 1000-1500 ml; *III – severe* – blood loss of more than 30% of VBC or more than 1500 ml.

A special form is differentiated - "***profuse***" AGIH with a significantly damaged large artery.

The ***profuse hemorrhage*** is seen as a simultaneous and rapid flow of large amounts of blood (up to 1 l) into the lumen of the digestive tract, which is accompanied by the development of a typical symptom complex: vomiting with blood, melena and collapse.

There are ***3 forms of profuse AGIH*** (Yu. E. Berezov):

1) *one-time, stops quickly;*

2) *recurrent, repeats within several hours or days*;

3) *continuous*.

Acute and *chronic* gastrointestinal hemorrhages (GIB) are distinguished. There is also *massive* bleeding and *non-massive* bleeding (the latter term is rarely used).

Clinical picture of the acute gastrointestinal hemorrhage syndrome (AGIHS). The clinical course of AGIHS includes *two periods*: *I – the latent period; II – the period of external manifestations of bleeding.*

The latent period corresponds to the initial manifestations of AGIH and is characterized by *common signs* of *acute blood loss*:

1) *subjective* – fatigue, cold sweat, dizziness, blurred vision ("seeing spots", "seeng colored circles", " seeng mesh"), head noises, fainting may occur, which are the manifestations of the brain hypoxia;

2) *objective* – lethargy and hypodynamia, skin pallor, changes in hemodynamic parameters – hypotension, tachycardia, deficient pulse (due to the developed of hypovolemia).

External signs of bleeding from the upper portions of the digestive tract are: vomiting with blood (haematmesis) or coffee-grounds vomiting (of brown colour) and tarry stool (loose, black) (melaena); in case of bleeding from ***the lower portions*** (the left half of the colon) *blood admixtures in the excrements or "hemafecia*".

Vomiting with blood (haematemesis) can be of one-time character, abundant, insignificant, multiple, with fresh red blood, with or without clots admixtures, such as "coffee grounds". The rate of bloody vomiting depends on the intensity and massiveness of bleeding, the degree of filling of the stomach with food, the individual peculiarities. As a rule, it is a sign of

severe gastric or esophageal bleeding. In case of significant bleeding with the location in duodenum, the discharge of blood into the stomach and the occurance of bloody vomiting cannot be excluded. Repeated hematemesis is more often observed with moderate hemorrhage and severe hemorrhage. In almost half of the patients, vomiting with blood is not a mandatory sign of bleeding and may be absent altogether.

Vomiting with blood, the one of the "coffee grounds" type, as a rule, indicates moderate bleeding from the upper digestive tract, and the poured blood finds time to be exposed to the hydrochloric acid of gastric juice.

The tarry stool (melaena) is the most constant (noted in 100% of cases) and a rather early sign of the onset of AGIH (it appears already 30-40 minutes after the onset of bleeding) and sometimes it may be the first manifestation of AGIH.

In case of acute profuse bleeding, the blood, which irritates the intestine and intensifies its peristalsis, quickly reaches the rectal ampoule, causing an involuntary act of defecation (which often develops during collapse) with blood or feces mixed with blood. In most cases, "*bloody stool*" (the presence of unchanged blood in the feces) indicates that the source of bleeding is localized in the distal colon.

Acute gastrointestinal hemorrhage quickly affects the general condition of patients. Elderly patients, who are physically weaker and have concomitant diseases, are more sensitive to the blood loss.

Mild bleeding is manifested by dizziness, short-term general fatigue, tarry stools. With a more massive acute bleeding, severe weakness, dizziness, cold sweat, cyanosis of the lips, acrocyanosis, severe skin pallor are observed. Hemorrhagic collapse with the loss of consciousness or with impairment of consciousness may quickly develop. As a result of acute anemia, severe visual impairment ("flashing seeing spots"), hearing and mental disorders due to progressive brain hypoxia may occur. There may be pain in the area of the heart due to myocardial hypoxia.

Clinical specification of the severity of AGIH

Bleeding degree stage I – mild. ***Complaints*** – moderate general fatigue, dizziness when making sudden movements, nausea, vomiting of "coffee grounds" type (or it may be absent), tarry stool 1-2 times, in small amounts. The general condition of the patient is relatively satisfactory, the consciousness is preserved. Mild skin pallor, pulse rate of up to 90-100 beats / min, blood pressure – normal or lowered up to 100 millimeters of mercury.

Bleeding degree stage II – moderately severe. Complaints – expressed general fatigue, vertigo at rest, visual disturbances, head noises, short-term loss of consciousness (relapce is possible), nausea, repeated vomiting of "coffee grounds" type, and sometimes, even bloody vomiting can be observed, repeated defecation with black loose feces. Lethargy, expressed skin pallor are observed, pulse is accelerated up to 100-110 beats / min, of weak filling, the blood pressure is lowered up to 90-80 millimeters of mercury.

Bleeding degree stage III – severe hemorrhage. It can be difficult to contact with the patient due to the patient's lethargy and even confusion; severe fatigue, hypodynamia, expressed dizziness at rest, visual disturbances, repeated fainting, repeated bloody vomiting, frequent loose black stool of significant volumes, thirst. The general condition is severe, severe skin and mucous membranes pallor, acrocyanosis, cold sweat. Pulse –120-130 beats / min, of weak filling, blood pressure –below 70 millimeters of mercury.

In case of ***profuse hemorrhage***: it is almost impossible to contact with the patient, continuously – vomiting with blood and melaena in large volumes; extremely serious condition, adynamia, acute skin and mucous membranes pallor, pulse – 130-140 beats / min., filamentous or not palpated, blood pressure is lower than 50 millimeters of mercury or not determined – clinical picure of hemorrhagic shock. If untreated, the patient may die within an hour.

Peculiarities of clinical manifestations of AGIH syndrome with various diseases. When starting clinical examination of a patient with AGIH syndrome and when interviewing each of the patient, it is necessary to identify the presence or absence of peptic ulcer (PU) of the stomach

or duodenum, first. It is also mandatory to inquire about other diseases of the digestive tract organs, liver, cardiovascular and respiratory organs, and the blood system. The doctor should ask if the patient took any drugs that can cause bleeding into the lumen of the digestive tract (aspirin, anticoagulants, corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs)); were there any other factors that may cause bleeding (alcohol, rough and spicy food, any stressful situations, physical stress, etc.).

Bleeding gastric ulcer and bleeding duodenum ulcer. According to the literature, ulcerative bleeding ranges from 37 to 88% of all the bleeding of the given location. Ulcerative bleeding, like the peptic ulcer itself, is more common in men (80%) than in women (20%). Acute inflammatory necrotic processes of ulcerative lesions of the wall of the stomach or duodenum lead to arrosion of the vessels at the bottom of the ulcer, which causes bleeding. Ulcerative bleeding, in addition to complaints characteristic of any etiology of the AGIH syndrome, is characterized with the *Bergmann syndrome* – with the onset of bleeding pain in the epigastric region subsides against the background of acute ulcer. When clarifying *the history of the disease*, it is necessary to find out if the patient has a peptic ulcer. If the answer is negative, it is necessary to try to identify the presence or absence of an "*ulcer history*" (connection of the appearance of pain in the epigastrium with food intake, "hungry", "night" pains, heartburn, "sodalgia" symptom, seasonal complications), and to ask the patient if there was a black stool before.

Objective symptoms for ulcerative AGIH – the appearance and behavior of the patient, as well as other objective symptoms depend on the degree of blood loss, the time passed from the onset of the bleeding, as well as on whether the bleeding stopped at the time of examination or is still ongoing.

Local status. The tongue is usually coated, often with a brown fur (after vomiting of "coffee grounds" type); in case of severe blood loss, the tongue may be dry. On examination of the abdomen, there are no signs characteristic of ulcerative bleeding (UB) – the abdomen is not swollen, it is involved in the act of breathing, and is symmetrical. On palpation, the abdomen is soft, moderate soreness is determined (it may not be present or it will be determined only on percussion of the abdominal wall – *Mendel's symptom*). It must be remembered that only superficial palpation and easy percussion are permissible. On auscultation, an important symptom is an increase in intestinal noise, which may indicate continuing bleeding (*Taylor's symptom*).

The clinical examination of the patient must necessarily be completed with a *digital examination of the rectum*, in which *traces of black feces* are found on the glove, which is the main objective sign of AGIH.

It is necessary to remember about the possibility of a combination of acute ulcerative bleeding and ulcer perforation. The clinical picture of ulcer perforation on the background of AGIH is often atypical – such characteristic symptoms of perforation as stabbing pain, rigidity of epy abdominal muscles ("wooden belly") and Shchetkin-Blumberg symptom may be absent.

An increase in the abdominal pain, muscle tension (even a small one) and pain during superficial palpation, absence of hepatic dullness on percussion are the early symptoms of perforation with the background of bleeding. At the slightest suspicion, it is necessary to carry out the radiographic examination of the abdominal cavity in order to detect free gas in it – *Jobert's symptom* (if the patient is in serious condition – in lateroposition); and in case it is absent, – the *Hennelt's test* (if the patient's condition allows). In difficult cases, urgent endoscopy (with insufflation of air into the crater of the ulcer) is indicated.

Bleeding from phlebectasia of the esophagus and cardia of the stomach due to portal hypertension. They make up from 6 to 37%, or more, of all gastrointestinal bleeding and rank second in frequency, second only to ulcerative bleeding. With liver cirrhosis, thrombosis of the hepatic veins, thrombosis, stenosis or compression of the portal vein or its branches, make the outflow of venous blood through the portal system difficult, the pressure in the portal system rises.

Due to the venous stasis in the portal system, compensatory collateral circulation gets involved through the veins of the cardial section of the stomach and the distal portion of the esophagus, the veins of the anterior abdominal wall dilate (as the disease progresses and the load on them increases, this produces varix dilatation) – *phlebectasia* in the esophagus and stomach develops. With a significant increase in portal pressure and the development of vascular insufficiency, they rupture and lead to severe bleeding. As a rule, vascular insufficiency accompanies the development of parenchymal liver failure, which is clinically manifested by the appearance of jaundice, enlargement and thickening of the liver, development of splenomegaly and ascites, signs of intoxication develop.

There are intrahepatic, extrahepatic, and mixed forms of ***portal hypertension***. *Intrahepatic portal hypertension* (the most common form) develops due to the ***liver cirrhosis*** – which is of alcoholic nature, due to the past history with viral hepatitis (A - Botkin's disease, C, D), malaria, syphilis, Budd-Chiari syndrome, liver tumors, etc.

Extrahepatic form is noted after various infections and inflammatory processes of the abdominal cavity, history with umbilical sepsis, which cause phlebitis or pylephlebitis of the portal vein or its branches, followed by their thrombosis and cicatrical stenosis. Tumors, scars, inflammatory infiltrates, and chronic pancreatitis, which lead to compression of the portal vein and its branches, are decisive in the development of the process.

Intrahepatic forms of portal hypertension are characterized by a more severe course, where the portal blood flow is disturbed, as well as the arterial blood flow. In case of the extrahepatic form, the portal blood flow changes, but the arterial blood supply may compensatory increase.

Portal hypertension is often accompanied by ascites and recurrent bleeding from phlebectasia of the esophagus and stomach. Varicose veins of the stomach and esophagus connect the portal and superior vena cava through the azygos and hemiazygos veins. High portal pressure and anatomical interconnection of the portal system and superior vena cava system contribute to the varicosity of the veins.

Veins of the lower and middle third of the esophagus, as well as of the cardiac part of the stomach, are exposed to varicosity, which can be explained both by the thin walls of these veins and by the dyshesion of the connective tissue in which they lie, and by the impaired innervation of the latter. Trophic changes in the wall of the veins and the adjacent mucosa develop due to the varicose veins of the esophagus.

In case of portal hypertension, the triggering mechanism for bleeding is a portal crisis of the portal system, which causes disruption of the decompensated veins, peptic factor and changes in blood coagulation system. Bleeding from phlebectasia of the esophagus and stomach often occur after a heavy meal or during sleep, when the blood flow into the portal system increases significantly. Patients often feel pain in the epigastric region, which is accompanied by heartburn,
colicky pain in the liver, together with bloody vomiting and jaundice.

The diagnosis of AGIH related to portal hypertension, is based on the comprehensive examination of patients.

Local status. On examination, the following is observed: weight loss, icteric sclerae and the skin, vascular spiders, varicose subcutaneous veins of the anterior abdominal wall – “arachnogastria”, abdominal swelling and flattness – “frog belly”, atrophy of the shoulder belt muscles; solid liver and spleen are enlarged (hepatolienal syndrome), or the liver is reduced due to atrophic cirrhosis, ascites. But these signs are not permanent, therefore, the diagnosis of portal bleeding cannot be successfully made without a comprehensive study which includes special techniques.

Hemorrhagic erosive gastritis. It is a common cause of gastrointestinal bleeding – from 5 to 14% of cases. Erosions are superficial ulcers with clean or brown coated fundus resulting from the formation of hemin influenced by hydrochloric acid of gastric juice. They are most often

localized in the prepiloric part of the stomach, but they may also appear in other portions of the stomach.

The *etiology and pathogenesis* of hemorrhagic gastritis is not yet fully understood. Most of the authors believe that gastric mucosa erosions occur as a result of systemic diseases or under the influence of local factors: in case of endocrine, infectious, toxic, cardiovascular diseases, circulatory disorders, sometimes in case of burns, long-term use of drugs (aspirin, nonspecific anti-inflammatory drugs – Diclofenac, Voltaren, adrenal hormones, anticoagulants, etc.), in the postoperative period and with the nervous system damage.

The major role in the pathogenesis of hemorrhagic gastritis bleeding belongs to biochemical changes in the blood coagulation system towards hypocoagulation and impaired metabolic processes, which cause an increased in the gastric mucosa capillaries' permeability.

The clinical picture of hemorrhagic gastritis does not include pathognomonic symptoms and consists of: general symptoms of acute blood loss; dull pain in the abdomen that occurs after the diet defects, alcohol intake, certain medicines; nausea, less often – vomit of "coffee grounds" type, melena. As to the intensity degree of bleeding, it is often mild, and it is not accompanied by collapse.

Bleeding in case of Mallory-Weiss syndrome. Mallory-Weiss syndrome occurs mainly in men aged 30 to 50 years, and is characterized by the formation of deep deep fractures (tears) of the mucous membrane of the cardio-esophageal zone along the lesser curvature. At the same time, the vessels are also damaged, which is accompanied by bleeding of different intensity (more often it is pronounced).

The *reasons for the development* of this syndrome are the intake of large amounts of alcohol and food with recurring massive vomiting, weight lifting (freight movers), etc. Thinning of the stomach in case of atrophic gastritis, as well as the advanced age (in this pathology) contribute to this. Vomiting leads to an increase in intragastric pressure, changes in blood circulation in the distended stomach due to anemization of the mucous layer with adequate blood circulation in the muscle layer and mucosal ruptures. Sometimes the development of the syndrome is preceded by hiccups, coughing episodes in bronchial asthma, on physical exertion after eating or chronic diseases of the stomach. The epigastric pain is usually weak or absent.

Bleeding in case of the stomach and esophagus cancer. According to many authors, stomach cancer causes gastrointestinal bleeding in 10-12% of AGIH cases. They are rarely of a profuse character, they usually occur in the late stages of the disease due to the tumour cavitation, often after gastric intubation to examine the gastric juice.The bleeding is not excessive, but it lasts long, and as a result, the patients gradually lose a significant amount of blood, while their condition remains clinically subcompensated. With the background of a single or double vomiting of "coffee grounds" type, tarry stool has long been observed, which is caused by the parenchymal bleeding from small vessels of the destructed tumor. Much less common is the profuse bleeding in the initial stages of cancer in case of large vessel arrosion due to the neoplastic process.

The diagnosis is made on the basis of the characteristic local and general symptoms of gastric cancer: the "Lesser signs syndrome by Savitsky" – progressive weakness, mild fatigue, irritability, loss of appetite, weight loss; gastric discomfort, dyspepsia, abdominal pain, which are little dependent on the food intake, foul-smelling eructation, palpable tumor in the epigastrium or detection of distant metastases (increased nodular liver, "Virchow metastasis" in the left supraclavicular fossa, umbilical Schnitzler and Krukenberg metastases) .

Bleeding in case of esophageal cancer is a late clinical manifestation of the disease, which indicates an advanced case and, most likely, the inoperability of the tumor. Bleeding occurs in stages III-IV of the disease, when the diagnosis is no longer doubtful – drastic weight loss, rapidly progressive dysphagia, pain along the esophagus are observed.

Bleeding in case of benign non-epithelial digestive tract tumors. They make about 1.5% of all the bleeding of non-ulcer genesis. Benign tumors, which cause bleeding, can be localized in the esophagus, stomach, duodenum, small intestine or colon. Bleeding from gastric tumors are

most often observed. In most cases, the cause of bleeding is leiomyoma of the stomach, less often are: fibromas, neurinomas, neurofibromas, etc.

As a rule, no clinical symptoms are observed, therefore, this pathology caused by AGIH is diagnosed with the help of additional research methods.

Bleeding in cases of the colon diseases. Such colon diseases (CD) as ulcerative colitis, diverticulosis, benign tumors (polyps) and cancer may be complicated by acute or chronic intestinal bleeding.

Intestinal bleeding (IB) is rarely profuse, but it is often pronounced, with red blood colour, when the source of the bleeding is localized in the left half of the large intestine; but when being localized in the right half – it comes in the form of melena (tarry stool). There may be hidden forms of bleeding, which are manifested by *anemia*.

The colon diseases (CD) are marked by a *common main clinical symptom complex*:

1) abdominal pain

2) bowel disorders – constipation, diarrhea, abdominal distension, tenesmus and others;

3) the occurrence of pathological foreign substances in the feces – mucus, blood.

The symptom complex has its own clinical features with each disease, which makes it possible to make the differential diagnosis.

Nonspecific ulcerative colitis is clinically defined by the occurrence of *signs of inflammation* – *complaints* of diarrhea in defecating with the release of mucus and pus, complaints of the abdominal pain, general weakness, fever. *On objective examination*: an increase in the pulse rate, a decrease in the blood pressure, tenderness on palpation along the large intestine, spasmodic sigma is often painful on palpation; swelling and pitting edema of the lower extremities frequently occur, sometimes the nail phalanges are thickened and look like the drum sticks.

The occurrence of complaints of rectal bleeding, detection of edema and swelling of lower extremities in such patients, complaints of thickening of the nail phalanges so that they look like the drum sticks, occurrence of *anorectal* (paraproctitis, fistula, anal fissures) and *common* (peripheral arthritis, damage to the organs of vision, skin and mucous membranes, liver disorders) definitive ***complications*** – make it possible to suspect nonspecific ulcerative colitis (NUC).

Being a possible cause of the intestinal bleeding (IB), ***diverticulitis of the large intestine (LI)*** is characterized by a *triad of symptoms* (especially in elderly people). They are colicky abdominal pain, flatulence, disturbed rhythm of defecation (constipation). All may occur in the absence of any objective stomach disorders or when paracolytic infiltrate or abscess is detected; internal gastrointestinal or external intestinal fistula may be detected in patients with intestinal bleeding.

The history of frequent small rectal bleeding for asymptomatic patients with *dysproteinemia* and of *anemia* in patients with intestinal bleeding (IB) may suggest the occurence of ***colon polyps***.

Epigastric pain complaints, frequent loose stool, discharge of blood and mucus from the rectum, prolapse of polyps from the anus, exhaustion, anemia, growth and physical development failure, late development of secondary sexual characteristics, changes in the nails, fingers and toes (they look like “drum sticks and watch glass”), pigmentation of the skin of the face and especially of the lips. All these symptoms make it possible to suspect ***diffuse colon polyposis*** in young people when intestinal bleeding occurs.

In patients over 40 years old with a clinical picture of intestinal bleeding, the survey reveals the “small signs of Savitsky syndrome”, complaints of abdominal pain (more often in the left half), symptoms of intestinal discomfort, persistent constipation, blood in the feces, and tumor is detected on palpation and on abdominal or digital examination of the rectum (especially *in the squatting position of the patient*) all these symptoms *make it possible to diagnose* ***colon cancer*** being the cause of the intestinal bleeding (IB).

Bleeding in case of Zollinger - Ellison syndrome. In case of this syndrome bleeding are caused by ulcers of the upper gastrointestinal tract, which are formed due to the *ulcerogenic pancreatic adenomas*, which secrete gastrin-like hormone 40 times stronger than gastrin as to the ability to activate gastric secretion and ability to develop ulceration.

Clinical picture. Severe hypersecretion (especially basal) and sustained recurrent nature of ulcerative lesions are characteristic of Zollinger-Ellison syndrome, even after radical surgical interventions being done. Ulcers are accompanied with severe pain, and are characterized by a tendency to bleed and to perforation. Due to the hypersecretion of the stomach, the pancreas function is disturbed, which is accompanied with steatorrhea.

The diagnosis of Zollinger-Ellison syndrome can be confirmed with ultrasound examination and CT, although the diagnostics is often complicated and the disease may often be detected with autopsy.

Bleeding in case of the Osler-Rendu disease. Profuse gastrointestinal bleeding can becaused by Osler-Rendu hemorrhagic angiomatosis, which is characterized by periodic bleeding from multiple telangiectasias and angiomas of the skin and mucous membranes. It is a heredo-familial disease, it runs in the blood in a dominant mode, it may occur sporadically. The etiology of the disease is not fully established. Most often, telangiectasia and angiomas occur on the mucous membranes of the nose and mouth, on the lips, tongue, wings of the nose and ear lobes, under the nail plate of the fingers. Less commonly, the mucous membrane of the trachea, bronchi, gastrointestinal tract, bladder and liver is affected.

Clinical picture. The disease is characterized by frequent nosebleeds that occur in early childhood, telangiectasias and angioma with a certain localization. Gastrointestinal bleeding can be profuse and even fatal.

Hemobilia is a bleeding from the biliary tract into the lumen of the gastro-intestinal tract (GIT). According to the *etiology*, hemobilia is differentiated as traumatic, postoperative, or it may be caused by diseases of the liver and biliary tract. The pathoanatomical ground for the hemobilia is the non-integrity of the blood vessels of the liver and biliary tract.

The clinical picture of hemobilia is characterized by paroxysmal pain, jaundice and cyclical bleeding (after 6-8 days). Bleeding from the biliary tract in case of damage of the large vessels can be severe and the only salvation to escape the mortal danger in such cases is surgery during which the diagnosis is made in most cases.

Bleeding in case of hemorrhagic diathesis. Hemorrhagic diathesis include*: thrombocytopenia – Werlhof disease; hemophilia A, B, C; capillary toxicosis – Schönlein-Henoch disease.*

The factors that underlie ***the Werlhof disease*** are presented by thrombocytopenia or functional inconsistency of platelets (they are often of autoimmune genesis). AGIH syndrome occurs with the Werlhof disease in 0.5-2.0% of patients. Bloody vomiting and black feces are often observed in these patients due to the ingestion of blood in case of bleeding from the nose and gums. Patients report prolonged bleeding with injuries, in case of teeth extraction and abundant metrorrhagia, bruise formation is common. Excessive subcutaneous hemorrhages (*a symptom of "spotted, tiger skin"*) are often observed.

The cause of the development of ***hemophilia*** is a congenital absence or insufficient blood levels of certain coagulation factors: with hemophilia A –VIII factor of antihemophilic globulin, with hemophilia B – X factor and with hemophilia C –XI factor. AGIH syndrome in patients with hemophilia develops in 6-24% of cases and it is quite pronounced. Such patients give the history of an increased bleeding from early childhood (it is often hereditary). The hemarthrosis result in partial or complete ankylosis of the joints.

The factors underlieing ***capillary toxicosis*** (*Schönlein-Henoch disease*) are fragility, tenderness (angiasthenia) or increased capillary permeability. Patients have positive symptoms of "bundle", "pinch sign ", petechial hemorrhage (ecchymosis) on the mucous membranes and skin.

According to standard schemes, ***the plan for additional examination (laboratory and instrumental) of a patient with gastrointestinal bleeding*** includes and identifies pathology.

1. Clinical blood analysis – a decrease in hemoglobin and red blood cells; in case of the Werlhof disease – thrombocytopenia.

2. Clinical analysis of urine – no specific findings.

3. Biochemical blood analysis – hyperbilirubinemia may occur in case of liver cirrhosis.

4. Coagulogram – blood coagulation disorders with the tendency for hypocoagulation, in case of hemophilia – the absence or insufficient level of coagulation factors VIII (antihemophilic globulin), X or XI.

5. Determination of blood loss – *circulating blood deficiency (CBD)* using various methods.

1) *Van Slyka-Phillips method* – according to relative density of blood, hematocrit, and hemoglobin. The relative density of blood is determined by using a series of standard solutions of

copper sulphate ($CuSO_4$) with a relative density from 1.030 to 1.070 when the patient's blood is immersed in them (taken on heparin). The calculation of the degree of the blood loss is done according to Table 21:

Table 21

Calculation of the degree of the blood loss

Blood loss, ml	Relative blood density	Hemoglobin		Hematocrit
		Sali units	GM/DL	
Up to 500 (I degree)	1,057-1,054	65-62	109-103	0,44-0,40
Up to 1000 (II degree)	1,053-1,050	61-50	101-83	0,38-0,32
Up to 1500 (III degree)	1,049-1,044	53-38	81-63	0,30-0,23
Over 1500 (IV degree)	Less than 1,044	Less than 43	Less than 72	Less than 0,23

2) *M. I. Borovsky and V. S. Zhukova method* – according to hematocrit and blood viscosity.

Evaluation by the formulas:

$$CBD_{male} = (1000 \times Y_0 + 60 \times Ht_0) - 6700;$$

$$CBD_{female} = (1000 \times Y_0 + 60 \times Ht_0) - 6000,$$

where: CBD is patient's circulating blood deficiency, Y_0 is the viscosity of the patient's blood, Ht_0 is the patient's hematocrit.

3) Determination of circulating blood volume (CBV) and its components according to *the dilution of the indicators* that are injected into the bloodstream – 1% solution of Evans blue (azofarb T-1824), polyglucin, radioactive erythrocytes labeled with ^{51}Cr and albumin labeled ^{131}I. Blue Evans and radioactive albumin are most commonly used, using special formulas to calculate CBV and its components (CPV, CEV, CHV, CPV)

4) *method developed at the State Institution "Institute of General and Emergency Surgery named after V.T. Zaitsev NAMS of Ukraine":*

$$CBD_{male} = 6152 - 158 \times \text{Ht of the patient},$$

$$CBD_{female} = 5456 - 156 \times \text{Ht of the patient}.$$

6. Determination of the blood group and Rh affinity.

7. ECG.

8.*Urgent FEGDS (Fiberoesophagogastroduodenoscopy).* The most currently recognized informative diagnostic methods for AGIH syndrome endoscopic examination methods are ***emergency esophagogastroduodenoscopy***, ***rectoromanoscopy***, ***colonoscopy***, which make it possible to establish the source of the bleeding and to treat the bleeding wound in 93-96% of cases. The endoscopic examination makes it possible to determine the type, size, number of ulcers of the stomach and duodenum, the hemostasis stage, the depth of the ulcer crater.

Classification of ulcers according to the localization (Johnson):

Type I – body, small curvature; *Type II* – gastric ulcer + duodenum; *Type III* – pyloric ulcers.

Endoscopic classification of hemostasis (Forrest).

1). Active bleeding:
a) spurting bleeding;
b) leakage from under the fixed clot.
2). Stopped bleeding:
a) thrombus vessels;
b) a fixed clot;
c) thrombosed vessels (black spots).
3). *No bleeding* – defects of the mucous membrane under the clot.

Endoscopic examination also reveals bleeding benign and malignant tumors of the stomach and esophagus, Mallory-Weiss syndrome, erosive ulcer and duodenitis, bleeding phlebectasia of the esophagus and stomach, tumors and ulcerative intestinal lesions, intestinal diverticulosis, etc. The patient's severe condition, tumors and ulcerative bowel lesions, intestinal diverticulosis, etc. The patient's severe condition cannot prevent from endoscopic examination of the stomack, because *endoscopic local hemostasis* can be used in this condition.

9. *X-ray examination* is indicated at suspicion on a combination of bleeding from an ulcer and its perforation – carrying out of fluoroscopy of the abdominal cavity (in case of a serious condition of the patient it is performed in the lateroposition) and detection of the *Jobert's symptom* (sickle-shaped free gas strip under the right dome of the diaphragm) indicates the occurence of perforation.

10. *Urgent ultrasound examination* of the abdominal organs and veins of the portal system together with dopplerography: it makes it possible to detect the liver cirrhosis, splenomegaly, ascites, thrombosis of the splenic vein, etc.

11. In case of the syndrome of portal hypertension complicated by AGIH, *the angiographic examination* proves to be a highly informative diagnostic method (evidence-based medicine); it is splenoportography (which is preceded by splenomanometry), which detects splenomegaly, thrombosis of the splenic vein, degree of the liver cirrhosis, phlebectasia of the esophagus and stomach.

Differential diagnostics (for more details, see the information below). The patients with the clinical picture of gastrointestinal bleeding, have to undergo the differential diagnostics with all the diseases that may be complicated by the AGIH syndrome in order to establish the etiological diagnosis.

For this, the data of the clinical examination are used – characteristic complaints, history of the disease, clinical features, the detection of pathognomonic symptoms in all these diseases (see the nformation above), as well as the data of additional research methods.

The most important clinical differential features in this pathology.

Reduction of pain or its disappearance with the onset of bleeding often indicates ulcerative lesions of the stomach or duodenum.

The occurrence of vomiting with blood in case of severe vomiting or vomiting with blood after a sharp increase in intra-abdominal pressure (height lifting, strong cough attack, a strong inner tension during defecation, etc.), which is accompanied with abrupt chest pains, Mallory-Weiss syndrome is suggested.

Bloody vomiting after acute pain in the epigastric region is also characteristic of the infringement of the stomach in the hernia of the esophageal opening of the diaphragm.

In case of bleeding from the decaying gastric tumor, the following history is common: weight loss, loss of appetite, rapid fatigue with increasing general weakness, loss of appetite, foul-smelling eructation (syndrome of "minor signs"). Most often the large decaying gastric cancerous tumors bleed.

At that, profuse bleedings can rarely be registered, they are usually not abandant, the blood looks like "coffee grounds. The hemorrhage usually occurs against the background of previous anemia and cancer cachexia. Active bleeding that occurs in the form of vomiting with the red blood colour and clots, may accompany cardiac gastric cancer and may manifest itself for

the first time. They are often noted after the gastric juice sampling. In case of bleeding from a decaying gastric tumor, pain is usually not felt.

The acute abdominal pain attacks of abdominal colic type, which are accompanied with a bloody stool and vomiting, and which produce obvious changes on the skin and mucous membranes in the form of hemorrhagic purpura. They are specifically attributed to vascular lesions of the gastrointestinal tract – Henoch-Schönlein purpura, periarteritis nodosa (Kussmaul's disease).

The history with bleeding, which occurs since childhood, makes it possible to suspect hemophilia or Randy-Osler disease. Associated changes in the joints present a valuable diagnostic feature.

In this regard, to clarify the causes of the bleeding, special attention should be paid to the skin and mucous membranes, lymph nodes, the size of the liver and the spleen, ascites, palpable abdominal mass in the abdominal cavity during an objective examination of the patient.

Treatment of patients with AGI syndrome. ***Pre-Hospital Emergency Medicine*** presupposes absolute rest of the patient, who should remain in a horizontal position, while excluding the ingestion of food and fluids through the mouth. For the purpose of hemostasis, cold can be applied to the epigastric region. 100-200 ml of a 1% solution of calcium chloride (if available under conditions of a reanimobile) intravenously and 5 ml of a 1% solution of Vicasol intramuscularly are indicated. In case of profound hemodynamic disturbances, limbs phlebostasis should be performed, with limbs being kept in the upward position. Cardiovascular medication and, if possible, transfusion of plasma substitutes are indicated.

A brigade of special medical aid should carry out emergency hospitalization, aimed at targeted treatment of the patients. The patient should be admitted to a surgical unit with all-day proper care of the team of surgeons, resuscitators, laboratory assistants. The round-the-clock emergency X-ray and endoscopic examination must be available.

Treatment in a surgical hospital. *The choice of the disease management*. *Patient-specific noninvasive therapeutic approach to the AGIH syndrome* is recognized in the surgical hospital.

Active surgical approach – life-saving operation is to be performed immediately after the patient is hospitalized and is taken from the emergency department to the operating room. This approach is indicated in the following cases:

1) in case of *profuse hemorrhage* of any etiology (repeated massive bloody vomiting, repeated abundant melena, ischemic encephalopathy, rapid decrease in blood pressure, up to collapse – the clinical pictre of hemorrhagic shock);

2) in case of *concomitant bleeding* from the ulcer or a tumor of the stomach, duodenum or intestine and in case of the perforation of the ulcer or tumor.

In most cases, the nature of gastrointestinal bleeding makes it possible to follow *noninvasive therapeutic approach* – to initiate the treatment with application of the conservative therapy. It is aimed at stopping the bleeding and it includes general and local hemostatic therapy and hemodynamics stabilization with simultaneous specification of the diagnosis.

Conservative therapy. Peptic ulcer and the ulcer of the duodenum:

1) patients are strictly confined to bed;

2) bowel rest with the moving to the Meulengracht diet (mashed chilled food)

3) application of cold on epigastrium (it is possible to use intragastric hypothermia in an open or closed way according to Wangesteen)

4) the application of *the hemostatic therapy* by way of:

a) intravenous infusions of 1% solution of calcium chloride 200.0 ml, 5% solution of aminocaproic acid (ACC) 100.0-200.0 ml, fibrinogen 1-2 g, ascorbic acid 2.0-4.0 ml – all twice a day;

b) intramuscular injections of 1% Vikasol solution with the dosage of 2.0 ml, Etamsylate or Dicynon with the dosage of 1.0 ml – 3 times a day;

c) local administration of hemostatic drugs – oral administration of cooled solutions of 5% ACC, Hemophobin, Thrombin (they may be combined – a mixture of 30.0 ml of each one – every 30-60 minutes);

5) the use of antacid drugs, inhibitors of proteolytic enzymes and application of other methods of anti-ulcer medication.

In recent years, local hemostasis has been successfully used for *urgent endoscopic examination* "at a height" of bleeding by way of irrigation of the source of the hemorrhage using hemostatic solutions (aminocaproic acid, Thrombin, Hemophobin), filmogenous drugs (sodium alginate, oxycellosol, lyfusol), diathermocoagulation, cryoelectroagulation or laser photocoagulation of the bleeding area, clipping the vessels lengthwise.

Liver cirrhosis in case of AGIHS. The conservative treatment of patients with bleeding from phlebectasia of the esophagus and stomach due to portal hypertension (most often it is specifically attributed to liver cirrhosis) includes (additionally to the above described) intravenous drip of 20 U of pituitrin dissolved in 200 ml of 5% glucose solution – for 20 minutes, with further intramuscularly introduction of 10 U of pituitrin every 3 hours.

For the treatment of hepatic insufficiency, (which is registered, as a rule, in such patients) concentrated glucose solutions with vitamins and *hepatoprotectors* Essentiale, Heptral, Hepabene, Syrepar, glutamic and lipoic acid, etc.) is administered.

The application of the *Blackmore tube* is most effective for local hemostasis. Before the introduction of the tube, the obturator is moistened in the 2% solution of novocaine, the cylinders are folded around the tube and are introduced into the stomach. Then, 100 ml of air are injected into the distal balloon and the probe is pulled upwards, which leads to obstruction of cardia phlebectasia.

After that, about 200 ml of air are injected into the esophageal balloon, which squeezes the phlebectasia of the lower third of the esophagus. In such a position, the probe remains for 1-2 days. Through the lumen of the probe, dynamic control over the nature of the gastric contents is performed and the patient is fed 3-5 times a day (150-200 ml of fluid diet, it should be rich in vitamins, carbohydrates and proteins). With the probe kept in the esophagus, the patient is prescribed narcotic drugs and antihistamines. Every 6-8 hours of air is drawn off from the esophageal balloon to prevent the possible occurrence of trophic disorders of the esophageal wall. Before removing the probe from the cylinders, the air must be removed, and the patient is given a spoonful of vaseline oil to drink.

In case of hemorrhagic diathesis, the conservative treatment is aimed at compensating the components of the blood coagulation system that the patient lacks. ***In case of hemophilia A***, which is marked by the deficiency of antihemophilic globulin (coagulation factor VIII), transfusion of freshly prepared plasma is indicated (as the unstable antihemophilic globulin is destroyed when stored within a few hours) or transfusion of the fresh frozen plasma is performed. ***In case of hemophilia B and C***, dry, freshly prepared and native plasma is used, since factors X and XI, which are the cause of these forms of hemophilia, can be stored for a long time. For all types of hemophilia, the transfusion of *frozen cryoprecipitate*, which contains about 50% of factor VIII, 20-40% of fibrinogen, factor XIII admixture and fibronectin, is effective. Commonly used hemostatics (like Vikasol, vitamin C, calcium chloride, etc.) are ineffective. ***In case of Werlhof's disease***, the most effective method applied in case of gastrointestinal bleeding is the transfusion of freshly prepared native plasma, platelets (thrombocytes), and a terminable *platelets (thrombocytes) concentrate* preparation. Other hemostatic drugs can also be prescribed.

In case angiographic facilities are available, ***the method of endovascular therapy*** – the packing of bleeding vessels – is possible to be applied.

There are the ***three variants of application of the noninvasive therapeutic approach***.

1. The *positive effect* that can be obtained after the hemostatic therapy – the appearance of clinical signs of the bleeding control: the patient does not have a sense of fear, his behavior is

more relaxed, nausea and vomiting stop, melena is not regestered, the hemodynamic parameters tend to normal.

In such cases, conservative therapy continues to be carried out. It is presented by hemostatic and replacement therapy (in case of II-III bleeding degree), anti-anemic and anti-ulcerous (in case of the peptic ulcer) or anti-inflammatory therapy (in case of nonspecific ulcerative colitis (NUC), diverticulitis). After which *the decision about further medical management should be made*. It can be an *"early"* (10 - 14 days after the onset of bleeding) *elective surgery* should be performed. In ths case the patient has indications, like long course of ulcer with frequent relapses, that is, ineffectiveness of the conservative treatment of ulcer, history with repeated AGIH, ulcer stenosis revealed during the examination, malignancy of the ulcer, ineffectiveness of conservative therapy for nonspecific ulcerative colitis (NUC), complicated forms of diverticulosis; benign and malignant tumors of GIT and others). Another option is *admission to a therapeutic unit* to complete the course of anti-ulcerous or other specific therapy (in case of stable hemostasis and in the absence of indications for surgery).

2. *No positive effect* from the ongoing hemostatic therapy – the patient has *clinical signs of prolonged ulcerative bleeding* (increase in general weakness, lethargy, physical inactivity, hypotension and tachycardia, continuing nausea and bloody vomiting or the vomiting of "coffee grounds" type, melena). In such cases, life-saving surgery (which has already been delayed) is indicated "at the height of bleeding".

3. A temporary stopping of bleeding (*temporary hemostasis*) with its subsequent *relapse* during the clinical course within any period of time (from several hours after the stopping of the bleeding and up to the discharge from the hospital). In such cases, life-saving surgery is also indicated "at the height of bleeding."

In case of degree I of the blood loss (with circulating blood deficiency (CBD) of up to 1000 ml) the therapy *is limited* to the above mentioned volume of *hemostatic conservative therapy*.

In case of degrees II and III of the blood loss (with circulating blood deficiency (CBD) of more than 1000 ml), *the hemostatic therapy after stopping of the the bleeding must be supplemented with the replacement therapy*, which includes blood transfusion (red blood cell mass) and infusion of blood-substituting solutions.

In recent years, not the whole preserved blood is transfused, but the *packed erythrocytes* (optimally *washed erythrocytes*) of early shelf life (up to 5-7 days) in the amount that corresponds to *the half of the blood loss*. The second half of the blood lost is restored by plasma-substituting drugs – native and dry plasma, albumin, Protein, Reopolyglucine, Gelatinol, Polyglucinum, 6% Hydroxyethyl starch preparations ("Refortan" and "Stabizol"), and other modern plasma-substituting preparations.

In case of massive bleeding (CBD of 30% or more), to control severe hypovolemia, the infusion therapy should be started with the infusion of Reopolyglucine or Polyglucinum preparations, they can quickly restore circulating blood volume (CBV). And along with the use of hemostatic agents and blood transfusion, protein losses (plasma transfusion, albumin, protein, etc.) should be restored.

Strict bed confinement should be observed for at least 10-12 days. Hunger is indicated at the height of bleeding. Bodily functions should be fulfiled in bed with the control over the nature of the stool. To eliminate the toxic effect of blood, which is decaying in the intestines, it is necessary to use *cleansing enemas in bed* 1 time a day in order to remove the blood and its decay products from the intestines.

When the bleeding stops, the regimen and food intake of patients should be as careful as possible. The diet should be well-nourishing, high-calorie and highly digestible according to the Meulengracht diet. The food intake should be carried out every 2-3 hours, 100-150 ml with a daily caloric content of no less than 1000-1200 kcal. Food in the stomach neutralizes active gastric juice, which reduces its adverse effects on the source of bleeding, it reduces the "hungry"

peristalsis, promotes the formation of a blood clot in the vessel and stimulates regeneration processes.

Comprehensive conservative therapy is effective in 90-92% of cases. In 6-10% of cases, the bleeding, despite complex conservative therapy, does not stop, the manifestation of bleeding continues (nausea and bloody vomiting, melena, the indicators of hemodynamics are not stable, the indices of hemoglobin, erythrocytes, viscosity and relative density of blood worsen), in these cases it is necessary to make a decision about an urgent surgery "at the height of bleeding".

Surgical treatment of patients with AGIHS (Acute gastrointestinal hemorrhage syndrome).

1). The severity of the patient's condition (depending on the degree of AGIH and the comorbidities – mild, moderate, severe, very severe) should be determined.

2). Indications for hospital admossion in the ICU (intensive care unit).

3). Emergency instrumental examination – urgent endoscopy, ultrasound.

4). Additional examination: clinical analyzes of blood, urine, biochemistry and blood coagulogram, determination of blood loss (circulating blood deficiency (CBD)), determination of the blood type and rhesus, ECG, pulmonary function, intragastric pH-metry, determination of the degree of operational risk.

5). Other professional medical advice.

6). The main approach to treatment is ***an individual surgical management***.

Bleeding gastric ulcer. *Depending on the time limit for a surgery performance*, the following types of surgical interventions and the corresponding indications for them are distinguished:

I. *Urgent life-saving operations or operations "at the height of bleeding"* are performed, which presuppose the following sympoms:

1) profuse bleeding

2) bleeding that does not stop;

3) recurrence of bleeding in the clinic;

4) perforation of the bleeding ulcer.

II. *Delayed operations*, which are performed after the stopping of bleeding within the period that not exceed 2-4 days, for the patients, who in most cases have *absolute indications* for surgical treatment of the peptic ulcer, when the threat of *recurrent bleeding* remains real (30-40% of cases):

1) the occurence of chronic callous gastric ulcer of large size, which penetrates into the adjacent organs;

2) the data as to the malignancy of the ulcer or suspicion as to it;

3) the occurence of chronic ulcers, complicated by severe pyloric stenosis;

4) massive blood loss, unstable hemostasis with a long ulcer history in elderly people.

III. *Early planned operations* (elective surgery), which are recommended for patients, who have relative indications for the surgical treatment of peptic ulcer, in case of having stable hemostasis for 10-18 days since the date of admission to the hospital:

1) a long ulcer history;

2) ineffectiveness of previous courses of conservative treatment with frequent relapses;

3) relapce of the bleeding;

4) the occurence of chronic callous gastric ulcer.

Surgical treatment is not indicated:

1) to young and middle-aged people with stopped bleeding degree I-II, with first time diagnosed non-calous ulcers, which have not been treated;

2) in case of extremely severe comorbidities, especially in the elderly and senile age.

Currently, in case of bleeding ulcers of the duodenum and stomach *organ preservation surgeries – excision of the ulcer together with gastro - or duodenoplasty in combination with one of the types of vagotomy* are most commonly performed (at that, the performace of stem or selective vagotomy requires *the mandatory performance of any type of draining operation –*

piloroplasty, gastroduodeno- or gastroenteroanastomosis – to prevent gastrostasis, which is not necessary when performing selective proximal vagotomy).

When preparing patients for the surgery "at the height of bleeding", it is necessary to achieve compensation of the hemodynamic indices. And the resuscitation measures that were initiated before the operation, should also be continued during the surgical intervention.

After the laparotomy a thorough revision of the abdominal cavity is carried out, starting with the stomach and the initial part of the duodenum. The blood in the stomach and intestines indicates bleeding in the digestive tract. The examination includes examination of the organs, palpation, and, if necessary, mobilization of the duodenum according to Kocher.

On examination of the stomach, special attention should be paid to the areas of hyperemic serosa, injectivity of the vessels, the occurence of inverted scars. When pathology is not revealed, gastrotomy (wide longitudinal) is indicated, which starts with a few departures from the pylorus and makes it possible to fully examine all the parts of the stomach and to carry out a digital examination of the part of the duodenum, which can be supplemented by duodenotomy if necessary.

After the detection of the source of the bleeding measures are taken to stop the bleeding as a choice of an optimal variant of stable hemostasis. To do this, it is necessary to suture the visible bleeding vessel, or to perform an eight-fold suture on the ulcer.

The patients, being in extremely severe condition, should justifiedly undergo only ***palliative operations***: suturing of the bleeding vessels from the side of the mucosa after gastrotomy, tamponade of the ulcer with the spare omentum, suturing of the stomach wall through withthe U-shaped sutures along the perimeter of the ulcer with subsequent covering of the through-sutures with seroserous sutures.

Recently, especially severe patients with ulcerative genesis of the AGIH syndrome, for whom urgent surgical treatment is undesirable "at the height of the bleedig", undergo urgent diagnostic endoscopy together with ***minimally invasive technologies*** that are used to stop the bleeding: *endoscopic clipping of the bleeding vessels in the crater of the ulcer*. In case there is an angiographic facility to stop the bleeding from gastric or duodenal ulcers, it is possible to use the *method of endovascular therapy*, ***which should be urgently performed*** *– selective catheterization of the bleeding arteries with the subsequent introduction of hemostatic preparations and their embolization (packing).*

In case of bleeding from gastric and duodenal ulcers (callous, penetrating, stenotic), ***the resection method can be applied to treat the peptic ulcer*** depending on the operating situation. Besides, conditions permitting, *Billroth's I operation becomes the method of choice*.

Bleeding in case of portal hypertension syndrome. In case of bleeding from phlebectasia of the esophagus and the stomach, minimally traumatic surgery is performed, it is gastrotomy with suturing of the bleeding varicose veins of the stomach and esophagus and ligation of the left gastric, splenic arteries and coronary vein.

The operation ***in the modification of N. D. Patsiora*** et al. (1974) is performed in following way: after the laparotomy, the stomach is opened up, 10-12 cm long, from the fundus and up to the lesser gastric curvature; the upper part of the anterior wall of the stomach is raised; the varicous bleeding veins are stitched with catgut through the mucous membrane with the interrupted sutures; pulling up the ligatures and visible mucous membranes, the veins of the lower esophagus extruding into the lumen are sutured; after this, the wound of the stomach is sutured. If the condition of the patient permits, the splenic and left gastric arteries are ligated closer to the abdominal trunk and the coronary vein of the stomach before the gastrotomy. In the postoperative period, therapeutic and prophylactic measures are carried out, which are aimed at restoring the blood loss and treatment of the liver failure.

In recent years, minimally invasive technologies have been used, such as endoscopic introduction of sclerosants into the esophageal varicose veins of the esophagus with subsequent temporary obturation using the Blackmore tube, as well as the embolization of the above mentioned arteries during the selective angiography.

Hemorrhagic erosive gastritis. The treatment of hemorrhagic erosive gastritis is mostly conservative, but if the bleeding continues, the salvage operation is performed "at the height of the bleeding". Depending on the severity of the pathology, the following operations are performed: vagotomy with pyloroplasty together with the ligation of the left gastric artery, subtotal gastrectomy or gastrectomy.

Patients with hemorrhagic erosive gastritis are subjected to the follow-up with endoscopic control observations.

The technique of clipping of vessels in a tear at urgent endoscopy is applied.

Bleeding in case of Mallory-Weiss syndrome. Surgical treatment in most cases involves suturing of the fissure of the mucous with a catgut suture, starting from its lower edge; the ligation of the left gastric artery is obligatory. In case of the bleeding that continues, the suturing of the fissure and the ligation of the left gastric artery are to be complemented by vagotomy.

The technique of clipping vessels in the fissure is applied in case of urgent endoscopy.

Bleeding in case of gastric cancer. The method and the extent of surgical treatment for bleeding gastric cancer is determined by the localization of the tumor, the stage of the disease and the degree of the blood loss. When the tumors are removed, either radical or palliative resection of the stomach, or gastrectomy are performed. In case of inoperable immobile tumors, their invasion into the neighboring organs, as well as in case of cancer peritonitis or with large metastasis, the treatment is limited to the suturing of the vessels around the tumor and the ligation of large arteries of the stomach. To prevent possible perforation around the tumor, the portion of the greater omentum is fixed.

In case of the bleeding from digestive tract diverticula (esophagus, intestines) large-sized diverticula, which recurrently bleed and the occurence of diverticulitis are the causes for surgical treatment – diverticulectomy or resection of the intestine is carried out in case of multiple diverticula.

In case of the bleeding from benign non-epithelial tumors of the digestive tract,the treatment should be only operative – resection of the stomach, excision of the tumor of the esophagus, stomach or intestine, resection of the stomach or intestine with a tumor.

In case of hemobilia the dissection, drainage and tamponade of the liver hematoma are performed. Besides, it is necessary to drain the common bile duct with the aim of the decompression and irrigation of the biliary tract. The most radical operation is opening of the hematoma with ligation of the bleeding vessels and the gallbladder, or liver resection.

In case of the bleeding due to Zollinger-Ellison syndrome, the surgical treatment involves the removal of the ulcerogenic pancreatic adenoma and simultaneous operative intervention for the bleeding stomach or duodenal ulcer (organ preservation surgery or resection methods).

Osler-Rendu disease. When the gastric bleeding is often recurrent and profuse, gastric resection is indicated.

Werlhof disease. Surgical treatment involves splenectomy, the absolute indications for which is a sustained repeated bleeding "at the height of the bleeding", despite the conservative treatment, and the threat of intra-cerebral haemorrhage.

II.10. DIFFERENTIAL DIAGNOSTICS IN CASE OF GASTROINTESTINAL HEMORRHAGE

Acute gastrointestinal hemorrhages (AGIH), which are combined under the name of "Acute gastrointestinal hemorrhage syndrome", and may occur as a result of various diseases. Most often the bleeding occurs in case of the peptic ulcer disease (60-85%). The gastrointestinal bleeding may also often occur due to the lesions of the vascular wall (impaired permeability, atherosclerotic changes, increased fragility, varicose dilation, aneurysms), in case of the impaired coagulation properties of the blood and its fibrinolytic activity. Bleeding into the lumen of the digestive tract is a serious complication registered with a large number of diseases, it is of critical and sometimes of life-threatening character. If earlier the sources of the bleeding were mainly

ulcerative lesions of the digestive tract organs, recently there has been a relative increase in the bleeding occurences, which are of non-ulcerous etiology (Saenko VF, Kondratenko P. R. et al., 1997).

The adopted clinical ***classification of Acute gastrointestinal hemorrhages (AGIH)***, is given above.

To recognize the gastrointestinal bleeding is usually not difficult. Before the common specific signs of the bleeding manifest themselves – vomiting with blood and melena are their clinical manifestations, which are quite vivid. Against the background of a relatively good condition, sudden weakness, sweating, dizziness and "seeing dark spots in one's vision", fainting, palpitations, nausea, thirst, a sudden urge to defecate develop. Acute blood loss is accompanied by the development of a characteristic syndrome, the main symptoms of which are a black and loose or a tarry stool, vomiting with blood or "coffee grounds" type vomiting.

The chronic bleeding, which is initially compensated due to the activation of the hematopoietic system, can further manifest itself by a slow increase in anemisation of the patient; though the blood in the feces can be detected only using the laboratory methods.

The occurence and severity of these signs of bleeding are determined by the localization of its sources, the volume and rate of the blood loss, the speed of its passage through the intestines, and the level of the acidity in the stomach. Therefore, bleeding in the amount of 25-50 ml can be detected only due to the laboratory methods, as to the amount of more than 60 ml – it can lead to the occurence of the black feces. Common melena (tarry stool) may occur when the bleeding is registered with the amount of approximately 500 ml.

Differential diagnostics. The group of "gastric AGIH". Esophagitis and peptic ulcers of the esophagus – are the secondary diseases that develop in the patients diagnosed with the duodenal ulcer, which is accompanied with gastroesophageal hernia of the diaphragm, as well as in case of tumors and esophageal achalasia, and also in case of surgical interventions into the cardia. The bleeding that occurs is rarely severe.

Malignant and benign tumors of the esophagus, as a rule, do not cause heavy bleeding.

Mallory-Weiss syndrome – ruptures of the mucous membrane and submucous membrane of the cardiac orifice and abdominal part of esophagus. The cause of the ruptures, which are often accompanied with a heavy bleeding from the arteries of the submucous membrane, is a sharp increase in intra-abdominal pressure, which usually occurs with vomiting and when lifting heavy weights. The factors contributing to it are: esophagitis, gastritis (especially atrophic gastritis).

Chronic ulcers of the duodenum and the stomach most commonly cause hemorrhages in patients diagnosed with gastrointestinal bleeding. The ratio of hemorrhagic gastric and duodenal ulcers is 1: 4 – 1: 3. The duodenal ulcers bleed most often, especially those, which are located on the postero-medial wall of the duodenum and in the postbulbar partition of the duodenum. The severe bleeding occur more often in the stomach, in case of callous gastric ulcer, which are located on the lesser curvature of stomach. It is related to the fact that large branches of the left gastric and gastro-duodenal arteries are located here. The frequency of bleeding depends on the duration of the course of the disease, but bleeding may also manifest the first sign of the disease, especially in the patients of older ages.

The recurrent peptic ulceration, which occurs after various surgical interventions for gastric and duodenal ulcers, may develop as a result of inadequate choice of the method of operation, technical errors in its implementation, as well as in case of the unrevealed Zollinger-Elisson syndrome before the operation. The peptic ulcers are usually located in the region of the gastrointestinal anastomosis, efferent intestinal loop and afferent intestinal loop near the anastomosis, less often they may occur in the cult of the stomach. The bleeding is often profuse.

Acute (or the so-called stress) ulcers often develop with the background of a cardiovascular accident (Myocardial infarction, thrombosis and great vessels embolism, etc.), severe trauma and burns, intoxication, long-term use of steroid hormones and NSAID (non-

steroid anti-inflammatory drugs), after major traumatic operations. In 30-40% of patients acute ulcers are complicated with bleeding, and often profuse bleeding.

Hemorrhagic erosive gastroduodenitis. In this pathology, which can be considered as a stage of the developing peptic ulcer, the bleeding rarely has a profuse character and it can be quite easily stopped using the conservative measures.

The stomach cancer is usually accompanied with bleeding, in the later stage of the disease when the tumor decays. In case of primary ulcerative forms of cancer, bleeding can be one of the first manifestation signs of the disease. In most patients, parenchymal bleeding from small vessels, of the decaying tumor takes place, which determines a low-grade or moderate-grade blood loss. In case of the arrosion of the vessels of the reborn stomach wall, there are heavy bleeding may occur.

Bleeding from the lower gastrointestinal tract is usually accompanied with the release of feces, stained with dark or red blood, or which look like a "raspberry jelly." Melena rarely occurs if the pathological process is localized in the right half of the colon. Bleeding are very seldom of a profuse character, if to be compared with the case when the bleeding occurs from the upper parts of the gastro-intestinal tract. Anemia develops in case of a prolongated blood loss.

Colon diverticulosis is a fairly common disease that is often complicated with bleeding. Most patients have episodic bleeding.

Nonspecific ulcerative colitis is accompanied, as a rule, with chronic blood loss, acute bleeding is rarely observed in this case.

Colon polyps are often accompanied with anemia due to the chronic blood loss. Intestinal bleeding can often be the first and the only sign of this common and asymptomatic pathology.

Malignant tumors of the colon and rectum is the most frequent (if to exclude the hemorrhoid) cause of bleeding from the lower part of the digestive tract. Bleeding often occurs when the tumor is localized in the left half of the colon (sigmoid colon, rectosigmoid angle); and the severe anemia (which occurs due to cancer intoxication) is more distinctive of tumors of the right half of the colon.

"Agastric AGIH". Syndrome of portal hypertension. Varicose veins of the esophagus and the cardial part of the stomach develop as a result of intrahepatic or extrahepatic occlusion of the portal system, this most often occurs with the liver cirrhosis. In the pathogenesis of bleeding from phlebectasia of the esophagus and the cardia of the stomach, a sudden increase in portal pressure and reflux esophagitis is of big importance. Violation of the integrity of varicose veins more often occurs in the lower, rarely in the middle third, of the esophagus and the cardial part of the stomach. Bleeding is usually massive.

Hemobilia – bleeding in the biliary tract occurs after open and closed liver injury, liver puncture, the bleeding may complicate the course of cholelithiasis, benign and malignant tumors, cysts and abscesses of the hepatobiliary zone. Hemobilia is manifested by bloody vomiting and melena.

Purulent pancreatitis, which is complicated with gastric fistula and arrosion hemorrhage of the stomach or of the splenic artery, leads to profuse, often fatal bleeding into the lumen of the digestive tract.

Diseases of the blood and blood vessels (*Werlhof's disease, leukemia, hemophilia, Henoch-Schonlein Purpura disease*) are other systemic diseases that are the relatively common causes of gastrointestinal bleeding, and which result from the disruption of vascular permeability, quantitative or functional platelet insufficiency, and coagulation disorders. Atherosclerosis and hypertension create conditions for increased vascular permeability, which can lead to acute bleeding. Sometimes it occurs as a result of increased fragility and rupture of the vessel.

Clinical manifestations. When starting clinical examination of patients with AGIH syndrome, it is, first of all, necessary to detect the gastric or duodenal ulcers, in case they are present, as well as the diseases of other organs of the gastrointestinal tract, of the liver, cardiovascular and respiratory systems and of the blood system. Each patient should be

interviewed in case he took any drugs that can cause bleeding into the lumen of the gastrointestinal tract (like aspirin, anticoagulants, corticosteroids, NSAID); or if there were any other factors that may cause bleeding (alcohol intake, rough and spicy food, stressful situations, physical exertion, etc.).

To recognise the gastrointestinal bleeding is usually not difficult. Such classical manifestations of the specific signs of bleeding as bloody vomiting and melena,are accompanied with quite bright general clinical manifestations of the gastrointestinal bleeding. Against the background of a relatively good health condition, the following signs may develop: sudden weakness, sweating, dizziness and "seeing dark spots in one's vision", loss of consciousness, palpitation, nausea, thirst, and sudden urge to defecate.

Medical history is important in the assignment of the causes of the gastrointestinal bleeding. In many patients it is typical for *the peptic ulcer*: epigastric pain after taking food or hunger night pains, heartburn, "the symptom of sodalgia", seasonally disease recurrence, bleeding relapses in the past, previous operations for the peptic ulcer. Bleeding often occurs against the background of acute peptic ulcer disease and is characterized by a decrease or disappearance of epigastric pain (Bergman syndrome) due to the blood being alkaline active. However, even the history of a long-term ulcer does not always indicate the ulcer as a source of bleeding. The bleeding ulcer, the hemorrhagic or erosive gastroduodenitis may occur in a combination.

To make the diagnosis of bleeding *peptic ulcer*, it is necessary to establish the nature of the major surgery that the patient underwent, the time and the nature of complaints. The ocurence of "gastric" complaints and weight loss often indicate *malignant neoplasms of the stomach*. However, such clinical picture may present the course of a large callous gastric ulcer.

There are some important moments in the medical history of patients, who give the history of bleeding from varicose veins of the esophagus and the cardia, which occur most often due to *the liver cirrhosis*. Vomiting with the blood of bright red colour, which occurs very often, is specifically attributed to the liver cirrhosis. It is important to pay attention to the fact if the patient suffered from hepatitis or other hepatobiliary diseases, or if he is an alcohol abuser. However, patients, who are diagnosed with cirrhosis, often suffer from the peptic ulcer. In the decompensation stage of the liver cirrhosis, bleeding from acute gastric ulcer can be registered.

The bloody vomiting, which occurs after repeated vomiting without blood admixture, after a heavy weight lifting, is considered to be a pathognomonic symptom for patients with *Mallory-Weiss syndrome*.

To establish the source of bleeding it is also important to gain the information about *the comorbid conditions* (rheumatism, collagenosis, deforming arthrosis and arthritis, venous thrombosis) and *medications* that were taken before (corticosteroids, salicylates, NSAID, as well as anticoagulants).

Objective examination of the patients, who underwent a detailed examination, is important if it necessary to recognize the causes of gastrointestinal bleeding, especially of non-ulcerous genesis. The following factors may cause the bleeding: coloration of the skin and visible mucous membranes, icteric sclera, acrocyanosis, telangiectasia and vascular spiders, intradermal and subcutaneous hemorrhages, petechial hemorrhage, varicose veins of the anterior abdominal wall (arachnogastria), "frog-like abdomen".

Palpation, percussion and auscultation of the abdomen can reveal tumors of the stomach or intestines, the enlarged liver and spleen, ascites, the enlarged lymph nodes.

Digital rectal examination is a mandatory method to make a diagnosis in patients with gastrointestinal bleeding. Judging from the feces formation and looks, the nature of the bleeding can be defined, and the diseases of the rectum, which could cause the bleeding, can be diagnosed.

The gastric intubation and the stomach lavage can make it possible to judge about the localization of the source of the bleeding and the intensity of the bleeding due to the nature of the gastric aspiration contents and the gastric lavage.

Endoscopic methods of examination go much beyond other diagnostic methods as to the ability to gather the necessary information, as they make it possible to identify the source of the bleeding in 95% of patients' cases. The active diagnostic tactics for patients with gastrointestinal bleeding involves the urgency of the endoscopic research to establish the source of the bleeding, as well as the bleeding activity and its possible stopping due to the exposure via the endoscope. Absolute contraindications for the endoscopy are cardiovascular decompensation, acute myocardial infarction and the stroke.

Esophagoscopy is a reliable method to make a diagnosis of the esophageal bleeding. When the varicosity of the veins is well-expressed, the esophagus has practically no lumen, in case of a more intensive swelling, bluish-colored protrudings into the lumen of the esophagus (beads-like swellings) are revealed. The site of the rupture of the varicose node is often covered by a clot, from where the dark blood enters the lumen of the esophagus.

Gastroscopy makes it possible to identify the source of the bleeding, which is located in the stomach, to identify the blood reflux from the pylorus, in case of the bleeding from the duodenal ulcers. In case of the Mallory-Weiss syndrome in the region of the cardial and subcardial parts of the stomach (in some cases, with the conversion to the abdominal part of the esophagus), the mucous fissures are detected, which are located along the axis of the esophagus and the stomach. When being examined in the early stages of the onset of the disease, active bleeding, up to the arterial one, can be detected, in case of the stopped bleeding – blood clots can be observed in certain sites of a fissure. At a later date, the base of the lesions is covered with fibrin.

Gastroscopy makes it possible to diagnose the bleeding gastric ulcer, to differentiate benign and malignant ulcers, and also to judge about the possibility of the bleeding recurrence due to the size of the ulcer, the occurence of the thrombosed vessels at the ulcer base. Endoscopic diagnostics of acute ulcers is not difficult, but it is not always possible to clearly differentiate them with major erosions of the mucosa.

Duodenoscopy makes it possible to identify the duodenal ulcerous bleeding or to partially judge about it, (if the ulcer is covered with a blood clot), to diagnose hemorrhagic and erosive duodenitis and to identify hemobilia.

Colonoscopy is indicated in case of the clinical picture of the bleeding from the lower parts of the digestive tract in order to identify its cause and, if possible, to stop it. Colonoscopy makes it possible to diagnose diseases, which presuppose bleeding into the lumen of the colon.

In some cases, it is necessary to carry out a differential diagnostics of the gastrointestinal bleeding with the bleeding from the upper respiratory tract and the lungs, which can simulate the bleeding, when the blood is swallowed by the patients. The red foamy nature of the blood that is released when coughing, percussion and auscultation of the lungs, as well as pulmonary fluoroscopy, all that can determine the cause of *the pulmonary hemorrhage*.

II.11. INTRA-ABDOMINAL INJURIES

The severity of the clinical course and the concequences of the traumatic injuries of the abdominal organs give the ground to define this pathology as one of the most severe in urgent surgery. Every year the ammount of traumatic injuries increases. Despite significant advances in the organization of emergency surgical care, the abdominal injuries are often fatal. Providing surgical care to the patients with abdominal trauma remains a difficult task, one of the main factors here is timely pre-admission diagnosis and emergency transportation of patients to the surgical hospital.

Abdominal injuries range from 0,5 to 20% as to be compared with other various types of injuries. Most often the damages occurs due to mechanical action.

There are *isolated injuries, multisystem injuries, concomitant injuries and combined injuries*.

The isolated **injuries** *represent one or several mechanical damages of one organ, if only one surgical correction of the organ is sufficient to repair the damage.*

Multisystem injuries *include mechanical damage to two or more organs or several injuries in one organ in the same cavity, if various individual surgical interventions are necessary to be performed o correct each damage.*

The ***concomitant*** *injuries (****polytrauma****) include mechanical damage to two or several organs in different cavities or simultaneous damage to an internal organ and the musculoskeletal system. The most common is the combination of abdominal trauma and craniocerebral injury (CCI); the more rare are the abdominal and musculoskeletal injuries, abdominal and chest injuries.The concomitant abdominal trauma is marked by an extremely severe level of the damage.*

The most severe among the listed above concomitant injuries is *the thoracoabdominal trauma*. Thoracoabdominal injuries can be penetrating into both serous cavities (pleural and abdominal) or in one of them, but they are always characterized by a damage to the diaphragm. Thoracoabdominal injuries are distinguished from *simultaneous injuries of the chest and abdomen*, which are independent from each other injuries, injuries of the chest wall or organs of the pleural cavity and the mediastinum and abdominal wall or organs of the abdominal cavity and retroperitoneal space, not including the diaphragm.

Combined injuries are the damages to the abdomen and other parts of the body, which are caused by exposure to two or more damaging factors (a combination of mechanical injury with thermal, chemical or radiation trauma).

Isolated damages to the abdominal wall amount to 40,5%.

According to the mechanism of an injury, the two groups of abdominal injuries are differentiated – ***open and closed***. Clinical manifestations, diagnostics and surgical approaches are quite different with open and closed abdominal injuries.

Closed abdominal injuries

Closed injuries of the abdomen are characterized by the absence of abdominal wall wounds, although multiple abrasions and subcutaneous hemorrhages on the skin of the abdomen and adjacent parts may be observed. Sometimes, instead of the term "*closed injuries of the abdomen*," another is used – "*blunt abdominal trauma*." These injuries can be caused by a blow to the stomach with a hard object, by compression of the abdomen, a fall from a height, during an earthfall, and due to the burst effect.

Closed abdominal injuries are subdivided ***into 3 groups***:

1. Damage to the abdominal wall.
2. Damage to the abdominal organs:

a) hollow organs;

b) parenchymal organs.

3. Damage to the retroperitoneal space.

Depending on the extent of the damage, there are ***4 degrees of the severity of a closed injury*** of the abdomen (J. Wedell, 1981).

I degree is marked by the absence of the shock and the absence of the "acute abdomen" signs.

II degree is defined by a light shock, the state of the abdomen is unclear.

III degree is characterized by a clinical picture of shock and the signs of intra-abdominal bleeding.

IV degree is accompanied with a severe hemorrhagic shock and acute hypovolemia.

Among the closed injuries of ***the abdominal wall***, bruising and muscle ruptures, as well as hemorrhages in the subcutaneous fatty tissues are differentiated. Muscle bruising wound is marked by traumatic hematomas with crushing injury. In case of muscle ruptures, a large hematoma of the abdominal wall is observed, which is characterized by a diastasis of torn muscle edges. Besides, the rupture of a large arterial vessel of the abdominal wall may occur, which represents a serious threat to the life of the victim.

Closed damages to ***the internal organs*** most often occur as *multiple closed damages.*

Damages to the parenchymal *organs occur being accompanied with the capsular transgression (fissures, ruptures, disinsertion and crush injuries) and without the capsular transgression (subcapsular and central hematomas).*

The rupture of the parenchymal organ, which occurs immediately after the injury, is called ***immediate*** or ***single-phased***.

Subcapsular hematomas can later be emptied into the abdominal cavity due to the rupture of a detached and stiff capsule, this is accompanied with the intra-abdominal bleeding. Such ruptures of the parenchymal organs are commonly called *double-stage* or double-phase ruptures, since they occur several hours or days after the injury. The central hematoma can attain a large size without any clinical manifestations, but severe functional impairment can be observed.

Fissures and ruptures of the parenchymal organs can be differentiated as linear or star-shaped, single or multiple, superficial or deep. Deep through ruptures, when being connected with each other, cause the separation of a part of the body, which can freely lie in the abdominal cavity or in the retroperitoneal space.

Crushing injury is an extreme degree of an organ damage when, due to the crushing or gunshot wounds, the surgeon discovers the remnants of the capsule, fragments of large vessels of the parenchyma.

A severe injury, whch is associated with a strong blow, can lead to a complete *detachment* of the organ (kidney, spleen) from its pedicle.

When the pelvic and spinal bones are damaged, the integrity of the blood vessels of these parts is violated, which causes retroperitoneal fiber haemorrhage – *retroperitoneal hematoma*.

The hollow organs damages are subdivided into *bruises, crushing injuries, complete ruptures and incomplete ruptures.*

A *complete rupture* is a defect in the wall of an organ, which has a linear or irregular shape.

An incomplete rupture is a damage to the serous or muscular membranes when the mucous membrane is preserved. Sometimes in case of closed injuries of the small intestine, there are numerous internal ruptures of the mucous membrane and of the submucosal layer. The intra-arterial vessels are also damaged and they bleed into the intestinal lumen. The visceral peritoneum and the muscular membrane of the intestine can be changed.

The hollow organs bruises look like separated hematomas. The colon bruise is characterized by a superficial subserous hematoma; the small intestine bruise is marked by a deep submucosa hematoma. The occurence of an extensive hematoma with all the intestinal membranes leaking with blood is indicative of the *crush injury* of the intestinal wall.

The patients with abdominal trauma must be hospitalized, since the symptoms of the damage to the internal organs are variable and clinically not pronounced in the early terms, especially during the first 2 hours after the damage.

Open injuries (wounds) of the abdomen are inflicted with a gun or a knife, or occur due to the secondary impact effects.

Table 22

The frequency rate of the damage to the abdominal and retroperitoneal space

An organ	%
Small intestine	31,8
Spleen	16
Liver	13,6
Colon	3,5
Stomach and Duodenum	1,6
Mesentery of small and large intestine	0,8
Pancreas	0,4
Kidneys	20,1

Open damages to the abdomen. (abdomenal injuries)

The wounds, which are inflicted with ***a bladed weapon***, are differentiated into *stab wounds, incised wounds (sword-cut) and avulsive wounds (tear of the tissue).*

The gunshot wounds are differentiated into *shot-gun-type wounds, missile wounds (perforated wounds, gutter wounds and nonperforating wounds).*

In car accidents and professional accidents, the following injuries, which occur due to the secondary impact effects can be differentiated – glass fragments, metal parts, etc. Such wounds are close to *tear-contused wounds* as to their character.

Open injuries of the abdomen are subdivided into ***nonpenetrating*** into the abdominal cavity and ***penetrating*** into the abdominal cavity, depending on whether the peritoneum remains intact or it is damaged.

In the case of a nonpenetrating wound to the abdomen, the anterior abdominal wall or soft tissues of the lumbar region are most often damaged. It happens extremely rarely that a particular organ, which is located retroperitoneally (duodenum, pancreas, kidneys, bladder) is damaged. In case of nonpenetrating gunshot wounds of the abdominal wall, the organs of the retroperitoneal space and the abdominal cavity can be damaged due to the action of the lateral impact of the vulnerary projectile. The nature of the damage to the organs in such cases is more attributable to the closed injury.

Penetrating injuries of the abdomen *are subdivided into the injuries without any damage to the internal organs and injuries with some damage to the internal organs.*

The following injuries are differentiated: ***the injuries of the hollow organs*** (the stomach, intestines, bladder and the gall bladder), ***the injuries of parenchymal organs*** (the liver, spleen, pancreas and the kidneys) and ***the injuries of the blood vessels*** (the main arteries and veins, the mesentery vessels, omentum and the retroperitoneal space).

Nonpenetrating wounds to the abdomen. Clinical picture. It is characterized by minor abdominal pains. If the pain intensifies during the transportation, but when being admitted to the hospital it decreases – this is actually a nonpenetrating wound. The patient's condition is relatively satisfactory; the skin is of normal color. The pulse and the blood pressure are normal. On the part of the abdomen, there are no symptoms of peritoneal irritation. The intestinal peristalsis is retained.

The digital examination of the rectum and urine examination are compulsory. In severe cases, laparocentesis and laparoscopy, the review fluoroscopy (fluorography) of the abdominal cavity are performed.

Treatment. *Initial surgical debridement* of the wound is to be carried out, during which it is determined whether the wound penetrates or does not penetrate into the abdominal cavity. If the wound is *nonpenetrating*, the treatment is limited to the initial surgical debridement and the tetanus and clostridial difficile infections prophylaxis measures are carried out.

Penetrating wounds to the abdomen. ***Clinical picture*** of the penetrating wounds of the abdomen presents the following *signs: shock, internal bleeding and perforation of a hollow organ. Objective signs of the injuries of the abdomen accompanied with damage to the internal organs are as follows*: protective tension of the muscles of the abdominal wall, positive symptoms of peritoneal irritation, obtundation in sloping parts of the abdomen, lack of hepatic obtundation. On the rectum examination – anterior mucosal prolapse of the ampula and its tenderness are observed. The presence of blood in the urine indicates some damage to the organs of the urinary system. Eventration of the omentum and intestines on the anterior wall of the abdomen suggests a penetrating injury.

Treatment. In case of a penetrating injury, *laparotomy according to Petrov, with a thorough revision of all the abdominal organs* (including their retroperitoneal parts), is carried out, and *providing any damage is detected, an appropriate surgery is to be performed*. In case *of the eventration of the greater omentum* on the anterior abdominal wall from the side of the abdominal cavity, the forceps are to be imposed upon the omentum, the omentum is cut off and is dressed. Its remnants on the anterior abdominal wall are removed.

The diagnostics and treatment of the injuries to various organs of the abdominal cavity are presented below.

Clinical classification and peculiarities of the injuries of various organs.

Injuries to the abdomen with the damage to the ***parenchymal*** organs.

Single and multiorgan injuries to the abdominal organs are differentiated. An example of a single injury is splenic rupture. In case, in addition to the rupture of the spleen, the surgical patient is diagnised the rupture of the small intestine, this refers to a multiorgan damage.

Each organ, in its turn, may have one wound or several wounds. In this regard, isolated *monofocal and polyfocal* injuries are differentiated.

There is a good practice to use the term "*multiple*" when there are several wounds of one organ (multiple ruptures of the small intestine).

When describing the wounds, ruptures and fissures of the organs, it is necessary to indicate their number and localization, using the generally accepted anatomical designations ("multiple ruptures of the lower pole of the spleen", "rupture of the antimesenteric part of the ileum", "perforating wounds of the liver in the 5th and 6th segments" etc.).

Liver injuries. The liver is often damaged, it makes from 10 to 20% of cases. Closed liver injury occurs due to a direct blow, countrecoup and compression.

Classification of the liver damages.

1. ***Closed injuries***.

A. Type of the damage:

a) liver ruptures with a damage to the capsule;

b) subcapsular hematomas, central ruptures or hematomas of the liver, damage to the extrahepatic biliary tract and blood vessels.

B. Degree of the damage:

a) surface fissures and ruptures of up to 2 cm deep;

b) ruptures of up to 2-3 cm deep half-thickness of the organ;

c) ruptures deeper than half-thickness of the organ and through ruptures;

d) split parts of the liver or liver fragmentation.

C. Damage localization (according to lobes and segments).

D. The nature of the damage to the intrahepatic vessels and bile ducts.

2. ***Open injuries:***

a) *the gunshot wounds* (missile wounds, shot-gun-type wounds and projectile wounds)

b) *stab and slash wound*.

3. ***The combination of closed and open injuries.***

Classification of liver damages (G. V. Mikolayev, 1955).

1. Damages to the liver without capsular transgression:

1) subcapsular hematomas;

2) deep and central hematomas.

