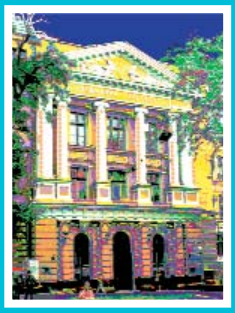

Бібліотека студента-медика
Medical Student's Library

FACULTY SURGERY

ОДЕСЬКИЙ МЕДУНІВЕРСИТЕТ

ОДЕСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ

THE ODESSA STATE MEDICAL UNIVERSITY

Бібліотека студента-медика

Medical Student's Library

Започатковано 1999 р. на честь 100-річчя Одеського державного медичного університету (1900–2000 рр.)

Initiated in 1999 to mark the Centenary of the Odessa State Medical University (1900–2000)

FACULTY SURGERY

A course of lectures

Edited by Honoured Doctor of Ukraine,
prof. B. S. Zaporozhchenko

Recommended
by the Central Methodical Committee
for Higher Medical Education of the
Ministry of Health of Ukraine as a manual
for students of higher medical educational establishments
of the IV level of accreditation

Odessa
The Odessa State Medical University
2009

ВВС 54.5я73
UDC 617(075.8)

Authors: B. S. Zaporozhchenko, V. V. Mishchenko, A. L. Dekhtyar, O. B. Zubkov, Yu. V. Grubnik, A. V. Snisarenko

Reviewers: prof. M. P. Zakharash, a fellow of the Academy of Medical Sciences of Ukraine, the head of the Department of Faculty Surgery N 1 of the O. O. Bogomolets National Medical University, MD

prof. V. I. Lupaltsov, a fellow of the Academy of Medical Sciences of Ukraine, the head of the Department of Faculty Surgery of the Kharkov State Medical University, MD

The manual contains a course of lectures on faculty surgery. They are elaborated within the scope of the curriculum on faculty surgery of the higher medical establishments.

Intended for lecturers and students.

Переклад з української за виданням: Б. С. Запорожченко, В. В. Міщенко, А. Л. Дехтяр [та ін.] «Факультетська хірургія». Одеса, Одеський державний медичний університет, 2005.

Рекомендовано Центральним методичним кабінетом з вищої медичної освіти МОЗ України як навчальний посібник для студентів вищих медичних навчальних закладів IV рівня акредитації, (Протокол № 1 від 10.02.2004 р.)

Факультетська хірургія : курс лекцій : навч. посібник / Б. С. Запорожченко, В. В. Міщенко, А. Л. Дехтяр [та ін.] ; за ред. Б. С. Запорожченка. — Одеса : Одес. держ. мед. ун-т, 2009. — 328 с. — (Б-ка студента-медика). — Англ. мовою.

ISBN 978-966-443-022-4

Навчальний посібник містить курс лекцій з факультетської хірургії. Вони відповідають навчальній програмі для студентів IV курсу медичного факультету вищих медичних навчальних закладів.

Для студентів і викладачів.

Табл. 1. Бібліогр.: 53 назви.

ББК 54.5я73

ISBN 978-966-7733-47-6 (серія)
ISBN 978-966-443-022-4
ISBN 966-7733-72-6

FOREWORD

Poor quantity and quality of modern textbooks, manuals published in the state language which would correspond to the curricula of surgery there is an important problem in the educational process of future surgeons traiing in Ukraine. The published textbooks and manuals contain classical data on various surgical diseases without the differential approach to study of surgical discipline; the sequence principle is therefore broken. Students of higher medical schools use the manuals written by skilled, world famous experts in surgery of our country and of the near abroad, but they do not fully correspond to the curricula on surgical disciplines, therefore cannot be considered sufficient for mastering surgery. Students have to use other sources, but due to shortage of time and scope of medical disciplines which are studied, it creates additional impediments in study. Besides, the curricula and typical programs on faculty surgery have no information on modern achievements of medicine and wide experience of our best experts in this branch.

All aforesaid has pushed us to writing and publishing of the course of lectures on faculty surgery as a manual on studying the bases of the surgical science. We should mention that many manuals on faculty surgery published in Ukraine, but there is still no lecture course.

The manual corresponds to the curriculum on faculty surgery. According to themes of the lecture course the material is given taking into account pecularities of this surgical discipline. A group of the authors have worked hard so that the course of lectures included the latest achievements of the surgical science.

We hope that the course of lectures on faculty surgery will promote further development of the national medical science and training of medical skilled personel.

Honoured Doctor of Ukraine,
MD, Professor B. S. Zaporozhchenko

INTRODUCTION

It is generally known that surgery is considered to be one of the leading branches of medicine. The study of it needs systemic approach and mainly depends on quality of general preparation of a student.

Knowledge from the gross anatomy, topographical anatomy, normal and pathological physiology, microscopic anatomy and other applied disciplines are like a bridge between theoretical knowledge and study of clinical disciplines, direct study of different diseases, including surgical. In the course of general surgery students get acquainted with the methods of anaesthesia, principles of prevention of the surgical infections, types of bleeding and methods of their control, general information is given about such groups of surgical diseases as injuries, purulent diseases, bone tuberculosis, tumours and others. Besides, in propedeutic clinics the students acquire skills of patients's examination. Thus, knowledge obtained in the previous courses allow to study specific surgery, most important and frequent diseases.

Clinic as a Basis of Faculty Surgery Teaching

During VII–VIII terms the students study faculty surgery, that is a base of specific surgery. During the course, the students are taught directly at the patient's bedside, to collect correctly anamnesis of disease, to reveal the basic symptoms of the disease, to make a differential diagnosis and case report.

Independent work of the students of higher medical educational establishments in the course of surgical diseases needs special pose of audience preparation to every practical class.

The students make independent work at the patient's bedside; in the manipulation room, in the dressing-room, in the operation-room, in the diagnos-

tic room, in the polyclinic, in the pathomorphological department; during duties in the clinic.

Practical classes consist of management of patients in the wards of the department, operation-room, dressing-room, and polyclinic. During curation of patients, the students take anamnesis and make objective examination of patient independently, using the methods of additional examination (laboratory, instrumental) etc., make a diagnosis and administer treatment. A student fills in case of every patient's history, reads it out at the practical classes, and then it is discussed with a participation of the whole group. The student prepares the case report according to the scheme and defends it at the end of the cycle in the presence of the professor or assistant professor.

The lectures present issues of etiology, pathogenesis, pathological picture, clinical course, differential diagnosis and treatment of surgical diseases. As to pathogenesis, special attention is paid to comparison of the existed theories devoted to this question. To give the clinical picture on the whole, it is necessary to teach a student to take anamnesis, to give estimation to the objective data, results of roentgenologic, instrumental and laboratory studies. In making diagnosis, a differential diagnosis is discussed. Differential diagnosis is considered the main peculiarity of course of faculty clinical surgery.

Study of clinical thinking is considered the most essential and responsible in the educational process.

To master the method of examination of a surgical patient thoroughly, to enrich knowledge with surgical pathology, a doctor should gradually accumulate experience of the clinical thinking. Formation of the clinical thinking takes place during active work of the student at the patient's bedside under the guidance of the teacher. The clinical thinking is perfected in the process of practical activity; application of new methods of diagnosis and treatment requires constant evaluation of different clinical information and perfection of the clinical thinking.

Experience of the faculty surgery course tution gives evidence that formation of the future specialist should be conducted with integration of theoretical and practical training. The methodological studies showed that some students who had high enough level of theoretical knowledge and were able to analyze the presented data sometimes could not apply them to the concrete patient, as they are not well informed with the methodological peculiarities of the clinical practice. Therefore, the work at the patient's bedside as well as an independent work of students and patients and the subsequent clinical discussion according to the established method is very important. Students develop practical skills during training in surgery.

Before working in the manipulation room, a student should learn the corresponding literature and get familiar with devices and equipment of the manipulation room, character of performing manipulations, and master the technique of their performance. While studying the blood transfusion method one should independently determine the blood group and Rh-factor, fill the system, etc.

In the dressing-room a student in practice get acquainted with its equipment, asepsics and antiseptics, the methods of work, independently (under guidance of a doctor or a teacher) makes dressings, removes and places sutures.

For better mastering the practical skills 2–3 students work in a few manipulation and dressing rooms. A teacher controls and assesses work of students, gives necessary consultations.

Before the class in the operating room, a student repeats material of the 3rd year as to equipment of the operating room, question of asepsics and antiseptics, methods of treatment of surgeon's hands, operating field, studies basic principles of technique of operations depending on the character of the disease.

In a diagnostic room, the students get acquanted with its work, equipment (endoscopy, US apparatuses, etc.). A teacher informs the students about the supposed diagnosis of a patient, purpose of examination, possibility of applying one or another method of diagnosis. Students take part in the endoscopic examination of patients.

In polyclinic the students get acquainted with documents, study to fill the case in, take part in reception of patients, dressing, manipulations, write out prescriptions of medicines, etc.; get recommendations concerning the peculiarities of examination of urgent and elective patients, render necessary aid at the prehospital stage. For mastering surgical manipulations (puncture, blockade), if there is no possibility to learn performing in the clinic, for example, due to difficulty of puncture of the heart or lack of such patients), sectional material and phantoms are used. There is a list of manipulations, necessary for mastering by students.

After questioning about the technique of manipulations, indications and contraindications to them, complications, which can arise during execution, every student performs these manipulations. It is necessary to do it in rubber gloves.

While being on duty in the clinic a student makes his daily round in the department and examine just admitted seriously ill patients. Besides, he takes part in administered procedures (enemas, stomach lavage). On admission of patients with urgent surgical pathology, a student should take part in examination and rendering a necessary aid. A teacher or a doctor on

duty assesses student's activity during his duty at the practical training. The mark is registered in the test list.

Along with high requirements to theoretical knowledge, maximum attention is paid to the effective use of the time given for independent work of students, in order to master necessary practical skills in accordance with qualification demands for a doctor.

It is important to know the wish of students as to mastering practical skills that is why the questionnaire is filled in after examination on surgery. The results of questionnaire give a possibility to have a picture of the educational process at the department and its timely correction.

Pathogenetic Conception in the Aspect of Clinical Material of the Course of Surgical Diseases

Surgery is a constantly developing science; moreover, it is a creative art. Every doctor must be able to give urgent surgical aid in damages, bleeding, asphyxia, acute retention of urine and some other acute surgical diseases, as well as organize timely hospitalization of patients with acute surgical abdominal pathology to the specialized medical establishments, which is of prior importance in the course of faculty surgery.

Undoubtedly, the urgent abdominal surgery, which seems to be one of the most developed themes of the clinical surgery, is still far from perfection. Even experienced surgeons has sometimes diagnostic difficulties and experience a sense of uncertainty in treatment of patients suffering from acute surgical diseases of abdominal organs. The years when with a diagnosis of "acute abdomen", the surgeon performed "explorative laparotomy", and specified a diagnosis only during operation have become a thing of the past.

Skill of a surgeon is perfected in ability to reveal and recognize the signs of the diseases, in preoperative specification of the diagnosis. It helps in detection more clear indications to the operation, limitation of the extent of operative intervention, introduction of sparing technique of the operation and new methods of diagnosis.

However, it is not necessary to be so pedantic. Doctors know that under conditions of "surgical urgency" even approximate diagnoses are, as a rule, considerably more useful than theoretical speculations with ambitious claims on exactness (especially in urgent surgery).

It is necessary to take into consideration that notion of the disease, as a nosologic unit is generalized, schematic. Every patient has his own peculiar

features. The main thing is not the treatment of the disease, but the treatment of the patient.

There are situations when there are indications to operation, but there is no diagnosis. The value of Legar's rule was not lost: severe acute pain in the abdomen that arose against a background of complete health and has lasted over 6 hours is an indication to urgent operation. Three signs that increase within an hour are indication to the operation: pain, tachycardia, irritation of the peritoneum.

A skilled doctor should not miss the moment when it is necessary to stop the study of manifestations and begin to fight against them. This is especially important in acute surgical pathology of the abdomen, because there cannot be any alternative to operation. However, a useless laparotomy in any case a creative error of a doctor. Therapy is the purpose of the medical activity. During treatment of acute surgical diseases of abdominal organs, a proper auxiliary symptomatic treatment is of great value along with surgery. Unlike etiotropic and pathogenetic therapy, it will not eliminate the principal causes of the disease, but helps an organism to fight with factors which complicate the course of the disease, and save life of the patients.

Lately progress of the biological science has allowed penetrating deeply to the intimate bases of vital processes, which take place in an organism, and clinical application of this knowledge showed the value of the integral organism in development of any pathological process. It armed a clinician with ways for detection and correction of some common disorders in an organism. It became clear that acute appendicitis, acute intestinal obstruction, perforated ulcer of the stomach, acute cholecystitis and others are the diseases of all organism, not only of separate organs. Everything is interconnected in an organism. "The life is a biological chain, and it is as strong, as stable its weakest link" (H. Selye).

The clinical picture of various types of acute surgical diseases of organs of the abdominal cavity has a number of general signs that only partly is a consequence of adjacent initially affected organs in the abdominal cavity. In general, similarity of the clinical picture is conditioned by the general reaction of an organism to the pathological process in the abdominal cavity. In final establishment of the diagnosis of acute surgical disease of organs of the abdominal cavity, its local specific manifestations are taken into account.

While examining the patient it is necessary to choose clinical physical examination as the most simple and easily done for the patient, and the so-called paraclinic methods of examination (laboratory, instrumental) should be considered auxiliary, although they sometimes can have decisive importance in making the diagnosis more precise.

Actuality of this theme is conditioned by prevalence of patients with acute abdominal surgical pathology, which needs surgical aid according to vital indications. Among the general amount of surgical operations, which are performed at the surgical departments, every third is performed for one or another acute surgical disease of organs of the abdominal cavity. In the near future this correlation cannot be lowered, unfortunately; today medicine does not have influential methods of majority of these diseases prophylaxis. It is difficult to name the other group of diseases, which would begin so quickly and dramatically and for such a short period.

Acute surgical abdominal pathology, as well as any disease, causes a number of disorders in the vital functions of the patient's organism, that are felt first of all by a patient, and then they can be noticed by people, surrounding him. Sensations and sufferings make the basis of complaints of the patient, his anamnesis, subjective picture of the disease. Sensations can be revealed only through verbal description of them, a word (language) is the mean of objectivity of consciousness of the patient. Disturbances of the normal state of the organism that can be noticed by other people must become the basis of the so-called objective picture of the disease.

Both subjective and objective picture of the disease consists of a chain of signs that are symptoms of the disease, based on analysis of which a doctor make a conclusion about the functional state of the organism and essence of the disease, i. e. make a correct diagnosis. The disease is recognized through a combination of symptoms.

As in the normal state of psyche of the patient the subjective picture of the disease is a reflection of the real disorders in an organism, it can be a source to understanding the pathological process. It is thus necessary to take into account the individual features of an organism. In most cases, subjective symptoms develop earlier than objective signs of the disease.

The study of disease history is a unique way of detection of dynamics, conditions, formation and development of separate symptoms of the disease, necessary for correct understanding of the disease. Naturally, the complaints of the patient have the subjective-objective character and while estimating them it is necessary to be careful, remembering that two subjects take part in their formation: a patient and a doctor are alive persons with the individual psyche, emotionality, feelings, and mood.

Clinical signs should be thoroughly analyzed. Evolution of a symptom during the course of the disease is of special value.

Skill of the doctor's art is an ability to reveal symptoms of the disease, and medical science must be able to explain the cause of origin and development of symptoms, their pathogenesis in the light of modern achievements of the medical science. Only understanding a symptom, knowing the cause

of its origin and mechanism of its development it is possible to recognize a pathological process that takes place in an organism. It is impossible to neglect the signs. They "take" revenge on the doctor for carelessness.

Let us consider some questions of methodology of differential diagnosis. In making the diagnosis, a doctor compares the clinical picture of the symptoms, revealed in a patient, with the described clinical signs, which have the same complex of symptoms characteristic of certain diseases.

In general, there are two methods of determination of differential diagnosis: by analogy and method of exception.

While making diagnostic hypotheses, a doctor has a possibility to compare the clinical picture revealed in the patient with the abstract clinical pictures fixed by science and human experience. This mental operation — comparison of clinical pictures — is the basis of traditional differential-diagnostic method that is based on analogy.

Thus, in the process of diagnosis the simple analogy of two casual diseases outgrows in the method of differential diagnosis, during differential diagnosis a doctor operates by hypotheses all the time. Having gained a certain complex of the symptoms revealed in a patient, the most similar abstract clinical picture, a doctor expresses a hypothesis about possibility of presence exactly such disease in a patient. If similarity in most symptoms of the clinical picture in the patient with the clinical picture of the abstract patient taken for comparison does not appear, a doctor makes a new hypothesis, expressing supposition about the presence of other disease in the patients.

It should be noted that because of comparison of symptoms it is still impossible to make a reliable conclusion about the diagnosis of the disease.

Therefore, a method of differential diagnosis by analogy is not exact; it allows making only preliminary conclusions. A diagnosis is considerably more reliable, if a possibility of some other disease is eliminated. The method of differential diagnosis is based on the search of difference between the given case and all possible cases after the exception of suppositions, which are not reliable.

Certainly, the starting point of the differential diagnosis by the method of exception is the choice of the most pronounced, leading symptom or syndrome. Then all diseases, which have this symptom or syndrome, are remembered. Comparing the observed picture of the disease with description of those diseases, with which it has similarity, with this symptom or syndrome, a difference is searched between the given case and the similar disease. Based on the revealed differences or contradictions all diseases are eliminated from a search group. In comparison and exception of all similar diseases, except one most similar by clinical symptoms, it is concluded that the patient has this disease.

Thus, distributive-categorical syllogism is of main value in differential diagnosis. Essence of diagnosis is that by the exception of some possible diseases, which were assumed, conclusion is made about most probability of one of them. Realization of this distribution, that is differentiation of diseases, is possible only by distributive syllogism.

For example, it is possible to use such a case: a patient is revealed to have the enlargement of the thyroid gland. In this case it is possible to make the following distributive syllogism: the enlargement of the thyroid gland can be a leading sign in thyreotoxicosis, endemic goiter, acute thyroiditis, tumour. Absence of characteristic symptoms eliminates supposition about presence of thyreotoxicosis, endemic goiter, and acute thyroiditis in the patient. Consequently, the patient has tumour of the thyroid gland. The presence of other signs is a hard tuberous tumour that germinated in the adjacent tissues, insignificant leucocytosis, elevated ESR, exhaustion and others — specifies the presence of cancer tumour. However, determination of correct distributive syllogism is possible only in the case, if all cases of diseases, which have a similar clinical picture, are taken into account.

Distributive and conditional syllogisms are used in a differential diagnosis, as a rule, together, but not separately. Therefore, a differential diagnosis integrates enough complicated combination of different conclusions in each of which, if not to observe laws and rules of logical thought, there may be a logical error. Only logically correct clinical thought can be a reliable means in recognition of extraordinarily rare and severe diseases.

For illustration of making the differential diagnosis, it is possible to give an example, which consists of complicated chain of conclusions: in the syndrome of "acute abdomen" in a patient, it is possible to think about the perforated ulcer of the stomach, acute cholecystitis, acute gastritis, acute pancreatitis, and acute intestinal obstruction. This distributive judgment is the initial basis of distributive-categorical syllogism.

For its making, another judgment is nesessary, in which all diseases are excepted but one. A number of conclusions, which are conditional-categorical syllogizms, are thus made. The thoughts are as follows: if it is a perforated ulcer, it very often occurs in men, it is characterized by presence of free gas under the diaphragm, tension of muscles in the hypochondrium and absence of frequent vomiting. This patient does not have gas under the diaphragm, and there is frequent vomiting. Conditional syllogism is correct, because taking aside the consequence; a doctor fits for objection of basis. Consequently, a conclusion that the patient does not have a perforated ulcer is reliable.

Similarly all pre-conditions are excepted, but one. In relation to the last supposition, it is necessary to think as follows: if it is pancreatitis, transver-

sal resistance of the abdominal wall, considerable leucocytosis, severe general condition, etc. are typical. This patient has acute transversal painfulness of the abdominal wall, considerable leucocytosis, grave condition, etc. Consequently, this patient may have acute pancreatitis.

As all forms of "acute abdomen" are considered in this case, a doctor, operating all initial conditions for construction of distributive syllogism now, can come to motivated conclusion logically: in the syndrome of "acute abdomen" the following diseases are possible: perforated ulcer of the stomach, acute cholecystitis, acute appendicitis, acute intestinal obstruction, etc. The patient cannot have perforated ulcer of the stomach, acute cholecystitis, and acute appendicitis. Thus, the patient has acute intestinal obstruction. Therefore, complementing conditional syllogism by a distributive one, the doctor passes from the possible diagnosis to the motivated reliable diagnosis.

Short Essay from Development of Surgery in Ukraine

The students get acquainted with history of surgery at the course of general surgery, history of medicine, etc. In development of this important link of medical science, our domestic scientists who created scientific schools made the outstanding contributions: in Kharkov — V. Kh. Grube, A. G. Pidriz, M. P. Trincler, in Kiev — V. O. Karavayev, Yu. K. Shimanovskiy, F. K. Berngaupt, A. Kh. Rinek, L. A. Malinovskiy, M. M. Volkovich, M. V. Sklifosovskiy, in Odessa — K. M. Serapin, K. M. Sapezhko, A. N. Shogolyev. All of them as founders of surgical departments of universities worked in the pre-war years. In the peace time the academicians M. M. Amosov, O. O. Shalimov, D. P. Tchukhrienko, etc. played a large role in development of surgical science in Ukraine.

M. I. Pirogov is one of the founders of higher medical education in Odessa, his efforts were continued by professors M. P. Sokolovskiy, Ya. M. Voloshin, M. L. Dmitryev, K. G. Tagibekov etc.

History of the Faculty Surgery Department

The staff of the Faculty Surgery Department of the Odessa State Medical University develops positive traditions of the world and domestic medicine, makes extraordinarily large and useful work training the medical personnel for the system of health care in our and foreign countries.

At the end of 1902 construction of the building was completed, and at the beginning of 1903 sanctification and inauguration of the surgical clinic of the medical faculty of the Novorossiysk University took place. This event was thoroughly described in one of the issues of the magazine "Niva" in 1903. Kyril Mikhaylovich Sapezhko (1861–1929) one of the best students of professor A. Kh. Rinek, the head of the faculty surgical clinic of the Kiev Medical Institute, was the founder of department of Faculty Surgery. In 1892 K. M. Sapezhko defended dissertation for the degree of the doctor of medicine on theme "Clinical material to the question about transplantation of the mucous membrane". This was highly estimated by P. I. Dyakonov. K. M. Sapezhko made a report on the theme of the dissertation in 1884 in Rome at the International Congress of Doctors. In opinion of doctors of many countries, the report was brilliant. Transplantologists from the Institute of Organs Transplantation and tissues consider K. M. Sapezhko the founder of mucous membrane transplantation.

K. M. Sapezhko was the first professor of surgery at the Medical faculty of the Novorossiysk University. He began intensive research and medical studies in clinic. Surgery of organs of the abdominal region was basic direction of his researches. He was one of the first surgeons in Ukraine who successfully began to perform operations with application of gastro-intestinal anastomosis in ulcerous disease. The scientist described his views in the monography "Surgery of the stomach. Ulcer and its complications. Y-gastroenteroanastomosis in ulcerative and tumoral constriction of the pylorus" (1901). Professor K. M. Sapezhko is the author of the operation in umbilical hernia, which considered outstanding in the whole world.

In 1914 a small book by K. M. Sapezhko "Two words about treatment of wounds" was published in Odessa, intended for "young people leaving for the battle field". The methods of treatment of gunshot wounds, which were progressive for that time, are given in this book; the method of drainage and irrigation of wounds is offered. Recommendations of the scientist were a considerable step ahead in treatment of septic wounds of different areas of the human body. This method quickly acquired wide distribution and was used not only during the 1st World War, but during also long time after it. Today due to considerable growth of incidence of purulent complications in surgery, the method of permanent irrigation of wounds acquired subsequent development.

K. M. Sapezhko was a wonderful lecturer, the active member of association of the Russian doctors in memory of M. I. Pirogov, and participant of all surgical congresses of the pre-revolution Russia.

A disciple of K. M. Sapezhko professor Anatoliy Andreevich Novikov, who worked in the Military Medical Academy since 1921, headed the department from 1919 to 1921.

From 1921 to 1931 professor Vladimir Leontyevich Pokotilo (1880–1931), a disciple of P. I. Dyakonov, headed the department. A wonderful doctor, talented leader, scientist, literary man, active public man, Vladimir Leontievich for short time managed to organize brilliantly the scientific, pedagogical and medical studies. The questions of fight against a surgical infection were developed at the department, as a result the method of surgeon's hands treatment was offered by the method of Pokotilo; plasty surgery, anaesthesia, surgical endocrinology. At that time the talented scientists worked in the clinic, they are L. A. Barinshteyn, A. A. Babskiy, S. A. Bokkal, A. B. Rayz and others. V. L. Pokotilo played a large role in organization of the Odessa Surgical Society.

After death of V. L. Pokotilo a disciple of K. M. Sapezhko professor P. G. Chasovnikov (1877–1954) headed the department and clinic. Under his guidance, the clinic was engaged in surgery of the liver and bile ducts up to 1936. More than 900 operations were performed on the bile ducts. This was a leading medical establishment in Ukraine. Questions of allergy in the mechanism of dysfunction of the liver were studied, postcholecystectomy state, morphological changes in the liver under the influence on vegetative nervous system, treatment of ulcerous disease of the stomach and duodenum, surgical treatment of diseases of the thyroid gland (174 operations were performed on the thyroid gland in clinic in 1936), as well as anaesthetizing and improvement of anesthesia devices, fight against surgical infection and treatment of burns.

During the period when P. G. Chasovnykov had been head the department there were prepared professors: E. Kh. Koch, A. Z. Kozdoba, A. G. Sosnovskiy.

From 1945 to 1964 a disciple of S. I. Spasokukotsky professor Michaylo Pavlovich Sokolovskiy headed the department. In 1911 M. P. Sokolovsky worked at the Department of Pathological Physiology of the Saratov University after graduation from Medical faculty of the Moscow University, which was headed by O. O. Bogomolets. At the end of 1913 M. P. Sokolovsky was made an assistant of the Faculty Surgical Clinic which was headed by S. I. Spasokukotsky. In 1920 he defended the dissertation made under the guidance of O. O. Bogomolets.

The scientific work was activated at the department after arrival of professor M. P. Sokolovsky. The problems of thromboobliterative diseases of vessels of the extremities, neurosurgery, pathophysiology of the wound process and treatment of wounds were developed in the clinic. When professor M. P. Sokolovsky headed the department, there were defended 13 candidate's and 2 doctor's (M. F. Kamayev, A. S. Sinovets) dissertations.

In 1964 a disciple of M. P. Sokolovsky Anatoly Stanislavovich Sinovets was elected to be the head of the department of faculty surgery. At that time, important problems of surgical pathology were intensively developed at the department, namely ileus and gastrointestinal stasis (dissertations of I. F. Lvov and A. A. Sinovets). A great attention was paid at the department to the study of different questions of pathogenesis of acute pancreatitis (experimental works of A. L. Dekhtyar, P. S. Redchits, I. F. Hudoley, V. A. Michurin, A. L. Veretennikov), to treatment of this disease, in particular, by inhibitor preparations. A methodical list was published on this theme. The questions of treatment of diseases of the arterial and venous vessels of extremities were intensively developed (dissertations of V. O. Nikulin, B. D. Morozov).

Special attention was paid at the department to the fight against surgical infection (immunoprophylaxis and immunotherapy of staphylococcal complications), treatment of wounds (dissertations of V. G. Us, A. V. Snisarenko, P. G. Litvinov). Methodical recommendations are also published on these themes.

Before creation of the department of urology in 1970, urology and scientific studies, devoted to diagnosis of chronic pyelonephritis, chronic renal insufficiency, adenoma of the prostatae at the department of faculty surgery were taught (dissertations of K. V. Velickanov, N. P. Chashina, D. F. Tuchin).

In 1984 due to surgery teaching reorganization in the Odessa Medical Institute the department of Faculty Surgery was reorganized to the department of Surgical Diseases N 3 headed by professor A. S. Sinovets. At that time the course of faculty surgery was taught to the students of the 5th year of medical faculty, and hospital surgery was taught at the 6th year. Such situation of teaching of surgical discipline had continued till 1990.

From 1964 to 1990 thirty dissertations were defended, five of which were doctoral; there were prepared and published 4 monographies at the department. Professor A. S. Sinovets headed the Odessa Regional Society of Surgeons for 19 years, was a member of all-union and republican societies of surgeons, participant of all-union and republican congresses of surgeons.

During 1989–1990 Igor Fedorovich Lvov temporally performed the duties of the head of the department.

In 1990 the department of surgical diseases N 3 was incorporated into the department of surgical diseases N 2, which was headed by a doctor of medical sciences, deserved worker of higher school of Ukraine, professor A. L. Dekhtyar since 1987, its basic clinical base was in the 11th Municipal

Clinical Hospital. Besides of a basic clinical base, teaching of faculty surgery was conducted at the clinical bases of the 10th and 12th Municipal Clinical Hospitals, 411th Military Hospital.

In 1997 due to changes in teaching of the course of surgical diseases at the Medical faculty of the Odessa State Medical University the department of Faculty Surgery was incorporated into the department of Faculty and Hospital Surgery of pediatric and dental faculties under the guidance of the honoured worker of science and technique of Ukraine, doctor of medical sciences, professor Sergiy Olexandrovich Geshelin.

In 1999 the department of Faculty Surgery of the medical faculty was headed by the doctor of medical sciences, professor Anatoliy Sergiyovych Son. A course of neurosurgery was also taught at the department.

Surgery of the massive gastrointestinal bleeding, purulent-septic surgery of diabetic foot, surgery concerning many forms of primary and secondary pathology of the brain vessels, as well as widely spread introduction of endoscopic technologies in diagnosis and treatment of many surgical diseases assumed ever greater importance. Associate professors A. S. Son and Yu. V. Grubnik defended doctoral dissertations on materials of scientific development of these themes; at present medical staff of clinic prepare a few candidate dissertations.

Due to requirement for training highly skilled surgical personnel and in order to save the unique doctrine of teaching of surgical science and new resolution of Ministry of Health and department of Education and Science of Ukraine concerning training of future doctors the Department of Faculty Surgery became independent in 2002. It is headed by honoured doctor of Ukraine, doctor of medical sciences, professor Boris Sergiyovich Zaporozhchenko.

Today the department of faculty surgery has powerful scientific potential. Development of treatment problems of hepatopancreatoduodenobiliary diseases is a priority direction. Five professors, doctors of medical sciences, three candidates of medical sciences work within its walls. For the last five years the three doctoral and five candidate dissertations were defended. Five candidate dissertations are planned and successfully preparing.

Lecture 1

SURGICAL DISEASES OF LUNGS AND PLEURA

ACUTE PYO-DESTRUCTIVE DISEASES OF THE LUNGS

Acute Abscesses and Gangrene of the Lungs

Acute abscesses and gangrene of the lungs belong to the group of so-called unspecific infectious destructions of the lungs, which are characterized by necrosis of the pulmonary parenchyma, progressive advance of purulent or putrid process, melting and formation of cavities in this area.

A cause of combination of abscesses and gangrene of the lungs in one group — diseases, different by their clinical manifestations, severity of course and prognosis, — is the same type of the initial phases. Development of characteristic morphological changes in the subsequent development of the pathological process is a reason to consider it to be one or another form of infectious destruction of the lungs.

Lung abscess is characterized by formation of the cavity in the pulmonary parenchyma separated from undamaged parts by a pyogenic capsule, that is formed in the process of development of purulent or putrid inflammation in it.

Pulmonary gangrene is characterized by necrosis of the large area of the pulmonary parenchyma, at the initial stage depending on the volume of which (segments or part of the lung), virulence of microflora, general state of the organism. The infectious-destructive process can be separated from the undamaged parts by the inflammatory-granulation "ban" or in absence of separation and progress of necrosis involve the whole lung (extended gangrene of the lung).

Abscess-forming pneumonia as a nosologic form takes an intermediate place between pneumonia and infectious destruction of the lungs. It is characterized by formation of small abscesses and purulent melting in the areas of pneumonic infiltration. Such areas of purulent inflammation can be fully completed, evacuating under favourable conditions through the bronchi,

or result in formation of greater areas of necrosis with development of abscess or gangrene.

Worsening of breathing hygiene of modern man that disturbs the protective mechanisms of the respiratory system, growth of rate of the immunodeficient states, caused by diabetes, alcoholism and other causes, increases of the causative agents antibioticoresistant strains dissemination are considered to be factors, which are favourable for development of infectious destructions of the lungs.

High incidence of lung abscesses is in pneumonias (from 2 to 5%), closed traumas of the chest (1.5–2%), gunshot wounds (1.5%). In spite of considerable progress in treatment of this pathology, lethality in lung abscesses makes 10%, and in pulmonary gangrene it remains very high — from 40 to 90%.

Etiology and pathogenesis. Acute infectious destructions of the lungs are, as a rule, unspecific, but there are sometimes mixed forms, when unspecific and specific infectious processes develop simultaneously. Mostly in infectious destructions of the lungs gram-negative and anaerobic microorganisms, as well as staphylococci are revealed. *Esherichia coli* is the most frequent representative of the gram-negative flora, proteus, pseudomonas aeruginosa. Staphylococcus is the causative agent of destructions, which complicate epidemic flu. Fuzospiril flora is determined in association with enumerated types of the causative agents in 25–50% of cases. It should be noted that identification of microorganisms causing pulmonary suppurations is still difficult due to wide application of antibiotics, complications of taking and culturing microorganisms under anaerobic conditions. However, penetration of pathogenic microorganisms in the pulmonary parenchyma is not enought to cause infectious destruction of the lungs, as due to cleansing function of the tracheobronchial tree that was well preserved under conditions of normal functioning of local and general protective mechanisms, such a microbal invasion does not have any clinical manifestations. The development of acute infectious destruction of the lungs is provoked by pathogenic factors, which disturb these mechanisms. They include:

— high virulence of pathogenic microflora;

— impairment of passage and drainage function of the bronchi;

— disorders of microcirculation in the area of infectious or posttraumatic inflammation of the pulmonary parenchyma.

The principal causes of impairment of bronchial passage are edema of the mucous membrane of the fine bronchial tubes in massive inflammation of the pulmonary parenchyma, aspiration of vomit masses in loss of consciousness, acute alcoholic intoxication, anesthesia, comatous state, epilep-

tic fits and so on. Massive aspiration of the sour gastric contents causes pronounced chemical affection of the mucous membrane of the bronchial tubes (general bronchospasm) and pulmonary parenchyma (syndrome of Mendelson) with formation of atelectasis, hemorrhagic edema and necrosis of the alveolar septums. Foreign bodies, benign and malignant tumours, purulent exudate with high contents of fibrin, enlarged parabronchial lymph nodes can cause obturation of the bronchial tubes. A leading role in development of infectious destructions of the lungs is played by chronic unspecific diseases of the bronchial tubes, alcoholism, neurologic diseases, which are accompanied by impaired act of swallowing, unconscious states, gastroesophageal reflux, complications during anesthesia, bronchoadenitis of different etiology, bronchial asthma.

Disorders of blood supply to the pulmonary parenchyma mainly develop in embolism of the branches of the pulmonary artery and are accompanied by infarct-pneumonia. The area of necrosis that is formed is subject to purulent or putrid disintegration as a result of vital functions of pathogenic microbe flora in the area of necrotic masses that penetrated together with septic embols or secondary. In this case a suppurative process may develop in the area of aseptic infarct. Septic endocarditis, thrombophlebitis of veins of extremities and pelvis, phlebitises during catheterisation of the peripheral and central veins, abscesses of different localization are the main diseases in such mechanism of formation of pulmonary destruction.

Operative interventions on infected tissues promote migration of septic blood clots. Disorders of microcirculation in the pulmonary parenchyma quite often arise in diabetes mellitus and thrombohaemorrhagic state of other etiology; these states are quite often accompanied by pulmonary suppurations, as disturbance of patient's immunobiological status is assumed (insufficient correction of carbohydrate metabolism, hormone therapy).

Rather high rate of infectious destruction of the lung in traumas of the chest is to a great extent caused by combination of many factors, which result in development of pulmonary suppuration: posttraumatic necrosis and direct microbe dissemination of the pulmonary parenchyma, impaired bronchial passage due to obturation by blood, mucus, damage of the bronchial tubes, reduction of the protective forces of an organism after trauma and large blood loss. Development of infectious destruction of the lungs often takes place against a background of previous respiratory viral infection, due to deep disorders of local protective mechanisms, which "open the road" to bacterial infection.

A modern look on pathogenesis of infectious destructions of the lungs is as follows. In the area of inflammatory infiltration of the pulmonary parenchyma, regardless of its origin (bacterial pneumonia, infarct-pneumonia,

septic infarct-pneumonia, and aspirative pneumonia), the bronchial tubes passage impairment as a result of edema of the mucous membrane, spasm, obturation by their pathological contents. Infiltration and progress of edema result in microcirculation disorder in the parenchyma of the lungs and thrombosis of the pulmonary vessels that causes necrosis of the pulmonary parenchyma and intensive development of pathogenic microflora in it. Posttraumatic abscesses and gangrene of the lungs are the exception, when there are "primary" necrosis of the pulmonary tissue, and a blood supply disorder in the area of trauma. In future, depending on predominance of elements of necrosis or purulent inflammation and demarcation in the course of the pathological process, its course takes place after the type of abscess or gangrene of the lungs.