2. *Damages to the liver,which are accompanied with the capsular transgression:*

1) single and multiple fissures;

2) isolated ruptures and ruptures, which are accompanied with fissures;

3) split liver or the liver fragmentation;

4) ruptures and fissures of the liver, which are accompanied with the damage to the allbladder and major bile ducts;

5) isolated damages to the gallbladder and extrahepatic bile ducts.

In case of ***isolated damages to the gallbladder and extrahepatic bile ducts, A.S. Rarenko's classification (1978) is used.***

1. *Isolated damages to the gallbladder and extrahepatic bile ducts*.

1) penetrating (intraperitoneal and extraperitoneal)

2) nonpenetrating;

3) gallbladder avulsion without its wall burst and its ducts damage.

2. *Joint damage to the gallbladder and extrahepatic bile ducts.*

1) co-damage to the gallbladder and extrahepatic bile ducts:
a) the separation of the gallbladder with a wall gap;
b) detachment of the gallbladder and damage to the extrahepatic bile ducts;
c) rupture of the gallbladder and extrahepatic bile ducts;
2) co-damage to the gallbladder and extrahepatic bile ducts with the damage to other organs of the abdomen.

The damage to the visceral surface of liver is accompanied with a heavy bleeding. Subcapsular hematomas develop on the diaphragmatic surface, and within the first 2 weeks after exercise, the capsule bursts and empties into the abdominal cavity (biphasic rupture). This manifests the clinical picture of the intra-abdominal bleeding. Central hematomas are emptied into the intestines through the bile ducts. The presence of the blood with bile in the duodenum (*hemobilia*) is a severe complication.

The specific characteristics of the liver wounds is *the heavy bleeding into the abdominal cavity with the development of* ***hemoperitoneum*** without a tendency to stop. The main complaints are pain in the right hypochondrium, weakness and dizziness. The skin is pale. The tremulous pulse is typical, the blood pressure decreases as the blood flow increases. The abdomen is retracted, and it participates partially in the respiratory movements. On palpation, the abdomen is soft or slightly tense; the pain is felt in the right hypochondrium. Some dullness is registered in the sloping parts of the abdomen. The phrenicus symptom is positive. The symptoms of peritoneal irritation are not vividly expressed.

Table 23

Clinicopathologic classification of the live injuries (E. Moore, 1986).

The damage extent	**The type and morphology of the damage**	**(AIS) Abbreviated Injury Scale System**
I	Hematoma. Subcapsular and stable. It occupies less than 10% of the surface. Is marked by a rupture (wound). The depth is less than 1 cm, with no bleeding.	2
II	Hematoma. Subcapsular, stable. It occupies 10-50% of the surface. It is central, stable, of less than 2 cm in diameter. Is marked by a rupture (wound). The depth is less than 3 cm, the length is less than 10 cm. Is defined by bleeding.	2
III	Hematoma. Subcapsular, stable. It occupies more than 50% of the surface. It is subcapsular and unstable. Can be of any diameter. Subcapsular, characterised with rupture and bleeding. It is central and stable, of more than 2 cm in the diameter. It is central, unstable, can be of any diameter. Is defined by a rupture (wound). The depth is of more than 3 cm.	3
IV	Hematoma. Central hematoma characterized by a rupture and bleeding. Is marked by the destruction of the parenchyma by 25-50% of the lobe or of 1 to 3 segments.	4
V	Is defined by a rupture (wound). Vascular damage is marked. There is a destruction of the parenchyma of more than 50% of the lobe or of more than 3 segments. Junction-hepatic lesions (inferior vena cava, portal vein, hepatic arteries, bile ducts)	5
VI	Vascular injuries are marked. Detachment of the liver occurs.	6

The specific feature of the liver wounds is a *heavy bleeding into the abdominal cavity with the development of* ***hemoperitoneum*** without a tendency to stop. The main complaints are pain in the right hypochondrium, weakness and dizziness. The skin is pale. The tremulous pulse is typical, the blood pressure decreases as the blood flow increases. The abdomen is retracted, and it participates partially in the respiratory movements. On palpation, the abdomen is soft or slightly tense; the pain is felt in the right hypochondrium. Some dullness is registered in the

sloping parts of the abdomen. The phrenicus symptom is positive. The symptoms of peritoneal irritation are not vividly expressed.

The leading ***diagnostic methods*** are ultrasound examination, laparoscopy, computed tomography.

The main task of the surgeon in case of the liver damage is to stop the bleeding. Nowadays, the following methods of *permanent hemostasis from the liver wounds* are widely used: tamponade with gauze gauze swabs, with the omentum on the pedicle, saturation of the edges of the liver wound, suturing of the liver edge to the peritoneum. The superficial wounds are sutured with interrupted catgut stitches. When the liver is decaying within the anatomical structures, a lobe or a segment of the organ is resected with the help of modern technologies.

Примечание [ПW1]:

Примечание [12]:

Примечание [13]:

Spleen injuries. The ruptures of the spleen amount to 15-30% out of all the injuries of other organs of the abdominal cavity. The favorable factors that contribute to its damage are its low mobility, organ plethora and insufficient strength of the thin capsule. Closed injury of the spleen occurs due to a direct blow to the left hypochondrium, compression of the lower parts of the chest, the inertia force (falling from a height). In case of a closed injury of the chest, a damage to the spleen by the rib fragments may occur.

Types of spleen injuries:

1) blows, which cause no damage to the capsule and the absence of the development of subcapsular hematomas;

2) blows the spleen, which cause no damage to the capsule and the development of subcapsular hematoma;

3) blows with central hematoma, which cause damage to the parenchyma while having the capsule intact;

4) single and multiple deep ruptures;

5) crush injury of the spleen;

6) the spleen detachment from the vascular pedicle.

American Association for the Surgery of Trauma (based on CT data) developed the following ***classification of spleen ruptures:***

Severity grade I – subcapsular hematoma of <10 % of the surface or damages of <1 cm deep;

Severity grade II – subcapsular hematoma of 10-50 % of the surface, damages of 1-3 cm deep, with no damage to the trabecular vessels;

Severity grade III – subcapsular hematoma of > 50 % of the surface, damages of > 5 cm deep;

Severity grade IV – damage that affects segmental or the major vessels with wide devascularization of > 25%;

Severity grade V – organ fracture, damage to the vascular portal of the spleen with complete organ destruction.

The clinical picture of the rupture of the spleen is manifested by *the intra-abdominal bleeding (hemoperitoneum)*. The patients are discomforted by the pain in the left hypochondrium, which radiates to the left shoulder, left shoulder blade, they feel weak and have a dizzy feeling. The skin is pale; tachysphygmia and low blood pressure are registered. The pain and slight muscle tension of the anterior abdominal wall in the left hypochondrium are marked. Dull percussion sound along the left side canal is typical. Rozanov's symptom (a “weeble wobble toy” symptom – when the patient tries to take a horizontal position, he immediately returns to the sitting position, which is caused by a significant increase in pain in the left hypochondrium when lying) is positive. On percussion, a dull feeling in the left hypochondrium is registered. When the blood is accumulated in the abdominal cavity, the dull feeling moves its location with the change in the position of the patient's body.

The symptoms of the ***subcapsular rupture*** of the spleen are scarce. The poured into the capsule blood causes distension of the capsule, which is the reason of the pain in the left hypochondrium. The intensity of these signs depends on the amount of the blood that has been

poured into the capsule. The pains can be quite intense. In some cases, such ruptures, especially small ones, proceed favorably and result in the formation of cysts. In the other cases, rupture develops in the capsule – *a double-stage rupture of the spleen*.

Laparocentesis, laparoscopy and ultrasound examination are of special importance in ***making the diagnosis***.

Surgical treatment in case of damages to the spleen is presented by *splenectomy, resection of the broken part of the organ, splenoraphy*. The indications for splenectomy are as follows: in case of the spleen destruction, multiple deep ruptures of its parenchyma in the perihilar, detachment from the vascular pedicle.

If possible, organ sparing surgery should be performed – resection of the destructed part of the organ, splenoropharya, autotransplantation in the greater omentum (transplanting of the parts of the removed spleen).

At the beginning of the twentieth century, the conservative method of treatment of closed spleen injuries was fatal in almost 100% of cases. Nowadays, the conservative method of treatment can give a favorable result in 24-43% of adult cases and in 63-90% of children cases. In case of the severity grade I of the spleen damage, non-operative treatment is possible in almost 100% of cases, in case of the severity grade II – in 53% of cases, in case of the severity grade III – in 4% of casess and in case of the severity grade V – in 1% of cases.

Pancreas injuries. Open and closed injuries of the pancreas occur rarely due to the pancreas anatomic location. Closed injuries occurs most often as a result of a direct blow in the stomach with the car steering wheel in the car crash (Motor Vehicle Crush). Besides, they may occur when falling from a height or excessive and rapid flexion of the spine. In the vast majority of cases, the pancreas injury is combined with the damage to the duodenum, liver or spleen. Isolated damages occurs in 1-5% of cases.

Примечание [ПW4]:

Classification of closed pancreas injuries.

1. *According to the nature of the damage*:
- bruises and hematomas of the pancreas, with no damage to the capsule;
- incomplete, partial ruptures of the parenchyma or gland capsules;
- complete ruptures;
- detachment of the gland.

2. *According to the localization*:
- head of pancreas;
- body of pancreas;
- tail of pancreas.

Clinical picture. Severe pain in the epigastric region radiating to the back and vomiting are observed. Pallor of the skin, rapid pulse, low blood pressure, tachycardia are registered. On palpation, the abdomen is sharply painful. The muscle tension is manifested 4-6 hours after injury. An increase in temperature and worsening of the intoxication indicate traumatic pancreatitis.

To make a diagnosis, it is of significant to determine the *level of blood enzymes* (amylase, lipase) and *bilirubin* in the blood; *laparoscopy, ultrasound examination and computed tomography* are also helpful.

To assess the severity of the damage, the anatomical localization of the injury should be taken into account: in the head of the pancreas, in the body of the pancreas or in the tail of the pancreas. The most severe are the damages to the pancreatic head.

Surgical treatment in case of the injuries of the pancreas. In case of the pancreas injury, procaine block with antibiotics and antienzymes is performed, and the peritoneal omental sac is drained. In case of incomplete rupture of the gland, the wound is sutured with U-shaped sutures and the peritoneal omental sac is drained. In case of a complete transverse rupture of the gland, the ends of the ducts are to be carefully sutured. The resection of the peritoneal omental sac should be carried out in the distal part of the gland, and drainage of the peritoneal omental sac and abdominal cavity must be performed.

Injuries of the vessels of the abdominal cavity and retroperitoneal space. Any damage to the abdominal cavity and retroperitoneal space is commonly accompanied with non-integrity of the blood vessels. The most frequent cases of the isolated injury of the blood vessels of the abdominal cavity are the injuries of the greater omentum and intestinal mesentery. Any damage to the retroperitoneal organ and the blood vessel is accompanied with the hemorrhage in the loose retroperitoneal cellular tissue. The blood loss in the retroperitoneal cellular tissue can amount to several liters.

Intraabdominal trauma with injured *hollow body organs*

In case of closed abdominal trauma with injured internal organs, the symptoms of peritoneal irritation due to effusion of gastric contents, bile, blood, and feces into the abdominal cavity, appear. Jejunum and ileum are injured most frequently, then the colon, stomach, rectum, duodenum trauma occur.

The pain sensation depends on the type of contents that has effused into the abdominal cavity.

Gastric injuries. *Closed injuries* of the stomach are observed in 1-4% of all abdominal traumas, and arise at severe impact in epigastric region, or in cases of fall from a height. Gastric overfilling with food leads to a hydrodynamic impact and promotes perforation of its wall. The blows, perforations, crush injuries of the visceral wall, and complete stomach detachment are distinguished. It is considered that the anterior wall of stomach is disposed to ruptures, and the posterior wall is disposed to crashes. In extremely rare cases, a complete separation of the stomach from the esophagus, or the stomach from the duodenum are observed.

Symptoms of *complete gastric perforation*:

- severe pain in the upper abdominal cavity;
- cold sweat, dry tongue, vomiting with an admixture of blood;
- board-like rigidity of the abdomen, which is excluded of breathing, tense anterior abdominal muscles, positive symptoms of peritoneal irritation;
- no liver dullness;
- superficial respiration;
- tachycardia.

The open *injury of stomach* is most common in case of thoracic and abdominal wounds. This is due to the fact that major portion of the organ anteriorly is under the protection of the costal skeleton, and is directly adjacent to the diaphragm from the top. Along with it, the left lobe of the liver or the spleen is often injured simultaneously.

Diagnosis. Plain abdominal X-ray examination – radioscopy (-graphy), fibrogastroduodenoscopy, laparoscopy.

Treatment. In case of injured anterior wall of the stomach, wound suturing is carried out transversely to the axis of the stomach with double-row sutures followed by obligatory inspection of the posterior wall.

***Injury of the duodenum*.** Isolated injuries of the duodenum (D) are extremely rare due to its anatomical position, even knife wounds of the duodenum are often combined with wounds of the pancreatic head, upper mesenteric artery, inferior vena cava, right kidney. Gunshot wounds and closed abdominal trauma result in major injury of the duodenum and neighboring viscera. Injury of the retroperitoneally located portions of the duodenum is usually accompanied by appearing hematoma, which very quickly leads to phlegmon of retroperitoneal cellular tissue. The inferior horizontal area is often injured.

Table 24

Classification of duodenal injuries by M.P. Postolov (1983)

№	Characteristics of injuries
1. Isolated injuries	
1.1.	Perforations of retroperitoneal duodenum
	a) involving peritoneum;
	b) excluding peritoneum.

1.2.	Perforations of intraperitoneal duodenum
1.3.	Crush injury
1.4.	Complete separation of the duodenum from the stomach
1.5.	Rupture of half of the diameter of intestine
1.6.	Isolated perforations
1.7.	Multiple perforations
1.8.	Hematomas (intramural) of duodenal walls
2. Combined injuries of duodenum	
2.1.	Involving parenchymal organs (liver, spleen, pancreas)
2.2.	Involving biliary and pancreatic ducts
2.3.	Combined with injured hollow organs
2.4.	Combined with fractures of the bones (ribs, spinal column, pelvic bones, etc.)

Table 25

Duodenal injury grading (Kline G. et al.)

Grade	**Characteristics of injuries**
Grade I	Minor hematoma or subserous perforation
Grade II	Major hematoma or small perforation
Grade III	Large perforation excluding ampulla of major duodenal papilla (Vater papilla)
Grade IV	Large perforation involving ampulla of major duodenal papilla (Vater papilla)
Grade V	Duodenal and pancreatic (head of pancreas) crush

Intraabdominal ruptures of the duodenum are manifested by symptoms of internal bleeding and peritonitis:

- severe pains in the abdomen;
- pale skin, dry tongue, nausea, vomiting;
- tense abdominal muscles (muscle guarding);
- positive symptoms of peritoneal irritation;
- dullness in the flat regions of the abdomen.

In case of the injury of the retroperitoneal duodenum:

- pain in the upper half of the abdomen, more right; then the pain disappears and there is a "lucid space"; in 6-8 hours the pain intensifies;
- tense local muscles in the right hypochondrium;
- mild symptoms of peritoneal irritation;
- nausea, vomiting;
- hepatic dullness is preserved;
- growing signs of intoxication: tachycardia, thirst, dry mouth.

It is difficult to establish ***diagnosis*** even during the surgery. Indirect signs indicating a retroperitoneal duodenal perforation: retroperitoneal hematoma and emphysema, swelling of tissues, saturation of them with yellow-green fluids (Vinivarter's symptom).

Plain *abdominal radiography, fibrogastroduodenoscopy and laparoscopy* are used for diagnosis.

Treatment. In case of injured duodenum, the wound is sutured transversely with a double-row suture. In some cases, after suturing the duodenal wound, anterior or posterior gastroenteroanastomosis is put additionally, and the abdominal cavity is drained.

If grades I-III of the duodenal injury are present, primary suturing is indicated. In case of grades IV-V injuries, intestinal exclusion, its diverticulization, or resection of the duodenum and the pancreatic head (Whipple's et al. surgery) are performed.

Small bowel injury. Among the injuries of the abdominal cavity, the small and large bowel injuries are the most prevalent and make up 29.0-35.3% of cases.

Small bowel injury is the most common type of injuries in the abdominal cavity. Gas and liquid contents in the intestinal lumen contributes to hydrodynamic impact injuring intestinal wall, sometimes a quite significant injury.

Closed abdominal trauma often leads to a single perforation of the small bowel loop. More often, separation of the bowel region from mesentery appears with intraabdominal

bleeding and subsequent intestinal necrosis. In other types of small bowel injuries, crush injury of small intestine with blood effusion in the wall may occur.

The following injuries *are distinguished*:

- injury of the bowel wall with hemorrhages from the side of mucous and serous membrane;
- injury of the wall with subserous hematomas and hemorrhages in the wall;
- injury of the wall with multiple hematomas;
- longitudinal ruptures of mesentery;
- transversal perforations and ruptures of intestinal mesentery;
- tears of the serous membrane;
- single or multiple perforations of the bowel wall exposing its lumen.

Emptying of the hollow organ into the abdominal cavity forms conditions for the ***development of peritonitis.***

Clinical picture:

- sharply pronounced pain spreading all over the abdomen;
- forced position - the patients lie on the side, legs bent in the knee joints and brought to the abdomen;
- abdominal wall in not involved in breathing, it is tense, positive symptoms of peritoneal irritation;
- absent liver dullness;
- dullness in flat parts of the abdomen.

In cases of separation of the bowel from the mesentery, the clinical signs of internal bleeding and acute anemia are prevalent: pale skin, dizziness, frequent and weak pulse, lowering blood pressure, and decreasing red blood count.

Diagnosis: laparocentesis, laparoscopy, plain abdominal radiography.

Treatment. Small wounds of the small intestine are sutured with double-row sutures transversely. Indications for bowel resection: separation of mesentery from the edge of intestine, its crushing, multiple wounds on a limited area of the bowel, large destruction of the intestinal wall. After resection of the intestine, side-to-side anastomosis is applied.

Large bowel injuries. The frequency of large bowel injuries is from 3.5 to 14.0%. They are combined with injuries of other organs. The injuries are caused by: traffic accidents, fall from height, direct blow to the stomach. Having less length, if compared to the small intestine, relatively protected by the ascending, descending colon and rectum cause rarer injuries of the large intestine. The mobile parts of it are often injured: transverse colon and sigmoid colon. Distention of the large bowel with gases and fecal masses contributes to the perforations.

Special group is formed by *injuries of rectum* in accidental or intentional introduction of foreign bodies and compressed gas through the anus, performing enema procedure. All injuries of the rectum are classified into intraperitoneal and retroperitoneal.

The following injuries are distinguished: hematomas of the intestinal wall; hematomas of mesentery; complete and incomplete rupture of the wall.

Classification of traumatic injures of the large intestine.

A. *By the mechanism of injury:*

1. Closed bowel injuries.
2. Cut/stab wounds of intestines.
3. Gunshot wounds of intestines.
4. Spontaneous perforation of bowel.
5. Iatrogenic bowel injury.
6. Thoracoabdominal wounds.

B. *By the extention of injury.*

1. Isolated injury:

a) of the right portions of the colon;

b) of the transverse colon;

c) of the left portions of the colon;

d) of the sigmoid colon;

e) of the rectum.

2. Combined with injuries of other abdominal organs (small intestine + large intestine, liver, spleen, pancreas, etc.).

3. Combination with injuries of other areas of the body (musculoskeletal system, brain, chest, etc.).

C. *By the nature of the injury.*

1. Injuries excluding penetration into the bowel lumen:

a) blows to the intestinal wall;

b) wound or tear of the serous membrane;

c) hematomas of the wall and its mesentery;

d) separation of mesentery with necrosis of bowel;

e) separation of mesentery free of intestinal necrosis;

f) hematoma of the retroperitoneal intestine.

2. Injuries penetrating into the bowel lumen:

a) perforation or wound of bowel that takes 1/3 of a circle;

b) perforation or wound of bowel that takes 1/2 of a circle;

c) perforation or wound of bowel that occupies more than 1/2 of a circle, or a complete perforation;

d) single perforations or injuries;

e) multiple perforations or injuries;

f) through injuries, penetrating into paracolon.

D. *By the presence of early complications.*

1. Clinical pattern of traumatic hemorrhagic shock.

2. Clinical pattern of peritonitis (abscess of the abdominal cavity).

3. Clinical pattern of retroperitoneal phlegmon.

4. Eventration of organs.

Clinical picture of perforation: severe abdominal pain; bradycardia; lowered blood pressure; pale skin; local pain in the abdomen palpated in the projection of injury, spreading then all over the abdomen; tachycardia; tense anterior abdominal wall (muscle guarding); positive symptoms of peritoneal irritation; absent liver dullness; dullness in the flat parts of the abdomen; decreased peristalsis.

In case of injury of the mesentery of the transverse or sigmoid colon, the phenomena of anemia and internal bleeding are registered. The most difficult is to diagnose the injury of the retroperitoneal portion of the colon, because of the subclinical disease pattern.

Diagnosis: rectal examination, radiography of the abdominal cavity, laparoscenesis, laparoscopy.

Treatment. A specific feature of the clinical course, that is the early development of peritonitis or phlegmon of retroperitoneal cellular tissue, has a great, even decisive, significance in determining surgical tactics in the cases of the colon injuries. Primary closing of the large intestine wound with a three-row suture is possible at a less than 2 cm long isolated wound. Lavage of the abdominal cavity, its drainage and transanal intubation of the colon with a thick probe is performed. At multiple, closely spaced colon injuries, resection is indicated. At signs of shock, massive blood loss, contamination, it is necessary to expose both ends of the intestine on the anterior abdominal wall as the end colostomy. Lavage of the abdominal cavity and its drainage should be done.

Intraabdominal trauma involving the *organs of the urinary system*

Injury of the kidneys. *Conventionally, the following injuries of the kidneys are distinguished:*

1. Renal injury non-penetrating the capsule.

2. Non-penetrating injury of the renal pyelocaliceal system with formation of adrenal hematoma.

3. Ruptures of the renal pyelocaliceal system with formation of urohematoma.

4. Crush injury of the kidneys and injuring its major vessels.

Clinical pattern. The classic triad of symptoms of the renal injury: pain, macrohematuria and retroperitoneal hematoma.

The pain is localized in the lumbar region and in the corresponding part of the abdomen. It is often dull, sometimes irradiates into the inguinal area, testicle, urethra. If the lumen of the ureter is obturated with blood clots, renal colic may occur including all its manifestations described above.

Macrohematuria may be accompanied by the appearance of vermiform blood clots, resulted from their formation in the lumen of the ureter. In case of numerous blood clots and if they fail to move away when urinating, the urinary bladder tamponade develops. In this case, the patient complains of pain in pubic region, and inability to empty the bladder. Sometimes tenesmus, painful urges for urination, are observed, which are due to irritation of the mucous membrane of the urinary bladder with blood clots. Hematuria may be profuse and accompanied by general signs of internal bleeding.

The retroperitoneal urohematoma is a consequence of a kidney rupture. However, subcapsular injury is free of it. The accumulation of blood and urine in the retroperitoneal cellular tissue occurs gradually. Fat tissue is being increasingly saturated with fluid. Urohematoma increases in size and exerts pressure on the back of the parietal peritoneum, raises it, bulges into the abdominal cavity, and irritates. Within the first hours after injuring, no signs of irritation of the peritoneum occur. The clinical picture is scarcely seen even in 12-14 hours. Only by the end of days 1 and 2, the signs of peritoneal irritation begin to manifest, but no clinical signs of peritonitis are seen. Gradually, they grow up to the beginning of the day 3 – quite noticeably. It distinguishes the symptoms of urohematoma from those of intraperitoneal injury, especially with enterorrhexis. In such patients, the symptoms of peritonitis increase with each hour. Vomiting, intestinal paresis and other symptoms of peritonitis appear.

The most effective method of ***diagnosis of closed renal injuries*** is the *excretory urography*. It allows the information about the side of the injury, the anatomical and functional state of the injured kidney, the degree of injury, presence and functional state of the contralateral kidney.

In the information is unavailable during the conducting excretory urography, the retrograde ureteropyelography may be performed.

In ***the diagnosis*** of the closed renal injury, such methods of examination as aortography and renal phlebography, ultrasonography, CT are used.

Treatment. About 80% of patients with isolated renal trauma get nonsurgical treatment in a hospital. Such treatment is indicated in cases when the general condition of the patient is satisfactory, and no profuse hematuria, symptoms of internal bleeding, signs of increasing hematoma and urinary infiltration are registered. The treatment is carried out in a hospital, keeping the bed rest for a patient for 10-15 days, and hemostatic, painkillers and antibacterial drugs administration.

Operative treatment is indicated in the following cases:

1) combined renal trauma with injured internal organs;

2) increasing signs of internal bleeding;

3) rapid increase of retroperitoneal hematoma;

4) intense and prolonged hematuria with worsening of the general condition of the patient;

5) appeared signs of acute inflammation in the injured kidney or paranephric tissues;

6) increasing hyperazotemia.

It is always required to attempt ***an organ-preserving operation*** - closing the wounds of the kidneys and of the renal pelvis, wound debridement in case of separation of the kidney segment, closing the capsule.

If the removal of the injured kidney (**nephrectomy**) is required, it is necessary to make sure that the second kidney is present and check its full functional value.

Injury of the urinary bladder. *Incidence.* Complete or partial lesion of the bladder wall is registered in 5-15% of all traumatic injuries. In children, it makes 4.4-11.5% of injuries of internal organs. More than 60% of all cases of bladder traumas are combined injuries. Non-traumatic injury to the bladder and rectum occurs in cases of fall on the sharp objects, in cases of gunshot wounds. In cases of the closed abdominal trauma, extraperitoneal and intraabdominal perforations of the bladder are seen, which mechanisms are different.

Classification:

- closed, open;
- isolated and combined (in combination with fractures of the pelvic bones, external genital organs, organs of the abdominal cavity, rectum, etc.);
- intraabdominal, extraperitoneal and combined;
- non-penetrating and penetrating.

Clinical manifestations. The main symptoms of bladder injury include *pain, urination disorders and bleeding.* In extraperitoneal bladder rupture, the pain is localized in the lower abdomen over the pubis with irradiation in the perineum, rectum, penis.

Examination shows abrasions of the skin of the lower abdomen, dulling percussion sound over the pubis, tenderness at the palpation of this area, anterior abdominal wall muscle guarding, symptoms of peritoneal irritation, absent or disordered urination act, admixture of blood in urine. ***To confirm the diagnosis***, *catheterization of the bladder*, *cystography*, *excretory urography* are used.

Treatment is operative or medical (nonsurgical). *Surgery is the basic method*.

Operative treatment. *In closed intraperitoneal injuries* the laparotomy, abdominoscopy (closing the ruptures of the parenchymal organs, then surgical intervention at the gastrointestinal tract are done). The wound of the urinary bladder is closed by a two-row suture. The abdominal cavity is drained. The permanent catheter is left in the bladder for 5-7 days.

In case of closed extraperitoneal injuries, medial suprapubic access is used. The paravesicular hematioma is voided, free bone fragments are removed. The urinary bladder ruptures are closed, a two-row ketgut suture is advisable. Epicystoma is applied. If necessary, the pelvic cellular spaces are drained.

Medical treatment is indicated in cases of blows and incomplete bladder perforations. In the hospital conditions the complete rest, cold on the stomach are indicated. Hemostatic, anti-inflammatory, analgesic agents are prescribed. In rare cases, a permanent urinary catheter is applied for 3-5 days, or 3-4 periodic catheterizations are performed.

According to the standard pattern, a ***plan for additional examination (laboratory and instrumental) of a patient with suspected abdominal trauma*** includes:

1) laboratory tests (clinical blood and urinary tests, daily diuresis, Ht, total blood protein, blood electrolytes);
2) X-ray plain examination of the chest and abdomen;
3) X-ray contrast study (of the digestive organs);
4) computed tomography (CT) of the abdominal cavity;
5) ultrasonography (US) of the abdominal cavity;
6) laparocentesis;
7) laparoscopy.

Summarizing the above, it is necessary to emphasize the following.

Clinical and diagnostic program for patients with suspected abdominal injuries

1. Interviewing of the patient or those persons who delivered him to clarify the circumstances and the mechanism of injury (blunt blow, fall from a height, running-down accident, compression).

2. Detection of indirect external signs of injury while examining, such as wounds, abrasions, hemorrhages on the abdominal wall and in the lumbar region.

3. Assessment of the patient's condition and detection of three main traumatic syndromes: shock, hemorrhagic and peritoneal.

Clinical picture of open and closed abdominal trauma depends on the degree of injury of the abdominal wall and the specific injury to the internal organs. In case of considerable trauma of the abdominal wall, the following features are revealed: abdominal pain in rest, intensifying at breathing, tenderness on palpation, muscular guarding, possible phenomena of peritonism.

In case of injury of parenchymal organs, clinical picture of intraabdominal bleeding is observed.

In case of injury of hollow organs, clinical picture of peritonitis is seen.

In case of trauma of the retroperitoneally located organs the picture is subclinical, yields little information, and is growing with the development of complications.

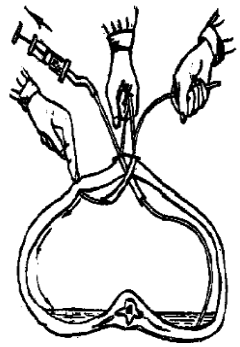


Fig. 60. Laparocentesis with "hunting catheter"

The diagnosis is based on clarification of the mechanism of injury, and if the clinical picture is pronounced, it is unmistakable.

General clinical methods of diagnosis do not always allow revealing and excluding injuries of the internal organs.

In unclear cases, when it is impossible clinically to exclude injury of internal organs, ***laparoscopy and laparocentesis are used*** *as evidence-based medicine methods*. Kharkiv Institute of General and Emergency Surgery proposed a high informative ***technique of laparocentesis that is a "hunting catheter"*** (a metallic S-shaped, bent at the end catheter) (Figure 60).

Diagnostic and therapeutic algorithm for blunt abdominal trauma.

1. Taking history.

2. Examination (visual, palpation, auscultation, per rectum).

3. Laparoscenesis with a "hunting catheter" and abdominal cavity drainage, dynamic monitoring. In case of the revealed blood, the Rouvielois-Gregoire test is performed.

4. Laparoscopy.

5. X-ray examination of the abdominal organs in the injured visceral organs can give a picture of free air present in the abdominal cavity.

6. In obscure cases – diagnostic laparotomy (if necessary with expanding the surgery volume aiming at the detection of injured organs).

Diagnostic and therapeutic algorithms for open abdominal trauma.

1) History taking.

2) Examination.

3) Determining the nature of the injury (penetrating or non-penetrating) with the use of research methods:

a) ISD (initial surgical debridement) and surgical wound exploration;

b) Contrast radiography of penetrating wounds;

c) Manometry of the wound injury;

d) Laparoscopy.

4) Laparotomy and a thorough revision of all organs of the abdominal cavity (including their retroperitoneal divisions) and diaphragm; in case of the detected injuries, their closing (see above).

Dynamic monitoring of pulse, blood pressure, blood parameters and coagulogram is required in case if no information about the injured viscera is available.

Treatment of a patient with an abdominal trauma is given above for each type of injury.

Postoperative complications:

- failure of sutures of the intestinal wound and interstitial anastomoses;
- abscesses of the abdominal cavity;
- suppuration of postoperative wounds with the eventration of intestinal loops;
- duodenal fistula;
- postoperative peritonitis;
- retroperitoneal phlegmon;
- postoperative pancreatitis;
- acute adhesive intestinal obstruction;
- pneumonia;
- acute renal and hepatic failure;
- bleeding from the wound, internal bleeding, gastrointestinal hemorrhage, secondary bleeding from the edges of the wound;
- postoperative pneumonia;
- thrombosis of the veins of the lower extremities;
- parotitis, bedsores, psychopathological syndromes.

II.12. DIFFERENTIAL DIAGNOSIS, TREATMENT AND PROPHYLAXIS OF ACUTE ULCER OF DIGESTIVE TRACT ORGANS

Acute ulcers of the digestive tract is a topical issue determined not only by the quantitative growth, but also by the lack of noticeable success in improving timely diagnosis and the results of treatment of patients with this disorder. Bleeding from acute ulcers and erosions are registered in 9-30% of all patients with acute gastrointestinal hemorrhage.

Acute erosions (AE) *are the superficial defects of the mucous membrane of different (linear, round, irregular) shape, 1-3 mm in size, 0.5-2.0 mm deep, penetrating only to the muscular layer of the visceral wall.*

Usually, the bottom of erosion is clear, bright red, sometimes with a hemorrhagic coating. The edges are low, often flattened. Hyperemia, swelling and mild vulnerability of the mucous membrane surrounding erosions are registered.

In contrast to erosions, ***acute ulcers (AU)*** of the *digestive tract (DT) can penetrate the submucosal, muscular, serous layers, and sometimes perforate the wall of the organ, as a rule, they are round or oval with a diameter of up to 1.0-1.5 cm, although sometimes reaching enormous size.*

Usually, no signs of inflammatory reaction are seen around the acute ulcers. Externally they resemble punched-out defects of the mucous membrane. This is due to the prevalence of necrotic over regeneration processes. Acute ulcers are often superficial, with smooth clear edges, the bottom is covered with red fibrin, the mucous membrane is elastic. Inflammatory swell, characteristic for chronic ulcers, is absent. In most cases the acute ulcers are wedge-shaped, with the vertex of the wedge facing deep into the wall of the stomach. However, there may be acute ulcers with an underlined suspended oral edge and a smooth anal edge. The necrosis and fibrin exudation of the bottom of the ulcer in the region of the underlined edge is much greater than at the opposite edge. Sometimes there are ulcers with underlined edges and mucous membrane which is fused over the ulcerative defect. As a result, the ulcer sections have a triangular shape with a vertex tropic to the space of the stomach. The bottom of acute ulcers is usually covered with necrotic masses, which are infiltrated by fibrin and abundantly impregnated with leukocytes containing a large amount of acidic glycosaminoglycans. Under the necrotic masses, there is a zone of fibrinoid necrosis. In acute ulcers, unlike those chronic, fibrinoid necrosis is not entire, but occurs in the small areas with weak positive PAS-reaction. A variety of microflora often appears in necrotic tissues.

Localization of acute ulcers. Acute ulcers and erosion in 2/3 of the observed cases are located in the stomach, and the rest 1/3 cases are found in other digestive organs. Selye in 1936 revealed that a stress ulcer in experimental animals is found not only in the stomach, but also in the large and small intestine, and even in the appendix. There have been described acute ulcers of the esophagus, small and large intestines, respiratory and urinary tract, as well as stress, which injures liver, pancreas, heart and other organs.

Acute erosions, as a rule, are multiple, located predominantly in the body and at the bottom of the stomach or in the initial portion of the duodenum. Meanwhile, they literally disseminate the entire mucous membrane of the affected area of the organ. Acute ulcers can be either single or multiple. Their localization is different: from the stomach and duodenum, which is more common, to the esophagus and intestines. According to V.P. Khokholya (1988), erosive ulcerative lesions in 83.2% of observations were located in the stomach, in 35.4% they were found in the duodenum, 12.4% in the esophagus, 3.6% in the small intestine and 0.8% in the large intestine. Moreover, in 35.4% of patients, acute ulcers and erosions were localized simultaneously in two or more organs. The author found that in 32.8% of cases, multiple acute erosions of the mucous membrane were detected, 41.9% were acute ulcers, 22.5% were ulcers combined with erosions, 5.4% were multiple hemorrhages in the mucous membrane, and 15.5% hemorrhages, acute ulcers and erosion. Single acute ulcers were in 32.7% of patients, and multiple in 67.3%. Moreover, in 69.7% of patients, their number ranged from 1-15 to 30 and more; those acute ulcers localized in the stomach (65%) were predominantly multiple (73%), and those localized in the duodenum (26%) were more often isolated (67.6%).

A distinctive feature of acute ulcer, unlike chronic, is the absence of cicatricial sclerotic tissue in the area of its bottom and around the ulcer itself.

Causes of erosion and acute ulcers. Acute ulcers and erosions may occur as a complication in severe diseases, sepsis, burns, injuries, shock, surgical interventions, intake of some medications, alcohol and other toxic substances.

Until the 1970s, acute ulcers and erosion of the upper digestive tract were referred to rare diseases. Over the past two decades, numerous reports have appeared on the occurrence of acute ulcers and erosions of the mucous membrane of the digestive tract followed the severe trauma, burns, traumatic surgical interventions against the background of severe somatic diseases, and resulted from cardiovascular, respiratory, hepatic, renal failure, sepsis, shock and a number of other causes that lead to significant homeostatic disorders. Acute erosive ulcerous lesions of the mucous membrane of the digestive tract can occur after the administration of various drugs (corticosteroids, acetylsalicylic acid, butadion, etc., total of more than 50 agents). Moreover, some drugs have a direct harmful effect on the mucous membrane, others destroy the protective mucosal barrier or stimulate the production of hydrochloric acid, creating favorable conditions for its destruction. However, it is not absolutely correct to attribute such injury "purely" to the drug-induced. It should be noted that drugs are used for a long time or in large doses, usually for the treatment of severe somatic diseases of the heart, blood vessels, lungs, liver, kidneys, other organs and systems.

The risk factors for acute ulcers and erosions of the esophagus, stomach, small and large intestine mucous membrane include: severe stress conditions, severe injuries, especially craniocerebral, burns, various types of shock, sepsis, some somatic, including severe, surgical diseases, accompanied by cardiovascular, respiratory, hepatic, renal failures, dehydration, hypovolemia, intoxication, as well as myocardial infarction, stroke, blood diseases, advanced stages of malignant neoplasms, atherosclerosis, hypertension, coronary heart disease (CHD), diabetes mellitus, some infectious diseases (influenza, etc.). Diseases of digestive organs in history, in particular ulcer, gastritis, gastroduodenitis, have a certain value in the acute ulcer development in mucous membrane. Quite often acute erosive ulcerative lesions occur in patients following the surgical intervention on the heart, lungs, pancreas, biliary tract, intestines, aorta and major arteries. Less common acute ulcers are found in patients who have undergone surgery, especially radical, on the stomach.

The study of the causes of acute ulcers and erosions allowed to establish that in addition to surgical intervention, the nature and severity of the underlying disease, its complications, comorbidities, as well as the quality of anesthesia and intraoperative infusion therapy are very important.

Surgical injury, that is an operational stress, is one of the most significant factors. The direct relationship between the frequency and severity of erosive ulcer lesions and the number of risk factors for ulceration was revealed. Since severe injuries, burns, various types of shocks, severe somatic diseases and other cause are regarded to be the risk factors for acute erosive ulcerative lesions of the digestive tract mucous membrane, this process should be considered as a form of organ failure in multi-organ or polysystemic insufficiency.

Free radicals of oxygen are of great importance in the occurrence of visceral disorders in cases of ischemia and subsequent reperfusion.

Disordered hemostasis, inhibition of protein-synthetic liver function due to deep disorders of general and local hemodynamics in seriously ill patients play a certain role in the occurrence of acute ulcerative erosive lesions of the mucous membrane of the digestive tract and the development of hemorrhagic complications. In the close postoperative period, the majority of patients develop *increased basal secretion of hydrochloric acid against the suppression of motility of the stomach. The erosive lesions appear, as a rule, when the pH level in the lumen of the stomach is less than critical (3.5-4.0).*

One of the factors contributing to the acute ulcerations and erosions of the mucous membrane of the stomach and duodenum is the change in its *resistance.*

Thus, the development of acute ulcers and erosions of the mucous membrane of the digestive tract is due to endogenous intoxication and is a manifestation of organ failure in polyorganic or polysystemic insufficiency. However, the acute ulcerations and erosions of the mucous membrane of the stomach contribute to a number of additional factors, which include hydrochloric acid, pepsin, motility disorders and evacuation of stomach contents, duodenogastric reflux. Perhaps precisely because of this, acute erosive ulcerative lesions of the mucous membrane are most often localized in the stomach.

Undoubtedly, an important role in the development of acute ulcers and erosions of the mucous membrane of the digestive tract is played by the sensitization of the organism, infection, various hormones, including gastrointestinal, prostaglandins, mediators and other biologically active substances, as well as the gender and age of patients, the value and place of which are to be further specified in the pathogenesis.

The findings concerning the frequency of acute ulcers of the mucous membrane of the digestive tract in different pathological conditions vary considerably. Most often, they are observed in cardiovascular diseases, as it is evidenced by the results of autopsy.

It should be noted that the ulcers of the mucous membrane may follow not only a rather traumatic intervention (reconstructive operations on the aorta and major arteries, surgery for pancreatic cancer and chronic pancreatitis, etc.), but also an appendectomy, removal of hernia, cholecystectomy. Moreover, the heavier and more traumatic the surgical intervention was, the earlier the ulcerative lesions of the upper parts of the digestive tract appear, and the greater degree and severity of the injury are registered, and the more often they are accompanied by complications, first of all by bleeding.

Numerous and various forms of ulcerative destructive processes, obviously, reflect the diversity of pathogenetic mechanisms affecting this process, and a different degree of resistance of those or the other areas of the mucous membrane.

Conventionally, the acute ulcers are classified into the following:

I. *Acute (symptomatic) ulcer of the stomach.*

1. Localization: L_1 - cardial portion; L_2 – subcardial portion; L_{13} - small curvature; L_4 - large curvature; L_5 - pyloric portion.

2. *Phase of the process*: F_1 - active; F_2 - cicatricial.

3. *Cause*: E_1 - burns (Curling's ulcers); E_2- myocardial infarction; E_3 - sepsis; E_4 - severe injury; E_5 - undergone operation; E_6 - use of medications; E_7 – central nervous system (CNS) disorder; E_8 – hypoxic state.

4. *Complications*: O_1 - acute bleeding; O_2 - perforation.

II. *Acute (symptomatic) ulcer of the duodenum.*

1. *Localization*: L_1- duodenal bulb; L_2 - behind the bulb.

2. *Phase of the process*: F_1 - active; F_2- cicatricial.

3. *Etiology:* E_1- burns (Curling's ulcers); E_2 - myocardial infarction; E_3 - sepsis; E_4 - severe injury; E_5 - undergone operation; E_6 - use of medications; E_7 - central nervous system (CNS) disorder; E_8 – hypoxic state.

4. *Complications*: O_1 - acute bleeding; O_2- perforation.

Uncomplicated acute ulcers and erosions of the gastric and duodenal mucosa are mostly asymptomatic. In 47% of cases, they are detected only on autopsy.

Clinical picture of acute ulcers and erosions of the digestive tract is highly changeable and depends on the depth and location of the lesion, the presence of complications, and some other factors. It is always combined with the symptoms of the underlying disease and is often masked by it. An opposite picture, however, is often seen: complications of acute ulcers (bleeding or perforation) are the dominant signs of exacerbation of the main disease.

Considerable number of patients have the clinical course of acute ulcers and erosions of the mucous membrane with no complications or accompanied by a slight, clinically unidentified bleeding. At the same time, 25% of observations, especially against the background of extremely serious diseases, manifest with a massive bleeding or perforation, which greatly aggravates the patient's condition, often leading to a fatal outcome. Curling's ulcers are complicated by bleeding in 2/3 of the cases, and 45% of them are profuse bleeding.

According to A.K. Ageev (1984), the infectious processes in the mucous membrane are essential for the development of acute ulcers and erosions, along with the impact of various stress states, in which the impaired microcirculation is observed. Herewith, the infection has both negative effect on the microcirculation, and a direct damaging effect on the tissues.

Interviewing of a patient shows the following ***complaints*** of the underlying disease: weakness, abdominal pain in the epigastric region, belching, nausea, sometimes vomiting, lack of appetite, flatulence, and often constipation. The cases free of complaints caused by other organs and systems are possible.

The history of the disease: an "ulcer history", the intake of certain medications, aggressive to the gastric and duodenal mucosa. It should be clarified, since when the patient has been ill, which treatment he got and the effect of it, the findings of previously performed additional methods of examination - laboratory, X-ray, instrumental. ***The past history:*** harmful habits and chronic diseases are specified.

Clinical physical examination. The general condition of the patient, as a rule, ranges from a relatively satisfactory to a medium severity. Skin and visible mucous membranes are of normal color. Patients are quiet. Cardiovascular system: rhythmic heart sounds, pulse – no specific findings, blood pressure normal.

Local status. The examination of the abdominal organs state (abdominal examination, palpation and percussion of the stomach, palpation of the intestines, liver, spleen, kidneys, pancreas, pelvic organs, auscultation of the abdomen) may show no specific features.

Perforation and bleeding are the frequent ***complications of AU***.

Clinical picture of the AU perforation in most patients is very scarce. Such classical symptoms as sudden severe pain, protective guarding of abdominal muscles, and the symptoms of peritoneal irritation are often absent or appear so weak that they do not always attract the doctor's attention. The perforation is usually accompanied by a worsening of the general condition of the patient, which is difficult to explain by the course of the underlying disease. There is a gnawing pain in the epigastrium, retrosternal pain, shortness of breath, sometimes with

short-term loss of consciousness, gastric and intestinal paresis, and also collapse as an acute circulatory failure, which cannot be helped with the aid of cardiovascular agents.

Clinical course of bleeding with AU and AE is also often characterized by very scarce and slight symptoms. The initial symptoms of bleeding (weakness, dizziness, nausea, vomiting, tachycardia) are often remained unnoticed or related to the manifestations of the underlying disease. Frequently, the first and the only sign of bleeding is a ***collapse***, which is usually associated with cardiovascular failure or myocardial infarction. The analyzed reference and clinical data showed that the most common clinical manifestations of bleeding from the acute ulcers of the mucous membrane were the following: weakness in 75-100% of patients, bloody feces in 59-88%, bloody vomiting in 63-68%, nausea in 35-37%, dizziness in 29-31%, abdominal pain in 21-30%, collapse in 10-13%. Bloody vomiting, as an independent symptom of bleeding, occurs in 12% of patients, bloody feces in 37%, and their combination in 51%. Vomiting with slightly changed blood and clots was registered in 58-64% observations, and with the 'coffee-grounds' content in 32-36%.

The other complaints (regurgitation, heartburn, tinnitus, 'flying flashes' in the eyes, drowsiness, lack of appetite, bloating, diarrhea, palpitations, chills) are relatively rare and thus have no significant value in the diagnosis of bleeding acute ulcers and erosions of the digestive tract.

In cases of postoperative bleeding from ulcers of the mucous membrane, such signs as bloody vomiting and melena are observed only in 2/3 of patients. The doctor's inadequate attention to these signs, and thus missing them, may lead to incorrect interpretation of the almost always concomitant bleeding hemodynamic disorders, which are considered as signs of cardiovascular failure.

According to some authors, frequency of ***perforation of bleeding acute ulcers*** is 6-34% in relation to bleeding acute ulcers. Quite often the perforation is combined with bleeding. In case of perforation of a chronic gastroduodenal ulcer, bleeding is revealed in 2.5-2.7% cases, if perforation of acute ulcers occurs, it is seen in 41-50%. In children, AU perforation is often preceded by bleeding.

In clinical practice it is often necessary to deal with some somatic diseases, the course of which is sometimes complicated by massive bleeding from acute erosive ulcer lesions of the mucous membrane of various organs of the digestive tract.

In this regard, the more detailed ***peculiarities of pathogenesis and clinical manifestations of acute ulcers and erosions in some diseases*** are described.

Diseases of the cardiovascular system. According to many researchers, symptomatic ulcers and erosions occur in approximately 20% of patients ***with atherosclerosis of the abdominal aorta*** and its atherosclerotic aneurysm and in 20-25% of patients with obliterating atherosclerosis of the vessels of the lower extremities, especially in men aged 50-70 years. Moreover, the risk of developing ulcers increases to 50% after the amputation of the lower extremities due to this disease. In the pathogenesis of the formation of acute ulcers and erosions of gastric and duodenal mucous membrane in patients with atherosclerotic lesion of the abdominal cavity vessels, the key role is played by the ischemia and hypoxia of the mucous membrane, and endogenous intoxication, as well. Such ulcerative lesions can sometimes be large or even gigantic and, as a rule, occur asymptomatic, manifested by massive gastrointestinal bleeding or ulcer perforations. In some observations the multiple ulcers localized in different organs. Medical treatment for acute erosive and ulcerative lesions of the digestive tract mucous membrane of atherosclerotic genesis is often inadequately effective.

Acute ulcers and erosions of the mucous membrane often complicate the course of chronic ***coronary heart disease***. The development of such lesions can be explained by the deteriorated blood circulation of organs and tissues due to heart failure. Increasing of the latter resulted from the diordered water-salt metabolism, increased volume of circulating blood, venous influx, etc. promotes an even greater impaired hemodynamics: increased venous

pressure, slowing down the blood flow rate, etc. All this leads to hypoxia, to the formation of acute erosive ulcerative lesions, more often of the stomach or other organs.

Quite often symptomatic ulcers and erosions are observed in patients with ***myocardial infarction***. Such lesions are found in 8-16% of patients who died of heart attack. According to L.T. Malaya et al. (1976), almost 30% cases of gastroduodenal ulcers are found in patients with abdominal form of the disease. Symptomatic ulcers and erosions are characteristic for the acute period of myocardial infarction, especially the severe and complicated course. The pathogenesis of ulcer formation is based on the disorders of microcirculation in the mucous membrane of the stomach and duodenum. These are: arterial constriction, intravascular aggregation of red blood cells, increased capillary permeability, and a number of other disorders. The listed changes develop as a result of general disorders of hemodynamics in patients with myocardial infarction and manifest an organ failure in polyorganic insufficiency. Since the days 2 and 3 of myocardial infarction, paresis of gastric and duodenal smooth muscles accompanied by decreased tone of the gastric vessels, the thrombosis development and formation of new destruction sites in the mucous membrane, serves as an additional factor of ulcerogenesis. In the late period of myocardial infarction, autoimmune disorders, as well as the prolonged use of drugs with ulcerogenic effect (salicylates, glucocorticoids, reserpine, and others) may contribute the development of acute ulcers and erosions. Clinical picture of secondary gastroduodenal ulcers in patients with myocardial infarction is characterized by obliterated manifestations and no specific symptoms. Often, the first manifestation of such disorders as myocardial infarction itself is a massive gastrointestinal bleeding or perforation of ulcers.

Acute gastroduodenal ulcers and erosions can develop in patients against the background of ***hypertension disease*** that existed before. Often, symptomatic erosions and ulcers arise in the face of severe hypertensive crises, cerebrovascular disorders, and characterized by obliterating clinical manifestations, no specific symptoms, frequent development of gastrointestinal bleeding (in 39% of cases), lower production of hydrochloric acid (V.H. Vasylenko et al. 1987). The earlier developed changes in blood vessels contribute to the formation of acute ulcers of the mucous membrane. According to A.L. Grebnyov et al. (1982), the observed "hypertonic microangiopathy" vascular lesions can reduce trophic processes in the mucous membrane of the stomach and create favorable conditions for the occurrence of ulcers and erosions, as evidenced by plasmorrhagia, hyalinosis, thickening of the basal membrane, proliferation of the vascular wall endothelium. These changes mostly explain the frequent gastrointestinal bleeding in the development of symptomatic gastroduodenal ulcers and erosions agaisnst the background of hypertension.

Diseases of the respiratory organs. The frequency of development of acute erosive ulcerous lesions, more often in the stomach, in patients with ***non-specific diseases of the lungs*** is 9-30%. The key role in their development is played by prolonged hypoxia of the gastric mucous membrane, caused by the underlying disease with all its consequences. In addition, the elderly patients and concomitant cardiovascular diseases also contribute to the progression of circulatory disorders in the stomach wall. The clinical pattern of symptomatic ulcers and erosions of the mucous membrane in patients with chronic non-specific lung diseases is characterized by obliterating manifestations, almost complete absence of pain syndrome and dyspeptic disorders. The course of such disorders is often complicated by gastrointestinal bleeding.

Development of acute erosive and ulcerative lesions of the mucous membrane of the digestive tract in patients with ***primary pulmonary tuberculosis*** is contributed by:

- reduced resistance of the organism against the background of tuberculosis intoxication;
- progression of chronic respiratory failure with subsequent hypoxia of organs and tissues as it occurs in patients with chronic non-specific diseases;
- taking medications that cause irritating effects on the mucous membrane of the gastroduodenal zone.

Tuberculosis of the lungs when combined with symptomatic ulcers and erosions is characterized by a very severe course. Quite often infiltrative, disseminated, destructive forms of the disease with active bacterial secretion are revealed, antituberculosis therapeutic effect is reduced, sputum abacillation is not always achieved. To treat pulmonary tuberculosis, it is necessary to use paraaminosalicylic acid with caution, which is better to be prescribed parenterally to patients with acute erosive ulcerous lesions of the mucosa without bleeding.

Diseases of the liver and pancreas. The number of patients with symptomatic gastroduodenal ulcers and erosions that developed against the background of chronic liver disease is quite large. Among patients with cirrhosis of the liver, they are found in 11-19% of observations. The frequency of the ulcerations in patients with cirrhosis of the liver, undergone the operation of portocaval bypass surgery, reaches 27%. In almost 60% of cases, "hepatogenic" acute ulcers and erosions are the cause of gastrointestinal bleeding in patients with cirrhosis of the liver. However, not all researchers share these ideas.

Symptomatic ulcerative lesions of the mucous membrane of the digestive tract may occur in various chronic liver diseases. Their incidence depends on the severity of the lesion of the liver. In ***chronic hepatitis*** it is lower than, for example, in cirrhosis of the liver. A number of authors believe that such etiological factors of liver cirrhosis, such as alcohol, viral infection do not affect the frequency of ulceration. Somewhat different is the opinion of Kirk et al. (1980), who found a high incidence of acute ulcers and mucosal erosions in patients with cirrhosis of the liver caused by viral hepatitis B. Disturbances of portal blood circulation, which lead to hypoxia of the mucous membrane of the digestive tract (more often of the stomach), decrease its resistance and development of dystrophic processes in it, which ultimately contributes to the formation of acute erosive-ulcerative lesions mucous membrane, play significant role in the pathogenesis of hepatogenic acute ulcers and erosions. The additional factors for the formation of ulcers are the following: gastric mucosal disturbances, duodenogastric bile reflux, decreased secretion of pancreatic bicarbonates, use of glucocorticoids (for the treatment of chronic active hepatitis and liver cirrhosis).

The clinical picture of acute ulcers and erosions of the mucous membrane *in patients with chronic liver disease is characterized by the atypical symptoms and obliterating manifestations.* Often, massive gastrointestinal bleeding is the first symptom of a disease or exacerbation of the underlying pathology. However, in this category of patients the intensive pain syndrome and dyspeptic disorders are described. It is likely that the severity of certain clinical manifestations of the disease in each particular case is determined by the nature and degree of injury to the liver itself, the level of acid production, localization of ulcers, the degree of morphological changes in the mucous membrane of the gastroduodenal zone.

Acute ulcers and erosion of the mucous membrane of the stomach and duodenum in patients with chronic liver disease are poorly subjected to medical treatment and are usually characterized by an inactive and torpid course. There are great difficulties in connection with the need for prolonged and continuous administration of glucocorticoid drugs. However, during the anti-ulcer therapy, the adverse effect on the liver function of a number of drugs (carbenoxolone, anabolic hormones, etc.) should be taken into account.

According to the reference sources, the incidence of symptomatic, pancreatic gastroduodenal ulcers and erosion in patients with ***chronic pancreatitis*** is 8-24%. Acute ulcers of the mucous membrane in chronic pancreatitis are mainly localized in the bulb of the duodenum. Decreased pancreatic production of bicarbonates, especially at a relatively high level of hydrochloric acid production are of particular importance in their pathogenesis. Inflammatory changes in the mucous membrane of the duodenum, duodenal reflux, enhanced release of kinins, hypergastrinemia are among the additional factors of ulceration. According to some studies, these patients have a rather pronounced pain syndrome. A connection can be traced with the pain and meals, and also a tendency to a persistent course. Gastrointestinal bleeding is somewhat less frequently observed than in other forms of acute ulcers of the mucous membrane. It should be noted that many issues of pathogenesis and clinical manifestations of acute ulcers and erosions

of the gastric and duodenal mucous membrane in patients with chronic pancreatitis have not yet been studied.

Quite a large group consists of patients with symptomatic ulcers and erosions that have arisen against a background of ***type II diabetes mellitus*** (insulin-independent). These erosive ulcerative lesions are characterized by obliterating pain syndrome, almost complete absence of dyspeptic disorders, poorly expressed exacerbation and remission periods, localization of the process predominantly in the stomach, multiple nature of ulcers, and high (in 32% of patients) incidence of gastrointestinal bleeding. In such patients the diabetic microangiopathy changes in the submucosal layer of the stomach are: plasmarrhagy, hyalinosis, thickening of the basal membrane, proliferation of peri- and endothelium, and others. They can play a role in the formation of symptomatic ulcers and erosions in patients with diabetes mellitus, the course of the latter is often burdened with concomitant hypertension and coronary heart disease.

Acute erosive ulcerative lesions can develop in patients with diabetes mellitus in extreme situations, in particular at hyperacidic or hyperosmolar coma, at surgical intervention. The appearance of ulcers in this category of patients is due to endogenous intoxication with multiple organ failure. In addition, the formation of acute ulcers and mucosal erosions in patients with diabetes mellitus may be due to an ulcerogenic effect of insulin, which causes increased acid production, impairs utilization of glucose in the tissues of the stomach, reduces the content of mucopolysaccharides and reduces trophic processes in the mucous membrane.

Kidney disease. Different disorders on the part of the digestive organs quite often occur ***against the background of the terminal stage of chronic renal failure (CRF) - uremia.*** In 1912, Moynichan described chronic rounded stomach ulcers in those died of uremia. The author associated them with decreasing resistance of the gastric mucosa due to atrophic changes. A morphological study by Takacs (1966) made it possible to establish that in 53% of patients with uremia, the hemorrhages were revealed in the gastroduodenal mucosa, in 20% ulcers, and in 27% submucosal edema. These changes are often combined with erosive gastritis. The author attributed the development of such a pronounced gastroduodenal disorder to the capillary stasis and increased vascular permeability, resulting in hemorrhages and edema. Different microbes can easily penetrate the injured tissue, which is probably one of the causes of necrotic processes and ulceration. In this case, as erosive gastritis, and acute ulcers often serve as a source of bleeding.

A number of researchers point to a fairly large number of those patients with chronic renal failure (CRF) combined with acute and subacute gastroduodenal ulcers, who were preparing for kidney transplantation. An increased frequency of acute erosive ulcerous lesions of the gastroduodenal mucosa, including complicated bleeding, was registered in patients with chronic renal failure, who were treated with recurrent hemodialysis, and after kidney transplantation, as well. However, not all authors agree with this point of view. So, Wiener et al. (1965) believe that hemodialysis does not contribute to the formation of ulcers, if it prevents the occurrence of *hypercalcemia*. In cases of its at least periodical development, a gastric hypersecretion and often an ulcer appears.

Hypergastrinemia is one of the causes contributing to the formation of both acute and chronic gastroduodenal ulcers. The latter, apparently, may be due to hypercalcemia and the loss by the severely affected kidneys an ability to destroy gastrin. However, not all patients with chronic renal failure have an increased production of hydrochloric acid. On the contrary, in many of them its secretion is reduced. In many respects it depends on the condition of the glandular apparatus of the stomach. Even excessive gastrin or other endogenous stimulation will not cause an increase in acid production if it develops marked degenerative and atrophic changes resulted from chronic renal intoxication.

Analysis of the reference literature allows to conclude that against the background of recurrent hemodialysis, the formation of ulcers and erosions in patients with chronic renal function is more common than in those treated only by medical methods. To a certain extent, this is due to the development of a complicated mechanism of gastric hypersecretion against the

background of hemodialysis, which creates preconditions for lesions of the gastroduodenal mucosa and the formation of ulcers. Important role in the formation of acute ulcers and erosions of the mucous membrane belongs to vascular lesions, and of trophic tissues disorders due to uremic intoxication. They result from the accumulation of toxic products and changed metabolic processes, which cause impairment of all organs, including stomach and duodenum.

All the above mentioned proves that *in patients with renal disorders, formation of the acute erosive ulcerous lesions of the mucous membrane of the gastroduodenal zone is due to endogenous intoxication. Acid-peptic factor and a dramatic suppression of the mucous membrane resistance play a certain role in this process.*

Rheumatoid arthritis. According to some authors, the frequency of gastroduodenal ulcers and erosions in patients with rheumatoid arthritis, treated with anti-inflammatory drugs, varies from 3 to 26%. Sun et al. (1974) found that 26% of patients with rheumatoid arthritis, treated with a combination of corticosteroids, metindol and butadion, had gastroduodenal ulcers. However, the use of only gold preparations practically completely prevented their occurrence. However, other researchers do not see any noticeable differences between the frequency of ulcers and the nature of the treatment. This fact was confirmed by Alwater et al. (1965), who revealed a more frequent occurrence of gastroduodenal ulcers (26%) in the severe course of rheumatoid arthritis, due to the effect of the underlying disease.

According to Gibberd (1966), in the early stages of rheumatoid arthritis, many patients develop dystrophic changes in the mucous membrane of the stomach. As the disease progresses, acute ulcers and erosion of the mucous membrane of the gastroduodenal zone may occur. The long-term intake of *ulcerogenic agents and especially their combinations* by such patients plays a significant role in their formation. It should be emphasized that the intensity of therapy is in direct relation to *the severity of the disease*. This is largely due to the complexity of evaluating the comparative value of a factor contributing to the erosive ulcer of the mucous membrane. Thus, the relationship between acute gastroduodenal ulcers and erosions and rheumatoid arthritis seems rather complicated, and a variety of different factors can contribute to the formation of ulcers in each patient.

Drug-induced ulcers. It has been established that not only the stressful effects, but *also the use of steroid hormones for therapeutic purposes*, may cause multiple hemorrhages, acute ulcers and erosion of the mucous membrane of the stomach. This issue is even more relevant due to the fact that glucocorticoids are widely used in some cases as the main therapeutic agent for various diseases.

The frequency of acute erosive ulcerative lesions of the gastric mucosa against the background of the intake of steroid hormones is of 0.2-5%. Most often they are located on a large curvature of the stomach and are multiple. Prednisone and prednisolone in doses greater than 40 mg per day have the most ulcerogenic activity, moreover, at prolonged use, steroid ulcers form the flattened wedge-like defects penetrating deep into the outer muscular or subserous layer.

Both the activity of the acid-peptic factor resulted from the use of steroids and inhibition of mucus formation, the reduction of regenerative processes, and the protective properties of the mucous membrane have a certain value in the pathogenesis of hormonal ulcers and erosion.

Clinical picture of ***steroid ulcers*** is quite diverse. Pain syndrome is observed in 42% of patients, asymptomatic course is registered in 25%, and other complications (bleeding, perforation, penetration) have developed in 3% of patients. The absence of pain in some patients can be explained by the initial severity of gastroduodenal hyperkinesia. The most frequent asymptomatic course is determined by the localization of ulcers in the stomach, which is largely due to the anti-inflammatory and analgesic effects of hormonal drugs. It has been established that the more severe the disease is, the more often are the complications of steroid ulcers, often with an unfavorable outcome. In case of a satisfactory general condition of the patient and no complications, as a rule, epithelization of acute ulcers and erosions is occurring rapidly.

In the reference literature there is information about the possibile transformation of acute steroid ulcers into chronic. Apparently, this is due to the ability of glucocorticoids to interfere the healing of acute ulcers and to the increase under their influence of the destruction of the walls of the previous chronic ulcer, which then returns to the original level.

In 1961, the first reports of gastric ulcer in patients taking large doses of ***aspirin or aspirin-containing drugs***, appeared.

The mechanism of the formation of acute ulcers and erosions of the gastric mucosa at the background of aspirin intake can be represented as follows. First of all, aspirin contributes to the direct destruction of the mucous barrier, resulting in reverse diffusion of hydrogen ions deep into the mucous membrane. Experimental studies by Sempl and Russel (1975) have shown that aspirin causes a decreased acidity and amount of secretion, which is associated not with inhibition of the activity of the overlaying cells, and with a sharp deteriorated mucosal permeability and the subsequently increased inverse diffusion of hydrogen ions in the interstitial space. Released with this, histamine changes the permeability of the capillaries, and conditions of acidic environment and in combination with alcohol are created. There are also suggestions about the ability of aspirin to suppress oxidative enzymes in the mucous membrane of the stomach, which significantly impairs the energy processes carried out in it. In addition, it is able to reduce the production of mucus, to increase the injury of epithelial cells, to cause bleeding, changing the structure and adhesive properties of platelets, increasing anticoagulant activity. In recent years, it has been shown that aspirin and similar drugs inhibit the synthesis of prostaglandins, which is associated with the negative effects of these agents on the gastrointestinal mucosa. The use of aspirin with synthetic prostaglandin E_1 or its synthetic analogue (Misoprostol) has been able to reduce the frequency of gastric bleeding from 80 to 20%.

Thus, the pathogenic effect of aspirin is variable. It manifests as dystrophic changes in the superficial epithelium with an increase in its permeability for hydrogen ions and inhibition of the mucus formation. In the case of bleeding, a certain role is played by the ability of the drug to lower the blood prothrombin, change the properties of the platelets, increase capillary permeability, and provide a cortisone-mimetic effect.

All salicylates have a negative effect on the gastrointestinal tract. Side effects caused by their intake can be divided into two groups. The first group involves a feeling of heaviness and pain in the epigastric area, nausea, heartburn, vomiting, decreased appetite, sometimes diarrhea. The second group includes impaired integrity of the mucous membrane: acute erosion and ulcers, exacerbation of chronic ulcers, gastrointestinal bleeding. Complications of the first type are more characteristic for all salicylic derivatives, and of the second type is precisely for acetylsalicylic acid.

Some pyrazolone derivatives may cause gastrointestinal complications. Butadion is the most important among them. Various complications are observed in 9-70% of patients treated with it. ***Butadion*** acute ulcers and erosions may develop in the first day of treatment or at the end of the course. They are localized, as a rule, in the stomach. In addition, butadion is able to provoke exacerbations of "old", chronic ulcers, which are usually located in the duodenal bulb and can result in a massive bleeding or perforation. It is believed that butadion is directly involved in the biochemical processes occurring in the gastric mucous membrane and impairs the synthesis of proteins in it. As a result, there are profound changes in trophic processes, which lead to ulcers. In addition, there is an opinion on the local irritating effect of the particles of this poorly soluble drug.

In a number of studies, it has been shown that erosion of the mucous membrane of the stomach also occurred after taking ***Indomethacin (Metindol).*** The drug has a more pronounced depressant effect on mucus production than aspirin. Mucus production was often disordered both in the fundus, and in the pyloric stomach, which contributed to the development of the destruction lesions in the whole membrane of the organ. Intake of Metindol causes the changes in the vascularization of the mucous membrane, associated with disordered microcirculation. On

average, the rate of development of acute ulcers of the gastric mucous membrane within the treatment with indomethacin is 2%. Their development is closely related to the duration of the drug intake and does not depend on its daily dose. However, the appeared pain and dyspeptic symptoms are more often observed at high doses of indomethacin. The development of this type of acute ulcers and erosion is favored by previous stomach diseases, and combined use of indomethacin with other potentially ulcerogenic substances (glucocorticoids, salicylates, Butadion), as well.

In conclusion, it should be mentioned that currently there are numerous drugs which are known to cause formation of acute erosive ulcer lesions of the gastrointestinal tract mucous membrane. In some cases, this is due to their direct damaging effects on the mucous membrane, but can often be due to direct or indirect stimulation of the secretion of hydrochloric acid and pepsin, inhibition of the gastric motility. In addition, the ulcerogenic action of some drugs is seen only against the background of pronounced degenerative changes in the gastroduodenal mucosa, which are a consequence of various diseases.

Diagnosis. Considering the fact that in most cases bleeding acute ulcers and erosion of the mucous membrane are located in the upper portions of the digestive tract, the main diagnostic method is an ***endoscopic examination***. With extremely high diagnostic information and accuracy, endoscopy can be used in extremely seriously affected patients, including those in the immediate postoperative period. As the morphological picture of acute erosive ulcerative lesions of the mucous membrane can change rapidly, many researchers emphasize the need for an earlier endoscopic study. Endoscopically acute ulcers, as a rule, are superficial, penetrating only to the submucosal base, small (up to 0.5-0.7 cm in diameter), round or oval. A crown of bright red color is determined around the ulcer. The inflammatory wall, characteristic for chronic ulcers, is absent. Touching with the instrument (endoscopic palpation) the edges of acute ulcer shows that they are mobile, the mucous membrane is elastic. Erosion is a superficial defect in the mucous membrane, round or oval in diameter up to 0.1-0.3 cm. The bottom can be clean, bright red or with a hemorrhagic coating, the edges are low or flattened. The mucous membrane around erosions is more often hyperemic, swollen, easily vulnerable.

Sometimes, an endoscopic study performed because of a massive gastroduodenal bleeding, finds neither acute ulcers nor erosion. However, the swollen mucous membrane is seen, from which, when pressed, the blood is oozing. In this regard, there always a question arises about the source of bleeding; whether the punctulated, invisible with naked eye erosions of mucous membrane are present, or considerably ruder pathology detected.

Pathogenesis of diffuse bleeding from the gastric mucosa has not been studied yet. They can both start quickly and stop quickly. In this case, in the *morphological structure of the mucous membrane of the stomach*, shortly after massive enough hemorrhages, *no changes are detected*. It can be thought that the appearance of such bleeding is associated with ***hemorrhagic diathesis*** due to various disorders of redox processes and manifests as a decreased activity of the coagulation system of blood, as well as an ***increased permeability*** of capillaries, small arteries of the mucous membrane and submucosal layer of the stomach. Based of clinical data, the cause and main mechanisms of this condition can not always be established.

According to B.S. Rozanov (1960), ***erosive hemorrhagic gastritis*** develops more often against the background of chronic stomach disease and is a manifestation of exacerbation of gastritis with ulceration. The multiple petechia of the mucous membrane is the source of bleeding in these cases. However, such a definition is valid for patients with history indicating gastritis or other diseases of the stomach. In this case, both the endoscopist and the pathologist find the substrate of the disease.

In some cases, the lesion of the mucous membrane of the stomach due to chemical influence, administration of certain drugs, pyloric stenosis and a significant enlargement of the stomach, septicemia, etc may be the cause of acute hemorrhagic gastritis. In most patients, a morphological study can determine the cause of diffuse bleeding.

However, there is also a third type of such bleeding, when not only visually, and also in the histological study, *neither petechia, nor erosion* revealed. In the history of these patients, there is no information about the diseases of the stomach and other possible causes of erosion formation. Bleeding with such functional gastritis, as a rule, is negligible and may occur among complete well-being. Along with this, sometimes very severe, profuse bleeding with a fatal outcome is observed. Sometimes, biochemical blood test can detect changes in one or another component of the coagulation system and vascular permeability. Apparently, these bleeding is based on ***hemorrhagic diathesis***. Gastric localization of bleeding *is associated with virtually imperceptible trophic changes in the mucous membrane against hypersecretion of hydrochloric acid*. According to Salmon et al. (1981), all erosion processes of the gastric mucosa have one genesis. At the initial stage, acute hemorrhagic gastritis may also occur with no apparent injury to the mucous membrane. The resulting bleeding is associated with impaired border arterioles of the stomach. Following the devloped trophic disorders, the erosions of the mucous membrane appear.

It should be mentioned that establishing the main disease is very conventional in many cases. This is explained by the fact that in many patients, except for one "main" disease, there are two, three, and sometimes four more others. In particular, heart disease is often combined with the lung disease, disorders of the liver, blood vessels, or vice versa. Therefore, in come combined diseases, preference is given to the one with the greatest degree of the functional disorder of the organ.

Plan of laboratory instrumental examination of an AU patient and possible changes.

1. Clinical blood test: possible anemia, decreased hemoglobin, erythrocytes.
2. Clinical urinalysis: may show no changes.
3. Determined by circulating blood deficiency (CBD) in bleeding (of varying degrees).
4. Biochemical analysis of blood (no identified changes possible).
5. Blood group and Rh affinity.
6. Coagulogram (no changes may be shown).
7. Plain X-ray of the chest (no changes may be shown).
8. Endoscopic examination of the stomach and duodenum – detection of AU (possibly several) or acute mucosal erosion.
9. Ultrasonography examination of the abdominal cavity (no changes may be shown).
10. ECG (in patients with cardiac comorbidity, appropriate changes may be found).

Differential diagnosis. If ulcer in history has characteristic symptoms, the diagnosis of acute ulcer is not complicated. However, acute and chronic ulcers, complicated and medication ulcers are distinguished, and differential diagnosis is complicated.

Treatment of a patient with uncomplicated acute ulcer is *mostly medical*. Nonsurgical treatment is aimed at:

- correction of hemodynamic disorders;
- decrease of the output of aggressive factors (hydrochloric acid, pepsin);
- increase of protective properties of the gastric mucous membrane.

The key role in the treatment of acute ulcers belongs to proton pump inhibitors (PPI). The indicative pattern of management for a patient with acute ulcer is as follows:

1) 3-7 days of Controloc intravenously at a dose of 40 mg every 12 hours;
2) then for 7 days, Controloc orally at a dose of 40 mg 2 times / day;
3) then for up to 4 weeks of Controloc, orally 40 mg 1 time daily (taking at 17-18 o'clock), De-Nol at a dose of 120 mg 4 times / day on average for 2 weeks.

Surgical treatment is carried out according to individual indications, *excision of acute ulcer combined with selective proximal vagotomy* is a surgical treatment of choice.

***Treatment of AU complicated by bleeding*. Medical treatment**. In spite of persistent long-term search, currently, treatment of bleeding acute ulcers and erosions of the mucous membrane of the digestive tract is a very difficult task. Among the methods of *local hemostasis, gastric lavage of cold physiological solution of sodium chloride*, 2% solution of sodium

bicarbonate, 5% solution of aminocaproic acid is a widespread method. This measure allows in many patients to achieve a temporary stop of bleeding, and often even to improve the conditions for endoscopic examination and use other methods of hemostasis. However, some researchers use irrigation of the stomach with warm salt solution. The warm fluid, in their opinion, increases the vascular reactivity and increases the coagulation properties of the blood while the icy solution gives the reverse effect. In addition, to stop the bleeding from acute ulcers and erosions, the administration of the *inhibitors of proteolytic enzymes (Contrykal, Trasylol, etc.), Adroxone, Dicynone, Thrombin, Ferricil, 0.5% solution of silver nitrate, and also 2-4 ml of Norepinephrine in 100-150 ml of isotonic sodium chloride solution in stomach through the probe is used.*

Endoscopic diathermy electrocoagulation, laser coagulation, film-forming agents, as well as injection of hemostatic drugs into the acute ulcer area are enough effective remedies for hemostasis, especially to stop bleeding acute ulcers.

Successful use of Pituitrin, Vasopressin, Somatostatin, as well as transcatheter embolization of the gastric arteries to stop bleeding from acute ulcers of the mucous membrane is reported in the literature. Pituitrin (0.5-1 ml), Dicynone (2 ml) and aminocapronic acid solution (5% 20 ml) are injected into the ventral trunk or arteries, in which the sources of bleeding are located in the vascularization zone. Somatostatin is administered intravenously at doses of 250 μg / hr and then at a rate of 3.5 μg / kg for 48-120 h. Vasopressin is used in a dose of 0.2 U / kg / h for 8 hours. In regard of the active participation of hydrochloric acid in the ulcer formation, it is considered appropriate to use antacids (Maalox, Aluminium hydroxide (Almagel), De-Nol, Sucralfate) internally at doses maintaining the pH of the gastric contents at a level not lower than 3.5-4.0. If a constant pH measurement is impossible, Almagel, in particular, is administered 1 tablespoon every hour. A good hemostatic effect is obtained from the use of H_2-histamine receptor blockers and Omeprazole.

Becker et al. (1979) reported for the first time about the successful use of the *secretin polypeptide* produced by the duodenal mucosa for treatment of bleeding acute ulcers and erosions. Suppressing acid production, secretin in doses of 0.25-1.5 U / kg body weight shows a 3-fold increased volume of pancreatic juice and 1-15-fold of the H_2CO_3 content in it. Already 30 minutes after the start of infusion of secretin, a virtually neutral pH in the stomach is achieved. According to the authors, the use of secretin in doses of 0.3 U / kg / h led to the stopping of bleeding from erosive ulcerative lesions of the mucous membrane in patients. In 44% of patients, recurrence of bleeding of moderate intensity was registered, in such cases positive results were achieved by repeated administration of secretin. V.P. Khokholya et al. (1982) recommend to use secretin as follows: 100U of the drug is diluted in 500 ml of Ringer's solution and slowly for 4 hours, infused dripping intravenously (25 U / hour). According to the authors, the effectiveness of secretin for hemostasis was 81%.