In formation of acute purulent abscess in the center of inflammatory infiltrate, there is purulent melting of necrotized masses. In small foci of necrosis and renewal of drainage function of the bronchial tubes of the abscesses are fully evacuated, forming fine (0.3–0.5 cm in diameter) cavities in the infiltrative pulmonary tissue ("abscess-forming pneumonia"). In more extensive area of necrosis purulent melting and infiltration of the pulmonary tissue in the demarcation area result in formation of the spherical cavity filled with pus and separated (to a greater or less degree) from the unaffected pulmonary tissue by a pyogenic capsule. In future, there is release of pus through the bronchial tubes (second phase of the course of the disease). In sufficient emptying of abscess through the bronchus the inflammatory process subsides, infiltration disappears gradually, the cavity becomes deformed, diminishes in size, the granulation tissue is formed on its walls, development of which in combination with reduction of the cavity size brings about complete obliteration of the latter. It is the most favorable result of infectious destruction of the lungs. Formation of the area of minimum pneumofibrosis as a result of acute abscess is considered to be a complete recovery.

Such course of acute abscess of the lungs is observed in 85% of cases. However, not infrequently in considerable size of the cavity, peculiarities of breathing technique in different parts of the lungs, state of the pulmonary parenchyma (emphysema, pneumofibrosis), disregarding complete emptying of abscess and disappearance of perifocal infiltration, the cavity is not obliterated but epithelizes from within at the expense of granulation of the bronchial epithelium. In this case, the question is forming of the real cyst. During stabilization of the cavity size without epithelisation of its walls a dry residual cavity is formed. Such result is estimated as clinical convalescence, however, the subsequent course of the process is unpredictable (lifelong asymptomatic cavities, obliteration of the dry residual cavi-

ty, its suppuration with formation of lung abscess). The result of transformation of acute purulent abscess into a cyst is observed in 5% of cases.

In inadequate treatment of infectious destruction of the lungs, more frequently associated with insufficient emptying of the purulent cavity in the area of perifocal inflammation of the pulmonary tissue, the connective tissue is formed, that forms a thick wall of abscess, which hinders approchement of the affected areas of the lungs, and obliteration of the cavity. The areas of fibrosis develop in the adjacent pulmonary tissue, that impare ventilation, deform fine bronchial tubes, and it results in a transformation of the process into chronic form — chronic lung abscess (10% of cases).

In pulmonary gangrene the area of necrosis quickly extends, involving a part, two parts or the whole lung. The numerous cavities of putrid disintegration, filled with stinking contents, are formed in the necrotized parenchyma, the pathological process involves pleura with development of empyema and conjunction with lumen of the bronchial tubes (pyopneumothorax). Due to absence of demarcation of the pathological process the products of putrid disintegration are absorbed in blood, causing severe intoxication, toxic affection of other organs, that quickly leads to death of a patient. In case of more favourable course, the area of ichoroze disintegration is gradually demarcated from unaffected pulmonary parenchyma, forming a cavity with offensive contents, which contains freely lying and necrotized tissues fixed to the walls (gangrenous abscess). The cavity, as a rule, is over 5 cm in diameter; its walls are irregular, bay-like, with pronounced perifocal infiltration. Melting and elimination of the necrotized tissues is slow. Because of great size of the purulent cavity and its insufficient emptying due to presence of tissue sequesters, a chronic abscess is formed as a rule in the lungs. Clinical convalescence ("dry" cavity) is observed very rarely in this case.

However, despite variety of types of pathogenesis and clinical course, classification of infectious destructions of the lungs is possible, which promotes determination of the most rational medical management.

Classification. According to etiology infectious destructions of the lungs are divided depending on the type of the microbe causative agent which is determined by modern methods of bacteriological studies, on the basis of clinical data, that substantiates antibacterial chemotherapy.

According to pathogenesis:

1. Bronchogenous (including aspirative and obturative).
2. Haematogenous (including embolic).
3. Posttraumatic.

According to the character of the pathological process:

1. Acute purulent abscess.
2. Acute gangrenous abscess (limited gangrene).
3. Gangrene of the lung (extended gangrene);
4. Chronic abscess.

According to severity:

1. Mild.
2. Moderate.
3. Severe.

According to the character of the course:

1. Uncomplicated.
2. Complicated (by empyema of the pleura, pulmonary bleeding, sepsis, pneumonia of the opposite lung, etc.).

In addition, abscesses can be:

1. Single.
2. Numerous.
3. Unilateral.
4. Bilateral.

Clinical course. The clinical course of infectious destruction of the lungs is very variable and depends on the individual (including immunologic) peculiarities of an organism, stages of the process, presence of causal and concomitant diseases. Two periods are distinctly differentiated in the clinical picture of "classic" acute abscess: a period of formation of the purulent cavity until its perforation in the bronchus and a period after the perforation in the bronchial tree.

On purposeful inquiry patients not infrequently mark the episodes of overcooling, previous acute respiratory infection, exacerbation of chronic bronchitis of a smoker, as well as situations, in which the aspirative mechanism of the onset of destruction could be realized for (operation under anesthesia, episodes of severe alcoholic intoxication, epileptic fits, comatose state, etc.).

The first period of a "classic" clinical form of the acute purulent abscess is characterized by acute beginning with the increase of the body temperature up to 38°C and more, pain in the chest and cough — dry or with small ammount of sputum. Physical symptomatology in this period is scarce: weakening of breathing in the area of affection, dry and moist rales of different caliber are determined. Changes in the peripheral blood characteristic of the purulent process of any localization with pronounced purulent intoxication, positive reactions of the acute phase of in-

flammation. Massive infiltration of the pulmonary tissue is determined roentgenologically, mainly within the limits of one-two segments or the lobe of the lung. The first period usually lasts for 7–10 days, and enumerated symptoms are considered as manifestations of acute pneumonia. However, already at this time, a doctor should take into account failed conducted therapy.

The second period is characterized by appearance of unpleasant odour from the mouth and a plenty of purulent sputum (up to 200–300 ml per day). It is accompanied by subsiding fever, improvement of the patient's general condition. Roentgenologic examination helps to detect the cavity (cavities) of destruction with the horizontal level of liquid against a background of infiltration of the pulmonary tissue that confirms the diagnosis of acute abscess of the lung. In some cases, the disease has at first a torpid course with discharge of small amount of purulent sputum and slow formation of cavity of destruction in unevenly infiltrative pulmonary parenchyma. In aspirative genesis of acute abscess of the lung there is extraordinarily intensive course of the disease: a plenty of stinking sputum, which develops from the first days of the disease, considerably more expressive manifestations of general purulent intoxication. In gangrene (gangrenous abscess) of the lungs the clinical picture of the disease is characterized, unlike the described one, by the grave condition of a patient, marked intoxication up to development of septic shock, respiratory insufficiency and, quite often, signs of polyorganic insufficiency come afore. A patient complains of pronounced weakness, loss of appetite, thirst, painful cough with stinking sputum (of brown, grey-brown colour), which after sedimentation divides into three layers: crumb-like sediment on the bottom; turbid, scarce — in the middle; muco-purulent, foamy — upper one.

Sometimes the odour is so unpleasant on breathing that it is impossible to stay close to a patient. Flaccidity, adynamias are characteristic. The skin is dry, grey. The lips and the nail phalanges are bluish. The affected side of the chest falls behind in breathing. Physical data depend on the volume of necrosis of the pulmonary tissue and expressiveness of disintegration — dullness of the percussion sound, a box-like tint above the cavity of destruction, located subcortically; there is the considerable weakening (absence) of respiratory sounds on auscultation, amphoric tint above the cavity drainaging through the bronchus, moist rales of different caliber.

Roentgenologically massive confluent infiltration of the pulmonary parenchyma within the limits of a part or the whole lung in the initial phases of gangrene of the lung is determined. In progress of disintegration against a background of infiltration of the pulmonary tissue, there are numerous cavital foci of destruction of different size and degree, filled by selection. On tomography of the lungs the tissue sequesters of irregular form are deter-

mined, located freely in most cavities of destruction. As a rule, the course of gangrene of the lung is complicated by haemophtysis, pulmonary bleeding, and empyema of the pleura (pyopneumothorax), that considerably worse the patient's condition.

Laboratory indices give evidence of severe intoxication and suppression of factors of unspecific defence. As a rule, there are observed acute leycocytosis (more than 30 thousand per 1 mm), increased ESR over 70 mm/h, changes of leucocytic formula of blood with predominance of young forms, pronounced toxic granularity of neutrophils, considerable anaemia. There may be hypoproteinemia, disproteinemia, disturbance of water-electrolyte balance, acute metabolic acidosis. If the indices of leucocytes of blood are normal at the height of severity of the state, the unfavorable sign that is evidence of deep suppression of the immune system is prognostic. The subsequent clinical picture of the disease depends on whether the area of necrosis demarcated from the uneffected pulmonary tissue, or becomes extensive gangrene. In the first case, the course of the disease is similar to manifestations of an abscess of the lungs; however, it has a torpid character and mostly results in formation of chronic abscess. In the second case, surgical intervention is usually necessary, and, as a rule, recovery is impossible without it.

Diagnosis. The diagnosis of infectious destruction of the lungs is based on anamnesis, evaluation of clinical manifestations, data of laboratory and roentgenologic studies. Data of the roentgenologic examination — roentgenoscopy, roentgenography, and tomography are the basic source of verification of the diagnosis, if possible — computer tomography should be made. It is difficult to estimate efficiency of the conducted treatment and to carry out correction of medical measures, without systematic repeated roentgenologic examination (in 1–2 days).

The bacteriological methods of research are important among the laboratory investigations, as their results influence the choice of optimum amount of etiotropic therapy. Research of sputum in presence of tubercular rods, mycotic flora is obligatory.

The list of obligatory instrumental researches should also include bronchoscopy that allows excluding a tumour nature of the process, to take the material for bacteriological and cytologic research. Fibrobronchoscopy in combination with an introbronchial biopsy from the walls of the cavity formation should be preference in invasive methods, as transthoracic intrapulmonary biopsy of the wall of abscess.

Differential diagnosis of infectious destructions of the lungs is very difficult due to variety of the clinical manifestations of the disease in different periods. First it is necessary to differentiate infectious destruc-

tions of the lungs with cavernous and some other forms of lung tuberculosis.

The differential diagnosis with cancer of the lungs should often be necessary. The negative results of bronchofibroscopy do not allow excluding the peripheral form of cancer of the lungs with disintegration. In presence of substantiated suspicion on a neoplasm there must be made morphological verification of diagnosis (different methods of biopsy).

Not infrequently there should made a differential diagnosis of acute abscesses of the lungs with different types of limited empyema of the pleura (limited pyopneumothorax, interlobular empyemia, apical empyema) and the so called pleuropulmonary cavities, in which one of the walls of the cavity is the pulmonary tissue, that disintegrates, the other — parietal pleura (empyema of the pleura with destruction of the lungs), subdiaphragmatic abscesses. Ultrasonic research and computer tomography are most informative in such cases.

It is sometimes necessary to differentiate the abscesses of the lungs with echinococcosis, especially in endemic unfavorable regions and in the corresponding anamnesis data, especially when the echinococcal cyst which became suppurated practically is an abscess of the lung, with abscess-formation in the pulmonary parenchyma in actynomycosis, aspergillosis of the lungs with formation of the cavity, that contains mycelium of the funges — the so called aspergilloma. Thorough investigation of the patient for mycosis allows to make a correct diagnosis.

Ttreatment of acute infectious destructions of the lungs includes pathogenetic and etiotropic therapy accordihg to the following scheme:

1. Maximally complete emptying of the cavities, which contain purulent content.

2. Antibacterial therapy taking into account sensitivity of microflora;

3. General treatment, directed at the elimination of intoxication, correction of all types of metabolism, stimulation of protective reactions of the organism. In unfavorable course of lung gangrene such treatment is a preparatory stage before the radical operative intervention. Success of operation to a greater extent depends on general treatment.

Noninvasive sanation of pulmonary abscesses should include:

1. Medicinal influence on the mucous membrane of the bronchial tubes for reduction of edema and elimination of bronchospasm (euphyllin, teofedrin, broncholytin). The optimum way of medicine introduction is inhalation by the ultrasonic method. Inhalation should contain hormones, which have marked antiedematous effect.

2. Dilution of viscid sputum (expectorants, including those of herbal origin: decoctions of termopsis, violet of three-colour, etc.). Besides, inha-

lations with inclusion of proteolytic enzymes (terilitin, teridecaze, tripsin, hemotripsin, RNA-element), acetylcystein in disperse solution are indicated.

3. Obligatory combination of the enumerated measures with permanent postural drainage. A patient is placed in such position, in which the lower point of purulent cavity is an ostium of the draining bronchus. It is expedient to complement the postural drainage with vibromassage (pating on a pectoral wall in the projection abcess). These procedures should be performed as frequent as possible especially intensively in the morning and in the evening. If sanation of abscess during permanent conduction of the indicated measures proved ineffective (a plenty of liquid in a cavity of abscess on roentgenoscopy, small volume of coughed sputum), it is necessary to make bronchofibroscopy, during which bougienage of the ostiums of subsegmental bronchial tubes is made by the closed biopsy forceps, and then brushes of different size. Procedure is completed by catheterization of the abscess cavity through the canal of bronchoscope and introduction of medical composition that contains antibiotics, antiseptics, proteolytic enzymes. It can be carried out in 60–70% of patients. Leaving of the catheter in the abscess cavity for 1–1.5 week does not have undesirable consequences, but frequent roentgenologic control is needed for the timely exposure of migration of catheter. In some cases, it is expedient to make a lavage of the abscess cavities through a catheter in postural position. Lavage of abscess during subanesthesia of hard bronchoscopy may result in a bronchogenic dissemination of the process.

Antibacterial treatment is given to influence microflora that vegetates in the abscess cavity and in the area of perifocal bacterial inflammation. However, there is no necessity in antibacterial treatment in absence of liquid in the abscess cavity and perifocal inflammation. The ways of introduction of antibiotics is chosen individually depending on the phase of the process, amount of introduction, patient general state. Expedience of introduction of antibiotics into the pulmonary artery is probably exaggerated. Intravenous introduction of antibiotics is no less effective than the mentioned method and does not need such difficult manipulations. Intratissue electrophoresis with antibiotics proves to be a very perspective method that provides introduction of them directly in the abscess cavity. Daily dose is introduced intravenously combined with galvanization of tissues by the aparatus “Stream-1” in the projection of the pulmonary abscess. It promotes moving of antibiotics under the influence of the electric field directly in the focus of affection.

The criterion of progress of treatment, that is given, is improvement of the patient’s general state, normalization of the body temperature, reduction of amount of sputum, disappearance of its unpleasant smell in roentgenologically confirmed absence of liquid in the cavity and reduction of its

size. As a rule, the systematic, persistent conduction of the indicated measures allows to obtain good results of treatment in majority of patients. In absence of positive dynamics during 2 weeks of complex therapy, operative drainage of the purulent cavity is indicated. It may be in detection of plenty of detritus, sequestra in the abscess cavity, which interfere with its natural emptying.

Transthoracal drainage of abscess by Monaldi is considered the most rational method of the noninvasing emptying of the lung abscess. For successful drainage, the following conditions are needed: its subcortical location; not less than 2 weeks from the beginning of the disease (time, necessary for development of adhesions, which prevent collapse of the lungs), determination of exact localization of abscess by roentgenoscopy. It is desirable to make manipulations under roentgen-television examination control. It is necessary to avoid puncture of abscess, as their efficacy is low, and probability of possible complications (air embolism of the cerebral vessels of the cerebrum, pyopneumotorax, internal and pulmonary bleeding, and phlegmone of the pectoral wall) considerably grows due to necessity of frequent punctures.

The technique of drainage is as follow. Infiltration anaesthesia of the skin, hypodermic fat and tissues of intercostal area is conducted in the marked place. The collected device that consists of the long needle (not less than 20 cm) through the stylet of the trocar puncture of abscess is carried out. To give novocaine along movement of the needle through the lung tissue is undesirable because of danger of air embolism. The piston of the syringe is slightly pulled up to take the content of the cavity. Then, not drawing out the needle cut the skin and hypodermic cellular tissue of 1 cm long and a trocar is introduced following movement of the needle, keeping it from moving forward. The introduction of the trocar in the cavity of abscess is controlled by length of the needle or, that is more effective, by roentgen-television examination. The stylet and the needle are taken out, and through the shell of the trocar a two-lumen drainage pipe the diameter of which exactly corresponds the internal diameter of the thin-walled shell is introduced in the purulent cavity. Drainage is fixed by a stitch that will simultaneously pressurize a wound round a tube, and joins the vacuum device. After drainage, the cavity of abscess contrast examination is necessarily made for visualization of the drainage pipe, control of position of the lateral opening in it. If necessary the drainage is pulled up to such a level, at which the lateral opening is fully at the wall of abscess that allows emptying it in postural position. For determination of "draining bronchi", the so-called retrograde bronchography is made.

After aspiration 5 ml of 2.5% solution of trimecaine is introduced in the cavity of abscess, a patient takes postural position and coughs out the intro-

duced solution. Then they fill the cavity of abscess by the water-soluble contrast substance and by polypositional roentgenoscopy, the introduction of it in the "draining" bronchi is traced. Dilution in the aspiration system should not exceed 15–20 cm of water column. Sanation of the pulmonary abscess through the drainage consists in its permanent emptying and introduction of medicins. Complete emptying of abscess is achieved by a permanent vacuum-aspirator in small (5–10 cm of water column) dilution; if possible, a patient is constantly in the postural position. The control of amount and character of secretion is obligatory. In intense haemorrhagic secretion from the cavity of abscess, it is necessary to stop aspiration because of possibility of intense bleeding from the arrosive vessels of the abscess wall. In this case, the cavity of abscess is irrigated with solutions of haemostatic preparations (epsilonaminocaprone acid, dicinon); subsequent drainage is made in the passive mode.

After emptying of abscess, lavage of the cavity fractional or permanent, is made in drop irrigation through the thin lumen of the drainage. In fractional lavage the amount of antiseptic solution, which is introduced is chosen individually, not to exceed the volume of the cavity because of danger of aspiration in other parts of the lungs, especially in the weakened patients with suppression of cough reflex. If there is thick pus in the cavity, detritus and sequestra, proteolytic enzymes are added in the antiseptic solution in fractional lavage. In 3–5 days, when the cavity of abscess is almost cleared up from pus, the mode of permanent aspiration with 2–3 times introduction of antibiotics. It promotes reduction and obliteration of the cavity. The drainage needs careful examination with daily bandaging, and in inflammation of the soft tissues, that begins round drainage, infiltration by antibiotic solution is made.

The criteria of efficacy of drainage are complete emptying of abscess, absence of sequestra, reduction of the cavity size and reduction of signs of purulent intoxication. Renewal of drainage through the bronchus is confirmed by abscessography and serves as a sign for removal of the tube and suturing of the drainage opening. In considerable inflammation of the soft tissues it is better not to suture a wound round drainage.

Drainage operations in infectious destructions of the lungs include pneumotomy. The conditions of its performance are the same as in lung abscess drainage by Monaldi. However, indications to pneumotomy are limited today: it is made mainly in progressing gangrene, when radical operation is impossible due to severe state of the patient, and transthoracal drainage by a tube due to the presence of numerous cavities will fail in any case.

Indications for resection of the lungs in infectious destructions are:

1. Lung gangrene (extended and limited after maximally possible sanation of abscess). Stabilization of the patient's state, correction of basic indices of homeostasis, sanation of the focus of destruction in a preoperative period is the condition of successful result of the operation.

2. Complications of acute lung abscess are pulmonary bleeding, profuse hemoptysis with the threat of development of pulmonary bleeding (by vital indications).

3. Chronic lung abscess.

In extended gangrene of the lungs, pneumonectomy is performed, and in gangrenous and purulent abscesses, lobectomy is performed as a rule.

Bronchoectatic Disease

Bronchoectasy (from Greek. *ectas* — stretching) is a morphological notion that means stable pathological extension and deformation of the bronchi. Congenital bronchoectasies are consequence of anomalous development of not only the bronchial tree but also of respiratory parts of the lungs. They often combine with malformations of other systems and organs; therefore, they are a component part of characteristic complex of pathological changes, which develop because of disturbance of embryogenesis: cystic hypoplasia of the lungs, Sievert — Kartagener's syndrome (bronchoectases, pansinusitis, "mirror lung"), Tourpine's syndrome (bronchoectasies, expansion of the gullet, malformation of the vertebrae and ribs). Combination of congenital bronchoectasies with polycystic pancreas sometimes occur as well as cleft lip, deafness-and-dumbness, congenital pathologies of the heart.

Stable extensions of fine peripheral bronchi can develop because of pathological processes in the adjacent tissues (chronic abscess, fibrous-cavernous tuberculosis, chronic pneumonia). Such bronchoectasies are called recurrent, underlining that this pathological process is a consequence and component part of basic disease.

Bronchoectatic desease is an acquired disease that is characterized by chronic purulent inflammation, which affects all layers of the bronchial wall with the irreversible change of its structure, functions, and arises up as a rule, in the lower parts of the lungs. It is pathology of mainly children's age.

Unlike congenital bronchoectasies (cystic hypoplasias of the lungs), which are initially structural and functional defective area of the bronchi, that results in development of suppurative inflammation, in bronchoectatic desease the indicated impairments arise and develop for a long time while at the initial stages they may be reversible. At the late stages of develop-

ment of bronchoectatic desease, these differences do not have the principle value for determination of the medical management.

Some pathological states occupy intermediate position between congenital pathology of the bronchi and bronchoectatic desease. They include the Williams — Campbel's syndrome (congenital underdevelopment of cartilaginous structures and elastic tissues of the middle bronchi, as a rule bilateral, generalized). Conditioned by pathology of the bronchial wall, hypotonic diskinesia of the bronchi in this disease (expansion in inhalation and expressive stenosis in exhalation) quickly results in development of emphysema and fusyform generalized bronchoectasies, which are localized at the level of the middle bronchi. There is considerable congenital extension of the trachea and large bronchi in Mounier — Kuhn's syndrome (tracheobronchomegaly) that is accompanied by diskinesia of the trachea and bronchi, forming diverticulum-like pockets. The bronchi of 5–6 generation remain little changed for a long time. In the indicated pathological states, their heredofamiliar nature character is often observed when changes of the same type are found during few generations.

However, a genetically determined tendency to the origin of bronchoectasies can be not always realized, and only at presence of the appropriate conditions (pneumonias, bronchoadenitis, etc.). In this case a pyo-inflammatory process inevitably develops, the progress of which causes irreversible changes of structures of the bronchial wall and complete loss of functions that is formation of bronhoectasies. The presence of pyo-inflammatory changes in the bronchi, which predetermine the clinical picture, justifies the fact that bronchoectasies and bronchoectatic desease belong to the same group of the diseases etiologically related to the infection in chronic unspecific diseases of the bronchi.

Etiology and pathogenesis. The leading role in development of bronchoectatic desease has impairment of patency of the bronchi (middle and fine), that results in formation of obstructive atelectasis. Some patients have, as it was specified above, congenital tendency to collapse of walls of the bronchi (underdevelopment of the cartilaginous rings and smooth muscle fibres), as well as reduced activity of the surfactant, increased viscosity of sputum (in cystic fibrosis), with formation of thick mucous plugs which obturate the lumen of the bronchi. In children the pliable wall of the bronchi is quite often compressed by enlarged lymph nodes in bronhoadenitis (more frequently tuberculous one), pneumonias (especially measles), other respiratory infections caused by hyperplasia of lymphoid tissue. Bronchial secretion begins to accumulate below the place of obturation.

At the first stage of development of the disease, moderate extension of the lumen of fine bronchi is marked by mucus bronchial secretion that is

accumulated, without the change of the epithelium of walls of the bronchi. Transition of the pathological process in the second stage is manifested by purulent inflammation in the occlusion part of the bronchus. It occurs due to reduced efficacy of the protective mechanisms of mucous membrane of the respiratory parts of the lungs (alveolar macrophagi, immunoglobulin A), under the influence of viral infection, avitaminosis and nutrition disorder. The other cause of development of the suppurative process is considerable, but not complete obturation of the bronchi, that prevents full emptying of abscess (expiration stenosis), but allows to penetrate the infected content into the blocked segments of the bronchi. The purulent inflammation unavoidably involves the walls of the bronchi, in which there is marked metaplasia of cylinder epithelium and ulcers of the mucous membrane. In progress of the pathological process the inflammatory changes spread to the deeper layers of the bronchial wall, there is cicatrious regeneration of the smooth muscles and submucous layer. At this very stage the pathological process becomes irreversible because of loss of the contractive and "cleansing" function of the bronchi by the muscular elements of the bronchial wall. Even elimination of obstruction of the bronchi at this stage already cannot result in renewal of their function because of the muscular layer regeneration. Thus, a pathological circle is closed: impairment of patency of the bronchi — pyo-inflammatory process — dysfunction — progress of purulent inflammation — strengthening of drainage dysfunction, etc. At this stage even the most energetic conservative methods of treatment cannot tear this "vicious circle". However, they can promote reduction of intensity of purulent inflammation and thus slow down inevitable transition of the pathological process into the final stage.

At the third stage of development of bronchoectatic desease, the pathological changes are marked in all layers of the bronchial wall and spread beyond the bronchial tree. The bronchi become sharply extended and contain purulent or purulent ichorous exudates in the lumen with unpleasant putrid odor. The cartilaginous frame of the bronchi undergoes regeneration, the connective tissue and the whole "fields" of granulation tissue appear at the places of mucous membrane, covered with ulcers. Such impairment of the structure of bronchi results in their reduced resistance and to the action of the so-called "bronchodilatation" forces, i. e. increase in pressure of the bronchial tree during cough, stretching of them by sputum, which is accumulated and in the end predetermines formation of sac-like expansions of the areas of the bronchial tree. The pathological process unavoidably involves peribronchial tissues (sclerosis of the peribronchial cellular tissue, hypertrophy of the bronchial arteries, bronchoadenitis) and pulmonary parenchyma (foci of pneumonia, pneumofibrosis). During angiography, the lumen of

the bronchial arteries increases 4–5 times, the amount of arterio-arterial anastomosis and width of their lumen increases, that results in the considerable release of arterial blood from the systemic circulation into the system of the pulmonary artery, there is pulmonary hypertension, and a "pulmonary" heart is formed afterwards. Thus, in the final stages of development of bronchoectatic desease the pathological changes do not differ from those in congenital pathology of the bronchial tree (cyctic hypoplasia), when there are also bronchoectasies in which a chronic purulent process is supported and a pulmonary parenchyma is structurally and functionally incomplete. The long existence of the purulent process in the lungs results in the origin and progress of systemic affections: formation of chronic pulmonary-cardiac insufficiency, diffuse chronic purulent bronchitis, emphysema of the lungs, dystrophy of the parenchimatous organs and anaemia.

The described mechanism of development of the bronchoectatic desease is not limited by all variety of pathology. Quite often bronchoectatic desease with formation of bronchoectasies in one lung is combined with development of purulent bronchitis in another one and formation of the obstructive syndrome. Some patients have a considerable pronounced expiration stenosis of the large bronchial tubes and trachea, the symptoms of bronchial asthma predominate in the clinical manifeststions of the disease. The bronchoectatic desease quite often develops against a background of primary tuberculosis of the lungs and bronchoadenitis related to it (post-tuberculous bronchoectasies).

Classification. A generally accepted classification of brochoectatic desease is not developed until now. The most acceptable classification was offered by. A. Beysebaev et al. in 1982.

I. According to the origin:
   1. Congenital (including those combined malformations — the syndrome of Sievert — Kartagener).
   2. Acquired (in bronchoectatic desease).

II. According to affection of structures of the lungs:
   1. With prevailing affection of the pulmonary parenchyma.
   2. With prevailing affection of the bronchi.

III. According to the form of bronchoectasies:
   1. Cylinder.
   2. Sac.
   3. Fusiform.
   4. Mixed.

IV. According to the clinical course:
1. Remission.
2. Exacerbation.
3. Continuous recurrent course.

V. According to the presence of complications:
1. Uncomplicated course.
2. Complicated course:
— by pulmonary bleeding;
— by hemoptysis;
— pyopneumothorax;
— abscess formation.

VI. According to the state of the function of the external breathing:
1. Without respiratory insufficiency.
2. Respiratory insufficiency of I, II and III stages.
3. Pulmonary-cardiac insufficiency.

It is necessary to make full diagnosis of bronchoectatic desease taking into account all signs listed in the classification, with localization and prevalence of the pathological process.

Clinical course and diagnosis. The clinical picture of bronchoectatic desease is various and depends on prevalence of the pathological process, stages of its development (including transformation of the inflammatory changes involving pulmonary parenchyma), phases (exacerbation or remission), and presence of complications (pulmonary bleeding, pyopneumothorax) that determines the treatment management.

As bronchoectatic desease is pathology of the children's age, adult patients with this pathology are rarely seen in the surgical department. As a rule, they ask for medical aid because of the complications development or due to chronic bronchitis, chronic pneumonia, and suspicion on tuberculosis of the lungs. It is related to the same type of basic clinical manifestations of bronchoectatic desease and afore-mentioned diseases. They include: cough with sputum, amount of which and character can change depending on the season, episodes of increased temperature of the body, chest pain, hemoptysis. The signs of chronic purulent intoxication can develop in prolonged course of the desease (untimely diagnosis): asthenisation, deformation of the fingers (as drumsticks) and nail plates (as "watch glass").

The basic complaint of patients, which brings them to the doctor, is cough. As a rule, it is permanent, productive, increases during overcooling, especially at the height of acute respiratory-viral diseases in the autumn-spring period. The amount of sputum can be considerable — up to 200–

300 ml per day, its character is mainly purulent in the period of exacerbation and mucopurulent with a rather yellow tint. Most of sputum is coughed off in the morning ("by a complete mouth") and less — during daytime or is increased on physical exertion and in the postrural position. There may be admixtures of blood in the sputum that is also a frequent cause of coming of the patient to the doctor.

Quite often there is shortness of breath on physical exertion, that is evidence of considerable involvement of the pulmonary parenchyma in the pathological process. The increased body temperature is often accompanied by the increase of amount of coughing off purulent sputum that is usually observed during exerbation of the disease. Carefully taken anamnesis of the disease is of importance for its recognition and determination of causal factors of the development of bronchoectatic desease (pneumonias, whooping cough, measles, and tuberculosis). It is necessary a purposeful cross-questioning of patient's parents, who can remember the beginning of development of chronic pulmonary pathology (chronic pneumonia, chronic bronchitis) even in the early child's age. It is necessary to ask about systems and organs, which quite often allows revealing the congenital character of pathology of other organs (failure of the heart, maldevelopment of the skeleton, etc.).

It is possible to reveal during examinations of patient: signs of deformation of the thorax that are evidence of long-term disease (sagged intercostals spaces, scoliosis), lag of one of halves of the thorax in breathing (massiveness of the pleura adhesions), cyanosis, signs of chronic purulent intoxication, deformation of the fingers and nails, sallow color of the skin, asthenisation. However, absence of the enumerated symptoms does not eliminate the presence of bronchoectasies. During the physical examination there may be tenderness of intercostal space on palpation (especially durig exacerbation), that is evidence of affection of the visceral and parietal pleura, and on percussion — a dull percussion sound in the projection of the affected parts of the lungs (atelectasis, pneumofibrosis). In the same parts, diminished breath sounds are heard, dry rough and moist rales the amount of which dicreases after coughing. The limitation of mobility of the pulmonary edge, related to massive pleurodesis in the basal parts of the pleural cavity, often develops in long-term course of the disease. Bronchography is still the only method of reliable verification of the diagnosis. To determine exactly prevalence of the process, it is necessary to make bronchograms for all parts of bronchial tree without exception with previous careful sanation of it and elimination of acute pyo-inflammatory process, as presence of viscid sputum and pus in the bronchi does not allow contrasting the bronchial tree fully and hampering interpretation of the bronchogram.

The modern technical equipment allows making cinematobronchography (videobronchography), which estimates dysfunction of the bronchi more exactly, rate of contrasting of substance eliminaton from the bronchi, place of its retention. Computer tomography has a high ability in revealling not only the affected bronchi but also the surrounding pulmonary tissue. The ordinary methods of roentgenologic research (sciagraphy in standard projections, tomography) also give valuable information that allows to suspect the presence of bronchoectasias and also judge about the functional state of the pulmonary parenchyma and determine the order of bronchography performing for different parts of the bronchial tree. Cluster extensions of the bronchi in cystic hypoplasia of the lungs can be noticed on survey sciagrams and tomogramas. Reduction of volume of the affected part or of the whole lung is a frequent roentgenologic sign of the bronchoectatic desease; compensate emphysema of the "intact" part, displacement of the mediastinum and interlobular fissures because of the change of architectonics of the bronchial tree.

For determination of the function of the affected parts of the lungs (pulmonary blood stream) angiopulmonography is used, and perfusion scanning. Bronchoscopy is of substantial diagnostic value.

In determination of medical managememt, the results of cytologic research and biopsy of the bronchial tubes are taken into account (degree of metaplasia of the bronchial epithelium, expressiveness of the inflammatory changes of the wall of the bronchi). The bronhoscopic signs of endobronchitis allow to judge about sanation of the affected focus quality in detail and to suspect the congenital character of the bronchial pathology (deformation of the bronchial tree, tracheo-bronchomegaly, etc.).

It is necessary to make a *differential diagnosis* with brocnhoectatic desease, bronhoectasis as manifestations of other pathological processes — chronic bronchitis, tuberculosis; by bronchoectasis in congenital pathology — cystic hypoplasia, tracheobronchomegaly, syndrome of Sievert — Kartagener, etc.

Treatment. The presence of bronchoectasias is extension of the bronchi that is accompanied by the chronic purulent inflammatory process in them. It requires immediate estimation of possibility and necessity of radical operation — removal of the pathological focus. It is necessary to take into account that the prolonged presence of purulent inflammation unavoidably results in spread of the pathological process to all parts of the bronchial tree (diffuse chronic bronchitis), respiratory parts of the lungs (chronic pneumonia, pneumofibrosis, emphysema), and then acquires expressive systemic action (dystrophy of parenchimatous organs, amiloidosis).

Surgery is contraindicated to patients with the I–II stage of bronchoectatic desease, if the prolonged and skilled conservative treatment can

"tear" the pathological circle that is formed, and to result in long-term remission and in some patients — to convalescence. This treatment includes intensive sanation of the bronchi (postural drainage, medicines, which facilitate discharge of sputum, inhalations sanation, bronchoscopy), antibacterial therapy according to indications (high fever, pneumonia, abcess formation). With prounced manifestations of purulent intoxication, the target infusion, substitutional and detoxicative therapy is conducted. Syrgery is impossible in patients with the widespread (total) character of affection of the bronchial tree and of little use in presence of accompanying obstructive bronchitis with strong respiratory insufficiency, "pulmonary" heart. Thus, its localization form is an indication to surgical treatment of bronchoectatic desease, that is if the function of remote parts of the lungs is compensated by the sufficient volume of pulmonary parenchyma, that was saved. The operation is possible even in bilateral localized bronchoectasyas (simultaneous resection or successive operations with some interval of time) ander condition of saving sufficient volume (not less than 8–10 segments) of the pulmonary tissue.

The age of 7–14 years is most acceptable to operative treatment, when there is a possibility exactly to determine the extent of affection of the bronchial tree and the risk of relapse due to the postoperative changes of architectonics of the operated lung diminishes. As a rule, in bronchoectasias, which are accompanied by the great changes of the pulmonary parenchyma, the resections of segments, a lobe are performed, and in the diffuse (but unilateral) character of the pathological process — pneumonectomy.

In localization of bronchoectasias in segmental and subsegmental bronchi and in absence of changes, the isolated resections of bronchi with preservation of the pulmonary parenchyma can be executed. The later experiences more or less pronounced pneumofibrous changes, however, the postoperative period is favourable, there are fewer changes in architectonics of the bronchial tree of the operated lung (and consequently, the risk of relapse of bronchoectatic desease and development of pulmonary hypertension decreases, especially in early children's age).

Conservative treatment in some patients, whom operation is not indicated, is basic, and in patients who need operative treatment it is preparatory.

Skilled clinical supervision, sanatorium-spa treatment increase of efficacy of treatment.

Lecture 2
LUNG CANCER

Lung cancer is a malignant tumor arising from the epithelium of the bronchial mucosa, its submucous layer, bronchioles and bronchial glands.

First, the casuistic description of lung cancer was made in 1805 and belonged to a French researcher Bale. In 30 years — in 1837 — Laénnec made a more detailed description of clinical development of this still rare desease of that time. In Russia, the most significant early works about lung cancer were the dissertations of A. Ebermann (1857) and pathologist G. V. Shora (1903). That time there were some systematization and enumeration of observations after the patients suffering from lung cancer which were small in numbers.