An *artificial hypotony* that is achieved *by a ganglionic blockade with the aid of Benzohexamethonium or Pentamine*, combined with intensive therapy of blood loss, is used successfully by a number of researchers to stop bleeding from acute ulcers and erosions. Administration of the mentioned drugs causes blood circulation decentralization, improves peripheral circulation, provides stable hypotension, slowing blood flow, secretory and motor rest of the stomach.

Provision of *adequate corrective therapy* (*remedial treatment) for homeostasis disorders caused by blood loss* is extremely important. However, it is necessary to take into account the initial state of the organism and background, which caused bleeding acute ulcers and erosion. Due to the fact that acute erosive ulcerative lesions of the mucous membrane are formed as a result of energy insufficiency, it is reasonable to inject hypertonic solutions of glucose and protein preparations into the stomach. A. A. Kurygin et al. (1990), to prevent the onset of acute ulcers recommended to injected into the stomach 100 ml of 40% glucose solution and twice a day 200 ml of Aminokrovin in the first 3-5 days after traumatic surgery. The authors found that it results in the pH increase, and the proteolytic activity in the body and the antral portion of the stomach is reduced. In the studied group of patients, in neither case, acute ulcers and erosions

were formed, while in the control group, this complication was observed at 21.5%. The diet in this category of patients should be thrifty and rich in energy, proteins, microelements and vitamins.

A number of authors mention the use of *hyperbaric oxygenation* in patients with bleeding acute ulcers and erosions, and hypoxia of the stomach wall and hypercapnia, which stimulates gastric secretion. According to Yu.V. Isakov (1991), this favors to the improvement of electrocardiographic and rheographic indices, the elimination of tissue oxygen failure.

The opinion on the promising use *of prostaglandins* for the treatment of bleeding acute ulcers of the mucous membrane is expressed. The experiment and the clinical course found that these substances, in particular, prostaglandin E_2, significantly (by 84-95%) reduces the secretion of hydrochloric acid, have cytoprotective effects and contribute to the healing of gastroduodenal ulcers. A number of clinical observations indicate the efficacy of prostaglandins in stopping bleeding from acute ulcers.

Summing up the described methods of medical treatment, it should be emphasized that such ***treatment must necessarily be comprehensive***. In order to stop the bleeding from acute ulcers and erosion of the mucous membrane, endoscopic methods of local hemostasis should be used, and in acute erosions, *selective intraarterial administration of vasopressors* should be performed. The latter is also effective in bleeding acute ulcers. Along with this, patients with acute ulcers of the mucous membrane in a complex of therapeutic measures should include *oral administration of local hemostatic drugs, Peritol, antacid and coating drugs, H_2-histamine receptor blockers or Omeprazole.* The use *of drugs that improve tissue respiration (cytochrome C) and antioxidants (vitamins E, C, etc.)* helps to reduce the level of endogenous intoxication and improves metabolic processes. *Secretin* can be used to suppress acid production and stop bleeding. *Artificial hypotension and hyperbaric oxygenation* are attributed to the auxiliary medications. Several authors recommend using *Methyluracil, Trichopol (Metronidazole), sodium nucleinate, anabolic hormones and protease inhibitors* to accelerate the healing of acute ulcers and erosions, which, due to local and central actions, increase reparative processes in the mucous membrane.

Surgical treatment. Most surgeons prefer medical treatment of bleeding acute ulcers and erosion and resort to surgery only in the case of ineffectiveness of medical therapy. However, some authors insist on more active tactics, considering that an emergency surgery is indicated during profuse bleeding (L.G. Anishchenko et al. 1964, L.V. Potashov et al., 1982). The issue of the choice of the time for performing surgical intervention has not been resolved completely. There are ideas according to which emergency surgical intervention is indicated in case of ineffective medical hemostatic therapy for 46 hours, in case of ineffective treatment with it for 5-6 days, in case of continued bleeding or relapsing bleeding after transfusion of 2500-3000 ml of blood.

According to the authors, emergency surgical intervention in patients with bleeding acute ulcer of the mucous membrane is indicated only in those cases when endoscopic hemostasis methods fail to stop the ongoing or relapsing bleeding. In hemorrhagic gastritis, the whole arsenal of complex hemostatic therapy should be used in connection with the extremely low effectiveness of surgical methods of treatment. It should be emphasized that the operation is an extremely serious additional trauma for the severe condition of a patient, due to the underlying disease and its complications, as well as the bleeding. In this regard, *in patients with acute erosions, and with acute ulcers of the mucous membrane, the advantage should be given to* ***medical treatment***. Due to this, the absolute majority of patients manage to avoid the need for emergency surgery, which significantly affects the patient's condition, and for some it is simply unbearable.

The need for a broad gastroduodenotomy to determine the source of bleeding is an important feature of surgical interventions for bleeding acute ulcers and erosions. This is due to the fact that fresh, with no inflammatory reaction, superficial ulcers, as a rule, are not defined

visually and by palpation. Failure to obey this rule should be considered a gross mistake that may be fatal to the patient.

Great difficulty arises *not only in determining the* ***indications for surgery****, but also in choosing the method of surgical intervention*. There are two pathogenetically determined approaches to operational tactics. The first involves the presence of common mechanisms for the formation of acute and chronic ulcers. It explains the relevance of the use of *vagotomy with drainage surgery or antrumectomy*, as well as for the removal of lesions of the stomach in the form of a *conventional or subtotal resection*, and in the case of ineffectiveness of the latter or major spread of lesions – to perform gastrectomy. Proponents of the second approach, considering the circulatory disorders as one of the most important factors in the pathogenesis of acute ulcers, perform ***interventions that influence on the local blood flow*** – *suturing of blood vessels, devascularization of the stomach.*

The 30-70% decrease in blood flow in the gastric mucosa after vagotomy was registered in the experiment, which allowed it to be used in the clinic to treat bleeding acute ulcers of the mucous membrane. A.A. Dotsenko et al. (1983) found that vagotomy, reducing acid formation, increases local hemostasis, contributing to stopping and preventing bleeding. *Currently, various types of vagotomy are used combined with pyloroplasty and retroclusion or removal of acute ulcers and erosions* (V.T. Zaitsev et al. 1984; A.A. Kurygin et al. 1990 and others). It should be mentioned that the implementation of vagotomy is more effective for prophylaxis than for the treatment of bleeding acute ulcers and erosions, since its use itself can contribute to the appearance of ulcers of the mucous membrane.

A number of researchers consider the pathogenetically grounded ***resection of the stomach***, in which the source of bleeding and a significant part of the mucous membrane, including the gastric production zone, is removed. Some surgeons use *subtotal resection of the stomach and even gastrectomy*. However, these interventions are extremely traumatic and very difficult to be tolerated by weakened patients with bleeding acute erosive ulcerative lesions of the digestive mucous membrane. One can not but agree with the opinion of Sullinvan et al. (1968) that gastrectomy, of course, allows reliably to stop the bleeding, but it is achieved too costly price for the patient. Both vagotomy and resection of the stomach are difficult to consider as radical operations, since they do not exclude the possibility of further ulceration in the stomach or its stump, as well as in other portions of the digestive tract. Thus, *relapse of bleeding* after the various variants of vagotomy is observed in 9.5-47.0% of patients, and after resection of the stomach this level is 20-44%.

Certain authors consider it reasonable to use the *minimal volume of surgical intervention, which consists of gastroduodenotomy, suturing of bleeding ulcers and devascularization of the stomach.* The number of relapses after this operation is of 9.0-37.5%. Devascularization of the stomach, as a rule, leads to decreased secretion of hydrochloric acid and significantly affects the motor function (while shortening of the periods of rest, prolongation of working periods, reduction of force of constrictions at their constant number). It was found that at the first 3-6 days after the ligation of vessels, the volume of the vascular bed of the stomach shows a 6-8 times decrease, with the largest changes observed in the mucous membrane of low curvature and body, less pronounced - in the pyloric portion. Despite the fact that after devascularization of the stomach there is a compensatory expansion of the remaining extra- and intraorganic vessels, the blood flow in the first time remains disordered. Arterial pressure is reduced by 4-69%, venous pressure shows a 5-8-fold reduction and reaches the baseline values only in 5-10 days. Virtually all of these changes are eliminated within 1-2 months after the operation, and sometimes a little later. Arterial ligation should be carried out in accordance with the localization of the source of bleeding, which is specified during endoscopic examination and surgical intervention after extensive gastroduodenotomy. In this case, acute ulcers or erosion should be fixed or cut.

Thus, the analysis of experimental studies and clinical observations suggests that partial and even subtotal devascularization of the stomach, as a rule, is not accompanied by life-

threatening consequences. However, due to a significant reduction in blood flow and acid production, these interventions can help stop bleeding and heal the acute ulcers of the mucous membrane.

Some surgeons perform only gastroduodenotomy and retroclusion of bleeding ulcers and erosions to stop bleeding. However, most researchers consider this intervention to be inadequate, which is confirmed by observations published by A. I. Gorbashko et al. (1977), Kunzman et al. (1970), Neidhardt et al. (1973) and others.

Thus, *the treatment of all patients with bleeding acute erosive ulcerative lesions of the mucous membrane of the digestive tract* ***should begin with medical (nonsurgical) methods***. Endoscopic methods of stopping and preventing relapse of bleeding were included in patients' comprehensive therapy: diathermocoagulation, laser coagulation combined with injection into the acute ulcer of hemostatic drugs and the application of film-forming substances onto it, only the application of film-forming substances. The given data prove the appropriateness and efficiency of application of complex medical treatment, which, besides the use of hemostatic and infusion corrective therapy, must necessarily include endoscopic methods of local hemostasis, anti-ulcer and vasoactive agents. ***Operative treatment is indicated in the case of ineffective medical therapy. In cases of profuse bleeding an emergency surgery is indicated.***

Treatment of the AU complicated by perforation, is similar to treatment of perforative ulcer in ulcer disease - ***urgent operation according to vital indications***; the method of choice is the *excision of acute perforated ulcers in combination with selective proximal vagotomy.*

According to many researchers, the problem of treating patients with acute ulcers and erosions is still far from the final solution. This is confirmed by the not in every case comforting results of the treatment of patients. Low effective in some cases, both medical and surgical treatment, requires further research in this area.

In order to prevent the formation of stress acute ulcers and erosions, especially after major and traumatic surgical interventions, it is recommended to use *antacids and enveloping drugs, H_2-histamine receptor blockers or Omeprazole in combination with protein hydrolysates, Peritol, Litonitum, or a combination of Metoclopramide, Methyluracil, Almagel and anabolic hormones.*

The key in the prevention of the development of acute ulcer is administration of proton pump inhibitors intravenously at 40 mg every 12 hours to stabilize the patient and transfer it to enteral nutrition.

The general measures aimed at preventing acute erosive ulcerative lesions of the digestive tract in surgical patients should include: thorough preparation of the patient for surgery; correction of disordered function of organs, systems of the organism and indices of homeostasis; adequate anesthesia during and after surgery; cost-effective surgical method; active post-operative management; prevention and active fighting against complications; aspiration of gastric contents; if no contraindications are found, then early enteral nutrition, including a through probe nutrition.

According to v.P. Khokholya (1998), a good effect in the prevention of acute ulcers and erosion of the digestive tract is got from the use of a *complex of medications*, which includes: Metoclopramide 2 ml intramuscular or intravenous before the operation, and then every 6-8 hours for 2-3 day; anabolic steroids (Retabolil, Nerobolil, etc.) 1-2 ml intramuscular one day before surgery; Methyluracil 0.5-1.0 g intake 3-4 times a day during or after meals a day before the operation and within one week after the operation; Almagel 1 tablespoon every 2 hours for 7-14 days after surgery.

To prevent the formation of acute ulcers of the mucous membrane in severe patients with concomitant pathology A.A. Khomichov (1992) recommends Litonit in a daily dose of 10 mg / kg after traumatic surgery. Its intake allows, in the author's opinion, to reduce the incidence of bleeding, improve the regeneration of the mucous membrane of the stomach and duodenum, stabilize the gastric acid production.

II.13. ACUTE SURGICAL COMPLICATIONS IN THE PATIENTS WITH INFECTIOUS DISEASES

Infectious diseases are quite common in the world, and their constant growth, including complicated forms, is observed. The mass migration of the population, which has been observed in recent decades contributes to the spread of infectious diseases.

This problem is particularly acute in relation to HIV / AIDS. Currently, there are more than 40 million people living with HIV / AIDS worldwide.

HIV** is a retrovirus that causes **human immunodeficiency. AIDS - Acquired Immunodeficiency Syndrome.

It takes about three months for the organism to produce antibodies, at the same time blood becomes HIV-positive, more precise to say, reactive. Nevertheless, blood already becomes infectious in 2 weeks. So, human blood can be very contagious, but not yet determined by tests - the so-called "window". Therefore, blood should always be considered conditionally HIV-positive.

The disease is serious enough, and those affected live with it for a long time. The number of infected people has been increasing. ***To prevent the infection** of medical personnel and HIV / AIDS patients in a medical facility, the following measures should be taken:* the use of masks with transparent plastic that protects the eyes, plastic apron, chainmail gloves (or two pairs of ordinary gloves), blunt atraumatic needles for closure of the abdomen; improve the surgical technique; as well, one of the most important conditions for preventive work is the correct organization of sterilization of surgical instruments and materials, the correct disposal of disposable syringes and medical waste with subsequent burning without dumping into household waste.

***Typhoid fever** is an acute infectious disease with a fecal-oral mechanism of transmission characterized by bacteremia, lesion of the lymphoid apparatus of the small intestine and has the course with fever, intoxication, and a typhoid maculopapular rash.*

The causative agent of the disease belongs to Salmonella group D - *Salmonella typhi.* The source of infection are patients with typhoid fever and those discharging bacteria. Typhoid pathogen enters the human body through the mouth, and then passes into the intestines and penetrates into the lymphatic formations of the small intestine - Peyer's patches and solitary follicles, which lead to the development of lymphadenitis and lymphangiitis. From the lymphatic formations the pathogen enters the bloodstream - bacteremia begins, that corresponds to the first clinical manifestations of the disease.

The clinical classification of typhoid fever *depends on:*

1) clinical forms - typical, atypical (abortive, obliterating);

2) the degree of severity - light, medium-grave, severe;

3) the nature of the course - cyclic, recurrent;

4) the presence of complications - uncomplicated, complicated (perforation of the intestine with the development of peritonitis, intestinal bleeding).

Any form of the disease may be accompanied by ***severe complications - perforation of the intestine with the development of fecal peritonitis.*** This complication develops more often at the weeks 2 or 3 of illness. The success of the treatment depends on the timing of the diagnosis, so it is necessary to know well the first signs of perforation and bleeding.

Intestinal perforation in 80% of patients with typhoid fever is associated with severe pain syndrome, but even minor abdominal pains should be a prerequisite for a thorough examination of the patient and consultation of the surgeon. Intestinal perforation is accompanied by worsening of the patient's condition, and even greater increase of the body temperature. Increasing flatulence, inhibition of peristalsis, and local muscular defense (in the intestinal perforation area) is a reason for a perforation to be suspected, that requires surgical intervention.

Intestinal bleeding complicates the course of typhoid fever, body temperature drops sharply, pallor of the skin and mucous membranes increases, tachycardia, decreased blood

pressure, bloating, and increased peristalsis. The appearance of liquid black stool or with fresh blood clots indicates a developed intestinal bleeding.

Key for ***specific diagnosis*** is *isolation of hemoculture*. Blood for bacterial inoculation is taken in all periods of the disease, 5-10 ml from the vein, and inoculate in 50-100 ml of bile broth or Rappoport's medium.

It must be remembered that a high body temperature in a patient during more than 5 days requires *a blood culturing*. The first blood culturing is desirable before the start of antibiotic therapy. *Serologic reactions* are used in the diagnosis - agglutination assay, indirect hemagglutination test, diagnostic titre - 1: 200 and above. To identify individuals with asymptomatic course of the disease, indirect hemagglutination test, with Vi antigen is prescribed.

Highly sensitive *methods of early diagnosis*, based on the *detection of antigens of the pathogen or antibodies to it* - enzyme immunoassay, CIEP – counterimmunoelectrophoresis, RIA (radioimmunoassay), etc. have been developed.

Treatment. Treatment of typhoid fever is *medical, if perforation developed – emergency operation for vital indications.*

Etiotropic drugs of choice include *fluoroquinolones* (ciprofloxacin 0.5 g 2 times a day) and *ceftriaxone* (rocephin 1-2 g / day intramuscular or intravenous). *The main etiotropic reserve drug* is ***Levomycetin***, which is administered orally in a dosage 0.5 g 4 times daily until the day 10 of normal temperature. Infusion therapy is used to eliminate intoxication.

In case of perforation of the intestine, ***an urgent surgical intervention*** *is performed - the closure of the perforation opening, sanation and drainage of the abdominal cavity*.

In case of intestinal bleeding, *absolute rest, starving, hemostatic therape and, in necessary cases, replacement therapy is indicated.*

Abdominal tuberculosis *is a specific disorder of the digestive system, peritoneum, mesenterial lymph nodes and retroperitoneal space.*

Among other localizations of tuberculosis, abdominal tuberculosis is a special and one of the most difficult chapters in phthisiology.

Abdominal tuberculosis most often (in 70% of patients) develops *secondary* as a result of lymphohematogenous infection with mycobacterium tuberculosis from other organs, primarily from the lungs. As a primary infection, it occurs through the alimentary spread of infection.

Clinical picture of abdominal tuberculosis is polymorphic, pathognomonic symptoms and clear diagnostic criteria are absent, therefore, as a rule, it goes being *masked with other diseases of the abdominal cavity* and is diagnosed only in a minority of the patients, while most remain undiagnosed. According to statistical data, the abdominal tuberculosis is revealed in only 2-3% cases in the structure of extrapulmonary tuberculosis. The frequency of tuberculosis affecting the organs of the abdominal cavity is different. More often (in 70% of patients) mesenteric lymph nodes are affected, less often it is peritoneum (12%). Isolated lesion of one organ is rare, often a specific process involves simultaneously several anatomical structures.

The most recognized ***classification of tuberculous mesadenitis***, proposed by V.G. Shtefko (1937), according to which the following types are distinguished: *caseous, fibroproductive and indurative forms of the disease.*

The tuberculous mesadenitis can have either *acute or chronic course*.

In the acute course there are abdominal pains of different localizations, but more often in the area of the navel and the right iliac region. The duration of the pain attack is from 2-3 hours to 2-3 days; the pain are so intensive that resemble a picture of an "acute abdomen". The abdomen is evenly blown, the anterior abdominal wall is involved in the act of breathing. Palpation of the abdomen is moderately tender, no muscular guarding of the anterior abdominal wall is found, the symptoms of irritated peritoneum are weakly positive.

It should be taken into account that enlarged lymph nodes can cause changes in the trophism of the appendix and cause the development of *secondary appendicitis*. Surface palpation of the abdomen is painless, when the deep palpation is done, the marked *tenderness in*

the navel area, especially in the Sternberg's points (on the left at the Ln level and on the right 1 cm above the McBurney's point).

Tuberculous mesadenitis*, McFanden's symptom* (pain in the navel region) and *Clinn's symptom* (abdominal pain shifts when the patient is moved to the left side) are characteristic. Sometimes, the palpation of the abdominal cavity may allow detection of conglomerates of enlarged lymph nodes as tumorous formations, moderately painful when palpated. Percussion over the conglomerate determines dull percussion sound. The examination of the patient should be performed on an empty stomach after preliminary emptying of the intestine (cleansing enema).

Tuberculosis of the intestine has long been considered as a severe complication of pulmonary tuberculosis. As Hippocrates said: "... tuberculosis patients die if diarrhea associates."

The most adequate systematization of ***intestinal tuberculosis is the classification developed by M.M. Alperin*** (1950), which distinguishes *asymptomatic, diarrheal, allergic, dyspeptic and general toxic forms of the disease.*

Pain at the intestinal tuberculosis is localized in the right iliac region and is characterized by persistency. They vary in intensity and duration, may appear independently or associated with meals and defecation. *Unstable stool* (up to 2-4 times a day) and *bloating* are the typical symptoms of intestinal tuberculosis. Pus, mucus and blood in feces are rare. Abdomen is evenly blown, soft at palpation, painful in the right iliac region, and often palpation causes a low-sounding borborygmi in the ileocecal region. Cecum can be bloated or thickened (compressed). Terminal loop of the ileum is palpated as a cord.

Tuberculous peritonitis. Clinical morphological classification of tuberculous peritonitis, which includes *papulose, exudative, adhesive, exudative adhesive and caseous ulcerous forms*, is generally recognized.

According to R. Seth et al. (1974), tuberculous peritonitis should be suspected in any patient with unclear abdominal symptoms. In its course it is a *chronic disease*, but it can be *acute.*

Acute course is characteristic for ***papulose tuberculous peritonitis***. The disease begins with a significant increase in body temperature, chills, abdominal pain, similar to acute infectious disease, typhoid fever, or paratyphoid, or it is taken as an "acute abdomen". Front abdominal wall is tense, positive symptoms of peritoneal irritation. Such patients are often performed an operative intervention, during which the tuberculous rashes are revealed on the peritoneum.

Exudative tuberculous peritonitis may result from papulous or paraspecific reactions to toxins of mycobacterium tuberculosis in any of its localization and is characterized by the presence of exudate in the abdominal cavity. The disease usually begins with the appearance of indeterminate, spasmodic abdominal pain, unstable stool, nausea and vomiting, subfebrile temperature. Against this background, the abdomen begins to grow, there is a feeling of fullness. As the fluid fills up, the abdomen can reach enormous proportions. At the same time, the navel is smoothed, then it protrudes, the abdominal skin becomes dry, enlarged venous network appears.

Adhesive tuberculous peritonitis is more often a consequence of exudative one and develops as a result of the organization of fibrin, but it may at first be plastic. At this form of peritonitis, the peritoneum is covered with fibrous layers in the form of ligaments and threads. These coverings at first are loose, then grown up with connective tissue, scarred, resulting in fusion with neighboring organs. Symptoms of adhesive peritonitis vary and depend on the severity of the adhesion process, cicatricial changes, disorders of bowel function. Patients complain of general weakness, abdominal pain of varying intensity, unclear localization, or localized, sometimes they are spasmodic. The patients' appetite decreases, vomiting, constipation or diarrhea often occur. The abdomen is enlarged in volume, but it can also be drawn-in *("armoured stomach"),* sometimes asymmetric. Front abdominal wall is often swollen. Palpation of the abdomen sometimes demonstrates a noise of peritoneal friction and soreness. The adhesive process can be limited to the formation of fibrinous overlays on the stomach

(*perigastritis*) or intestine (*pericolitis*). In the case of wrinkling of mesenterium, epiploon, adhesion of the intestinal loops between themselves and with other organs of the abdominal cavity, the tumor-like forms arise.

In case of ***exudative-adhesive form of tuberculous peritonitis*** the *fluctuating tumors - encapsulated exudate* is found in the abdominal cavity. Abdominal percussion reveals a *symptom of "chessboard "* (alternating timpanic and dull percussion sounds). The general condition of patients, despite such changes in the abdominal cavity, remains relatively satisfactory for a long time.

Caseous-ulcerative form of tuberculous peritonitis is characterized by the presence of areas of *clotted necrosis in the parietal and visceral peritoneum* among adhesions and intergrowth. Often, there is a tendency to *decay the caseous masses*. This is the most severe form of tuberculous peritonitis, more commonly seen in children, in which the sharply pronounces symptoms of tuberculous intoxication are seen. The general condition of patients is extremely grave. The body temperature rises to 39° C. Often there are profuse sweats. When intestinal obstruction occurs, paroxysmal pains, bloating appear. Patients are exhausted, facial features are sharpened. Skin is dry, sometimes swollen. Often anasarca is observed. Palpation of the abdomen reveals borborygmi in the intestine and crepitation, especially expressed in the navel, as well as tumor-like formations in different parts of the abdominal cavity.

Among the complications of abdominal tuberculosis, it is necessary to note *bleeding, formation of external and internal fistulas, intestinal perforation, fecal peritonitis, intestinal obstruction, amyloidosis of internal organs.*

Diagnosis. *X-ray changes* in case of intestinal tuberculosis are divided into *functional and morphological* and are detected both in oral and in rectal administration of contrast media. The most common *functional features* include segmental hyperperistalsis disease, local spasm, spastic filling deficiency (Shtirlin's symptom), early and late typhlospasm, retained barium suspension in the caecum or ileum, straightening and "hatching" of the small intestine loops, hyperperistalis with spastic contractions of the terminal branch of the ileum or ileocecal region, segmental expansion of intestinal loops.

Colonoscopy with a biopsy of the modified site of the mucous membrane is performed to clarify the changes in the lumen of the colon. Valuable information can be obtained with *laparoscopy* (papulous rash on the peritoneum, adhesive process).

The clinical picture of abdominal tuberculosis has no specific features, so it needs to be ***differentiated*** from some diseases of the abdominal cavity. *Acute forms of abdominal tuberculosis* should be distinguished from *acute appendicitis*, *acute non-specific mesodenitis, acute cholecystitis and pancreatitis, acute intestinal obstruction.* In this case, it is necessary to take into account the presence of signs of tuberculosis intoxication up to acute abdominal pain. *In a chronic course of abdominal tuberculosis, it should be differentiated from chronic gastritis, cholecystitis, pancreatitis, peptic ulcer, chronic non-specific mesadenitis, enterocolitis, chronic gynecological diseases, endometriosis, Crohn's disease, malignant tumors, systemic lupus erythematosus.*

Some difficulties arise in differential diagnosis ***with chronic nonspecific mesenteric adenitis.*** For diagnostic purposes, immunological studies are carried out: anti-TB immunity is studied in serological reactions and blast-transformation reaction with tuberculin; reaction of non-specific hemagglutination is also used. The diagnosis is specified on the operating table and histological examination of the material obtained during the operation.

When differentiating abdominal tuberculosis and ***chronic enterocolitis***, it is necessary to take into account that common symptoms occur only when the small intestine is affected and the exchange of vitamins, proteins, fats and carbohydrates is disordered. When the process progresses, the mineral exchange also suffers. The diagnosis is specified on the basis of the X-ray examination results, with the evidence of the detected swelling of the mucous membrane of the small and large intestine, while in cases of tuberculosis there is segmental lesion.

Treatment of abdominal tuberculosis is carried out in accordance with the general principles of treatment of tuberculosis. *The main method is* ***chemotherapy***. Concurrently with specific treatment, the pathogenetic and symptomatic therapy, a nutritious diet that includes the vitamin-rich foods, and excludes hard-to-digest foods are prescribed. In case of exudative tuberculous peritonitis, puncture of the abdominal cavity is indicated. *Complications of abdominal tuberculosis and persistent abdominal pain* are ***indications for surgical treatment (****laparotomy*). The material obtained during the operation is sent to a histological and bacteriological study.

Sanatorium and resort treatment for patients with abdominal tuberculosis is carried out mainly in local sanatoria.

Pseudotuberculosis (Far East scarlatiniform fever) *is an acute infectious disease characterized by polymorphic clinical manifestations with a predominant lesion of the gastrointestinal tract of the skin and of the musculoskeletal system*.

Clinical forms are determined on the basis of the predominant syndrome affected the organ. There are *abdominal, icteric, arthralgic, exanthemic, catarrhal, mixed, generalized, obliterated and latent forms*, and each of them may have symptoms of any other form, but they are not dominant. Each clinical form gives orientation in the approach to etiotropic and pathogenetic treatment, it allows to determine the complex of laboratory and functional examination.

Abdominal form occurs with predominance of gastrointestinal tract syndrome - abdominal pain, nausea, vomiting, diarrhea, signs of terminal ileitis, mesenteric adenitis, appendicitis.

Abdominal pains are localized in the ileocecal region, their intensity is different, and sometimes abdominal pain is a dominant symptom. Pain and borborygmi are observed in the ileocecal region at palpation of the abdomen. In addition, in some cases in the right iliac region, the percussion sound is shortened, tension in the anterior abdominal muscles and the symptoms of peritoneal irritation are observed. This symptom is due to the development of mesenteric adenitis, terminal ileitis or appendicitis.

Icteric form shows pain in the right hypochondrium, darkening of the urine, jaundice of the skin and sclera, enlargement of the liver, bilirubinemia, hypertransaminasemia. Often, patients complain of heaviness and pain in the right hypochondrium. The enlarged painful liver, icteric skin and sclera are determined, darkening of the urine, urobilinemia, hyperbilirubinemia, increased activity of transferase are observed. The spleen increases in 10-18% of patients.

Clinical diagnosis is based on a combination of signs characteristic of pseudotuberculosis: acute onset of the disease, syndrome of general intoxication, fever, skin lesions (exanthema, symptoms of "hood", "gloves", "socks"), catarrhal inflammation of the oropharyngeal mucosa, signs of gastrointestinal disorder, syndrome of hepatitis, impaired joints, allergic symptoms, changes in blood and urine.

Laboratory methods - *bacteriological and serological* methods have a decisive role in the final diagnosis. Pathogen may be isolated from the patient in the acute period of the disease, possibly in faeces, urine, oropharyngeal smears of mucous membrane, removed appendices and mesenteric lymph nodes. At present, modern methods *such as immunoblotting and polymerase chain reaction (PCR*) have been widely used for the diagnosis of pseudotuberculosis.

In case if a clinical picture of *acute appendicitis develops as a surgical complication of pseudotuberculosis*, the patient is *indicated urgent surgical treatment - appendectomy*. *In case of the development of hepatitis - medical treatment with detoxification therapy, hepatoprotectors, etc.*

Yersiniosis (intestinal yersiniosis) *is an acute infectious disease characterized by a predominant lesion of the gastrointestinal tract with a tendency to a generalized lesion of various organs and systems.*

The causative agent of yersiniosis belongs to the family of intestinal bacteria *(Enterobacteriaceae Yersinia* species *Yersinia enterocolitica*).

Clinical classification of yersiniosis has not been developed. According to the major syndrome, it is possible to distinguish several ***clinical forms***, in particular *localized* (*gastroenterocolitical*), the clinical picture of which is limited to the predominant lesion of the gastrointestinal tract and generalized (*icteric, exanthematous, arthralgic, septic*) forms.

Gastroenterocolitic form. Occurs more often than others. Its share accounts for about 70% of cases. An acute onset of disease is observed, the body temperature rises to 38-39° C. Headache, malaise, insomnia, anorexia, chills are characteristic. Abdominal pain, diarrhea, and sometimes vomiting appear simultaneously with the intoxication syndrome. The sool is liquid with strong unpleasant odor, sometimes with an admixture of mucus, blood. The frequency of the bowels varies from 2 to 15 times per day. Severe course is rare. Most often body temperature is subfebrile or normal, the syndrome of general intoxication is mild, the stool is 2-3 times a day, abdominal pain is slight. Such patients are actively diagnosed in cases of group illnesses. The course of this form can be seen as enteritis, enterocolitis and gastroenterocolitis. This form of yersiniosis lasts from 2 days to 2 weeks.

The icteric form develops either simultaneously with gastroenterocolitic, or 2-3 days after intestinal dysfunction. In this form, the symptoms of liver lesions appear on the foreground, *toxic hepatitis* develops. The patients complain of the heaviness and pain in the right hypochondrium, sometimes itchy skin. Jaundice of the skin and sclera develops. The liver is enlarged, it is painful when palpated. There is darkening of urine, discoloration of feces. Hyperbilirubinemia and hypertransaminasemia are registered.

Complications in intestinal yersiniosis occur more often at the week 2-3. They include allergic *exanthema* (*urticaria, nodular erythema), Quincke's edema, arthritis (*mainly large joints*), myocarditis, urethritis, conjunctivitis, appendicitis.*

For laboratory confirmation of the diagnosis, cultures are performed on the nutritional media of blood, feces, liquor, inflamed mesenteric lymph nodes and appendicular processes. The following *serological methods* are used: agglutination reaction, indirect hemagglutination reaction with erythrocytic diagnosticums, latex agglutination, and immunoenzyme assay.

Differential diagnosis is performed with acute gastrointestinal infectious diseases (acute dysentery, escherichiosis, salmonellosis, pseudotuberculosis, viral hepatitis, scarlet fever, rubella, toxic allergic erythema). Yersiniosis can be clinically diagnosed on the basis of acute onset, intoxication, fever, symptoms of acute gastroenterocolitis in combination with exanthema, jaundice, arthralgia, and epidemiological conditions.

Treatment** of yersiniosis is medical* and includes a diet, infusion-transfusion, antibiotic therapy, etc. When *acute appendicitis occurs as a complication of yersiniosis,* ***urgent surgical treatment - appendectomy *is indicated.*

Opisthorchiasis *– is a helminthosis, affecting mainly the hepatobiliary system and the pancreas, characterized by prolonged course, with frequent exacerbations leading to the onset of primary liver and pancreatic cancer.*

The causative agents of opisthorchiasis are two types of trematodes of the family *Opisthorchidae: Opisthorchis felineus* and *Opisthorchis viverrini*. *O. felineus* (synonyms: catliver fluke, Siberian liver fluke). Opisthorchiasis is a naturally occurring disease. Opisthorchiasis caused by Opisthorchis felineus is often found in the population of the Ob and Irtysh basins (Western Siberia, Kazakhstan), Kama (Perm region), the Dnieper (some regions of Ukraine), registered in the basins of the Volga, Don, Donets, and Nothern Dvina, Neman. Infection of human and animal mammals occurs in case of eating raw, improperly roasted and slightly salted fish with helminthic metacercarias. Ingested with fish, Opistorchis larvae in the human intestine come out from their surrounding envelopes and in the common bile and pancreatic ducts penetrate the liver, gall bladder and pancreas, where in 2 weeks they reach puberty, and begin to lay eggs in a month.

The main role in the pathogenesis of opisthorchiasis is played by: allergic reactions (especially pronounced in the early phase of the disease) that arise from the released products of helminth metabolism; the mechanical effect of helminths, which consists of the injury of the

walls of the biliary and pancreatic ducts and the gall bladder with bothria and spinelets that cover the surface of the helminth body. The accumulation of parasites causes a slowdown the flow of bile and secretion of the pancreas; nervous-reflexory effects due to stimulation of the nervous elements of the ducts by helminths, resulting in pathological nerve impulses that are transmitted primarily to the stomach and duodenum; occurrence of conditions (dyskinesia of the biliary tract, accumulation of parasites, eggs, exfoliated epithelial cells, temporary and complete cessation of the flow of bile), favorable for the secondary infection of the biliary tract; glandular proliferation of the epithelium of the bile and pancreatic ducts, which should be considered as precancerous.

Incubation period for opisthorchiasis lasts 2-4 weeks. In the early phase of opisthorchiasis fever may develop, pain in the muscles and joints, vomiting, diarrhea, pain and enlargement of the liver, sometimes enlarged spleen, allergic skin rashes, leukocytosis with eosinophilia in the blood, often a leukemoid eosinophilic reaction are observed. In the late phase of opisthorchiasis, the main complaints of patients are pain in the epigastrium and right hypochondrium; in many cases they irradiate in the back and sometimes in the left hypochondrium. Often pain is exacerbated by attacks of biliary colic. Disorders of the motor function of the gallbladder in opisthorchiasis can occur in the form of hyperkinetic, hypertonic or hypokinetic dyskinesia. Three quarters of patients with radiologically proven disorders of the gallbladder motility have a hypokinetic type of dyskinesia. For such patients, dull bursting pain in the right hypochondrium, expressed dyspepsia, constipation (*cystic failure syndrome*) are characteristic. In patients with hypertonic and hyperkinetic types of dyskinesia, the syndrome of *biliary (acalculous) colic is more common,* ***gall bladder is not enlarged****.*

It is difficult to diagnose opisthorchiasis according to the clinical picture of the disease due to the absence of specific symptoms and syndromes, characteristic only for this disease. It is not difficult to recognize opisthorchic invasion in a month after infection, when helminths begin to lay eggs (ovoscopic examination of feces and duodenal juice of the patient). Great difficulties are encountered in recognizing the early phase of opisthorchiasis. The presence of fever, hepatomegaly, eosinophilic leukocytosis in new residents in an intensive focus of opisthorchiasis makes it suspect the early phase of this helminthosis. Eggs of catliver fluke in those invaded are more often detected in duodenal intubation than in feces. In cases of mild invasion, they are sometimes found only during repeated intubations.

The ***complications of opisthorchiasis*** include *purulent cholangitis, perforation of cystoid enlarged bile ducts followed by biliary peritonitis, acute pancreatitis, primary liver cancer.*

In cases of the development of complicated opisthorchiasis, i.e. ***cholangitis and biliary peritonitis***, the patients undergo an *open operative* ***treatment,*** according to the indications: *cholecystectomy, debridement and drainage of biliary ducts and abdominal cavity.*

Amoebiasis *is a disease caused by the pathogenic strains of Entamoeba histolytica; it is widespread in the world, mainly in tropical and subtropical countries.*

This infection is ranked second (after malaria) in the world as for the mortality associated with parasitic diseases. About 480 million people in the world are carriers of *E. histolytica*, 48 million of them develop colitis and extraintestinal abscesses, and in 40-100 thousand patients the fatal outcome is registered.

Pathological changes and clinical manifestations of invasive amoebiasis vary widely from colitis with mild clinical manifestations to fulminant colitis and amoebic abscess in the liver. ***Amoebic colitis and amoebic abscess of the liver*** *are the most common clinical manifestations of invasive amoebiasis****,*** and the amoebic colitis is found 5-50 times more often than the amoebic abscess of the liver.

Liver abscess and fulminant colitis are the main causes of the mortality due to amoebiasis.

The complications of intestinal amoebiasis include: *perforation of the intestine*, more often in the area of the cecum, less often in the rectosigmoid region, that can *cause peritonitis and abscess of the abdominal cavity; amoebic appendicitis; massive intestinal bleeding* due to

dissolution of major artery by an ulcer*; amoeboma*, that is a tumor-like lesion in the wall of the large intestine, mainly in the ascending colon, cecum and rectum. The *strictures* are usually isolated and located in the area of the cecum or sigmoid colon, sometimes contributing to the development of constipation and partial intestinal obstruction.

In patients with ***amoebic liver abscess***, the past history of intestinal amoebiasis is revealed only in 30-40% of cases, and amoebae in feces are found in no more than 20% of patients. The amoebic abscess of the liver is more common in adults than in children and in males more often than in females. Single or multiple abscesses are formed more often in the right lobe of the liver. The abscess consists of three zones: *central zone of necrosis* containing liquid necrotic masses with an admixture of blood, usually sterile (2-3% of cases of secondary bacterial infection*); the middle zone* consisting of stroma, and *the outer zone* containing amoebic trophozoites and fibrin.

The clinical course of the amoebic abscess of the liver is characterized by a fever with chills and excessive sweating at night, hepatomegaly and pain in the region of the liver projection, a moderate leukocytosis. In case of large abscesses, jaundice may develop, which is a poor predictor. If diaphragm is involved in the process, elevated diaphagm, restricted diaphragmal mobility, possible development of atelectasis are seen. Relatively often (in 10-20% of cases, there is a prolonged latent or atypical course of the abscess (for example, only fever, pseudocholecystitis, jaundice) with a possible subsequent perforation, which can cause peritonitis and affect the chest organs.

The simplest and most reliable method for diagnosing ***intestinal amoebiasis*** is the *microscopic examination of feces* to detect vegetative forms (trophozoites) and cysts. If clinical findings indicate a possible bowel affection, *recto- or colonoscopy* is recommended. Recto- and colonoscopy is targeted at getting a biopsy from the affected areas of the intestine in order to detect amoebae and for differential diagnosis, in particular, with carcinoma. These methods can detect the presence of ulcers in the intestine, amoeboma, strictures and other pathological lesions. *Focal, not diffuse type of lesions is a characteristic feature of changes in amoebiasis.*

Diagnosis of extrahepatic amoebiasis, in particular ***liver abscess***, is carried out by *ultrasonography and computed tomography*, which allow to determine the localization, size and number of abscesses, and also to control the results of treatment. X-ray examination reveals elevated diaphragm, effusion in the pleural cavity, abscesses in the lungs. If necessary, an aspiration the contents of the abscess is done. Amoebae are rarely located in the center of necrotic masses, they are usually found in the outer walls of the abscess.

To diagnose amoebiasis, *serologic tests* may be used to detect specific antibodies.

Treatment. In general, all drugs used for the treatment of amoebiasis can be divided into 2 groups: "contact" or "compartment" (affect the intestinal compatrment forms), and systemic tissue amoebocides.

The heterogeneity of the pathological process and clinical manifestations of amoebiasis in different geographical regions, the presence of strains resistant to standard chemotherapy regimens with 5-nitroimidazoles require varying treatment patterns based on the experience gained in a particular area. After successful chemotherapy of the liver abscess, residual cavities usually disappear within 2-4 months, however, persistence of cavities up to 1 year is possible. In severe patients with ***amoebic dysentery***, due to possible *perforation of the intestine and the development of peritonitis*, additional antibacterial drugs that are active against the intestinal microflora should be prescribed. *Aspiration (or percutaneous drainage*) is recommended for large abscess sizes (greater than 6 cm), localization of the abscess in the left lobe of the liver, or high in the right lobe of the liver, severe abdominal pain and abdominal wall tension because of the potential threat of abscess burst, and in ineffective chemotherapy within 48 hours from its beginning. Aspiration is also recommended for abscesses of unclear etiology. In the absence of closed drainage, burst of the abscess and the development of peritonitis, an open surgical treatment is performed.

Ascariasis is a helminthosis, which has been known since ancient times in the population of countries with moderate, warm and hot climate, provided with all-year sufficient humidity. Ascariasis is the most frequent helminthosis, common across the globe. In countries with a dry climate it is rare, no cases were registered beyond the Arctic Circle. *The causative agent of ascariasis is a common roundworm Ascaris lumbricoides*. Infection occurs in case when mature eggs are swallowed. Epidemiologically significant are mainly vegetables, on the surface of which are particles of soil. Currently, there is a great danger for the spread of ascariasis in the gardens, where sometimes non-disinfested human feces are used for fertilizing the soil. From mature eggs that a person swallowed, larvae come out in the small intestine, which penetrate into the intestinal wall and then pass into the blood capillaries, then hematogenously migrate into *the liver and lungs*. In addition to the intestines, liver and lungs, the Ascaris larvae were found in the brain, the eye, and other organs. They are intensely fed with blood serum and erythrocytes. In the lungs, the larva is actively released into the alveoli and bronchioles, moving through the small and large bronchi with the help of ciliated epithelium to the oropharynx, where swallowing of sputum with larvae occurs. Getting into the intestine, the larva reaches puberty for 70-75 days. The life span of the adult acaridium reaches the year, after which it dies and along with the feces it is excreted out. Severe manifestations occur in case of *penetration of ascaris into the liver, pancreas and other organs*. Adult helminths can injure the intestinal wall with their sharp ends, and clusters of ascaris sometimes cause mechanical obstruction. Irritation of nerve endings, the toxic effect on them of life-threatening worms sometimes causes spastic bowel obstruction. Migration of ascarides to other organs create conditions for the secondary bacterial infection followed with the *purulent complications (abscesses, cholangitis, pancreatitis, etc.).* A feature is mentioned in cases of reinfection that such pathologoanatomical changes are much less pronounced than at the primary infection, which may indicate a peculiar immunity with ascariasis. Immunity to reinsvasion lasts for several months. Antibodies to ascarid proteins can be identified 5-10 days after infection, in 3 months they are usually not detected.

Clinical manifestations of ascariasis depend on the localization of parasites and the intensity of the invasion. In the clinical course of ascariasis there are *two phases - early (migratory) and late (intestinal).* The first phase coincides with the period of migration of larvae, while the second is due to the parasitizing of helminths in the intestine and possible complications.

Complications of ascariasis. Frequent complications of ascariasis is *obstruction of the intestine,* due to the closure of the lumen of the intestine with a mass of ascarides, or due to a disordered neuromuscular regulation of the intestinal tone. Palpation of the abdomen in patients with such complications can reveal round tumour of pasty consistency that is a mass of ascarides, which can be localized in any portion of the intestine. In some cases, in the thin abdominal wall, you can palpate the bodies of individual helminths in the lumen of the intestine.

Penetration of helminths into the bile ducts and gall bladder is the severe complication of ascariasis. In these cases, there are severe pains that are not stopped even by narcotic analgesics. Against the background of these attacks, vomiting often occurs, and helminths are sometimes isolated with vomiting masses. In cases of cholangiohepatitis and mechanical obstruction of the common bile duct with ascarides there is *jaundice.*

The temperature during the development of complications may be septic, with shocking chills. It results in secondary bacterial infection, *purulent cholangitis and multiple abscesses of the liver* often occur, which in turn can be complicated *by peritonitis, purulent pleurisy, sepsis, abscesses in the abdominal cavity.*

Penetration of ascarides into the pancreatic ducts causes *acute pancreatitis*. Reaching to the appendix causes appendicitis or appendicular colic with no inflammatory manifestations.

Diagnosis of ascariasis in the *migration stage* is based on the identifying eosinophilic infiltrates, considering clinical radiological, hematological and immunological findings. The *radiological picture* of these infiltrates can simulate tuberculosis, pneumonia, and tumor of the lung. The main difference of infiltrates in ascariasis is their rapid disappearance without any

residual effects. Similar infiltrates can be found in other helminthiasis - ancylostomiasis and strongyloidiasis.

Reliable diagnosis of ascariasis in *the first phase* is based on the *detection of ascaris larvae in sputum and completing immunological reactions to detect specific antibodies in the blood of patients.*

In the *intestinal stage* of the disease, the main method is *copro-ovoscopic method for ascarid eggs*. If eggs are found in duodenal contents, this may indicate parasites in biliary and pancreatic ducts. However, sometimes in the intestine there are parasites of the same sex, in such case they can be detected radiologically. After the contrast entered, the mass of ascarides in the form of strips of X-ray translucency 0.4-0.6 cm wide appear on the screen.

*In case if the **surgical complications of ascariasis** develop (intestinal obstruction, cholangitis, mechanical jaundice), patients undergo **surgical treatment**: obstruction repairing, debridement and drainage of the biliary duct system.*

CHAPTER III. ACUTE SURGICAL PATHOLOGY OF RESPIRATORY ORGANS AND HEART

III.1. MODERN INVESTIGATION METHODS FOR ACUTE SURGICAL DISEASES OF THE RESPIRATORY AND CARDIAC ORGANS

Non-invasive methods for diagnosis of the respiratory system disorders

X-ray methods

Among non-invasive methods of examination of the patients with pulmonary disorders, pride of place goes to **X-ray examination.**

Roentgenoscopy should be considered an essential requirement of X-ray examination of the patients with pulmonary disorders.

First of all, X-ray diagnostics includes rough examination of the lung fields, while doing that region and extent of damage are evaluated. Attention is focused on the instability of the cupula of diaphragm, condition of the rib-diaphragmatic sinuses, clear lung fields, structural properties of the roots of the lungs, size and configuration of the medial shadow, which is formed by mediastinal organs.

Further X-ray examination should deepen and extend understanding of the nature of changes in the lungs and thoracic cavity. These studies are made in the *frontal (antero posterior view) and lateral views*, adding, if necessary, the *images of oblique view and enlargement X-ray films*. An important advantage of radiography is objectivity and reliability of the results obtained, possible comparison to the data of the previously performed X-ray examination, that is, the *possibility of a case follow-up*. The latter is often one of the most important factors for correct diagnosis.

X-ray examination in two views (Fig. 61) allows to determine with maximum accuracy the localization and spread of lung injuries, changes in the mediastinum, condition of interlobar fissures, assessment and comparison of the transparency level of lung portions in its various divisions, small focal lung lesions, what is especially valuable during differential diagnosis of pulmonary tuberculosis, sarcoidosis, pneumoconiosis and other diseases that produce similar radiological changes.

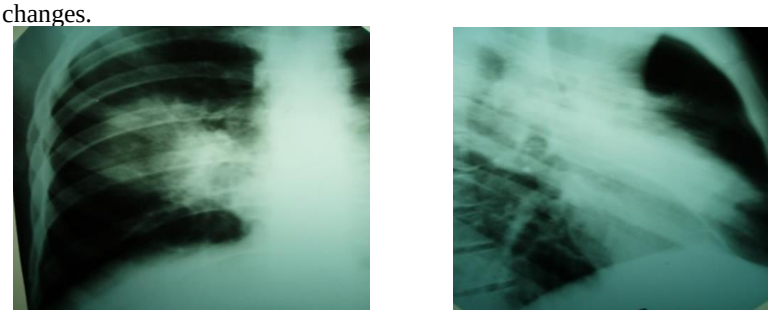


Fig. 61. X-ray image in frontal and right lateral views. Inflammatory syndrome of the middle part

The so-called *overexposed* images of the thoracic cavity are used to improve visualization of the tracheobronchial tree and indurated lymph nodes of the mediastinum. They extra clearly show the elements of the lung root on the background of the pulmonary pattern as if it was put on the "back line".

Some clinics began to use **electroroentgenography** in order to obtain the first, most general information on the nature of radiological changes in the lungs and chest cavity (along with fluorography). The very important advantage of the method is its high economic efficiency, no expenses for equipment of special photolabs and reagents necessary for X-ray studies, the process is independent of water supply, recording of large-format images is made in the light. The image is reproduced on the selenium plate and then put onto a paper.

Electroroentgenography has undeniable advantages during urgent X-ray diagnosing.

The tomographic (layer-by-layer) examination of the chest cavity is also performed in frontal and lateral views. The number of tomograms, performed in case of different pathological changes in the organs of the chest cavity, depends on the tasks in hand and is determined for each patient individually. If necessary, the tomograms made in frontal views can be completed with tomograms in oblique views. The best scans are the ones in the range of 8 to 12 cm, going from the spinal processes of the vertebrae. Tomographic study allows us to clarify the data obtained from the plain radiography: exact localization and nature of pathological process in the lungs, its distribution, relation to the vessels and bronchi of the lungs, pleura and mediastinal organs are determined. With the help of tomography, it is possible to assess permeability of the trachea and bronchi, to detect neoplasms, a foreign body in the lumen of the respiratory tract. Tomography is an important supportive practice to study the nature of changes in the lymph nodes of the mediastinum, in the bony formations that form a framework of the chest cavity.

The potential of X-ray diagnostics has considerably increased due to the formation and clinical use of **computed sectional roentgenography**. If compared with conventional X-ray, it has dozens of times higher resolution and allows to distinguish the tissues with a difference in density up to 0.5%. This makes it possible to define clearly the border between normal and pathologically changed pulmonary tissue. On computed tomograms of the chest organs it is possible not only to localize a pathological nidus, but also to determine its size, estimate the density and homogeneity, relation between adjacent anatomical formations and organs.

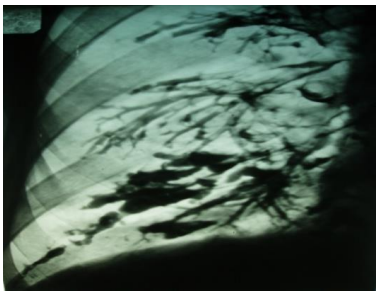


Fig. 62. Bronchography. Cystic bronchiectases of the lower part of the right lung

X-ray contrast imaging study methods of the lungs. Bronchography (Fig. 62). The main indications for the administration of this method are currently the following: suspicion of the pathology of the trachea and bronchi, bronchiectases, other chronic inflammatory diseases of the lung, tuberculosis, some forms of pneumosclerosis.

The method of bronchography. For successful bronchography special preliminary training of patients, especially those that produce significant amounts of sputum, is required, because in this case it is not possible to have good filling of the bronchi.

For the disimpaction of tracheobronchial tree from pathological content the drugs (expectorants, mucolytics, spasmolytics) and special methods – steam inhalations, inhalations with mucolytics, enzymes, endotracheal infusions, therapeutic bronchoscopy are used.

The study begins only in case of confirmation of full-value and effectiveness of the initiated preparation. For the study contrast medium iodolipol is used. In most cases, the study is conducted under local anesthesia. For this purpose, solutions of Dicain, Trimecainum, novocaine are used. For the introduction of a contrast medium, standard steerable catheters of Rozenstrauh-Smulevich are used.

After anesthesia of the upper respiratory tract, anesthesia of the bronchial tree of the examined side in various positions of a patient is performed: on the back, on the abdomen, side-lying with a head down. Later, depending on the tasks performed, the bronchial tree of an entire lung, its lobe or a segment are examined. Bronchial filling is performed under control of the X-ray television equipment. X-ray pictures are made in frontal, lateral and oblique views in the patient's upright position or lying on the lateroscope.

After completion of the examination, the bronchial tree is cleaned from the contrast medium, washed with a warm antiseptic solution with the help of an electric suction machine, which is connected to the catheter introduced into the bronchial lumen. During next 24 hours a patient undergoes steam inhalation. On next day control roentgenoscopy is advisable. It allows to assess the level of clearance of the bronchial tree from the contrast medium, and, what is equally important, may show changes which have not been detected during examination (residual cavities, cysts, in which the contrast medium is left).

Lung bronchography can be performed under anesthesia. Its specific features are that the patient lays on the back, artificial ventilation of the lungs is carried out in conditions of myorelaxation. Contrast substance is injected through a tracheal tube and X-ray pictures of both lungs in frontal and oblique views are made in the period of short apnea.

Aerosol bronchography is also performed with **tantalum powder as a contrast medium**. Tantalum, covering the mucous membrane of the trachea and bronchi, allows to identify very precisely the details of its structure – folds, glandular apparatus. Contrast substance in the form of fine powder is introduced into the lumen of the respiratory tract with the help of a special device – nebulizer. Good tolerability of the procedure by patients, high informational value of the received results are observed. Aerosol bronchography is especially good in the cases when it is necessary to accurately assess the pattern of the mucous membrane of the tracheobronchial tree, to diagnose stenosis of the trachea and large bronchi, especially in patients with low functional reserves, including those who have undergone lung surgeries.

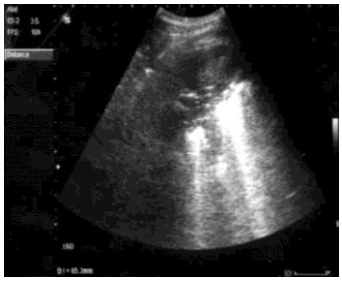


Fig. 63. Ultrasound in the left pleural cavity. Adhesion process in the left pleural sinus

Ultrasound examination of the pleural cavity

The first report in the USSR on the use of ultrasound for the diagnosis of lung and pleural pathology was published in 1964 by M.D. Gurevich and D.I. Tsurupa. The authors noted that with the help of ultrasound the thickness of induration can be accurately determined, the fluid in the pleural cavity can be recognized, the size of a localized empyema can be evaluated. Ultrasound is even more significant, according to the authors, for the diagnosis of postoperative pleural complications.

All diseases of the thoracic cavity can be grouped into four ultrasound syndromes, united on the ground of one principally basic echo-symptom for each of them (Shakhov B.E., Safonov DV., 2002). The names of the syndromes correspond to the main ultrasound signs:

- *syndrome of pleural effusion;*
- *syndrome of the parietal formation;*
- *syndrome of airless pulmonary tissue;*
- *syndrome of a changed parietal hyperechoic line.*

Most often, an ultrasound examination of the pleural cavity is performed in case of pleural effusion syndrome.

During echo scanning of ***hemithorax*** on the side of the pathological process, it is possible to prognose the type of pleural effusion according to the echo signal data.

1. *Sero-hemorrhagic effusions* usually have a homogeneous echostructure with low echogenicity.

2. *Free pleural effusion* (until the development of fibrin formation) forms an echogenic zone, which is clearly visible on the scanner screen in the form of anechoic strips of different size and length.

3. The diverse structure of the exudate is characteristic for a *sero-fibrinous effusion*, what is manifested during ultrasound scan in the form of hyperechoic signals of an islet nature.

4. Echo structure of the *purulent and serous-purulent effusion* is always complex due to multiple echo signals disseminated around the area of effusion.

Ultrasound examination of the pleural cavity allows to diagnose effusion successfully both in large (200 ml and more) and in small quantities. The minimum amount of fluid found during ultrasound examinations of the pleural cavity – 10 ml.

Positive aspects of ultrasound examination of the pleural cavity should include the advances of this method in *differential diagnosis between the adhesive process and effusion in the pleural cavity*. In the presence of the adhesion process in the pleural cavity (Fig. 63), linear echostructures of high echogenicity are clearly visualized. It is also possible to detect a fluid encapsulated among adhesions with ultrasound. The formation of the adhesion process in the pleural cavity, in some cases, allows to avoid unreasonable diagnostic punctures.

In case of the development of *encapsulation* in patients examined with ultrasound it is possible to clearly visualize its borders with inhomogenous internal echostructure, caused by purulent content, fibrin, detritus. The effusion in case of empyema produces internal echo signals, which "float" moving in sync with breathing and when the position of a patient's body is changed. Thickened pleura sheets are well contoured for a significant length.

In some cases, adhesions do not cover the pleural cavity completely, but seem to hang down in its lumen, shifting in sync with breathing. They represent fibrin mesh that can be quite dense.

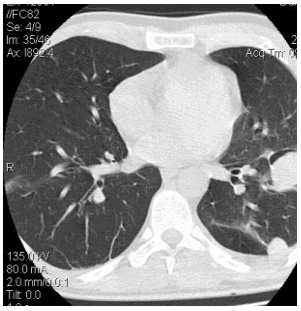


Fig. 64. Computed tomography of the chest organs. Cyst of the left lung (cavity with fluid)

The mentioned ultrasound data are especially important in the puncture method of therapy of multichamber empyema. The positive aspects of ultrasound investigation should include the definition of the distance between the chest wall and the fluid in the pleural cavity.

Ultrasound examination of the pleural cavity is a highly informative diagnostic method in the complex of pleural effusions diagnosis. Its administration is effective:

- to determine the borders, structure, localization of encapsulation;
- to determine the location of the puncture of the pleural cavity;
- to make differential diagnosis between the adhesion process and free fluid in the pleural cavity.

Concurrent administration of X-ray and ultrasound examination of the pleural cavity increases the effectiveness of diagnostic measures and reduces radiation exposure on the patients.

Computed tomography of the chest organs.

Computed tomography (Fig. 64) gives an opportunity to obtain the most complete X-ray data about tumors and cysts of the mediastinum, vascular changes in the lungs, aneurysms, the state of the tracheobronchial tree, nature and structure of "spherical formations in the lungs", pathological changes in the pleura, thoracic wall, especially in case of its malignant lesions.

Invasive methods of diagnosis of the respiratory system diseases

Bronchoscopy

The endoscopic examination of the tracheobronchial tree is given a very significant value, since with its help most patients receive reliable information on the nature of changes in the tracheobronchial tree, what is especially important in the diagnosis of tumor diseases of the trachea and bronchi.

The diagnostic capacities of bronchoscopy grew with the development and creation of new models of bronchoscopes with *rigid construction*. Brüning's and Mezrin's bronchoscopes gradually gave place to the more convenient and safe model of *Fridel's bronchoscope.*

The latter provides adequate ventilation of the lungs during bronchoscopy. The bronchoscopes of such design (so-called "respiratory") have been widely used in bronchial, pulmonary and surgical practice up to the present day.

The use of optical devices combined with reliable, stable ventilation of the lungs, completion of respiratory bronchoscopes with the sources of "cold" light largely defined wide use of rigid models of bronchoscopes. Undeniable advantage of respiratory bronchoscopes is the possibility of performance not only diagnostic but also some *therapeutic manipulations on bronchi* with their help – removal of foreign bodies and small benign tumors with the help of a set of forceps, treatment of mucous membranes with medical preparations, including treatment of bronchial fistulas. Respiratory bronchoscopes of rigid construction allow to perform a puncture transbronchial biopsy of bifurcation and other groups of intrapulmonary lymph nodes with a long needle. With the help of respiratory bronchoscopes, *therapeutic obstructions of the bronchial tree* are performed in case of suppurative destructive lesions of the lungs, pneumothorax, and *the drainage of the bronchi* is also performed.

However, diagnostic capabilities of rigid models of bronchoscopes are limited to the main, lobar and initial divisions of segmental bronchi, that is, centrally located parts of the respiratory tract. It is caused by the stiffness of the construction and its relatively large size. Optical devices allow to view only initial parts of the segmental bronchi and exclude the possibility of biopsy. In addition, an increased traumatism of the examination of the tracheobronchial tree with the help of rigid models of bronchoscopes demands anesthesia during their use.

Further development of bronchoscopic diagnosis was manifested in connection with the creation and use of fibre-optic endoscopes – *fibrobronchoscopes.*

Among the most important advantages of fibrobronchoscopes there are high elasticity of the device, small size of the diameter of the diagnostic tube (up to 6 mm), reliable system of a powerful "cold light" flow. The diagnostic channel of a fibrobronchoscope and a set of biopsy forceps and scarifiers allow to obtain material for morphologic examination not only from the tracheobronchial tree divisions, accessible for visual control, but also from more distal ones. With the aid of fibre-optic models of the fibrobronchoscope, a detailed survey of the bronchial tree and obtaining of material for morphological study including the segmental bronchi bacame possible, and for individual divisions (lower, middle, partly upper segments) – the subsegmental bronchi.

The diagnostic value of fibrobronchoscopy increases significantly by taking the material for *morphologica*l (histological and cytological) *examination* as an addition to the visual examination: *forceps and scarification biopsy, targeted aspiration of lavage* from the regions of supposed lung injury.

The design of modern videobronchoscopes allows to do wide-ranging examinations of the tracheobronchial tree with administration of local anesthesia.

The appearance of new terminal anesthetics with a broad spectrum of therapeutic action and low toxicity (Lidocaine, Trimecainum) also contributes to bronchial examinations. They replaced previously administered for these purposes drugs such as Dicain, which often caused severe general disorders in the examined patients.

Local anesthesia by inhalation of *aerosol with lidocaine solution* is important for the use of a bronchoscope. Inhalation aerosol anesthesia is convenient to use with an aerosol inhaler, which provides creation of an aerosol solution flow. After anesthesia, lidocaine solution is distributed on the surface of the mucous membranes of the trachea and bronchi. As a rule, to the moment of anesthesia offset there appears a feeling of "discomfort when swallowing" and a peculiar sensation of "the relief of inhale depth."

Despite the advantages of bronchoscopy under local anesthesia, it is not possible to refuse anesthesia completely during this study. In many patients only under anesthesia, in conditions of complete relaxation, the availability of visual diagnosis of changes in the tracheobronchial tree is provided. Among such patients there are persons with unstable, emotionally labile frame of mind, children, as well as the patients with signs of allergy to drugs used for local anesthesia. In addition, bronchial examination should be performed under anesthesia in the cases when the diagnosis is complicated: an examination lasts for a long time or it is necessary to have a shared examination and discussion of the revealed changes by several specialists.

Diagnosis of changes in the tracheobronchial tree, definition and evaluation of the signs of the disease development represent rather important task and require the use of special methodical techniques. This is more important because the information obtained during the examination of the tracheobronchial tree determines much the entire course of a subsequent bronchological examination, and sometimes – a therapeutic approach as well. Therefore, during examination via fibrobronchoscope a single system should be followed, which provides the most complete data on the condition and changes in the tracheobronchial tree.

The condition of all tracheobronchial tree divisions available for viewing is compulsory for evaluation. Particular attention is paid to the region of the changes, registered during an X-ray examination.

Technique. After insertion of the bronchoscope tube into the larynx, the vocal cords are carefully examined. At the same time, the nature and degree of their movement are evaluated: limitation of movement (paresis) of the vocal fold may indicate involvement in the pathological process of the recurrent laryngeal nerve.

In the trachea, its walls are examined in details (frontal, posterior, lateral) along the entire length – from the subglottic region to the bifurcation – the place of its division into the right and left main bronchi.

The information received at this stage of the bronchoscopy can be somewhat extended by means of scheduled visit, which allows to evaluate the movement of different parts of the trachea and bronchi (walls, rugae). If during investigation by rigid models of bronchoscopes shifting of walls and rugae of the trachea and bronchi with the endoscope tubes is carried out without much effort, then it is difficult to perform this by the elastic endoscopes with fiber optics.

After completion of the examination of the trachea, we proceed to the study of the carina of its bifurcation, the primary portions of the main bronchi. In this case, along with the assessment of the condition of the mucous membrane of bifurcation, special attention is paid to its configuration. Thus, flattening or "expansion" of the carina of the trachea bifurcation, its laterodeviation, limitation or lack of motion often indicate significant changes in the adjacent groups of lymph nodes of the mediastinum: their enlargement, formation of dense non-mobile conglomerates. All these allow to estimate the spread of pathological changes in the lungs, mediastinal organs.

After examination of the trachea bifurcation we should proceed to the examination of bronchi. It is procedurally correct to start it with the study of the lumen of lung respiratory passages, which does not have pathological changes. This approach allows to develop general understanding of the condition of the mucous membrane of the lumen of the respiratory tract in one particular patient (chronic bronchitis, atrophic bronchitis, etc.). Then, knowing these "background" changes, during examination of the bronchial mucous membrane of the affected lung, it is easier to detect even small changes caused by the disease (signs of inflammation, surface transformation).

Within the borders of the lungs, an examination of the main bronchi, lobar bronchi which branch from it and further – segmental, subsegmental and smaller ones, is made by a single system, with the obligatory registration of the condition of the rugae in the areas of their division, as well as the respiratory movements of the bronchi, the degree of contractile ability of their ostia. An examination of the bronchial tree within each lung is carried out down from the top: from proximal bronchi to the basal pyramid bronchi.

In case of clusters of the pathological content in the lumen of the respiratory tract, which disrupts an examination, aggravates data interpretation, it is removed by means of a suction apparatus. If necessary, the bronchial tree is washed with a physiological solution of sodium chloride or a solution of antiseptic (Furacilin).

Already only on the basis of a bronchovideoscopy examination (Fig. 65), there is an opportunity to get an accurate picture on the nature of pathological changes in the lungs in more than 60% of patients. Thus, in case of a tumor in the bronchus lumen, "plus-tissue" is determined as nodular, fungiform, papillomatous, granulomatous, polypoid mass; in the presence of infiltrates of the bronchus wall, a flat or nodular with a smooth or rough surface protrusion, as well as a narrowing of the bronchus lumen from either side or of a concentric nature, can be observed. As a rule, the mucous membrane is also distorted in the region of changes: it is loose, easily bleeding, breathing motionless. Documentation of the results of the examination with fibrobronchoscopy allows to make pictures of the detected changes in the tracheobronchial tree.

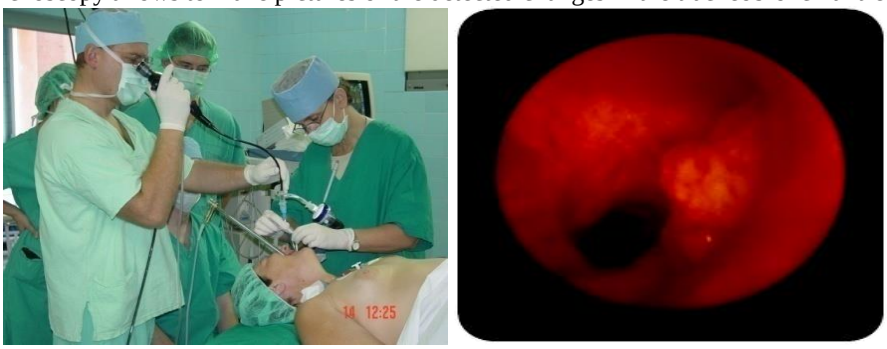


Fig. 65. Bronchoscopic examination:1 - bronchoscopy;
2 - tumor formation of the right main bronchus (endophoto)

Along with a detailed examination and evaluation of pathological changes in the tracheobronchial tree, an endoscopic examination of the patients with lung disorders provides ***sampling for the morphological verification of a disease***. In most patients, it allows to complete successfully etiological and topical diagnosis of the disease. Particularly important are the results of morphological diagnosis at suspicion on the lung cancer. Used in the initial period of a patient's examination, they make it possible to establish a correct diagnosis and determine indications for the most reasonable treatment in the short term.

The great practical value of the results of morphological studies of the material, obtained during bronchoscopy, determined the development and improvement of special devices to take it. They include sets of biopsy forceps, curettes, scarificator brushes, needles for transbronchial biopsy, various aspirators.

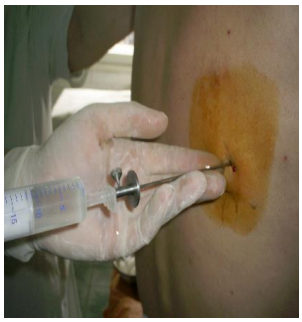


Fig. 66. Puncture of the right pleural cavity

Diagnostic puncture of the pleural cavity

Puncture of the pleural cavity (Fig. 66) is one of the most widely used diagnostic methods for the diseases of the lungs and pleura. The information obtained during it, often allows us to evaluate the nature of pathological changes and determines further therapeutic tactics. In addition, the removal of the exudate or air from the pleural cavity often enables further examination with the use of more complex techniques and methods, both due to the improvement of the condition of patients, and as a result of the restoration of normal anatomical interrelate

ion of the chest cavity organs, spread of the collapsed or atelectated lung.

Indications for a puncture are defined, when physical and x-ray methods show accumulation of fluid or gas in the pleural cavity. Depending on the nature of these findings, the location and tasks of the pleural puncture are specified.

In case of the accumulation of fluid in the pleural cavity, the puncture is performed in the patient's sitting position with a support stand under the arm from the healthy side of the trunk. The place for the puncture is marked from the back, in the lower chest regions, most often – along the seventh intercostal space between the midaxillary and scapular lines. *The classical point for the puncture of the pleural cavity in the presence of fluid – the seventh-eighth intercostal space along posterior axillary line on the upper edge of the lower rib*.

At the point marked for the puncture, infiltration anesthesia of the tissues of the chest wall isperformed. A mandatory requirement for the pleural puncture is prevention of the cavity's connection with the external environment – air should not enter the pleural cavity.

The type and character of fluid in the cavity, its quantity often solve many issues of diagnosis (pus, chylous fluid, blood, serous exudate) already in the process of puncture. However, special study of the liquid is of fundamental importance. Its density, biochemical and cytological composition, nature of the microflora and its sensitivity to antibiotics are determined. Part of the liquid is recommended to leave in the test tube for one day for macroscopic evaluation of the residual matter.

In the presence of air in the pleural cavity, the puncture is performed *anteriorly in the second intercostal space along the mid-clavicular line*. Use of the pneumothorax apparatus for this purpose allows not only to remove air from the pleural cavity and determine its amount, but also to clarify the character of the connection between the pleural cavity and airways. This is often a key factor in the solution of issues of further therapeutic tactics. For this purpose, *pleuromanometry* is administered, which makes it possible to determine the degree and stability of negative pressure in the pleural cavity at various stages of air evacuation.

In case of clusters of air and fluids in the pleural cavity pleural punctures from several points are often required. The choice of the location of the puncture is determined by the area of accumulation of fluid or gas, diagnosed with X-ray examination. It is recommended to complete the pleural cavity with a control X-ray examination, which allows to estimate the effectiveness of the punctures, and may also provide additional information on the nature of changes in the lungs or pleural cavity, which was previously difficult to obtain (on the background of exudate or air).

Diagnostic pneumomediastinum

Pneumomediastinography is used to specify the location of the formations, which adhere to the organs of the mediastinum in X-ray pictures and tomographic images, merging with them. In addition, this method can be used to identify different neoplasms of the mediastinum more clearly, to define their relations with the organs located here. By pushing the mediastinal pleura from the mediastinum, it is possible to clarify the location of neoplasms in relation to the lung in the cases where the symphysis in the pleural cavity does not allow pneumothorax to be placed.

For application of *frontal pneumodiastinum* a patient lies on the back with a small bolster under the shoulder blades and his head pulled back. In the sternal notch area local anesthesia of the skin and subcutaneous tissue is made. A thin needle is introduced with anesthetic solution behind the sternum. Making sure that the end of the needle is in the mediastinal tissue, and not in the vessel, the pneumothorax apparatus is attached to its pavilion and slowly, dispensing for 7-10 minutes, 500-750 ml of air or oxygen are introduced. The patient is put in a position on the back with elevated feet, and then – on the healthy side, what is necessary for more targeted gas distribution within the cellular spaces. After that an X-ray examination is performed.

For placement of the *posterior pneumomediastinum* presacral access is used, from where the gas through the fat of the retroperitoneal area reaches the back regions of the mediastinum.

For this purpose, in the face-down position of a patient below the apex of the coccyx local infiltration anesthesia is performed. A long, thin needle, supplying the solution of anesthetic, is introduced under the coccyx and then upwards to the frontal surface of the sacrum. After checking that the end of the needle is located in the presacral tissue, and not in the rectum,

with the help of pneumothorax device up to 1500-2000 ml of air (not oxygen!) is gradually introduced. After completion of this stage of the examination, a patient keeps plantigrade or semisitting position for an hour. Periodically with an X-ray method the movement of gas in the ascending direction to the posterior mediastinum is defined. After gas reaching the cellular spaces of the posterior mediastinum, an X-ray examination is performed.

Pleurography and fistulography

Pleurography is a method of study, which helps to detect, precisely localize, determine the size and configuration of the residual pleural cavity in case of empyema, rigidity of the lung, etc. (Fig. 67).

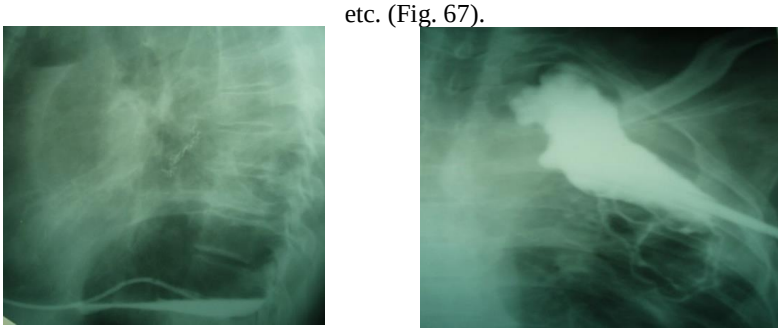


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Fig. 67. Pleurography – encapsulated pleural empyema

The fistulography *is performed to determine the direction and length of the fistulous passages, definition of the supporting them causes (foreign body, rib osteomyelitis), as well as for definition of the shape and size of the residual cavity during its combination with the lumen of the bronchial tree.*

As a contrast agent, water-soluble substances are used – Verografinum, Urotrast, and others.

Into the pleural cavity, the contrast substance is introduced by puncture at the point previously marked according to X-ray pictures or to radioscopy, and in the case of a pleuropulmonary fistula – through a catheter or needle with a club shaped end inserted into the fistulous passage. After filling the cavity, the outer hole of the fistula is closed by a strip of adhesive plaster.

X-ray examination is performed standing (in two projections) and lying on a lateroscope.

Pleurography and fistulography are currently the most common methods which make possible to identify, localize and measure the size of the final pleural cavity. It should be kept in mind that the size of the cavities, as a rule, exceeds their contrast images in X-ray pictures.

Angiopulmonography

Angiopulmonography *is a method of a contrast study of the vessels of the pulmonary circulation.*

More often, indications for angiopulmonography are defined as differential diagnostic ones: in case of congenital abnormalities of the lungs, purulent diseases, blastomatous process. It also provides an opportunity to determine the functional state of parenchyma of the lung, to evaluate hemodynamics.

There are four methods of angiopulmonography:

1) general, performed by intravenous introduction of contrast media or by angiocardiography from the right ventricle of the heart;

2) selective, performed from the trunk or branches of the pulmonary artery;

3) superselective, which includes contrast enhancement from the lobar, segmental branches of the pulmonary artery;

4) occlusal, which is performed when the subsegmental or lobular branch of the pulmonary artery becomes wedged by the end part of the cardiac catheter or during blockage of the main branch of the pulmonary artery by the catheter bag.

Angiopulmonography is performed in X-ray operation rooms, which provide video X-ray and physiological control, equipped with automatic injectors for rapid, remote and synchronous with radiography introduction of the contrast media (Urotrast, Verografinum, etc.) into the blood vessels and the heart. The cardiac catheter is introduced into the blood vessels usually by a percutaneous, intrarfemoral way.

It is recommended to perform **bronchial arteriography** (Fig.68) in combination with catheterization of the pulmonary artery and study of pressure indicators, oximetry in the places above and below the found anastomoses of two systems of blood circulation of the lungs.

During the **contrast study of mediastinal vessels**, the signs of affected lymph nodes located here in case of lung cancer can be found.