Complete absence of an opportunity not only to help but also to reveal timely this disease before occurrence of late signs, determined constrained interest of the doctors-clinicians to lung cancer at that time. Pathologists mainly did the description of such observations. Besides, the number of the patients was very limited, and cases of the disease occur very rarely at that time. Until the end of the first decades of the XIX century about 400 observations of lung cancer were published in the world literature, among which the reports made by pathologists prevailed.

Lately cases and mortality rate from lung cancer have increased everywhere ten times. It is especially marked in the industrially developed countries, where morbidity rate of lung cancer has got a social disaster. Therefore, in Great Britain the number of patients with lung cancer has increased more than 40 times for the last fifty years. The same situation is marked in other countries (Poland, Norway, Sweden and Japan). The high morbidity rate of lung cancer is constantly recorded in the Baltic countries, Moldova, Transcaucasia. Up to 100,000 men fall ill with lung cancer annually in Russia.

Etiology and pathogenesis. Search for causes of development of cancer has old history. The search of mechanisms bringing about malignant trans-

formation of tissue cells and organs of the human organism proceeds now.

The important step in research of the causes of development of tumors was formation and development of experimental oncology. This section of science began developing since the middle of the last century. Then the intensive researches on morphology of malignant tumors were carried out by a professor M. M. Rudnyev at the department of Pathological Anatomy of the Medico-Surgical academy in St.-Petersburg. He was one of the first who raised the question that cancer is a tumor of the epithelial origin, contrary to the used opinion of the foreign morphologists led by R. Virchov about an opportunity of development of cancer from the connective tissue. That is why M. M. Rudnyev began to learn histogenesis of malignant tumors under the experimental conditions. Under his guidence, a researcher M. A. Novinsky was engaged in a question of transplantation of tumors in the veterinary department of the Academy. The successful solutions of this problem have made M. A. Novinsky an ancestor of experimental oncology. In 1876 he defended the dissertation "To a question of inoculation of malignant neoplasm" and for the first time in the world the opportunity of inoculation of tumors to animals was proved. He succeeded in transplanting a tumor taken from an adult dog to a pup. The transplanted tumor not only successfully grew; it could be transplanted to other dogs. The microscopic research of these tumors established their complete identity with the initial one. Researches of M. A. Novinsky were a basis of creation of the first experimental models of tumoral growth; they are widely used now in all experimental oncologic laboratories of the world, being the conventional model in oncologic researches.

The assumptions that oncologic diseases can be caused by various factors, were made at the end of the 19th century. For example, an outstanding Russian surgeon N. A. Velyaminov was of the same opinion. The founder of native oncology N. N. Petrov also considered that development of malignant tumors could be caused by several etiological factors, not by one specific agent. The immunological theory of tumor development claims to be universal. It is considered that disorder of immune protection or its deficiency may be connected with a series of factors, among which the leading place belongs to the immunodepressive action of various chemical, physical carcinogens and some viruses. Immunodepression can become a result of such action, which is the universal mechanism realizing development of a malignant tumor in an organism.

The hormonal background of an organism exerts a marked influence on arising rate of tumors, their development in the latent period and biological properties.

The data, which have been collected until present time, give evidence that malignant transformation of normal cells can result from the numerous external and internal causes. One of leading places among them is a problem of healthy way of life, including its ecological aspects.

Smoking is a generally accepted and leading factor in development of lung cancer. The combination of smoking and staying in the polluted enviroment most frequently results in development of this disease. The tobacco tar represents the greatest danger to the smoking people. The structure of tobacco tar includes various substances and resins capable of causing development of a malignant tumor. There is carcinogenic hydrocarbon benzpyren among them, which contents in smoking of 1000 cigarettes is quite enough for development of a malignant tumor in a laboratory animal, and a man smoking a pack of cigarettes per day, passes through the lung about 800 g of tobacco tar for one year. The degree of smoking of cigarettes is also of significance. It was revealed that especially many (three times as much) carcinogenic substances are accumulated in the last third of the cigarette in its smoking. In smoking of the cigarette up to the end, the greatest amount of combustion materials of tobacco gets in the respiratory tracts. The efficacy of tobacco filters has appeared low, and the efforts to make a "harmless" cigarette failed. Besides, the American scientists isolated a radioactive isotope polonium-210 with the large period of half-life from tobacco. In smoking, it passes into tobacco smoke and stays in an organism for a long time. A heavy smoker receives a general radiation dose of an organism sufficient to cause changes in the chromosomal apparatus of tissue cells of the lungs, resulting in their cancer degeneration.

The changes which take place in the mucous membrane of the bronchi in smokings are investigated now in details. The normal mucosa of the bronchi has a two-layer cylindrical ciliated epithelium. Movement (fibrillations) of the epithelial cilia carry out the important function of self-cleaning of the bronchial tree. Due to constant fibrillation of the cilia of the cylindrical epithelium, small dust particles and drops of mucus can be displaced at the distance of 1 meter within one hour. Therefore, normal functioning of the epithelial integument of the mucous membrane of the bronchi provides rather reliable physiological cleaning of deep parts of the lungs.

The daily and repeated influence of the tobacco smoke on these gentle structures naturally results in reorganization of the bronchial epithelium. The function of the ciliary epithelium appears insufficient to counteract the amount of the tobacco smoke, temperature and chemical influences. Not only superficial, but also deeper layers of the bronchial wall are damaged, that results in protective reorganization of the epithelium: the cilia gradually disappear, the number of the cell layers is enlarged and the cells themselves

get plant and multiangled form. The cylindrical epithelium is transformed into the multilayered squamous one which is more stable to adverse influences.

The foci of the squamous bronchial epithelium can constantly be found in the mucosa of the bronchi of heavy smokers (L. M. Shabard). It partly explains the fact that majority of the patients suffering from lung cancer have epidermoid cancer, while in norm the squamous epithelium is absent in the bronchial mucosa.

The cancerogenic action of the tobacco smoke is rather malignant for non-smoking people. According to the data of an American researcher Everet Koop, published at the end of the 80s, about 23 thousand people die from lung cancer annually due to "passive smoking".

It was established that in families where men smoke in inhabited rooms, their nonsmoking wives fall ill with lung cancer much more often in comparison with families without this harmful habit.

Smoke of the cigarette left unextinguished in the ashtray is more harmful. It contains carbon dioxide, benzpyren, nitrozamines, heavy metals, nicotine in large concentration. A man with a cigarette inhales approximately a fourth part of cancerogenic substances, formed in it, and the rest of their volume is dissolved in the air and acts on the lungs of people nearby.

A man smoking 20–30 cigarettes daily within 25 years has probability of falling ill with lung cancer 22 times as much in comparison with non-smokers. Heavy smokers who smoke two packs of cigarettes per day have chances to fall ill with lung cancer 24 times higher. Such intensive smoking for over 20 years, especially started in childhood since 12–14 years old raises the risk of falling ill with lung cancer 52 times as much.

The examination of 187 thousand of men smoking tobacco, which has been carried out by a certain program, revealed that mortality from lung cancer among them is 20 times as much as in non-smokers (USA).

Various diseases capable of causing different changes in the tracheobronchial tree and disturbing normal processes of self-cleansing, removing pathological admixtures may be considered as additional adverse factors promoting long-term action of carcinogenic substances and development of lung cancer. It is possible to find out consequences of various inflammatory diseases in the past, long-term chronic bronchitis, pneumonia in many people who fell ill with lung cancer. Frequent diseases, such as influenza with pulmonary complications, especially in smokers, should be conditionally related to precancerous condition. Such repeated inflammatory processes developing in the organs of respiration, not only result in damage of the

ciliated epithelium of the bronchi, but also weaken both local and general resistance of an organism concerning influence of the adverse factors of environment. In chronic bronchitis, which as a rule is accompanied by disorder of the drainage function of the bronchi, there is retention of the bronchial secretion more often in the deepest and least ventilated areas of the lungs. It results in accumulation of carcinogenic substances, their longer influence on the bronchial mucosa. Apparently, it can explain development of lung cancer in man mainly in the upper lobes and more often in segmental bronchi especially those coming from the lobes at acute angle and obstructing easily with difficulties of outflow of the inflammatory or mechanical origin. Chronic pneumonia results in reorganization of the bronchial epithelium, similar in structure to a malignant degeneration. The consequences of pulmonary tuberculosis also cause changes of the tissue structure of an organ with functional and organic reorganizations of the mucosa of the bronchi, until its transformation into cancer.

Lung cancer is a disease of the second half of life. The overwhelming majority of patients are people of the elder age group. Such fact is explaned that constantly working adverse factors require long time, sometimes tens of years to be realized as a tumour. Probably, some protective mechanisms of a human organism, including organs of respiration become limited and are partially lost with years and change of way of life style.

The men are mainly fall ill with lung cancer. The correlation between the number of ill men and women is steadily kept in proportions 1:4–1:7.

The reason of it consists in the fact that mainly men work at harmful manufactures all over the world and have a habit of smoking.

Thus, the information taken from etiology and research of relationships of cause-and-effect development of lung cancer, gives enough bases for distinguishing a specific “group of risk” concerning development of this disease, meaning such criteria, as age and sex, long-term work of harmful manufactures, smoking for a long time, previous deseases or frequently recurrent catarrhal and other inflammatory diseases of the organs of respiration.

The research of lung cancer morbidity has shown that depending on the degree of its differentiation and histological structure for the period from the moment of development of tumour to its size of 1 cm takes a rather long period. For small cell carcinoma it makes 2.4 years, for adenocarcinoma — 7.2 years, for squamous cancer — 13.2 years. The further growth of a tumour occurs in a geometrical progression and is mainly determined by pathogenesis of the disease.

There are three periods of lung cancer development:

I — a so-called biological, covering time from the beginning of development of a tumour to its first morphological signs;

II — preclinical, or asymptomatic;

III — clinical, characterized not only by pronounced morphological signs of the disease, but also by various clinical signs caused by disfunction of the affected lung.

Long asymptomatic development of the tumour in the lung, covering I and II periods, is associated with peculiarities of the structure of the pulmonary tissue. Germination of the tumour in the wall of the bronchus and its destruction results in blood in sputum. The blocking of the bronchial lumen by the tumour is accompanied by hypoventilation of the corresponding site of the pulmonary parenchyma with development of inflammatory process in this area. Then there are signs, characteristic of pneumonia. Blastomatous changes involve the main bronchus resulting in exclusion of large areas and even the whole lung from gas exchange; it is accompanied by compensatory changes of respiration, cardiac activity. The transfer of the tumour or development of its metastases in the mediastinum can be accompanied by compression of the upper caval vein, esophagus and large nervous trunks located here (n. vagus, diaphragmatic nerve) with development of corresponding disorders.

The certain attention to clinical signs and the course of the disease renders the character of tumour innidiation in the organs posed outside the thoracal cavity and feature of tumour structure.

Classification. Two criteria are fixed in the basis of existing classifications of lung cancer: histomorphological and topographo-anatomical. They allocate three basic histological types of lung cancer. Each of them is shaped and occurs from various elements of bronchial epithelium: squamous cell carcinoma (planocellular) — from covering epithelium, adenocarcinoma — from glandular cells, nondifferentiated — from basal cells. Most convenient for clinical practice became histological classification developed by academician N. A. Karayevsky and his colleagues in 70s years. According to it (a) planocellular cancer (with keratinization and without), (b) adenocarcinoma (high-differentiated, moderate-differentiated), (c) undifferentiated cancer (microcellular, macrocellular, small-differentiated with elements of planocellular, glandular, with cancer-like structures) are distinguished.

The histological classification of nondifferentiated and glandular forms of lung cancer is being developed now. Dimorphous and trimorphous forms of growth, separate subtypes of undifferentiated cancer are distiguished. However, in opinion of many researchers, now such allocation has no practical importance.

The original features of clinical development are noticed for each histological type of lung cancer. Planocellular cancer grows rather slowly and to the less extent belongs to an early and extensive innidiation. Adenocarcinoma develops rather slowly, but differs by early hematogenous dissimination. Undifferentiated cancer, especially microcellular, is characterized by fast development, high degree of malignancy, rough and extensive innidiation. In this type it is quite often possible to note a backlog in growth of primary tumour from quickly growing metastases. Infiltrating, peribronchial and perivasal growths are frequently observed.

The majority of modern clinical and pathomorphologic classifications divide lung cancer into central and peripheric. It is not easy to differentiate them, nevertheless clinic and morphology of the precisely outlined forms of this tumour is very characteristic and quite justifies such division completely, which is important in practice.

Peripheric cancer begins its development from the epithelium of bronchioles or the most distal subsegmental and terminal departments of bronchial tree branching. Here, on periphery of the lung, such tumour freely spreads to all directions, acquiring a spherical or ball-shaped form. For a long time it does not destroy bronchial patency, does not notably influence the pneumaticy of lung departments, directly involved in the blastomatous process.

Central lung cancer arises from the epithelium of the mucous integument of large — segmental or lobar bronchi. Such a tumour, even having not reached large sizes in early terms, obturates or squeezes the bronchial lumen, destroying its patency. It results in hypoventilation and atelectasis of the appropriate department of the lung: its segment, a group of segments or a lobe. Characteristic changes of a pyramid or a wedge shape inverted to the center of the organ appear in a growth zone of the central tumour.

Location of a tumour at various distances from the bronchial tree, its development with or without involving various structures of the lungs (pleura, large bronchi, blood vessels etc.) determines features of the clinical picture, diagnostic and medical, especially surgical management of the disease. So, central lung cancer with a pronounced endobronchial type of growth results in a rapid blockage of one or another site of airways, hypoventilation, atelectasis and inflammation of the appropriate lung department. Paracancrosic destruction of the tissue develops in it quite often. The peribronchial type of development of central cancer damages the airways at the great extent, but quite often it does not cause disorders of gas exchange for a long time. Peripheric cancer, spreading more or less even in the parenchyme of the lung, gets spherical form, rather early infilt-

rates visceral pleura and goes beyond the limits of the lungs, infiltrating the thoracal wall, diaphragm and other anatomic structures located nearby.

Lung cancer spreads by lymphatic ways early and widely. The lymphogenous metastases of tumour damage the regional lymph nodes with the same frequency — both in central, and peripheric lung cancers. Up to the moment of the reference of the patients for help such metastases already exist approximately in a half of them.

Lymphogenous innidiation occurs mostly in accordance with natural, physiological movement of lymph from each lobe. The lymph nodes damaged by a tumour are involved in pathological process stage by stage. At the beginning the intrapulmonic regional lymph nodes, then — posed in the field of the root of the lung, further — in the mediastinum, on the same side with the lung.

In the later period the metastases spread to the groups of lymph nodes of the opposite part of the mediastinum, and with an ascending lymph flow — in supraclavicular lymphatic collectors. The mediastinal lymph nodes are basic receiving lymph from lung, and a part of lymphatic vessels only here meets these original biological filters for the first time. Within the limits of the mediastinum each of lung lobes has own regional groups of lymph nodes. It is bifurcation and periesophagial for the lower parts, for middle lobe and lingular segments of the left lung — bifurcation, prepericardial, tracheobroncheal, for the upper lobes — tracheobroncheal, paratracheal on the right, preaortocarotid and tracheobroncheal on the left.

Only at late stages of the disease, when innidiation causes disorganization of the lymph stream from the lung, the collateral and transversal lymphatic ways open, innidiation of lung cancer becomes asymptomatic, retrograde, and contralateral. As a rule, at this time, hematogenous metastases in various areas of a human organism with formation of new growth foci of a tumour frequently are also found out. Most frequently such foci arise in the liver, the kidneys, the adrenal glands, the brain, the bones of the skeleton.

The conception about stages of lung cancer development is relative. It can vary during inspection of the patients and reception of additional information. It is not always possible to separate different stages of the disease clearly, because of the variety of tumoral growth signs in each of the analyzed symptoms and in general estimation of several of them.

The greatest spread in our country has two classifications of lung cancer: clinical (1956) and international — recommended by the International Anticancerogenic Union, which is periodically reconsidered and supplemented. In the international classification separate criteria describing malignant growth of a tumour are designated by symbols *T, N, M* and numbers (Table).

Table. **Classifications of lung cancer according to disease development stages**

Characteristics of changes in lung cancer	Clinical classification	International classification
Tumour up to 3 cm in diameter, there are no metastasizes in lymph nodes	I stage	T1N0M0
Tumour 3.1–5 cm in diameter, metastases are in lymph nodes of lobes, single metastases are in root lymph nodes	II stage	T1N1M1 T2N0M0 T2N1M0
Tumour 5.1 cm and more, can spread to adjacent anatomic formations, multiple metastases in lymph nodes of the root, metastases in lymph nodes of the mediastinum	III stage	T1N2M0 T2N2M0 T3N0M0 T3N1M0 T3N2M0
A large tumour spreading to the ajacent organs, multiple metastases in lymph nodes of the mediastinum of the own and opposite site, dissimination throughout the pleura, hematogenous spread to organs	IV stage	T4N1M0 T4N2M0 T4N2M1 T4N2M1 T4N2M1

More exact definition of the stage of lung cancer development is possible only during an operation and after morphological research of the received material. In these cases in designation of each symbol of the international classification an additional designation — "p" should be written before them; pTNM, that means the presence of histological confirmation of parameters of criteria.

Clinical course. Changes and disorders arising in patients with lung cancer manifest themselves in a different way, depending on the stage of tumour development. Long absence of any significant disorder of health is the most typical during the course of the disease. Such situation corresponds to so-called biological and preclinical (or asymptomatic) periods of malignant tumour development. The subsequent course of the disease with the development of lung cancer is accompanied by the appearance and increase of various signs and is called the period of clinical manifestations. The first precursory symptoms of the disease usually disturb patients a little, do not attract their attention, as do not break everyday life and, as a rule, do not become an occasion for the reference to the doctor.

Purposeful study of diverse signs of lung cancer, undertaken by an academician A. I. Savitsky, and disclosing of the concept "the latent course of disease", has allowed to determine most typical syndromes arising in lung cancer,

and to designate them with original "masks" of disease. The study of clinic of lung cancer together with these data has shown that a true "latent period" in the pathogenesis of the disease occurs much less often than it is considered. "Masks" of clinical course of lung cancer differ depending on the stage of the disease. Decrease of work capacity, increased fatigue, and decrease of interest to occurring events, so a circle of sensations quite often is united by the concept "decrease of a vital tonus" belong to the number of such precursory symptoms.

In the majority of the patients lung cancer (especially central) is hidden under the "mask" of various acute or chronic respiratory diseases: bronchites, catarrh of the upper respiratory ways, pneumonia, repeated episodes of influenza and others. As a rule, at the profound and purposeful inspection of the patient in these cases it is already possible to determine signs of accompanying nonspecific paracancrosic pneumonia. At this time temperature of the body periodically increases to subfebrile numbers, a light malaise arises and soon passes. As a rule, after taking antipyretic and anti-inflammatory drugs or even without medicamental treatment at home conditions all these phenomena soon pass and consequently do not become an occasion for the reference for medical help. The alarm usually is caused by the frequent repetition of such malaises during short — 1–1.5 months — period of time. It is considered that such nonspecific manifestations of pulmonary cancer last on the average up to 8–10 months.

Cough is considered to be one of the main signs of lung cancer. At the beginning it is dry, from time to time — becomes hoarse. Later it is accompanied by excretion of mucous or mucopurulent sputum. With time the cough gets more constant, excruciating and hoarse and then becomes the main complaint of the patients. The reason of cough more often is involvement of the bronchial wall of moderate and larger calibre in the blastomatous process, their stenosis or obturation by a tumour.

Haemoptysis or appearance of blood in sputum is caused by destruction of blood vessels' wall of the bronchus. These changes always cause an alarm in the patients and, as a rule, force them to ask for help. However, more often this sign, which the patients specify as the first sign of health disorder, is not an early display of the illness and occurs in the majority of the patients already in II and III stage of tumour development. Sometimes "an authentic sign of lung cancer" is accepted as a form of sputum reminding "raspbarry jelly", which is especially not the early sign and more often corresponds to IV stage of lung cancer.

Stethalgias is a sign usually arising in the part of the lung damaged by a tumour. It is various in intensity. As a rule, appearance of pains is connected with involvement in the process of the parietal pleura, intrathoracic fascia, inter-

costal nerves and ribs. At the beginning these pains are short-term, transient, but with tumour progressing they become constant, depriving patients of rest even at night. Location of a tumour in the apical segment of the superior lobe of the lung and its transition to the pleura, and then on nerves of a humeral plexus and the trunk of sympathic nerve causes especially excruciating pains which are in this case one of a sign complex, pathognomonic for peripheric cancer, such as Pancoast's tumour, for which the following combination is characteristic: superlobar localization, spherical form, plexitis, Horner's sign.

Dyspnea, steadily increasing respiratory discomfort, feeling of incompleteness of inspiration and shortage of air, especially at movement or arising the stairs, and in later period of tumour development these complaints are present even in the state of rest, increase with time because of atelectasis of many departments or all lung, accumulation of pleural exudate are characteristic at this time. General weakness, adynamia, as a rule, accompanies other local displays of lung cancer and is characteristic for late stages of disease development. In some patients products of tumoral metabolism, the various functional disorders of the endocrine glands of an internal secretion (adrenal glands, thyroid, gonads) result in original clinical changes and appearance of special "masks" of the disease, but some of them have got their own names: Pierre Marie-Bamberger's syndrome — excruciating pains in large joints of extremities, their pronounced tumescence, phenomenon of ossifying periostitis in the field of tibial bones; the Schwartz — Bartter's syndrome — production of tumoral, with small cells structure, hormone-like polypeptide, which secretion is accompanied by pronounced muscular weakness, hyperosmolarity of urine due to increased excretion of sodium, steady hyponatremia, delay of extra- and intracellular liquid, mental disorders as excitement or opression with various neurologic signs.

The Lambert — Eaton's syndrome appears at low-differentiated lung cancer. It is characterized by muscular weakness similar to myasthenia, but arising mainly in proximal departments of the inferior extremities function, pelvic organs disorder. It is characterised by the sign of "involving into work": if with myasthenia the proceeding muscular efforts result in complete loss of forces, at a Lambert — Eaton's syndrome the repetition of movements is accompanied by complete restoration of the lost muscular force. Lambert — Eaton's syndrome frequently arises 3–6 months before appearance of other signs of pulmonary cancer.

Cushing's syndrome, which is accompanied by increased secretion of serotonin, adrenocorticotropic and antidiuretic hormones, calcitonin and series of others is also added to the number of paraneoplastic synd-

romes, lung cancer "masks". The correct interpretation of various paraneoplastic syndromes is important for revealing a true nature of the disease — a malignant tumour of the lung behind these "masks" — and frequently allows to avoid gross mistakes in explanation of clinical picture of the disease.

The involvment of anatomic formations of the mediastinum results in appearance of rather original clinical signs in patients with lung cancer. So, prelum or germination of the esophagus is accompanied by dysphagia. The involvement of recurrent branches of vagus nerves (inferior laryngeal) results in wheezing of the voice. The prelum of the mediastinum by the lymph nodes damaged by metastases of cancer, the superior caval vein of the mediastinum is accompanied by disorder of bloodflow to the right departments of heart, congestion with edema of the face, neck, upper part of the trunk and formation of roundabout flow of the venous blood in the extended hypodermic veins of a frontal surface of the breast (syndrome of the superior caval vein).

The hematogenous metastases of lung cancer to the brain, the liver, the kidneys, the bones of skeleton are accompanied by appearance and development of clinical signs inherent to the disorder of appropriate organs and anatomic structures. Quite often these disorders become the first cause for visiting a doctor at apparent complete well-being of respiratory organs.

Natural (without treatment) course of lung cancer rather quickly ends with lethal outcome. According to the data of academician I. S. Kolesnikov, among the patients those who for different reasons did not receive treatment, only 52% could live more than 1 year, 3.4% of patient lived for three years, and less than 1% — 5 years.

A histological structure of lung cancer has essential influence on life span in these cases: 48% of patient with planocellular, high-differentiated cancer lived for more than 6 months; and with undifferentiated cancer of the same structure — only 12%.

Diagnosis. The efficiency of medical help to patient with lung cancer is determined by duly diagnosis of the disease and it can be the most productive at the early, initial period of development, at I stage of the disease (T1N0M0), which is almost often asymptomatic, and the absence of essential disorders of health in the patients does not induce them to the reference for medical care. Such a tumour can be revealed only with use of modern roentgenologic methods of inspection, so mass purposeful dispensary inspections of the population, especially of those selected in the group of increased risk concerning lung cancer is very essential. The leading place in efficiency of such inspections and diagnosis of initial attributes of lung cancer belongs to a large picture frame photoroentgenography of the thoracal organs allowing to reveal preclinical signs of the tumour.

Preventive photoroentgenographic inspection is annually carried out by healthy population and by everybody, addressed in polyclinic concerning various diseases of the respiratory organs, selected in the group of risk of lung cancer. At such organization of diagnostic work it is possible to reveal lung cancer on the average in 2–3 men out of 10,000 prophylactic examination and in 38–40 out of 10,000 those addressed for medical assistance in polyclinics.

Notable increase of diagnostic opportunities in detection of lung cancer at dispensary inspections became possible with formation of special advisory commissions. Roentgenologist, phthisiologist and oncologist must take part in the work of the comission. On the joint basis the members of the commission carry out the analysis of photofluorograms and other results of patients' examination, in which pathological changes in the lung were revealed for the first time. If it is necessary, such patients are immediately sent for the profound inspection and specific diagnosis in special clinics. Such organization of diagnostic work allows to reveal lung cancer at early (I–II) stages of the disease. Nevertheless according to the data of numerous oncologic symposiums and congresses, despite of successes achieved in this field, the number of patients in the initial stage of the disease at prophylactic inspection, still makes only a little more than 40% among all patients with pulmonary cancer, and the III stage of tumour development is diagnosed in half of patients who addressed to the doctor for the first time, and the IV stage of the disease is found in each ten patient.

For the majority of the patients with cancer of lung final decision on character of the disease and its spread can be made only after the profound inspection in specialized hospitals.

During the examination of the patient with lung cancer it is possible to pay attention to acyanotic, grey colour of dermal integuments, to note loss of body weight, decrease of muscular tonus, inherent to many oncologic patients. More pronounced changes, such, as the forced patients position and restriction of movements because of pain in the backbone, instability of gait, hypodermic veins extension of the upper part of the trunk, edema of the neck and the face — indicate damage of the lung by a tumour with wide-spread lymphogenous or hematogenous metastases.

During palpation it is possible to determine decrease of tissues turgor, to estimate a condition of regional lymph nodes accessible for palpation, including supraclavicular and more removed — axillary, inguinal. At palpation it is always necessary to pay special attention to areas which the patient specifies as a source of pain or appeared unusual inspissations, growth and

so on. Their site can be rather various, frequently — outside the thoracal wall.

The opportunities of percussion in diagnosis of lung cancer are not great, but this method with enough high accuracy allows assuming presence of atelectasis of appreciable departments of the lung, clump of exudate in the pleural cavity.

With the large reliability it is possible to draw a conclusion of diffusion both blastomatous and paracancrosic changes on the character of respiration determined at auscultation, presence or absence of rales, decrease of respiratory murmur above the lung damaged by a tumour.

Roentgenologic inspection has a leading role in diagnosis of lung cancer. It includes lung X-rays in two projections, level-by-level tomographic research, and a computer tomography under the indications. The study of roentgenograms enables to determine, to specify localization, sizes, structure of pathological changes in the lung, their diffusion, relation to tracheobroncheal tree and large vessels of lung root and mediastinum, pleura and other anatomic formations of the thoracal cavity.

The necessity of computer tomography in patients with lung cancer should be always proved by the results of usual, traditional X-ray inspection. The computer tomography should be applied purposefully for solving a special task. Such tasks more often include detailed topography of pathological changes in the lung and thoracal cavity, their malignancy and diffusion. Computer tomography can also be applied for specification of nature of extrapulmonary changes; especially suspicious on cancer spread to the liver, brain, kidney etc.

The additional information about diffusion of tumoral changes within the limits of mediastinum can be given by X-ray — contrast research of the esophagus. Its shift or narrowing frequently testifies to an extensive lymphogenous innidiation of lung cancer on regional collectors of the mediastinum.

Bronchography has been rarely used in lung cancer diagnosis lately, but the results of this research can be useful in difficult cases, differential diagnosis.

Endoscopic bronchologic research is obligatory for each patient with prospective or fixed lung cancer. It is undertaken after X-ray inspection that most precisely focuses a direction of survey concerning a location of pathological changes in the lung. Bronchoscopy enables to reveal a condition of the lumen of the respiratory ways in details. The modern fibre-optic bronchoscopes equipped with a complete set of kelectomes, brushes-scari-

ficators and apparatus for aspiration of bronchial secretion can help a visual estimation by taking a material for morphological study. The exact verification of character of pathological changes in lung reaches 95% of observations with the help of fibrobronchoscopy with pinching and scarificating biopsy.

Diagnosis of lung cancer includes not only tumour revealing but always assumes its morphological verification with establishment of degree of malignancy. At absence of an opportunity to receive these data during a bronchoscopy, additional diagnostic receptions are applied to recieve necessary information. First of all it is transthoracic needle biopsy of a tumour that is used more often in peripheric pathological changes in the lung, near to visceral pleura.

Mediastinoscopy, parastemal mediastinotomy, biopsy of the supraclavicular lymph nodes, transbronchial needle biopsy of bifurcation lymph nodes are diagnostic means which are carried out as so-called "small operations" to reveal lung cancer metastasing to the regional and more distant lymphatic collectors.

Ultrasonic research of the liver and kidneys, radioisotope hepatography and hepatoscanning, enabling to reveal metastases of lung cancer in the parenchymatous organs, are used for diagnosis of distant hematogenous lung cancer metastases. A puncture biopsy of the liver, laparoscopy and diagnostic laparotomy is applied in more difficult cases for diagnosis of lung cancer metastases. The last one enables to survey not only the liver but also organs of retroperitoneal space in details, to confirm or to exclude involvement of the kidneys and adrenal glands. This operational method of diagnosis is traumatic, but is considerably more reliable and productive.

It is necessary to note that a variety of methods offered and used in pulmonary cancer diagnosing is caused partly the fact that that any of them has no prior importance in definition of medical policy of this disease. Only the results of all examinations with high reliability allow to judge spread and features of tumoral process. Diagnostic tasks also include definition of functional and reserve opportunities of respiration, cardiovascular and other basic systems of life supply of the patient's organism. These data are necessary for medical policy and choice of treatment method, taking into consideration data, received during inspection of the patient.

Treatment. It' s well known that the surgical treatment is the most successful in I–II stages of lung cancer, when a tumour and its lymphogenous

metastases has not yet gone out of limits of a damaged lung or its lobe. Five years' survival rate among the radically operated patients with lung cancer of the I stage reaches 85–90%, in the II stage — about 60%. Unfortunately, the number of patients with lung cancer of I–II stage among all patients, directed to surgical clinic, is rather insignificant and makes no more than 40–45% from total number of patients suffering from this disease. Purposeful study of features and laws of lung cancer innidiation on regional lymphatic collectors of the mediastinum (I. S. Kolesnikov, S. A. Shalaev, I. A. Chalisov, L. N. Bisenkov etc.) has allowed to develop and to use into clinical practice rather effective operative measures in patients with the III stage of lung cancer — the extended pneumonectomy — an erasion of the lung damaged by a tumour or its part with a wide dissection of lymph nodes in the cellular tissue of mediastinum, the metastases in which are found out in half of patients. While involving in tumoral process of extrapulmonary anatomic formations — the thoracal wall, diaphragm, esophagus, pericardium, wall of the superior caval vein, vagus and diaphragmatic nerves etc., the extended operative measures are supplemented by a resection of these organs. The introduction into clinical practice of such extended and combined pneumonectomies has appreciably allowed increasing efficiency of surgical treatment in this group of the patients. 21–26% of the patients live after such operations for more than 5 years. The development of bronchoplastic reconstructive operative measures promoted expansion of operative treatment of the patients with lung cancer, especially among the persons of the senior age groups. Its usage has increased operability of patients with lung cancer among those for whom pneumonectomy represents a direct vital risk, and lobectomy is technically impracticable and oncologically unjustified.

Radiation therapy is performed when a tumoral process has not left the limits of the lung and thoracal cavity, but its diffusion excludes surgical intervention. Frequently this method of treatment is recommended to the patients who have refused an operation for different reasons or it can not be performed because of severity of their general condition caused by accompanying diseases.

Application of gamma-therapeutic apparatus, in which telecobalt is used became a new stage in the development of lung cancer radiation therapy. This allows prolonging life up to five years in 5% of patients. The subsequent development of apparatuses for radiation therapy and ways of irradiation, the creation of rotatory gamma therapy have increased efficiency of irradiation and more than 7% of patients live for over five years after a course of rotatory gamma therapy.

The application of linear and cyclic accelerators with the use of brake or electronic fascicles with generation energy within the limits from 4 up to 45 MeV appeared to be especially successful in deep tumours, including lung cancer. It was noted that the majority of patients have complete regress of a tumour at a focal dose 60 Gy. However, the application of electrons has not obtained a wide recognition in clinical practice yet.

The development of interstitial radiation therapy techniques is undertaken: implantation of isotopes as needles or granules in the tissue of the lung tumour. Thus a considerable regress of a tumour is marked: 40% of patients with I–II stages of lung cancer and 7% of patients with III stage of disease live for more than 3 years. However, intensive irradiation frequently results in occurrence of serious complications (bleeding, empyema of the pleura, lung abscess, bronchial fistulas), that constrains clinical application of the method.

As it was possible to establish on the basis of radiobiological researches, that the success of lung cancer radial treatment is determined not only with exposure dose, but also with special properties of tumoral tissue. It is proved, that high resistance of hypoxic fraction of tumoral cells against influence of ionizing radiation is one of the reasons low efficiency of irradiation. While introduction of large doses of oxygen under the increased pressure to a tumour the radiosensitivity of such hypoxic tumor departments grows considerably and medical effect is enlarged. The experience of radiation therapy realization under conditions of hyperbaric oxygenation at oxygen pressure of 2–3 atmospheres has shown considerable increase of rate and size of tumour regress in the overwhelming majority of patients.

The result of radiation therapy carried out according to a full program to patients suffering from lung cancer, was the prolongation of life span in more than 12% of patients for over five years. In I–II stages of the disease this parameter has made 33%, and in III stage — more than 8%.

Chemotherapeutic treatment of the patients suffering from pulmonary cancer was finally acknowledged during last 35–40 years. Various preparations used for this purpose cause in the tumoral tissue oppression of mitotic activity and morphological changes in chromosomes of divided cells, disturbance of DNA and RNA synthesis, block synthesis of nucleic acids, inactivate enzymes, which provide various parts of intracellular exchange, detain separate phases of the mitotic cycle.

The common disadvantage of drugs used for chemotherapeutic treatment of the patients with lung cancer, is the absence of their selective, purposeful influence on nothing but tumoral cells. These drugs to some

extent render also damaging action on other intensively proliferating tissues of a human organism. So, frequent signs of such action are disorders of haemopoiesis, immunodepression etc.

The efficiency of such treatment depends on the mass of the tumour and its histological structure: small cell carcenomas are highly sensitive to chemotherapeutic preparations and not small cells carcinome are practically resistant to this method of treatment.

Basic preparations used now for treatment of lung cancer are cyclophosphamide, methotrexat, nitrozomethilurea, vincristin, adriamicin, platidiam, vepezid, goloxan.

The indication for chemotherapeutic treatment is considerable cancer spread, excluding both surgical intervention and radial treatment; considerable decrease of respiratory function and cardiovascular systems; various personal motives forcing the patients to refuse an operation or radiation therapy.

The reason for refusal of chemotherapy by the patient suffering from lung cancer is pronounced cachexia and tumoral intoxication, extensive tumour dissemination in the lung with progressing respiratory failure, disintegration of the tumour with pneumorrhagia menacing transition in a pulmonary bleeding, and also long-term previous use of antituberculous remedies lowering erythropoiesis, causing leukopenia. Lung cancer metastases damage the liver, bone marrow, and other internal organs that appreciably reduce opportunities of chemotherapeutic treatment. That is why in each case the question on the indications and contraindications to chemotherapeutic treatment concerning lung cancer are decided individually.

The most complete medical effect is achieved with the chemotherapy concerning lung cancer which is carried out at short cycles repeated every 3–4 weeks and with the use of a combination of several preparations. The complete course of chemotherapeutic treatment enables to receive not only immediate improvement of patients' health, but also to prolong life span to some of them. Life span of the patients with lung cancer, who has received a course of treatment with chemopreparations, on average makes about 8 months, and 2% of them live for more than three years.

The medical effect of surgical intervention concerning lung cancer can be increased by additional realization of radial or chemotherapy. Such combination of surgical operation with other methods of antitumoral effect has received the name of combined treatment. The combination of surgical intervention with radial treatment or chemotherapy can be carried out both

before the operation, and after its performance. Each of these tactical approaches has the substantiation.

Preoperative irradiation or chemotherapy provides a decrease of biological activity of all tumoral population at the expense of cytogenetic changes of cancer cells in the primary center and metastases in regional lymphatic collectors. Such cells remaining in a zone of operative intervention and even acting in blood channel, substantially lose an ability to implantation and formation of metastases of lung cancer. Besides, it is marked that the immune processes arising in reply to destruction of a part of tumoral cells, can also handicap implantation of the alive cells.