Contrast study of mediastinal vessels includes *phlebography* and *aortography*. Phlebography is performed taking into account the character of the pathological process, which is detected by X-ray examination, tomography, bronchography. If there is a possible suspected disease of the lymph nodes of the anterior mediastinum – *upper cavography* and contrast study of the internal thoracic veins are performed, and at suspicion on the disease of lymph nodes of the posterior mediastinum – *azygography.*

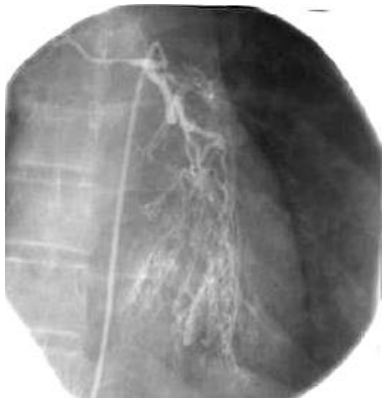

Fig. 68. Bronchial arteriogram of a patient with bronchiectasis (hypervascularisation of pulmonary tissue with significant arterial plexus, aneurysmatic dilation of the segmental branches of the bronchial arteries (arrow))

During analysis of angiopulmonograms, attention is paid to the phasal movement of the contrast agent in different regions of the lung: pulmonary artery, capillary bed, venous system of the pulmonary circulation.

By the combination of angiographic signs it is possible to characterize the appearance and extension of the pathological process (in case of two-projection seriography – at the segment level) in the lung, with attention paid to the presence of vascular deformation, displacement, contours, dilation of the vascular pattern (rarefication), degree of development of the vessels of certain segments, parts, the lung, the character of the location of the chambers of the heart, heart rotation and others.

Comprehensive assessment of a number of indicators (pressure in the right chambers of the heart and the pulmonary artery system, oxygen saturation of the arterial and mixed venous blood, as well as in different regions of the lesser circulation, etc.) allows to determine reserve capacities of a pulmonary circulation and to plan the treatment of pulmonologic patients efficiently. In combination with the quantitative assessment of the regional pulmonary blood flow, the definition of pulmonary volume of blood significantly expands and deepens the idea about reserve capacities of the body.

Diagnostic surgical interventions

Transtoracic needle biopsy of the pleura. The indication for the puncture biopsy of the pleura is most often the assumption of tuberculosis or blastomatous disease of the pleura.

At the point marked according to the X-ray pictures and tomograms, infiltration anesthesia of the chest wall is performed, after which with the help of a specially designed for this purpose Abrams needle (M. Abrams), which has an outer diameter of 4 mm, a soft-tissue cylinder of the chest wall with parietal pleura is cut by a circulatory movement. The received material is sent for histological study.

The fundamental disadvantage of this method is that in fact, it is performed blindly. Therefore, in case of the focal nature of the disease characteristic for pulmonary pleurae, during etiological diagnosis only a positive result of the study should be taken into account.

Transthoracic puncture biopsy of the lungs. An indication for puncture biopsy is the development of the so-called "spherical formations" in the peripheral portions of the lung, the morphological structure of which is unclear, and thoracotomy is associated with increased risk.

The biopsy is performed in an X-ray room under control of the electrooptic image converter display. The shortest distance from the skin surface to the formation in the lung is determined, and infiltration anesthesia of the tissues of the chest wall is performed at the assigned point. The Silverman needle or its various modifications are used for the puncture. For this purpose, a trocar needle with a tip in the form of a cutter is also applied. Under control of an X-ray screen, a needle with a trocar is introduced into the formation in the lung. After the correct position of the needle end has been confirmed, the trocar is pulled out, and the needle is slowly pushed forward, being rotating clockwise. The cutting milling end excises a part of the formation. Then a small vacuum, which holds biopsy material in the lumen of the needle, is created with a syringe, and the needle is pulled out. The obtained material is subject to morphological study.

The most common ***complications of lung transthoracic biopsy*** include: *pneumothorax, small-volume hemoptysis*; in case of an infected examined formation (formation of abscess in it) it is possible to transfer infection via the needle route with the development of the *phlegmon of the thoracic wall*. The emergence of *implantation metastases* during the puncture of a malignant tumor is among very rare complications.

Thoracoscopy. This diagnostic technique is important in the examination of patients for pulmonary tuberculosis, spontaneous pneumothorax. In addition, with the help of thoracoscopy, it is possible to define the localization and character of broncho-pleural fistulas in case of chronic pleural empyema, as well as examination of the parietal and visceral pleura, at suspicion on their affection by tumor with direct sampling for morphological study.

The study is performed in the dressing or operating room. Before thoracoscopy pneumothorax is placed on the side of the injury. The patient is laid on a healthy side with a hand put behind his head. The most convenient place to introduce the device is the area of intersection of the third-fifth intercostal space with the middle axillary line, but depending on the study objectives and the nature of X-ray changes, it can be altered respectively. In case of a formed pleurodermal fistula, the endoscope is introduced into the pleural cavity via this channel. The skin after anesthesia is punctured with an acute scalpel, and through other layers of the chest wall we permeate into the pleural cavity by a trocar with a mandrin from the thoracoscope set. After removal of the mandrin through the trocar, the thoracoscopy is performed. Turning the thoracoscope around the axis and changing the angle of its inclination, an examination of the lungs, visceral and parietal pleura is performed. With the help of biopsy forceps directly under visual control, the material for morphological study is taken.

Fibrobronchoscope can be used as a thoracoscope, the small outer diameter of the first, systems of reliable and intensive light and control, as well as devices for biopsy, increase potentialities of the method.

Upon completion of the examination, the endoscope and the trocar are removed, sutures are applied to the chest wall. Air is removed from the pleural cavity. Among possible complications of thoracoscopy the formation of reactive effusion is sometimes observed, the treatment of which is performed by punctures.

Abscessoscopy. The fibre-optic endoscope can be used to assess the condition of the lung abscess cavity, for examination of a draining bronchus, dynamic control of detersion and repair of the purulent cavity.

To perform the study, an endoscope is introduced into the abscess cavity through the formed fistulous passage or by thoracocentesis. Before putting a trocar, it is necessary to be sure that the lung in the region of abscess localization adheres to the parietal pleura. After that, the puncture of the abscess cavity is performed. The trocar with a mandrin is inserted through the wall of the thoracic cavity along the way of the needle. After the mandrin removal purulent contents are collected with the help of a rubber catheter and an electric suction machine, and the

cavity is washed out. An endoscope is inserted through the trocar. The cavity is examined, the material for morphological and microbiological study are taken.

Biopsy of supraclavicular lymph nodes. The indication for this diagnostic method is most often the evaluation of the spread of a lung tumor or other organs of the chest cavity. Certainly, the biopsy of the supraclavicular lymph nodes is important for differential diagnosis of the diseases in which there are formerly similar changes in the lymphatic apparatus (lymphogranulomatosis, tuberculosis).

Prescalenic biopsy is used to find out the condition of the lower cervical lymph nodes. In this case, under local or general anesthesia the fiber with the lymph nodes located in it of the other fatty tissue neck area is removed. This anatomical region is limited by the internal jugular and subclavian veins in the place of their merging into the brachiocephalic venous trunk and inferior belly of the omohyoid muscle. The lymph nodes located here (lower deep jugular ones) are excised and submitted to histological examination.

A possible variant of morphological assessment of the condition of supraclavicular lymph nodes is ***puncture biopsy***. This less traumatic technique is used mainly for the study of altered lymph nodes – enlarged, clearly palpated.

Mediastinoscopy. Mediastinoscopy is intended to solve the issues on the condition of the lymph nodes of the mediastinum (paratracheal, tracheobronchial, pretracheal, bifurcation) by means of their visual examination, instrumental palpation and *puncture or direct biopsy*. Thus, the main task of mediastinoscopy is evaluation of the nature and degree of the blastomatous process.

The technique. The examination is performed under anesthesia. In the dorsal position of a patient, with a bolster, put under his shoulder blades, and the head drawn back over the suprasternal notch of the sternum, transverse skin resection is made, fascial formations are dissected. Bluntly, with the help of a finger and instruments, the fiber behind the sternum is dissected and in the anterior mediastinum a channel for endoscope (mediastinoscope) introduction is formed. Anatomical structures of the anterior mediastinum, the groups of regional lymph nodes located here are examined. With the help of a needle, biopsy forceps or grasping forceps the changed or suspicious tissues and lymph nodes struck by the pathological process are taken for morphological study. The study is complete with hemostasis, wound suturing.

Mediastinoscopy can be accompanied by a number of ***complications,*** such *as severe bleeding, paresis of the recurrent nerve, damage of the esophagus.* There have been cases of *mediastinitis, pneumothorax*.

However, it should be kept in mind that mediastinoscopy is not able to provide a complete picture of the condition of the mediastinum along its whole length. Examination of its dorsal departments, lymph nodes in the region of the "aortic window" is complicated. It is not always possible to determine the relation of a primary tumor to the vessels of the lung root, pericardium.

Parasternal (frontal) mediastinotomy. Some dissatisfaction with the potentials and results of the examination of the mediastinum with the help of mediastinoscopy is the basis for the development and recommendations for diagnostic parasternal (frontal) mediastinotomy.

Parasternal mediastinotomy provides better access to the lymph nodes of the mediastinum, makes it possible to study the cellular space in front of the superior vena cava and arc of the aorta, "aortic window" and the root of the lung, more free visual control, extended palpation examination of the mediastinum, and sampling of several lymph nodes from different parts of regional basins for the study.

The study is performed in the operating room. In most observations, when in case of mediastinotomy radical operation is advised, immediately after its completion thoracotomy should be started.

Technique. Under anesthesia, in the dorsal position of a patient, with a bolster under the shoulders and the head, on the right or on the left from the edge of the sternum, in parallel and retreating from it by 1 cm outside, a vertical incision of the skin between the Ist-IIIrd ribs is

performed. The cartilage of the IInd rib is reached layer-by-layer, which is subperichondrially subject to the resection of 2.5-3 cm in length. If deep examination of the lower edge of the root of the lung and around periesophageal groups of the lymph nodes is necessary, the cartilage of the IIIrd rib is additionally resected. Internal chest arteries and veins are separated and secured. The mediastinal pleura is bluntly flapped outside. From the formed route an examination of the mediastinum and the organs located in it is performed (upper vena cava, aorta, regional lymph nodes, lung elements). If necessary, fat and particular lymph nodes are collected for histological examination. The study is completed with hemostasis, and in case of disruption of anatomical continuity of the mediastinal pleura and development of pneumothorax – with the drainage of the pleural cavity. After mediastinotomy, control X-ray examination is performed.

Non-invasive methods for the diagnosis of surgical heart disease

Cardiomarkers. Historically, for the diagnosis of ACS, the activity of one of the enzymes synthesized in our body was used (and continues to be used) – *creatine phosphokinase (CPK).* More specifically, one of its components is the *MB fraction of creatine phosphokinase (CPK-MB).* Increased activity of CPK-MB is the most specific for myocardial infarction (MI). In case of MI, the increase of the activity of CPK-MB begins already in 4-8 hours after an acute attack and reaches maximum in 12-24 hours; on the third day the activity of this fraction returns to normal. In case of the enlargement of the region of the myocardial ischemia, the activity of CPK-MB remains high much longer, what allows to diagnose MI during longer follow-up periods.

However, an increase in the CPK-MB fraction is also observed during non-surgical diagnostic cardiac manipulations. Radiotherapy of the chest may also cause a slight increase in the enzyme level. But arrhythmia, tachycardia and heart failure practically do not affect the level of CPK-MB.

An invaluable contribution to solving of all the above mentioned issues was an introduction of the evaluation of *myocardial markers* in the blood serum (plasma) into clinical practice – *troponins I and T* – absolutely specific and highly sensitive for the situation of acute coronary syndrome (ACS). *At present, leading cardiological associations of Europe and the United States have adopted recommendations in which the determination of the concentration of troponin T (in serum, plasma) in blood is considered a very important diagnostic criterion of MI.*

In recent years the *brain natriuretic peptide (NTproBNP)* has become another guaranteed cardiomarker. It was reliably and evidently substantiated, that the detection of its increased concentrations indicates that a patient has heart failure (HF). Therefore, in the diagnosis of HF in the world, evaluation of the level of this peptide has become the standard. It is worth noting that in a number of large international studies it has been shown that the use of the tests "NT-proBNP" and "BNP" in clinical practice allows to reduce the cost of patient examination and to prognose accurately the presence or absence of HF, even before echocardiography. Another important fact: the test is now widely used not only in case of cardiovascular pathology, but also in some clinical conditions, which are accompanied by the decrease of the contractile ability of the heart muscle.

Electrocardiography is used for:

a) diagnosis of myocardial hypertrophy;

b) diagnosis of heart rhythm disorders;

c) diagnosis of coronary circulatory disorders;

e) ECG stress tests.

In practice, it is mandatory to use 12 leads – 3 standard, 3 doubled monopolar and 6 chest ones. To specify the localization of focal changes, to establish the size of postinfarction and necrotic areas, to study their dynamics, precordial cartography is used (35 electrodes are placed in 5 horizontal rows). In addition, ECG can be registered with esophageal leads.

For the patients for whom stress tests are contraindicated (physical activity, pharmacological tests, stress test, with transoesophageal electrostimulation*), 24-hour ECG (Holter) monitoring* is performed.

Phonocardiography *is a method of graphic recording of tones and noises of the heart*. Most often, it is used in the diagnosis of congenital and acquired heart defects. Normal FCG consists of tones I and II, between which there is a straight line that corresponds to systolic and diastolic pauses. During the diastolic pause, sometimes tones III and IV are registered.

Echocardiography *is a method based on the recording of ultrasound signals reflected from the moving heart structures.* It is used with the aim of:

a) diagnosis of the disorders of morphology and mechanical activity of the heart;

b) monitoring of cardiac activity;

c)control of minimally invasive intracardiac surgical interventions (ablation of arrhythmogenic focus).

Echocardiography allows to:

1) identify the heart valves, their mutual position;

2) to recognize the inter-anterior and interventricular septum, to trace their continuity, to assess the type of movement;

3) to evaluate anatomical location of the heart valves and interventricular septum;

4) to evaluate the movement of the valve leaflets;

5) to carry out measurements and determine changes in the thickness of the walls and the sizes of the heart chambers to determine the presence and severity of dilatation of the heart cavities and myocardial hypertrophy of the left and right ventricle;

6) to conduct dopplechocardiography, combining it with two-dimensional echocardiography, to detect manifestations of valve regurgitation, narrowing in the path of blood flow and intracardiac shunts.

Stress ECG is used to replace ECG stress tests, most often with exercises. Also, *contrast echoCG* is used (contrast is administered intravenously). In addition to *transthoracic echoCG*, *transesophageal echocardiography* (including three-dimensional) and *intracardiac echocardiography* are used.

X-ray methods of heart examination allow to assess the heart shadow, to detect its increase or increase of particular heart chambers, to estimate pulmonary circulation. With the appearance of echoCG, the significance of the X-ray examination decreased.

X-ray contrast ventriculography allows to estimate the volume of ventricles, ejection fraction, systolic and diastolic functions.

Computed tomography is a method based on the use of X-rays to visualize the heart chambers, pericardium, the presence of thrombi in the heart cavity, tumors. Recently, common CT scan has been completely replaced by **spiral CT** or **NMRI.**

Electron beam computed tomography is a fundamentally new technology of CT scan, which allowed to reduce the time of examination significantly as compared to customary CT scan. It is mainly used to diagnose proximal occlusions of coronary arteries, anomalies of coronary arteries transposition, as well as their aneurysms. The disadvantage is the complexity of visualization of distal stenoses of the coronary arteries.

Multispiral computed tomography (MSCT) for the diagnosis of atherosclerotic vascular disease has been used since the 90s of the last century. In the beginning of the development of this method, direct visualization of the coronary arteries was impossible, due to low resolution and high percentage of motion artifacts. Therefore, the atherosclerotic disease of the arteries was evaluated by calculation of the content of intravascular calcium.

Quantitative evaluation of coronary calcinosis is based on the coefficient of X-ray absorption and the area of dense calcium. The calcium index (CI) by the Agatston method is defined as the formation of the area of calcific lesion by the density factor. It was observed that CI reflects the prognosis of a cardiovascular disease and correlates with the incidence of atherosclerosis development directly: the higher the rate, the greater the risk of the

atherosclerotic lesion. For example, with low CI of 10 units and below – probability of atherosclerosis of the coronary arteries is not more than 5-10%. With moderate CI from 11 to 100 units, the probability of presence of 50% narrowing – no more than 20%, with CI 101-400 units – 75%, that is, moderately high risk of atherosclerosis. And, with high CI, more than 400 units – the probability of atherosclerotic lesions of the coronary arteries is about 90%. CI is a predictor of the development of future cardiovascular accidents; the frequency of cases is significantly increasing with the indicator increase.

With creation in 1999 of 4, and later 8 spiral computed tomographs in 2001, it became possible to diagnose not only static objects with the evaluation of indirect signs of atherosclerotic injuries, but also direct visualization of the coronary bed. However, only on the edge of 2005 non-invasive 64-MSCT appeared in the arsenal of a doctor, what allowed to get pictures with the reconstruction of a three-dimensional image less than 0,5 × 0,5 × 0,6 mm even faster.

64-MSCT is used in diagnosis of the cardiovascular system in case of:

1) coronary heart disease (CHD);

2) diseases of the aorta (coarctation, aneurysms, dissection, etc.);

3) lesions of the peripheral arteries (obliterating atherosclerosis of the arteries of the lower extremities, atherosclerosis of the carotid arteries, etc.);

4) myocarditis;

5) pericarditis;

6) infectious endocarditis;

7) thromboembolism of the pulmonary artery;

8) congenital anomaly of the development of the cardiovascular system;

9) acquired heart defects (for example, calcinosis of the aortic valve with the development of stenosis or insufficiency, etc.);

10) arrhythmias.

Coronary arteries of the heart. High capability of 64-MSCT allows:

a) to visualize coronary arteries reliably, with adjustment of the localization of atherosclerotic lesion, to identify anomalies of the development of coronary vessels of the heart;

b) to determine the possibility of aorto-coronary shunts and intravascular endoprostheses (stents);

c) to calculate the calcium index in order to clarify the prognosis of the disease;

d) to distinguish violations of perfusion and viability of the myocardium in patients in the early and late stages of myocardial infarction;

e) to evaluate the heart contractility;

e) to study the state of the pericardium, heart valves.

Visualization of atherosclerotic lesions of coronary arteries with MSCT is an alternative to invasive cardiography and is used as in case of the confirmed CAD, of suspected CAD as in asymptomatic patients for diagnosis, detection of risk groups and determination of their future prognosis.

In particular***, indications*** for the study are:

1) atypical pain in the chest;

2) presence of risk factors:

- arterial hypertension;
- hyperlipidemia;
- obesity;
- diabetes;
- smoking;
- high CI;
- family history of coronary heart disease, sudden death, peripheral artery disease;

3) acute coronary syndromes, myocardial infarctions without ST increase to assess the lesions of coronary heart arteries.

64-MSCT allows to diagnose possible complications in case of myocardial infarction, for example, rupture of the interventricular septum, as well as the development of left ventricular aneurysm.

64-MSCT angiography is based on the scanning technique during the arterial phase and rapid transport of the contrast agent.

Technique. Any special preparation for a patient is not required for the examination. The MSCT is performed on an outpatient basis. The patient is in prone position. After preliminary scanning, necessary for accurate determination of the location of organs in the examined region, contrast iodine substance is introduced by bolus intravenously, then, with a slight delay, a scan is started under control of ECG for a little more than 10 seconds, and the subsequent data is processed by the computer. Full time of the patient examination takes about 15-20 minutes. The final data is further available for analysis and building of four-dimensional images.

The development of the method was supported by: high diagnostic value, relative ease of use, short time of information uptake, convenience for a patient (no premedication, preliminary tests, etc.), and also that the MSCT is the first non-invasive technique of visualization of the coronary arteries, which does not require hospitalization, without the risk of intra- and postoperative complications. The latest results confirmed the equivalence of the results of MSCT and coronary angiography in the diagnosis of coronary artery atherosclerosis.

Thus, modern 64-MSCT is not inferior to the invasive methods of CAG and AAG, which are the "gold standard" for the diagnosis of peripheral and coronary arterial diseases, taking precedence over the selective CAG:

- in the relative simplicity of the diagnostic procedure;
- in the absence of possible intraoperative and postoperative complications;
- in the time of examination and information uptake;
- in the absence of the demand for hospitalization and premedication.

In addition to the above presented advantages over CAG, MSCT provides an additional characteristic of the atherosclerotic plaque (detection of "soft" plaques, degree of calcinosis, etc.), determines the function of the heart systole (by the values of diastole and systole volume of the left and right ventricles, an accurate calculation of ejection fraction is made), detects zones of left ventricular myocardial dyskinesia (during building a four-dimensional volume image) with an additional assessment of the anatomy of the heart and blood vessels. And, what is important for the prognosis, MSCT maintains the option to determine perfusion capacity and evaluate the viability of the myocardium, what is especially important for the patients with myocardial infarction and the patients with heart failure.

Magnetic resonance imaging is considered a "gold standard" in the diagnosis of the global and regional functions of the left ventricle. *MRI coronary angiography* has a number of limitations – it diagnoses only hemodynamically significant stenoses of coronary arteries (more than 50%) and is limited to the visualization of only proximal and middle coronary arterial regions. New direction of MRI – it is *catheter-associated MRI visualization*. It combines catheter angiography and NMRI. It allows to evaluate the vessel wall as a whole, visualize an unstable atherosclerotic plaque, determine the rate of blood flow and evaluate the functional state of surrounding tissues.

Optical coherence tomography (OCT) is the latest gold standard of intraoperative diagnostics (including the molecular level) and visual control of balloon angioplasty, stenting, athertomy. It is designed for the visualization of blood vessels with the help of light radiation, which is directed through an intravascular catheter sensor. Since 2013, it has been used in Zaycev V.T. Institute of General and Emergency surgery of the National academy of medical sciences of Ukraine.

Radionuclide methods are based on the introduction of albumin or erythrocytes into the vein with a radioactive label (^{201}tallium or ^{99m}technetium). These methods allow to clarify the contractile function and blood supply to the myocardium, to identify ischemia and focus of myocardial necrosis, cicatricial areas.

Invasive methods for diagnosis of the cardiovascular system diseases

Catheterization of the heart and major vessels – the method allows to determine main hemodynamic indicators: blood pressure and blood oxygen saturation in different parts of the heart and blood vessels, shock volume and cardiac output, vascular resistance in systemic circulation and pulmonary circulation, localization, volume and direction of the pathological blood flow, presence and degree of valve regurgitation, size of valve openings, pressure in the heart cavities and vessels.

Coronary angiography is a technique that allows to determine the presence, manifestation, localization and distribution of coronary stenosis, as well as to identify its cause (atherosclerosis, thrombus, spasm). It is considered the "gold standard" in the diagnosis of coronary atherosclerosis. It belongs to the *selective angiography*, is performed with special catheters, as a rule, by retrograde approach through the femoral artery.

Indications:

a) stable severe angina pectoris (Grade 3 by the classification of the Canadian Cardiovascular Society);

b) stable angina pectoris (Grade 1 or 2) if a patient had myocardial infarction or there are ischemic episodes with little physical exertion;

c) in case of stable angina pectoris in patients with the blockage of the branches of His band, if the areas of ischemia are detected with scintigraphy;

d) the patients whom coronary vessels surgery is administered;

e) in case of severe ventricular arrhythmia;

e) in case of relapses of moderate or severe angina pectoris in patients who have undergone myocardial revascularization (catheter angioplasty or aorto-coronary bypass surgery).

The data of coronary angiography allows to determine the scope and tactics of surgical intervention. Therefore, without coronary angioplasty, it is impossible to perform any surgical operation, regardless of whether it is coronary angioplasty with stenting or aorto-coronary bypass surgery.

Coronary angiography is an invasive procedure associated with a certain risk! During coronary angiography ***complications*** are possible, but the risk of development of these complications is very small – the tiny fractions. However, sometimes bleeding at the site of the puncture, *allergic reaction to a contrast*, *heart rhythm disturbances*, *myocardial infarction* develop. Before coronary angiography, some patients need to undergo certain examinations: general blood test, blood group, Rh factor, hepatitis B and C virus tests, HIV, RW, 12 lead ECG, echocardiography, and also to purchase an elastic bandage.

Technique. Under local anesthesia (during the whole procedure, a patient is conscious), an artery puncture (femoral, axillary, radial) is performed, A small hollow tube (introducer) with a diameter less than 2 mm is introduced into the artery. Through the tube, a catheter is conducted to the coronary arteries of the heart. The contrast medium is brought via the catheter and under X-ray control of the special apparatus for angiography, recording of the examination is made. After the coronary angiography, the patient achieves hemostasis-stopping of bleeding at the puncture site, and an elastic bandage is used for this. So far, the procedure lasts from 10 to 20 minutes. If necessary, simultaneous balloon dilatation and / or placement of vascular endoprostheses – stents – may be performed, "without removal of a patient from the table". After the examination is completed, a specialist demonstrates recording of the coronary artery to the patient, explains the degree of coronary artery disease and recommends further treatment regimen.

III. 2. ACUTE PURULENT SURGICAL PATHOLOGY OF THE LUNGS AND PLEURA

Acute pulmonary suppuration occurs more often in adulthood, mainly in males who suffer from diseases 3-4 times more often than females due to alcohol overuse, smoking, increased susceptibility to hypothermia, and harmful working environment.

In 60% the right lung is affected, in 34% – the left one and in 6% – an injury is bilateral. The greater incidence of the injuries of the right lung is induced by special features of its structure: the broad right main bronchus seems to continue the trachea, what contributes to the exposure of the right lung to the infected material.

The classification of purulent diseases of the lungs is adopted and used in the clinical practice ***(Shalimov O.O.).***

1. *Acute purulent lung diseases:*
1) acute interstitial pneumonia;
2) acute solitary abscesses;
3) acute multiple abscesses;
4) acute gangrenous abscesses;
5) disseminated gangrene.
2. Chronic purulent lung diseases:
1) chronic pneumonia;
2) chronic solitary abscesses;
3) chronic multiple abscesses;
4) pyoinflammatory bronchiectases;
5) pyoinflammatory pulmonary cysts;
6) pyoinflammatory parasitic cysts of the lungs (echinococcus);
7) pneumosclerosis;
8) pyoinflammatory polycystic lung disease;
9) mycotic pyoinflammatory processes (actinomycosis, aspergillosis).
Purulent inflammatory bronchiectases *by etiology:*
a) acquired;
b) congenital;
c) cylindrical;
d) cystic;
e) mixed.

Acute abscess of the lungs

Acute abscess (simple, gangrenous) of the lung belongs to the group of purulent-destructive injuries of this organ and is initially manifested by purulent melting, appearance of the necrosis of the pulmonary parenchyma. Later, depending on the resistance of a patient's organism, the type of microbial flora and the ratio of alterative-proliferative processes, either sequestration and localization of necrotic areas or progressive purulent putrescent melting of surrounding tissues occur, and one or another form of acute pulmonary suppuration develops.

The pathological process in the lungs is characterized by dynamic character in this case and one form of the course of the disease may change to another.

Acute (simple) abscess of the lungs (ALA*) is purulent melting of necrotic areas of the pulmonary tissue, often within a single segment with the formation of one or more cavities filled with pus and surrounded by perifocal inflammatory infiltration of the pulmonary tissue. Purulent cavity in the lungs is often separated from unaffected areas by a pyogenic capsule.*

Gangrenous abscess *is a purulent putrescent disaggregation of the necrotic area of the pulmonary tissue (of a lobe, a segment), characterized by predisposition to sequestration and localization from undamaged areas. Gangrenous abscess is also called a circumscribed gangrene.*

Abscesses may localize in different areas of the lungs, but most often – in the upper lobe of the right lung. More often abscesses are localized "centrally" (closer to the bronchi), "peripheral" abscesses, which are located closer to the pleural cavity, occur more rarely.

In case of ALA, the onset of the disease may be acute both on the background of healthy condition and on the background of improvement (after previous ARVI, pneumonia), high fever, chills occur.

Clinical manifestations. The clinical picture of acute abscesses in the lungs is determined by many factors, the main of which are the nature and virulence of the infection, the state of the body defense, as well as the phase or the period of the process development.

The first period – the formation of an acute abscess is characterized by purulent infiltration and melting of the lung tissue, formation of dense connective tissue capsule, which clearly separates the development of a round formation, which within 4-10 days transforms into a cavity containing liquid pus with a small amount of gas above it. In this phase, the clinical picture of an abscess is similar to severe pneumonia. *Complaints* of chest pain on the injured side, high temperature of possibly hectic character, cough, shortness of breath. *Objective findings.* The condition of a patient is severe, in the elderly and young elderly patients, the consciousness may be confused due to the high temperature and intoxication.

Status localis. Slowing down of the chest during breathing on the injured side, tachypnoe, dull percussion sound over the region of the abscess formation, weakened breathing at the same time during auscultation.

The described clinical signs increase during 4-10 days, after which the abscess opens unilaterally into the nearest bronchus, what is manifested by the release of a large amount of purulent sputum (up to 200-500 ml) during an attack of coughing (usually in the morning after sleep) and *the second period of the abscess* begins – *drainage into the bronchus*. In the clinical picture – there is an improvement: drop of temperature, reduction of pain, coughing with purulent sputum. Sputum is often with a bad odor, with blood, contains a large number of bacteria, after defecation it is divided into three layers: the lower layer consists of pus and sphacelous tissues (detritus), the middle layer – of yellowish transparent fluid, the upper one is foamy. *By percussion* after bursting of an abscess into the bronchus the region of reduction of a percussion sound decreases, tympanic shade of percussion sound appears, *by auscultation* – appearance of "amphoric" breathing. The frequency of respiratory movements depends on the amount of lung tissue damage – different degrees of tachypnea intensity.

In case of bursing of the "peripheral" acute abscess into the pleural cavity, an ***emergency condition*** of a patient due to the development of ***complication*** occurs – ***acute pyopneumothorax*** *requiring emergency care* (pleural puncture and drainage of the pleural cavity).

The third period can occur in different ways, depending on the degree of emptying of the abscess cavity, body protective forces and effectiveness of treatment. Under favorable conditions (good drainage of a cavity, high level of immune protection, adequate treatment), this period ends with recovery due to the gradual subsidence of the cavity and its cicatrization. Under unfavorable conditions (insufficient drainage of the cavity, low degree of protective forces of the organism, inadequate treatment), the third period lengthens out, the cough with purulent sputum persists, the temperature does not normalize, signs of intoxication remain unchanged and most often the result is *the transition of the process to the chronic stage* – formation of the chronic pulmonary abscess.

Main clinical syndromes *are chest pain and dyspnea.*

In accordance with the standard schemes, ***the plan for laboratory and instrumental examination of a patient with acute abscess includes:***

1. Clinical blood test.
2. Clinical urine test.
3. Biochemical blood analysis.
4. Coagulogram.
5. Immunological tests.
6. Bacteriological study of sputum, contents of an abscess cavity.
7. X-ray examination of the chest organs in 2 views, tomography, bronchography – if medically required.
8. Spirography.
9. Diagnostic and sanitary fibrobronchoscopy.

Characteristic pathological changes.

1. *Clinical blood test:* moderate anemia, high leukocytosis with the left shift of the white blood cell count, accelerated ESR.

2. *Clinical urinalysis:* changes are nonspecific.

3. *Biochemical blood test:* reduction of total protein, dysproteinemia.

4. *Coagulogram*: hypercoagulable disorder with tendency to hypercoagulation.

5. *Immunological tests:* characteristic decrease of the parameters of cellular immunity, reduction of A / G coefficient, increase of CIC.

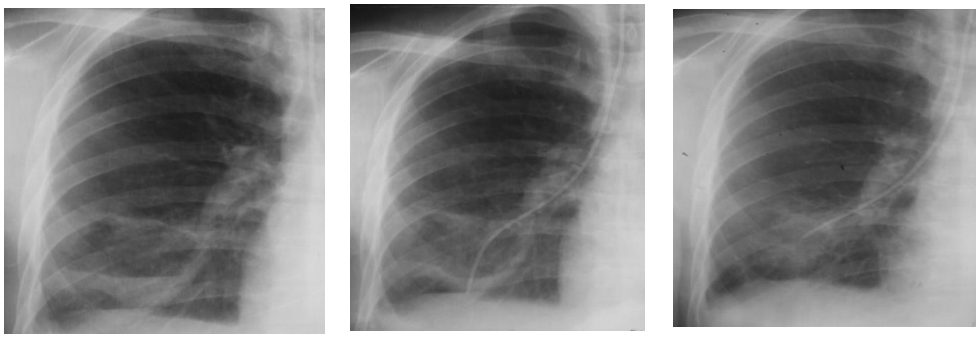

Fig. 69. X-ray study of the right lung: a - abscess of the lower lobe; b, c - microtracheostomic catheterization of the abscess for the sanitation of its cavity

6. *Bacteriological study of sputum, abscess cavity contents:* allows to determine the pathogens of the purulent-destructive process and evaluate the sensitivity of the latter to antibacterial drugs.

7. *X-ray examination of the chest organs in two views.* In the phase of purulent-necrotic melting (acute infiltration phase), the infiltration of the pulmonary tissue in the form of a focal (round) shadow is determined in the X-ray picture. In the phase of abscess drainage through the bronchus one or more destruction cavities are clearly defined on the x-ray (Fig. 69), most often with a horizontal level of fluid and perifocal inflammatory infiltration of the pulmonary tissue around the cavity. Overexposed images or tomograms help to detect destruction cavities in the lungs. With the help of tomography, lung sequesters are diagnosed.

During drainage of the peripheral abscess to the pleural cavity and the development of *acute pyopneumothorax* in the pleural cavity in the lower parts an opacification is detected in the X-ray picture (Fig. 70) due to the liquid pus and above it – lucent area due to the air, different degrees of lung compression.

8. *Spirography* – reduction of VCL and MPV by 25-30% from the proper values, reduction of CIO_2 below 25 when oxygen saturation of arterial blood is reduced by less than 90% .

9. *Fibrobronchoscopy*: in the phase of drainage of the abscess through the bronchus the symptoms of purulent tracheobronchitis are determined. The localization of the abscess according to the duct of the draining bronchus may be determined.

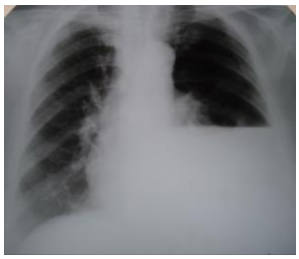

Fig. 70. Acute pyopneumothorax on the left

Differential diagnosis in acute abscesses should be made between cavernous tuberculosis, actinomycosis, echinococcosis, suppuration of pulmonary cyst, interlobar encapsulated pleurisy, focal pneumonia, as well as tumors and primary bronchiectases in the abscessing phase.

Cavernous tuberculosis is usually excluded during the study of anamnesis, absence of Koch's bacilli and specific for tuberculosis X-ray changes in the lung outside the cavity with liquid level.

For ***actinomycosis***, the presence of druses in the sputum is characteristic, which is difficult to detect, and this is the reason why subsequent thorough examination is required. Characteristic involvement in the process of adjacent organs, chest wall.

In case of ***suppurated parasitic (echinococci) and congenital pulmonary cysts***, the condition of a patient is not as grave as in case of acute abscess, there is no prior lung inflammation; during X-ray examination, smooth, round, clear shadows with no perifocal inflammation are determined. In the sputum, chitinous coats, secondary cysts, and hooks are found.

Particularly difficult is the diagnosis of ***interlobar pleurisies***, opened into the bronchus, and in case of ***encapsulated pleurisy***. In such cases, the follow-up multi-axial X-ray examination can be effective, by means of which you can clarify the diagnosis.

It is also difficult to distinguish acute abscess from the ***focal pneumonia*** before emptying its part through the bronchus. In these cases, dynamic monitoring of patients, repeated radioscopy may help, what allows to define beginning of the cavity formation in the center of intense opacification.

It is characteristic for the abscesses, which develop on the background of ***the lung tumor***, that they are detected on the background of atelectasis of the lobe of an entire lung, and in x-ray pictures, and especially in tomograms, the shadow of a tumor is visible in the drainage region of the bronchus.

Abscesses during ***primary bronchiectasies*** are the late phase of the development of bronchiectatic disease, and therefore they are easy to distinguish from acute abscesses of the lungs according to the anamnesis data.

It should be remembered, that the acute abscess could be distinguished from ***the gangrene of the lung*** by the clinical course, because all the signs of the acute abscess are expressed more significantly, as well as severe intoxication. X-ray examination for gangrene of the lung reveals the presence of the complete opacification of the part of the lung on the injured side with a gradual change to the normal pulmonary pattern along the periphery of the opacification.

Treatment of a patient with acute pulmonary abscess. *Choice of therapeutical approach*. Treatment of the patients with acute abscesses of the lungs should be performed only in the hospital. The treatment tactics depends on the stage of purulent destructive process and the efficacy of its treatment.

At the stage of acute infiltration – conservative therapy in combination with bronchological methods.

At the stage of the drainage of the abscess through the bronchus – active use of bronchiological methods of sanitation (including the most effective ones – microtracheostomy) on the background of conservative therapy. In case of insufficient drainage of the abscess through the bronchus or its complete absence (blocked abscess), the puncture and draining surgical techniques are used.

Pathogenetically grounded ***conservative therapy*** is focused on the control of a pathogen, which caused purulent destructive process in the lungs.

1. *Regimen* – partial bed rest.

2. *Diet* with high-energy value and high protein dietary intake. The food should contain an increased amount of vitamins.

3. *Drug therapy*:

1) antibiotics for empirical therapy (before obtaining the results of bacterial swab test and evaluation of antibiotic susceptibility), synthetic penicillins, macrolides (Sumamed), fluoroquinolones of IIIrd-IVth generations, cephalosporins of IIIrd-IVth generations are most often used;

2) non-specific anti-inflammatory drugs (NSAIDs – Movalis, Ketoprophenum and its derivatives Oruvail, Ketonal), which are administered in the forms of injections and tablets;

3) immunocorrective therapy (Levamisole / Decaris by 0.15 daily for 3 days with intervals of 14 days, for 4-6 months);

4) direct anticoagulants (Heparin, Fragmin, Fraxiparine);

5) drugs that improve the escalating function of the lungs (Lasolvan, Fluimucil, ACC);

6) detoxification therapy (Neohemodez, Sorbilact, Rheosorbilact, Reamberin);

7) infusion therapy (plasma to increase the body defense, amino acid solutions for protein exchange correction, Ringer solutions, officinal saline solutions for the correction of electrolyte balance, glucose-insulin mixtures).

During the administration of broad-spectrum antibiotics and in the beginning of the treatment in the early phase (up to 6-8 weeks from the moment of abscess formation), it is possible to achieve success by conservative methods in 65-70% of patients with acute abscesses. This allows to recommend conservative treatment to the patients with acute pulmonary abscess for 6-8 weeks from the moment of its formation, if there are no special indications for the operation. This treatment involves the administration of broad-spectrum antibiotics according to antibiogram data, which are used alone and in combination and are usually administered intramuscularly or intravenously.

During the conjugation of the abscess with the bronchus, it is necessary to provide regular and sufficient removal of pus from the abscess cavity via the bronchoscope or with the help of *"postural drainage" ("drainage with position"),* which is performed in the following way: taking into account the location of an abscess in the lung and the place of transposition of the draining bronchus, a patient is laid in the position, in which the draining bronchus is the lowest point of the abscess, and the patient is made to cough out sputum. After such emptying of the abscess from pus, antibiotics are administered intratracheally. Proteolytic enzymes (Trypsin, Chymotrypsin, Ribonuclease), which are administered via *inhalation, by microtracheostomy or therapeutic bronchoscopy*, are used to increase the efficiency of the bronchial tree sanitation.

Recently, due to the resistance of microflora to antibiotics and development of dysbiosis, as well as allergic reactions, *combined treatment with antibiotics and modern antiseptics* is recommended – intravenous administration of solutions of Dioxidine, chlorhexidine, Chlorophyll, diluted with physiological solution. A method of treatment with *Dimexid* is used. A mixture of 15-20 ml of 20% Dimexid and 200 000-300 000 units of antibiotics are injected into the bronchi at intervals of 4-5 days. In 50% of patients, after two to five procedures, microflora disappears, especially pneumococci and hemolytic streptococci.

In case of the "peripheral" acute pulmonary abscess, *the intrapulmonary method* of treatment is used: after puncture aspiration of pus, a cavity is washed with 50% solution of Dimexid until content is clear, then 20-30 ml of 70-90% solution of the medication together with antibiotics are injected into the abscess cavity. It is possible to establish a permanent drainage of the cavity of such abscess using Seldinger technique.

In case of the central localization of an acute pulmonary abscess, the method of microtracheostomy catheterization of an abscess is effective for the permanent sanitation of its cavity.

If within 3-8 weeks (individual approach) from the moment of abscess formation conservative treatment does not give a positive result, ***operative treatment*** is administered for the *removal of a damaged area of the lun*g. There are resection methods: *segmentectomy* (removal of a segment), *lobectomy* (removal of 2 lobes), *bilobectomy* (removal of 2 lobes). In severe cases (with multiple abscesses) *pulmonectomy* (removal of the lung) is performed.

Causes of unsatisfactory results of conservative treatment and *indications for surgical intervention*:

1) insufficient drainage of ALA through bronchial tree;

2) "peripheral" lung abscess that cannot be drained;

3) "peripheral" lung abscess, the cavity diameter of which exceeds 5 cm;

4) complications (acute pyopneumothorax);

5) spread of purulent destructive process (gangrene of the lung);

6) chronization of the abscess.

Postoperative complications are possible: a) postoperative hemoptysis and pulmonary haemorrhage; b) pleurisy; c) pleural empyema; d) failure of the bronchial stump after resection methods and development of bronchial fistulae.

Gangrene of the lung

Gangrenous lung *(GL) is the necrosis of pulmonary tissue under the influence of toxins and nourishment failure that has no clear borders.*

The acute pulmonary abscess and gangrene of the lung have much in common, but nonetheless, most authors consider these diseases independent. In case of ALA, the inflammatory reaction and purulent focus are limited in character and in case of pulmonary gangrene – it is the necrosis of pulmonary tissue, which has no clear borders. In case of gangrene, necrotic process diffusely extends into the pulmonary tissue. The areas of normal tissue without noticeable borders change into altered, dark, without a clear structure pulmonary tissue, which also changes into a dark mass without clear borders. At the same time, pulmonary tissue looks like a gray-green dirty mass that produces bad odor. A lobe, two lobes or the entire lung are usually affected. Polymicrobial flora causes gangrene of the lung: staphylococci, gram-negative bacteria, nonspore-forming anaerobes. Disturbances of the secure bronchus with the development of atelectasis, disturbances of blood circulation; formation of a cul-de-sac in the atelectasis area and arrest of bronchi elimination of infection with coughing; and, in particular, the influence of a large number of toxins of the microorganisms developing on the lung tissue contribute to the development of gangrene.

Clinical picture. Everything related to the acute pulmonary abscess, to the full extent pertains to the gangrene of the lung with the only difference that the first place is taken by the sharp intoxication of the body of a patient. The disease is accompanied by constant high temperature, which does not decrease for a long time, or sharp fluctuations in the morning and in the evening. Painful cough with especially putrid sputum is indicative. Putrid character can be observed before sputum appearance.

Sputum looks like a foamy liquid, it is of dirty grayish color, sometimes with raspberry or chocolate shade, what is caused by parenchymal bleeding from the disintegrating tissue. During defecation of sputum it is divided into 3 layers: the upper one – liquid, the medium – serous, the lower – thick, consisting of crumbly mass and fragments of the pulmonary tissue. A large amount of sputum is usually excreted in the morning and is accompanied by a painful cough.

Patients complain of severe pain in the affected half of the chest. This results from the damage of the pleura, rich with nerve endings.

Objective findings. The condition of patients with pulmonary gangrene is always complicated. They quickly weaken, get exhausted, sweating, lack of appetite, progressive anemia are observed. There is a frequent and small pulse, muffled heart sounds, decrease of blood pressure.

Status localis. During the examination of a patient retardation of the injured half of the chest is observed. By percussion – dull percussion sound with obscure diffuse borders. By auscultation – a lot of various rales is heard.

At the beginning of the disease, hyperleukocytosis with left shift of the leukocyte formula is registered *in the blood*. As the gangrene progresses, leukocytosis may reduce.

During an X-ray examination, there is a total cloud on the part of the lung with a gradual change to the normal pulmonary pattern along the periphery.

If the gangrene progresses, then peripheral lung portions get injured, and at the same time the parenchyma of the lung breaks down in the form of sequesters and gets into the pleural cavity. This leads to the development of *putrid pyopneumothorax* and the disease takes a septic course.

Treatment of the patients with pulmonary gangrene. The treatment of the pulmonary gangrene is exactly the same as of an acute pulmonary abscess, with the only difference that it should be more intense.

Conservative treatment with PG is performed:

1) in case of advanced forms of pulmonary gangrene, expressed pulmonary heart disease and in case of bilateral injury;

2) if there is the transformation of gangrenous abscess into the normal adequately drained acute abscess;

3) uncomplicated gangrene of the lungs with an expressed favorable clinical and radiological time course on the background of the treatment performed.

In all other cases, conservative treatment for acute pulmonary gangrene should be considered as a preoperative period.

Intensive therapy in case of PG includes infusion therapy for parenteral nutrition, correction of volemic disorders, improvement of rheological properties of blood, maintenance of energy balance and detoxification. The support of energy balance in this case is provided by the introduction of concentrated glucose solutions 25-40% up to 1 liter. Restoration of protein loss is most often accomplished by the introduction of solutions of amino acids, fresh frozen plasma, albumin solutions, and others.

For detoxification and improvement of rheological properties of blood, improvement of capillary blood circulation, infusions of Neohaemodes, Rheopolyglukin, Reamberin are administered.

For the correction of anemia, transfusions of erythromass (the best-washed erythrocytes), iron preparations are administered.

For extremely serious patients with pulmonary gangrene and the clinical picture of ***septic shock*** and manifestations of ***multiple organ failure*** combined therapy with the administration of drugs, that influence cellular regulation of the antioxidant immune response, is indicated: Cytoflavin 10 mg twice a day per 200 or 900 ml 5% or 10% glucose solution with subsequent addition of cycloferon 4 ml twice a day after stabilization of the patient's condition. In severe cases, introduction of Pentaglobin (USA) 10 or 20 ml IV for a solvent of 50 ml or 100 ml (contains complete and biologically intact immunoglobulins in a stable form).

In case of unsuccessful conservative therapy, patients with pulmonary gangrene should undergo surgical treatment.

Methods of ***surgical treatment*** are divided into *resection and drainage*. Drainage operations are less traumatic. They are tolerated even by seriously ill patients, but their positive effect is less pronounced. Drainage can be performed with thoracocentesis and a drainage tube. At present, drainage is performed by *miniinvasive thoracoscopic methods*. Only cavities with liquid pus and small sequesters can be drained in case of gangrene. During thoracoscopy, pus, all sequesters are removed, complete sanitation is made. However, if general condition of a patient allows, then radical treatment is a resection of the lung lobe, two lobes or removal of the lung – pulmonectomy.

Acute pleural empyema

Acute empyema of the pleura *is a limited or diffuse inflammation of the visceral and parietal pleura, which occurs with accumulation of pus in the pleural cavity and is accompanied by the signs of purulent intoxication and often respiratory insufficiency.*

Nonspecific empyema of the pleura is caused by various suppurative or putrifactive microorganisms. Staphylococci are most often detected – up to 77% in the pleural cavity. This is explained by their pronounced virulence and resistance to most antibacterial agents. In 30-45% of cases, in pus culture from the pleural cavity, gram-negative microorganisms get growth – these are various strains of the colibacillus, Pseudomonas aeruginosa, Proteus. Up to 80% of the cases are anaerobic non-clostridial flora (bacteroids, fusobacteria, peptococci, peptostreptococci, and others).

Pathogenetically primary and secondary pleural empyema are distinguished. In case of the primary pleural empyema, the focus of inflammation is localized in the pleural cavity from the very beginning, in case of the secondary one – it is a complication of some other purulent inflammatory disease.

Primary pleural empyema occurs on the background of unchanged, healthy pulmonary pleurae as a result of the violation of their barrier function with the introduction of microflora. This happens during chest injury, after manipulations in the pleural cavity and pulmonary operations.

According to many authors, in 85-90% of cases, ***secondary pleural empyema*** appeared as a complication of pneumonia, acute and chronic purulent lung diseases. From the very beginning pneumonia may proceed with the development of purulent pleurisy (parapneumonic pleural empyema, or empyema of the pleura develops at the end of pneumonia and acquires the character of an independent disease (metapneumonic).

In case of lung abscesses, the empyema of the pleura develops in 8-11% of patients, and in case of pulmonary gangrene – in 55-90%.

In particular cases, empyema may develop as a complication of suppurative or parasitic lung cyst, decaying cancer, spontaneous pneumothorax.

Secondary pleural empyema may develop by contact, in case of inflammation of chest wounds, osteomyelitis of the ribs, spine, sternum, chondritis, lymphadenitis, mediastinitis, pericarditis.

In rare cases the source of contamination of the pleura may be acute inflammatory diseases of the abdominal cavity (subdiaphragmatic abscess, cholecystitis, pancreatitis, etc.). The penetration of microbes from the abdominal cavity into the pleura occurs through the lymphatic vessels and fissures in the diaphragm, the so-called "hatches" (dilated lymphatic vessels), or by hematogenous way.

The clinical practice accepts and uses the following classification of pleural empyema:

I. *By etiology:*

1. Nonspecific: purulent; putrid; anaerobic
2. Specific: tuberculosis; fungal; syphilitic
3. Mixed.

II. *By pathogenesis:*

1. Primary: traumatic; postoperative.
2. Secondary: para- and metapneumonic; contact; metastatic.

III. *By clinical course:*

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

IV. *By the presence of pulmonary destruction:*

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

V. *By contact with the external environment:*

1. Closed.
2. Open:

- with bronchopleural fistula;
- with a pleurodermal fistula;
- with bronchopulmonary fistula;
- with the ethmoidal lung;
- with other hollow organs.

VI. *By extension:*

1. Separated: apical; paramediastic; superdiaphragmatic; interlobar; parietal.
2. Generalized: total; subtotal.

Clinical picture. Usually the disease begins acutely on the background of injury or previous acute inflammation of the pulmonary tissue. Permanent symptoms are general weakness, poor appetite, insomnia, fever up to 38-39 °C, chest pain and dyspnea. Pain syndrome occurs, as a rule, on the injured side, is of permanent or abnormal character, increasing with deep breathing, coughing, changing of the body position. Sometimes there are abdominal pains due to

the irritation of the diaphragm with irradiation along the abdominal nerve. Cough – often with a discharge of a large amount of sputum, depending on the degree of a lung injury, presence of bronchopleural fistula.

*Objective statu*s. General condition of a patient is more often of moderate severity – severe. Consciousness is usually clear. The skin is pale. The patient takes a forced position – sitting or lying on a healthy side. Cardiovascular system disorders: typical palpitation. Tachycardia is associated with an increase of the body temperature. Heart sounds are muffled. There is a tendency to hypotension. The development of pulmonary heart failure with increasing decompensation of blood circulation and hypertension in pulmonary circulation is possible, what is indicated by the accent of IInd tone on the pulmonary artery.

Status localis. There is a restriction of respiratory movements of the affected half of the chest, smoothing of intercostal spaces, local edema of the skin and subcutaneous tissue over the area of accumulation of pus in the pleural cavity. Later, the tissues of the thoracic wall in this area become dense, tenderness increases, hyperemia of the skin appears. During percussion over the zone of fluid accumulation, dullness is determined. In the absence of air and adhesions in the pleural cavity, the upper border of dullness corresponds to *the Damoiseau-Ellis line*. By auscultation, weakening of vesicular breathing is registered, until its complete absence over large accumulation of fluid. Above the compressed lung area – bronchial breathing, sometimes various wet rales are heard, sometimes the noise of the pleura friction due to fibrinous pleurisy around the empyema cavity. If bronchopleural fistula is present and the cavity is well drained through the bronchus, then amphoric breathing can be heard. An increase in bronchophonia above the area of fluid accumulation is very typical.

Leading clinical symptoms and syndromes *– chest pain, shortness of breath, intoxication.*

In accordance with the standard schemes ***the plan of additional laboratory and instrumental examination of a patient with acute pleural empyema:***

1. Clinical blood test.
2. Clinical urine test.
3. Biochemical analysis of blood.
4. Coagulogram.
5. Study of blood electrolytes.
6. Immunological tests.
7. Bacteriological study of sputum, bronchial washings, abscess cavity content.
8. X-ray examination of the chest.
9. Spirography.
10. Fibrobronchoscopy.

Characteristic pathological changes.

1. *Clinical blood test*: moderate anemia, leukocytosis with neutrophilosis, shift of leukocyte formula to the left, increase of ESR.

2. *Clinical urinalysis*: changes are nonspecific – presence of signs of toxic nephropathy: albuminuria, cylindruria.

3. *Biochemical blood test*: pronounced hypoproteinemia, dysproteinemia.

4. *Coagulogram*: disturbances of blood coagulation tending to hypercoagulation with a decrease of the time of blood coagulation, a significant increase in the level of fibrinogen.

5. *Study of blood electrolytes*: hyperkalemia is observed, what is explained by disintegration of tissues and formed blood elements.

6. *Immunological tests*: typical reduction of indicators of the organism reactivity.

7. *Bacteriological study of sputum*, bronchial washings, content of the pleural cavity: allows to determine causative agents of the inflammatory process and the sensitivity of the latter to antibacterial drugs.

8. *X-ray examination of chest organs:* it is the most important, allows to accurately verify the diagnosis and determine the immediate therapeutic tactics for the patient. The most informative is polypositive radioscopy (Fig. 71), what allows to localize injuries, to determine precisely the degree of lung collapse and displacement of the mediastinum, amount of fluid, to detect pathological changes in the pulmonary parenchyma, to select a point for adequate drainage of the pleural cavity.

It should be emphasized that radioscopy is often a diagnostically sufficient examination for making decision on the implementation of urgent (emergency) therapeutic measures – puncture or drainage of the pleural cavity for its decompression during tension pyopneumothorax. In case of its absence, if the patient's condition allows, it is possible to perform *lateroscopy*, which allows to determine the vertical dimensions of the cavity precisely, to assess the condition of the basal segments of the lung, "covered" with the level of the fluid. If laparoscopy on a healthy side is associated with the danger of aspiration of the contents of the abscess (in fragile patients), for determination of the lower point of the cavity the X-ray examination in the lateral view is sufficient, with the body bent forward or in a frontal view with the body bent to a healthy side.

Tomography allows to answer the above mentioned questions, however, this study is not informative on the background of lung collapse or the presence of significant amount of fluid in the pleural cavity. Therefore, it should be reasonably performed after drainage of the pleural cavity and emptying it from pus. If the lung is collaborated by more than 1/4 of volume, the interpretation of tomographic data becomes complex. Under these conditions, tomography may be performed during connection of the drainage to the aspirator operating in the "advanced vacuum" mode.

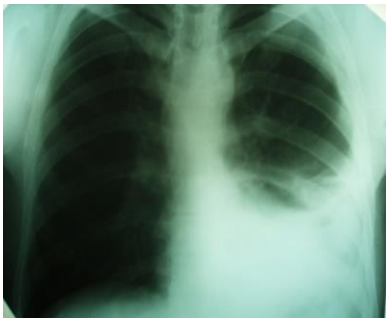

Fig. 71. Left-sided acute empyema of the pleura (the Damoiseau line)

9. *Spirography* – low indicators of VCL, reduction of compensatory capacities of the lungs in combination with hyperventilation, decrease of the saturation of arterial blood with oxygen.

10. *Bronchovideoscopy*: provides information on the degree of severity of the inflammatory process in the tracheobronchial tree, which allows to determine opening of the draining bronchus (in case of the presence of destruction of the pulmonary tissue). The resolution of bronchoscopy increases with the introduction of stain (water solution of methylene blue) into the pleural cavity in healthy side-lying position. This allows to determine which bronchi are involved in drainage of the destruction area of pulmonary tissue, what is very important for planning the level of temporary endobronchial obstruction.

Differential diagnosis is made between pneumonia, obturator atelectasis, hydrothorax, pulmonary abscesses, subdiaphragmatic abscess, lung cancer in the decaying stage and presence of cancer pleurisy, pleural tumors, suppurative cysts, echinococcus, diaphragmatic hernias.

Treatment of a patient with acute pleural empyema. *Choice of disease management*. Currently, in the treatment of acute pleural empyema, a combination of surgical methods of treatment focused on the evacuation of purulent contents from the pleural cavity, and conservative therapy, which is considered the main part of preoperative preparation and postoperative management of patients, is used.

Pathogenetically grounded ***conservative therapy*** is focused on the control of three major pathological factors – suppuration, losses and resorptions. It is impossible to treat patients with pleural empyema only by local therapy (surgical methods of sanitation) without influence on an entire organism, without normalization of the dysfunctions of organs and elimination of occurring complications, without the therapy of primary and concomitant diseases. Complex therapy is administered to such patients, and includes etiological, pathogenetic, symptomatic and

local treatment. The intensity of the treatment and significance of its components varies depending on the general condition of a patient, presence of concomitant diseases and complications, on the stage of purulent process and nature of empyema itself.

The main measure in the conservative treatment of acute empyema of the pleura is re-puncture of the pleural cavity, which is performed in the traditional place – in the VIIth-VIIIth intercostal space along the posterior axillary line along the upper edge of the lower rib.

During the course of punctures, evacuation of purulent content, lavage and sanitation of empyema cavity with solutions of antiseptics and antibiotics with leaving the latter in the pleural cavity is performed. The frequency of punctures depends on the rate of accumulation of purulent exsudate.

An important etiotropic method of therapy is the *administration of antibiotics, sulfanilamides and antiseptic drugs.* Antibiotic therapy should include 2-3 drugs, which influence pathogenic aerobic and anaerobic microorganisms.

Antibiotic therapy is supplemented with the administration of Metronidazole (Trichopol) at a dose of 1.5-2 g / day, because this drug has high bactericidal activity against anaerobic bacteria.

Pathogenetic therapy includes administration of anti-inflammatory drugs (NSAIDs), bronchodilators (Lasolvan, ACC, Bronchobru, Fluimucil), taking measures for correction of disturbances of protein (transfusion of fresh frozen plasma, amino acids), mineral (infusion of crystalloid solutions), carbohydrate metabolism (infusion of glucose solution 5%); administration of oxygen therapy (5-7 liters per minute), detoxification treatment (sorbilact, rheosorbilact, reamberine).

In the complex treatment of the patients with pleural empyema, adequate nutrition with high content of proteins and vitamins is very important. To improve appetite, administration of hydrochloric acid, acidine-pepsin, tincture of magnolia-vine, Chinese ginseng, pantocrine is advised. In debilitated patients, anabolic hormones (retabolil 50 mg, Superanobolon 1-2 ml IM once a week, Nerobol 0,005 g t.i.d. per os, etc.) are administered.

Pleural punctures are quite effective in the cases without bronchopleural fistula present (in case of primary pleural empyema). In the absence of an effect within 1-2 weeks of puncture therapy, it is necessary to turn to surgical methods.

Methods of operative treatment and indications for them. Surgical methods of treatment of acute empyema are divided into *"closed" and "open"*.

Closed methods include *different methods of drainage of the pleural cavity*. Drainages of the pleural cavity are divided into *"passive" and "active"*.

"Passive" drainage – when purulent discharge from the pleural cavity flows out passively through a drainage tube to a lower placed vessel with sterile furacilinum solution according to the law of communicating vessels; at the end of a drainage tube there is a valve, made from a rubber glove finger, the end of which is cut and creates two petals, which, sticking in case of occasional withdrawal of the tube from the solution, prevent the air influx into the pleural cavity. This is a *passive drainage by the method of Bülau.*

"Active" pleural drainage – when negative pressure is created in the drainage system and *active aspiration* of purulent discharge from the pleural cavity occur. This is a *double-ampoule Subbotin's system*, aspiration with small motors (for example, for aquariums), plastic corrugated devices ("accordions") are the most widely used.

Active drainage of the pleural cavity with a constant suction of exudate and air (in presence of bronchopleural fistula) should be considered the most effective method of treatment.

Technique of drainage: after an X-ray examination and confirmation of the presence of free exudate in the pleural cavity, under local anesthesia an incision of the skin with a scalpel (up to 1 cm) is made at the traditional point of the pleural puncture, then we bluntly penetrate with auriscalpium through the chest wall into the pleural cavity, through which a special drainage tube with the holes on each end is introduced. It is directed along the costal surface of the pleural cavity upwards, fixed on the skin by a U-shaped or purse suture and connected to the aspiration

system. To improve the quality of the procedure, it is recommended to use X-ray drainage and drainage under the ultrasound control.

After draining 3-4 times a day OD intermittent lavage of the empyema cavity is performed.

If the defect of the bronchus (in the presence of bronchopleural fistula) is significant and through it a large amount of air comes, then active aspiration from the pleural cavity makes no sense, because vacuum conditions cannot be created, and intense air suction leads only to the increased disorders of external respiration. In such cases, passive underwater valvular Bülau drain is used.

The results of aspiration methods of treatment were significantly improved due to implementation of the methods of *temporary obstruction of bronchial fistula by foam or collagen obturators*. Selective sealing of the bronchial system by temporary obstruction of the bronchi allows to make difference between purulent processes in the lungs and pleura. Indications for this method are acute pleural empyema with destruction of the pulmonary tissue and extensive air dump via drainages, acute failure of residual stump of the bronchus or pulmonary tissue in conditions of acute empyema after pulmonary surgery.

In recent years ***minimally invasive thoracoscopic techniques*** have been widely used and they allow pus aspiration, pleural effusion, destruction of intrapleural drainage, removal of pseudomembranes from the visceral pleura, elimination of adhesions between the pulmonary pleurae, directed drainage of the pleural cavity.

The patients with broncho-pleural fistulas undergo electrocoagulation of the fistula or (in case of their large diameter) temporary obstruction of the bronchi.

In case of ineffectiveness of "closed" and minimally invasive methods of treatment of acute empyema ***"open" surgical method is used*** – thoracotomy with pleurotomy for mechanical sanitation of the pleural cavity (possible resection of a lung portion containing a fistula), what results in the wound suture and drainage of the pleural cavity.

In the postoperative period, as a rule, conservative therapy, which has been started earlier, is continued. The main tasks of the therapy in the postoperative period are the following: providing of adequate ventilation of the lungs and prevention of hypoxia; correction of volemic disorders and restoration of adequate hemodynamics; prevention of hypercoagulation and infectious complications.

Possible *postoperative complications:*

1) postoperative bleedings;
2) development of the residual cavity;
3) suppuration of the postoperative wound;
4) failure of the bronchus stump after resection methods;
5) development of peristump abscess.

After completion of the course of treatment in a hospital, patients with acute pleural empyema are mandatorily observed by a pulmonologist at the polyclinic at their place of residence. Operated patients are observed also by a surgeon.

Chronic pleural empyema

The currently accepted clinical term ***"chronic pleural empyema"*** (*CPE) means a purulent destructive process in the residual pleural cavity with rough and persistent morphological changes characterized by prolonged periodic exacerbations.*

The incidence of chronic empyema is observed in 4-20% of patients with acute pleural empyema. The great differences in the frequency of chronic pleural empyema of such origin are caused, first of all, by the absence of a consolidated standard on the criteria of migration of an acute form of this disease to the chronic one. Histologic studies of the material obtained during pleurectomy, decortication and resection of the lung have proved that persistent and irreversible morphological changes in the pleura and underlying tissues develop only to the result for 2-3 months from the onset of the disease. At the same time, there are signs of violation of regenerative mechanisms and acerbation of a purulent process.

Proceeding from this, those cases of chronic empyema of the pleura should be considered the most reasonable, during which the empyema persists for more than 3 months and the course of the disease has characteristic clinical signs – periods of subsiding and aggravation of purulent process.

Alternatively to an acute form, mixed flora with the predominance of gram-negative bacteria (intestinal, Pseudomonas aeruginosa) is much more common during chronic pleural empyema. The formation of a residual cavity may be caused by several reasons. This, first of all, is an incomplete lung spread due to the presence of exudate in the cavity, as well as the result of:

1) commissure of the collapsed portions of the lung with dense, organized fibrous masses, which are not subject to lytic therapy;

2) significant induration and sclerosis of the pulmonary tissue;

3) discrepancies in volumes of the resected lung and pleural cavity;

4) atelectasis of a lung portion due to obstruction of the bronchial tree.

If during acute empyema the lung is not completely spread, then a cavity between the pleural cavity remains, the walls of which are covered with granulation tissue. Over time, this tissue matures and turns into fibrous connective tissue, that is, becomes denser. The lung in the initial stage of the disease remains floating and expands in case of emptying of the pleural cavity from exudate, and in case of accumulation of exudate the lung deflates again. During a long-term course of exudative inflammation, the lung becomes covered with connective tissue, as a shell, and loses an ability to spread. These fibrous layers on the pleura are called *commissures*. During a long course of the disease, they reach significant thickness (2-3 cm or more) and density. Therefore, *long-term inflammation is one of the causes of chronic pleural empyema.*

The clinical picture of chronic pleural empyema develops gradually. There is no clear border between clinical manifestations of acute and chronic pleural empyema, because acute inflammation gradually becomes chronic. The pronounced difference of clinical manifestations is typical only for extreme forms of acute and chronic pleural empyema.

Usually, the disease begins gradually. Constant symptoms are an increase of body temperature up to 37-38 °C, moderate chest pain, shortness of breath, cough, and presence of secretions from the pleural fistula. Pain syndrome – appears on the injured side, is of nagging nature, which increases with deep breathing, cough, changing the body position. Cough often occurs with discharge of various amounts of sputum, depending on the degree of a lung injury, presence of bronchopleural fistula.

In the anamnesis of the disease – acute pleural emphysema.

Objective findings. General condition of a patient is often of moderate severity. The severity of the condition is related to the size of a residual cavity, in which pus remains, and is determined by the degree of intoxication. Skin is pale with waxy shade, face puffiness, cyanosis. *Clubbed fingers, deformation of nail plates in the form of "watch glass".* Cardiovascular system disorders include characteristic palpitation, tachycardia (associated with an increase of body temperature), muffled heart tones, tendency to hypotension is observed, cardiac deviation is possible.

Status localis. Limitation of the respiratory movements of an affected half of the chest is observed, volume reduction of an injured half, intercostal spaces are narrowed. Often there is a pleural fistula with purulent discharge. By percussion over the area of fluid accumulation dullness is defined, over the area of air accumulation – timpanitis. By auscultation – weakening of vesicular breathing is observed up to its complete absence over a large fluid concentration. Above the area of the compressed lung is bronchial respiration, sometimes various wet rattles are heard. Increase of bronchophonia over the area of fluid accumulation is a characteristic feature.

Leading clinical symptoms and syndrom*es – chest pain, shortness of breath, intoxication.*

In accordance with the standard schemes the plan of ***additional laboratory-instrumental examination of a patient with chronic pleural empyema:***

1. Clinical blood test.

2. Clinical urine test.
3. Biochemical analysis of blood.
4. Coagulogram.
5. Study of blood electrolytes.
6. Immunological tests.
7. Bacteriological study of sputum, bronchial washings, abscess cavity content.
8. X-ray examination of the chest.
9. Spirography.
10. Fibrobronchoscopy.

Characteristic pathological changes.

1. *Clinical blood test*: moderate anemia, leukocytosis with neutrophilosis, left shift of leukocyte formula, increase of ESR.

2. *Clinical urinalysis*: changes are nonspecific – presence of signs of toxic nephropathy: albuminuria, cylindruria.

3. *Biochemical blood test*: pronounced hypoproteinemia, dysproteinemia.

4. *Coagulogram*: disturbances of blood coagulation tending to hypercoagulation with a decrease in the time of blood coagulation, increased fibrinogen level.

5. *Study of blood electrolytes:* hyperkalemia is observed, what is caused by the destruction of tissues and formed blood elements.

6. *Immunological tests*: characteristic reduction of indicators of reactivity of the organism.

7. Bacteriological study of sputum and content of a residual cavity allows to establish inflammatory pathogens and determine the sensitivity of the latter to antibacterial drugs.

8. *X-ray examination of the chest organs*: X-ray examination in case of chronic pleural empyema is highly significant, allows to verify the diagnosis accurately and determine immediate therapeutic approach. The most informative is polypositional radiography, which allows to localize injuries, to determine the amount of pus in the cavity precisely, to detect pathological changes in the pulmonary parenchyma, to find a point for adequate drainage of the residual cavity. *Tomography* allows to answer the above mentioned questions.

Rather informative method of research is *pleurography* in three views. It allows to evaluate the size of a residual cavity, nature of its walls, presence of sequesters and fibrinous layers. During pleurography in the position on a healthy side, the areas of the bronchial tree are often contrasted, what is very important for further therapeutic measures (temporal endobronchial obstruction, optimization of lavage regimen of the pleural cavity).

Bronchography allows to evaluate the condition of the bronchial tree, to determine the localization and character of bronchopleural fistulas, determine a cause of the chronic process (bronchiectases, chronic abscess, etc.). Bronchial signs of significant changes in the lungs are:

1) presence of "empty space" through non-contrasted bronchi in collapsed lung departments;

2) coaptation of bronchi with a decrease in the angles of their branching;

3) various types of deformations, flexures of the bronchial tree, often with bronchiectasis formation.

9. *Spirography* – low indicators of VCL, reduction of compensatory lung capacities, decrease of oxygen saturation of arterial blood.

10. *Fibrobronchoscopy:* provides information on the degree of severity of the inflammatory process in the tracheobronchial tree, allows to determine an opening of the draining bronchus (in presence of destruction in the pulmonary tissue). The resolution of bronchoscopy increases with the introduction of stain (water solution of methylene blue) into the pleural cavity in lying position on a healthy side. This allows to determine which bronchi are involved in drainage of the destruction area of pulmonary tissue, what is very important for planning the level of temporary endobronchial obstruction.

Differential diagnosis is made between osteomyelitis of the ribs, sternum and spine, chronic lung abscess, lung cancer, echinococcus and pulmonary tuberculosis.

Treatment of a patient with chronic pleural empyema. At present, in the treatment of chronic pleural empyema, a combination of surgical treatment methods aimed at the evacuation of purulent contents from the pleural cavity, and conservative therapy, which is considered as a major part of preoperative preparation and postoperative management of patients, is used. When choosing treatment methods for chronic pleural empyema it is worth to remember that conservative treatment rarely results in recovery. Special difficulties appear in the case when a bronchial fistula opens into the residual cavity. Only surgery can provide the success of treatment in such cases.

Pathogenetically grounded ***conservative therapy*** is focused on the control of three major pathological factors – suppuration, losses and resorptions. It is impossible to treat patients with chronic pleural empyema only by local treatment (surgical methods of sanitation) without influence on the entire organism, without normalization of the dysfunctions of organs and elimination of occurring complications, without the therapy of primary and concomitant diseases.

Complex therapy is administered to such patients, and includes etiological, pathogenetic, symptomatic and local treatment. The intensity of the treatment and significance of its components varies depending on the general condition of a patient, presence of concomitant diseases and complications, on the stage of purulent process and nature of empyema itself.

An important etiotropic method of therapy is the administration of antibiotics, sulfanilamides and antiseptic drugs. Antibiotic therapy should include 2-3 drugs, which influence pathogenic aerobic and anaerobic microorganisms. Antibiotic therapy is supplemented by the administration of Metronidazole (Trichopol) at a dose of 1.5-2 g / day, because this drug has high bactericidal activity against anaerobic bacteria.

Pathogenetic therapy includes administration of anti-inflammatory drugs (NSAIDs), bronchodilators (Lasolvan, ACC, Bronchobru, Fluimucil), taking measures for correction of disturbances of protein (transfusion of fresh frozen plasma, amino acids), mineral (infusion of crystalloid solutions), carbohydrate metabolism (infusion of glucose solution 5%); administration of oxygen therapy (5-7 liters per minute), detoxification treatment (sorbilact, rheosorbilact, reamberine).

In the complex treatment of the patients with pleural empyema, adequate nutrition with high content of proteins and vitamins is very important. To improve appetite, the administration of hydrochloric acid, acidine-pepsin, tincture of magnolia-vine, Chinese ginseng, pantocrine is recommended. In debilitated patients, anabolic hormones (retabolil 50 mg, Superanobolon 1-2 ml IM once a week, Nerobol 0,005 g t.i.d. per os, etc.) are administered.

Methods of operative treatment and indications for them. Surgical treatment is used only when conservative treatment is ineffective due to inflexibility of a chest wall, in case of fixation of a collapsed lung by commissures, in case of a large bronchial fistula, open into the pleural cavity, and in weakened, debilitated patirnts with decreased regenerative potential.

The main task of surgical treatment is the elimination of a residual cavity and closure of the pleurobronchial fistula.

The main methods of local preparation for radical operations are the methods of pus removal from the pleural cavity and its sanation. Depending on the size of the cavity, pus removal can be performed both by punctures and by drainage. Drainage of empyema cavity is better because it allows to establish constant active aspiration of purulent contents and, if necessary, continuous intermittent irrigation of a purulent cavity with solutions of antiseptics.

Recently, ***thoracoscopic techniques*** have been widely used, they allow to perform pus aspiration, washing of the empyema cavity, destruction of intrapleural encapsulations, removal of pseudomembranes from the visceral pleura, destruction of adnations between pulmonary pleurae, directed drainage of the empyema cavity. Electrocoagulation of a fistula or (in case of

its large diameter) temporary obturation of the bronchi are performed for the patients with bronchopleural fistulas.

In case of ineffectiveness of minimally invasive methods of treatment, ***surgical interventions*** conditionally divided into several groups, are performed.

1. *Restorative operations* (pleurectomy and lung decortication).

2. *Resection-restorative operations* (pleurectomy and decortication of the lung with lobectomy, segmentectomy, sphenoidal or saggital resection).

3. *Resections* (pleuropneumonectomies).

4. *Plastic and reconstructive surgeries* (thoracoplasty and muscular plastic surgery with resection of the lung or with plastic operations on the bronchial stump).

5. *Obturation and reamputation of a bronchial stump* from transpleural, transsternal, contralateral or transmediastinal accesses.

6. *Thoracotomy with sanitation and drainage of the empyema cavity.*

*In the postoperative perio*d, as a rule, conservative therapy, which has been started earlier, is continued. The main therapeutic tasks in the postoperative period are: to provide complete ventilation of the lungs and prevention of hypoxia; correction of volemic disorders and restoration of adequate hemodynamics; prevention of hypercoagulation and infectious complications.

Possible *postoperative complications*:

1) postoperative bleeding;

2) pneumonia;

3) suppuration of postoperative wound;

4) relapse of a bronchial fistula;

5) development of acute postoperative empyema.

After undergoing the course of treatment in a hospital, patients with chronic pleural empyema are mandatorily observed by a pulmonologist at the polyclinic at the place of residence. Operated patients are observed also by a surgeon.

III.3. INJURIES OF THE RESPIRATORY SYSTEM (LUNGS, PLEURA, BRONCHI, TRACHEA, LARGE BLOOD VESSELS, LARGE LYMPHATIC DUCT)

In the literature of the last decade, a significant number of works is devoted to the diagnosis and treatment of traumatic injuries of chest cavity organs. In these works special attention is paid to combined closed chest injuries, which differ by specifics and severity of clinical course (Florikyan A.K, 1998).

Published by various authors, statistical data show that still a very high mortality caused by severe thoracic and combined injuries is observed (Bisenkov L.M., 2004, Tishchuk I.A., 2005). So, from 100,000 US residents who annually die from injuries, almost 25% are the people with chest injuries. Fatal cases, making 4-42% of isolated chest injuries, reach 30% for combined injuries (Hank I., 2003). Similar data are presented in the work by V.V. Lebedev et al., (2003), according to which mortal consequences of the isolated trauma make 1,5-2%, and of the combined one – 28,6%.

As the research has shown, most of the injured die after admission to medical institutions (Florikyan A.K., 1998). Moreover, fatal consequences occur in the first 6 hours after trauma, and the most critical for the injured are the first four days. From the total number of the patients, survived after severe combined injury, about a half die later from respiratory failure caused by *pneumothorax or hemothorax, – main severe complications of traumatic injury of the thoracic skeleton and chest organs* (Bisenkov L.M., 2004).

Hemothorax

Hemothorax (HT) *– is an accumulation of blood in the pleural cavity.* The cause of it is a closed or open trauma of the chest of different etiology and volume with injuries of vessels of the chest wall (intercostal, internal chest artery), organs (lungs, heart, diaphragm), large vessels (aorta, hollow veins and their intradermal branches).

Pathogenesis – an internal bleeding, which results in the accumulation of blood in the pleural cavity and compression of a lung on the injured side with a possible dislocation of the mediastinal organs, leading to the development of acute blood loss and anemia, acute respiratory and cardiac insufficiency.

Classification of hemothorax.

1. *Depending on the volume* (P.S. Kupriyanov, 1950):

1) *small* – up to 500,0-600,0 ml of blood within the costodiaphragmatic recess (VIIth intercostal space, angle of scapula);

2) *average* – up to 800.0-1000.0 ml of blood (up to the IVth rib anteriorly, the Vth intercostal space, middle of scapula posteriorly);

3) *large* – up to 1000,0 -1200,0 ml of blood (up to the IInd rib anteriorly);

4) *total* – up to 1500.0 ml of blood and more (up to the apex of the lung).