The postoperative radiation therapy compensates inaccuracy of operation, which oncologic radicalism is quite insufficient, that frequently occurs in patients with lung cancer.

During extended operative measures in this category of patients it is difficult to follow principles of ablastics, because a complex of tumoral cells together with lymph can get into the operative area from widely open regional lymphatic collectors and pleural cavity. The study of the remote results of combined treatment in patients with III stage of lung cancer has shown that it is possible to prolong life span in 30% of patients for more than five years, in comparison with 26% of patients who received only surgical treatment.

The preoperative radiation therapy occupies now a rather small place in the combined treatment of the patients with lung cancer. It is caused by the fact that the most complete representation about true spread of blastomatous changes in the lungs and the stage of the disease can be determined after detailed histological study of operational material, and also that the irradiation considerably reduces reparative processes and the number of postoperative complications in the previously irradiated patients grows considerably.

The reason for chemotherapy usage in pre- or postoperative period are like to those for radiation treatment: destruction and devitalization of tumoral cells getting into the zone of surgical action or to the blood, prevention of new tumour foci formation. It is obligatory with operation concerning small cellular lung cancer at any stage of the disease development; the combination of a radical surgical intervention with chemotherapy enables to increase life expectancy.

Sarcoma of the lung. The mesenchymal component of lung structure is seldom a place of development of tumoral process, which has epithelial elements of bronchi and bronchioles. Probably to some extent it is connected

with a frequent influence of oncogenous factors immediately on epithelial structures, while the mesenchymal formations are protected by epithelial integument and are protected from influence of the exogenous factors of oncogenesis through blood and lymphatic vessels. Apparently, for the same reasons sex, smoking and action of penetrating radiation do not influence the frequency of primary lung sarcoma's development. The influence of internal causes of predisposition to blastomatous process and malignant transformation of benign neoplasms (fibromas) is more probable.

The first detailed description of a primary lung sarcoma was made in 1856 (Poisson, Robin). In Russia the first report on this disease belongs to M. A. Lvov (1899). More than 500 cases of primary lung sarcoma have been published to the present time in the world literature. This disease can arise at any age and is observed much more often than it was supposed earlier. Its rate in relation to lung cancer widely ranges: from 1:20 up to 1:100. Such difference in these parameters to a certain extent reflects difficulties in explanation of morphological data, especially in the late stages of tumour development.

According to the form of tumour growth we distinguish ball-shaped, characterized by expansive growth, and infiltrative sarcoma of the lungs. Endobronchial sarcoma is separately distinguished; it can be both polypoid and diffuse.

According to histological signs we distinguish differentiated sarcomas of the lungs (angiosarcoma, chondrosarcoma, lymphosarcoma, neurosarcoma, myxosarcoma, reticulosarcoma, leiomyosarcoma), undifferentiated sarcomas of the lungs (round-cellular, spindle-shaped-cellular, polymorphocellular) and mixed primary lung sarcoma. In a number of cases the opportunity of transformation of lung cancer in sarcoid growths (carcinosarcoma) is not excluded, and metastases of such tumours can have a structure of both cancer and sarcoma. It is possible that a source of these sarcomas development is the interalveolar connective tissue.

Lung sarcoma is differentiated according to localization of primary tumoral centers to peripheric and central.

Pathological anatomy. In most cases primary sarcoma of the lung looks like a massive tumoral node occupying a part or all lobe of the lung, quite often affecting the whole lung. The tumour can be limited from environmental tissues, owing to its expansive growth, but can infiltrate them, involving the large bronchi. The definition of initial tissue, in which originally there was a focus of sarcomatous growth in the lung, quite often, is rather inconvenient, especially at the late stages of development of sarcomatous process. Most frequently, especially with localization of

sarcoma in peripheric departments of the lung, the tumour is ball-shaped, sometimes reaching large sizes and quite often clearly limited from environmental parenchyme. Infiltrative growth of lung sarcoma occurs much less often, diffusion of tumoral process on peribronchial and perivascular structures of lobar bronchi lays in its basis.

In these cases tumour quite often infiltrates into the thoracic cage, fatty tissue and lymph nodes of the mediastinum.

The great connection of morphological tumour structures with blood vessels is marked during histological exam of preparations in all kinds of primary lung sarcoma, therefore the term "angiosarcoma of the lung" up to a known degree is a collective concept uniting all kinds of lung sarcoma, originally outgoing from various elements of blood vessel wall. Fibrosarcomas of the lung are the next following on frequency, proceeding from the elements of connective tissue of bronchuses or peribronchial tissues. Lymphosarcomas of the lung, proceeding from the lymph nodes of the bronchopulmonar system occupy the third place. Other kinds of primary lung sarcomas occur much less often.

Clinical course. The clinical displays of primary lung sarcoma are similar to symptomatology of lung cancer and have poorly specific signs. The true nature of pathological changes in the lungs is frequently connected with long unsuccessful treatment of presupposed pneumonia or homogeneous shadowing in the lung, found out during a routine inspection and roentgenophotography. However, quite often there is no sign of organ damage, but osteoarthropathy as a progressing thickening of bones of extremities, periostites, polyneurites. The reasons of such systemic lesions have been studied insufficiently: it is believed that intoxication, hypoxia, endocrine disorders and influences of other factors pay an important role in their origin, as these damages are not specific to a primary lung sarcoma and are observed in a number of other diseases.

Changes in the peripheric blood in primary lung sarcoma can be absent or can be shown by anemia, increased ESR, leukocytosis. Change of thrombocytic formula of peripheric blood which increased number of old forms of platelets plays great importance (F. G. Uglov).

Roentgenologic signs are determined by localization of the primary tumoral center (central, peripheric), presence or absence of accompanying changes and complications (atelectasis, inflammation of lung metastases environmental parenchyme), character of tumour growth (endobronchial, peribronchial, intrapulmonic). Roentgenologic inspection should be complex and roentgenotomography should include angiopulmonography in many cases. To differentiate diagnosis of primary lung sar-

coma it is necessary to define that the overwhelming majority of them are notable for slow growth and metastases arising only at late stages of the disease, with the exception of lymphosarcomas, angiosarcomas and myxosarcomas which manifest in fast growth and early innidiation.

Peripheric sarcomas at an early stage, as a rule, are asymptomatic and are found out occasionally while preventive inspections. At further stages the clinical signs are caused by mechanical pressure and growth of tumour having usually infiltrative character. At these stages lung tumour frequently infiltrates the neighboring organs and anatomic formations (the esophagus, the trachea, large blood vessels, the thorax, the mediastinum, the pericardium etc.), that is manifested by corresponding clinical signs.

Clinic-roentgenologic symptomatology of central lung sarcoma is similar to a clinic-X-ray picture in lung cancer. It is determined by sizes and character of tumour growth (endobronchial, peribronchial, intrapulmonic). At late stages of central lung sarcoma the clinical signs of involvement of on the next organs, such as dysphagia, superior caval vein syndrome, hemorrhagic pericarditis, hemorrhagic pleuritis etc., appear.

Differential diagnosis of primary lung sarcoma is carried out with lung cancer, echinococcosis, tuberculoma, unparasitogenic cysts of the lung. Sometimes there is a necessity in differential diagnosis of central lung sarcoma with a malignant thymoma.

The basic method of *treatment* is surgical (lobectomy, atypical pulmonectomy, pulmonectomy). Despite the fact that almost half of the patients does not have metastases of the tumour, modern oncology treatment of lung sarcoma should be complex, that means a combination of operative measures with radiation and chemotherapy.

Polyploid sarcoma of the pulmonary artery trunk. Among primary sarcomas of the lung this kind of sarcoma is allocated in a separate nosological form. For the first time it was spread by Mandelstamm in 1923, and in the home literature by S. V. Ryzhkov in 1957. In the world literature about 100 observations are described up to the present time.

The source of tumour formation is the valves of the pulmonary artery or its wall. The tumour has a polypoid form and is closely connected to the valves or the pulmonary artery trunk. Growth of a tumour rather frequently to the right and left pulmonary arteries. At later stages there can be invasion in the aorta, mediastinum and parenchyme of the lungs. Morphologically the tumour has signs of undifferentiated sarcoma, leiomyosarcoma, fibrosarcoma, myxosarcoma, rhabdomyosarcoma. Remote metastases in the rain, the adrenal glands, the pancreas occur most often.

Clinical course. Women at the age of 50–55 years prevail among all patients. A clinical picture of the disease is rather variable, but most often clinical signs are dyspnea, stethalgias, pneumorrhagia, cyanosis and systolic hum in the precardial area. Intravital diagnosis of the disease remains rather difficult and most informative is radiopaque angiopulmonography, at which the defects of filling in the trunk of the pulmonary artery or its main branches are found out. The life span of the patients can last from several months up to 20 years, but the overwhelming majority of them dies within one year after appearance of the signs listed above.

Treatment is surgical in a combination with chemotherapy.

Lecture 3
ESOPHAGIAL CANCER

Anatomy and physiology. The esophagus is a muscular tube about 35 cm long, covered with mucosa and surrounded by connective tissue through which nutrition from the pharynx gets into the stomach. It starts at the level of VI cervical vertebra and reaches XI thoracic vertebra. Esophagus is divided into three departments: cervical (5–6 cm), thoracic (16–18 cm) and abdominal (1–4 cm). The entrance of the esophagus is posed at the level of the cricoid and at a distance of 14–16 cm from the edge of the upper incisors. The upper esophageal sphincter — the first physiological narrowing — is placed there. The second physiological narrowing is at the level of trachea bifurcation and crossing of esophagus with the left primary bronchus, the third — corresponds to the level of esophageal aperture of the diaphragm.

The esophagus is posed to the left of the medium line at the cervical and at the beginning of the thoracic department up to the level of the aortic arch. In mediothoracic department, it deviates to the right and lays to the right of the aorta, and in infrathoracic department, it again deviates to the left of the medium line and above the diaphragm, it is posed in front of the aorta. Such anatomic location determines following operational ways: to the cervical department — left-hand, to mediothoracic— right-hand transpleural, to infrathoracic — left-hand transpleural. The mucosa of the esophagus is formed by laminated flat epithelium, which passes in cylindrical gastric epithelium at the level of a gear line posed a little bit above the anatomic cardial part. The submucosa is composed of connective and elastic fibers. The muscular environment consists of internal circular and outside longitudinal fibers. The muscles are striated in the upper 2/3 of the esophagus, in the lower third of it they are smooth. From the outside the esophagus is surrounded with a quaggy fibrous connective tissue, in which there are lymphatic, blood vessels and nerves. There is a serous layer only in the abdominal department of the esophagus.

Blood supply of the cervical department of the esophagus is carried out from the inferior thyroid arteries, of the thoracic one — from the esophageal arteries departing from the aorta, branches of the bronchial and intercostal arteries, of the abdominal one — from ascending branch of the left gastric and a branch of the inferior diaphragmatic arteries. The blood supply of the esophagus in the thoracic department has a segmental character, so its allocation on the considerable extent from the surrounding tissues during operative measures can result in the esophagial walls necrosis.

The outflow of the venous blood from the lower department of the esophagus passes into the splenic vein and further into the portal vein, from the upper departments of esophagus — into the lower thyroid, azygos and hemiazygos veins, further in the superior vena cava system. Thus, in the area of the esophagus there are anastomoses between the portal and the superior caval veins system.

Lymph outflow from the cervical department of the esophagus is carried out up to the peritracheal and deep cervical lymph nodes. The lymph flows into tracheobroncheal, bifurcation, juxtaspinal lymph nodes from the thoracic department. Regional lymph nodes are paracardiac, and also the nodes located in the field of the left gastric and celiac arteries for the lower third part of the esophagus. A part of the esophageal lymphatic vessels opens just into the thoracic duct. It can explain in some cases earlier occurrence of Virchov's metastasis, but not metastases in the regional lymph nodes.

The branches of vagus nerves, which form anterior and posterior plexuses on its surface, carry out innervation of the esophagus. The fibers, forming an intramural muscular-intestinal nervous interlacement (Auerbach's) one and submucous one (Meisner's) are separated from them by the esophageal wall. The cervical part of the esophagus is innervated by recurrent nerves, the thoracic one by the branch of vagus nerves and fiber of sympathetic nerve, the abdominal one by the branch of splanchnic nerve. The parasympathetic department of the nervous system carries out the regulation of the esophagus motor function and lower esophageal sphincter.

Physiological importance of the esophagus consists in realization of nutrition from pharyngeal cavity in the stomach exercised by a swallowing reflex. Thus, the important role in normal activity of the esophagus belongs to duly opening of the cardial part that normally happens 1–2 sec after a swallow. The relaxation of the lower esophageal sphincter provides free entering of nutrition into the stomach under the influence of peristaltic wave. After the passage of alimentary piece into the stomach, there comes restoration of tonus of the lower esophageal sphincter and its closing. The lower esophageal sphincter forms a barrier between acidic contents of the stomach and esophageal mucosa, which is very sensitive to it.

The diseases of the esophagus first of all are connected with the disorder of nutrition passage, peristalsis, function of upper and lower esophageal sphincters: the rising of tonus of the upper esophageal sphincter results in occurrence of Zenker's diverticulum; the rising of pressure in the lower esophageal sphincter causes achalasia and absence of relaxation reflex (formation of epiphrenal diverticulum); the decrease of pressure in the lower esophageal sphincter together with other factors promotes development of reflux-esophagitis, peptic ulcer of the esophagus; promoted pathological processes in the esophagus, especially cancer, cause dysphagia and complete obstruction of the esophagus.

Methods of research. The contrast X-ray inspection of the esophagus by water suspension of barium sulphate (at suspicion of perforation with water-soluble contrast) is the basic method of diagnosis. It is carried out at various turns of the patient around vertical axis. They pay attention to the character of contours, peristalsis, relief of mucosa, function of the esophageal sphincters.

The computer tomography enables to determine interrelation of the esophagus and formations outgoing from it, with environmental organs (for example, invasion of the esophageal tumour into the trachea, bronchi, metastases in the lymph nodes and so on).

Esophagofibroscopy allows examining mucosa of the esophagus at all its extent, to make biopsy from suspicious sites, to make smears for cytologic research. The removed slices of tissues are subjected to histological research. Esophagoscopy with a rigid esophagoscope is applied in the medical purposes for extraction of foreign bodies, sclerosing of dilatated veins and so on. Esophagotonokimography — a graphic record of reductions and tonus of the esophagus walls and its sphincters — is a diagnostic method of functional and some organic diseases of the esophagus sphincters (achalasia of cardial part, esophagism, hernia of the esophageal aperture of diaphragm etc.). pH-metry — a definition of gastroesophageal reflux intensity with the help of a special probe or radiopill, which are placed 5 cm above a cardial part. There is a sharp decrease of pH in the esophagus and intensification of pain syndrome in gastroesophageal reflux.

Etyology and pathogenesis. Cancer occurs in 60–80% of the esophagus diseases. This disease usually develops at the age of 50–60 years, mainly men suffer, and in the more senior age group (older than 60 years) — women. Men get ill 2–3 times more often than women. Esophagus cancer arises most frequently among the inhabitants of northern and east regions. The highest rate of esophageal cancer is marked in the Central Asia, the lowest — in Moldova, Lithuania. Among the mortality from malignant neoplasms, esophageal cancer occupies the third place after

carcinoma of the stomach and lung cancer, and this parameter is the highest in Turkmenistan, and the lowest — in Moldova. In the world it is the highest in France, the lowest — in Norway, Sweden.

The development of esophageal cancer is mostly provoked by chronic inflammation of the esophageal mucosa owing to mechanical, thermal or chemical irritation. Traumatisation of the esophagus mucosa by nutrition containing dense alimentary masses, small bones, and also excessive amount of spices, very hot food and alcohol, smoking, especially on an empty stomach, can promote chronic esophagitis being a precancerous condition. The analysis of the reasons for non-uniform frequency of esophageal cancer spread in different latitudes and increased morbidity in separate regions specifies some features of local feeding habits. The inhabitants of northern areas, for example, are more inclined to use hot food, than in south areas, and in some regions the inhabitants of north eat kindled bacon, dried fish containing ossicles, traumatizing esophageal mucosa. Another group of reasons causing cancer includes such precancerous diseases, as cicatrical esophageal strictures after chemical burns, hernia of the esophageal aperture of the diaphragm accompanied with reflux-esophagitis, leukoplakia, various benign neoplasms, and long existing ulcerations of esophageal diverticulums. A precancerous disease is also sideropenic Plammer — Winson's syndrome, shown by hypochromic anemia, achlorhydria, atrophy of mucosa with subsequent development of hyperkeratosis of oral cavity mucosa, pharynx and esophagus, and also papilloma of the esophagus.

Pathological anatomy. Esophageal cancer develops more often in places of physiological narrowings: esophageal ostium, at the level of trachea bifurcation above the physiological cardial part. The medium third of the esophagus thoracic department (60%) is the most affected, tumours in the infrathoracic and abdominal (30%) and suprathoracic (10%) departments of the esophagus are less frequently found out. Three basic forms of esophageal cancer are distinguished microscopically. In scirrhous, or infiltrating form of cancer (10% of patients), the tumour infiltrates the esophageal wall at regular intervale and passes into the normal tissue. It looks like a dense white thing, expanding circularly, covers The esophagus and is characterized by plentiful development of stroma. In large tumours ulceration and perifocal inflammation takes place in the center.

Medullary or ulcerative carcinoma is diagnosed in 30% of patients, grows into the lumen of the esophagus and easily breaks up. The tumour has precise borders and quickly ulcerates, circularly sprouts the esophageal wall only in late cases, but it early metastasizes into the regional and remote lymph nodes.

Nodulous carcinoma makes about 60% of all cancers of the esophagus. It has exophitic growth, expands as a cauliflower, easily breaks up and bleeds. However, more often there are mixed forms of tumour, which have elements of endophitic and exophitic growth, with early disintegration and ulceration. The extremely infrequent form is papillary cancer.

According to histological structure there are: epidermoid cancer with keratinization and without it, which occurs most frequently in 96% of patients. Adenocarcinoma occurs less frequently (in 3.8%), more less — colloid cancer. Adenocancroid and carcinosarcoma (0.04%) are extremely seldom forms.