2. *By etiology*: traumatic (including gunshot injury); pathological (caused by various diseases); postoperative.

3. *By dynamics*: increasing; non-increasing.

4. *In case of complications:* clotted; infected.

5. *By the period of formation* (M.M. Abakumov):

stage I – formation (the 4-5th day from the moment of injury – gelatinous mass of fibrin, blood elements, loose adhesions);

stage II – development of clotted hemothorax (the 5-30th day – clot retraction, separation of liquid and dense blood, appearance of new connective tissue);

stage III – suppuration (the 30-60th day, this stage does not take place during intensive antibiotic treatment);

stage IV – fibrothorax (the 90th day – pleuropneumocirrhosis with severe violations of the functions of external respiration).

Clinical picture. During an interview with a patient, complaints depend on the size of hemothorax. In case of a *small hemotho*rax, the symptoms are reduced to pains in the injured area and some limitation of respiratory movements, mild clinical manifestations of acute hemorrhage. Small hemothorax (up to 100-200 ml) in most cases is not clinically diagnosed, later it usually resolves with the formation of pleural adhesions. In case of *moderate hemothorax* cough, shortness of breath, chest pain, delay in the act of breathing on the affected chest side, clinical manifestations of acute hemorrhages of moderate severity are observed. In case of *large and total hemothorax*, among the complaints of patients, the symptoms of massive intrapleural bleeding, which correspond to acute blood loss of severe degree: pronounced weakness, dyspnea, dizziness at rest, cold clammy sweat, short-term loss of consciousness and visual impairments; anxiety, pain in the chest on the injured side, delay in the act of breathing in the chest on the injured side, possible protrusion of intercostal spaces, are observed.

Patients with chest injuries even without objective signs of penetrating wounds are examined in a sitting position and should be hospitalized.

In most cases, HT develops acutely immediately after an injury.

Objective status. General condition of a patient may vary from relatively satisfactory to extremely severe, depending on the HT size. Clinical symptoms depend on the intensity of internal bleeding, compression or dislocation of the lungs and mediastinal organs. The root and deep lung lacerations are accompanied by massive bleeding, surface injuries – insignificant.

Status localis. In case of small and moderate hemothorax, moderate pallor, delay of an affected part of the chest in the act of respiration are observed, by auscultation weakening of breathing and dull percussion sound are heard on the injured side. In case of the large and total hemothoraces: pallor of skin and mucous membranes, tachycardia, shortness of breath, decreased blood pressure, what shades the picture of the main damage. Cyanosis of the skin, protrusion of intercostal spaces, cough, sometimes bloody, difficulty breathing, noticeable delay in the act of breathing in the chest; by percussion dullness of percussion sound is heard, breathing cannot be heard. The degree of anemia depends on the amount of blood loss.

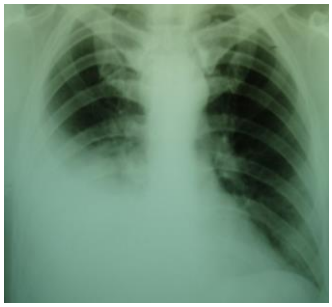

Fig. 72. Dextral post-traumatic hemothorax (after a closed chest injury)

Leading symptoms and syndromes – chest pain, hemorrhagic syndrome, shortness of breath.

Additional study methods:

1. Clinical blood test.
2. Coagulogram.
3. Radiography of the chest organs.
4. Ultrasound examination of the pleural cavities.
5. Computer tomography of the chest.
6. Videothoracoscopy.
7. Determination of blood group and Rh affinity.
8. CBD.

Characteristic pathological changes.

1. *Clinical blood test* – increased leukocytosis, ESR, decrease of hemoglobin.

2. *Coagulogram* – manifestations of hypocoagulation with continued bleeding, signs of hypercoagulability with clotted hemothorax.

3. Radiography of the chest organs – detects shadows in the pleural cavity with a corresponding horizontal level during moderate and large HT (Fig. 72), total shadow of the entire pulmonary field on the injured side during total HT.

4. *Ultrasound examination of the pleural cavity* (Fig. 73). Pathognomical ultrasound sign of pleural effusion in hemothorax, on which the search is focused during examination of post-traumatic patients, are thrombotic clots. They are represented by medium or hypoechoic regions of indefinite shape and various sizes, with often rough, partially indistinct boundaries, which in

5. *Computer tomography of the chest organs (Fig. 74).* A homogeneous shadow in the pleural cavity with a lung collapse on the injured side is observed.

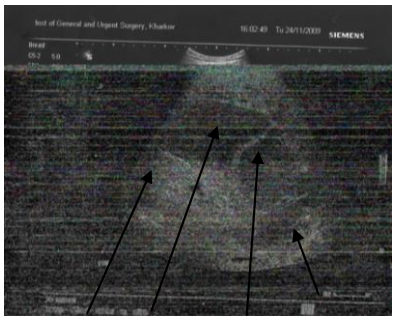

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Fig. 73. Ultrasound examination. Right-sided post-traumatic hemothorax (after closed chest injury):
1 – collapsed lung;
2 – anechoic liquid content;
3 – fibrinous echogenic thread;
4 – parietal echogenic thrombotic clot

6. Videothoracoscopy. Indications for videothoracoscopy are: lung injury, complications of hemo- and pneumothorax, suspected injury of the pericardium, heart and vessels of the chest wall, as well as thoracoabdominal injuries. In case of low localizations of chest wounds on the left, it is recommended to use thoracoscopy to evaluate the condition of the diaphragm. Thoracoscopic examination in this category of patients includes an examination of the pleural cavity, evacuation of blood, clots. In the presence of both air and blood in the pleural cavity, the latter forms a horizontal level – in this case, the diagnosis of ***hemopneumothorax*** is made. In the absence of active bleeding, an operation ends with Bulau drain of the pleural cavity.

Differential diagnosis of HT is performed between pneumothorax of different etiology, intercostal neuralgia, fracture of ribs, post-traumatic pneumonia, pleurisies.

Treatment. The first medical aid at the pre-hospital stage in case of HT includes application of aseptic bandages using large sterile bandages (included in B-1 dressing kit), application of an occlusive dressing in case of an open hemothorax, administration of pain relievers, cardiovascular and respiratory analeptics. In case of pulmonary arrest due to asphyxia,

a mouth should be cleaned from mucus, blood and soil, artificial respiration is induced using an S-shaped tube or an airway with a valve (AW).

Choice of therapy approach. Patients with hemothorax should be immediately hospitalized at a surgical hospital (preferably with a specialized polytrauma department). *Conservative treatment*, the basis of which is a pleural puncture, is administered for small hemothorax in combination with haemostatic therapy under clinical and X-ray control. *Indications for surgical treatment are:*

1) ongoing bleeding;

2) recurrent accumulation of blood after its aspiration by pleural puncture; blood discharge through a pleural drainage in a volume more than 500 ml in 2-3 hours;

3) clotted large hemothorax, which prevents a lung spread;

4) injuries of vital organs.

Conservative treatment: analgesics, hemostatic (in case of mild blood loss) and substitution therapy with urgent hemotransfusion (in case of moderate and severe blood loss), immunocorrection, symptomatic therapy, general and local antibiotic therapy for prevention and treatment of contamination of HT, administration of fibrinolytic drugs for prevention and treatment of clotted hemothorax.

Puncture of the pleural cavity during hemothorax is a therapeutic and diagnostic procedureth and is performed in the VI-VIIth intercostal space along the posterior axillary line (in a sitting position or in a lying position) with strict adherence to asepsis rules. Blood from the pleural cavity is completely removed and at the end, a solution of a broad spectrum antibiotic is introduced.

During a pleural puncture in patients with hemothorax, the following **tests** are performed:

1) *Ruvilua-Gregoire test*: if the blood in a test tube or a basin coagulates, then it is a sign of ongoing bleeding, blood incoagulability is a sign of stopped bleeding;

2) *Efendiyev sample*: 5-10 ml of blood from the pleural cavity and an equal amount of distilled water are poured into a test tube, what results in blood hemolyzing; if the hemolysate is evenly colored ("laky" blood) – the blood is not infected, if it reveals a cloudy suspension, flakes – it is infected;

3) *Petrov sample*: contents of the pleural cavity are poured into a test tube and diluted fivefold with distilled water and the obtained mixture is shaken; in the absence of suppuration there will be a transparent hemolyzed liquid in the test tube; if the liquid gets muddy, this

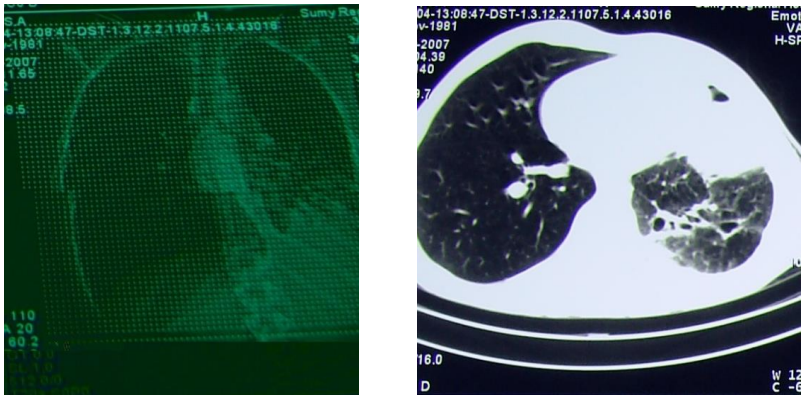

Fig. 74. CT – left-sided small post-traumatic hemothorax (after a closed chest injury). Arrow indicates hemothorax

indicates that the content is suppurating.

Surgical treatment. *Closed* (various methods of drainage of the pleural cavity) and *open* (thoracotomy) methods of surgical interventions are used.

In case *of moderate and large* hemothorax, drainage of the pleural cavity is performed at the traditional place, in combination with hemostatic and substitution therapy under clinical and X-ray control. **2 types of drainages** are used: *passive (Bulau) and active (aspirational).*

In case of *unarrested intrapleural bleeding, thoracotomy is indicated* for detection of the source of bleeding and its reliable elimination. ***Indications for thoracotomy*** are also: wounds of the lungs and the heart; suspected wound of the heart or a large vessel; injury of the large bronchi or the esophagus; tense hemopneumothorax, which is not eliminated by punctures and drainage; foreign bodies in the pleural cavity.

During thoracotomy hemostasis is achieved, the pleural content is evacuated; an operation ends with the drainage of the pleural cavity.

The diagnosis of a clotted hemothorax is made according to the clinical picture (shortness of breath, pain, fever) and typical radiological picture (presence of a homogeneous and intense opacification on the injured side of the lower parts of a pulmonary field or non-homogeneous opacification with fluid levels). *Thoracotomy and an evacuation of clotted hemothorax*, performed in the first 5 days, prevent the development of pleural empyema, contribute to the most adequate restoration of the functional capabilities of the lungs.

At present, in case of chest injuries ***minimally invasive surgical operations*** are used more and more often – videothoracoscopic operations: lung resection in case of its injury, complicated with hemo- and pneumothorax, ligation of large vessels of the chest wall in case of their injuries, etc.

Possible ***complications of hemothorax***: infection; fibrothorax.

Pneumothorax

Pneumothorax (PT) *is a collection of air in the pleural cavity as a result of the violation of a sealing capacity of the lung or chest damage.*

Classification:

1. *Depending on an injury of anatomical structures*, the following are distinguished:

1) *closed pneumothorax* – air enters the pleural cavity from a damaged lung;

2) *open pneumothorax* – air enters the pleural cavity from the external environment during inhaling via the injured chest wall and parietal pleura, and during exhaling it is pushed outside from the pleural cavity;

3) *valvular pneumothorax* – air enters the pleural cavity from external environment during inhaling through a damaged chest wall or a bronchial wound, and during exhaling, it is not pushed outside from the pleural cavity, intrapleural compression increases, and displacement of mediastinal organs is observed.

2. *Depending on the mechanism of occurrence:*

1) *traumatic pneumothorax* – occurs as a result of a trauma, an injury of the lung or thoracic wall;

2) *spontaneous pneumothorax* – develops as a result of a pathological process in the lung tissues (most often during bullous emphysema of the lungs);

3) *iatrogenic*:

a) *accidental* (as a result of various manipulations – thoracocentesis, subclavian vein catheterization, etc.);

b) *"natural, expected"* (during thoracotomy, thoracoscopic examination);

c) *therapeutic* (previously widely used in the treatment of pulmonary tuberculosis).

3. *Depending on the volume:*

1) *small* – in the pleural cavity there is a small amount of air, the lung is collapsed by 1/3 volume;

2) *moderate* – the lung is collapsed by 1/2 volume;

3) *total* – the lung is fully collapsed.

Clinical picture. When a patient complaints of a sudden pain in the chest on the injured side, feeling of air hunger, shortness of breath, sometimes dry heavy cough. Development of pneumothorax is acute.

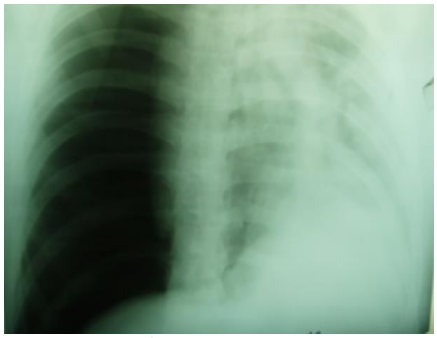

Fig. 75. Chest X-ray. Left tension pneumothorax (collapse of the lung for 2/3, displacement of mediastinal organs to the right)

Objective status. Cyanosis, tachycardia, in rare cases – fall of blood pressure. Delay of one half of the chest during breathing. Sometimes extrusion of an affected half of the chest occurs. On the injured side palpatory-defined voice tremor is absent, the box percussion sound is determined (in case of large pneumothorax – timpanitis), breath sounds are weakened or not audible.

The severity of symptoms depends on the type and degree of pneumothorax.

Closed traumatic pneumothorax develops if the air ingress into the pleural cavity through the wound of the chest wall or damaged bronchi has been short-termed. In case of a small collection of air in the pleural cavity, clinical symptoms are poorly evident. Closed pneumothorax, characterized by a collection of large amounts of air in the pleural cavity, causes severe ventilation disturbances due to a collapsed lung and displacement of mediastinal organs.

An open traumatic pneumothorax occurs in case of a wound opening in the chest wall, through which the pleural cavity is intercommunicates freely with external environment, moreover during breathing air is inhaled into the pleural cavity, and when exhaled it goes out. In this case, the patient's condition is heavy, which is caused by the collapse of a lung and its exclusion from the act of breathing, as well as by displacement of the mediastinum to a healthy side and change of its location with each breathing in and out (*Jakobsen syndrome – "mediastinal flutter"*). During open pneumothorax, cyanosis, dyspnea (sometimes up to 40 – 50 respiratory movements per 1 min) are observed. Pulse is rapid, weak, BP is lowered. During coughing, blood with air bubbles flows out from the wound.

In case of a small injury of the chest wall, half-covered with soft tissues, as well as in case of closed chest trauma with lung injury, a ***valvular pneumothorax*** may occur. Intraperitoneal pressure in this case exceeds atmospheric pressure. Displacement of the mediastinum leads to significant disturbances of blood circulation, and compression of a lung – to sharp breathing difficulty. The condition of a patient with valvular pneumothorax is extremely severe; *acute dyspnea, cyanotic face, increase in pulse rate, increasing subcutaneous emphysema* in the area of the chest, back, neck, face, abdomen, and sometimes limbs are observed.

Leading syndromes and symptoms – chest pain, dyspnea.

Additional study methods.

1. Clinical blood test.
2. Coagulogram.
3. Radiography of the chest organs.
4. Computed tomography of the chest organs.
5. Videothoracoscopy (according to indicators).

Characteristic pathological changes.

1. *Clinical blood test* – increased leukocytosis, ESR.
2. *Coagulogram* – symptoms of hypercoagulation.
3. *Radiography of the chest organs*. X-ray (Fig. 75) reveals massive air accumulations in the pleural cavity, a collapsed lung, displacement of mediastinal organs to the opposite side, gas collections in the mediastinal tissues, thoracic walls, neck, etc.

4. Computed tomography of the chest organs reveals accumulation of gas in the pleural cavity, a collapsed lung, as well as the displacement of mediastinal organs to a healthy side.

Differential diagnosis of pneumothorax is made between hemothorax, intercostal neuralgia, rib fracture, posttraumatic pneumonia, pleurisies.

Treatment. ***The first medical aid for closed PT at the prehospital stage***: for anesthesia, Analgin is administered, in case of significant level of pain – morphine, Omnopon; oxygen therapy is performed. For suppression of cough reflex, Codeine, Libexin, Tusuprex are administered. ***In case of an open PT***, the first aid includes immediate adjustment of a hermetic (occlusal) bandage on a chest wound with the help of an adhesive plaster or an adhesive pad from the emergency medical packet, which is fixed to the edges of a wound by an adhesive and gauze (roll gauze) dressing. An injured person should be provided with oxygen inhalation, introduction of anesthetics (morphine, etc.), antibiotics. ***In case of a valvular PT***, *externally* open, a hermetic bandage of an adhesive plaster is applied on a wound of the chest wall and the patient is transferred to a hospital. In case of a progressive deterioration in the patient's condition (increased shortness of breath, sudden fall of blood pressure, etc.), caused by a ***valvular pneumothorax***, opened *inside* (no defects of the thoracic wall), an urgent pleural puncture is administered in the second intercostal space along the midclavicular line (in the absence of a doctor, this manipulation may be performed by a paramedic) and leaving of a thick needle in the pleural cavity during urgent transportation to the hospital.

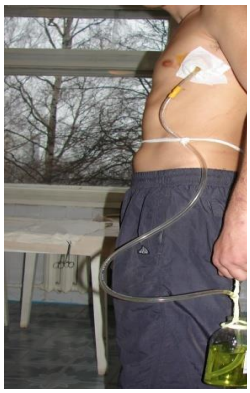

Fig. 76. Drainage of the left pleural cavity by the Bulau method at the Bogush point in a patient with pneumothorax

A patient with symptoms of a pneumothorax should be urgently transferred to a surgical hospital, preferably to a specialized department of shock and polytrauma.

In a hospital for a closed PT, a doctor performs a pleural puncture in the IInd intercostal space along the midclavicular line or at Bogush point (in the IInd or IVth intercostal space along the anterior axillary line), with further aspiration of air. In case of the inability of lung spreading, the pleural cavity is drained according to Bulau method using a trocar (Fig. 76).

For open PT, sharp debridement of the wound with sealing of a chest wall defect by layered closure or plasty with surrounding tissues and drainage of the pleural cavity in the IInd intercostal space along the midclavicular line with a constant active air aspiration are performed, and if the latter is impossible, – Bulau drain is performed. It is necessary to monitor carefully the drainage, to change the antiseptic solution daily, into which a free end of the drainage is immersed.

In case of a significant damage of the lung tissue, inability to spread the lungs with the help of aspiration or drainage, as well as during intra-pleural bleeding, *thoracotomy*, exploration or management of the lung tissue (bronchus), its sealing are made. Sometimes resection of a damaged part of the lung is necessary.

As in case of hemothorax, at present, more and more successfully for the treatment of pneumothorax, ***minimally invasive surgeries are performed – videothoracoscopic operations.***

Possible ***complications of pneumothorax***: development of pleural empyema; development of pleurocardial shock.

Chylothorax

Chylothorax (CT) *occurs as a result of an injury of the chest lymphatic duct or large lymphatic ducts which are its tributaries, what is accompanied by lymphorrhea into the pleural cavity.*

Classification.

1. *By etiology*: traumatic and nontraumatic Chylothorax (Perelman M.I. et al., 1984).
2. *By localization* (Rajkevich M.P., 1980):
a) right-sided (occurs during a trauma below the IIIrd-IVth thoracic vertebrae);
b) left-sided (observed during trauma above IIIrd-IVth thoracic vertebrae);
c) bilateral (extremely rare).
3. *By the course of chylothorax* (Shchiglinsky G.M., et al., 1975):
a) sharp;
b) subacute;
c) chronic.

Clinical picture. During an interview in case of an *acute* course of chylothorax a patient *complains of* pronounced dyspnea, pain on the affected side, pallor of the skin; in case of *subacute* course of chylothorax, lymph in the pleural cavity accumulates within 5-9 days after an injury, shortness of breath and chest pain gradually increase; during a *chronic* course, clinical symptoms are not pronounced.

In some cases, during a closed injury of the lymphatic chest duct the lymph flows into the mediastinal cellular tissue with the formation of "*mediastinal chyloma*". Due to the bursting of a "mediastinal chyloma" into the pleural cavity, a shock-like picture with symptoms of intra-pleural tension develops.

The history of the disease indicates the presence of traumatic injuries of the chest, in most cases the development of chylothorax is acute.

Objective symptoms depend on the rate of accumulation of lymph in the pleural cavity, compression or displacement of the lungs and mediastinum.

Status localis. A noticeable delay of an affected part of the chest in the act of breathing is observed, weakening or absence of breathing and dull percussion sound, tachycardia, dyspnea, fall of blood pressure.

Leading symptoms and syndromes – *chest pain, dyspnea.*

Additional study methods:

1. Clinical blood test.
2. Biochemistry of blood (protein).
3. Coagulogram.
4. Radiography of chest organs.
5. X-ray contrast study of chest lymphatic duct.
6. Computed tomography of the chest.
7. Diagnostic puncture of the pleural cavity.

Characteristic pathological changes.

1. *Clinical blood test* – increased leukocytosis, ESR.
2. *Blood protein* – hypoproteinemia.
3. Coagulogram – signs of hypercoagulation.
4. *Radiography and CT* of chest organs – signs of hydrothorax, with "mediastinal chyloma" – an increased opacity of the mediastinum. In case of the bursting of chylomedistinum out into the pleural cavity, the reduced opacification of mediastinum is observed, increase of the level of free fluid in the pleural cavity.
5. *X-ray contrast study* of the chest lymphatic duct is essential for the diagnosis of a topical damage of the chest lymphatic duct. At present, the method of choice for lymphaductography is an antegrade route of administration of Lipiodol Ultrafluid or Etiodol.
6. During *the puncture of the pleural cavity*, an incoagulable, odorless, milky color opalescent fluid is obtained.

In doubtful cases, spetial agents are used (a common lipstick, proposed by R. G. Klepsner, J. E. Berry) at a rate of 1 g per 250 g of butter. This mixture in the form of a sandwich is proposed to a patient as a meal. After 2 hours after ingestion, the pleural cavity is punctured and in case of a coral stain of the punctate, the diagnosis of chylothorax becomes definite. During settlement 2 layers are formed: the upper one is thick, the bottom one – liquid pink. The

obtained fluid has an alkaline reaction, specific gravity – 1012-1028, contains 6% fat and more, up to 4-9% protein; by microscopy, drops of fat and lymphocytes are detected. Rivalta test is positive.

Differential diagnosis of chylothorax is made between pneumothorax, hemothorax, rib fracture, posttraumatic pneumonia, pleurisy.

Treatment. Patients with chylothorax should be immediately referred to a hospital. There are two methods of treating chylothorax: conservative and operative.

Conservative treatment – removal of chylous pleural fluid by the puncture method before the onset of thrombosis of a chest lymphatic duct and restoration of its losses. This method requires long-term treatment – from several weeks to several months. Complex therapy includes plasma transfusion, hemotransfusion, infusion therapy, restriction of fat consumption by patients, restriction of fluid intake per os. In order to reduce venous pressure, diuretics, cardiac glycosides were prescribed.

Operative treatment*. Indications for surgery:*

1) trauma of the chest lymphatic duct, revealed during intrathoracic operation for a closed or penetrating chest wound;

2) ineffectiveness of conservative therapy for 2 - 3 weeks;

3) exhaustion of a patient, which begins after medication, substitution therapy;

4) daily loss of hilus in adults of 1500-2000 ml / h, in children – 100 m / year for a year of life for 5 days;

5) clotted fragmented hemochylothorax.

Operative interventions are divided into *palliative and radical*.

Palliative surgical intervention – tamponade of a duct or a suspicious place of damage of a lymphatic duct by a muscle flap or a pedunculated flap. It is performed in case of a severe condition of a patient.

Radical surgeries:

1) ligation of a chest lymphatic duct;

2) application of the duct opening "end to end";

3) implantation of a thoracic duct into one of the large veins (unpaired, brachycephalic, etc.).

Possible ***complications of chylothorax***: development of pleuro-cardial shock; development of a pleural empyema.

Patients who were treated conservatively and operated for hemothorax undergo regular medical check-ups of a surgeon and a pulmonologist.

The prognosis for spontaneous pneumothorax, if treatment is started on time, is usually favorable. The patients operated for chylothorax undergo case follow-ups by a surgeon and a pulmonologist.

III.4. CARDIAC INJURIES

Traumatic cardiac injuries, taking the second place by frequency after lung injuries, remain one of the actual and challenging problems of urgent surgery and resuscitation.

The most difficult diagnosis is a closed heart injury, many aspects of which have not been resolved yet. According to the literature, a closed heart injury is observed in 6.4-64% of cases. Larger variations in statistics are explained, first of all, by the fact that in case of other severe concomitant injuries of the body, some types of heart trauma are not detected.

Although the proportion of open heart injuries in the structure of penetrating chest wounds in peacetime is relatively low and ranges from 3.7 to 16%, the absolute number of injuries with heart wounds amounts thousands. Current tendencies of the increasing crime in the country, increase of wound traumatism and frequency of military weapon wounds should be taken into account, what requires a deeper study of the issues of their diagnosis and surgical treatment.

An essential characteristic of heart injuries is a high risk of death of the injured due to untimely treatment and maintenance of the capacity for work in the persons who survive after timely and qualified medical care.

Overall mortality due to cardiac wounds continues to stay high, making out 8.3 – 40%, and in the part related to the injured – up to 90%.

Young age of the overwhelming majority of people with heart injuries indicates a high socio-economic significance of this problem, what does not make surgeons indifferent to it. The main cause of death of patients after heart injuries in the acute period is bleeding and cardiac tamponade. Surgical tactics and technique of surgical intervention in these situations largely determine not only the survival of a patient at the moment, but also influence the course of the postoperative period and development of a number of complications.

Clinical anatomy and physiology of the heart. The heart with its larger part (2/3) is located in the left half of the anterior mediastinum, and with its the smaller one is in the right part. Its anterior-posterior axis is directed diagonally from top to bottom, dextrosinistrally and from back to front. A distinction is made between ***the base of the heart*** (*base cordis*), *apex* and *four surfaces*: *anterior* (sternocostal), formed by the right atrial appendage, right ventricle together with the beginning of the pulmonary artery and the anterior interventricular sulcus and a part of the left ventricle; *lateral* ones (pulmonary), formed from the left by the posterior wall of the left atrium and the left ventricle, and on the right by the posterior wall of the right atrium; *the lower*, or diaphragmatic surface, which is formed mainly by the left and partly by the right ventricle.

The right border of the heart extends archwise from the IIIrd to the lower edge of the Vth rib cartilage at a distance of 2-2.5 cm from the right sternal line. It is formed by the superior vena cava and the right atrial appendage. The left border of the heart at the level of the IInd rib extrudes by 2 cm from the left edge of the sternum, and then along the IIIrd – Vth ribs runs by 1-1.5 cm medially to the midclavicular line. The pulmonary artery, the left atrial appendage and the aortal arch form it. The lower border of the heart extends from the Vth rib cartilage on the right to the Vth intercostal space on the left and does not reach 1 cm to the left midclavicular line. It is formed by the left and partly by the right ventricle.

The right and left atria are separated from each other by the atrial septum (*septum interatriale*), in the center of which is an oval fossa (*fossa ovalis*).

The right and left ventricles are separated from each other by a well-developed interventricular septum in which a difference is made between the muscular part (*pars muscularis*) and a small upper part, layered from both sides by the endocardium, – membraneous (*pars membranacea*).

The right atrium, which the upper and lower hollow veins enter, is connected to the right ventricle by the atrioventricular orifice. Here lies the right *atrioventricular valve (valvula tricuspidalis).* The left atrium, in which the pulmonary veins enter, is connected to the left ventricle by the left atrioventricular orifice. *The mitral valve (valvula bicuspidalis)* is located in it. The narrowing of the orifices or insufficiency of the valves indicate acquired and congenital heart defects.

The walls of the heart consist of three layers: the endocardium, the myocardium and the epicardium. The most developed middle layer is the myocardium, which consists of two layers in the atria (superficial and deep), and of three layers – in the ventricles (superficial layer consisting of longitudinal fibers, middle circumferential layer and internal longitudinal one).

The main source of *heart blood supply* is right and left coronary arteries. *The right coronary artery (a. coronaria dextra)* branches from the aorta. It begins between the arterial cone (conus arteriosus) of the right ventricle and the right appendage. Next, in the coronary sulcus, on the border between the right atrium and the ventricle, it runs to the posterior surface of the heart, bifurcating the posterior interventricular branch (*r. interventricularis posterior*) near the posterior interventricular sulcus. The latter reaches the apex of the heart along this sulcus. The right coronary artery territory is the right atrium, the posterior wall of the left ventricle, the

interatrial septum, the posterior third of the interventricular septum, papillary muscles of the right ventricle, the posterior papillary muscle of the left ventricle. *The left coronary artery (a. coronaria sinistra)* emerges from the ascending aorta, is located on the border between the left atrium and the ventricle behind the pulmonary trunk, and then passes between the left appendage and the wall of the left atrium. Next to the aorta, it is divided into two branches: the anterior interventricular (*r. Interventricularis anterior*) and circumflex (*r. circumflexus*). The anterior interventricular artery anastomose with the posterior one in the region of the heart apex. The left coronary artery territory is the left atrium, a part of the anterior wall of the right ventricle, the anterior and greater part of the left wall of the left ventricle, the interventricular septum. *The veins of the heart* merge into the coronary sinus (*sinus coronarius*), which enters the right atrium.

Lymphatic vessels accompany coronary arteries.

Innervation is carried out by means of nerve plexuses, which are formed from the vagus nerve and the sympathetic trunk. Heart contractions are generated by the autonomous, main heart system. In the normal condition, a disorder occurs in the sinus node, and then an impulse passes through the interventricular routes of the atrium and reaches the atrioventricular node. In the ventricle, the impulse is conducted through the His band via its right and left branches and through the Purkinje fiber network reaches the muscle fibers.

The heart, along with the vessels that branch from it, lies in the pericardial cavity, the reserve cavities of which are called sinuses. The largest of them are: anteroinferior, located at the point of transition of the anterior wall of the parietal layer to the lower (*sinus pericardii anterior*), and transverse, which is limited by the aorta and the pulmonary trunk anteriorly, and by the upper vena cava and the wall of the right atrium – posteriorly. The oblique sinus is located between the left atrium and the posterior pericardial wall. Exactly in these spaces, effusion or blood can be accumulated.

A number of *physiological parameters* are used to characterize cardiac activity.

The volume of blood per minute is the amount of blood passing through the heart to the aorta for 1 min. The minute volume coming to the large and small circles of blood circulation is the same in the absence of heart defects. Its physiological values are 5-6 l / min.

Stroke (systolic) volume is an amount of blood released into the aorta for one contraction. It is calculated by dividing the minute volume of the heart by the number of cardiac contractions. In the normal condition, the value of the stroke heart volume is 60-70 ml.

Cardiac index (CI) is the ratio of the minute blood volume to an area of the body surface. It is calculated according to Du Bois tables, depending on the mass and height of the person. The CI norm is 2.4-4.2 l / min × m^2.

Table 26

Physiological values of pressure in the heart chambers

Heart chambers	Pressure, kPa (mm Hg)	
	systolic	diastolic
Right atrium	0,7 (5)	0
Right ventricle	3,3 (25)	0,3 (2)
Left atrium	1,1 (8)	0,3 (2)
Right ventricle	16 (120)	0,3 (2)

All traumatic heart injuries ***are classified*** into *closed injuries and heart wounds*.

Closed traumatic heart injuries

It is advisable to divide them into four types: *concussion of the heart, cardiac contusion, traumatic myocardial infarction, heart ruptures*. Each of these types of heart trauma has specific mechanogenesis, clinical picture and functional disorders (A.P. Borisenko, A.P. Golikova, E.O. Pisarenko, 1977).

Pathogenesis, severity of traumatic heart injury depend on many factors: condition of the myocardium and coronary vessels before trauma, a phase of cardiac activity at the moment of an injury, age of a patient, etc. However, the force of mechanical trauma in the anteriorposterior direction is the most important, leading to the hemorrhage in different parts of the heart or rupture of its walls. Sometimes these violations are caused by direct force action of the ends of broken ribs and sternum, metabolic disorders, energy changes in the myocardium, and influence of stress reactions on the heart. Morphological changes are diverse during a heart injury.

These are various punctate or more extensive hemorrhages in the muscle, under the epicardium, in the septa, around native heart vessels. All these violations lead to various changes in the fibers of the heart muscle and vessels in the form of swelling, disintegration, necrotic areas, parietal thrombi, etc.

In contrast to the *cardiac concussion in case of a contusion, traumatic infarction, rupture of the heart, anatomical violations of different intensity also occur along with functional disorders.*

In case of ***a cardiac contusion*** as a result of the hemorrhages in its various departments quite rough morphological changes in the fibers of the heart muscle occur in the form of edema, disintegration, ruptures, etc. These morphological changes are manifested by various symptoms within several hours or days after an injury and accompanied by pains in the heart or behind the sternum.

The ***pathogenesis of traumatic myocardial infarction***, which is observed extremely rare, is of particular interest. The disease begins acutely. A few hours and even days after a chest injury above the heart area characteristic pains appear, a collapse is possible. Clinical picture, diagnosis and treatment of traumatic infarction and myocardial infarction of other genesis do not differ significantly. However, transmural traumatic infarction, in contrast to myocardial infarction of nontraumatic origin, has its own localization and is manifested mostly in the region of anterior or anterolateral wall of the left ventricle of the heart, what depends on the mechanism of an injury.

The most severe patients requiring surgical resuscitation measures are the ones with ***heart ruptures***. Most of them die at the place of an injury or during transportation; only the patients with small cardiac ruptures are delivered to a clinic, preventive measures of cardiac tamponade of the heart are applied to them.

Depending on the disruption of continuity of one of its walls, partitions or valves a difference is made between *external and internal* heart ruptures. In case of an *external rupture* of the heart wall, a connection is formed with the adjacent organs of the chest, and in case of an *internal* one – with different heart cavities. As it is shown by the analysis of sectional and clinical observations, sometimes the wall of the heart muscle is not ruptured completely. Therefore, when a diagnosis is made, it is advisable to distinguish *complete, incomplete, external and internal heart ruptures.*

The right half of the heart is more vulnerable than the left one, what can be explained by muscle weakness.

The clinical course of various types of closed heart injury can be divided into *three periods: acute, subacute and rehabilitation* – with various duration for each type of the injury. The most dangerous is an acute period, because sometimes various manifestations of closed heart injury are not diagnosed during this period, what may lead to fatal consequences.

Open traumatic injuries (wounds) of the heart

The classification of heart wounds by W. Schmidt (1961) is used in the clinical picture.

1. Isolated non-penetrating heart wounds.
2. Wounds of coronary vessels (isolated and with wounds of the myocardium).
3. Penetrating heart wounds.
4. Injuries of internal structures (valves, partitions).
5. Multiple heart wounds.
6. Stab heart wounds.

A characteristic feature of open chest injuries in peacetime is a predominance of left-sided wounds, what increases the risk of a heart damage. Entrance perforations of wound canals in case of cardiac injuries are located mainly in the region of a heart projection or in the precordial region. According to I.I. Hrekov, "*the area of possible heart injury*": above – the IInd rib, below – the left subcostal area and the pit of the stomach, on the left – the midaxillary line and on the right – the parasternal line. However, the size and projection of chest wounds do not always give an idea of the direction and depth of a wound canal. The wounds, located parallel to the edges of the ribs, should always warn a doctor, since a deeper penetration of a traumatizing object and a serious damage to the organs may occur.

Hemodynamic disorders accompanying heart wounds. Heart wounds will lead to *hypovolaemia, tamponade and are accompanied by severe violations of cardiohemodynamics*. At the same time a significant increase in the frequency of heart rate occurs, although the rate of blood circulation is slowing down. However, the change of the pulse rate during heart injury is not typical. The state of its strength and changes in the rhythm of a pulse wave, which depend on the degree of the heart compression, are important.

In case of severe wounds of the pericardium and the heart, *profuse intraperitoneal bleeding* often occurs, which may lead to ***hemorrhagic shock***. In many respects, adaptation of a patient to the blood loss determines the changes in the capacity of the venous system (which contains up to 75% of the blood volume in a healthy person). However, the capacities for blood mobilization from the blood pool are limited: in case of a loss of more than 10% of CBV, CVP begins to decrease and venous return to the heart also decreases. A low cardiac output syndrome occurs, what leads to a decreased perfusion of tissues and organs. In response, non-specific compensatory endocrine changes appear. Release of ACTH, aldosterone and ADH leads to retention of sodium, chlorides and water by kidneys during parallel increase of potassium loss and decrease of diuresis. Epinephrine and norepinephrine ejections result in peripheral vasoconstriction. Less important organs (skin, muscles, intestines) are excluded from the bloodstream, and blood supply to vital organs (brain, heart, lungs) is maintained, that is, centralization of blood circulation occurs. Vasoconstriction leads to deep tissue hypoxia and development of acidosis. In these conditions, proteolytic enzymes of the pancreas enter the bloodstream and stimulate the formation of kinins. The latter increase the permeability of the vascular wall, what contributes to the transition of water and electrolytes to the interstitial space. As a result, in the capillaries an aggregation of erythrocytes occurs, what establish a foothold for the formation of thrombi. Irreversibility of shock actually follows his process.

Hemorrhagic shock leads to severe ***polyorganic disorders***. As a result of hemorrhagic shock, the lungs are affected with the development of acute pulmonary insufficiency by the type of a "shock lung". During a hemorrhagic shock the renal blood flow dramatically decreases, hypoxia of the renal tissue develops, the "*shock kidney*" is formed. Especially unfavorable influence of a hemorrhagic shock is on the liver, in which morphological and functional changes cause the development of the "*shock liver*".

If a wound is small and plugged with a clot, ***the hemopericardium*** develops in a patient, the result of which depends on the rate of blood accumulation in the pericardial sac. The accumulation of blood in the pericardial cavity prevents venous return to the right atrium and the right ventricle, what causes venous hypertension, which is manifested by the swelling of the cervical veins, face, increased central venous pressure. Since an ejection of the right ventricle is regulated mainly by the volume of the blood entering it, the blood volume entering the left half of the heart via pulmonary circulation is significantly reduced as well. This contributes to a decrease of the diastolic filling of the left ventricle, and since during the diastole, the pressure in the heart chambers should be low, an increased pressure in the pericardium cavity negatively affects this phase of a cardiac cycle. Cardiac ejection and pressure in the aorta sharply decrease, what leads to tissue hypoxia, acidosis and coronary blood vessel deterioration – factors that contribute to a decreased myocardial contractility, arrhythmia and cardiac arrest.

Taking into account the above mentioned, we can distinguish the following mechanisms of the cardiovascular homeostasis involved in the preservation of the level of arterial pressure: increase of total peripheral resistance and a number of cardiac contractions; increase of the blood flow to the right heart by increasing the volume of circulating blood. Centralization of blood circulation and release of deposited blood contributes to it.

The patients, who can be delivered to a clinic, have a blood clot in a wound, which plays a role of an obturator. Due to the thromb and an increased pressure in the pericardial cavity, which does not practically decrease due to the limited elasticity of the latter, systolic blood pressure rises to the level of 50-70 mm Hg. It is assumed that the frequency of blood clot formation is directly proportional to the thickness of the heart wound wall and therefore inversely proportional to the frequency of the tamponade occurrence.

Violation of hemodynamics does not always depend on the intensity of bleeding and blood loss. In case of accumulation of even a small amount of blood in the pericardium, a clinical picture of ***cardiac tamponade*** develops. A small size of a pericardial wound, pericardial displacement in relation to the heart wound contribute to it. Depending on the size of a hemopericardium, its wound from the rounded one turns into the fissural, what further complicates an outflow of blood from the pericardial cavity to the pleural cavity. In case of locaization of the pericardial wound near the pleura or the posterior surface of the chest wall, a blood outflow is also difficult. The causes of a difficult blood outflow from the pericardial sac cavity are a small wound size, its high location, presence of valve mechanism and a blood clot. Exactly due to the cardiac tamponade even isolated pericardial wounds pesent a considerable threat. A full-blown picture of a heart tamponade is observed during accumulation of 200 ml of blood in the pericardial sac and in case of acute bleeding (up to 500 ml), heart arrest in the systole may occur.

Clinical picture. During an interview, a patient complains of chest wounds with open heart injury; pain in the region of the chest and in the region of the heart, often with irradiation to the left shoulder, axillary region, neck, and less often to the epigastric region; feeling of intermissions in the heart region, dyspnea, loss of consciousness, sudden general weakness, apprehension, deficiency of air. In case of multiple and combined injuries, patients may complain of coughing sometimes with a foamy red blood discharge, "crunching" of ribs at the site of fractures, abdominal pain.

Objective signs. General condition of a patient, as a rule, varies from average severe to extremely severe. Pronounced pain syndrome often leads to traumatic shock. Patients are anxious. Sometimes impairment of consciousness until coma is observed. Pale skin, cyanosis of the lips and mucous membranes, acrocyanosis, clammy cold sweat.

Status localis. During examination of the chest in the heart projection a wound in case of an open heart damage, hematomas, and scratches are observed. The deformities of the ribs and sternum in case of fractures can be determined. Pain during palpation at the places of fractures. An increase of the pulse rate up to 180 per 1 min, poor blood volume and tension are characteristic. Blood pressure is low. By auscultation, heart tones are soft, arrhythmia is possible. In case of a heart rupture, systolic murmurs are observed by auscultation. By percussion, extension of the borders of cardiac dullnes is observed.

Clinical signs of heart wound are manifested by *Beck triad*: 1) a wound in the heart projection; 2) sharp fall of blood pressure; 3) muffled or absent heart sounds (signs of cardiac tamponade).

Leading clinical syndromes – hemorrhagic, chest pain.

In accordance with the standard schemes, a plan for additional laboratory and instrumental examination of a patient with heart injury:

1. Clinical blood test.
2. Clinical urine test.
3. Definition of the CBD.
4. Biochemical analysis of blood.

5. Blood group and Rh affinity.

6. Coagulogram.

7. Survey radiography of the chest organs in two views.

8. ECG.

9. Bronchoscopy (in case of a combined injury according to indicators).

10. Measurement of the CVP.

11. Ultrasound examination of the pleural cavity and heart.

12. Puncture of the pleural cavity.

13. Pericardium puncture.

14. Thoracoscopy.

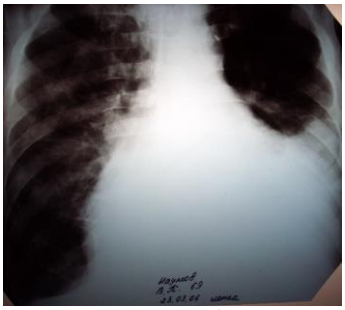

Fig. 77. X-ray picture of a patient with hemopericardium (tamponade) of the heart

Characteristic pathological changes.

1. *Clinical blood test* – in case of the complication of a chest trauma with hemorrhage, anemia is possible (reduction of Hb, erythrocytes), elevated ESR.

2. *Clinical urinalysis* – there may be no changes.

3. CBD is determined in case of bleeding.

4. *Biochemical blood test* – possible increase of transaminases, C-reactive protein. For a heart injury, an increase of creatine phosphokinase, lactate dehydrogenase, and the most specific increase of MB-izoenzyme creatine phosphokinase serum are characteristic.

5. *Blood group and Rh affinity*.

6. *Coagulogram* – there may be no changes.

7. *Review X-ray of chest organs* in two views – fractures of the ribs, sternum can be determined. X-ray symptoms of a heart injury include:

1) *hemopericardium symptoms* – extension of the heart borders, flattness of the cardiac arches, increase of the intensity of the heart shadow (Fig. 77. Radiograph of a patient with hemopericardium (tamponade) of the heart);

2) *a symptom of pneumopericardium* – presence of an air stripe between the shadow of the heart and the pericardium;

3) *a symptom of hemopneumopericardium* – an increase of the heart size, flattness of its arches, a horizontal level of fluid between the shadow of the heart and the pericardium;

4) change of cardiac pulsation.

As a rule, a heart wound is accompanied by appearance of large hemothorax (classification of hemothorax, depending on the size of intrapleural bleeding, as below). The radiographic picture *of hemothorax* is rather specific – characterized by intense homogeneous opacification on the injured side with a transverse upper contour (Damoiseau line), the phreno-costal sinus is not visualized.

8. *ECG* – tachycardia, signs of myocardial hypoxia. The appearance of a negative T wave in the precordial electrocardiogram is observed, sometimes it becomes biphasic, displacement of the interval S-T from the isoelectric line, decrease of voltage and deformation of R wave.

9. *Bronchoscopy* in case of a combined trauma: inflow of red blood from the bronchus in case of a lung injury. Bronchial and tracheal injuries can be visualized.

10. *Measurement of CVP*: sharp decrease of CVP is observed during massive blood loss, during cardiac tamponade CVP increases.

11. *Ultrasound examination of the pleural cavity*: in case of a chest trauma complicated with hemothorax the fluid in the pleural cavity is observed on the injured side. The ultrasound examination of the heart is the most informative in case of a heart trauma. A decrease of contractile function of the myocardium is observed, external and internal heart ruptures can be seen.

12. *Puncture of the pleural cavity* serves as a diagnostic and therapeutic manipulation for suspected hemopneumothorax.

13. *Pericardial puncture* is a therapeutic diagnostic method indicated for cardiac tamponade in order to reduce intrapericardial pressure.

14. *Thoracoscopy* is a highly informative method, which allows to specify the nature, localization of damage and to choose therapy approach.

Puncture of the pleural cavity. Puncture of the pleural cavity serves as a diagnostic and therapeutic manipulation for a suspected hemopneumothorax.

To remove air, the puncture is performed under local anesthesia with a solution of novocaine in the second intercostal space along the midclavicular line in a sitting position of a patient. If, due to the difficult condition a patient can not sit, the puncture is performed in a lying position on a healthy side in the Vth-VIth intercostal space along the midaxillary line.

To remove the blood, the puncture is performed in the VIIth-VIIIth intercostal space along the posterior axillary line in a patient's sitting position. To avoid damage to intercostal vessels, a puncture is performed along the upper edge of an underlying rib.

The examination of contents of the pleural cavity – *the Ruvilua-Gregoire test*, is evaluated as positive in case of coagulation of the drained blood from the pleural cavity and indicates continued bleeding into the pleural cavity.

The Larrey's pericardiocentesis is performed in the point corresponding to the vertex of an angle between the left rib arch (insertion of a cartilage of the VII rib to the sternum) and a base of the metasternum on the left. After local anesthesia with novocaine, a long needle connected with a syringe, is introduced in the puncture point in a cranial direction at an angle of 45° to the body surface. The skin, subcutaneous tissue, abdominal rectus muscle with aponeurosis are perforated.

After a puncture of the anterior wall of the vagina of the abdominal rectus muscle, the direction of a syringe and a needle are changed in parallel with the area of the sternum, and after that a needle is pushed upwards by 2-3 cm. The direction of a needle – from bottom upwards and slightly back. In this case a needle passes through the muscular fascicles of the sternal region of the diaphragm, the lower surface of the pericardium. A sense of pulsation suggests heart closeness. During pushing of a needle, a syringe plunger is periodically pulled out to fix the moment of a pericardial puncture, after what a needle advance should be stopped to avoid damage to the heart. The fluid from the pericardial cavity is sucked out very slowly, so as not to disrupt the work of the heart.

Differential diagnosis of heart wounds in some cases does not make much difficulty, and in others it is extremely difficult. It is explained by many factors: lack of reliable symptoms or variability of many signs in acute period, unusual localization of a wound, satisfactory condition of a patient, frequent alcohol intoxication, which disguises a clinical picture, extreme severity of the condition, especially in case of multiple injuries, combined heart wounds and abdominal cavity organs, as well as relative rarity of such wounds and mistakes, and sometimes lack of experience in a surgeon.

The diagnosis is based on the study of the mechanism of trauma, clinical picture, assessment of the severity of a patient's condition, the stage of shock and the detection of symptoms of cardiac tamponade.

Treatment of a patient with a heart trauma.

First medical aid at the prehospital stage for a closed heart injury: *pain syndrome management* – intravenous slow administration of Droperidol and Fentanyl in a solution of glucose or Omnopon; *for disturbances of rhythm and conduction* – Lidocaine intramuscularly (in case of frequent ventricular extrasystoles), slow intravenous administration of Isoptin or Trasicor (in case of fibrillation or atria fluttering, supraventricular tachycardia), Novocainamide intravenously with normal saline (in case of paroxysmal ventricular tachycardia, combined with hypotonia – in a mixture with a Mesaton solution); in case of signs of a left ventricular failure or cardiogenic shock during unarrested attack of paroxysmal tachycardia (or ciliary arrhythmia),

during ventricular fibrillation – urgent external shock therapy is carried out; in case of pulmonary edema *without manifestations* of cardiogenic shock – intravenous solution of Strophanthinum with a sodium chloride or a glucose solution, Lasix solution; *oxygen therapy is carried out; in combination with a pulmonary edema and a cardiogenic shock* – Strophanthinum in a mixture with Polyglukin or Rheopolyglukin, Droperidol in a mixture with Mesaton or norepinephrine intravenously by drop infusion; after stabilization of arterial pressure – Lasix, a solution of sodium bicarbonate.

The first medical aid at the pre-hospital stage in case of a heart wound. The general appearance of a patient, nature and localization of an injury, frequency and character of respiration, pallor or cyanosis, as well as the presence of blood admixture in the sputum will help to determine a patient's condition. *Resuscitation measures*: restoration of breathing and blood circulation, control of external bleeding; in case of an open (gaping) wound – adjustment of dressing using polyethylene, rubbered fabric (wound air resistance), and in case of continued bleeding – adjustment of a tight bandage. In presence of paradoxical movements of a chest wall (in case of several rib fractures), a compression bandage should not be adjusted in any case, because it will cause sudden respiratory affection. Anesthetics should be used only if the injuries of the abdominal cavity organs are completely excluded.

Patients with chest injuries are hospitalized in specialized multi-speciality hospitals. They are transported in a lying position, with a bolster, a bundle from outer garments, placed under a head.

Choice of medical approach. There are conservative and surgical ways of treatment of heart injuries. In case of concussion, heart impact and traumatic myocardial infarction, the treatment is usually conservative. In case of heart wounds, external and internal heart ruptures the treatment is surgical.

In general ***conservative treatment of heart trauma*** is similar to intensive therapy of acute coronary insufficiency or myocardial infarction. First of all, it includes control of pain syndrome, introduction of cardiac glycosides, antihistamines, drugs that improve coronary circulation and normalize myocardial metabolism. If necessary, antiarrhythmic and diuretic drugs are administered. Infusion therapy is conducted under control of central venous pressure.

Surgical treatment. After confirmation of a heart wound or a heart rupture, urgent surgical treatment according to vital signs under endotracheal anesthesia is indicated as soon as possible. It should be emphasized that a decrease of hemodynamic parameters, degree of blood loss and shock should not prevent from an operation, because its delay in order to improve these parameters leads to cardiac arrest due to a tamponade.

Access to the heart must be broad, extremely careful and requires the shortest time spent. Incorrect choice of surgical access often leads to dangerous complications – increased bleeding, coronary retroclusion.

All of the above mentioned requirements fully correspond to the anterolateral intercostal transpleural access – a standard thoracotomy along the IVth-Vth intercostal space on the side of a wound localization on the chest wall. An operating field should be prepared taking into account possible extension of an incision across the sternum to the opposite side of the chest, that is, a lumbar sternotomy and an opening of the opposite pleural cavity.

In case of ***hemothorax*** detection (***rupture of the pericardium***), a surgeon starts with extensive revealing the pericardium anteriorly from the diaphragmatic nerve, inserts his left arm into the pericardial cavity, using Lejars heart enveloping, and since most wounds are located on the anterior surface, a thumb of the left hand (other fingers and the palm can be used for massage) swabs a rupture heart area and stops ongoing bleeding. Only after this an assistant can begin to draw blood from the pleural cavity for reinfusion.

If a wound is located on the back surface, it is swabbed with an index finger. In case of small heart ruptures, especially of its right departments, the hole is often swabbed with a blood clot and no visible bleeding is determined. Therefore, a thorough operative exploration of all sections of the heart walls in case of hemopericardium detection is mandatory. An examination

of the heart, especially its posterior departments, should be carefully performed to prevent a significant angulation of intrapericardial vessels.

Traumatic ***external heart ruptures*** mostly have irregular, torn edges. Only crushed tissues are subject to excision. Heart wounds are usually closed with loop or U-shaped stitches with an atraumatic needle capturing an entire thickness of a damaged heart muscle. Superficial suturing, although stops bleeding, may further contribute to the onset of heart aneurysm and various thromboembolic complications. In case of large wounds, the mattress stitch may be used. To prevent cutting of sutures, flaps of the pericardium or small nylon pads are used. In case of heart wounds in the region of coronary vessels the bypass U-shaped stitch is used, and if a large vessel is damaged – the vascular stitch.

In case of ***internal heart ruptures***, depending on anatomical disturbances, primary delayed and late interventions with the use of extracorporeal circulation are indicated.

Surgical access, tactics and operational technique for heart wounds and its rupture in case of a closed chest trauma are identical.

It is advisable to emphasize a number of characteristic features: due to the severity of the condition it is better to make a standard left-sided anterolateral thoracotomy along the fourth or fifth intercostal space; during suturing of a heart wound, additional caution is required regarding coronary arteries and proper heart vessels; the pericardium is sutured with rare single stitches, it is mandatory to perform an additional pericardial incision (counterpuncture) with a length of 3 cm in the region of the posterior cardiophrenic angle.

Treatment of the patients, subject to various surgical interventions for traumatic chest injuries, is performed individually. A different approach to the management of patients in the *postoperative period* is determined by the features of mechanical trauma, nature and volume of damage of an intrathoracic organ, thoracic skeleton, combination of injuries, severe shock, functional disorders of respiration, circulation, trauma of other vital organs, type of surgical intervention, age of a patient, complications developed during an operation, etc.

The results of surgical interventions for damaged intrathoracic organs showed that *prevention of postoperative complications* should be started yet in the operating room. Before extubation special attention is paid to the sanitation of the tracheobronchial tree and restoration of adequate independent breathing. Multiple sanitations of the tracheobronchial tree during an operation and before the removal of an advanced airway, especially in case of a lung injury, is carried out using Metras catheters reaching the main and lobar bronchi.

If a surgery was performed for one of the intrathoracic organs, it is necessary to expand all the lung divisions on the operating table. Only after the restoration of adequate autonomous breathing and gas exchange, controlled by the indicators of blood gases, acid-alkaline state, hemodynamics of large and small circles of blood circulation, absence of rales in the lungs, an advanced airway is removed.

For the patients with multiple chest injuries after an extensive operation for one of the intrathoracic organs with massive blood loss and severe cardiopulmonary disorder, as well as for elderly people, *prolonged artificial pulmonary ventilation* is administered. Early restoration of autonomous breathing does not provide an oxygen regimen of tissues and may lead to acute decompensation of cardiac activity. Such patients after careful sanitation of the bronchi with an advance airway are transferred to the intensive care unit, where artificial ventilation continues. Prolonged AV is performed on the basis of the study of indices of homeostasis, respiratory mechanics, pulmonary volumes. During artificial ventilation for breathing synchronization of a patient Promedol, Thalamonal, GHBA are introduced by a machine, muscle relaxants are not administered. In case of indications for a longer AV, an issue about the tracheostomy placement is raised. The nearest efficacy of a surgical intervention for the organs of the chest cavity depends on the active management of patients, therapeutic exercises and sanitation of the tracheobronchial tree via percutaneous tracheal puncture, bronchial catheterization or bronchoscopy.

It should be emphasized that *postoperative bronchial sanitation* depends on the severity of a patient's condition, combination of injuries, intraoperative and immediate postoperative complications (atelectasis, pneumonia), intensity of the cough reflex, and others.

If a general condition is satisfactory, there are no hemodynamic disorders and damages of the thoracic skeleton, a patient is forced to expectorate, holding the wound by hands and pressing on the sternum in time with cough movements. If the cough reflex is preserved, a puncture of the trachea with fractional administration of an antibiotic solution, bronchodilators, enzymes, irrespective of the volume of an intervention and lung sealing capacity on the operated side is more effective. In some cases, a cervical vagosympathic blockade is performed in advance.

In patients with poorly manifested cough reflex and a severe general condition, sharply reduced cardiorespiratory parameters, the sanitation of the bronchial tree by the tracheal puncture is usually not effective. Bronchial sanitation in this involved patient population is carried out by nasotracheal catheterization of bronchi by Metras catheters or bronchoscopy. However, the most effective sanitation bronchoscopy can not be used in patients with a combined craniocerebral trauma and fractures of the cervical spine as often as it is sometimes necessary.

In the postoperative period prevention of *atelectases, pneumonia, pulmonary abscesses, pleural empyema, pericarditis, thromboembolism of the pulmonary artery, suppuration*, and others are extremely important.

Prevention of atelectases and pneumonia in the patients, especially with pulmonary ruptures, should be performed at all stages of surgical treatment. Before surgery – aspiration of the contents of the trachea, bronchi, normalization of homeostatic indices. During a lung surgery – careful operating, adherence to the anatomical features of the segments, exposure of a lung along the whole length with obligatory securing of a pulmonary ligament, multiple aspiration of contents of the tracheobronchial tree and expansion of all remaining lung divisions. In the nearest postoperative period – inhalation of enzymes, bronchodilators, active expectoration of sputum or its aspiration with creation of low positive pressures in the bronchial tree. The applied method of prevention and treatment of these complications reduced the number of postoperative atelectases greatly.

Prevention of pleural empyema is carried out by rational drainage of the pleural cavity. In case of a pleural empyema, drainage of the pleural cavity with active aspiration of the contents, its lavage with antiseptics and administration of antibiotics, depending on the sensitivity of the microflora, are performed.

Prevention of thromboses and embolism should be performed during operations and in the postoperative period. In patients with increased coagulation, increased concentration of fibrinogen, especially fibrinogen B, administration of anticoagulants of direct effect until stable normalization of a coagulogram indicators is necessary.

The study of indicators of AAB, electrolyte metabolism, bioenergetic processes on the 1, 3, 5, 7th and 14th day after an operation revealed their relationship with the nature and volume of damage of an intrathoracic organ, an operation performed, amount of blood loss, cardiorespiratory disturbances, and others. That is why the correction of disturbances of these homeostasis indicators should be performed individually and in a targeted manner: introduction of hypertonic solutions of glucose with insulin, polyionic solutions, vitamins of group B and C, transfusions of blood, plasma, protein blood substitutes, Rheopolyglukin, cardiac drugs, bronchodilators, sanitation of the bronchial tree, oxygen therapy, inclusion of products rich in potassium and calcium in the diet.

In case of a heart injury, occurrence of ***such emergencies as total hemothorax, hemorrhagic shock, cardiac tamponade***, during which an urgent thoracotomy is indicated for the elimination of a source of bleeding.

Complications that occur in the postoperative period in case of heart injuries are divided into *early and late.*

The early ones include: myocardial infarction, purulent myocarditis, cutting out of myocardial sutures, acute cardio-pulmonary insufficiency, acute heart failure due to an injury of intracardiac structures, pulmonary atelectasis.

The late ones include: pericarditis, cardiac aneurysm, pleurisy, purulent tracheobronchitis, pneumonia, pleural empyema, suppuration of a postoperative wound.

The study of long-term results of treatment confirms that those who suffered from a heart wound are not a socially unpromising group of patients. Working ability is particularly important in the evaluation of the results of surgical therapy of a heart wound. On average 80% of the operated patients return to previous work after such surgeries, 15% move to easier work and 5% leave a former job.

CHAPTER IV. ACUTE SURGICAL DISORDERS OF THE MAJOR VESSELS OF THE EXTREMITIES

IV.1. MODERN METHODS OF RESEARCH IN SURGICAL DISEASES OF THE MAJOR VESSELS OF THE EXTREMITIES

Instrumental methods of examination

Ultrasound examination of vessels (*Doppler ultrasonography)* is a method of examination that, due to the presence of a Doppler sensor, allows to evaluate the vascular lumen, the thickness of the vascular wall, the presence of an obstacle to the blood flow (embolism, atherosclerotic plaques), and also to determine the direction, nature and volumetric blood flow, to determine the age of blood clots, etc. Today, it is *one of the principal methods for assessing the status of major vessels.*

Angiography is an X-ray contrast examination of vessels with a special X-ray unit (angiograph) which is the main method that allows the most accurate diagnosis of location and extent of injury of the arterial bed and to make a decision about the possibility and nature of surgical reconstruction of the affected arteries.

Several *types of angiography* are used:

1) Seldinger's percutaneous femoral arteriography;

2) the Dos Santos technique of translumbar puncture abdominal aortography;

3) catheterization chest aortography.

When the lesion on the lower extremity is localized more distal than the inguinal ligament, and when the femoral arterial pulsation is determined, it is recommended to perform a *Seldinger's percutaneous puncture femoral arteriography* (1953) using a special Seldinger's puncture needle and a transition tube connecting the needle with an automatic injector syringe. The femoral artery is punctured immediately below the inguinal ligament, 40-45 ml of a contrast medium (Urotrast, Triombrastum, Omnipaque, etc.) are injected, and video recording of the contrast on various segments of the arterial bed of the limb (on the femoral, popliteal, and arteries of the shin) is performed.

If Leriche's syndrome is seen (with occlusion of the aortoiliac segment), when the femoral arterial pulsation is not felt, it is necessary to perform *translumbar puncture abdominal* aortography by Dos Santos (Dos Santos 1929), using a special long mandibular puncture needle. Site of puncture at the patient's prone position – the left costovertebral angle 6-7 cm more laterally than the spinous processes of vertebra, the direction of the needle – to the spine, slipping from the body of the vertebra, the needle punctures the aorta, 60-80 ml of contrast is injected, and the permeability of the contrast along the abdominal aorta, the common, external and internal iliac arteries and arteries of different parts of the limb (femoral, popliteal, and of the shin) is recorded.

When the lesion is localized in the branches of the arch of the aorta (in the case of Takayasu disease), *catheterization thoracic aortography* is performed.

*The main **angiographic signs** of occlusive arterial involvement* are the following*:*

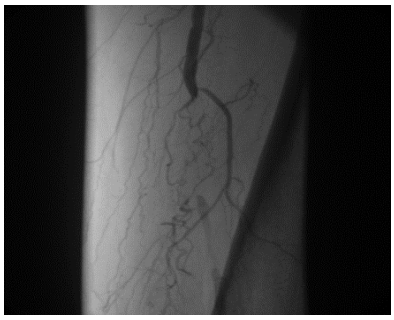

Fig. 78. Puncture femoral arteriography – occlusion of the superficial femoral artery in the middle and lower third of the thigh ("amputation stump" symptom)

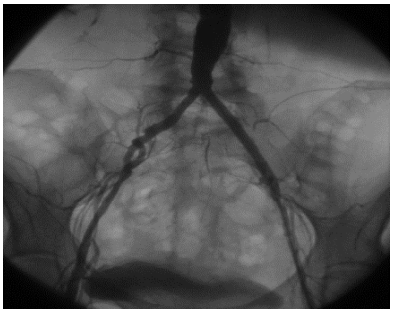

Fig. 79. Abdominal aortography – Leriche's syndrome, stenosis of the left external iliac artery

1) ***atherosclerosis*** (fig. 78, 79) – the arteries of the large and middle caliber (iliac, femoral, carotid) are affected in the form of segmental stenosis or segmental occlusion with an abrupt breaking the contrast entering to the arteries (the symptom of "amputation", "the stump of the vessel"), the expansion and voluminosity of the arterial segment, irregular narrowing, " indentures" of the inner contour due to the protrusion of atherosclerotic plaques in the lumen, the presence of pronounced collaterals;

2) in ***endarteritis, nonspecific arteritis*** - regular diffuse narrowing of the lumen or total occlusion of the distal arteries (of the foot, leg, subclavicular).

These two above-mentioned methods are the most informative and are of paramount importance in the diagnosis of chronic limb ischemia syndrome, the others, which are listed below, are only auxiliary techniques that are used mainly in outpatient settings.

Currently, a highly informative, non-invasive method of research is computed tomography in angiography mode.

Oscillography is based on recording the pulse fluctuations of the arterial wall with varying degrees of compression by the cuff of the device for measuring blood pressure. It characterizes *major blood flow*. The permeability of the artery and the tone of its wall are assessed by the nature of the curve. The maximum, average and minimum blood pressure (BP) (for the middle third of the leg, the norm is M_x 120-140, M_y 80-105 and $M_м$ 60-80 mm Hg) are determined, the main parameter is the oscillographic index (OI) which is measured in mm of the height of the highest spike, which normal value in the middle third of the shin and radial artery is of 10-15 mm, in the popliteal artery it is of 20-25 mm, in the femoral artery it shows 30-35 mm. *According to the shape*, the normal, pointed, blunt, and plate-like oscillograms are distinguished.

Longitudinal segmental rheovasography records resistance pulse fluctuations of limb tissues against the low frequency alternating current. Due to the small electrical resistance of the blood in the bloodstream during systole, the resistance of the limbs tissues decreases; and in case of outflow during diastole it increases. It characterizes the *general (trunk and collateral) bloodstream in the extremity*. The form and shape of the rheovasograms depend on the nature and velocity of the pulse wave, which varies depending on the peripheral resistance and systolic blood volume. A normal rheographic wave has a high, abrupt anacrotic peak, a segment that goes down by several "respiratory" waves, and a dicrotic spike. The main parameter is the rheographic index (RI) - this is the ratio of the main rheographic wave (anacrotic) value in mm to the magnitude of the calibrated impulse of the device. In norm, the foot PI is of 0.9-1.0, the shin value is of 1.5-1.6, the thigh PI is of 2.5-2.7.

At the major artery stenosis there is a decrease in the amplitude of the oscillographic and rheovasographic curves, the decrease of the OI and PI, extending and disappearance of additional (respiratory) waves on the reovasogram.

Radioisotope method for determining the blood flow rate:

1) *clearance method* supposes the study of limb muscle flow by entering ^{131}I isotope with hipuranium (1-2 μKu) into the calf muscle and determination of half-life of the isotope (N - 11-16 min, in case of pathology it shows lengthening of time);

2) studying the *rate of blood flow in the extremity* by administering to the vein a serum albumin labeled with ^{131}I (0.75 μKu / kg in physiological saline) and determining the time of the isotope appearing on the foot or hand (the norm is 20-30 sec). The time of saturation of the feet (the rate of 3-5 minutes) is also determined. In case of pathology it shows extension of time of the mentioned parameters.

Capillaroscopy - the toe nail bed is examined under a microscope: a pink base normally, capillaries in the shape of a female hairpin, in pathology – a pale background, deformation of the capillaries, sinuous, cliff, they are coma- and dot-shaped, sometimes – only their scraps.

Thermometry (thermography) is an assessment of the temperature of the surface of the limb with a special electrothermocouple or registration of its infrared radiation with a thermal imager. Normally, in distal (foot) temperature is 29-31º C, and in the pathology - 22-24º C. Due to the high dependence on external factors, the greatest temperature difference is in symmetrical areas. For the same reason, it has a low diagnostic value.

IV.2. ACUTE LIMB ISCHEMIA SYNDROME ARTERIAL EMBOLISM AND ACUTE ARTERIAL THROMBOSIS OF THE EXTREMITIES

Treatment of the patients with acute limb ischemia is one of the most urgent problems in the present vascular surgery. According to L. Norgren, W.R. Hiatt et al. (2007), the incidence of acute limb ischemia is 140 cases per million population per year.

The clinical practice of the last decades indicates the ongoing increase in acute thromboembolic lesions of the major vessels. Untimely diagnosis and late start of treatment mostly determine a high mortality rate, that ranges from 20 to 35%, and in the aged and elderly patients it is up to 45%. The number of amputations of the extremities in patients with this urgent vascular pathology reaches 12-28%.

The injured major arteries is one of the most dramatic issues of the polytrauma, as patients with major vessel injuries die, most often in the immediate hours since the moment of injury, due to bleeding. This is a complex issue because of a number of managing and surgical problems, resulting in a percentage of unsatisfactory results of treatment reaching 27-75%, which, against the background of a steady increase in the number of injured with vascular traumas, shows this problem in the category of important social tasks.

The success of the treatment of this type of injured is largely determined by the timely delivered qualified surgical care. Most patients with major vessel injury die even before the specialized angiosurgical care is given. It is caused by a massive blood loss, which leads to hemorrhagic shock.

The firm knowledge of the basic modern principles of diagnosis and treatment of the patients with acute limb ischemia syndrome (ALIES) is an urgent task in the clinical training of a physician.

The acute limb ischemia syndrome (ALIES) (synonym – limb acute arterial occlusion syndrome – LAAOS) *is a syndrome which is based on acute disorder of arterial circulation in the extremity; it results from the termination of blood flow in one or more major arteries and leads to the development of acute ischemic disorders in the limb tissues, which cause ultimate outcome (in non treated cases), the development of irreversible necrobiotic changes in the tissues of distal limbs, that is the development of* ***gangrene****.*

ALIES is a heterogenous and polyetiological concept. Its development is caused by acute vascular diseases – *arterial embolism and acute arterial thrombosis, as well as traumatic injury of the limb arteries,* sudden extravasal compression of the blood vessel, etc. This various vascular pathology combines a sudden tissue trophic disorder of the limb resulted from a decrease or complete cessation of blood flow in its major arteries with the development of acute

ischemia of the distal limb tissues. At the same time, being very similar clinically, these diseases differ etiopathogenetically, that affects their clinical manifestations, and their specific features allow the differential diagnosis of them.

Arterial embolism (AEM) of limbs *is a sudden cessation of blood flow in the major artery caused by a blockage of a normal before artery with a thromboembolus, which is detached and brought by a blood stream from the cavities of the heart or large arterial trunks, that leads to severe circulatory disorders in the limb and development of severe acute ischemic lesions of tissues, which prior to that had a normal blood supply.*

AEM is a complication of various *embologenic diseases*. In 90-95% of patients, the large circulatory circle embolism of the arterial vessels is caused by *the heart disease*, in which thrombotic masses are formed in its cavities.

Atherosclerotic lesions of the heart, the so-called "*atherosclerotic cardiopathy*" (acute myocardial infarction, diffuse and postinfarction cardiosclerosis, postinfarction aneurysms of the heart, etc.) are currently ranked first in frequency, and approximately 55% of cases are the cause of acute arterial occlusion (AAO) of limbs. *Rheumatic impairment of the valves of the heart* (and mainly mitral stenosis among them) ranks second and makes up 43%. *Septic endocarditis* and *congenital heart disease* are the cause of AEM in only 1-2% of cases. At the same time, in patients with atherosclerotic heart disorders, embolic thrombotic masses are most often localized in the left ventricular cavity, and in the patients with rheumatic defects it is in the left atrium, rarely thrombi are formed on the valves (in cases of septic endocarditis, prosthetics of the valve).

Among *extracardiac embolic diseases* the first place is occupied by *aneurysms of the aorta* and its large branches (3-4% of cases), *aortic atherosclerosis* at the thrombonecrotic stage (the formation of mural thrombi), extremely rarely peripheral AEM may occur in pneumonia, pulmonary tumors (pulmonary vein thrombi are the source), in acute thrombosis of peripheral veins (the so-called *"paradoxical embolism*", when, due to congenital septal defects and patent ductus arteriosus (open arterial duct), an abnormal movement of the thrombus is observed from the right to the left cavities of the heart), in cases of thyrotoxic cardiomyopathy with atrial fibrillation, etc.

Most often (in approximately 75% of cases) AEM are observed in patients with a *disordered cardiac rhythm*, especially with different types of *fibrillation arrhythmia*, which in the patients with atherosclerotic cardiopathy and rheumatic heart defects contribute to the formation of blood clots in the cavities of the heart due to significant disorders of intracardiac hemodynamics.

The intracardiac thrombus detachment occurs most often at increased cardiac activity, due to emotional and motor stimulation, intake of cardiac glycosides, after defibrillation in order to eliminate arrhythmias (the so-called post-conversion embolisms); an increase in the fibrinolytic activity of the blood may also contribute to the fragmentation and detachment of thrombus (and this is the reason why the use of powerful thrombolytics for the treatment of AEM is considered inappropriate (V.S. Saveliev, 1974).

It is important to emphasize that in all embologenic diseases, there is a tendency to *recurrent embolism* (unless the radical treatment of the underlying disease is being performed).

The thrombus, which was detached by the blood flow, can be carried into any artery of a large blood circulation. It is registered that most often the thromboembolus is brought into the branch of the arch of the aorta (including in the cerebral arteries) - 36%, the second place is seen in bifurcation of the aorta and main arteries of the lower extremities - 24%, in the visceral arteries - 22%, in renal - 18%, less often - in the arteries of the upper extremities.

Usually, the emboli occlude the main vessels in the area of their bifurcations or junction to the large branches, where the vessel diameter decreases dramatically, and so they are called *"riding emboli"* (or *"saddle emboli").* Therefore, the most common places (the so-called *"surgical strata")* of embolic occlusions are (Figure 80): bifurcations of the common femoral, common iliac, popliteal arteries, bifurcation of the aorta, brachial artery, junction to the shoulder artery, deep brachial artery, etc.

Embolism of the arteries of the shin and forearm happens more often than it is diagnosed, and occurs as hidden embolism. Embolus can migrate distally from one 'floor' to another (after the introduction of anesthetics, antispasmodics, during transportation), that explains the cases of some eventual improvement of the limb condition.

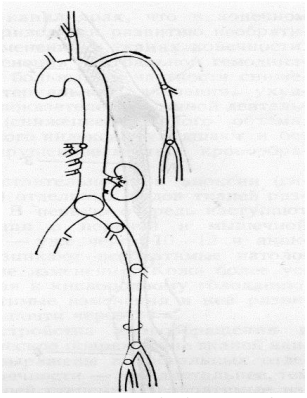

Fig. 80. 'Surgical strata' of arterial embolism

"Stratum" embolism is often observed: AEM occurs simultaneously at different levels of major vessels of one extremity, "*combined*" embolism - AEM of different limbs, *"multiple"* embolism - when visceral or cerebrovascular vessels are affected simultaneously.

Thus, the following aspects play a role in *the pathogenesis of AAO of limbs on the basis of AEM*:

1) since the embolus is localized in the area of bifurcation or junction of large trunks, it usually leads to the exclusion of all major arteries from the circulation, that is, more distal than embolic occlusion, *arterial blood flow practically ceases*;

2) due to the fact that a healthy artery is exposed to acute occlusion, acute ischemia occurs in tissues, which before had not suffered from insufficient blood supply, and therefore at the time of the event (termination of the main blood flow more distal than occlusion) no collateral network is found, that could at least compensate to some extent the disturbed major blood flow, which, together with the first moment, causes *practically the termination of arterial blood circulation;*

3) at AEM, local blood circulation disorders are exacerbated by disorders of central hemodynamics due to the severe disease of the heart or large vessels.

In connection with the mentioned pathogenetic features, AEM is *characterized by acute onset, severe clinical symptoms (the proximal limit of ischemic disorders is clear and most often corresponds to the level of embolization), usually a severe degree of acute ischemia and its rapid progression with the development of gangrene.*

Acute arterial thrombosis (AAT) of the limbs *is a sudden cessation of blood flow in the major artery caused by the local formation of an intravascular thrombus that completely obstructs the lumen of the artery in its segment, which as a rule was affected (stenotic) by chronic occlusive process, that leads to acute disturbance of blood circulation in the extremity and the development of acute ischemic lesions of tissues, which until then were more or less under chronic ischemia.*

The main cause of AAT of limbs is *chronic occlusive disease of their arteries* - obliterating atherosclerosis (most often), obliterating endarteritis, thromboangiitis obliterans, leading to progressive stenosis of major arteries and an increase in chronic ischemia of tissues.

In the *pathogenesis* of pathological intravascular development of thrombi, the leading role belongs to three main factors known as the *pathogenetic Virchow's triad* :

1) the narrowing of the lumen of the vessel and slowing down of blood flow in it;

2) changes in the vascular wall (disordered smoothness and nonwettability of the inner membrane as a result of mechanical, thermal, chemical, bacterial effects, disorders of microcirculation), increased thromboplastic activity of the affected arterial wall, including reduction of the synthesis of *prostacyclin*, a potent platelet aggregation inhibitor, and increase in the synthesis of *thromboxane* - a powerful inducer of platelet aggregation activity;

3) disorder of the functional state of the hemocoagulation system and increased coagulation properties (primarily, the growth of aggregation activity of platelets) and inhibition of anticoagulant and fibrinolytic activity of the blood.

Pathogenesis of AAO of the limbs due to the AAT distincts from AEM, that influence on the degree of severity of acute ischemia that develops:

1) in cases of AAO, as a rule, acute occlusion affects at first a limited segment of one arterial line, the most stenotic due to chronic occlusion process, while the blood flow continues in the other arteries;

2) complete occlusion of the arteries occurs against the background of the chronic tissue ischemia and collateral circulation in the limb developed due to it, that to some extent compensates the stopped major blood flow in one of the arteries;

3) severe disorders of central hemodynamics, which exacerbate local disorders, are rarely observed in such patients.

Thus, in AAT, in contrast to AEM, *more distal to acute occlusion, the blood supply to tissues does not completely stop, although decreases, resulting in the development of acute ischemia of tissues in distal limbs which most often is not so pronounced and severe, gangrene develops rarer and much later, proximal border of acute ischemia is not so clear, there is no strict compliance with the localization of the thrombus and the level of ischemic disorders.*

AEM and AAT of the major arteries of the limb lead to the development of acute ischemia of the tissues more distal to the occlusion, to the disordered function of the limb and to the neurological impairments in the extremity.

Classification of degrees (stages) of acute ischemia of limb tissues in case of LAAOS - limb acute arterial occlusion syndrome (ALIES - acute limb ischemia syndrome) was developed by V. S. Saveliev et al/(1987). It includes "ischemia on exertion", degree I, degree II, and degree III ischemia, which, in turn, are divided into 2 stages - A and B. Each of them is characterized by certain subjective and objective signs**.** ***Ischemia on exertion*** - no signs of ischemia in rest, but they appear on exertion. At the ***degree I ischemia*** there are no disturbances in sensitivity and movements in the affected limb: ***degree IA ischemia*** is characterized by the feeling of numbness, coldness, paresthesia, in cases of ***degree IB ischemia*** pains in rest in the distal parts of the limb develop. For ***degree II ischemia***, sensory disturbances, as well as disordered active movements in the joints from the paresis ***(degree IIA)*** to the plegia (***degree II***) are characteristic. ***Degree III ischemia*** is characterized by initiated necrobiotic phenomena, which are clinically expressed in the subfascial muscle edema (***degree III A***), and then - muscular contracture: partial (***degree IIIB***) or total (***degree III***).

Depending on *the clinical course*, the authors identify the following types of ischemia:

1) progressive;

2) moderately stable;

3) regressing ; (the first type results in gangrene, the second and third – in chronic arterial insufficiency).

V.S. Saveliev et al. proposed the following classification of the stages of development of acute ischemia of the limb tissues (depending on its duration):

Stage I - *acute ischemic disorders* developing in the close future after embolism or acute thrombosis, this stage is characterized by pronounced signs of ischemic limb impairment, clinically it manifests with all the above symptoms;

Stage II - *relative collateral compensation of the circulation of the limb*, this stage develops in a few hours (6-12); the compensation for collateral circulation led to reduced pain in the limbs, decreased pallor of the skin, the limb warms, the movements and skin sensitivity appear in it (it is seen, mainly in acute thrombosis, less often in embolism);

Stage III - *decompensation of the circulatory system* develops in approximately 8-14 hours, but may occur directly after the first stage; it is characterized by the appearance of signs of the beginning of non-fibrotic changes in the muscles, which manifests in subfascial edema, muscular pain at palpation, rigidity and restriction of passive movements in the joints up to the muscular contracture;

Stage IV - *irreversible changes in the limb tissues, the stage of gangrene* develops at the end of 24 hours and more, since the moment of acute occlusion; it is characterized by total contracture of large joints, muscle edema, sharp pain at palpation and passive movements, the

appearance of necrotic changes in the skin on the periphery or demarked area, general intoxication of the organism.

It is practically convenient to use *the classification* of acute ischemia degrees proposed by O.O. Shalimov et al. (1979), distinguishing *4 degrees of acute ischemic lesion of limb tissue*:

I - mild degree includes cases of suppressed and mild clinical manifestations, such as pain on exertion or poorly pronounced pain at rest, paresthesia, coldness of the limbs, as well as the initial stage of acute ischemic disorders that occur immediately after obstruction and are characterized by intense pain in the limb, coldness and paresthesias, but *no pronounced impairment of sensitivity and motor limb function are observed*; timely performed operation in case of AE leads to complete restoration of the function of the limb;

II - moderate degree is characterized by signs of *ischemic injury*, mainly of the *nervous apparatus*, disordered pain and tactile sensitivity, limitation of movements in the fingers or lack of active movements up to complete paralysis, however, *no rigidity, contracture, muscle swelling are seen*; the restoration of blood flow at this stage usually leads to restoration of the function of the limb, but an ischemic neuritis may develop that requires a long-term treatment;

III – *severe degree* *(or "complete ischemic syndrome")* is characterized by signs of initial *necrobiotic changes in the muscles*: rigidity, pain in muscles at palpation, restriction of passive movements in the distal joints of the limbs due to contracture of individual muscle groups; subfascial edema of certain groups of muscles may be observed; "complete ischemic syndrome" is clinically characterized by the emergence of a prognostically threatening combination of three "A" - akinesia, areflexia, anesthesia; restoration of blood circulation in the extremity in such patients usually leads to the development of post-traumatic disorders of local and general nature, possible necrosis of individual muscles and muscle groups, followed by their fibrosis; no complete restoration of the function of the limb is usually observed (the following consequences such as Volkmann's ischemic contracture, muscular weakness, ischemic neurites, as well as gangrene of individual toes and foot may develop);

IV - stage of gangrene or irreversible changes in the limbs tissues is characterized by *total contracture of the large joints* (ankle, knee, radial, elbow), sharp pain in the muscles at palpation and passive movements, their edema, irreversible changes in the tissues of the distal limbs with the demarked area appeared and necrotic changes in the peripheral skin, general intoxication of the body; in these cases, the thrombotic process extends to muscle arteries and venous vessels (secondary thrombosis); the only possible treatment is *limb amputation*.

The degree of expression of acute ischemia of the limb tissues in the development of LAAOS (ALIES), and their interconnected clinical picture as well, depend mainly on the cause of acute occlusion, the degree of development of collaterals, concomitant arteriospasm, the development of prolonged thrombosis, the state of central hemodynamics .

The AAO (acute arterial occlusion) syndrome of the limbs has a vivid clinical picture. The most characteristic symptom is a *sudden severe pain in the extremity*, that is so intense that the patients compare it with the "blow with whip," and some affected patients ask to "cut off the leg". The characteristic complaints are the following: of coldness, numbness, pallor of the leg, tingling; the weakness of the limb develops, which deprives the patient of the opportunity to walk and even stand, then paresis or paralysis of the limb may appear.

Additional questioning about *system complaints* allows suspecting or determining the cause of an acute arterial obstruction: there is one of the embologenic diseases (atherosclerotic cardiopathy, heart defects, cardiac arrhythmia, etc.), or signs of a chronic occlusive affection of the arteries of limbs – ‘intermittent claudication symptom’, trophic disorders, etc.

Local Status:

1) discoloration of the skin (from pallor to cyanosis and ’marble’ skin);

2) pronounced decrease in skin temperature (compared to the healthy limb);

3) impaired sensitivity (first the reduction of tactile and painful sensitivity is seen, then deep to complete anesthesia develops);

4) disordered motor function of the limb (from a decreased muscle strength and limited active movements in the toes to the absence of active motions initially in the distal joints, and then in more proximal up to the complete paralysis of the limbs*: akinesia, areflexia* develops);

5) development of subfascial edema of the muscles, restricted passive movements due to the development of rigidity and contracture of individual muscle groups, pain at palpation of the leg muscles (signs of initial necrobiotic changes in tissues);

6) further development of total contracture of large joints, edema of the limbs, necrotic peripheral changes and gangrene of a limb as a final outcome of ischemia of limbs, the appeared borderline area.

Fig. 81. Determining pulsation of major arteries of the limbs and head

The main objective symptom is *no pulsation of arteries distally to the place of occlusion, which allows to determine its level*.

Clinical symptoms in acute limb ischemia syndrome are *variable*. The urgency of the development, the degree and the dynamics of the development of clinical manifestations (as well as the degree of acute ischemia) depends, first of all, on the cause (AEM - arterial embolism or AAT - acute arterial thrombosis), as well as on the caliber of the vessel (that is, the place of occlusion), the nature of the blockage (complete or partial), the severity of arteriospasm and the ***degree of acute limb ischemia***, which, in turn, depends on all of these factors.

As it was above mentioned, arterial embolism is characterized by acute onset, severe clinical symptoms, the proximal border of ischemic disorders is clear and most often corresponds to the level of embolization; usually a severe degree of acute ischemia is seen and its rapid progression with the development of gangrene is registered.

The most severe clinical picture is observed in the *aortic bifurcation embolism*. Severe circulatory disorders in the lower limbs and pelvic organs occur, that manifest by general and local symptoms: acute onset - sudden intense pains appear in both lower limbs and in the lower half of the abdomen, which often lead the state of shock; pain is irradiated in the sacrum, lower back, perineum. The feet rapidly grow cold with their numbness and muscle weakness; the skin (up to the inguinal folds and buttocks) becomes pale, then – ‘marble’; all types of sensitivity get disordered quickly; the lower limbs are in elongated position, no active movements are seen, the feet hang down, the toes are contracted claw-like; pulsation is absent in all segments of the arterial bed of the limb. Due to ischemia of the pelvic organs, false positive or involuntary urination and defecation may happen. The picture of ischemia may first be more pronounced in one limb (as the embolus has the form of a "rider" and may not fully engage one of the common arteries). If no qualified emergency care is given, the disease progresses rapidly, ascending continuous thrombosis causes the portal occlusion of renal arteries, and acute renal failure develops, the gangrene of both extremities quickly emerges and the patient dies of endogenous intoxication and multiple organ failure.

Embolism of the iliac arteries. Embol is most often localized in the area of bifurcation of the common iliac artery or over the inguinal ligament, the clinical picture resembles the embolism of the aortic bifurcation, but on the one side (the aorta pulsation and major arteries on the other extremity is determined).

Embolism of the femoral arteries. The limbs are the most frequent localization. Often, the embolus is localized in the region of the bifurcation of the common femoral artery (in the place of branching of the deep artery of thigh) or over the entrance to the Hunter canal. The clinical picture is manifested by the symptoms of acute ischemia of the shin and foot (or the foot and distal half of the leg).

Embolism of the popliteal artery in the area of its bifurcation in the upper third of the shin or *arteries of shin* is less common, it does not always give a clear clinical picture (pronounced pain in the foot and lower portions of shin), often it is manifested by minor disorders of the skin sensitivity and motor function of the ankle joint and foot, although sometimes an ischemic necrosis of the muscles (tibial group) may develop, that is associated with increasing swelling of the muscles, which causes edema of the tissues and vessels in a tight fascial bed.

In acute arterial thrombosis of the extremities, unlike AEM, the onset of the disease is more often more gradual, and clinical manifestations are less pronounced - when walking or while other physical activity (and sometimes at rest), patients experience moderate intensive muscle pain in shin, foot, unpleasant sensations of cold, numbness of the limbs, sharp weakness, convulsive muscle contraction; in some cases the pain can develop gradually over several hours, and sometimes days, gradually progressing; the degree of sensory impairment (superficial and deep) varies widely - from a slight hyposthesia to complete anesthesia; prognostically threatening combination of three "A" - akinesia, areflexia, anesthesia (such cases usually result in gangrene) may develop. The phenomena of decompensation of the blood circulation and the development of the gangrene of the limb usually occur within 5-10 days from the onset of AAT and occur relatively less often than embolism (due to the pathogenic features – present at the time of acute thrombosis collaterals and acute occlusion of only one vessel in patients with COADL - chronic obliterating arterial diseases of the limbs) .

As it was above mentioned, in AAT, the proximal border of acute ischemia is not so clear, there is no strict compliance with the localization of the thrombus and the level of ischemic disorders, the manifestations of acute ischemia are less pronounced, the severe degree of acute ischemia and gangrene is rarer than in AEM.

Diagnosis of AAO (acute arterial occlusion) syndrome of limbs in most patients is not difficult, because it is characterized by a vivid clinical picture. Some difficulty can be caused by establishing of etiology, that is arterial embolism or acute arterial thrombosis. In this regard, significant findings are provided by the history data - embolic diseases or chronic occlusive diseases of the arteries of the limb in the patient's history. It is important to establish *the level of acute occlusion*, which can be determined clinically by palpation of the limb at the projection points of the major arteries (determination of the presence or absence of pulsation).

The leading clinical syndrome *that is characteristic for the ALIES (acute limb ischemia syndrom) is acute pain in the leg.*

In accordance with the standard schemes, the plan for ***additional laboratory and instrumental examination of a patient*** with acute limb ischemia syndrome in a specialized vascular surgical department includes revealing of characteristic pathological changes:

1. *Clinical blood test* - possible leukocytosis and elevated ESR (erythrocyte sedimentation rate).
2. *Clinical analysis of urine* - possible signs of toxic nephropathy.
3. *Biochemical blood test*: in atherosclerotic lesions of the heart and major arteries, an increase in the content of cholesterol, low density lipoprotein are characteristic.
4. *Coagulogram:* characterized by hypercoagulation (reduced coagulation time; increased prothrombin index, fibrinogen A, fibrinogen B, fibrin, etc.).
5. *Oscillography* (in a policlinic setting) - sharp decline of oscillographic index (OI).
6. *Longitudinal segmental Rheovasography* (in a policlinic setting) - sharp decrease of the rheographic index (RI), changed shape of the rheographic curve.
7. *Ultrasonic examination of vessels* (including Doppler ultrasonography), which allows to determine the localization and timing of the blood clot formation.
8. *Angiography* can most accurately determine not only the level and extent of acute occlusion and chronic stenosis, but also get clear findings of its length, severity of concomitant arteriospasm, the intensity of collateral blood circulation; conducting Seldinger puncture

arteriography of thigh, puncture translumbar abdominal aorta (by Dos Santos), catheterization aortography (in impaired brachiocephalic arteries).

*Angiographic signs in **AEM*** with partial arterial obturation, the embolus is well circulated with contrast and the arteriogram shows an oval or round shape; in case of complete occlusion, there is a sharp "breakdown" of the shadow of the vessel (*a symptom of 'amputation stump'*) with a well-defined convex upper border of embolus (symptom of "inverted cup" by V.S. Saveliev), the contours of the afferent limb of the occlusive artery are even, smooth, no collaterals, as a rule , occur (Figure 82); *at **AAT*** (Figure 83), the thrombosis level has the shape of an uneven line with grooves, the arterial pathways which are proximal to occlusion, are rugged, filling with contrasted blood is unequal, sometimes a phenomenon of the contrast medium gradual disappearance is seen, collateral net is often well-developed.

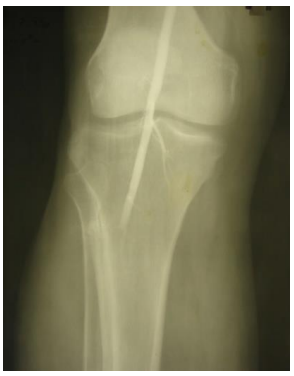

Fig. 82. Popliteal artery embolism in the site of its bifurcation

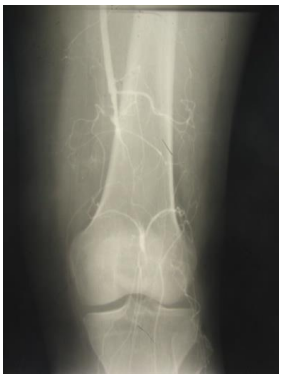

Fig. 83. Acute thrombosis of superficial femoral artery in the lower third at its atherosclerotic impairment

Differential diagnosis. Differential diagnosis is performed between the syndrome of acute limb ischemia and the aortic exfoliating aneurysm, with "white pain phlegmasia (static gangrene)", between arterial embolism and acute arterial thrombosis of the limb (Table 27).

Table 27

Differential diagnosis of AEM and AAT

No.	Differential diagnostic signs	Embolism	Thrombosis
1.	Major disease	Atherosclerotic and rheumatic heart disease, fibrillation arrhythmia	Chronic occlusive disease of limb arteries
2.	Beginning of the disease	Sudden	Relatively slow
3.	Pain syndrome	Very intensive	Mostly moderate
4.	Acute ischemia	Sharply pronounced, precisely limited	Moderate, sharply outlined
5.	The color of the skin of the limb	Pale, almost pale or 'marble'	Pale
6.	Skin temperature	Sharply decreased	Moderately reduced
7.	Motor function	Ischemic paralysis	Decreased muscle strength, paresis
8.	Skin sensitivity	Anesthesia	Decreased, but preserved
9.	Angiography	Contrasted segment of the major vessel with unaltered wall, with a clear level of breakage, with a convex and smooth upper limit shaped like an "inverted cup", no collaterals	Contrasted segment of the major vessel with uneven "eroded" contours, the line of the breakage is uneven, collateral network is pronounced

Treatment of patients with ALIES. *Choice of medical tactics.* Treatment for patients with ALIES should begin immediately after the diagnosis is made.

The first medical aid given at the prehospital stage (or in the clinic and in the non-specialized hospital) includes medications aimed *at preventing the progression of acute ischemia and thus, the extension of the the viability of the limb tissues*. Intravenous *promedol or even morphine* is administered to relieve pain; intravenous administration of large doses of *antispasmodics (no-spa, papaverine, complimin, halidor, etc*.) is used for arteriospasm and for the purpose of opening collaterals; intravenous direct anticoagulant heparin 5-10 thousand U is administered to prevent further growth of the thrombus (prolonged thrombosis) by creating a hypocoagulation state. Intravenous cardiac *glycosides (corglicon, strophanthin*); according to indications *antiarrhythmic drugs (novocainamide, etc.)* are used to *improve central hemodynamics.* The massage, warming the distal limb parts are prohibited, the patient should be hospitalized ugrently in a specialized vascular department.

In a specialized hospital, the choice of method of treatment depends, first of all, on the *cause* of the acute ischemic syndrome of the limbs and on the *degree of acute ischemia*. *Arterial embolism* in acute ischemia of any degree is an *indication for urgent surgical intervention.* In *acute arterial thrombosi*s, the choice of treatment depends mainly on *the degree of acute ischemia*: at a *mild degree I of ischemia*, medical therapy is indicated; at *stage II of ischemia (moderate)* - medical therapy is tried, in case of no effect: the urgent delayed surgical treatment is indicated; at the *severe degree III of* acute ischemia and the *degree IV* gangrene stage - an urgent surgical treatment after the necessary minimal examination and short-term preoperative preparation is administered.

Treatment of patients with arterial embolism. The diagnosed "arterial embolism" with degree I-III acute ischemia is an indication for urgent surgical intervention - the operation of embolectomy, that is, AEM should be treated urgently surgically.

The patients with absolute contraindications to this operation are excluded:
1) extremely severe (pre- or agonal) state of the patient, which excludes providing of any volume of surgical intervention and requires the conclusion of a medical council consultation;
2) in case of embolism of small diameter arteries (legs and forearms) when medical therapy is used;
3) the refusal of the patient and relatives from the operation.

All other possible contraindications are relative and are considered individually in each individual case.

Surgical treatment of AEM. For the treatment of AEM of limbs and aortic bifurcation, embolectomy is used currently, which can be considered a radical treatment.

Two methods of embolectomy are performed:

1) *direct embolectomy* (Figure 84) with exposed area of acute occlusion of the artery in easily accessible separated segments - in the region of the bifurcation of the common femoral artery on the thigh below the inguinal ligament, the shoulder arteries, and, less frequently, the popliteal artery;

2) *indirect embolectomy* is performed in acute occlusion of the major arteries in segments that are difficult to be accessed - bifurcation of the common iliac artery, aorta, at the localization of the embolus in the superficial femoral artery above the entry to the Hunter canal, in the shoulder artery on the shoulder, etc., is carried out with a *Fogarty balloon catheter* (thin 2.0-2.5 mm diameter elastic probe with latex balloon-blown at the end – Fig. 85).

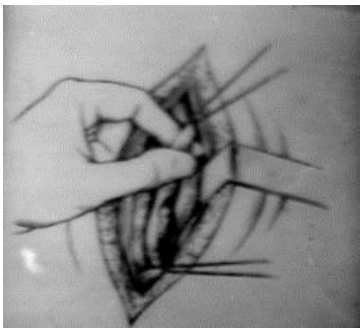

Fig. 84. "Direct" embolectomy

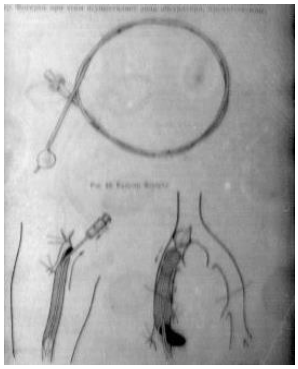

Fig. 85. "Indirect" embolectomy with a Fogarty balloon catheter

The most common *accesses* for both types of embolectomy are (Figure 86): in the upper third of the thigh below the inguinal ligament with the bifurcation of the common femoral artery, in the upper third of the forearm below the elbow flexion with the bifurcation of the shoulder artery, in the upper third of the shin on the posterior surfaces below the popliteal fossa.

If the balloon catheter is not available, polyethylene catheters of different diameters can be used for indirect embolectomy to *exhale the embolus*.

The *"retrograde drainage"* technique (on the lower limb by separating by the anterior ankle and catheterization of the posterior tibial artery, and radial artery on the upper limb) may be used to remove the distal prolonged thrombi or distal "surface" emboli.

After performing direct or indirect embolectomy, the artery suture (most often by the Carrel treatment) is done if transverse arteriotomy was performed; after longitudinal arteriotomy, to avoid stenosis and postoperative thrombosis - the arteriotomy opening is closed by sewing a "patch" from the autovein (a segment of a large subcutaneous vein).

All types of embolectomies are performed under local anesthesia, which allows to perform it without special risk, even in severe patients.

In the postoperative period - anticoagulant therapy (heparin, preferably - LMWH - low molecular weight heparins), disaggregation drugs, hemocorrectors for prophylaxis of postoperative thrombosis, treatment of the underlying disease, considering the probable radical correction to prevent possible recurrence of AEM.

It should be noted that in this area of urgent vascular surgery, minimally invasive operations are introduced currently in this area of urgent vascular surgery - *percutaneous aspiration embolectomy*.

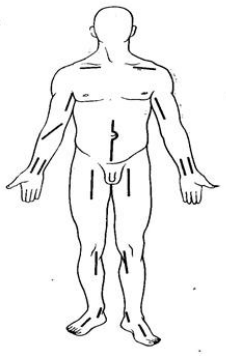

Fig. 86. Typical surgical approaches at embolectomy operations in case of arterial embolism of upper and lower extremities

Treatment of patients with acute arterial thrombosis. Medical treatment is indicated:

1) at a mild degree of acute ischemia of limb tissues;

2) as a trial therapy at a moderate degree of ischemia;

3) when the severity of the patient does not allow the surgery, despite the evident indications for it;

4) when a patient refuses surgery;

5) at acute thrombosis of peripheral arteries of small diameter (legs and forearms).

The task of medical treatment are the following: lysis of thrombus - restoration of permeability and blood flow in the artery with thrombus, prophylaxis of thrombus growth and spread, improvement of blood circulation and tissue metabolism in the zone of acute ischemia,

improvement of the function of vital organs, prevention of progression of the underlying disease (atherosclerosis).

Pathogenetically grounded therapy with AAT is thrombolytic and anticoagulant therapy, aimed at restoring the permeability of the occluded artery, against the growth and spread of the prolonged thrombosis, thereby improving blood circulation and tissue metabolism in the affected limb.

Thrombolytic drugs have a high effect of lysis, either directly affecting the thrombus, or activating the own fibrinolytic system of the patient, as activators of inactive plasminogen proactivators (fibrinolysin) and blocking the effect of inhibitors of fibrinolysis.

The following medications are used as thrombolytic drugs: *indirect thrombolytics - kabikinase, streptokinase*, etc., *direct natural urinary thrombolytic agent urokinase and direct thrombolytics (synthetic tissue plasminogen activators) - Actilise* (active ingredient - *alteplase*), *Metalise (tenecteplase), Reteplase*.

Intravenous administration of thrombolytics is possible for *systemic thrombolysis* and more effective *regional thrombolysis* - delivery of the drug to the site of thrombosis with the aid of an infusion catheter.

Considering the antistreptrococcal antibodies which are present practically in all people in the blood, *thrombolytic therapy by an indirect thrombolytic (streptokinase*) is started with the intravenous administration of the initial "inactivating" dose of the drug at 250 000 U per 300 ml of saline, or 5% glucose solution for 30-40 minutes , followed by systemic thrombolysis - continuous intravenous infusion of the therapeutic dose of the drug, which can reach 1 500 000 - 3 000 000 U of the drug (rate of infusion - 750 000 U for 8 hours); it is conducted under angiographic control with the subsequent heparin therapy.

Actilise (alteplase) is a direct synthetic thrombolytic, which is usually initially injected in a dose of 10 mg intravenously within 1-2 minutes, then 90 mg intravenously for 2 hours. The total dose of the drug is 100 mg for 2 hours. For the patients whose weight is less than 65 kg, the total dose should not exceed 1.5 mg / kg.

To avoid the main *complication of thrombolytic therapy* - various *bleedings*, careful laboratory control of the state of the hemocoagulation system is required: every 8 hours the coagulogram parameters are studied: *blood coagulation time, fibrinogen A concentration, thrombin time, fibrinolytic activity of blood.*

The rate of infusion of the thrombolytic drug should be such as to maintain thrombin time 2-5 times longer than the control time.

To reduce the toxic and antigenic effects of thrombolytic, the hormones (prednisolone, etc.) and desensitizing agents (suprastin, tavegil, pipolphen, etc.) are used.

In case of *hemorrhagic complications* (bleeding of wounds, micro-and macrohematuria, coagulogram parameters) – administration of thrombolytic agent is temporarily stopped; injection of *fibrinogen, 5% solution of ε-aminocaproic acid, 1% solution of calcium chloride, native or fresh frozen plasma, 1% Ambene solution is used as an antidote therapy*. After the termination of the manifestation of hemorrhage, thrombolytic therapy is continued until a therapeutic effect is obtained, but by a slower administration of the drug.

Anticoagulant therapy, which is started after the end of the thrombolytic therapy, or which is used as the main method in the treatment of AAT with localization below the inguinal ligament, has *antithrombotic effect* (inhibits thromboxinase and inactivates thrombin) and causes a decrease in the coagulation potential of the blood, prevents the formation and growth of prolonged thrombus, collapse of collaterals , has an antispasmodic effect and reduces the viscosity of the blood, but has no effect to dissolve the formed thrombus, and does not eliminate the embolus (although the vascular permeability may partially renew due to the further clot retraction under hypocoagulation of blood).

Anticoagulant therapy, as a rule, begins with administration of a *direct anticoagulant – heparin* - only parenteral administration, optimally intravenous, 5000-10000 units (1-2 ml) every 3-4 hours (as it is excreted from the body during this time), the daily therapeutic dose ranges

from 40 000 to 80 000 units (depending on the caliber of the occluded vessel); the course of treatment with heparin lasts 3-5 days, is decreased gradually in dose and is subsequently changed to indirect anticoagulants.

Heparin, similarly to thrombolytics, can cause *hemorrhagic complications* (although this is registered relatively rarely due to the fact that it is not cumulative in the liver). Therefore, when it is used, a careful laboratory control is also required: a ***daily*** study of the main levels of the coagulogram (the main control level is the *time of blood coagulation* by Lee-White, which is extended for effective therapy to 20-30 minutes), and microscopy of the urine for the detection of *microhematuria*.

When ***signs of overdose*** appear (bleeding of wounds, micro- and macrohematuria, evidences of the coagulogram), 1-2 injections of heparin are missed, a solution of ***protamine chloride or protamine sulfate*** (intravenously 5-10 ml in physiological solution) is used ***as an antidote***, then heparin therapy is continued with lowered doses. *It is unacceptable to discontinue heparin therapy abruptly*, as the opposite complication is possible to develop, that is, the so-called *"rebound events"*, when after abrupt withdrawal of heparin, blood coagulation increases dramatically and rethrombosis may develop.

Recent years heparin has been successfully replaced by *low molecular weight heparins* (LMWH - products of heparin fractionation) - fraxiparine (30 mg - 0.3 ml in a syringe - 2-3 times a day), Clexane (enoxaparin - 20-40-80 mg-0.2-0.4-0.8 ml in a syringe - 2-3 times a day); these drugs are administered only subcutaneously in the area of the anterior abdominal wall.

The course of treatment by direct anticoagulant is currently combined with indirect anticoagulant therapy (phenylene, syncumar, omepin, or warfarin), which have, besides the used antithrombotic properties, some negative effects: the highly variable sensitivity in different patients (some of them demonstrate elevated sensitivity, that is shown as bleeding even for normal doses and in short-term use; in others it shows tolerance, even the increased doses have insignificant effect), the ability to accumulate in the liver and in the long-term use cause massive bleeding. Such combination therapy should last at least 4-5 days.

In this regard, for each patient, the therapeutic dose of the indirect anticoagulant should be selected *individually*, starting with the standard dose, and then, every day, checking the level of the prothrombin index, it is necessary to select a dose that will support this level of the coagulogram required for the therapeutic effect - 35-40 %. To avoid dangerous cumulation of the drug, the course of treatment should last no more than 7-10 days.

Standard dosage: warfarin 5 mg (2 tablets) 1 time a day in the evening after meals, phenylene 0.3 g 2-3 times a day, syncumar 0.4 g 2-3 times a day. *Taking into account the different sensitivity of the body to indirect anticoagulants, it is recommended to choose the dose individually!*

The rules of therapy with indirect anticoagulants are the same as for the direct anticoagulants: they can cause similar complications - *hemorrhages* (in case of overdose or prolonged use), *"rebound effect"* (at a sudden withdrawal of administration), and considering the above features (different sensitivity, cumulativity), these complications can be even more unpredictable. Therefore, very careful laboratory control is required: after a dose selected, every 3 days, the main checking rate of coagulogram for indirect anticoagulants - INR (international normalized ratio) or the prothrombin index (which should not be lower the therapeutic level of 35-40%) are studied, and microscopy of urine for microhematuria is performed, and by the end of the course the dose is decreased gradually to the minimum (0.5 tabl 1-2 times a day).

If before, the indirect anticoagulants were started at the last day of those direct administration (the so-called "intersect" rule, due to the "latent period" of the effect from indirect anticoagulants, calculated from 12-14 to 24 hours, when their hypocoagulation effect is not yet manifested), currently, the indirect anticoagulants (warfarin, syncumar, phenylene) are recommended 2 days after the start of treatment with UFH (unfractionated heparin) or with LMWH (low molecular weight heparins) under the control of the *international normalized ratio (INR)*. The injection of indirect anticoagulants is ceased after the achieved INR ranged from 2 to

3. If this method is not available, the level of prothrombin index is monitored, which is recommended to be reduced to 35-45%. The control frequency is once per 2-3 days.

If signs of an *overdose* of an indirect anticoagulant appear (bleeding of wounds, micro- and macrohematuria, evidences of coagulogram), 1-2 doses of the drug are missed, 1% vicasol solution is given as an antidote (5-10 ml intravenously), then the treatment is continued with reduced doses. *It is unacceptable to abruptly stop the intake of an indirect anticoagulant*, because, as with heparin therapy, the development of the opposite complication is possible, that is the so called "*rebound effect*", when after the sudden withdrawal of the drug the blood coagulation increases sharply and rethrombosis may develop.

At present, *new standards of thrombolytic and anticoagulant therapy* are proposed, which allow to improve the immediate and long-term results of "systemic" thrombolysis by improving the so-called adjuvant anticoagulant and antiplatelet therapy which is used concurrently with thrombolytics.

A prerequisite for the use of anticoagulants during thrombolytic therapy (the so-called anticoagulant escorting of "thrombolysis") is the release of thrombin from the thrombus during fibrinolysis, which leads to an increased pre-thrombotic state and increases the risk of rethrombosis on the surface of an unstable plaque.

The advantage of low molecular weight heparins (LMWH), mainly enoxaparin (clexane), to increase the clinical efficacy of thrombolytic therapy has been proved, and it has been shown to be superior compared to unfractionated heparin (UFH), and not only for fibrin-specific thrombolytics, but also for streptokinase.

The appropriateness of prolonged (up to 7-8 days) anticoagulant therapy with enoxaparin or direct anticoagulant of the new class of *Arixtra (Fondaparinux* - synthetic selective factor Xa inhibitor) has been proved.

The complex medical therapy includes *hemocorrectors* (rheopolyglucin, etc.), disaggregant (*acetylsalicylic acid, clopidogrel, ticlopidine*), antispasmodics, cardiac glycosides, antiarrhythmic drugs (if medically indicated), correction of acid-alkaline state, measures to normalize the function of the kidneys (if it is impaired).

Medical therapy is used as an independent method of treatment and is a necessary complement to surgical treatment.

Surgical treatment of AAT of the limbs uses the following *restorative operations: the "ideal" thrombectomy* (rarely) or its combination with endarterectomy, *endarterectomy with autovenous plasty, endarterectomy using the "twist" method followed by reimplantation of the autotransplant (in case of thrombosis of the aortic bifurcation), resection of the artery with prostheses and constant bypass shunting* (in the area of bifurcation of the aorta and iliac artery – with synthetic prostheses, on the limb – with autovein). Operations are performed under general anesthesia.

In the case of contracture of the muscles of the shin in acute arterial circulatory disorders (and in case of embolisms and thrombosis), in order to *reduce the risk of the development of threatening post-ischemic disorders*, it is recommended, along with the removal of the thrombus-embolus and prolonged thrombus, to carry out *phlebotomy* of the adjacent major vein with

bloodletting and revision of its balloon catheter with the aim to remove toxic products of disintegration and blood clots, lavage through the *artery-vein system*, or *regional perfusion of the limb*. In the case of *developed muscular swelling* for the purpose of decompression and improvement of blood flow in the muscles, extensive *fasciotomy* is also indicated.

In case of the development of the degree IV acute ischemia - the gangrene stage, both at AEM, and AAT, *amputation of the limb* is indicated. When choosing the level of amputation it is necessary to remember that ischemic muscle injury is usually expressed to a greater extent and it is more proximally than this of the skin. It is sometimes advisable to supplement amputation with embolectomy, which reduces the level of limb amputation.

Prevention of embolism and acute thrombosis of the limb arteries involves radical surgical correction of embologenous diseases (commissurotomy in case of mitral stenosis, excision of postinfarctional aneurysms of the heart, aortic aneurysm resection, etc.) and chronic occlusive diseases of the arteries of the extremities (reconstructive operations). In cases of severe general condition or the age of the patients do not allow these interventions, life-cycle anticoagulant therapy with indirect dose regimens with constant dose and constant laboratory control, in which the prothrombin index does not exceed 60%, is indicated. For these purposes, warfarin is most often used.

Patients who underwent AEM and AAT of the limbs should be followed clinically. Twice a year, and if indicated more often, such patients should be examined by a family doctor and a vascular surgeon. The coagulogram levels are obligatory to be monitored. If necessary, the vascular surgeon prescribes ultrasonography of the major arteries of the extremities.

IV.3. ACUTE THROMBOSIS OF THE MAJOR VEINS OF THE LIMBS THROMBOEMBOLISM OF THE PULMONARY ARTERY

Diseases of the veins are an urgent surgical issue which has not been solved completely yet.

It is topical due to large spread of vein diseases which are observed 7-8 times more often than diseases of the arteries of the limbs. The patients with this disease make a significant percentage of polyclinic surgical visits.

Worldwide statistics show steady increase of acute venous occlusive disease that is considered to be associated with the growth of cardiovascular and oncological diseases, with emerging and increasing spread of complex surgical interventions on the heart and major vessels, in the transplantation of organs requiring prolonged intravenous infusions, resulting in increased traumatism and blood loss, with widespread use of medications which increase coagulation of blood, etc.

At present, according to national and Russian scholars, acute deep vein thrombosis (ADVT) and their complications affect 1.5% of the population, mostly of active working age. 160 cases of acute venous thrombosis are registered per 100 000 population (K.M. Amosova et al.., V.S. Saveliev).

Acute venous thrombosis (revealed by *radiometry* and ultrasonography) is diagnosed in 5-20% of patients with myocardial infarction, 60-70% with a cerebral stroke, 10-15% with disorders of the internal organs.

ADVT (acute deep vein thrombosis) and their threatening complications (pulmonary artery thromboembolism) is reaching a leading position among the postoperative complications. According to Bergqvist D. et al. (2005), ADVT is developed after orthopedic surgery in 50-75% of patients, after prostatectomy in 40%, after abdominal and thoracic surgery in 30% of patients. Similar data are presented by Russian researchers (V.S. Saveliev, R.D. Konstantinova, 2006): acute thrombosis of deep veins develops after general surgery, on average in 29% of those operated on (that is, every third patient), after gynecological surgeries in 19% of patients, after urological operations (for subcutaneous adenomectomy) in 38%, after traumatological surgery (in cases of hip fracture) in 53%, after orthopedic surgery (hip replacement) in 59% of those operated on; after surgery for malignant neoplasms in patients aged over 60 acute deep vein thrombosis occurs in 66% of cases.

In recent years, national and foreign literature has increasingly used the term ‘venous thromboembolism’ (VTE), with thrombosis of deep veins (DVT) of the lower extremities as its most frequent manifestation and the most life-threatening pulmonary embolism (PE). These two manifestations of VTE are closely related: asymptomatic PE is found in 40% of DVT patients, and 70% of symptomatic PE patients have DVT of the lower extremities (White R.H., 2003).

VTE is ranked third among the causes of death resulted from cardiovascular disease after myocardial infarction and ischemic cerebral stroke (Heit J.A. et al., 1999), that determines

the exceptional importance of improving its diagnosis, treatment and prevention (V.G. Mishalov et al., 2008).

Clinical practice uses ***the L.I. Klioner's acute venous thrombosis classification*** (1969), with the following criteria: primary thrombosis localization, ways of its spread, etiology, clinical course, trophic and hemodynamic disorders.

I. Considering the primary thrombotic process localization and the ways of its spread, AVT is classified as follows:

A. Thrombosis of the inferior vena cava system:

1) thrombosis of the veins that drain the muscles of the shin;

2) iliofemoral thrombosis;

3) thrombosis of the suprarenal, renal, and infrarenal segments or thrombosis of the entire trunk of inferior vena cava;

4) caval iliofemoral thrombosis;

5) combined total thrombosis of the entire deep venous system of the inferior vena cava.

B. Thrombosis of the anterior vena cava system:

1) thrombosis of the trunk of the upper vena cava at the level of the opening of the azygos vein, anterior and inferior;

2) thrombosis of the entire trunk of the anterior vena cava;

3) thrombosis of the trunk of the anterior vena cava and innominate veins;

4) thrombosis of the axillary and subclavian veins;

5) total thrombosis of all deep veins of the upper limb.

II. Considering etiological factor:

1) after infectious diseases;

2) after injuries;

3) after operations;

4) after childbirth;

5) at varicose dilation of superficial veins;

6) at allergies;

7) in case of intravascular congenital and acquired factors (partitions, diaphragms, adhesions, atresia);

8) in case of intravascular congenital and acquired factors (compression of veins by arteries, tumors, aneurysms).

III. Considering the clinical course character:

1) acute thrombophlebitis;

2) subacute thrombophlebitis;

3) post-thrombophlebitis syndrome;

4) acute thrombophlebitis against the background of post-thrombotic syndrome.

IV. Considering the degree of trophic and hemodynamic disorders:

1) mild form;

2) medium form;

3) severe form.

Acute venous thrombosis is appropriately classified as ***acute thrombosis of superficial veins and acute thrombosis of deep veins of extremities***. Thrombosis of superficial veins is termed '*thrombophlebitis of subcutaneous veins',* which is common in patients with varicose veins of the lower extremities (in 34-65% of cases), and the term 'acute varicothrombophlebitis' may be used for them. The development of thrombophlebitis of superficial veins promotes infections, injuries, malignant neoplasms.

O.O. Shalimov considered it reasonable to define *3 consecutive stages of venous thrombosis: acute* (7-14 days), *subacute* (to 3 months) and *chronic* – post-thrombotic syndrome (after 3 months).

Acute superficial venous thrombosis (ASVT) of the extremities

The main surface veins of the lower extremities (major and minor subcutaneous veins), either unchanged, or varicose dilated, are affected mostly. As a result of intravenous injections (taking samples for analysis, infusion therapy, including through constant venous catheters), thrombosis of the superficial veins of the upper extremities (*post-injection thrombophlebitis*) develops.

Clinical manifestations. The history taking characterizes the patient by the *complaints* of discomfort, malaise, general weakness, subfebrile fever (sometimes chills may occur), pain and painful induration along the affected segment of the superficial veins of the shin or thigh, walking which is somewhat difficulties due to pain in the leg.

In ***acute varicothrombophlebitis***, patients complain of pain in one or more varicose nodes of the shin or pain in the thigh in the region of the thrombosed trunk of the major subcutaneous vein of the thigh. It is reported that the affected sites become painful, indurated and do not disappear in a horizontal position.

History of the disease: most often, thrombosis of the superficial veins of the lower extremities, both in the unchanged, and varicose, is developed acutely. Among the factors contributing to the development of acute thrombosis, the recent infectious diseases, operations, traumatic injuries, present malignant neoplasms, allergic diseases are of value. The course of the disease is acute, progressive.

Objectively: general condition, as a rule, is relatively satisfactory, subfebrile fever is observed. The patient is somewhat adynamic due to pain in the extremity, which is more pronounced when walking.

Local status. Examining of the affected lower limb with ***previously healthy veins***, shows hyperemia and swelling of the skin, painful "cord-like" induration in the affected segment of the vein of the shin or thigh, that is usually clearly separated from the surrounding tissues. In case of thrombosis of the segment of the trunk of the *major subcutaneous vein*, the pathological focus is located on the anterior-inner surface of the leg or thigh. In case of thrombosis of the segment of the trunk of a *minor subcutaneous vein*, the focus is on the posterior-external surface of the leg. The circumference of the affected limb, as a rule, is not changed, that means that the swelling is not characteristic.

Acute thrombosis of unchanged superficial veins of the limb, especially in case with no factors in history (surgery, trauma, etc.) should be interpreted as a possible *Fischer symptom*, which indicates a high probability of having a malignant neoplasm of any localization in the patient (especially at the age over 40) and the need for an urgent examination to reveal it.

In acute thrombosis of *varicose veins*, hyperemia and swelling of the skin in the region of thrombogenesis of varicose nodules (more often on the shin) and in the projection of the trunk of the affected varicose vein (more often on the thigh), which become dense, painful when palpated, do not disappear when raising the foot upward.

In *postinjection thrombophlebitis of the superficial veins of the upper limb along the affected vein* above the elbow flexion the band of hyperemia and palpable dense painful cord-like induration are determined.

Leading syndrome *– pain in the leg*, ***leading clinical symptoms*** *- pain and painful induration in the projection of subcutaneous veins of the limb.*

According to the standard schemes, ***plan of additional laboratory-instrumental examination of a patient with acute thrombosis of the superficial veins of the limbs*** includes:

1) clinical blood test;
2) clinical urinalysis;
3) biochemical blood test;
4) coagulogram;
5) urgent Doppler ultrasonography examination of lower limb veins.

Possible pathological changes.

1. *Clinical blood test*: changes are nonspecific, possible leukocytosis, a slight increase in ESR (erythrocyte sedimentation rate).

2. *Clinical urinalysis*: no characteristic features.

3. *Biochemical blood test*: no characteristic features.

4. *Coagulogram:* disordered blood coagulation towards hypercoagulation.

5. *Urgent ultrasonography examination* makes it possible to determine with absolute accuracy the presence, localization and true extent of thrombosis; special attention is paid to the proximal margin of thrombosis, due to the possibility of thrombus detachment and PE (pulmonary embolism) development.

Differential diagnosis is carried out with other pathological processes, most often of inflammatory nature, which are localized in the skin and subcutaneous tissue, such as: lymphangitis, erysipelas, nodular erythema, allergic dermatitis, constricted femoral hernia.

Treatment of patients with acute thrombosis of superficial veins of the limbs.

Therapeutic measures should be aimed at solving the following tasks:

1) prevention of the spread of thrombosis, including deep veins, and development of pulmonary embolism;

2) rapid relief of inflammatory events in the walls of the vein and surrounding tissues;

3) exclusion of recurrent thrombosis of superficial veins.

Choice of medical tactics. In acute thrombosis of superficial veins of the lower extremities *urgent surgical treatment is indicated* to exclude thrombosis relapse and pulmonary embolism development. A short-term delay of the operation (1-2 days) is possible for medical therapy, aimed at removing the pronounced process of periphlebitis to improve the conditions for surgery. *Medical therapy* is performed in case when a patient refuses surgery or at serious contraindications (extremely grave general condition, etc.), and as a preoperative preparation as well. Medical therapy is also carried in case of post-injection thrombophlebitis of brachial subcutaneous vein.

Pathogenetically grounded ***medical therapy*** for acute thrombosis of the superficial veins of the lower extremities is aimed at relieving inflammatory and local thrombotic processes, preventing the distal and proximal spread of thrombosis from the primary focus of occlusion and on deep veins, and at the prevention of the development of pulmonary embolism, as well.

1. Regimen - 2-3 days bed rest following with the partial bed rest with obligatory elastic bandage of the affected limb from the foot to the groin, which provides more intense blood flow in deep veins, preventing the development of thrombosis in them and prevent the development of pulmonary embolism.

2. Diet - no alcohol, no spicy foods.

3. *Medication therapy:* ***nonspecific anti-inflammatory drugs (NSAIDs)*** - *diclofenac* and its derivatives (*voltaren, ortofen, arthrotec*) and *ketoprofen* and its derivatives (*oruvail, ketonal, fastum*) that can be used as injectable, tablet and ointment; ***disaggregants and hemocorrectors*** - intravenous administration of *rheopolyglucin* 400 ml once daily, *trental* in a daily dose of 800-1200 mg, small doses of *acetylsalicylic acid*; ***anticoagulants***: *better local - lioton gel, hepathrombin, etc.; direct anticoagulants - heparin, fragmin, fraxiparine* are used in case of stable recurrence of thrombophlebitis in patients with pathology of hemostasis; ***venotonics*** - *Venoruton, Troxevasin, Detralex, Diovenor*, representing protectors of the venous wall and have an anti-inflammatory effect; ***oral polyenzymatic mixtures*** - *Wobenzym, Phlogenzym* are used due to the ability of hydrolytic enzymes to suppress the inflammation process, to present antiedemic and immunomodulatory effect, to remove immune complexes which are fixed in tissues and to activate fibrinolysis.

If in the process of medical therapy the evidences of the ***spread of thrombotic process*** (despite treatment) *proximally* (*ascending thrombophlebitis* of unchanged or varicose veins) are seen, *an urgent operation for life indications is prescribed* considering the real threat of the development of pulmonary embolism (through saphenofemoral junction). Either *minimal palliative surgical intervention* under local anesthesia - a *Trojanov-Trendelenburg's operation* when a patient refuses a radical operation is performed, or a radical operation (under general anesthesia).

Surgical treatment can be ***palliative and radical***.

The purpose of the ***palliative operations*** is to prevent the transition of thrombosis to deep veins through the saphenofemoral or saphenopopliteal junctions and prevent the development of pulmonary embolism. In this regard, a classic Trojanov-Trendelenburg's operation or its modification is performed.

Trojanov-Trendelenburg's operation (Figure 86 b): ligation and resection of the area of a major subcutaneous vein in the place where it falls into the femoral vein (the vein section is resected between two ligatures). Modern modification of this operation: high ligation of a major subcutaneous vein with obligatory ligation of all subostial branches and excision of the trunk of the subcutaneous vein within the wound. In case of spread of thrombosis above the ostial valve, thrombectomy is performed from the general femoral or even external iliac vein with pressing the external iliac vein above the top of the floating embolus.

Radical surgical treatment with *acute* varicothrombophlebitis involves not only eliminating the risk of developing deep vein thrombosis and pulmonary embolism and treatment of thrombophlebitis itself, but also treatment of varicose veins: conventional radical operation - ***Babcock-Narat phlebectomy*** (Figure 87) which is being started by modern modification of Trojanov-Trendelenburg's operation (in case of thrombosis of minor subcutaneous vein - ligation and its crossing it in the place of junction into the popliteal vein), carry out the obligatory removal of all varicose dilated veins (thrombosed and non-thrombosed) from *Narat* projection accesses with obligatory ligation of perforated veins with valvular insufficiency; to remove clotted segments of the trunk of the subcutaneous veins, the *method of "tunneling"* is used.

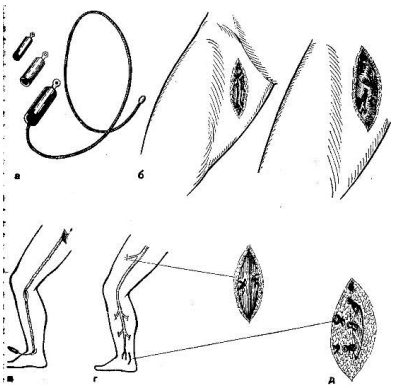

Fig. 87. Babcock-Narat surgery for varicose veins: a - one of the Babcock's probe modifications; b - Trojanov-Trendelenburg's operation; c - removal of the trunk of a major subcutaneous vein with the Babcock's probe, d - removal of varicose nodes and varicose dilated lateral branches from Narat projective access; e - Cockett's operation (suprafacial ligation of communicative veins over the ankle area).

Radical surgical treatment for *acute ascending thrombophlebitis of the unchanged* major subcutaneous vein includes the ***Trojanov-Trendelenburg's operation*** and the *removal of the entire thrombosed venous segment* from *Narat's* projection access using the *"tunneling" method*.

In the postoperative period, the introduction of low molecular weight heparins continues (fraxiparine), NSAIDs, disaggregants and analgesics. Moderately active motion regime with using elastic bandage is recommended for patients from the first day after the operation.

Possible ***complications*** of acute thrombosis of subcutaneous veins - abscess, spread of thrombosis to deep veins of the limb, development of pulmonary embolism.

Acute Deep Vein Thrombosis of the Lower Limbs (ADVT)

Acute thrombosis of deep veins of extremities is dangerous due to the consequences of the disease. Major veins of significant diameter are feasible to form a large-sized thrombus at risk of embolus in them, and intensive blood flow creates conditions for its easy detachment and

development of pulmonary embolism with possible fatal outcome. In a large percentage of cases, thrombosis of deep veins of the lower extremities leads to the development of post-thrombotic syndrom (PTS), which manifests with chronic venous failure, often with the development of trophic ulcers, which significantly reduces the working capacity and quality of life of patients.

Clinical manifestations. At the interview, the patient presents complaints of edema, cyanotic limbs, bursting pain in the shin of the leg, in the popliteal fossa, on the thigh, which increases with movement, local increase in skin temperature, fever, general weakness, headache, palpitations.

Case history: most often the acute thrombosis of deep veins of the lower extremities develops acute. The important factors contributing to the development of acute thrombosis are recent history of infectious diseases, operations, traumatic lesions, malignant neoplasms, allergic diseases, and the often cause in women are tumors of the uterus and adnexa, complicated childbirth and postpartum period. The course of the disease is acute, progressive.

Objectively. Common general condition is of moderate severity. The forced position is noted due to pain difficulty of walking, febrile temperature. Patient is somewhat adynamic due to pain in the extremity. Tachycardia.

Local status. *The main symptoms* are: varying degrees of severity of edema of the limb (depending on the localization of thrombosis), cyanotic coloration of the skin and elevated skin temperature of the affected limb, enlarged subcutaneous venous network. *The main symptom is pain at palpation in the projection of affected deep veins*: on the shin along the posterior surface between the venter of the calf muscle, in the popliteal femur, in the anterior-inner thigh, in the femoral triangle, in the inguinal-iliac region of the abdomen.

In thrombosis of the deep veins of the shin, a number of *pathognomonic symptoms* are identified: *Homans symptom* (Homans, 1941) which includes the appearance of severe pain in the calf muscle with a sharp dorsal flexion of the foot; *Meyer's symptom* that is the pain at palpation along the inner edge of the tibia in the middle third of the shin; *Pair's symptom* which includes the pain at palpation of the medial surface of the arch of the foot; *Opitz-Ramínes* symptom of pain at squeezing the calf muscle with your hand; positive *Lowenberg's test* (Lowenberg, 1954) that means appeared severe pain in the calf muscle at compression of the shin in the middle third with a sphygmomanometer cuff even at a minimum pressure of 30-40 mm Hg; *Moses test* (Moses, 1946) which includes appearance of pain at compression of the shin with your hands in the anterior-posterior direction and no pain at compression from the sides.

It should be noted that the clinical symptoms of ADVT are significantly variable, it is possible to diagnose the localization of thrombotic lesions of deep veins of the limb according to the severity of symptoms and, especially, *the level of localization of edema*.

So, in case of thrombosis of deep veins of the lower limb below the mouth of the deep vein of the thigh *("critical point"*) due to the relatively high efficiency of the collateral compensatory blood circulation, the symptoms and, in particular, edema of the limbs will be expressed moderately: in case of thrombosis of one or two deep veins of the shin, swelling will be determined only at the level of the foot and ankle joint; if thrombosis of the popliteal vein occurs, it is in the region of the foot and shin; thrombosis of the superficial femoral vein below the mouth of the deep vein of the thigh cause swelling which will spread to the distal portion of the thigh.

In acute thrombosis of *all deep veins of the shin* (anterior and posterior tibia, fibula), as well as at the thrombosis of the popliteal vein on the shin, there is a marked disorder of the venous blood outflow, the shin becomes swollen, tight, its perimeter, as compared with the healthy shin, increases 4-5 cm and more, the pronounced pain syndrome is registered, the patient is concerned about the bursting feeling, tension in the shin, skin cyanosis appears on the foot and in the lower third of the shin; in 2-3 days the swelling somewhat decreases and enlarged subcutaneous veins on the shin, sharp pain of calf muscle during palpation, pain in the popliteal region are seen, all the above-described symptoms are positive.

When the process spreads to the *superficial femoral vein*, and at its primary thrombosis, as well, the patients register the pain on the medial thigh surface along the projection of the Hunter canal; here the pain is determined by palpation, the area of which approximately corresponds to the length of the thrombotic site of the vein; venous blood circulation in the area of the knee joint can be disturbed, the pain appears, the contours are smoothed, its volume increases, function abnormalities are determined; swelling and enlargement of the hypodermic venous network in the distal thighs are seen.

A separate nosological form, the ***acute iliofemoral venous thrombosis of the lower extremities*** (acute occlusion of the common femoral, and of external, and sometimes of the common iliac veins, above the *"critical point*"), that, due to the low efficiency of the compensatory collateral circulation, demonstrates the most pronounced clinical picture of acute disturbance of venous blood circulation in the extremity. It often develops in women in the early postpartum period (especially after complicated delivery requiring manipulation on the uterus); it has an initial prodromal stage (compensation stage) and a stage of marked clinical manifestations (decompensation stage).

The main symptoms in the prodromal stage are the elevated temperature and pain of various localization (in extremity, in the lower abdomen, in the lumbosacral region); pulmonary embolism is possible.

The *stage of pronounced clinical manifestations* is characterized by a classic ***triad of signs***: *pain, swelling, discoloration of the limb.*

Extensive *pain*, localized in the inguinal region, along the anterior-medial thigh surface, can spread to the calf muscles due to stretching of the walls of the major vein due to venous hypertension and the thrombus present in the lumen of the vein, as well as the development of periphlebitis; the patients note the feeling of heaviness, tension in the extremity, its rapid increase in volume.

Edema of the limb is the most pathognomonic symptom of acute iliofemoral venous thrombosis, due to the high localization of venous occlusion, it is disseminated, *involving the entire lower limb* (from the foot to the inguinal ligament), often spreading to the lower abdomen and the buttocks; both venous stasis and disordered lymph flow contributes to its development.

Discoloration of the skin is more often diffuse, less often it is "spotty" cyanosis, and sometimes pallor of the skin is observed (diffuse cyanosis is due to the dilation of venules and capillaries, as well as increased oxygen utilization associated with the slowing down of blood flow in the extremity; spotty cyanosis is due to stretching of the small veins and extravasates from erythrocytes; milk-white coloration of the skin is caused by severe lymphatic drainage disturbances, rarely arteriospasm).

Intensified pattern of hypodermic veins on the thigh and, especially, in the inguinal region is a frequent and very important symptom of acute iliofemoral venous thrombosis, but it is not at the same time an early symptom (it appears at days 3-5 of the disease after reduced edema of the limb).

Some patients may experience *symptoms of "psoitis"*, i. e. pain and tenderness in the iliac region at maximum hip flexion, flexor contracture of the thigh, *symptom of "adhered heel"* (due to pronounced periphlebitis in the region of the total iliac vein, which is in proximity to the iliolumbar muscle).

The special clinical forms of acute thrombosis of the major lumbar veins include "*white and blue pain phlegmasia" (phlegmasia alba dolens and phlegmasia cerulea dolens).*

*"**White phlegmasia***" or "*white tumor of thigh*" is characterized by fast-growing edema of all tissues of the lower limb. It occurs at thrombosis of the iliac and femoral veins with *simultaneous total arteriospasm*, resulting in prevailing clinical symptoms of limb ischemia, resembling embolism or acute thrombosis of the major arteries of the limb, and it explains why phlegmasia alba is also called *pseudo-embolic or ischemic phlebitis*. Most often, it happens as a postpartum complication of septic endometritis. A puerpera, after a fever and a temperature increase to 39-40º C, develops a rapidly increased volume of the limb, which begins from the

thigh and descends to the ankle and foot; the edema by the end of the day reaches much larger sizes than in the common acute iliofemoral venous thrombosis, often spreading to pudendal lips, skin is tense, shining, milky white, very painful at contact; no pulsation of the major arteries is felt; simultaneously, severe pains in the whole limb develop. The course of phlegmasia alba dolens is severe and prolonged (2-3 weeks or more), the limb edema subsides very slowly in its elevated position; for 6-8 weeks, the patients remain bedridden, despite the fact that the course of the disease is relatively benign and rarely leads to fatal outcome.

"Blue phlegmasia" (or ***Gregoire's disease***), in contrast to white phlegmasia, has an extremely severe course with threatening complications and it often results with an unfavorable (fatal) outcome (mortality reaches 60%), which is due to massive thrombosis at the level of venous macro- and microcirculation, and even total thrombosis of the entire vascularization of the lower limb, sometimes involving the arterial system. The beginning of the disease is acute, suddenly an intense, *"throbbing*" pain in the limb and in the buttocks, a sharp feeling of *"bursting"* appear (according to the patients, it is an impression that the skin of the leg is "ready to burst"); the *movements in the limb are impossible* due to acute pain, *swelling* develops rapidly and becomes pronounced within several hours and does not decrease with raised position of the limb; the skin is tense, shining, not folded. At the same time, violet tinge *cyanosis* appears, which involves not only the lower limb, but also the lower abdomen and buttocks on the side of the lesion, often it is steady and "spotty" in nature; diffuse ***hyperesthesia of the skin*** develops on the hip and shin, at the same time on the foot it is reduced or absent; the skin is cold to the touch, *no pulsation of the major arteries is not determined*. On the skin there are areas *of petechial hemorrhages*, which, merging, form the purulent cyanotic ***spots*** with subsequent epidermal detachment and the formation of *hemorrhagic blisters* with a stinking smell, indicating the onset of *venous gangrene*. The general condition of patients reaches the extreme degree of severity, hyperthermia and hyperleukocytosis are observed, often hepatorenal or multiple organic failure, hypovolemic and septic shock clinical manifestations add, often pulmonary artery thromboembolism develops, and patients rather rapidly die.

Acute thrombosis of the inferior vena cava. Primary thrombosis of the inferior vena cava (IVC) due to its large diameter and high blood flow rate is rare. Only some patients develop primary IVC obstruction. These are individuals with congenital IVC anomalies as partitions, diaphragms, atresia. Thus, in most patients, *IVC thrombosis results from ascending iliofemoral thrombosis.*

Clinical picture *depends on the level of the IVC trunk affected.* Typically, the thromboses of the subrenal (infrarenal), renal and hepatic segments are classified.

The general condition of the patient depends on the level of IVC occlusion. At high occlusions of the IVC trunk, the state of patients is extremely grave. In case of the infrarenal IVC segment affected, the state of patients varies between the severe and of moderate severity. Consciousness is preserved.

Complaints (in case of affected *infrarenal portion*) are as follows: extended abdominal pain that disseminates to the lower extremities, inguinal areas, buttocks; edema of the lower extremities, external genital organs, buttocks, anterior abdominal wall; pronounced pattern of enlarged subcutaneous veins on the thighs and lower abdomen.

Case history: a characteristic beginning of the disease starting with acute thrombosis of deep veins of the lower limb (more often of the iliofemoral segment) with its subsequent ascending spread. In most patients, IVC thrombosis is a consequence of ascending iliofemoral thrombosis, in rare cases the primary thrombotic localization is in IVC.

In the *thrombosis of the IVC renal segment*, the first clinical sign is pain in the lumbar region in the projection of the kidneys. Then oliguria, sometimes anuria with signs of uremia develop.

Thrombosis of the IVC liver segment leads to the development of a severe clinical picture due to impaired liver function, portal hypertension, thrombosis of the portal vein, which are the symptoms of the so-called *Budd-Chiari syndrome*, which develops in the primary

thrombosis of the hepatic veins. Clinically, the enlargement of the liver, spleen, ascites, pronounced enlargement of the superficial veins of the anterior abdominal wall and the lower half of the chest, marked edema of the lower extremities, discoloration of the skin, up to jaundice, dyspeptic disorders are registered clinically.

According to the standard patterns, the ***plan of additional laboratory and instrumental examination of a patient with acute thrombosis of the major veins of the extremities*** includes:

1. Clinical blood test.
2. Clinical urinalysis.
3. Biochemical blood test.
4. Coagulogram.
5. Plasma D-dimer tests (D-dimer-test, dimer-test - thrombotic process activity study).
6. Urgent Doppler ultrasonography examination of the veins of the lower or upper limbs, abdominal and pelvic organs.
7. Chest fluoroscopy.

Possible pathological changes.

1. *Clinical blood test*: changes nonspecific: possible leukocytosis, a slight increase in ESR.
2. *Clinical urinalysis*: no characteristic features.
3. *Biochemical blood test*: no characteristic features.
4. *Coagulogram*: Coagulation indexes more often correspond to hypercoagulation, however, the "*hypocoagulation of consumption*" can be determined.
5. Increase of D-dimer level in plasma more than 500 μg / liter.
6. *Ultrasonography examination of venous vessels* (Figure 88) gives an opportunity of accurate determination of the presence, localization, timing and true length of thrombosis; special attention is paid to the proximal margins of thrombosis, because a possible detachment of the embolus and the pulmonary embolism development. Ultrasonography of the pelvic organs may reveal a tumor as the cause of thrombosis.

Differential diagnosis is performed with other pathological processes, in which symptoms are swelling and pain in the extremity, such as: blood circulation failure, lymphostasis, traumatic edema, anaerobic phlegmon, tumors of bones and soft tissues, small pelvic tumors, arthrosoarthritis, acute arterial obstruction, gestational edema, prolonged tissue compression syndrome.

Treatment of a patient with acute thrombosis of deep veins of the lower extremities. In-patient treatment in hospital is obligatory, and advisable in a specialized vascular unit in accordance with the recommendations of the Ukrainian National Consensus (2006). Therapeutic measures should be aimed at solving the following *tasks*: to stop the spread of thrombosis; prevent the disease; prevent the progression of edema and thereby prevent a possible venous gangrene and loss of limb; to restore the permeability of the veins in order to avoid the further development of PTS. Therapy is conducted in accordance with the recommendations of the "Ukrainian Consensus on the Diagnosis, Treatment and Prevention of Venous Thromboembolism" (2010).

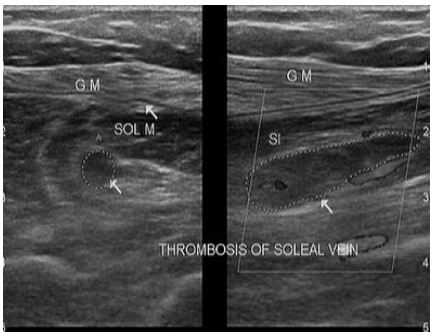

Fig. 88. Ultrasonography – acute thrombosis of deep vein of the shin

Choice of medical tactics. Currently, due to the available effective thrombolytic and anticoagulant drugs, even if acute thrombotic process are localized in the major trunk veins of the extremities, and in case of the timely hospitalization of the patient, the *medical method of treatment* is a *method of choice.*

Thrombolytic therapy is indicated in fresh massive iliofemoral venous thrombosis, as

well as in caval thrombosis, when there is a high risk of developing pulmonary embolism and venous gangrene. The duration of thrombosis in this case should not exceed 5-7 days.

Anticoagulant therapy is performed in ADVT localized on the thigh and shin.

However, none of the existing methods of medical therapy for thrombosis of the IVC system can serve as a reliable prophylaxis for such threatening complications as pulmonary embolism of the liver and kidney vein mouths. Therefore, recently, surgical treatment has been indicated more often in massive thrombosis of iliac vein and inferior vena cava with present floating blood clots in case of no possibility to carry out thrombolysis (high risk of developing a massive pulmonary embolism with a fatal outcome). Operative care is aimed not only at the prevention of complications, but, first of all, on the restoration of blood flow through the IVC and its branches.

Pathogenetically grounded ***medical therapy***.

1. Regimen: the first 3-5 days of severe bed rest (till the pain reduced and walking become possible, lowering the temperature, reducing the probability of developing pulmonary embolism), then moderately active (dosed walking) with obligatory elastic bandage on the limb.

2. Diet - restriction of spicy dishes, alcohol and food allergens.

3. ***Medication therapy.*** Pathogenetically grounded and most effective is ***thrombolytic therapy,*** which restores the permeability of the thrombotic vein. It is used for massive ADVT with the involvement of the iliocaval segment and the presence of floating blood clots for up to 5 days, as well as in cases of combed ADVT and PE.

The most commonly used method of *systemic thrombolysis* (intravenous drip infusion). At present, the most effective is thrombolytic therapy with a *direct plasminogen activator* ***Actilyse*** (a synthetic drug at a dose of 100 mg for 2 hours) followed *by the subsequent treatment with direct anticoagulants*. Other thrombolytic drugs are used - *indirect thrombolytics, streptokinase, kabikinase, direct natural thrombolytic urokinase*, etc.

One of the methods of thrombolytic therapy is *regional thrombolytic therapy (catheter-controlled thrombolysis*). After the angiography is performed and the diagnosis is confirmed, through the catheter, left in the vein, the infusion of the thrombolytic drug is done in proximity to the lesion focus, followed by angiographic control until the complete thrombolysis.

Thrombolytic therapy is controlled by the coagulogram findings - *the concentration of fibrinogen A and B, thrombin time, fibrinolysis activity.*

After the thrombolytic therapy is completed, the ***anticoagulant therapy*** starts, and supporting the state of hypocoagulation, prevents the recurrence of thrombosis, ascending thrombosis and pulmonary embolism. It starts with the use of *direct anticoagulants*.

The following treatment scheme is effective for the usage of *unfractionated heparin (UFH)*: during the first few days, UFH is administered by intravenous infusion *under daily monitoring with the Lee and White or Buerger test for blood coagulation time:* the target figure is considered to be 1.5-2 fold increase, and then subcutaneously (in the paraumbilical area) every 4-6 hours. The average daily dose is about 30-40 thousand units, the duration of therapy is up to 5 days.

If the *low molecular weight heparins (LMWH) - clexane* (enoxaparin), *fraxiparin* (nadroparin), are administered, the recommended dose is 1 mg / kg body weight every 12 hours, or 1.5 mg / kg once daily, duration of treatment on average up to 5 days. The advantage of LMWH therapy is that it requires no special laboratory control and practically causes no hemorrhagic complications.

After direct anticoagulants, the treatment is continued with *indirect anticoagulants*.

If earlier, the indirect anticoagulants were started at the last day of those direct administered (the so-called rule "intersect" due to the "latent period" of indirect anticoagulants, calculated from 12-14 to 24 hours, when their hypocoagulation effect is not manifested yet), currently the indirect anticoagulants *(warfarin, syncumar, phenylene*, etc.) are recommended 2 days after the start of treatment with UFH or LMWH *under the control of the international normalized ratio (INR).* Administration of indirect anticoagulants is ceased after the INR from 2

to 3 is achieved. If this method is not available, the control is the prothrombin index value, which is recommended to be reduced to the therapeutic level of 35-45%. Urinalysis for microhematuria is an obligatory monitoring. The frequency of the control is once in 2-3 days.

Dosage: *warfarin* 5 mg once daily, *phenylene* 0.3 g 2-3 times a day, *syncumar* 0.4 g 2-3 times a day (taking into account the different sensitivity of the body to indirect anticoagulants, it is *recommended to choose the doses individually!).*

The criterion for the effectiveness of anticoagulant therapy is the absence of ascending thrombosis and relapses of ADVT, the development of PE, as well as acute regression of the main symptoms of ADVT.

During the thrombolytic and anticoagulant therapy, there are serious complications: *hemorrhagic* - from increased bleeding of the gums while cleaning the teeth, nasal bleeding, small subcutaneous hematomas, microhematuria to intracranial and intracranial hemorrhage, severe macrohematuria with a reduced Hb of more than 30 g / L. In this regard, control is based not only on the coagulogram values, but also on regular urine tests (microhematuria), wound care (the appearance of hemorrhagic discharge).

In case of the development of hemorrhagic complications, the *antidotes of direct anticoagulants are protamine sulfate and protamine chloride* (intravenous infusions of 5-10 ml in the physical solution), *an antidote of indirect anticoagulants is vicasol* (intravenous administration of 3-5 ml in the physical solution).

The opposite complication, the so-called *"rebound effect*" is possible, when after an abrupt withdrawal of anticoagulants, ***rethrombosis*** *develops*, in connection with which in case of the developed hemorrhagic complications, the anticoagulant administration is *temporarily* interrupted, an antidote is applied, and then anticoagulant therapy is restored at lower doses, possibly with an increased time between administrations. For the same reason, gradual removal of anticoagulant medications (gradual dose reduction) is required.

For more information on thrombolytic and anticoagulant therapy, see Section below.

Obligatory components of complex medical therapy are *hemocorrectors and disaggregants* used for improving blood microcirculation, reducing its viscosity, reducing the aggregation of formed elements - *rheopolyglucine, pentoxifylline, trental, aspirin in small doses, thiclopidine, plavix. Non-specific anti-inflammatory drugs (NSAIDs) are diclofenac* and its derivatives (*voltaren, ortophen, arthrotec), and ketoprophen (oruvel, ketonal, fastum*) that can be used as injectable and tablet forms. *Venotonics - venoruton, troxevasin, detralex*, etc., which are venous wall protectors, improve venous hemodynamics and have anti-inflammatory effect.

Symptomatic therapy - analgesics, cardiotropics, and other drugs.

Surgical treatment. The tasks of surgical interventions in acute venous thrombosis of deep veins are restoration of the venous permeability and prevention of massive pulmonary embolism.

The following ***types of surgical intervention are used:***

Ideal thrombectomy (which can be considered a *radical operation* in case of ADVT) in case of early hospitalization (up to 5-7 days) of the patient and with thrombotic process localized in major veins (iliofemoral segment, iliac veins, inferior vena cava). Direct and indirect thrombectomy are available. *Direct (open) thrombectomy* (Figure 89) is performed on an easily accessible segment of the major deep veins: in the region of the common femoral vein (immediately below the inguinal fold in the femoral triangle) by phlebotomy, removal of thrombotic masses with subsequent venoplasty. *Indirect (semi-open) thrombectomy* (Figure 90) is carried out with the help of a *Fogarty catheter* in case of the localization of thrombosis in hard-to-reach segments of the major veins (in iliac veins and the inferior vena cava).

2. *Palliative operations* aimed at preventing pulmonary embolism. This is an *application of the inferior vena cava* - the partial occlusion of the inferior vena cava technique (F. C. Spenser, 1959 and M. Ravitch, 1964). The essence of the operation is that the lumen of the vein is divided into a number of channels with a diameter of a bit more than 3 mm using suturing

devices or individual sutures. The operation on the *endovascular introduction of caval filters* is more effective (Figure 91).

Ligation of major veins is a kind of operative intervention aimed at blocking only the part of the venous channel, from which the pulmonary embolism is threatened (currently practically not used).

In the postoperative period, an early moderately active motion regimen with obligatory use of elastic bandage on the lower extremities, anticoagulant therapy (first with direct, then with indirect anticoagulants), administration of disaggregants and hemocorrectors, symptomatic therapy is used.

Fig. 89. Ideal thromboectomy from the common femoral vein

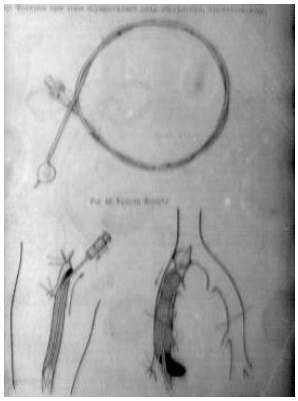

Fig. 90. Indirect thromboectomy from the common femoral and external iliac veins with the Fogarty catheter

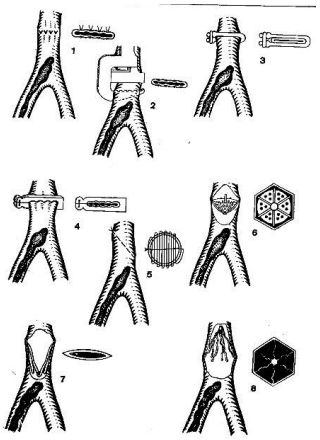

Fig. 91. Methods of partial occlusion of the inferior vena cava (1-5) and various types of caval filters (6-8)

Possible *postoperative complications*:
- bleeding during surgery;
- intraoperative PE;
- postoperative rethrombosis;
- inflammatory wound complications;
- injured nerves and lymph vessels.

The most threatening ***complication of acute deep vein thrombosis is a massive pulmonary artery thromboembolism (PE).*** All kinds of sharp movements and stresses of the patient contribute greately to the detachment and floating embolus, most often - when the patient, after prolonged severe bed rest, rises or sits for the first time (sometimes - during bowel movements). Especially dangerous is the period of incomplete thrombosis of IVC, since the preserved venous blood flow in the thrombotic vein creates favorable conditions for the detachment of embolus from the thrombus.

Thromboembolism of the pulmonary artery

At present, in Europe, the incidence of pulmonary embolism is 11-25%. In particular, in France, every year, up to 100,000 cases of pulmonary embolism are registered, in England and Scotland, 65,000 patients are hospitalized annually for PE, and 60,000 in Italy. According to the largest US clinics, 3% of the USA population is observed for this condition.

According to the Framingham study, pulmonary embolism accounts for 15.6% of the total internal hospital mortality, among them surgical patients account for 18% , and 82% are the patients with therapeutic pathology.

According to numerous pathological anatomy studies, in 50-80% of cases, pulmonary embolism is not diagnosed at all, and in many cases only a provisional diagnosis is made. PE takes one of the leading places in obstetric practice: the mortality from this complication ranges from 1.5 to 2.7% per 10,000 births, and in the structure of maternal mortality it is of 2.8-9.2%.

Having received no timely and adequate treatment, many patients die in the first hours of the onset of the disease.

Thromboembolism of the pulmonary artery *- occlusion of the lumen of the main trunk or of the branches of the pulmonary artery by an embolus that is detached from the thrombus that has been formed in the veins of a major blood circlulation or of the right heart chambers and floated into a small circle of blood circulation. Mechanical blockage of the trunk or branches of the pulmonary artery by embolus is accompanied by generalized spasm of the pulmonary arterioles, which leads to a sharp restriction of blood flow in the lungs.*

The pathophysiological basis of the pulmonary embolism is the obstruction of the pulmonary arteries by the embolus with the subsequent development *of hypoxia and pulmonary arterial hypertension.* As a result, the blood circulation of the lungs is severely disturbed, the right half of the heart is overloaded with the development of *acute or subacute right ventricular failure*, and major neuroreflectory disturbances develop.

The clinical course of the pulmonary embolism, above all, depends on whether the main trunk and main branches, or medium and small branching are occluded, and also on the completeness of occlusion.

According to the clinical course, four forms of PE are distinguished (M.B. Rzaev, 1970):

1) *peracute disease*, sudden death of the patient occurs (within 10 minutes) due to acute asphyxia or cardiac arrest;

2) *acute* - with the sudden appearance of severe retrosternal pain, severe shortness of breath, development of collapse; if no treatment is given, patients die within 24 hours;

3) *subacute* - develops more gradually and clinically manifested in a pulmonary infarction, the main symptom of which is *hemoptysis*;

4) *chronic* - develops gradually and clinically manifested in chronic lung and heart failure.

Depending on the ***volume of injury of the pulmonary vessels,*** PE may be (Saveliev V.S., 1990):

1) *supermassive* - more than 70% of the pulmonary vessels are affected (sudden loss of consciousness, diffuse cyanosis of the upper half of the body, stroke, convulsions, respiratory arrest);

2) *massive* - more than 50% of pulmonary vessels are affected (loss of consciousness, shock, fall of arterial pressure, failure of the right ventricle);

3) *submassive* - from 30 to 50% of pulmonary vessels affected (shortness of breath, normal arterial pressure, right ventricular function disturbed to a lesser degree);

4) *nonmassive* - injury to less than 30% of the vessels of the lungs (shortness of breath, right ventricular function does not suffer).

The clinical picture of the pulmonary embolism depends on the size and location of the embolus. General, cardiovascular and pulmonary symptoms are distinguished.

Common symptoms: severe chest pain, anxiety caused by fear of death, increased body temperature (Michael's syndrome), loss of consciousness, general weakness, decreased diuresis. *Cardiovascular symptoms:* growing tachycardia (Mahler syndrome), arterial hypotension (up to the development of collapse), cyanosis of the face, neck and upper half of the trunk, swelling of the cervical veins, increased central venous pressure (CVP), pain in the heart and enlargement of the liver.

Pulmonary symptoms: shortness of breath, cyanosis, cough, hemoptysis, pleural friction rub. Characteristic ***clinical syndromes are distinguished***.

1. ***Pain in the chest*** at PE is observed in 52-86,9% of patients. The pain is localized behind the sternum, not irradiating, anginous in character, accompanied by fear of death. PE is a *shock* resulted from vascular obstruction, "dam" *syndrome,* due to a disordered hemodynamics in a minor blood circulation as a result of thrombotic formation and reflex spasm of blood vessels. There are ***3 variants of pain syndrome*** in pulmonary embolism: *anginous; pulmonary pleural; mixed*.

2. ***Syndrome of acute respiratory failure***, which is characterized by suddenly occurring respiratory disorders - from a feeling of lack of air to abrupt shortness of breath with the cyanosis, bronchospasm development, sense of fear. The development of shortness of breath is explained by the reflex response of the respiratory center to the onset of pulmonary hypertension and stimulation of pulmonary receptors, as well as an increase in the functional alveolar dead space by reducing lung perfusion.

3. ***Syndrome of acute vascular insufficiency***. It is explained by a pulmonary-depressor reflex - a reflex fall of blood pressure in a major circle of blood circulation in response to a sharp increase in minor. Another factor is a decreased blood supply to the left ventricle and decreased in cardiac output due to significant pulmonary obstruction and mechanical blockage of the blood flow.

4. ***Syndrome of acute heart failure (right and left ventricular).*** One of PE clinical manifestations is an *acute pulmonary heart disease syndrome*. The pathophysiological mechanisms of the development of acute cor pulmonale include the fact that thromboembolic occlusion of the pulmonary artery leads to reflex spasm of arterioles, a sharp increase in pressure in the system of minor blood circulation, resulting in an overloaded right ventricle.

5. ***Syndrome of acute heart rhythm disorders***. Tachycardia in 70-100% of cases, extrasystole, tremor and atrial fibrillation.

6. ***Acute coronary failure syndrome***. ECG in part of patients with pulmonary embolism, in addition to the classic signs of acute cor pulmonale, shows changes in the QT segment and the T wave in the left chest, indicating acute ischemia.

7. ***Cerebral syndrome***. Acute disorder of cerebral circulation, manifested by loss of consciousness, seizures, hemiplegia, involuntary urination and bowel movements.

8. ***Abdominal syndrome***. Symptoms of the false "acute abdomen" are acute pain in the right upper quadrant of the abdomen, belching, vomiting, vague symptoms of irritation of the peritoneum.

None of the listed syndromes is strictly pathognomonic for PE. At the same time, the absence of such symptoms as shortness of breath, tachycardia, chest pain put in doubt the diagnosis of PE. The significance of these symptoms increases significantly in the detection of signs of deep vein thrombosis.

Questioning of the patient reveals the complaints of sudden, often incomprehensible ***shortness of breath*** - the most characteristic symptom of thromboembolism, that is a reflection of acute respiratory failure and is of inspiratory nature, orthopnea is not observed; shortness of breath occurs in varying degrees of severity - from a feeling of lack of air to a very pronounced.

Pain syndrome occurs in several variants. In 42-87% of patients there is a sharp stabbing retrosternal pain. Total duration of the pain syndrome varies from several minutes to several hours. In case of embolism of the main trunk of the pulmonary artery, tearing retrosternal pain often occurs due to the irritation of the receptors of the pulmonary arterial wall. In some cases of massive thromboembolism, acute pain with broad irradiation resembles pain syndrome of dissection of aortic aneurysm. Sometimes the pain may be anginal, that is associated with a sharp decrease of coronary blood flow due to reduced beat and minute volume of the heart.

In case of the development of *massive infarctions of the right lung*, sharp pains in the right hypochondrium can be observed, associated with acute right ventricular failure, acute congestion swelling of the liver, combined with intestinal paresis, symptoms of irritated peritoneum.

In case of *infarction of lung*, acute chest pain is observed, aggravating by breathing and coughing, hemoptysis.

In case of *embolism of small branches of the pulmonary artery*, the pain may be absent or covered by other clinical manifestations, the main manifestation is ***hemoptysis.***

History of the disease: in most cases, the development of pulmonary embolism occurs suddenly against the background of acute thrombosis, often deep veins (diagnosed or undiagnosed); Past history may indicate the undergone acute venous thrombosis of the extremities, the presence of post-thrombotic syndrome, varicose disease of the lower extremities, and possibly a previous pulmonary embolism.

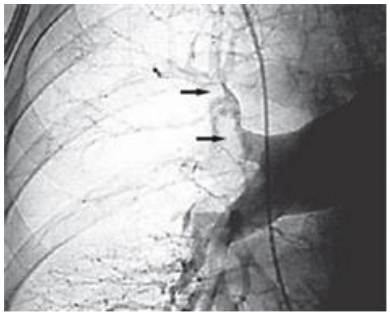

Fig. 92. Pulmonary angiography: emboli in upper lobar branch and in the mouth of the right middle lobar pulmonary artery

Objectively. The general condition of the patient depends on the localization and massiveness of the thromboembolism of the pulmonary artery and may be both of moderate severity and extremely severe, preagonal, and loss of consciousness may occur. The position of the patient is forced - semisitting, facial expression is suffering. There is a pronounced cyanosis of the skin of the face, neck and upper trunk. Tachicardia is increasing in the vast majority of patients with pulmonary embolism, the main cause of which is an acute decompensated pulmonary heart. The pronounced tachypnea, dyspnea are observed. The objective symptoms of acute right ventricular heart failure in PE are swelling of the cervical veins, positive ventricular pulse, and liver enlargement. The overload of the right ventricle can be evidenced by the appearance of epigastric pulsation, increased heart beat, displacement of the right border of the heart, sometimes pulsation in the second intercostal space to the left. Auscultation shows loud second heart sound and its split (gallop rhythm) on the pulmonary artery, where systolic, and sometimes diastolic murmur are heard. At the same time, ***central venous pressure (CVT) is increased significantly.***

It is believed that sudden cyanosis combined with shortness of breath, tachycardia, chest pain is a reliable sign of pulmonary embolism, and the development of cyanosis of the face, neck and upper half of the body indicates a massive pulmonary embolism with a poor prognosis.

Leading syndromes and symptoms *- chest pain, local cyanosis, arterial hypotension, shortness of breath, hemoptysis (signs of acute cardiac and respiratory failure).*

In accordance with the standard schemes, the ***plan for additional laboratory and instrumental examination of a patient with suspected pulmonary embolism*** in a specialized vascular surgical department includes:

1. Clinical tests of blood, urine, blood biochemistry.
2. Coagulogram.
3 Electrocardiogram.
4. Chest X-ray.
5. Echocardiography.
6. Doppler ultrasonography of the major veins of the lower extremities and pulmonary artery.
7. Perfusion lung scan.
8. Pulmonary angiography.

Specific pathological changes.

1. Clinical blood test - increased leukocytosis, ESR.

2. *Coagulogram* – hypercoagulation phenomena.

3. *Angiographic examination – pulmonary angiography* is a gold standard in the diagnosis of pulmonary embolism, which confirms or excludes the diagnosis. The most characteristic angiographic evidence of the pulmonary embolism is a *filling defect* in the lumen of the pulmonary artery or its branch, or the *symptom of "amputational stump"* (vessel contrast break, Figure 92).

4. *Radiographic signs of pulmonary embolism* (Figure 93). The most characteristic symptoms of the acute pulmonary heart are enlargement of the superior vena cava, enlargement of the pulmonary artery cone, flattening of heart. There may be an increase in the right chambers of the heart, protrusion of the pulmonary artery arch, no pulsation, and enlargement of the root of the lung, decreased pulmonary pattern in the branching region of the thrombotic pulmonary

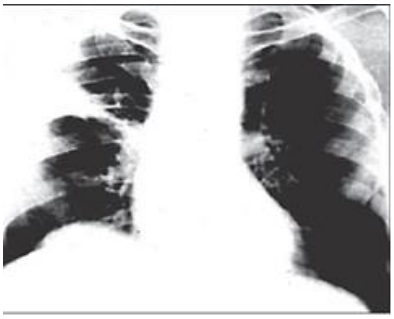

Fig. 93. X-ray signs of PE

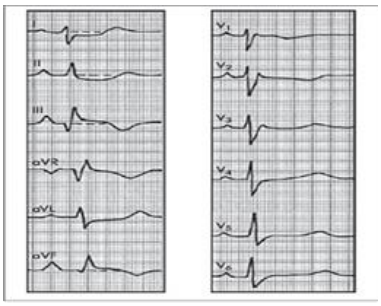

Fig. 94. ECG signs of PE

artery (*Westermark's sign*, 1958), the presence of a wedge-shaped shadow, high standing of the diaphragm dome and its restricted mobility on the side of the disorder (*Zweifel's syndrome*).

5. *Electrocardiographic diagnosis (*Figure 94). Changes are characteristic for *acute right ventricular failure.* There are changes in the QRS complex, characteristic of turning the heart around the longitudinal axis clockwise. In lead I, the wave S appears or intensifies, and the lead III is wave Q (SI, QIII syndrome). In the III lead, too, there is a high wave R. A characteristic shift of the transition zone to the left to V5 or V6, often incomplete or even complete right His bundle branch block. In some cases, a small rise in the segment S-T and inversion of the T wave in the III, AVF, V1-3 leads are seen. The signs of the right atrial overload are characteristic too, as increased amplitude of the wave P in leads II, III, AVF. All of the mentioned ECG changes that occur in the acute pulmonary heart, as a rule, are temporary and disappear after a few days. It should be considered that sometimes (even with a massive pulmonary artery embolism) ECG may not show any specific symptoms.

6. *Echocardiography* allows visualizing blood clots in the right chambers of the heart, evaluate right ventricular hypertrophy, and degree of pulmonary hypertension. Echocardiography is very important for the evaluation of the regression of embolic blockage of the pulmonary blood flow during treatment, as well as for differential diagnosis with the diseases similar to the

clinical manifestations (myocardial infarction, pericardial effusion, cardiac tamponade, aneurysm, aneurysm of the thoracic aorta, thrombosis of the superior vena cava).

7. *Doppler ultrasonography* of the main veins of the lower extremities is carried out to diagnose the source of pulmonary embolism - thrombi of the main venous vascularity of the lower extremities.

8. *Perfusion lung scan* (PLS) is based on the imaging of the peripheral vascularity of the lungs by albumin macro-aggregates labeled with ^{99m}Tc or ^{131}I. At the same time, defects of perfusion corresponding to the area of blood supply to the affected vessel, may be detected. Specificity of PLS increases when comparing results with X-ray findings. No disorders in perfusion of lungs allow, with sufficient certainty, excluding pulmonary embolism.

Differential diagnosis of pulmonary embolism is carried out with myocardial infarction, dissection aneurysm of the thoracic aorta, croupous pneumonia, exudative pericarditis, spontaneous pneumothorax.

Treatment of a patient with pulmonary embolism. Therapeutic measures *are aimed at restoring the permeability of the pulmonary artery - thrombolysis or surgical removal of the thrombus.* However, they require time which may be deficient. Of three died of pulmonary embolism, two patients die within the first 2 hours after embolism.

Therefore, ***in the peracute phase of severe pulmonary embolism, the measures aimed at maintaining vital (vital functions) of the body - hemodynamics and gas exchange*** - are very important.

First medical emergency treatment for PE.

1. *Life support in the first minutes of massive pulmonary embolism - heart massage, artificial lung ventilation,* inhalation of oxygen. Closed-chest cardiac massage not only provides blood circulation in vital organs, but also allows fragmenting clot or "pushing" it along the pulmonary trunk and reducing the degree of obstruction of the pulmonary vessels.

2. *Fighting with fear and pain - neuroleptanalgesia (*eliminates pain, fear, catecholaminaemia, reduces the oxygen demand*), narcotic analgesics (*morphine, omponon, promedol, etc.), strong *non-narcotic analgesics with antispasmodics* (triggan, baralgin), analgin combined with *antihistamines* (dimedrol).

3. *Fighting arterial hypotension - infusion therapy with blood substitutes with hemodynamic effect, cardiotropic therapy, etc.*

4. Removal of vasovasal intrapulmonary reflex - myotropic antispasmodics (euphylline 2.4% 10-20 ml IV, no-spa, papaverine), beta-2 adrenomimetics (alupent, astmopent, salbutamol), anticholinergics (platyphyllin); remedies to reduce the blood flow to the right atrium - nitroglycerin, isoket, 5-nitro.

Therapeutic tactics for pulmonary embolism in a specialized vascular unit. Currently, *the method of choice is medical therapy, which is based on effective* ***thrombolysis****, which restores the permeability of the pulmonary artery and its branches.* If medical thrombolytic therapy is ineffective, surgical treatment is indicated.

The main pathogenetically grounded ***medical therapy is thrombolytic therapy,*** aimed at restoring the permeability of the pulmonary artery, as well as ***anticoagulant therapy***, aimed at creating hypocoagulation and cessation the growth of the obturating thrombus.

Thrombolytic therapy*. Direct and indirect activators of endogenous fibrinolysis are used* for thrombolysis: ***indirect thrombolytics*** - *streptokinase, streptase, kabikinase;* ***direct thrombolytics*** - *urokinase, tissue plasminogen activators - actilyse, metalyse*.

Streptokinase treatment begins with intravenous injection of 250,000 U *("inactivating dose*") of the preparation in 300 ml of a 5% glucose solution for 30 minutes, then the infusion of the treatment dose of 750,000 U (at 100,000 U / hour) lasts for 8 hours. To prevent allergic reactions, 60-90 mg of prednisolone is administered simultaneously with streptokinase. *Urokinase*: during the first 15-30 minutes intravenously administered 4400 U / kg of the patient's weight, then by 4400 U / kg per hour for 12-24 hours.

Since the 90s of the twentieth century, the active ingredient (actilyse - synthetic tissue activator plasminogen) has become the *gold standard for thrombolytic therapy*. The disadvantage is a short elimination half-life (4-5 minutes), which requires constant drip infusion of the drug for 90 minutes. Scheme of actilyse administration: intravenously 10 mg for 2 minutes, the next 60 minutes - 50 mg, then for 2 hours, another 40 mg (100 mg for 3 hours). Possible intravenous administration of 100 mg of the drug for 2 hours. Thrombolytic therapy in patients with severe PE with actilyse has advantages over streptokinase and urokinase as it gives a faster clinical effect.

Metalyse (tenecteplase) is a modern third-generation thrombolytic agent that provides *bolus thrombolysis*. The elimination half-life comparison with alteplasa lasts 20 minutes more. It is administered within 5-10 seconds as a single intravenous bolus. Metalyse is prescribed in individual dosage (depending on the weight of the patient), which makes the drug the safest. It is used at the prehospital stage, that improves the prognosis significantly. It should be considered that in order to prevent rethrombosis against the background of metalyse bolus injection, aspirin, heparin should be administered, as the revascularized artery intima injury remains for a long time.

During thrombolysis, the following ***complications*** may occur:

1) pyrogenic and allergic reactions (urokinase, actilyse and metalyse are virtually free of antigenic properties);

2) hemorrhagic complications in 45-50% of cases;

3) there is a high probability of pulmonary embolism recurrence while the treatment with plasminogen activators, as fragmentation of other venous thrombi in thromboembolic disease, as well.

The thrombolytic drug infusion rate should be such as to maintain thrombin time 2-5 times longer than the control time. The effect of thrombolysis is assessed according to regression of clinical manifestations, electrocardiographic disorders, and control pulmonary angiography. After the thrombolytic therapy is complete, heparin or low molecular weight heparins are prescribed.

Anticoagulant therapy. Currently, the main drug for the treatment of pulmonary embolism is heparin (both unfractionated and low molecular weight). It suppresses the growth of blood clots, promotes their dissolution and prevents rethrombosis. In addition, it has antiserotonin and anti-bradykinin action, and therfore plays an important role in eliminating vascular and bronchodilating effects.

First, 10-20 thousand units of heparin are injected in bolus intravenously, then 5000 U every 4 hours. It is more efficient to use high doses of heparin: intravenously bolus 20,000 U (300-500 U / kg) followed by infusion of 5,000 U / hour. *Control over heparin therapy*: APTT-activated partial thromboplastin time (achievement of blood non-coagulation for more than 100 seconds) 4 times a day. Then a gradual decrease in the dose of 500-1000 U / hour. Daily dose should be 30000-60000 U.

Duration of the course is 5-7 days, because lysis and organization of thrombus occur within these terms. 3 days before heparin is cancelled, *indirect anticoagulants (phenylene, warfarin)* are prescribed, since they initially lower the level of protein C, which can cause thrombosis. The duration of treatment with indirect anticoagulants should be *at least 3 months.*

In recent years, *low molecular weight heparins* (LMWH) have been successfully used - *fraxyparin, clexane, fragmin*. Positive moments in LMWH administration are high bioavailability, rapid absorption in the subcutaneous injection, small number of times of administration - 1-2 times a day, good tolerability, rare development of complications, less thorough laboratory control.

For therapeutic purposes, fraxiparin is given 2 times a day with an interval of 12 hours in the following doses, depending on the weight of the patient's body (Table 28).

Table 28

Body weight, kg	**Amount of fraxyparin per injection**

	(2 injections per day), ml
45	0.4
55	0.5
70	0.6
80	0.7
90	0.9
100	1.0

Treatment continues through the high risk period until the moment of complete restoration of motor activity of the patient, but not less than 10 days.

Contraindications for LMWH administration are: acute bacterial endocarditis, thrombocytopenia in the presence of bleeding or predisposition to it (except the consumption coagulopathy), gastric and duodenal ulceration (in the acute period), cerebrovascular hemorrhages. The drug should be used with caution in pregnant women and patients with renal and hepatic failure.

While the LMWH treatment, drugs containing acetyl-salicylic acid, non-steroidal anti-inflammatory drugs should be prescribed with caution due to the possibility of potentiation of the effect.

Supportive antithrombotic disaggregant therapy: *rheopolyglucin* (400 ml), *trental* (5 mg/kg), *nicotinic acid* (2 mg/kg/day) for 5-7 days. New effective disaggregant is *plavix* (*clopidogrel* - 75 mg per day).

Infusion therapy. The controlled, slow infusion of liquid (*colloidal solutions*) is CVT-monitored. If CVT increases more than 15 cm water column, infusion should be temporarily stopped. Infusion therapy improves hemodynamics and tissue oxygenation.

Inotropic support. *Dobutamine* in a dose of 5-10 mg / kg / min provides adequate preload, increases the cardiac index, eliminates hypotension, reduces the heart rate, reduces the resistance of the pulmonary vessels. Dopamine in a dose of 5-17 mcg / kg / min has similar effects, but its administration may cause increase of pressure in the pulmonary artery, and in some patients tachycardia develops. *Noradrenalin* effectively provides inotropic function of the heart, improves cerebrovascular and coronary perfusion pressure, is well controlled. *Mesaton* is used to restore vascular tone and increase rapidly blood pressure during collapse.

Maintenance of strict bed rest. Providing the correct position in bed - half-sitting.

Surgical treatment. In case of the thromboembolism of the trunk of the pulmonary artery, at ineffective medical thrombolytic therapy, the surgical treatment - ***direct embolectomy under artificial blood circulation*** is indicated.

Currently, an alternative to open surgical intervention is ***a non-invasive operation - indirect thrombectomy from the pulmonary artery via a Fogarty catheter from the peripheral access.*** After pulmonary angiography, establishing the localization and size of embolus under X-ray video monitoring, a probe is introduced and mechanical removal of the thrombus is carried out with the subsequent administration of thrombolytics.

Prevention of the occurrence of pulmonary embolism.

1. *Non-medicated* prophylaxis includes: early activation of patients in the postoperative period, at myocardial infarction, cerebral stroke; bandaging with elastic bandages of the legs and hips; intermittent pneumatic compression with cuffs applied to the legs.

2. *Drug correction of the hemostasis system* is carried out with the help of small doses of heparin, which is administered subcutaneously at 5000 U every 6-8 hours. Treatment begins 2 hours before the operation and continues for 7-10 days after it, then indirect anticoagulants are started up to the discharge of the patient from the hospital, if necessary, continuing their use in outpatient conditions. The use of heparin reduces the risk of non-fatal pulmonary embolism by 40%, fatal by 65%, deep veins thrombosis (DVT) by 30%.

In patients with a high risk of hemorrhagic complications (after operations on the brain and spinal cord), instead of heparin, daily infusions of low molecular weight ***dextran (rheopolyglucin***) are administered intravenously at a rate of infusion of 10 ml / kg during the

first 24 hours, then at 500 ml/day for 2-3 days. Aspirin was revealed to be ineffective in preventing DVT.

In the last decade, for the prophylaxis of post-operative DVT, LMWHs are widely used. Their administration does not require regular laboratory control, and it is less frequently than standard heparin is accompanied by the development of bleeding and thrombocytopenia. In this case, the dose of the drug is determined depending on the risk of developing PE: at high risk, the dose is increased compared with that in case of moderate risk. For prophylactic purposes, LMWH is administered subcutaneously once a day, taking into account the body weight of the patient: *fraxiparin* 0.3-0.6 ml, *Clexane* 0.2-0.4 ml, *fragmin* 2500-5000 IU. Preventive administration of this drug should be at least 10 days (sometimes up to 3 months).

3. *Surgical prophylaxis - implantation of caval filters.* Currently, the most widely used *transcutaneous implantation of caval filters*, the indications are as follows:

- Contraindications to anticoagulant therapy or severe hemorrhagic complications during its use;
- Relapse of PE or proximal proliferation of phlebothrombosis against the background of adequate anticoagulant therapy;
- thrombembolectomy from the pulmonary artery;
- protracted "floating" thrombus in the iliocaval venous segment;
- DVT or PE in patients with low cardiopulmonary reserve and severe pulmonary hypertension;
- high risk of developing deep vein thrombosis (DVT) or pulmonary embolism (major surgery, fractures of the bones and the spine) in patients who had previously had pulmonary embolism;
- PE in pregnant women as an addition to heparin therapy, or in case of contraindications for the use of anticoagulants;
- failure of previously used methods of treating DVT or PE;
- thrombendarterectomy in patients with post-embolic pulmonary hypertension;
- DVT or PE in patients undergone kidney or heart transplantation.

At PE of degrees I and II of severity and adequate treatment, the prognosis is favorable, at degree III and especially IV mortality is high, as adequate help is usually delayed. In case of recurrent chronic pulmonary artery embolism, treatment with indirect anticoagulants is indicated, in case of indications - introducing a caval filter. The patients should be followed up.

IV.4. INJURIES OF THE ARTERIAL AND VENOUS MAJOR VESSELS OF THE EXTREMITIES

The history of surgery of the injured vessels has a thousand years of experience. Methods of hemostasis were improving over time from cauterization of vessels and tamponade of wound to methods that restore blood flow. Due to numerous wars that humanity led the methods of hemostasis improved.

So, M.I. Pirogov after the Crimean company released the fundamental work for that period "Wounds of blood vessels and traumatic bleeding", which described the clinical manifestations of various types of vascular injuries and methods of combating bleeding. Improving the destruction weapons required improvements in the surgery of injuries.

The experience of fighting with vascular trauma in the Great Patriotic War is reflected in the monograph "Surgical treatment of vascular injuries" written by B.V. Petrovskiy (1949). The development of industry and the growing various types of transport leads to an increase in industrial and traffic injuries, which, in turn, makes it necessary to improve the methods of angiosurgical assistance in the trauma of vessels of different localization.

In peacetime mortality from various types of injuries ranks third after cardiovascular and oncological diseases in economically developed countries. Share of injuries of trunk vessels in peacetime is 2% in the overall structure of injuries.

Injury of the major arteries is one of the most dramatic parts of the polytrauma, as patients with major vessel injuries die, most often, in the immediate hours from the moment of injury. The results of treatment for this category of patients depend not only on the area of injury, but also on how quickly and adequately the aid is provided at the prehospital stage and stages of non-specialized and specialized care. The majority of patients with polytrauma combined with injured major vessels die before the stage of specialized angiosurgical care. It is caused by the massive blood loss, which leads to hemorrhagic shock.

Most often, injuries to the vessels of the limbs combine with the injury of the tubular bones. According to our data, it happened in 23.4% of cases of all injuries to the arteries of the limbs. In cases of the bone trauma, if there is no active bleeding, in the first post-traumatic hours, disordered circulation of the limb remains often undiagnosed. Application of a plaster bandage, concomitant injury to the nerves and administered analgesics worsen the monitoring of the viability of the injured limb and contributes to the progression of acute ischemia, if there is an injury to the arteries.

In connection with the above mentioned, all patients with bone trauma need their pulse in the distal limb parts to be controlled and, at the slightest doubt, it is necessary to carry out Doppler examination or consult the vascular surgeon. In case if deteriorated vessel integrity and acute limb ischemia are revealed, the patient should be immediately operated. If simultaneous intervention is planned for bone injury, then first it is necessary to provide reposition and immobilize the bone fragments, and then to restore the blood flow. If this is done in reverse order, manipulation on the bones can lead to a disorder of the restored blood circulation.

To achieve the best results in treating patients, it is essential to acquire the methods of hemostasis either by medical workers or among the general population. First of all, among the soldiers, police officers, fire brigade, Ministry of Emergency response, drivers and workers in other areas where there is an increased risk of traumatism, as well as among those engaged in extreme sports.

Injuries of the major arteries of extremities

Vascular injuries are registered most often in males of young and middle age. On the one hand, it facilitates the work of the vascular surgeon, since the case is unaffected with arteriosclerosis, but on the other hand, such trauma causes breaking the blood flow in tissues and organs that are not prone to chronic ischemia and have no collateral circulation developed.

Therefore, irreversible changes in organs and tissues occur quickly and irreversibly. Invalidation among these patients is associated with both the loss of various segments of the limbs, and partial or complete loss of the function of the limbs.

Impaired function of the limbs is caused by the accompanying mechanical injury of the tendons, nerves and muscles, and subsequently the ischemic disorders add to them. Nerve tissue is more likely to suffer from ischemia more often than other tissues. Usually, no restoration of the impaired limb function caused by acute ischemia occurs. Life-hazardous struggle to save the limbs of the patient may be ineffective and result in loss of physiological functions of the limb, therefore, such risk must be justified.

Efforts of medical workers should be oriented, first of all, to the salvation of the patient's life and compensation of the vital functions of the body, and then to fight for the vitality and functional activity of the limb.

Which is better: to bring the patient to a specialized center or to carry an angiosurgeon to the place of hospitalization of the injured people? It depends on the type of injuries (polytrauma or isolated vessel injury), the state of the patient, the time elapsed from the moment of injuring to the moment of the expected arrival of the angiosurgeon, from the distance of location of the injured person to the nearest center for the polytrauma treatment or vascular department, as well as from the level of the regional development of air medical service or similar structures, staffed by the narrow specialists who are able to provide resuscitation and various types of surgical care in remote areas.

The types of transportation of the injured patients are also of importance. Most experts support the idea of approaching of specialized care to the patient if he is in shock. In case of stable hemodynamics and no contraindications for transportation of the patient, then it is reasonable to deliver him to a specialized center.

The current tendency to increase the number of catastrophes makes to think also about improving the skills of general surgeons, mastering methods of control of bleeding from the major vessels and temporary prosthetics of vessels, and the skills of vascular suturing, as well.

For more than 40 years, the emergency angiosurgical care, including aid for vascular trauma, is provided at the "Institute for General and Emergency Surgery". According to our data, the injury of the vessels of the lower extremities was diagnosed in 54.6% of the injured individuals, and the vessels of the upper limbs - in 31.1% of the total number of hospitalized for injuries of the major vessels.

According to most authors, the superficial femoral artery is often affected - 30-35% of cases, then - the shoulder - 15-20% and popliteal artery - 12-15%. Wounds of the vessels of shin occur more often than the vessels of the forearm. The share of carotid, subclavian and iliac vessels accounts for no more than 10-15% of the total amount of injuries.

The outcome of the treatment depends on the size of the vascular wound, the type of vessel (arterial or venous), the type of vascular injury (closed or open), its diameter, full intersection or partial, isolated injury or combined, the degree of blood loss, the shock stage, the distance of the accident from the medical centers, time passed from the moment of injuring and the adequacy of the provided medical care at all stages.

Polymorphism of traumatic vascular injuries makes it possible to differentiate them according to various features and thus results in a variety of classifications. Now in the clinical treatment the ***classification proposed by M.A. Shor in 2007*** (Table 29) is used.

Questioning the patient shows the most characteristic *complaint* that indicates arterial injury: it is a particularly strong, "fountain", pulsating bleeding from the wound at admission or in the close history. If interstitial hematomas form, the patients complain of the present in the injured vascular area a tense, sometimes pulsating swelling of considerable size. Hematoma often compresses the vascular-nerve bundle, which leads to the appearance of "shooting" pains and edema of the limb. The latter is sometimes so pronounced that the clinical course of such injury resembles a *crush syndrome*.

Objectively. As a rule, injury of the large arterial vessels is accompanied by a *hemorrhagic shock* clinical picture. Patients are in a state of excitement or, conversely, may be inhibited in case of massive blood loss. The pallor of the skin is seen, it is cold, with sticky sweat. Low level of blood pressure, tachycardia is registered.

Local status. Local signs of arterial injury include: presence of a wound with an external pulsating bleeding; presence of coagulated or pulsating hematoma (in case of closed arterial injury) in the affected area over which systolic-diastolic murmur, which coincides with the heart rhythm, can be heard; signs of tissue ischemia: pallor or cyanotic skin coloration, decreased skin temperature, delayed filling of capillaries after compression, decreased pain sensitivity, impaired muscular function as an ischemic contracture; absent or weakened pulse is more distal than the site of injury. The intensity of peripheral pulsations and the degree of ischemia depend on the nature of the arterial impairment (lateral defect, complete intersection) and its localization. Neurological symptoms indicate mechanical injury to the nerve or ischemic neuropathy, since nerve fibers are the first to respond to hypoxia.

*The hemorrhagic syndrome is **the leading syndrome** at arterial bleeding in the limb, the **main clinical symptom** is the presence of external pulsating bleeding or growing hematoma of soft tissues.*

In accordance with the standard schemes, the ***plan for additional laboratory and instrumental examination of a patient with injured major arteries of the limbs*** includes ***and reveals characteristic pathological changes.***

1. *Clinical blood test* - reduced red blood cell count, hemoglobin, hematocrit in the peripheral blood.

2. *Clinical urinanalysis* - unchanged.

3. *Coagulogram*: the most common is a disorder of blood clotting towards hypercoagulation.

4. *Doppler* - method which allows to reveal the presence or absence of blood flow in different segments of the major vessels. This method is easy to use and it can be provided in medical centers of general profile to determine the rationale of hospitalization of a patient in a specialized vascular department.

Table 29

Classification of injuries of the major vessels of the extremities

Time passed after injuring	**The cause of the injury**	**Character of the injury and its localization**	**Character of injury of the surrounding tissues**	**Clinical manifestations**
Within 3 days (recent injury). From 3 to 30 days (complicated injury).	1) Mechanical effect: - cut/stab wounds - gunshot wounds; 2) blunt trauma effect; 3) iatrogenic injuries; 4) electric current or radiation injuries.	1) Open (vascular injury): - tangential, non-penetrating in the lumen wound; - lateral wound; - perforating wound; - complete break of the vessel. 2) Closed: - concussion of vessel with intramembraneous hematoma; - rupture of some layers; - rupture or splintering of all layers of vessel; - compression of the vessel by the bone fragment or dislocated segment of the limb; - perforation of the vascular wall with an osseous fragment. The name of the injured vessel should be given.	1) No such injuries; 2) considerable destruction of the soft tissues 3) injuries of nerves; 4) injuries of bones; 5) injuries of soft tissues, bones and nerves (partial or complete amputation of the limb).	1) Recent injuries: - external or internal bleeding; - hematoma; - shock; - anemia; - ischemia of a limb. 2) Complicated injury: - hematoma of soft tissues, including pulsating hematoma; - suppuration of soft tissues and hematoma; - secondary (arrosive) hemorrhage; - contraction in joints of the injured limb; - arterial or venous failure of a limb.

More than 30 days (long-term results of the vascular injury).		Open or closed injury of a vessel with mentioning of its localization.	Isolated or combined injury of artery or vessel.	- Traumatic aneurysm (arterial or arteriovenous); - disease of the injured (ligated) vessel; - Volkmann's contracture; - post-thrombotic syndrome.

5. *Color duplex scanning* is a method that allows to visualize both the walls of vessels and surrounding tissues. This method can determine the location of vascular injury; assess the condition of the upper and lower segments; the presence, volume and distribution of hematoma. With duplex scan, muscular ruptures can be detected. As a rule, this method of research is enough to diagnose the injury of the major vessels of the extremities and to decide on further tactics and the volume of surgical treatment.

6. *Angiography* is used to diagnose arterial lesions less often than the ultrasound techniques are used. Angiography demonstrates a sudden breakdown of the contrast of the major artery with possible paravasal contamination of the contrast agent. If a patient is under the state of the shock, this method is unlikely possible, since it is invasive. The diagnostic procedure can transform into a non-invasive intervention for a temporary or permanent hemostasis in case of continued bleeding or at unstable hemostasis in one of the major vessels. It should be considered that in case of tangential wound of vessel and controlled bleeding, the angiograms may not help to identify the signs of injury. Therefore, the most important diagnostic measure for the detection of major vessel injuries is the wound revision during ISD (initial surgical debridement).

Differential diagnosis of arterial bleeding is firstly performed with venous bleeding.

Clinical manifestations of external arterial and venous bleeding from the vessels of the limbs are fairly easily to differentiate. But it is not so easy in the closed vascular injuries, which have polymorphic manifestations and are often undiagnosed immediately within hours from the moment of injuring, especially in case of polytrauma.

It should be considered that no obvious signs of bleeding may not exclude the trauma of the vessel, that sometimes calms the medical staff who provide the first medical care, and thus leads to untimely diagnosed limb ischemia, and in the long-term period - to the pseudoaneurysm.

In case of arterial bleeding through the injured skin, the blood pours out with a pulsating stream of red color. In case of venous bleeding, a stream of blood is not pulsating and the color of blood is dark cherry. It is not always possible to differentiate arterial bleeding from venous immediately. At prolonged arterial bleeding and low blood pressure, a pulsating blood stream may not be as pronounced as at the beginning of bleeding. Also, under conditions accompanied by poor oxygenation of arterial blood, its color may be dark.

Fig. 95. Angiogram: extravasate in case of injured arteries of the right shin

In disruption of the continuity of the artery with no injuring the skin, a bruise, or swelling appear at the site of injury, which is rapidly growing in size, usually eccentrically located relatively to the surface of the limbs and course of blood vessel. Palpation of the formation will determine pulsation, and it systolic-diastolic murmur, which coincides with the heart rhythm, will be heard. If the major veins are broken due to trauma with no damage to the skin, a large hematoma is formed at the site of injury. In case of contusion of major veins, thrombosis of the injured vein segment is most often accompanied by a clinical picture characteristic for venous thrombosis.

In case of open lateral injury of the artery of the limb, no characteristic external pulsating bleeding may be observed. The pulse on the distal artery of the limb in

such situations is sometimes preserved, that makes the surgeons to put off their guard. The same is possible with small size pointed or bladed wounds or barbed-cutting injuries that are compressed by surrounding tissues. Later in a few weeks, a false aneurysm is formed that requires surgical treatment.

When a limb artery is injured, a ***clinical picture of acute ischemia*** develops. Below the site of injury there is no pulsation of the major arteries, the distal limbs becomes pale and cold to the touch, all types of sensitivity are impaired and the active movements in the fingers first disappear, then active movements in the distal joints of the injured limb become limited. The muscles of shin and forearm become dense, sharply painful when palpated.

In the far-advanced stages of acute ischemia, ischemic contracture of the peripheral joints is formed, when even passive movements in the joints are not possible. In this case, it should be considered that during polytrauma pulsation at the peripheral arteries may be absent due to shock and centralization of blood circulation. For the same reasons, and in connection with massive blood loss, the peripheral parts of the limbs will be cold and pale. If the patient is unconscious, it is impossible to identify tenderness in the peripheral muscles and to check the presence of active movements in the distal parts. Pain in the muscles may be due to the injury of the most soft tissues and hematoma. Both in cases of injured peripheral nerves, or in acute limb ischemia caused by trauma of an artery, all types of sensitivity will be impaired, and active movements in the distal joints of the limbs will be impossible.

An acute onset of ischemic contracture, which appears a few hours after the moment of injuring and as a rule, shows irreversible changes in the muscles of limbs is the only reliable sign of arterial injury. Arterial injury diagnosed in the final stage of acute ischemia causes despair in angiosurgeons and, as a rule, liberates them from any further attempts to restore blood flow, especially if it is known that ischemic contracture occurred more than a day ago.

Treatment of a patient with arterial bleeding of the limb. *Choice of medical tactics.* The tactics of therapeutic measures should be based on the extent of injury received, the general condition of the patient, and threaten of vital functional decompensation due to the injured organs.

First medical care at the prehospital stage. If the open vascular injury is present, it is necessary to make every effort for ***temporary control of the bleeding***, that may be achieved by the following methods: application of the hemostatic tourniquet proximal to the place of injury in case of arterial bleeding and distal to the site of injury in venous bleeding; or by more reliable methods of hemostasis, such as clipping the wound or ligation of the injured vessels (if possible); tight compressing dressing packed tightly the wound with possibly sterile drapes (mainly in venous bleeding).

Fig. 96. Application of a thigh tourniquet

Application of a hemostatic tourniquet (Figure 96) or similar aids should be aimed at positioning it as close as possible to the wound. Especially it is obligatory to register the time of applying the tourniquet, and in winter time every hour, and in summer - every two hours, to ease it up for a few minutes before the onset of severe bleeding, and tight it again. If the easing of the tourniquet provoke no bleeding, it should be left on the limb as a so-called provisional tourniquet. At the slightest signs of resuming bleeding, it should be tightened again.

Occasionally, an incorrectly placed tourniquet brings more harm than injury itself. This is due, first of all, to the injuring by the tourniquet of nerves and soft tissues. It is necessary to put a bandage or a cloth under the tourniquet on a skin for less traumatism.

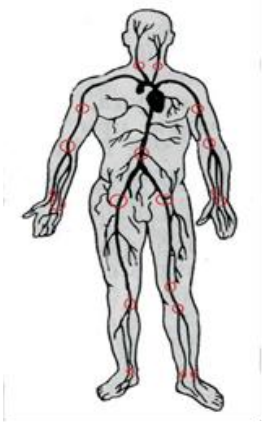

Fig. 97. Places of digital occlusion of arteries

Fig. 98. Pressing the abdominal aorta

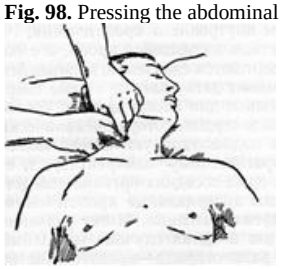

Fig. 99. Digital pressure of a vessel in case of bleeding from carotid artery

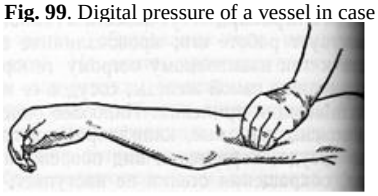

Fig. 100. Digital pressure of a vessel at bleeding from the brachial artery

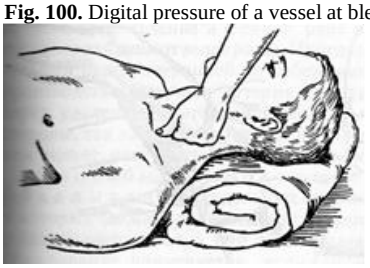

Fig. 101. Digital pressure of a vessel at bleeding from subclavicular artery

Fig. 102. Digital pressure of a vessel at bleeding from femoral artery

If it is possible to stop the bleeding with a tight compressing bandage packing the wound with sterile drapes, then this method should be preferred to the applying the tourniquet.

If the care is provided in a medical facility, the optimal type of temporary control of the bleeding is clipping the injured vessel. Applying a hemostatic clamp, it is necessary to try to apply it not on soft tissues, but directly on the wall of the vessel, as close as possible to its injured ends. Otherwise, restoring the integrity of the injured vessel by an end-to-end anastomosis will be unrealistic. The line of depression from a ligature or clamp applied can require the following artery bypass grafting. It is more traumatic for the patient, extends the time of surgical intervention and sometimes leads to worse long-term results than an end-to-end anastomosis.

Usually, the above-mentioned methods of temporary hemostasis are preceded by a digital occlusion of the upper vessels, which must be done in places most suitable for pressing, i.e. where the vascular-nerve bundle is located most superficially and can be pressed to deeper bone structures (Figure 97-102).

The wounds of the neck and its organs are difficult for diagnosis and taking tactical decisions. According to our data, cervical vessels are wounded in 9.7% of all peacetime injuries of blood vessels.

Pressure bandage, wound packing or application of hemostatic clamps are required to stop bleeding from the wound of the neck.

Such injuries can be combined with wounds of the esophagus, trachea, pharynx and larynx, as well as the pleural cavity and thoracic lymphatic duct. Therefore, if the patient's condition allows, chest X-ray with water-soluble contrasted esophagus is recommended before the examination of the neck wound.

Cerebral ischemia combined with injury of carotid arteries is one of the peculiarities of the injury of cervical arteries. As a rule, the probability of this complication is rather small, since injuries mostly occur in young people with no atherosclerotic comorbidities affecting carotid arteries.

In cases of congenital anomalies of the circle of Willis, as well as at prolonged hypotension against the background of the cessation of blood flow in the internal carotid arteries, cerebral circulation disorder may be observed. Taking it into account, in such cases of stopped blood flow in the internal carotid artery, during the operation, an attempt should be made to preserve the blood supply to the brain using an internal shunt.

If measuring of the invasive arterial pressure in the internal carotid artery is available during the operation, and it reveals above 40 mm Hg, there is no need in the internal shunt. In such a situation, surgery is best performed under controlled hypertension using thiopental anesthesia or gas anesthesia.

If the injury of the carotid artery results in clinical signs of ischemic stroke with marked neurological deficiency in its blood supply (extremely complicated diagnosis of this condition during shock and polytrauma), the need to restore blood flow in this region is doubtful, since cerebral revascularization can lead to fatal bleeding in the ischemic area.

Injuries of external carotid arteries and external jugular veins require no restoration of blood flow. In such situations it is enough to stop the bleeding by dressing or stitching.

Surgical treatment of arterial bleeding of extremities. *The permanent hemostasis.*

While rendering aid with anesthesia in an operating theater or dressing room, aimed at revealing an injured major vessel, and for the convenience of further surgical steps on the vascular nerve bundle, any transverse wound of the skin should be transformed into the wound which is projected to coincide with the course of blood vessels. If immediate hemostasis with hemostatic clipping fails, it is necessary first to perform a digital occlusion of the upper segment of the vessel or apply a hemostatic tourniquet.

If no restoration of the injured vessel is suggested in the close future, it is advisable to ligate it as close as possible to the place of injury, or to apply a tourniquet. It is considered better to apply tourniquet with rubber strips instead of traditional ligatures, to result in less trauma of the arterial wall.

According to V.V. Kovanov (1957), the ligation of the common femoral artery in 81%, and of the popliteal artery in 72.5% of cases leads to the gangrene of the limb. Loss of the upper limb occurs at 56% after the ligating of the brachial artery above branching of its deep branch and in 43% after the ligation of inguinal artery. If one of the arteries of the shin or arteries of the forearm remains ligated, this will not lead to loss of limb or its function, provided with the permeability in left arteries of the forearm or leg. Therefore, it is necessary to make sure that they are pulsating. If there is no pulsation, or if it is doubtful, it is necessary to examine the arteries in the distal parts of the limbs.

In spite of the threat of the loss of limb, it is necessary to repair the injured arteries when the patient is out of shock: stabilized all vital functions, restored CBV (circulating blood volume), stabilized blood pressure, normalized respiration, stable diuresis, and only then make attempts to restore the integrity of the vessel. Otherwise, any, even correctly performed, reconstruction is threatened with thrombosis, and the struggle for the salvation of the limb can lead to the death of the patient.

In such cases, *the use of temporary shunting of injured arteries* may be a compromise (Fig. 103). It gives an opportunity to gain time to stabilize the vital functions, deliver the patient to a specialized in-patient department and prevent acute ischemia in the traumatized artery system.

Fig. 103. Method of fixation of a temporary shunt by rubber ligatures

Temporary shunts are usually made of available drainage tubes, infusion systems and even urinary catheters. It is better to use for temporary shunting sterile silicone tubes of the appropriate diameter. All measures to stop the bleeding and establish a temporary shunt should be carried out without additional traumatism of the upper and lower segments of the arteries, since sometimes inexpert application of a temporary shunt leads to injuring of the artery at a length that requires a large volume of restorative surgery. In the clinic, a temporary shunt of our design is proposed and applied (Figure 104), in which two silicone tubes are interconnected through the T-joint, and, preferably, if the tee has a three-way valve. In the absence of a vent, a tube should be put on its free end, and instead of a grip, a surgical clamp is used.

First, one of the ends of the temporary shunt is introduced into the proximal end of the injured artery and the entire shunt is filled with blood, forcing the air out from the tubes. After that, the second end of the shunt is inserted into the distal portion of the injured vessel on the blood stream. The ends of the temporary shunt are introduced into the lumen of the artery by 3-5 cm. The temporal shunt is fixed in the lumen of the artery using tourniquets.

The remaining third free end of the shunt (or three-way valve) can be used to control the function of the shunt, to introduce drugs directly into the artery, and to take blood for analysis.

When the patient is out of shock, all efforts should be directed to the revascularization of the injured organ. According to A.V. Pokrovskiy et al. (1997), the results of restorative operations are worse if more than 6 hours have passed after injury. At the stage of specialized angiosurgical care, when the volume of the injured vasculature has been restored and primary surgical debridement of the wound has been performed, it is necessary to determine the type of surgical intervention that restores blood circulation.

In unstable hemodynamics after reaching hemostasis, all measures are required to withdraw the patient out of shock - to stabilize all vital functions. First of all, it is necessary to fill the CBV (circulating blood volume), raise the blood pressure, normalize the respiratory function, achieve stable diuresis, and only then try to restore the integrity of the vessel, if necessary.

Fig. 104. Provisional stunt designed in clinic

Ligation of the common femoral artery in 81%, and popliteal artery in 72.5% of cases leads to gangrene of the limb. Loss of the upper limb occurs at 56% after ligation of the brachial artery above its deep branch and in 43% after the ligation of inguinal artery.

If one of the tibial arteries or arteries of the forearm remains ligated, it will not lead to loss of limb or its function, provided by the permeability in left arteries of the forearm or the legs. Therefore, it is necessary to make sure that they are pulsating. If there is no pulsation, or if it is doubtful, it is necessary to examine the arteries in the distal parts of the limbs.

In case of contaminated or infected wounds, it is necessary to give preference to shunting operations. At the same time, as a shunt, it is necessary to apply autovein, since synthetic grafts in the purulent wound are contraindicated because of the threat of the development of arrosive bleeding from anastomosis.

In case of full intersection of the vessel, restoration of its integrity must be performed by applying end-to-end anastomosis. This type of connection is physiological and hemodynamically optimal. Before applying anastomosis, it is necessary to cut economically with scissors the torn edges of the injured vessel at an angle parallel to each other (Figure 105). This technique prevents narrowing of anastomosis.

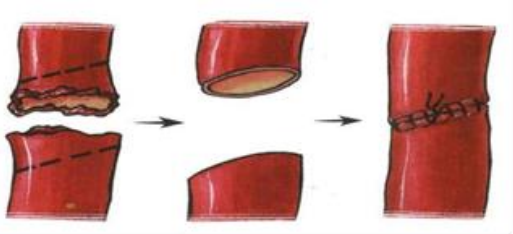

Fig. 105. Edging of the injured vessel

In case of lateral injury of artery and even edges of an arteriotomic wound, it is necessary to apply a continuous or nodular suture with an atraumatic monofilament unsoluble thread. When crossing the vessel more than ½ of diameter, or in case of the torn wounds of the vascular wall, it is desirable to cross the plane of the vessel and, after the treatment of its edges, to apply end-to-end anastomosis. When wounds are located along the vessel, or in case of long-term injuries that go obliquely, it is worth to stitch-in an autovenous patch. If contusion of a vessel exceeds 3-5 cm, an end-to-end anastomosis, as a rule, fails even after mobilizing its ends from soft tissues and ligating the branches from the vessel. Such situations require replacement

of the vessel segment with the autovein. Most often, a reversed segment of major subcutaneous vein is used as a transplant. In case of injured deep veins, the autovein segment should be taken from the intact limb. Major subcutaneous vein on the side of injured deep veins can participate in collateral circulation, so it is better to save it.

The peculiarity of the restoration of blood flow *in children* is that applied circular suture may form a stenosis in the site of anastomosis as a result of the child's subsequent growth. To prevent this complication, it is recommended to repair vessel integrity using instead of a circular continuous suture, a nodal suture at least at one of the semicircles of the anastomosis. In case of lateral trauma, a continuous suture is allowed to be applied.

The further increase in the amount of the affected patients with vascular lesions of different localization requires from the medical staff who are involved in treatment of this pathology, to increase constantly the level of their knowledge and skills both in diagnosis and tactics at all stages of rendering medical aid in vascular injuries.

Injuries of the major veins of the extremities

Diagnosis and treatment of venous bleeding is a topical and complicated issue of vascular surgery. The main causes of venous bleeding are the following: cut/stab and gunshot wounds, venous injuries due to bone fractures, trauma of superficial varicose veins of the lower extremities.

Diagnosis and surgical treatment of *injuries of major veins*, especially large (vena cava superior and inferior), and their consequences are rather complex. The success of treatment depends on the timely qualified specialized surgical aid given to the patient. According to statistics, the isolated injury of veins caused fatal outcome of about 40% of patients with injured blood vessels in emergency situations.

The frequency of revealed isolated injury of veins in peacetime is from 12 to 30% of all wounded vessels. However, combined injuries of major veins with homogeneous arteries, nerves and bones are observed in peacetime more often.

The incidence of injuries of major veins of upper and lower limbs is approximately the same and makes up about 50% of the total population. Injury of vena cava inferior occurs in 2-3% of the affected patients, and of the vena cava superior in 1.5%. Injuries of the major veins of neck (jugular veins) is registered in 12-15% of patients with vascular injuries.

Traumas of *varicose veins of the lower limbs* are rather common (especially in the rural area), however, due to a rapid hemostasis at prehospital stage are rarely recorded by specialized medical centers.

Clinical picture. The injury of veins is manifested with the main symptom - *bleeding*. General and local clinical signs depend on its intensity.

Injury of major veins in about a half of cases is accompanied by phenomena of hemorrhagic shock. Injuries of vena cava inferior and vena cava superior in 50% of cases lead to a fatal outcome.

Injured protuberant varicosities of the lower extremities is manifested by bleeding of medium intensity from the injured varicose vein.

It should be noted that for other equal conditions, bleeding occurs in most cases and blood loss is more massive in cases of incomplete rupture and lateral injury of veins.

Formation of *hematoma* is another important clinical sign. Its dimensions depend on the diameter of the vessel, the nature of injury (complete transverse rupture, lateral or penetrating wound), anatomic topographical characteristics. So, especially massive hematomas can be observed on the lower limbs (thigh). Venous hematomas usually have no clear borders, the pulsation over them is not determined, auscultation murmurs are not audible.

The complications of injury of the major veins, which are to be mentioned, are the following: air embolism and blue phlegmasia.

Air (gas) embolism is an extremely important, as a rule, a fatal complication, which develops in case when the major veins are wounded: subclavicular, innominate vein, internal

jugular, inguinal. In this case, the air enters the right half of the heart, the pulmonary artery. The cases of its penetration into a large circle of blood are described. Clinical picture is characterized by the presence of whistling sound when passing through the wall of the injured vein. At the same time, the patient is pale, breathing becomes superficial, irregular, blood pressure reduces, the pupils dilate, convulsions, cardiac and respiratory arrest appear. Most often, air embolism in peacetime is observed in complicated fractures of large tubular bones.

Blue phlegmasia (venous gangrene) is observed mainly in injured deep major veins of limbs.

Plan of additional examination (laboratory and instrumental) of the patient with injury of the major veins and bleeding from them includes:

1. Clinical blood test.
2. Clinical urinalysis.
3. Coagulogram.
4. Urgent Doppler ultrasonography of veins.
5. Phlebography.

1. *Clinical blood test*: reduced hemoglobin, red blood cell count, hematocrit.
2. *Clinical urinalysis*: no characteristic features.
3. *Coagulogram*: disturbance of blood coagulation towards hypercoagulation.
4. *Urgent ultrasonography*: especially informative in the presence of hematoma.
5. *Phlebography*: there is a sudden breakdown of the contrast of the major vein with possible paravasal entering of the contrast medium.

4. *Provision of a rationale and formulation of the clinical diagnosis* (taking into account the classifications of the disease, the presence of complications and comorbidity) - one of the options:

1) main - open fracture of the right femur with injured superficial femoral vein (or: varicose disease of the right lower limb, stage of decompensation with injured varicose node);

2) complications - external first degree venous bleeding.

3) Comorbidity (if there is any).

5. *Treatment* of patients with injured major veins and bleeding from them.

Methods of hemostasis

In clinical practice, it is common to distinguish between *spontaneous* hemostasis, when the bleeding stops due to the involved biological hemostatic factors of the body itself, and *artificial*, in which the bleeding stops as a result of external intervention. The latter, depending on the time of termination of the bleeding under the influence of an external factor, is classified into *temporary and final*.

Spontaneous hemostasis of venous bleeding is possible in case of deteriorated integrity of small caliber venous vessels, or at extravasation of blood in the intermuscular spaces which results in hematoma formation, when the blood enters the surrounding tissues before the equilibrium between tissue and venous pressure is established, after achieving such a balance, bleeding from the vessel stops and the thrombotic process takes place.

In clinical practice, bleeding is controlled *artificially*, that is carried out using mechanical, physical, chemical or biological effects both on the vascular wall and on the blood. In this case, bleeding may be controlled by stopping blood flow to the site of injured vascular wall and / or closing the defect in the vascular wall.

Temporary control of a bleeding is connected with the provision of urgent first medical aid and can be performed by: application of a pressing tourniquet more distal than the site of bleeding (standard Esmarch's tourniquet, or any other), maximum bending of the limb in the joint, packing the wound (if possible – with sterile gauze swabs), compression of the vessel along the length, pressing the vessel in the wound with fingers, clipping the vessel. It is performed at the prehospital stage or in non-specialized therapeutic or surgical hospitals.

The final hemostasis can be performed by: ligation of the vessel by ligature, vascular suture or vascular prosthetics, tight packing of wound, twisting of the vessel, using high or low

temperature, using ultra-high frequency current (electrocoagulation), as well as using chemical and biological agents (hemostatic sponge, etc.). It is performed in specialized vascular units.

At the prehospital stage, the patients with injuries of varicose veins of the lower extremities are provided with pressing bandage. Quite often the final control of the bleeding is achieved by this manipulation. However, considering the risk of recurrent bleeding, these patients should be hospitalized and undergo surgical treatment (venectomy of the injured veins).

At the prehospital stage, the patients with cut/stab and gunshot wounds of the veins are provided with one of the techniques for temporary control of bleeding, followed by emergency hospitalization in a specialized vascular unit for surgical treatment. At the stage of qualified and specialized medical care, the main tasks are the final hemostasis and prevention of complications.

Both ends ligation of the injured vein is actively used in cases when the superficial veins are injured and is considered as an ultimate measure for injured deep veins, and in case of portal vein injured, it is extremely dangerous and leads to a possible fatal outcome. Such hard-to-reach deep veins, as a gluteal and and internal iliac veins can be ligated.

The technique of temporary intravascular prosthesis is quite widely used, when proximal and distal ends of the injured vessel (veins) are separated, provisional ligatures are made, and then a synthetic prosthesis is inserted into the lumen of the remained vessel and is fixed with ligatures.

At the stage of specialized surgical care, the vascular suture on the injured areas of vessels, the end-to-end anastomosis (in case of complete injury of the vessel), and either synthetic prosthesis or autovein prosthesis at the injured area are the methods of choice.

Control of bleeding from the major veins in cases bone fracture should be performed under conditions of a specialized traumatologic department by the called vascular surgeon (if necessary using the air medical service).

In order to avoid undiagnosed vascular injuries, careful examination of the wound canal is necessary, especially if it passes through the projection of the vessel. If a vellel is found passing the wound, it must be separated through the whole wound and from all sides. Otherwise, in case of unstable hemodynamics it is impossible to find bleeding, especially if it is localized on the lateral or posterior wall. In addition, it is necessary to remove hematoma and clots of blood. They can cover injured vessel wall, masking its wounds. This technique will also help to avoid wound infectious complications.

The major veins of the limbs under a stable patient's condition, especially in an isolated injury, should be repaired, provided that the patient is located in a specialized medical center.

Otherwise, the patient is at risk of chronic venous failure, the severity of which will depend on the level of ligation of the vein and the spread of further venous thrombosis. The greater the venous blood vessel is excluded from the bloodstream, the more pronounced chronic venous failure will develop.

In case of simultaneous injury of the major arteries and veins, the aid should be aimed at the restoration of venous blood flow, because of the possible thrombosis of the reconstructed arterial segment associated with a disturbed venous outflow and increased peripheral resistance in the injured arterial segment.

If the patient’s condition is unstable, especially in the case of polytrauma, the restoration of venous blood flow must be rejected, since such attempts, especially in general surgical hospitals, lead only to increased blood loss and destabilize hemodynamics of the patient. Sometimes it turns more problematic to control venous bleeding than arterial, even in specialized units. In such cases, the ligation of the major veins is fully justified and saves the life of the injured person.

IV.5. ASSESSMENT OF PAIN SYNDROME IN THE LIMBS DURING DIAGNOSING AND DIFFERENTIAL DIAGNOSING OF VASCULAR DISEASES, INJURY OF THE NERVOUS SYSTEM AND PATHOLOGY OF THE MUSCULOSKELETAL SYSTEM

Limb acute pain syndrome (LAPS) can be observed at a variety of diseases. It may be due to irradiation of pain in diseases of the internal organs of the chest, abdominal cavity, spine, and may be a sign of diseases of the bones, joints and peripheral nerves of the limbs, as well.

But most often LAPS is observed in acute diseases and trauma of major arteries and veins of extremities.

WHO statistics in recent decades indicate steady growth of acute thromboembolic lesions of the major arteries of the extremities and aortic bifurcation, which are complications of a number of severe cardiovascular diseases. The results of the treatment of this complex vascular pathology with the leading LAPS at present remain unsatisfactory: mortality due to AAO (acute arterial occlusion) of the limbs ranges from 25 to 35%, and each fifth survived patient (approximately 20% of patients) develops gangrene of the limb, which requires high amputation and leads to a steady disability of the patients. In Ukraine, the frequency of amputations with AAO of the limbs is 12-28%, and mortality reaches 25%.

LAPS is often caused by acute thrombosis of deep and superficial veins of the extremities. There are 160 cases of acute venous thrombosis per 100,000 population. The most frequent life-threatening complication of venous thrombosis is PE (pulmonary embolism) which ranks third among the causes of death of patients with cardiovascular disease.

Taking into consideration that the urgent pathology is found by doctors of all specialties, they should be well aware of the clinical signs of limb acute pain syndrome (LAPS), be able to diagnose it, be able to conduct differential diagnosis of this syndrome in order to establish an etiological diagnosis between the diseases that may be the cause of the LAPS development that are the following: acute arterial and venous obstruction, trauma of the arteries and veins of the extremities, as well as diseases of the nervous system and pathology of the locomotor apparatus. This knowledge will enable the timely diagnosis of an urgent patient, to provide competent first medical emergency aid and timely send such patient to a specialized vascular surgical department for qualified medical aid.

Consequently, ***acute pain in the limbs*** *can be observed at a variety of diseases, either of the extremities, or to be a manifestation of diseases of various internal organs, the spine, nervous system disorders.*

According to the ***R. Hegglin classification (1977), the pain is classified by origin***:

1. *Irradiating pain:*

- in case of the *chest organs diseases* (diseases of the heart and large vessels, illness of the lungs - almost always in the arm, in the back);
- in case of the diseases of the liver, gall bladder and bile ducts (in the arm, in the back, rarely in the legs);
- in case of the diseases of the spleen (in the arm, in the back, rarely in the legs);
- in case of the diseases of the digestive organs (mainly in the back, legs);
- in case of the diseases of the spine (in the arms and legs);
- in the affected shoulder girdle with neurovascular syndrome (in the arms).

2. Pain *in case of the diseases localized in the spine and extremities*.

Those irradiating pains in the group I of diseases are studied along with the disorders of the internal organs of the chest and abdominal cavity.

In the group II of diseases, when ***the spine is affected***, acute pain in the limbs occurs most often as a result of an *intervertebral disc hernia* and is characterized by acute pain in the lumbar spine and lower extremities that arise after an "failed" sharp movement or lifting the weight or other physical activity (the patients characterize them as "lumbago"). Pain increases at

moving, raising the legs, coughing, sneezing, laughing. This disorder is diagnosed by X-ray of the lumbar spine and magnetic resonance imaging (MRI).

Acute pain in the limbs can be observed ***in diseases of the spinal cord*** - tuberculosis and tumors. The acute pain is irradiating. The condition is diagnosed by MRI.

Acute pain in the limbs can be observed ***in neuralgia***. Characteristic for this pain to appear along the peripheral nerves of the limb. The pain is "tearing", aching, it may last from one second to a minute. The pain disappears between the attacks. No motor and sensory disorders are observed. Diagnostic method used: pressing on the sensory nerves, and in the places of nerve output (Valleix's points), the pain intensifies.

Acute pain in the limbs may be due to ***acute inflammation of the joints*** - *rheumatic polyarthritis, rheumatoid arthritis, with exacerbation of gouty arthritis, in degenerative diseases of the joints - acute arthritis, coxarthritis.* In all cases, except acute joint pain, there are other signs of joint inflammation - swelling, hyperemia, increased skin temperature, limitation of movements in the joint due to edema and pain. The pulsation on the major arteries is preserved. Diagnosis: X-ray of the joint, and special diagnostic methods, as well.

Acute pain in the limb is often a main symptom of ***acute occlusive diseases of the major arteries and veins of the extremities***. These include:

- acute arterial obstruction of the upper and lower extremities (acute arterial thrombosis and arterial embolism);
- acute thrombosis of deep veins, including Paget-Schroetter syndrome (acute thrombosis of the deep veins of the upper limb), white and blue (Gregoire's disease) pain phlegmasia of the lower extremities;
- acute thrombophlebitis of superficial veins;
- acute varicose thrombophlebitis.

Acute disorder of the blood flow in the major arteries is characterized by a sharp pain in the affected limb, which is hardly relieved by narcotic drugs. The skin of the extremity is pale, cold to the touch, no edema is seen. The pain does not decrease in the raised position of the limb (in case of acute venous thrombosis, the pain decreases with the raised position of the limb and increases at the lowering the limb or standing position). There are disorders of all types of sensitivity and motor limb function, *there is no pulsation in the major arteries*.

The main symptoms of ***arterial embolism (AE)*** due to the development of ***acute ischemia in the tissues*** distal to acute occlusion, are the following: sudden pain in the affected limb, its numbness, the limb turns pale and cold, impaired motor function of the limb of varying degrees (up to its paralysis, when the patient is not able not just walking, but even standing). Objective symptoms are similar to subjective: pronounced pallor of the skin with cyanosis, and later on with "marble" skin color of distal limbs; lowering of skin temperature; disordered all types of sensitivity (pain, tactile, deep); impaired active movements in the limb of varying degrees from the reduced muscular strength and limited volume to full plegia; a prognostically threatening three ‘A’ combination - *akinesia*, *areflexia*, *anesthesia*. At the late stage of severe ischemia, passive movements (due to the rigidity of the muscles and contracture of the joints) may be absent, the sharp tenderness of the leg muscles during palpation is determined by their subfascial edema and development of focal irreversible destructive changes in the tissues, that result in the development of the *gangrene* of the *limb.*

The most important objective symptom of arterial embolism is the absence of pulse in the segments of the major arteries, located distal from the levels of blockage, sometimes there is an increased pulsation on the segment of the artery located proximal from the place of embolism. For the differential diagnosis of arterial embolism from other diseases, which determine the possibility of the occurrence of LAPS, it is worth remembering that AE is *a complication of the so-called "embologenic" diseases,* therefore, in the *history* of such patients, there will be indications that they suffer from either one of the forms of *atherosclerotic cardiopathy* (atherosclerotic cardiosclerosis, postinfarction aneurysms of the heart, etc.), often with *cardiac arrhythmia*, or *rheumatic heart disease*, or *aneurysm of the aorta* and, most often, it is revealed

that before AE, the *limb of the patient did not disturb* (was healthy). In connection with the peculiarities of pathogenesis, AE *is characterized by the most acute onset, severe clinical symptoms (and the proximal limit of ischemic disorders is clear and most often corresponds to the level of embolization), usually a severe degree of acute ischemia and its rapid progression with the development of gangrene for 1-2 days.*

Clinical manifestations of ***acute arterial thrombosis*** (AAT) are similar to those with arterial embolism, but due to the peculiarities of pathogenesis, which differ from the pathogenesis of AE, *the symptoms are less pronounced, acute ischemia of the tissues in the distal limb parts is often not so pronounced and severe, gangrene develops rarer and much later (within 5-7-10 days), the proximal limit of acute ischemia is not so clear, there is no strict correspondence with the localization of the thrombus and the level of ischemic disorders.*

For the differential diagnosis of acute arterial thrombosis from other diseases (including from AE), which determine the possibility of LAPS, it is worth remembering that AAT is often a *complication of chronic occlusive diseases of limb arteries* (obliterating atherosclerosis, endarteritis, thromboangiitis), which lead to progressive stenosis of the major arteries of the limb and create conditions for the emergence of the pathogenetic triad of Virchow's pathological intravascular thrombi formation in the segment of the affected *artery with a critical stenosis, that is, until the development of the AAT.* Therefore, *in the history* of such patients there will be indications of the intermittent claudication symptom or pain in the limb at rest, especially night pain, that disable the patient of a complete sleep and forced him to use painkillers and somnolent agents, there may be complaints and objective signs characteristic of trophic disorders of the limb tissues (atrophy of the skin and muscles, hair loss, fissures, trophic ulcers, focal necrosis), that is, the extremity was affected before acute occlusion.

Differential diagnosis of different types of acute arterial obstruction is presented in Table 30.

Table 30

Differential diagnosis of different types of acute arterial obstruction of the limbs

Differential diagnostic sign	**Embolism**	**Acute thrombosis**	**Spasm**
Main disease	Rheumatic and sclerotic disorders of the heart. Atrial fibrillation.	Atherosclerosis, trauma, chronic occlusive diseases of the arteries of the extremities; malignant neoplasms	Heart disease, trauma, acute venous thrombosis.
Onset	Acute	Subacute	Acute
Pain	Very intense	Moderate	Intense
Ischemia	Pronounced	Moderate	Moderate
Skin sensitivity	Anesthesia	Reduced, but preserved	Reduced, but preserved
Motor functions	Ischemic paralysis	Lowered muscle strength	Lowered muscle strength
Skin temperature	Lowered	Moderately lowered	Moderately lowered
Color of skin	Pale, almost white or marble-like.	Pale	Pale, but in case of blue phlegmasia – purple or blue.
Limb edema	Rarely, may be observed after the first day of necrobiosis development and only on the shin.	No	Occurs early in the acute thrombosis of the iliac / femoral veins and involves the thigh; in the other forms is absent
Sphygmography	Presence of two zones: 1) zone of normal variations; 2) "silent" zone.	Presence of three zones: 1) zone of normal variations; 2) zone of collateral pulse; 3) "silent" zone.	Presence of two zones: 1) zone of normal variations; 2) "silent" zone.

Angiography	Contrasted proximal segment of the major vessel is unaltered, with a clear and smooth broken level, prominent upwards (an "inverted cup" syndrome). No collaterals.	Contrasted proximal segment of the major vessel with uneven «corroded» internal contours, notched line of break. Numerous convoluted collaterals, syndrome of segmental occlusion of the major vessel.	Contrasted major vessel with smooth disappearing of the contrast, or contrasted along the entire major vessel with uninterrupted contrast, but with sharply dilated diameter. No collaterals.

Differential diagnosis between the embolism of the aorta bifurcation and the detaching abdominal aneurysm of aorta.

In case of ***embolism of the bifurcation of the aorta***, the general condition of the patients is extremely severe, acute pain occurs in both lower limbs and in the lower abdomen, up to the development of pain shock. Pain is irradiated in the sacrum, in the perineum. Both limbs are white, and then marble-like; rapid disorder of all types of sensitivity appeared, the lower extremities are in an elongated position, no active motions in them are observed, the feet are dropped, *no pulsation is registered on all segments of the arteries of both lower limbs*, involuntary defecation and urination due to acute ischemia of the pelvic organs are possible. If no qualified aid is provided, the patients die against the background of severe intoxication and multiple organ failure.

If the aortic bifurcation embolism is combined with the ***embolism of the mesenteric arteries*** (or *occlusion of their bifurcation due to prolonged thrombosis* occurs), ***acute arterial ischemia of the intestine up to its necrosis*** develops as a result of acute disorder of mesenteric circulation. All above mentioned is accompanied by severe abdominal pain, liquid stool with an admixture of blood and pieces of mucus (ischemic colitis). It is accompanied by vomiting and collapse. The mortality rate is high in these patients.

In case of the ***abdominal aorta aneurysm detachment*** which may simulate the embolism of the bifurcation of the aorta, the general condition of the patients is slightly milder. The detachment of the aorta may occur due to the rupture of the inner lining near the base of the atherosclerotic plaque with medionecrosis and aneurysm of the aorta. The main difference in the clinical picture compared with the aortic bifurcation embolism is predominant localization of the suddenly occurring pain in the back, lower back and, as a rule, *irradiation in the lower extremities*. *Acute ischemia in this case is not so acute* as in case of embolism - movements are available, there are no marble-like maculas on the skin. In case of abdominal aorta aneurysm detachment, the sensitivity disorder is localized above the level of coldness.

In order to clarify the diagnosis, Doppler examination, ultrasonography, and abdominal aortography are required to be performed, as well.

The frequent cause of LAPS (limb acute pain syndrome) is ***acute venous thrombosis of the extremities*** (in more than 90% of patients the pathology is localized on the lower extremities).

Clinical manifestations of ***acute thrombosis of the superficial veins*** of the limbs are the following: discomfort, malaise, general weakness, low-grade fever (sometimes chills), pain and the painful seizures along the affected segment of the superficial vein, difficulties of walking due to pain in the foot.

In ***acute varicothrombophlebitis***, patients notice pain in one or more varicose nodes on the shin or pain in the thigh in the region of the thrombosed trunk of the large subcutaneous vein of the thigh, it is emphasized that the affected nodes became dense and anymore disappear in a horizontal position.

Examination of the affected lower limb with healthy veins reveals hyperemia and swelling of the skin over painful cord-like induration in the area of the vein segment on the shin or thigh, usually clearly separated from the surrounding tissues. In case of segmental thrombosis of the large subcutaneous vein, the affected site is located on the anterior-interior surface of the

leg or thigh, in case of segmental thrombosis of a small subcutaneous vein, the site is located along the posterior-external surface of the leg.

In acute thrombosis of varicose veins, hyperemia and swelling of the skin in the projection of the trunk of the affected varicose veins (more often on the thigh) or in the region of thrombosed varicose nodes (more often on the shin), which become dense, painful at palpation, do not disappear when raising the leg up, are registered.

The circumference of the affected limb, as a rule, does not change, that means no characteristic swelling.

Thrombosis of superficial veins of the upper extremities is rare and mainly is provoked by multiple punctures for the administered drugs or prolonged introduction of catheter in the superficial vein (so-called post-injection thrombophlebitis). In this case, along the affected vein above the elbow flexion, a strip of hyperemia is determined and a dense painful cord-like induration is palpated.

Diagnosis of acute superficial venous thrombosis often does not cause difficulties, and ultrasonography diagnostic technique introduced into practice allows this modality to be used in evidence-based medicine for diagnosis of this common condition.

Differential diagnosis of superficial venous thrombosis is carried out with Winiwarter-Buerger disease, lymphangitis, nodular periarthritis, erysipelas, hypodermic phlegmon, and also acute thrombosis of deep veins of the limbs.

Differential diagnosis of this pathology considers that the development of acute thrombosis of the superficial veins of the lower limbs, both of the unchanged and varicose veins, is acute; the contributing factors include the undergone recent infectious diseases, operations, traumatic lesions, presence of malignant neoplasms, allergic diseases.

In patients with ***thromboangiitis obliterans (Winiwarter-Buerger disease),*** the pathological process affects the superficial veins with migrating thrombophlebitis (thrombophlebitis migrans) and also affects the distal portions of major arteries and accompanies their deep veins. In subcutaneous veins there are single or multiple painful nodal indurations, the skin over them is hyperemic, swollen. The developed nodules are not migrating, but soon they appear on new areas of limbs, on the skin of the chest. The course of the disease includes periodic remissions and relapses several times a year, formation of new foci of thrombosis. Pulse on the arteries of the foot is weakened or absent.

In contrast to thrombosis, in case of ***lymphangitis,*** there is a painful pale pink narrow strip at the skin along the inflamed lymphatic vessels expanding more often from an infected wound or acute purulent process in the distal limb to the enlarged pain regional lymph nodes. The patients are disturbed by itching, burning sensation and tension along the affected lymphatic vessels. No induration or pain at palpation of superficial veins are found.

Nodular periarteritis is an allergic disorder affecting minor arteries and arterioles. In the skin and subcutaneous tissue, the painful nodules from millet to nuts in size, along small arterial blood vessels are found, and they do not remain for long time. The nodules appear during the period of exacerbation, which alternates with remission intervals for many years. Node biopsy is decisive in diagnosis.

Erysipelas inflammation is characterized by the formation of a small red spot first, which then gradually increases. The margin between healthy and injured skin is sharply outlined, and the area of the affected skin looks as if elevated above healthy areas; high body temperature to 38.5-39.0 ^{0}C is observed; headache, intoxication.

In case of ***subcutaneous phlegmon***, local infiltration and hyperemia with the formation of swelling of the surrounding tissues are determined locally. Usually, a pronounced general reaction manifested as chills and high body temperature, severe lymphangiitis and regional lymphadenitis are seen.

In thrombosis of deep veins of the shin, patients complain of headaches, palpitation, fever to 38.0-39.0 ^{0}C, swelling, cyanosis of the limb, pain in the shin, in the popliteal region, the

bursting pain. In both cases, the onset of the disease is acute, sudden, the causes of their development are similar.

Instrumental and laboratory diagnosis. For a very long time, thrombophlebitis of superficial veins was diagnosed by a physician based on only clinical symptoms, because, in fact, there were no non-invasive methods of venous blood flow examination.

Ultrasonography diagnostic methods introduced into the practice have opened a new stage in the study of this common disease. It should be considered that ultrasonography methods of diagnosing venous thrombosis involves ***duplex scanning*** as a determinant technique, since only this method makes it possible to outline thrombosis exactly, determine the degree of the thrombus formation, the permeability of deep veins, the state of communicants, and the valve apparatus of the venous system. Unfortunately, the high cost of the required equipment so far sharply limits its practical use in outpatient and inpatient settings.

This examination is indicated, first of all, for the patients with a suspected *embologenic thrombosis*, i.e. in case when the thrombus passed from superficial to deep venous system through the saphenofemoral or saphenopopliteal anastomosis. The study can be conducted in several projections, and it increases the diagnostic value greatly.

Phlebographic study. At acute thrombosis the indications for phlebography are extremely narrowed. The need to perform it arises only in the case of the thrombus spread from the major subcutaneous vein to the common femoral and iliac vein. Moreover, this study is conducted only in those cases where the results of duplex scanning are doubtful and their interpretation is complicated.

Laboratory diagnostic methods. In the common clinical analysis of blood, attention is paid to the level of leukocytosis and the ESR level. *Coagulogram, thrombelastograms* are obligatory to be performed, the parameters of which characterize the state of coagulation-anticoagulation blood system, which are most often are evidence of *hypercoagulation*.

Acute deep vein thrombosis (***ADVT) of the lower extremities*** is more often developed in elderly patients suffering from cardiovascular diseases, diabetes mellitus, obesity, in the elderly and oncologic patients; they often develop in severe trauma, traumatic and prolonged operations, in pregnant women before and after childbirth. They can complicate the course of infectious and purulent diseases. These conditions are the risk factors for thromboembolic diseases and complications.

Clinical picture of ***acute thrombosis of deep veins of the shin*** for 1-2 days is often obliterated. The general condition of the patients remains relatively satisfactory, there are mild pains in the calf muscles, which increase when moving, a small swelling of the lower third of the shin, pain in the calf muscles during palpation. ***Characteristic symptoms of thrombosis of deep veins of the shin***: *Homans' sign, Meyer's, Opitz-Ramines'; positive Lovenberg's and Moses' tests.*

The clinical picture becomes pronounced when all *three paired deep veins of the shin*. It is accompanied by a sharp pain, a feeling of bursting, tension, edema of the shin, which is often combined with cyanosis of the skin and elevated body temperature.

When thrombosis extends to the ***femur vein,*** there is a hip swelling that is never significant, unless the mouth of the deep thigh vein which has a rich network of anastomoses with branches of the femoral vein, is blocked. Palpation along the thrombosed vein is painful. In case of combined thrombosis of the femoral and popliteal veins, sometimes there is swelling, pain, limited movement in the knee joint.

In case of ***iliofemoral venous thrombosis***, patients are concerned about pain in the anterior-internal thigh surface in its upper third, in the inguinal region of the abdomen. The extremity increases in volume, swelling extends from the foot to the inguinal fold, sometimes passing to the buttocks and lower abdomen. The color of the limb varies from pale to cyanotic. Tenderness along the major veins on the thigh and in the inguinal region is palpated. 3-4 days after the onset of the disease, the edema slightly decreases and an intense pattern of subcutaneous veins appears, due to the complicated blood outflow from the deep veins.

Sometimes the disease begins suddenly with acute throbbing pains in the limb, its coldness and numbness, as at arterial embolism. Total edema increases rapidly, the movements of the toes become limited, the sensitivity and skin temperature of the distal limb segments decrease, the pulsation of the arteries weakens or disappears. This form of iliofemoral thrombosis is called *pseudo-embolic,* or ***white pain phlegmasia*** *(phlegmasia alba dolens),* it occurs at combined deep vein thrombosis with severe spasm of the arteries of the affected limb. In the commonest case it occurs as a complication of septic endometritis after delivery. The clinical course, although severe and prolonged (up to 3-4 weeks), but benign (the patients, as a rule, recover).

Unlike white phlegmasia, ***blue pain phlegmasia (phlegmasia cerulea dolens)*** has an extremely severe course with the development of terrible complications and often finishes with an adverse result (mortality reaches 60%), due to massive thrombosis at the level of venous macro- and microcirculation, and even total thrombosis of the entire veins of the lower limbs, sometimes involving the arterial system. The onset of the disease is acute - suddenly an intense, '*throbbing' pain* in the extremity and in the buttocks, a sharp *'bursting'* feeling appear; *movements in the limb are impossible* due to acute pain; *swelling* develops rapidly and reaches a sharp degree within several hours; the skin is cold to the touch, the *pulsation of the major arteries is not determined*; *violetish cyanosis appears* (it involves not only the lower limb, but also the lower abdominal and buttocks on the side of the lesion, often is of a steady "spotty" character), then the areas of *petechial hemorrhages* that merge into a *purple-cyanotic spots* with subsequent epidermal detachment and formation of *hemorrhagic blisters* with a stinking smell, indicating the onset of ***venous gangrene***. The general condition of patients reaches the extreme degree of severity; hyperthermia and hyper-leukocytosis are observed, often clinical picture of hepatic and renal or multiple organ failure associates, hypovolemic and septic shock, thromboembolism often develops in the pulmonary artery, and patients quickly die.

Ascending thrombosis of the inferior vena cava is a complication of thrombosis of the major pelvic veins. Swelling and cyanosis at the same time involve a healthy limb and spread to the lower half of the body. Pain in the lumbar and hypogastric areas is accompanied by a guard tension of the muscles of the anterior abdominal wall.

Diagnosis of acute thrombosis of the major veins of the lower extremities is based on clinical picture of the disease and findings of instrumental examination. Ultrasonography duplex scanning is the most simple and safe method for detecting phlebothrombosis. It helps to "catch" the lumen of the inferior vena cava, iliac, femoral, popliteal and calf veins, to specify the degree of narrowing of the lumen of the vein, its type (occlusive, non-occlusive), to determine the length of the thrombus and its mobility (*floating thrombus*). Thrombosed vein becomes rigid, not tense, its diameter increases, it is possible to visualize intravascular inclusions (thrombotic masses) in the lumen. In occlusive thrombosis, blood flow in the lumen of the veins is absent, in case of non-occlusive thrombosis it is possible to observe how the contrast substance flows around the thrombus through the narrow, preserved lumen of the vein. In case of floating thrombosis there is an incomplete fixation of the thrombus to the venous wall, the movements of the top of the thrombus is seen in the beat of breathing.

Ultrasound duplex scanning is used to differentiate iliofemoral venous thrombosis from edema of the lower extremity of another etiology (lymphedema, compression of veins by tumors, by inflammatory infiltrates).

Phlebography is crucial in the diagnosis of floating blood clots, especially in cases of not clear duplex scans visualize the tip of the thrombus. The main *angiographic signs* of acute thrombosis are the *absence of contrast* or the *symptom of the "amputation stump"* of the major veins, *filling defects* which are present in the lumen of the vessel. The last sign indicates non-occlusive thrombosis. Visible thin layers of the contrasting substance which circulate around the thrombus, and visible around it strips are called *"railroad" symptom.* Extending top of the thrombus may float over the surface of the occluded segment or spread to the lumen of non-occluded vein. Dilated deep calf, popliteal and femoral veins, continuous retention of contrasting

substance in them are considered to be indirect signs of obstruction of the iliac veins, revealed at distal phlebography. The nature of the pathological process, which prevents the venous outflow from the calf and femoral veins, is determined by *proximal (pelvic) phlebography*.

Instead of conventional X-ray contrast phlebography**,** ***magnetic-resonance phlebography*** can be used in complex cases for differential diagnosis. MR-phlebograms show thrombotic masses in non-occluded thrombosis like filling defects against the background of a bright signal from the moving blood. If the thrombus occludes the lumen of the vein, no MR signal from the venous segment excluded from the circulation is registered.

Most often ***differential diagnosis*** of acute thrombosis of lower limbs deep veins has to be performed with acute arterial thrombosis and arterial embolism, neuritis of the femoral and sciatic nerve, lymphostasis, femoral phlegmon, hematogenous osteomyelitis.

Considering the ***differential diagnosis*** of acute thrombosis of deep veins of the lower extremities, it is necessary to take into account that among the most frequent causes which contribute to its development (and in superficial thrombosis, as well) the following factors are of value: those undergone recently infectious diseases, operations, traumatic injuries, the presence of malignant neoplasms (especially the tumors of small pelvis), allergic diseases; this condition is often results as a complication of childbirth, especially if it was accomplished with applying of forceps, manual handling, etc., if the course of the post-partum period proceeds with the phenomena of endometritis.

In case of ***neuritis of the femoral or sciatic nerve***, the pain is localized on the anterior or posterior surface of the limb along the corresponding nerve trunks.

Lymphostasis of the lower extremities usually develops along with the inflammatory process and lymphadenitis. There is no pain syndrome and dilated superficial venous vascularity. Swelling of the lower extremities, associated with the development of ***cardiac and renal failure*** are characterized by the same features; they are often symmetric.

Differential diagnosis of ADVT at acute venous iliofemoral thrombosis and in case of ***phlegmon of the thigh***.

Both diseases have ***similar general symptoms***: high fever, chills, headache, general malaise; pain, swelling and discoloration of the skin of the limb. However, at acute venous iliofemoral thrombosis, *general phenomena* are observed in the prodromal period, whereas in case of the phlegmon of the thigh they are observed before the phlegmon is tapped.

Pain at the acute venous thrombosis (AVT) is intense, it is localized in the inguinal region, along the anterior surface of the thigh; it can spread to the calf muscle, due to venous hypertension, and the development of periphlebitis, as well. In case of the femoral phlegmon, pain is also observed on the anterior and medial surfaces of the thigh, the patients complain of the feeling of weight in the extremity, but *more local*, and in case of AVT pain is also obligatory registered in the shin.

Edema at AVT has a disseminated character, involving the entire lower limb (from the foot to the inguinal ligament), often passing to the bottom of the abdomen, buttocks. If the phlegmon of the thigh develops, then swelling is on the thigh is seen and it may extend to the knee joint area, but it rarely spreads to the shin.

Discoloration of the skin in case of AVT is more often of a cyanotic color, less often as a "spotty cyanosis," and sometimes pallor of the skin can be observed, which is due to lymphatic drainage disturbances, and rarely arteriospasm is registered. If the phlegmon of the thighs develops, there is diffused redness of the skin over the inflammatory process, increased skin temperature, the pain is intensified precisely at the place of phlegmon formation; later *a fluctuation* appears in this place, but it does not happen at AVT.

If the patient develops acute erythematous venous *thrombosis,* within 5-7 days from the onset of the disease an *increased pattern of subcutaneous veins* appears after the edema of the limb decreases. In case of phlegmon a fluctuation appears.

The additional methods (ultrasonography and Doppler examination of the major veins of the limb) help to differentiate these diseases, especially in the initial stages. In case of

hematogenous osteomyelitis, the specific clinical picture depends on age, localization, protective forces of the patient's body, the nature of the infection, virulence of the pathogens, and on the treatment, as well. Within 1-2 days, the patient complains of general malaise, aches in the limbs, muscle aches, headache, sometimes vomiting. General condition of the patient: chills, high fever, tachycardia, tachypnoe, often loss of consciousness is observed. The temperature is high - 39-40 ^{0}C, especially in the first days, then, as the process progresses, it shows a remitting character with large variations during the day. Leukocytosis reaches 19×10^9 / L, there is a shift in the leukocyte formula to the left, decreased eosinophils and monocytes, increased anemia. *Local symptoms* develop from the first day of the disease. The local symptoms include bursting pain in the affected limb. Patients are lying in bed completely motionless, because the slightest movement and the touch to the diseased limb cause dramatic pain. When the site of the pain is located close to the joint, the limb takes the semi-arched position, and tapping the joint (elbow, knee) or the leg causes a sharp pain. In the next 1-2 days local swelling of soft tissues appears, which rapidly increases; there is hyperemia, swelling of the skin, local temperature elevation, regional lymph nodes are enlarging. By the end of the week 1 (in children) and in 2 weeks (in adolescents) the *fluctuation* appears.

To diagnose osteomyelitis, an *X-ray examination* and X-ray contrast is used - ***fistulography*** (if fistula is present). Using these methods, the localization, length and nature of the pathological process are determined.

X-ray signs of osteomyelitis: detachment of the periosteum (a thin linear shadow with a smooth outer and slightly uneven inner contour, raised above the shadow of the cortical layer of diaphysis); blurring of the bone structure; vague bone crosses; the alternation of the rare and dense areas (the rare areas have an elongate shape, respectively, along the Haversian canals, the cortical layer lying against the periosteal rim, loses smooth contours and looks corroded with some defects).

Complete X-ray pattern in case of osteomyelitis becomes apparent by the end of the second month of the disease, and sequestration - by the end of second-fourth months. Fistulography is of special significance if the fistula has formed - it helps to clarify the localization of the sequestration and to detect it in those cases when conventional X-ray studies are not clear enough; the following X-ray contrast agents are used: iodolipol, sergosin, triombrast et al.

Radioactive scanning using radioactive technetium is considered to be the most precise to identify the affected area of the bone.

Surgery of the upper extremities is often caused by ***Paget-Schroetter syndrome*** - *it is an acute thrombosis of the subclavian vein with its dissemination the armpit and veins of the shoulder and impaired venous outflow in the upper extremity.*

The development of the disease is favored by a number of features of the subclavian vein, which is surrounded by osseous and tendon muscle formations. Under severe stress of the muscles of the shoulder girdle, combined with movements in the shoulder joint, the size of the subclavian space is diminished, and traumatising of the proximal part of the subclavian vein in the costo-subclavian osseomuscular canal. People with well-developed musculature who deal with sports and heavy physical labor suffer more often. Men are affected more often than women (1: 4). Especially favorable conditions for impairment of the outflow on the subclavian vein, and, consequently, for thrombogenesis occur at *high position of the first rib, hypertrophy of the subclavian muscle and muscle-tendon portion of the minor thoracic muscle.*

Classification of Paget-Schroetter syndrome. The ***acute and chronic stages*** of the disease are classified, and in the acute stage, there are distinguished three forms that reflect the parameters of venous hypertension, which characterize the acute disorders of the blood outflow. In the *mild form* of the disease venous pressure does not exceed 300 mm of water column, *in a moderate form* - there is a venous hypertension ranged from 400 to 800 mm of water column, and in *severe* venous pressure reaches 1200-1300 mm of water column. Hypertension in the veins of the extremities is most pronounced in the first days of the disease, and as the collateral

outflow of blood and the recanalization of the thrombosed portion of vein gradually diminishes. In the chronic stage of the disease in a state of rest, hypertension in the veins of the affected limb is moderate, but the blood outflow failure is clearly manifested at the muscular exertion.

Paget-Schroetter syndrome is characterized with the acute onset and rapid development of the disease, often after considerable physical exertion shortly before. Patients complain of the appearance of edema without any previous unpleasant sensations, but which is accompanied by pronounced cyanosis of the skin and a feeling of heaviness, bursting, up to painful sensations. Often there is a discrepancy between the pronounced local manifestations and the general condition of the patient, which practically does not suffer, no rise in body temperature is observed. Subcutaneous veins of the upper limb and shoulder girdle of the respective side are tense and dilated, dense, moderately painful swelling along the inguinal and sometimes the femoral vein, pain at palpation in the projection of these veins is observed. In case if the thrombosis spreads on the inguinal and femoral veins, the course of the disease becomes severe. The duration of the acute stage does not exceed 2-3 weeks. During this period, acute events subside, collateral vascularity develops in the shoulder region and anterior chest wall of the corresponding side. The further course of the disease is stable with periodic worsening of the condition and becomes a post-thrombophlebitis disease of the upper limb.

In severe cases venous gangrene is possible and rarely thromboembolism of the pulmonary artery. The pulsation on the radial artery is preserved, but it may be weakened by edema of the arm.

Phlebography is a valuable ***diagnostic method.*** The phlebogram determines the localization of the thrombus, its extent and degree of development of collateral ways of blood outflow. Thrombosis radiologically manifests itself in the form of "*amputation*" of the major vein and the *no contrasting of it*, which is characteristic for all cases of occlusion of the subclavian, inguinal and femoral veins. *Phlebomanometry* is combined with phlebography. In case of Paget-Schroetter syndrome, venous pressure on the affected limb, depending on the degree of severity of the disease, can reach 800 and more mm of water column (norm - no more than 150 mm of water column).

Also, *ultrasound duplex angioscanning* is a valuable method of diagnosis, which, unlike phlebography, is safe for the patient; chest and cervical spine *X-ray* is necessarily to be performed.

Differential diagnosis should be carried out, first of all, with acute arterial obstruction of the upper limbs (with embolism and acute thrombosis of the major arteries).

Acute pain limb syndrome also occurs in ***acute trauma of major vessels (arteries and veins) of the extremities.***

Injuries of the major vessels in peacetime ranges from 0.25% to 0.6% of the total number of patients with mechanical trauma, and refers to severe life-threatening traumas. The treatment of patients with vascular injuries is complicated due to the objective difficulties of diagnosis, insufficient personal experience of doctors providing first aid. Untimely diagnosis, unqualified measures of the physician increase the severity of injury, lead to serious, life-threatening complications.

Under certain circumstances, during traumas of the vessels, first aid and urgent surgical intervention are performed by non-vascular surgeons (as appropriate), and by general surgeons, traumatologists, neurosurgeons and other specialists in the surgical procedure. A doctor of any surgical specialty in his day-to-day work may be in a situation when it is necessary to decide independently the issues of tactics and surgical treatment of patients with vascular injuries, and should be prepared for this.

Classification of vascular injuries. Arterial injuries are subdivided into: *closed and open injuries* (and *gunshot wounds* are distinguished as a separate group, both due to the features of the mechanism of injury of the vascular wall, and the area of affection which can be much larger than in other types of trauma and extend beyond the limits of macroscopically visible changes); *penetrating and non-penetrating traumas*.

In the case of ***closed injuries***, broad hematomas are formed, signs of internal bleeding are observed, in ***open injuries*** - external bleeding, often significant is registered.

Penetrating injuries are characterized with a complete anatomical disorder of all layers of the vascular wall, which is accompanied by bleeding. In case of ***non-penetrating injuries***, there is no complete disruption of the vascular wall, there may be an isolated wound of the outer layers of the vascular wall, ruptures and detachment of the intima, contusion, compression and traumatic spasm of the vessel. *An acute arterial obstruction negative for signs of bleeding* is a ***characteristic clinical syndrome,*** and the clinical picture of obstruction can be revealed not immediately after injury, but later, when thrombosis appears. In peacetime, non-penetrating injuries make up 20-30% among other vascular injuries, as a rule occur due to blunt trauma, contused sites, they are often observed at combined trauma, and accompany the osteoarticular injuries. Erroneous diagnosis and treatment tactics, untimely diagnosis and surgical treatment in these injuries are observed especially often.

Depending on the nature of the injury and complications of the vascular injuries, the diagnosis can either be complicated or uncomplicated. ***Complications:*** *blood loss, shock, development of tense hematoma, acute arterial failure.*

Diagnosis of vascular injury both can present no difficulties or can be complicated, due to the variety of clinical symptoms, depending on the type of injury that accompanies the lesion to soft tissues, nerves, bones, combined trauma of another anatomical region. The diagnosis may be complicated due to the fact that the signs of combined injuries, such as craniocerebral, thoracoabdominal and osseous usually prevail in the clinical picture.

Diagnosis of vascular injury is based on the analysis of the mechanism of injury, the identification of characteristic ***local symptoms*** *of open and closed vascular trauma.*

Injuries of the limb arteries are often observed: femoral, brachial, antebrachial, popliteal, iliac arteries; less commonly the wounds of cervical vessels occur. Injuries of aorta, the inferior vena cava and their visceral branches are rare, but they are the most severe traumas.

Local symptoms of open injuries of arterial vessels: localization of the wound in the arterial projection area, external bleeding, development of pulsating hematoma, absent or weakened pulse below the site of injury, the following signs of arterial failure: discoloration and decreased temperature of the skin of the limbs, impaired sensitivity. The injury of the blood vessels caused by cutting objects is often accompanied by bleeding. Such patients arrive as a rule, with a tourniquet applied on the limb. Signs of significant blood loss, and renewal of bleeding after the tourniquet is released, as well, allow diagnosing the condition precisely. If there is no external bleeding, but there is a wound in the projection of the artery, the diagnosis is clarified by *additional examination and during the revision during the operation*. In case of gunshot wounds, external bleeding may be absent, but there are often signs of acute obstruction of the major arteries.

Diagnostically more complicated are *closed penetrating injured vessels*, with the following clinical signs:

1) pulsating hematoma;

2) absence or disappearance of a previously defined peripheral pulse;

3) signs of acute arterial failure - pain below the injury site, pallor and cyanosis of the skin, impaired sensitivity and disordered motions.

It is important to know that arterial injury can often occur at the following injuries of the bones and joints.

1. Process extensor brachial fractures with displacements of the fragments; sharp edges of the splinters can injure the brachial artery at the moment of injuring or during the repositioning of the pieces, or compression of the vessel results from the developed hematoma at the site of the fracture and tissue edema.

2. Process femoral fractures with a typical displacement of distal posterior fragment that injures popliteal vessels.

3. Posterior dislocation in the knee joint, fracture-dislocations of the tibial process with displacement of the fragments back in position, that injures popliteal artery.

4. Dislocation of the shoulder or hip with a large displacement and hyperextension of the arteries, resulting in a rupture of intima and thrombosis or complete interruption of the arteries.

5. Diaphyseal fractures of long tubular bones are often open with large displacements of fragments.

Acute arterial failure with acute ischemia of tissues is the prevalent syndrome *of non-penetrating injury of the arterial vessels of the extremities.*

The degree of ischemia depends on the state of collateral blood circulation and the time that has passed since the injury occurred. In the early period, signs of ischemia can be insignificant, so injury of the vessels is not diagnosed timely.

It is especially difficult to diagnose non-penetrating arterial injuries in patients with fractures and dislocations of bones. Pain, impairment of the function of the limb, hematoma, edema and other signs of osteoarticular trauma mask the symptoms of vascular injuries.

Displacement of blood vessels, compression of bone fragments, that are anatomically non-damaging, also cause severe disturbances of the circulation of the limb. General clinical findings may be inadequate for diagnosing; ***angiographic methods of examination*** should be wider used for these patients.

Contusion of artery, rupture of the inner and middle layers are revealed only if *blood vessel thrombosis occurs* with the development of limb ischemia. Traumatic spasm while anatomical integrity of blood vessels can cause a picture of acute arterial thrombosis. For differential diagnostics of these injuries, *angiographic examination*, *revision of blood vessels* in doubtful cases should be performed, and the value of medical therapy (novocaine blockade, administration of antispasmodics, anesthetics, etc.) must not be overestimated, unless they have a rapid effect.

In case of gunshot, and stab wounds when the bleeding is stopped, the injury of blood vessels is most strongly confirmed or excluded during the revision of the vessel during surgery. *The wound in the projection of major vessels should always be suspected concerning a possible vascular trauma, even in the absence of bleeding.*

Indications for emergency angiography in case of the trauma of the vessels of the limbs are the following: blunt trauma of the limb with ischemia of the lung, suspected trauma of the artery or vein at fractures of the tubular bones or dislocations; signs of limb ischemia after the reduction of bone fragments, after reduced dislocation; unclear localization of injury of vessel; late post-traumatic thrombosis of the major arteries.

Angiography is also indicated after surgery, if the patient has no pulse in the peripheral vessels after a restorative operation. Angiography is performed by transcutaneous puncture of the femoral or brachial arteries.

Besides the condition of the patient, the *degree of limb tissue ischemia and its duration* is a very important factor, which determines the treatment tactics of trauma of major vessels.

In practice, it is not always possible to determine reliably the degree of reversibility of tissue changes in case of severe ischemia, however, as a rule, contracture of the muscles in most cases is an evidence of irreversible changes in tissues, the risk of involving the ischemic limb in the bloodstream, and requires primary or secondary amputation of the limb.

If the case of injury occurs at LAPS, it is necessary:

1) to determine the cause of pain;

2) if the injury of the vessel has been revealed, to determine its nature;

3) to determine the degree of acute tissue ischemia and the viability of the limb.

Diagnostic algorithm of LAPS in case of trauma.

Stage I *The purpose is to determine the cause of pain:*

1) bone and muscle injury;

2) nerve injury;

3) artery injury.

This goal is solved with a careful interviewing of the patient to get a history and studying the nature of the influence of the impact force.

In case of bone injuries, the pain is very sharp at the time of injuring, sometimes it causes shock, and increases at the slightest change in the position of the limb. Local pain at palpation, deformation or shortening of the limb, pathological crepitation is determined. X-ray findings confirm the nature of bone injury.

Injury of the nerve can be considered according to the nature of the injury in the place of its projection. The injury of the nerve is manifested as a complete or partial impairment of nerve conduction, including intermittent movements, sensitivity and autonomic functions, which are subsequently accompanied by trophic disorders. Pain and unpleasant sensations in case of nerve injury can be mild and often patients do not mention them.

Injury of the artery is characterized by a wound in the area of the projection of a vessel, bleeding, the appearance of pulsating swelling at the site of injury, absence of pulsation distal to the injury site. Ischemic pains appear a little later and are accompanied by pallor and coldness of skin, impairment of sensitivity and movements in the extremity. The findings about the artery injury are confirmed by angiography.

Stage II *The purpose is to determine the nature of injury of the arterial vessel:*

1) closed, open (including gunshot wound);

2) uncomplicated, complicated;

3) combined.

Complicated arterial injuries are characterized by significant blood loss, hemorrhagic and traumatic shock, acute arterial failure with irreversible massive mechanical and ischemic injuries of soft tissues, and others.

Combined injuries of major arteries are characterized by a clinical picture of injury of other organs or tissues of this anatomical region (bones, nerve, internal organs, etc.). Osteovascular lesions have extremely severe course and are accompanied by shock, therefore timely diagnosis of vessel injury may be complicated. This is why, in *every patient with bone fractures and dislocation of joints, it is always necessary to examine the pulse, sensitivity and movements.*

After the repositioning of the fragments or reducing of the dislocations, after the skeletal traction is set, it is necessary to perform these studies once more. If a complete clinical examination does not exclude vascular trauma, it is necessary to perform angiography.

Stage III *The purpose is to determine the degree of acute arrhythmia (acute ischemia) and tissue limb vital capacity.*

The degree of ischemia depends on the nature and length of the anatomical injury to the major artery (full or partial interruption), the degree of injury of the soft tissues, the state of collateral blood circulation and the time passed after the injury:

I - a mild degree includes cases of mild ischemic manifestations in the form of a feeling of numbness, paresthesia and pain during functional exertion, decreased skin temperature of the distal parts of the limb; in general, the circulation of the limbs at rest is relatively compensated;

II – medium degree is characterized by signs of an impairment, mainly of the *nervous apparatus*: pronounced pains at rest, disordered pain and tactile sensitivity, restricted or absent active movements in distal joints, but no rigidity, contracture, muscle edema; extremity is pale, skin temperature is lowered; blood circulation in the extremity is decompensated, however, tissue changes are reversible, the restoration of blood flow at this stage usually leads to complete restoration of the function of the limb;

III - severe degree is characterized, in addition to motor paresis and impairment of sensitivity, by the beginning signs of *necrobiotic changes of the muscles*: tenderness in the muscles at palpation, restriction of passive movements in the joints of the distal limb parts due to rigidity, initial signs of developing contracture, of individual muscular groups; restoration of blood circulation in the extremities in such patients usually results in the *development of the*

"syndrome of inclusion" of the ischemic limb, complete restoration of the limb function is usually not observed, there are consequences in the form of Volkmann contracture, fibrosis of muscles, ischemic neuritis; gangrene of individual fingers or distal portion of foot (hand), as well as necrosis of muscle groups may develop;

IV - the degree of irreversible tissue changes is characterized by sharply expressed contracture of the muscles, their subfascial edema, later by the disorder of passive movements in major joints as a result of their contracture, appearance of necrotic changes of tissues of distal limbs and demarcation zones - *development of gangrene*, general features of intoxication.

Differential diagnosis between the III and IV degree of acute ischemia, determining the viability of the limb tissues can present significant difficulties. In practice, this is essential for the purpose of determining the treatment tactics, since the use of restorative surgery is possible at a grade III, and in patients with degree IV ischemia, the amputation of the limb is clearly indicated. Unfortunately, at present there are not enough reliable and accessible special express methods for determining the viability of tissues and the practical surgeon has to focus mainly on clinical manifestations.

Acute trauma of ***venous vessels of extremities*** manifests by various clinical signs, the most important of them are the following:

1) severe bleeding is clearly venous (non-pulsating, dark blood);
2) hematoma in the vein projection area;
3) cyanosis of the skin of the limb to the periphery from the place of injury;
4) swelling of peripheral subcutaneous veins;
5) edema of the limb.

These signs are usually combined with the localization of trauma in the projection of a major vein. In case of bleeding from the veins of a large caliber the bleeding is characterized by abundant flow of dark blood, which can lead to the death of the injured. In case of injury of the veins of medium and small caliber, bleeding is less dangerous due to the decreased veins, the coagulation of blood in them and obturation of the lumen of the wound canal with blood clots.

Often injury of the veins is combined with wounds of the homonymous arteries and the frequency of such injury varies from 40 to 70% in relation to all injuries of the blood vessels. If venous blood has no possibility to flow freely outward, the *internal hematomas* are formed.

Venous hematomas in case of wounding subcutaneous veins are diffuse and, unlike arterial hematomas, they *do not pulsate*. Localization of the wound must raise awareness, because the injury of the vein may be revealed not immediately, but in a few hours, and sometimes in a day. Diagnosis is particularly complicated at combined injury of the veins with the homonymous artery, because in such cases, the symptoms of injury of the arteries are in the first place. In such cases, the pulsation of the arteries is weakened or disappears, and acute ischemia increases.

Air embolism is a less frequent complication, especially when injuring the axillary, subclavian, jugular or brachiocephalic veins. The hiatus of the lumen of the vessel leads to the aspiration of air in the respiratory movements, which with the blood flow penetrates into the right chambers of the heart, and then into the pulmonary artery, causing embolism of its branches.

In the case of trauma to the veins, when the permeability of many superficial and deep veins is disturbed, ***"venous gangrene***" (necrosis of the limb***)*** may develop.

3 phases of "*venous gangrene" are distinguished:*

- ***in the first phase of "white phlegmasia"*** there is a white edema of the limb and a visible pattern of subcutaneous veins, indicating spasm of the arteries and compensatory venous circulation;

- at thrombosis or spasm of the collateral veins, a ***second phase or "blue phlegmasia",*** manifested by cyanosis of the limb, pain and the appearance of necrosis; both of these phases are reversible;

- with the progression of thrombosis there is a total occlusion and blockade of the entire venous system in the distal parts of the limb and the ***third phase - the gangrene phase.***

Diagnosis of vein injuries is based on the history findings and correct assessment of clinical symptoms.

Among the instrumental examination the leading are ***Doppler ultrasonography and phlebography***. They allow determining the nature, extent and localization of injury.

At the prehospital stage, when ***providing the first urgent medical care,*** *it is important to* ***choose the optimal method for temporary hemostasis***. Possibly, the *advantage should be given to the tight wound tamponade*, which reduces the risk of disturbance of collateral blood flow and additional injury to the surrounding tissues.

If tight tamponade is ineffective, it is necessary to *apply a tourniquet more distal to the place of injury.* This is the simplest but most traumatic way to stop bleeding temporarily.

Observations show that it is used more often than it is necessary, in particular when only venous vessels are injured, when the hemostasis can be provided with a well-applied pressing bandage. The time of the tourniquet applied should not exceed 2 hours. It is necessary to try to provide the rapid replacement of the tourniquet by another one, better way of temporary stopping of the bleeding, in the first place by pressing bandage. *The method of providing a limb or a body of a certain position* can also be applied, in which the bleeding vessel is compressed by surrounding anatomical formations.

The following should be considered as ***emergency measures*** in case of vascular trauma of the extremity: antishock therapy, adequate restoration of blood loss, measures to prevent the progression of limb ischemia and the development of wound infection. In the complex of treatment of ischemic disorders, it is advisable to use vasodilators and anesthetics, novocaine blockades, agents that improve blood rheological properties (rheopolyglucin, hemodez), heparin under condition of reliable hemostasis. Applying of the local hypothermia of the limb is also recommended.

In case of severe trauma of extremities, there should be an individual tactical decision concerning indications for savings operations, depending on the massiveness of the destruction of bones, soft tissues, vessels and other anatomical formations, the period from the moment of injury, age, general condition of the patient, and also the presence of the necessary conditions for operations to be performed and postoperative period to be provided.

Currently***, replantation of the upper limb, the hand, the fingers, in some cases of the foot, is real.*** Macroreplantation is performed under the conditions of the vascular departments, as well as the ambulatory team of angiosurgeons in surgical and traumatological hospitals.

The detached part of the limb should not be disinfected, rinsed with antiseptics, it must be wrapped in a clean or sterile napkin, a scarf and placed in a plastic bag. There is also a place to put other individual tissues, for example, scalped skin. For prolonged transporting, the polyethylene bag with the part of the limb should be placed into another plastic bag in which iced water is poured. A dry pressing bandage should be applied on the stump. The bleeding is stopped by pressing, tamponade. It is necessary to avoid applying a tourniquet or a clamper on vessels, if necessary, anti-shock measures should be done.

Terms within which a successful replantation can be performed under conditions of preservation of the "amputated" part at + 4° C: for the entire upper limb - 6-8 hours, for a hand or foot - 10-12 hours, individual fingers - up to 20 hours. In this case, it is necessary to take into account the time of the operation until the restoration of arterial blood flow. Indications for replantation are determined by various factors, the condition and age of the patient, the state of the "amputated" part (it should not be smashed, the slaughtered place) and the stomachs, the duration of the anoxia period, the possibility of restoring functionally important structures, especially the nerve trunks, muscles, localization and character of the injury, and the patient's desire, as well.

Indications for re-plantation: amputation of the fingers I and II of the hand, forearms, multiple finger separation in the injured patients, usually up to 40 years of age, hands in children;

type of injury - sharply cut (cut, chopped, electric saw). ***Relative indications for re-plantation***: amputation of individual fingers III, IV, V, terminal phalanges of the fingers, separations, contusion and scalped injuries. ***Contraindications:*** macroreplantation in patients with severe shock, high blood loss, combined injuries with another anatomical region, significant injury to the nerve trunks, hopeless in the sense of their recovery; microreplantation of individual fingers with injured joints.

IV.12. ANTICOAGULANT AND THROMBOLYTIC THERAPY IN SURGICAL DISEASES OF THE VESSELS OF THE EXTREMITIES. PREVENTIVE ANTICOAGULANT THERAPY

Pathological intravascular clotting, with the leading three main factors in its pathogenesis which are known as the pathogenic Virchow's triad, despite thorough and long-term scientific study and certain achievements, remains a problem which is relevant and is not solved finally either diagnostically or therapeutically, or in preventive aspect, not only in scientific and practical angiology, but also for all medicine on a global scale.

This threatening pathology affects the arterial and venous vascular systems, both organ vessels and trunk vessels of the extremities, and, the latter location is especially important practically, due to its widespread distribution.

Acute deep vein thrombosis (ADVT) of extremities *is an acute disorder of the permeability of these vessels with polymorphic clinical manifestations from asymptomatic flow to the venous gangrene of the limb.*

The expressiveness of clinical symptoms and signs of ADVT depends on the localization of thrombotic masses, their distribution, compensatory possibilities of the venous bed, the features of the placement of blood clots in the lumen of the vessel (non-occlusive-floating, parietal or occlusive thrombosis), time of its existence, the state of the hemostasis system and the disease that led to the development of thrombotic complications. Typically, ADVT is a complication of various diseases, among which the most common are oncological diseases, septic conditions, chronic inflammatory processes, infectious diseases, prolonged bed rest of patients or treatment with an immobilizing bandage, trauma of the lower extremities and pelvis, thrombophilic states. ADVT can also cause the diseases of venous system - varicose veins (VV), PTS – post-thrombotic syndrome or post-thrombotic disease, congenital abnormalities of veins, failure of deep venous valves. ADVT with unrevealed causes is referred to idiopathic phlebothrombosis.

The modern concept considers ADVT and pulmonary embolism (PE) as a manifestation of the same pathological process - ***venous thromboembolic disease (VTED) or venous thromboembolic complications (VTEC).*** Annually in over 25 countries in Europe, more than 680,000 cases of ADVT and over 430,000 cases of PE are registered. Pulmonary embolism (PE) is responsible for 10-12% of all deaths at hospitals, but in 70-80% of cases no clinical diagnosis of pulmonary embolism is established. In Europe, the annual rate of ADVT remains at 160 cases per 100,000 population. Annually in the National Registry of the USA, ADVT and PE are recorded at 600,000, including 275,000 new cases and the incidence of the disease reaches 50.4 per 100,000 population. In economically developed countries of Europe, 40-50% of ADVT is complicated by pulmonary embolism, which in 10% of cases within one hour after the onset of clinical signs can result in the death of the patient. Unfortunately, there are no epidemiological data concerning the prevalence of ADVT in Ukraine. However, the extrapolation of the medical statistics of other countries to Ukraine, then in our country the frequency of ADVT should be approximately 77,000, PE - 48,000 and fatal pulmonary embolism – 12,000 per year.

These findings give the evidence of the ADVT spread in many countries, including Ukraine, it conceals the danger of severe complications - PE and post-thrombotic disease. Frequency of fatal PE is within 5-20%.

One of the serious consequences of ADVT is ***post-thrombotic disease***, which is clinically manifested by ***chronic venous failure syndrome***, which occurs after undergone acute

thrombosis in 90% of patients, may cause a 20% recurrence of ADVT and lead to disability in every third patient. The presented findings present an evidence that ADVT remains both medical and social problem.

Diagnosis and treatment of ADVT, its complications and consequences are the topical and unresolved issues of modern angiology and angiosurgery. The development of this chapter of medicine has made a significant contribution by outstanding and talented national and foreign researchers. However, so far, the tactic of treating ADVT remains completely non-standardized both in our country and abroad (although attempts to resolve this problem are constantly ongoing, especially recently).

For the treatment of ADVT patients, medical, x-ray endovascular and surgical methods are used. However, despite the development and introduction into clinical practice of various medical and surgical methods, as well as modern minimally invasive X-ray endovascular technologies, scientists and practitioners have not yet come to an agreement concerning the choice and time of the treatment for this disease.

Treatment of patients with ***acute limb ischemia syndrome***, frequently caused by acute arterial thrombosis (AAT), is today one of the most urgent problems for modern vascular surgery. According to L. Norgren, W. R. Hiatt et al. (2007) the incidence of acute ischemia of the limbs is 140 cases per 1 million population globally per year.

The clinical practice of the last decades shows a continuing increase of acute thromboembolic lesions of major vessels, including arteries. Diagnosis which was done not in due time and late start of treatment largely determine the remained high mortality - from 20 to 35%, and in aged and elderly patients - up to 45%. The number of the limb amputations in patients with urgent arterial vascular disorders is of 12-28%.

Chronic occlusive diseases of the limb arteries (obliterating atherosclerosis is prevalent, obliterating endarteritis, thromboangiitis obliterans, etc.) are the main cause of AAT of limbs, that lead to progressive stenosis of the major arteries, to the growing chronic ischemia of tissues and in case of the development of critical stenosis – up to the AAT, which results in, especially with no treatment, the development of limb gangrene, which requires its amputation and leads to persistent disability of the patient.

The physicians in Ukraine choose medical tactics in accordance with the Ukrainian National Consensus "Arterial, venous thrombosis and thromboembolism. Prevention and Treatment" (2006).

Anticoagulant therapy for acute thrombosis of deep veins of the limbs

To date, in most countries, the main method of treating ADVT – acute deep vein thrombosis is medical treatment, whose main role is prolonged anticoagulant therapy (ACT) under the control of changes in the values of the coagulogram. *The main objective of the ACT is to prevent the further progression of thrombosis and the possible development of pulmonary embolism, as well as prevention of post-thrombotic disease and the recurrence of ADVT.* Failure to follow the rules of anticoagulants can lead to the spread of thrombosis and occurrence of pulmonary embolism or development of hemorrhagic complications. The main principles of the ACT are timeliness, adequate dosage, duration of treatment, laboratory monitoring and monitoring of comorbidities.

To perform ACT, ***direct and indirect anticoagulants*** are used.

Direct anticoagulants include *unfractionated heparin (UFH) and low molecular weight heparins (LMWH).*

Contraindications to the use of UFH are the following: (Mashkovskiy M.D., 2005):

- hemophilia;
- hemorrhagic diathesis;
- increased vascular permeability;
- bleeding of any localization (except for hemorrhage at embolic pulmonary infarction of the lungs or kidneys);
- subacute bacterial endocarditis;

- severe disorders of liver and kidney function;
- acute and chronic leukosis;
- aplastic and hypoplastic anemia;
- acute aneurysm of the heart;
- venous gangrene;
- allergic reaction.

Caution is required for ulcer and tumor lesions of the gastrointestinal tract, cachexia, high arterial pressure (over 180/90 mm Hg), in the immediate puerperal and postoperative period (within the first 2-3 days), except for heparin therapy necessary for vital indications.

Historically, the first *unfractionated heparin (UFH)* was used for the treatment and prevention of ADVT. It is an acid mucopolysaccharide consisting of residues of glucuronic acid and glucosamine, esterified with sulfuric acid, has a relative molecular weight of about 16,000 dalton. UFH has a number of positive effects, however, it is not lacking negative properties.

*The **positive** biological effects of UFH include:*

- anticoagulant;
- antithrombotic;
- suppression of platelet aggregation;
- stimulation of fibrinolysis;
- anti-inflammatory action;
- increase of negative electrical potential of endothelium;
- normalization of rheological properties of erythrocytes;
- stimulation of angiogenesis;
- activation of lipoprotein lipase and hepatic lipase;
- suppression of aldosterone secretion;
- the possibility of binding to macrophages and proteins of the acute phase of inflammation.

***Negative** biological effects of UFH include:*

- low bioavailability when administered subcutaneously;
- unexpected clearance rate;
- binding of the drug to plasma proteins and endothelial cells;
- unpredictable anticoagulant effect;
- development of heparin-induced thrombocytopenia of immune genesis;
- the phenomenon of withdrawal / rebound;
- achieving optimal effect in less than 25% of patients;
- development of the "tolerance" phenomenon, osteoporosis, secondary hypoaldosteronism, triglyceridemia at long-term treatment with subcutaneous injections;
- problematic use at home.

Treating DVT with UFH can be done by prolonged intravenous infusion or subcutaneous injections. The treatment should last at least 5-7 days, under the control of blood coagulation rates, or so far the *international normalized ratio* (*INR*) will be at least 2.0 within 24 hours. UFH is initially injected intravenously in the bolus dose of 80 U / kg or 5,000 U, and further as a continuous intravenous infusion (starting at 18 U / kg / h or 1300 U / hour with a dose selection that is adequate to increase the APTT - *activated partial thromboplastin time* (Table 31).

Table 31

Nomogram of administration of UFH using relative changes in APTT (in relation to the reference value of a particular laboratory), Chest, 2008

Initial dose	80 U / kg bolus, then 18 U / kg / hour
APTT<1.2 control value (<35 s)	80 U / kg bolus and to increase infusion rate by 4 U / kg / hour
APTT<1.2-1.5 control value (35-49 s)	40 U/kg bolus and to increase the infusion rate by 2 U / kg / hour
APTV<1.5-2.3 control value (50-70 s)	No changes

APTV<2.3-3.0 control value (71-90 s)	To reduce the infusion rate by 2 U/kg/hour
APTV>3.0 control value (>90 s)	To stop infusion for 1 hour, then continue it, having reduced infusion rate by 3 U / kg / hour

The APTT value 3 hours after infusion should show a 1.5-2 fold exceed of the normal control value for a particular laboratory and be consistently maintained at this therapeutic level during all time of infusion. If the APTT target level is not achieved, its correction is done according to this nomogram. The next value of APTT level is determined every 6 hours after the next dose correction.

After reaching the target values of the APTT for 2 days, the control is continued every 12 hours; then, provided that the values of the APTT ranged 46-70 seconds are preserved, it is possible to determine it once a day, every day in the morning.

Subcutaneous administration of NFP, depending on nosology and clinical status, has several regimens. The most common is introduction of 5000 U every 4 hours; also subcutaneous injections 5000, 7500, 10000 U every 6-8 hours, 10000-12500 U every 12 hours are administered (Ukrainian Consensus, 2006). Abroad, it is recommended to introduce first 17500 U or 250 U / kg twice a day and further select the dose adequate to the APTT increase.

Table 32

Nomogram of UFH introduction in subcutaneous injections with APTT relative changes (in relation to the reference value of a particular laboratory)

APTT <40 s	To increase the next NFP dose by 50 U/kg
40≤ APTT <49 s	To increase the next NFP dose by 25 U/kg
50≤ APTT <75 s	No changes
76≤ APTT <100 s	To reduce NFP dosage by 25 U/kg
APTT <100 s	To stop at 50 U/kg. If hemorrhage begins, it is necessary to administer IV 5.0-10.0 ml 5% solution of protamine sulphate

The ATPP level should be checked every 12 hours, approximately 1-2 hours before the next introduction. Provided that the APTT values of 50-75 have been achieved, it is possible to determine this level once a day in the morning.

If it is decided to use uncontrolled laboratory subcutaneous injections of UFH for the treatment of the patient, then it is recommended first to introduce 333 IU / kg, and then 250 IU / kg 2 times a day. UFH administration is not recommended regardless the patient's body weight.

During the first 3 days of treatment with UFH, *the therapy with* ***indirect anticoagulants*** should be initiated. NFP may be discontinued if the International Normalized Ratio (INR) reaches the therapeutic level (2.0-3.0 for two consecutive days), but not earlier than 4-5 days after the indirect anticoagulants are prescribed.

When treating NFP, *side effects and complications* may occur:

- bleeding
- thrombocytopenia (when high doses used);
- skin necrosis at the injection site;
- hyperkalemia;
- hypertriglyceridemia;
- osteoporosis in case of prolonged treatment.

The control of platelet count at the UFH treatment should be performed once a week (at a high risk of thrombocytopenia - once per day). In the case of a sudden sharp decrease in platelet count by more than 50% or if their level does not exceed 100×10^9 / l, the introduction of UFH should be stopped.

In the case of impossibility to provide regularly monitoring of the level of APTT and the platelet count in the course of treatment, low molecular weight heparins (LMWH) should be used.

In cases of overdose or in case of bleeding, ***protamine sulfate or protamine chloride*** should be used as a ***heparin antidote***, which is to be administered intravenously or intramuscular calculated as 0.1 ml of 1% solution per 100 IU of UFH.

In ADVT patients it is preferable not to use UFH for initial treatment, but to provide subcutaneous administration of ***low molecular weight heparins*** (LMWH) 1 or 2 times a day, in both in-patient and out-patient settings. LMWH are fragments of UFH and consist of a sum of glycosaminoglycans with a molecular weight of 1800 to 8000 dalton. Components with a molecular weight of 1800-5000 dalton inhibit, suppress the Xa factor, and components with a molecular weight of 5000-8000 dalton: IIa factor (thrombin) and antithrombin. The ratio of anticoagulant activity, anti-Xa / antiIIIa, is 1: 1.5 to 1: 4 for LMWH. The pronounced Xa inhibition and a slight factor IIa inhibition in LMWH allows the long term antithrombotic effect of these drugs to be obtained without increasing the risk of developing bleeding.

LMWH have several advantages compared to the UFH:

- high bioavailability (90-98%);
- longer half-life (2-4 hours);
- administration once per day calculating the dose to body weight;
- no need for constant laboratory control;
- low incidence of osteoporosis (prolonged use of UFH can lead to asymptomatic vertebral fractures in 2.2% of patients);
- low frequency of development of heparin-induced thrombocytopenia (0.1% vs. 2-3% with UFH);
- insignificant elimination with breast milk;
- extremely low penetration through the placenta.

Administration of the LMWH does not require control of the APTT or determination of anti-factor Xa activity. During the first week of treatment, it is enough to determine the platelet activity 2 times.

Contraindications for the use of LMWH (Mashkovskiy M.D., 2005):

- acute bleeding
- hemorrhagic diathesis;
- fresh ulcers of the gastrointestinal tract, peptic ulcer in the stage of exacerbation;
- thrombocytopenia associated with the introduction of LMWH in the history;
- condition after recent operations;
- pronounced arterial hypertension;
- septic endocarditis;
- acute disorder of cerebral circulation;
- tuberculosis of the lungs (cavernous forms);
- the first trimester of pregnancy.

Patients with GFR and severe renal insufficiency should be preferred to non-LMG, and UFH.

Table 33

Different patterns of the LMWH administration taking into account the characteristics of the drug

Name of NMH	Average molecular weight	Bioavailability,%	Dose
Nadroparin (Sodium fraxiparine)	4300	99	86 IU / kg IV / bolus, then 86 IU / kg every 12 hours or 190 IU / kg (forte) 1 time daily subcutaneous
Dalteparin (fragmin)	6000	75	120 IU/kg subcutaneous every 12 hours or 200 IU/kg once daily
Enoxaparin (clexane)	4500	90	1 mg/kg subcutaneous every 12 hours

The following *side effects* are possible when using LMWH:
- bleeding of different localization;
- allergic reactions;
- thrombocytopenia (rarely)
- rarely - skin necrosis at the injection site.

In the event of side effects it is necessary to stop administration of LMWH temporarily. In cases of overdose or bleeding to neutralize the drug, it is necessary to administer *the protamine sulfate* in an amount that is 30% greater than it is needed for neutralizing UFH.

Patients treated with heparin administration require *transition to the administration of* ***indirect anticoagulants***. These are prescribed 4-5 days before the supposed cessation of heparins. The action of indirect anticoagulants, which are vitamin K antagonists, is aimed at reducing the synthesis of prothrombin, proconvertine (factor VII) and other factors of blood coagulation (IX, X).

Contraindications for the use of indirect anticoagulants:
- hemorrhagic diathesis;
- lowered ability of blood clotting;
- increased vascular permeability;
- pregnancy;
- breastfeeding period;
- hemorrhagic stroke;
- arterial hypertension, resistant to hypotensive therapy;
- varicose veins of the esophagus;
- peptic ulcer of the stomach and duodenum, for which the patient received no treatment;
- liver failure;
- renal failure (creatinine level over 140 μmol / l);
- mental disorders;
- alcoholism.

Indirect anticoagulants must not be prescribed in case of:
- lack of sufficient contact of the doctor with the patient;
- the impossibility of regular reliable laboratory control of treatment;
- absence of antidotes and impossibility of provision of timely medical aid in the development of bleeding.

The following indications for the use of indirect anticoagulants are recommended (Table 34).

Table 34

Indications for the use of indirect anticoagulants

Indications	**INR value**
Prevention of venous thromboembolism. Treatment of venous thromboembolism. Treatment of PE. Prevention of systemic thromboembolism: - at fibrillation arrhythmia; - at heart defects;	2.5 (2.0-3.0)
- at prostheses of the valves of the heart	2.5-3.0 (2.5-3.5)
-at recurrent thrombophlebitis; -at the risk of idiopathic thrombosis in family disposed to thrombosis. Secondary prophylaxis of myocardial infarction if the indications for the use of acetylsalicylic acid are present. Critical ischemia of the lower extremities. Prevention of rethrombosis after reconstructive	2.5 (2.0-3.0)

operations on major vessels.	
Prevention of thrombosis at antiphospholipid syndrome, congenital or acquired deficiency of coagulation factors.	3.0 (2.5-3.5)
Prevention of thrombosis against the background of chemotherapy for the stage 4 breast cancer.	1.6 (1.3-1.9)

Today, ***warfarin*** is the *drug of choice* for most multicenter studies (***warfarin*** is one of monocoumarin derivatives, which provides a stable effect on the blood coagulation process and has the lowest risk of side effects compared with other indirect anticoagulants).

Duration of treatment is prescribed individually and it is at least 3 months (except for cases of short-term effects of risk of thrombosis, such as hormonal therapy or short-term immobilization) (Table 35).

Warfarin therapy should be started at a dose of 5 mg per day. Lower doses are used in patients over 60 years of age, in patients with impaired liver and kidney function, severe arterial hypertension, severe congestive heart failure, and in case of concurrent medication that increases anticoagulant effect.

A patient who receives indirect anticoagulants should keep a diary in which the INR values, the dose of the drug, concomitant therapy, changes in well-being are registered. The patient should report to the doctor any changes in well-being that occur during the administration of indirect anticoagulants.

Table 35

Duration of therapy of thrombosis by indirect anticoagulants

The category of the patients	Duration of the therapy
- The first episode of thrombosis in the case of a short-term risk factor (surgical intervention, trauma, immobilization, hormonal therapy). - The first episode of thrombosis in the case of a heterozygous activated protein C resistance.	3-6 months (the age and present comorbidity should be also considered).
- The first episode of thrombosis of unknown etiology	6-12 months (the same)
- Rethrombosis of any origin. - The first episode of thrombosis in patients with oncopathology, antithrombin, protein C and S deficiency, anticardiolipin antibodies, homozygous activated protein C resistance.	No less than 12 months The maximum term is not limited, in fact, up to the development of contraindications for the use of coumarins.

Regular laboratory control during the treatment week 1 should be performed daily, while the first two months of treatment it should be done weekly, further: if INR is stabilized it should be povided monthly. The hypocoagulation effect of coumarins decreases with a significant intake of vitamin K, which is present in large quantities in vegetables and fruits. In this regard, it is advisable to limit the use of foods with vitamin K high content. Also, chronic alcoholism, the use of antacids, antihistamines, barbiturates, oral contraceptives, cyclosporins reduce the effectiveness of indirect anticoagulants. Inadequate vitamin K intake due to disordered intestinal absorption, increased catabolism of coagulation factors II, VII, IX, X, C and S proteins, acetylsalicylic acid, omeprazole, ranitidine, simvastatin, anabolic steroids, amiodarone, sulfanilamides, disaggregants, thrombolytics increase anticoagulant activity of indirect anticoagulants .

Indirect anticoagulants are prescribed considering the risk of developing bleeding. ***The risk factors for bleeding*** include: age over 75 years, gastric bleeding in history, arterial hypertension, cerebral vascular disorders (aneurysms), renal and hepatic failure, malignant tumors, alcoholism, concomitant administration of other antithrombotic agents.

*In the event of **excessive hypocoagulation**, the following measures should be taken.*
I. In case of an INR high rate without bleeding:

a) if the INR value is more than 5.0 but less than 9.0:

- skip 1-2 doses of the drug, control the INR, resume treatment at INR therapeutic values , or skip 1 treatment, vicasol 15 mg per os;

- if urgent correction is required - vicasol 30 mg per os, if the INR retains high values during the day - additionally vicasol 15 mg per os;

b) if the INR exceeds 9.0:

- skip 1 intake of the drug, vicasol 30 mg per os;
- if the INR maintains high values within 24-48 hours - additional vicasol 15-30 mg per os;
- resume treatment at therapeutic values of INR.

II. Small bleeding (hematuria, nosebleed):

- to stop taking the drug for 1-2 days;
- vicasol 1-2 ml of 1% solution IM or 15-30 mg per os.

III. Dangerous for life bleeding (intracranial or gastrointestinal):

- stop taking the drug;
- vicasol 1-2 ml 1% solution IM, if necessary, repeat;
- IV administration of factors II, IX, X concentrate or fresh frozen plasma (15 ml / kg).

Thrombolytic therapy in surgical diseases of the vessels of the extremities

The most important task of treating acute DVT is to restore their permeability in order to reduce the degree of venous failure and reduce the severity of post-thrombotic syndrome (PTS) and reduce the disability of patients in the long-term period. However, thrombolytic therapy for patients with lower extremity DVT is considered ambiguous, especially when it regards the duration of thrombotic occlusion, its localization, extent, choice of thrombolytic drug and the way of its administration.

Thrombolytic drugs have a high lytic effect, either directly affecting the thrombus, or activating the own fibrinolytic system of the patient, as activators of inactive plasminogen proactivators (fibrinolysin) and blocking the effect of fibrinolysis inhibitors.

The thrombolytic therapy was begun with the national medication ***Fibrinolysin*** which was administered intravenously in a daily dose of 20,000-100,000 units in physiological saline (sodium chloride) with obligatory addition of heparin (10,000 units for each 20,000 units of fibrinolysin) for 3-4 hours during 2-3 days. It was quickly given up due to low effectiveness (it weakly activated fibrinolysis, did not cause lysis of the thrombus, at the same time led to hypercoagulation and increase platelet aggregation, and was quickly inactivated in the body by antiplasmin).

Streptokinase (Germany) (a highly purified protein produced by an actively growing group C hemolytic streptococcus) was found to be a significantly more effective thrombolytic drug which has properties of the activator of the plasminogen proactivator and causes lysis of the thrombus, that is an ***"indirect" thrombolytic agent***. Other drugs in this group are the following: ***Kabikinase*** (Sweden), ***Streptodecasum, Streptolyase***.

The increase in the effectiveness of thrombolysis is associated with the creation in the 90-ies of the twentieth century of a new *fibrin-specific thrombolytic drug - a synthetic tissue activator plasminogen actilyse* (active ingredient - *alteplase),* then ***metalyse, reteplase, tenecteplase*** (a group of ***"direct" thrombolytics***).

Contraindications to thrombolytic therapy are standardized and explicated in the Ukrainian National Consensus "Arterial, venous thrombosis and thromboembolism. Prevention and treatment "(2006).

Absolute contraindications for thrombolytic therapy:

- hemorrhagic stroke in history;
- ischemic stroke for up to 6 months;
- severe trauma, craniocerebral trauma, surgical intervention for up to 3 weeks;
- tumor of the central nervous system;
- gastrointestinal bleeding during the last month;
- documented disorders of coagulation;
- aneurysm of the aorta;

- intolerance to thrombolytics.

Relative contraindications to thrombolytic therapy:

- transient ischemic attack occurred within 6 months;
- taking oral anticoagulants (at INR more than 1.4);
- pregnancy or first week after childbirth;
- puncture of the major artery, which is not available for compression;
- traumatic cardiopulmonary resuscitation;
- refractory arterial hypertension (systolic pressure above 180 mm Hg);
- progressing liver disease;
- infective endocarditis;
- exacerbation of peptic ulcer disease;
- bleeding hemorrhoids.

Thrombolytic therapy is indicated at fresh massive iliofemoral venous thrombosis, and at caval thrombosis, as well, when there is a high risk of developing pulmonary embolism and venous gangrene. The duration of thrombosis in this case should not exceed 5-7 days. Possible intravenous thrombolytics for systemic thrombolysis, as well as its delivery to the site of thrombosis by an infusion catheter.

Systemic and local thrombolysis are distinguished depending on the location of the thrombolytic agent injection. The local thrombolysis is classified ***into regional and catheter-controlled thrombolysis***.

To provide regional thrombolytic therapy, in case of thrombosis of the popliteal and superficial femoral veins, local anesthesia is used, and access is made through the posterior tibial vein, in which an infusion catheter is inserted and controlled by distal ascending phlebography. Gradually promoting the infusion catheter, an initial dose of 250,000 IU of streptokinase (farmakinase) in a 10 ml isotonic sodium chloride solution is introduced in the middle of the thrombotic mass, trying to pierce the thrombus through. Then the catheter tip is set 3-5 cm below the proximal edge of the thrombus. After that, the catheter is fixed to the skin and streptokinase (farmakinase) is continued to drip at a dose of 100,000 IU / h. The drug urokinase, after the initial dose, is administered in a dose of 240000 U / hour, the medication alteplase (actilyse) - 1 mg / hour.

The position of the tip of the catheter is corrected considering the lysis of thrombotic masses. If the catheterization of the deep venous system is not available, regional thrombolytic therapy is carried out through the punctured and catheterized subcutaneous vein of the foot. In addition, three tourniquets at the lower and upper third of the shin, as well as at the lower third of the thigh are applied to direct the flow of thrombolytic agent into the deep venous system. In this case, the compression lasts on average 30-40 minutes and depends on the subjective sensations of the patient.

For ***catheter-controlled thrombolysis***, the percutaneous puncture of the popliteal vein with the antegrade introduction of the infusion catheter under ultrasound control is optimum access. This access can be used also for *mechanical thrombectomy*. If the popliteal vein is thrombosed, the infusion catheter can be inserted antegrade, through the posterior tibial vein, which increases the chance of restoring blood circulation in the popliteal vein. In order to perform catheter-controlled thrombolysis, retrograde access is also proposed, in which through the subclavian or jugular vein the infusion catheter is inserted into the right atrium, then into the vena cava inferior and then directly into the thrombus, which occludes iliofemoral or femoral popliteal venous segment.

Infusion of the thrombolytic drug is carried out using an automatic injector which can control the infusion rate of thrombolytic agent. In the case when ***streptokinase (farmacokinase)*** is prescribed, this thrombolytic agent in a dose of 250,000 IU, initially as bolus, is injected directly into the thrombotic mass. After that, infusion is performed at a rate of 100,000 IU / h. If ***urokinase*** is prescribed, after introduction of an initial dose of 4400 U / kg, it is administered at a rate of 120,000 - 240,000 U / hour. If ***actilyse (alteplase)*** is prescribed, then it at a dose of 5 mg

is initially administered as a bolus to the thrombotic mass, and then infused at a rate of 1 mg / hour. As dissolution of the thrombus occurs, control phlebography is performed at least once every 12 hours and the catheter is promoted to the middle of the detected thrombus.

During catheter-controlled thrombolysis, the level of fibrinogen should be monitored, which, depending on the risk of hemorrhagic complications, should not be lower than 2.0-1.5 g / l. After the critical level of fibrinogen has been reached, thrombolysis should be stopped for a period of the normal value of this parameter is restored. *When conducting thrombolysis, the coagulogram should be performed every 6 hours, and at the slightest suspicion of hemorrhagic complications, as well.*

Administration of thrombolytic agent at catheter-controlled thrombolysis lasts on average 24-48 hours. Longer-term administration of a thrombolytic drug conceals the risk of hemorrhagic complications. To prevent pulmonary embolism in patients with floating thrombus, in the process of preparation for catheter-controlled thrombolysis, caval filters, predominantly temporary, can be used. Upon completion of the thrombolysis process, the venous bed should be evaluated, using angiography and ultrasonography for residual stenosis that can provoke an ADVT relapse. Stenotic changes are usually found in the left common iliac vein, which is under compression from the right side of the common iliac artery, known as the May-Thurner syndrome. In order to prevent the ADVT relapse, it is advisable to perform correction of this congenital disorder with cardiovascular balloon angioplasty and stenting of the right common iliac vein.

In some patients with a disseminated proximal acute DVT (less than 14 days), in somatically healthy patients with a life expectancy of more than 1 year, at a low risk of developing bleeding, in the absence of possibility to provide catheter-controlled thrombolysis, *systemic thrombolysis* is recommended to reduce the severity of the symptoms and the likelihood of PTS development.

Some patients with disseminated ADVT (e.g., iliac and femoral segment less than 14 days, in somatically healthy patients with life expectancy of more than 1 year), at a low risk of developing bleeding, in case if appropriate technical facilities and experience are available, the *catheter-controlled thrombolysis* is recommended to relieve the symptoms and the probability of developing PTS. After successful catheter-controlled thrombolysis in ADVT patients, balloon angioplasty or stenting is recommended to eliminate the factor that has become the cause of the DVT development.

Foreign experts, if technical facilities and experience are available, recommend not an isolated catheter-controlled thrombolysis, but ***pharmaco-mechanical thrombolysis***, which includes fragmentation and / or aspiration of the thrombus, to reduce the duration of the intervention.

The duration and intensity of anticoagulant therapy after successful catheter-controlled thrombolysis in patients with acute DVT should not be different from the recommended standard.

To carry out ***systemic thrombolysis***, the peripheral vein of the upper limb is used, which is inserted with the catheter of corresponding size and continuous infusion of the thrombolytic drug in high doses is administered. The following thrombolytic drugs are used: ***indirect thrombolytics*** - kabikinase, streptokinase, etc., a ***direct natural thrombolytic agent urokinase*** and ***direct thrombolytics*** (*synthetic tissue plasminogen activators)* - *actilyse* (active ingredient - *alteplase), metalyse (tenecteplase), reteplase.*

Due to the anti-inflammatory antibodies which are present in the blood of practically all people, thrombolytic therapy with *indirect thrombolytics*, such as ***streptokinase, kabikinase*** etc., is started with intravenous administration of an initial "inactivating" dose of 250,000 U per 300 ml of saline or 5% glucose solution for 30-40 minutes, followed by systemic thrombolysis - continuous intravenous infusion of the therapeutic dose of the drug, which may reach 1 500 000 - 3 000,000 U of the drug (infusion rate is of 750,000 U in 8 hours), conducted under angiographic control, followed by conversion to heparin therapy. *Regional thrombolysis* by intraarterial drip of

thrombolytic drug in the small arterial branch closest to the occlusion site, with special apparatus for intraarterial prolonged infusion is more effective.

Farmakinase, made by the national OJSC "Farmak", is prescribed to adults with occlusions of peripheral venous and arterial vessels. For short-term thrombolysis, farmakinase in an initial dose of 25,000 IU, dissolved in 100-300 ml of 0.9% sodium chloride solution (or 5% glucose solution) for 30 minutes is injected, followed by a maintaining dose of 1200,000 IU in 500 ml of solvent every hour for 6 hours is injected. Six-hour infusion of the drug can be repeated the next day, depending on the therapeutic effect.

Repeated introduction of thrombolytics under no circumstances should be carried out later than 5 days after the first course.

If no positive clinical effect is observed within 3 days, thrombolytic therapy should be discontinued.

The long-term lysis is an alternative to short-term lysis is in the treatment of peripheral vascular occlusion. The initial dose of the drug farmakinase of 250,000 IU is administered within 30 minutes, followed by a maintenance dose of 10,000 IU every hour. The duration of therapy depends on the distribution and localization of the occlusion of the vessel. In case of occlusion of peripheral vessels, the maximum course of treatment lasts 5 days.

Natural endogenic physiological activator of plasminogen - ***urokinase***, which is obtained from human urine, and is favorably different from the previous agents because of no toxic and antigenic properties, but considering the difficulties of preparation and high cost, has limited application. If urokinase is used, the drug is dissolved in a physiological solution of sodium chloride or in a 5% solution of glucose. Within 20 min the dose of 250,000 U is administered intravenously, then a continuous infusion of 750000 U or 4400 U /kg / hour for 12 hours is given. Daily dose of the drug is from 250,000 to 2700000 units. Administration of urokinase is combined with continuous intravenous heparin infusion at low doses (1000 U/kg/h).

Since the 90s of the twentieth century, the medication ***actilyse (alteplase,*** *that is a synthetic tissue activator of plasminogen)* has become the ***gold standard for thrombolytic therapy***. Its disadvantage is a short half-life (4-5 minutes), which requires constant drip infusion of the drug for 90 minutes. Dosing schedule for actilyse: intravenously 10 mg for 2 minutes, 50 mg for the next 60 minutes, then another 40 mg for 2 hours (100 mg for 3 hours). Intravenous administration of 100 mg of the drug for 2 hours is possible. Thrombolytic therapy in patients with severe PE with actilyse has some advantages compared to streptokinase and urokinase as it gives a faster clinical effect.

Metalyse (metalyse - tenecteplase) is a modern third-generation thrombolytic agent that provides *bolus thrombolysis*. Compared to alteplasa, its half-life is 20 minutes more. It is administered within 5-10 seconds in the form of a single intravenous bolus. Metalyse is prescribed in an individual dose (depending on the weight of the patient), which makes it the safest drug. It is used at the prehospital stage, which significantly improves the prognosis. It should be considered that in order to prevent rethrombosis against the background of metalyse bolus injection, it is necessary to administer aspirin, heparin, as the injury of the revascularized artery intima persists for a long time.

During thrombolysis, the following ***complications*** may occur:

1) pyrogenic and allergic reactions (urokinase, actilyse and metalyse have virtually no antigenic properties);

2) hemorrhagic complications in 45-50% of observations;

3) there is a high probability of recurrent pulmonary embolism in the treatment with plasminogen activators, due to the occurring fragmentation, and other venous thrombi in thromboembolic disease.

To avoid the various ***bleedings*** as a main complication of thrombolytic therapy, careful laboratory control of the state of the hemocoagulation system is required: ***every 8 h*** coagulogram parameters: *blood coagulation time, fibrinogen A concentration, thrombin time, and fibrinolytic activity of the blood* must be studied.

The rate of infusion of the thrombolytic drug should be such as to maintain thrombin time 2-5 times longer than the control value.

To reduce the toxic and antigenic effects of thrombolytics, hormonal (prednisolone, etc.) and desensitizing (suprastin, tavegil, pylophen, etc.) agents are used.

The effect of thrombolysis is evaluated by the regression of clinical manifestations, electrocardiographic disorders, *control angiopulmonography*.

After the end of thrombolytic therapy, heparin or low molecular weight heparins are prescribed according to the schedule.

In case of ***hemorrhagic complications*** (bleeding of wounds, micro- and macrohematuria, coagulogram values) introduction of thrombolytic is stopped temporarily; as an ***antidote therapy***, administration of *fibrinogen, 5% solution of epsilon-aminocaproic acid, calcium chloride 1% solution, native or frozen plasma, 1% Ambene solution* are *used*. After the manifestation of hemorrhage is ceases, thrombolytic therapy should be continued until the therapeutic effect, but with slower administration of the drug.

Currently, ***new standards of thrombolytic and anticoagulant therapy*** are proposed, which allow to improve the immediate and long-term results of "systemic" thrombolysis by improving the so-called adjuvant anticoagulant and antiplatelet therapy, ***co-administered*** with thrombolytics.

A prerequisite for the use of anticoagulants in thrombolytic therapy (the so-called anticoagulant "thrombolysis") is the release of thrombin from a thrombus during fibrinolysis, which leads to an increased pre-thrombotic state and elevated risk of rethrombosis on the surface of an unstable plaque.

In order to increase the clinical efficacy of thrombolytic therapy, the advantage of using low molecular weight heparins (LMWH), mainly enoxaparin (clexan), as compared to UFH - unfractionated heparin, was demonstrated, not only with fibrin-specific thrombolytics, but also with streptokinase.

The prolonged (up to 7-8 days) anticoagulant therapy with enoxaparin or direct anticoagulant of the new class of *arixtra (* a synthetically selective factor Xa inhibitor *fondaparinux*) has been proved to be rational.

Prevention of venous thromboembolic complications (VTEC) in general surgery

To date, the following ***risk factors for VTEC*** have been identified:

- episodes of ADVT and PE in history;
- age over 60;
- surgical intervention;
- injury;
- immobilization for more than 3 days;
- overweight (obesity);
- taking estrogen, combined oral contraceptives;
- thrombocytosis;
- polycythemia;
- anesthesia with muscle relaxants;
- pregnancy and childbirth;
- dehydration;
- heparin-induced thrombocytopenia;
- malignant tumors;
- cytostatic therapy;
- acute and chronic lung diseases;
- acute and chronic infectious diseases;
- stroke;
- myocardial infarction;
- decompensated heart failure;
- hemophilia

According to *the risk level of VTEC*, patients should be divided into 3 groups. According to *the risk level of VTEC*, patients should be divided into 3 groups. ***The low-risk group*** consists of patients with no VTEC risk factors, which are to undergo a small volume surgical intervention.

A moderate risk group includes the patients over the age of 60 who are to undergo a high-volume surgical intervention for benign disease.

A high-risk group includes the patients over the age of 60 with additional risk factors.

1. General provisions for the prevention of VTEC.

It is not recommended to use *aspirin* as a mono-prophylaxis of VTEC.

When deciding *to prescribe and choose the dose of LMWH* or other antithrombotic agents that are excreted in the urine, *kidney function should be considered*, especially in elderly patients, patients with diabetes mellitus or those with a high risk of bleeding. Depending on the circumstances, it is recommended to avoid prescribing anticoagulants that accumulate in the body in case of renal failure, to reduce the dose of the drug or to control the content or degree of the effect on the coagulation system. In those patients with severe renal failure (creatinine level greater than 2 mg / l), in most cases UFH (unfractionated heparin) should be the medication of choice for anticoagulant therapy.

Mechanical methods of VTEC prophylaxis are recommended prior to patients with a high risk of bleeding or as an additional method in the VTEC prevention schemes on the basis of anticoagulants, along with special attention to the correct disposal and observance of recommendations for their use.

2. ***Prevention of VTEC in patients scheduled for operative treatment.***

No specific prevention of VTEC in patients with *low risk*, except rapid activation and adequate hydration, is indicated.

Patients with *moderate risk* are indicated the VTEC prophylaxis with LMWH (according to the scheme recommended by the manufacturer), low-dose UFH (5000 units before surgery and 2 or 3 times daily in the postoperative period), fondaparinux or intermittent pneumatic compression (IPC) in combination with Graduated Elastic Compression (GEC).

Patients with high-risk are indicated the prophylaxis of VTEC with LMWH (according to the scheme recommended by the manufacturer), UFH in low doses (5000 units before surgery and 3 times a day in the postoperative period) or fondaparinux.

Patients with *multiple risk factors for thromboembolic complications*, which form a group of especially high risk of VTEC, are indicated combined prophylaxis with medication (low dose of LMWH, UFH 3 times daily, or fondaparinux) and mechanical (HES - hydroxyethyl starch and / or intermittent pneumatic compression - IPC) methods according to the optimal scheme.

For patients *at high risk of bleeding*, mechanical prophylaxis of VTEC is adequately selected by HES or IPC. After reducing the risk of bleeding, mechanical methods of prophylaxis of VTEC are recommended to be replaced or supplemented with medication prophylaxis.

For patients who underwent major surgical intervention, the prevention of VTEC should be conducted throughout the term of the hospitalization. In some patients with very high risk, in particular after major surgical interventions for oncological diseases or with history of venous thromboembolic episodes, the prophylaxis of VTEC with LMWH should be continued until 28 days after discharge from the hospital.

3. ***Prophylaxis of VTEC in patients scheduled for laparoscopic surgical intervention.***

The patients with no additional risk factors for VTEC, routine prophylaxis, other than rapid activation, is not represented.

The patients with additional VTEC risk factors are recommended prevention by at least one method (LMWH, low-dose UFH, fondaparinux, IPC or HES).

Antithrombotic therapy in acute occlusive diseases of trunk arteries of extremities

In case of acute arterial thrombosis of the extremities, as well as in *acute thrombosis of vascular prostheses and shunts* in the remote postoperative period during the first 12 hours

from the moment of complication, a thrombolytic therapy should be prescribed, if no contraindications for its use are revealed, similar to the treatment pattern for acute myocardial infarction.

As a thrombolytic, intravenously, streptokinase is injected at a dose of 1500,000 U in 100 ml of 5% glucose solution or 0.9% sodium chloride solution for 60 minutes. When prescribing a ***farmakinase,*** the drug is administered in a dose of 1000 to 2000 IU at intervals of 3 to 5 minutes. The duration of the administration depends on the dissemination of lesions and localization of acute occlusion of the peripheral artery, and is performed up to 3 hours at a total dose of a maximum of 120,000 IU of farmakinase. If necessary, transcutaneous transluminal angioplasty may be performed simultaneously.

If ***actilyse (alteplasa)*** is prescribed, the drug is administered intravenously at a dose of 15 mg bolus for 1-2 minutes, followed by intravenous infusion 0.75 mg / kg (but not more than 50 mg) for 30 minutes or 0.5 mg / kg (not more than 35 mg) for 60 minutes. As an additional treatment, immediately after the infusion of alteplasa (actilyse), the accompanying therapy with UFH is indicated from the rate of 60 U / kg (but not more than 4000 U), then infusion at a dose of 12 U / kg for 1 hour (not more than 1000 U / hour) during not less than 48 hours. Target prolongation of APTT - activated partial thromboplastin time - up to 50-70 seconds.

As an alternative, administration of ***clexan*** *(enoxaparin)* in a dose of 0.3 mg intravenously is possible, then 1 mg / kg subcutaneously every 12 hours. After 48 hours of intravenous infusion, UFH should be changed to its subcutaneous administration at a dose of 100 U / kg 2 times a day or at LMWH at doses corresponding to body weight for 5-7 days.

In case of a high risk of thromboembolic complications, certain categories of patients are *simultaneously prescribed* ***indirect anticoagulants***, the period of their joint use with UFH should continue until the target EER and reach at least 4 days. The dose of indirect anticoagulant is selected in order to achieve prolongation of prothrombin time to the INR of 2.0-3.0, their administration should last until the patient's discharge from the hospital.

After reaching the positive effect of thrombolytic therapy, it is recommended to use ***LMWH*** 2 times daily for 5 days *followed by long-term* ***clopidogrel*** (75 mg per day).

In the case of ineffectiveness of thrombolytic therapy, surgical intervention is indicated - thrombectomy followed by the administration of LMWH and clopidogrel as in the above pattern.

In case of acute peripheral artery *embolism*, thrombolytic therapy should not be prescribed for more than 14 days after symptoms occurred. After thrombolysis or surgical embolectomy, heparin therapy should be prescribed, followed with indirect anticoagulants (INR 2.0-3.0) for up to 3 months.

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