Esophageal cancer spreads by immediate germination, lymphogenous and hematogenous metastatic way. A tumour dissiminates in the esophagus upwards and downwards, sprouts in all layers of its wall, squeezes the next organs. Rather late complication is the germination in the organs with a possible formation of fistulas. Lymphogenous metastatic spreading in cancer of the esophagus occurs first of all in the paraesophageal lymph nodes, and tumours, posed in cervical and suprathoracic departments of the esophagus, metastasize mainly in the mediastinal, supra- and subclavial lymph nodes. Cancer of the lower third of the esophagus dissiminates the lymph nodes around the esophagus and cardial part, retroperitoneal lymph nodes, along the celiac arteria and its branches and into the liver. If a tumour localizes in the mediothoracal department of the esophagus, metastases spread into the peritracheal, bifurcation, lower esophageal lymph nodes. In cancer of esophagus there can be metastases also on the parietal and visceral pleura. Hematogenous metastases occur in the late stage of the disease and more often are found out in the liver, lungs, bones and other organs.

Classification. There are distinguished four stages of esophageal cancer.

The I stage — a precisely limited tumour involving only the mucous and submucous layers. The permeability of the esophagus is not broken. Metastases are absent.

The II stage — tumour sprouts into all layers, but does not go beyond the limits of the esophageal walls, with conciderable disorder of the esophagus permeability. There are individual metastases in the regional lymph nodes.

The III stage — tumour occupies large hemicircle of the esophagus or circularly covers it, sprouts into the esophageal wall, and it is connected with nearby organs. The permeability of the esophagus is completely broken. Multiple metastases occur in the regional lymph nodes.

The IV stage — tumour sprouts into all layers of the esophageal wall, goes beyond its limits, penetrating into nearby organs. There are multiple

metastases in the regional lymph nodes and distant organs. International classification of cancer provides the characteristic of tumour according to TNM system, taking into account the following factors: primary tumour, depth of invasion, condition of the regional lymph nodes, remote metastases.

Element T — primary tumour:

T0 — there are no displays of a primary tumour;

Tis — preinvasive carcinoma;

T1 — tumour up to 3 cm long;

T2 — tumour from 3 up to 5 cm long;

T3 — tumour from 5 up to 8 cm long;

T4 — tumour more than 8 cm long.

Element P — depth of invasion:

P1 — cancer infiltrates only mucosa;

P2 — cancer infiltrates a submucous layer, without penetration into a muscular one;

P3 — cancer infiltrates a muscular layer, but does not penetrate into periesophageal cells;

P4 — cancer goes beyond the limits of the organ.

Element N — regional lymph nodes:

N0 — signs of regional metastatic spread are absent;

N1 — an individual metastasis occurs in the regional zone;

N2 — multiple operable metastases occur in the regional zone;

N3 — multiple inoperable metastases are in the regional zone.

Element M — remote metastases:

M0 — no signs of remote metastatic spread;

M1A — a solitary metastasis in the lymph node accessible for erasion;

M1B — inoperable remote metastases in the lymph nodes;

M2 — metastases in other organs.

Clinical course. In clinical manifestation of the esophagus cancer three basic groups of signs are distinguished: local, dependent on esophageal wall damage; secondary, arising because of spread to nearby organs, and general signs.

The disorder of nutrition permeability (dysphagia) is the first and late sign of disease. It is connected with narrowing of its lumen by a tumour, arising only in the tumoral damage of not less than 60% of the esophagus. Occurrence of dysphagia can be preceded by sensation of a foreign body appearing at swallowing of firm food, feeling of "scratching" behind a breastbone, "sticking" of food to the surface of the esophageal mucosa. In the initial period of the disease dysphagia arises at swallowing of dense

food. Patients feel temporary delay of alimentary piece at the certain level. A gulp of water usually eliminates these phenomena. Further, even well chewed food doesn't pass, dysphagia becomes constant and arises even after the use of liquid.

Pain (33%) — is a frequent sign of esophageal cancer. It arises during reception of food, localizes behind the breastbone and have a dull character, can irradiate in to the back, neck. Occurrence of pains is caused by mechanical injury by nutrition of the inflamed esophageal wall. Constant pains, dependent on the reception of food or intensifyed after a meal, are caused by germination of tumour into nearby tissues and organs, development of periesophagitis and mediastinitis. Regurgitation with food, or "esophageal vomiting" (23%), occurs in considerable stenosis of the esophageal lumen and clump of food above the place of narrowing. Some patients artificially cause vomiting for the relief of pressing sensation behind the breastbone and pains, appearing during the meal. Hypersalivation arises in 6–7% of cases and is the result of reflex exaltation of vagus nerves. General signs of the disease — weakness, progressing weight loss are the consequence of starvation and intoxication. The signs of esophageal cancer complication arising because of nearby organs involvement, belong to late manifistations of disease and usually testify to surgically incurable tumour. Increasing dysphagia, excruciating pains, pronounced intoxication belong to them. During germination of recurrent nerves, patients have hoarseness, in the damage of sympathetic nerve, Gorner's syndrome is noted. The prelum of the vagus nerve can cause bradycardia, attacks of tussis, vomiting. Larynx invasion is accompanied by change of voice, dyspnea and stridorous respiration. Perforation of tumour into the mediastinum causes purulent mediastinitis, and in germination of large vessel there comes serious (usually lethal) bleeding. The formation of esophagotracheal and esophageal-bronchial fistula is shown by tussis while taking some liquid. This complication usually comes to an end with pneumonia, abscess or lung gangrene.

Diagnosis. Authentic diagnosis of the disease is frequently established only by comparison of the complex esophagus research and clinical data. Disease history, general condition of the patient are very important. Patient's appearance and data of objective inspection at early stages of the disease, as a rule, reveal no pathological changes. Therefore, basic diagnostic method of the esophagus cancer is X-ray inspection, which reveals the following characteristic signs: disorder of the mucous membrane, detection of filling defect, presence of a shadow of the tumoral node, absence of peristalsis of the esophageal wall. Authentic representations about the condition and features of the esophagus

at various levels can be received by X-ray inspection in various positions, and with the help of a double contrast study and parietography. X-ray inspection in the conditions of pneumomediastinum, roentgenotomography in the straight line and lateral projections are applied to define tumour dissimination.

Esophagoscopy is indicated to all patients suffering from dysphagia and in any suspicion of esophageal cancer. Endoscopic exam reveals the reason of dysphagia, the level of the esophagus damage, the form of tumour, the degree of esophageal stenosis, presence of disintegration or tumour bleeding. An obligatory capture of material for cytologic and histological researches allows to determine morphological structure of the tumour in 92–96% of cases. It is necessary to remember that negative result of morphological exam does not exclude presence of cancer, especially in its initial stages.

Radioisotope diagnosis is based on the principle of more intensive accumulation of radioactive substance in malignant cells. Radioisotope scanning of the esophagus determines localization and extent of the site of the esophageal wall with increased intensity of radioactive phosphorus accumulation. Lymphoductography, azygography, mediastinoscopy are auxiliary methods of research allowing to judge about the condition of the lymph nodes and the degree of blastomatous process in the mediastinum.

Differential diagnosis should be carried out with those diseases, the leading sign of which is dysphagia, and first of all with esophageal achalasia, cicatrical narrowing and ulcer of the esophagus, peptic constrictive esophagitis, benign tumours and diverticula of the esophagus, sclerosing mediastinitis. Long term anamnesis and X-ray picture of esophagus stricture are characteristic for strictures after burns, traumas of the esophagus. Patients with peptic esophagitis and ulcers of the esophagus in anamnesis frequently have peptic ulcer accompanying with signs of reflux-esophagitis. In benign tumours of the esophagus dysphagia appears very slowly and general condition of the patients, as a rule, does not change, roentgenologic and endoscopic researches specify absence of changes in the esophageal mucosa above the tumour. The varicose phlebectasia of the esophagus is characterized by repeated bleedings, signs of liver cirrhosis and disorder in the portal vein system circulation prevail.

Treatment. The basic methods of esophageal cancer treatment are operative treatment and radiation therapy. The choice of treatment depends on tumour localization, stage of the process, presence of accompanying diseases. Positive results of surgical treatment occur at the I stage of the disease and are considerably worse during operations in II–III stages. Un-

fortunately, esophageal cancer is seldom verificated at early stages and patients, as a rule, address for help at late stage of the disease. Thus, radical operative treatment is executed only in a rather small number of patients. In cancer of cervical and suprathoracic departments of the esophagus the tumour quickly sprouts into environmental tissues. Cancer of this localization is more successfully treated with radiation therapy. In cancer of mediothoracic department of the esophagus a one-moment esophagectomy with applying of anastomosis between the rest of the esophagus and stomach, displaced to the pleural cavity, multi-stage operation of Dobromyslov — Torek is carried out. In these cases a thoracal department of the esophagus is deleted and gastrostomy is imposed by the transpleural access. 3–6 months later they make an artificial esophagus of the large or small intestine. In cancer of infrathoracic department of the esophagus, esophagectomy with a one-moment applying of intrathoracic esophagogastric anastomosis in the pleural cavity is performed.

Remote results of combined (radiation and surgical) treatment are a little bit better, than only surgical. The cooperative dose of preoperative radiation therapy makes 3,000–5,000 Gy. In inoperable tumours or in the presence of contraindications to radical treatment palliative operations are carried out to restore permeability of the esophagus. Palliative esophagectomies, canalizations of a tumour, establishment of gastric fistula belong to them. In the presence of tumour metastases in the remote lymph nodes the radiation treatment is inexpedient. If surgical and radiation treatment is impossible chemotherapy (5-fluoruracil or fluorophur with metatrexate and colchamicin) can be applied. However, positive results of such therapy are not very significant.

Prognosis. Esophageal cancer proceeds slowly in most cases. If radical treatment is impossible, prognosis is always unfavourable and average life span makes 5–10 months. Operative treatment gives insignificant percent of a five years' survival rate (about 10%). The prognosis concerning working capacity of the patients who got radical treatment, always remains doubtful.

Lecture 4
DISEASES OF THE MAMMARY GLAND

Anatomy and physiology. The mammary gland is the largest adenogenous organ on the surface of a female organism, which is located in soft tissue formation called mamma (from Latin — breast). The form and the sizes of the breast are various and depend on individual features of woman, her age, functional condition, amount of preceding pregnancies, delivery, and duration of children feeding. On the average, in optimum delivery period (20–30 years) transversal size of a female breast is 10–15 cm, longitudinal — 10–12 cm, thickness — 4–6 cm.

The gland body which is enclosed by a fatty capsule determines the form and sizes of the breast. These formations are located in connective tissue capsule, formed with superficial fascia. Subcutaneous fatty tissue is less expressed here. The gland body is separated from pectoral fascia by stratum of friable fat, which help to displace the brest according to the chest. There is no fatty capsule on the most raised place of the breast, here nipple places — the mamma's nipple, which is more often of conic, cylindrical or pear-shaped form. The pigmented peripapillary circle — the mamma's areola — makes the basis of the nipple. The breast is divided into 4 quadrants by crossing perpendicular lines, which passed through the nipple.

The gland body is significantly less than the breast, reminds a flat-convex disk with a rough frontal surface, and rather smooth back one. It is made of 15–20 radially located lobes, which are separated from each other by fatty and filamentary connective tissue. The main mass of adenogenous parenchyma is located in back departments of the gland, the passages predominate in the centre.

A number of dense connective bands, being a prolongation of *ligg. suspensoria mamme* (Cooper's ligaments) is directed from the frontal surface

of gland body, through the superficial fascia, which forms a fatty capsule, and subcutaneous fat to deep layers of the skin and clavicular.

A structurally functional unit of the mammary gland is acinus, representing a tubular epithelial formation. It consists mainly of the alveolar ducts, covered with single-layer cuboid epithelium, which passes into lactiferous ducts. A number of incorporating acinuses, form a lobule, which contains adenogenous parenchyma. Later, when lactiferous ducts merge in *ducti lactiferi colligens*, which radially are directed to the nipple, lobules form one of adenogenous mass lobes. The collective passages, the system of which contains a lot of smooth muscules, open on the top of the papilla, forming an extension — *sinus lactiferus.* All these formations form a so-called milk-removing system. In accordance with the distance from distal ramifyings of passages the cuboid epithelium becomes columnar, in collective passages — stratified, and on top of the nipple passes in stratified epithelium of the skin.

The nipple is a ledge of the skin with pigmented epithelium. The skin of the nipple and areola is rich in the nervous endings. There are circulating smooth muscular cells grouped in the basis of the nipple and in the nipple-forming sphincter around an opening of the lactiferous ducts. Muscular cells located radially, promote a protrusion of the nipple. In the area of the areola there is a fair quantity of sweat glands, which secretory activity increases during pregnancy and especially after delivery.

Blood supply of each gland proceeds from 4 sources: through *a. thoracica interna* from subcutaneous artery, which gives 6–7 perforating arteries; through *a. thoracica suprema* from axillary artery; from its departs *a. thoracica lateralis* giving branches to side quadrants of the gland; at last, the fourth group of feeding vessels proceeds from 4–6 intercostal arteries. All arteries form wide anastomoses, derivating surface and deep lobular arterial network. Therefore the damage of any blood supply source as a result of trauma or operation doesn't influence on the blood supply of adenogenous tissue.

The veins of the breast form two vascular networks. The outflow from gland body is carried out in deep veins carrying blood to axillary, subclavicular veins and vena cava superior. From the superficial network the outflow goes in two main directions: in the skin veins of the shoulder and lateral chest wall and vein of the neck area. Blood supply of the mammary gland is increased in premenstrual period, achieves maximum during pregnancy and lactation and is reduced in involute period. That is why the superficial venous network is well visible during lactation, when the breasts enlarge and the skin is stretched.

In different periods of woman's life the condition of lymphatic formations of this zone (capillary tubes and vessels) has essential differences.

The degree of their development depends on the age and functional condition of the ovaries, it is connected with menstrual cycle, period of pregnancy, which progresses together with modifications of the gland body. It should be taken into account in evaluation of pathological process spread (infection, tumoral growth).

They distinguish intraorganic and extraorganic lymphatic system. The intraorganic system removes lymph both from the gland body, and exterior surface of the breast — the skin and the subcutaneous fat. It begins with capillary tubes in intralobular connective tissue, which cover lobules in the parenchyme of the gland. In deep anterior departments of the gland body the lymphatic vessels form a network, which is connected with retromammarial lymphatic plexus. From anterior departments along the milk ducts the lymphatic vessels go forwards and run in subareolal plexus. Here comes the outflow of the lymph from the skin of central departments of the breast, and this stream predominates in general. Lymph flows from peripheric departments of the gland body in lymphatic vessels of the frontal chest wall and subcutaneous vessels of the breast on the opposite side.

The extraorganic lymphatic system is performed by ramifying vessels and regional lymph nodes. They distinguish iliac path (37% of lymphatic flow), subclavicular path of outflow (internal departments, upper and back departments of the gland body), parasternal path (internal departments of the gland) and more rare: intercostal, cross, in which lymph can outflow in contralateral lymph nodes and Gerota's path (1897) — in preperitoneal fat. Iliac, subclavicular and parasternal lymph nodes are considered to be the first collectors of extraorganic lymphatic system.

Innervation of the breast skin and the gland body is carried out from II–VI intercostal nerves and the filaments of the chest department of the simpathetic trunk. Sensory and secretory filaments branch out, forming a nervous plexus enclosing the gland. The nervous endings are mostly concentrated in the area of the nipple and near the nipple circle, creating a high-power reflex field. The nervous filaments depart from the plexus to blood vessels, milk ducts, adenogenous cells.

The main function of the mammary gland is excretion of milk (lactation). It undergoes three stages in the development: mammogenenesis — development of the gland from II month of embryonic period till the beginning of excretion of milk; lactogenesis — the origin of excretion of milk; lactopoiesis — development and maintaining of milk production.

Mammogenesis is stipulated by hormones influence of the anterior lobe of the pituitary gland (follicle stimulating and luteinizing), the ovaries, the adrenal glands and the placenta before the period of sexual maturing. In children it is irrespective of the sex in the beginning. Then the mammary

gland undergoes involute modifications — in boys and men it is similar to the structure of a newborn gland.

In girls a noticeable enlargement of the mammory gland happens at 10–13 years, when the growth of ducts and stroma rapidly increases under the influence of hormonal effects (estrogens). Up to 15–17 years the gland body reaches its usual sizes. With the beginning of menstruation progesterone of the yellow body is involved in the process of mammogenesis. Sex hormones regulate the processes of hyperplasia and regression of gland structures that is observed monthly in the reproductive period (readiness to lactation).

At the beginning of pregnancy mammogenesis is finished under the influence of placenta hormones (gonadotropin and prolactine), and also true hormones of the yellow body; proliferation of alveolar ducts with formation of terminal alveoles is finished in acinus. They are bubbles covered with large cells which look like a truncated prism. Outside of them basket-shaped myoepiteliosites locate, which reduction ensures clearing of alveoles and alveolar ducts from milk, formed in them. Total volume of the gland body simultaneously increases and the amount of fatty tissue and connective tissue decreases, the amount of lymphatic vessels increases, blood supply of the gland amplifies. The areola increases from 1.5 up to 8.0 cm in diameter, nipple hypertrophises. After completion of mammogenesis the gland body turns in the alveolar-tubular gland of apocrine type with expressed synthesis of protein, lipids and other milk components.

Lactogenesis happens rhythmically and is closely connected with the process of milk discharging, which is conditionally divided into two phases. The first is connected with irritation of nipple and areola receptors during sucking. As a result weakening of the pappillary sphincter takes place with active reaction of smooth muscle elements of ducts and sinuses. The second phase includes a hormonal unit: reflective excretion of hormone of pituitary gland back lobe — oxitocine in the blood, results in reduction of myeloepitheliocytes, therefore the alveoli contract. The milk comes through the ducts into sinuses and becomes accessible for sucking. The final formation and fastening of milk-discharging reflex happens up to the end of the first week after delivery.

Lactopoisis: the amount of breast milk increases and achieves a maximum up to 8–10 weeks after delivery and remains stable for some time, making 1–1.5 l per day. After the end of feeding amount of alveocytes and mass of gland tissue decrease, an amount of the connective tissue increase, the gland begins to be subjected to effect of estrogens. In the climacteric period, the level of estrogens is reduces, involution of adenogenous structures occurs gradually, the milk ducts get atrophy.

MALFORMATION OF THE MAMMARY GLAND. INFLAMMATORY AND DYSHORMONAL DISEASES

Malformations of the Mammary Glands

Amastia — absence of one or two mammary glands and atelia (natural absence of the nipples) is rarely encountered. Hypoplasia of the mammary glands is more frequently encountered (aplasia, micromastia). These anomalies may be combined with hypogenesia of female genitals and decrease of hormonal functions of the glands.

The additional number of the nipples (polytelia, hypertelia) and mammary glands (polymastia, hypermastia) is more frequently encountered. These additional formations are located along the so-called "mammary line" which unites the armpit with the armpit fold.

There may be development of inflammatory processes and tumours in additional mammary glands. That is why their preventive excision is rational.

Areolitis, Galactooophoritis

Areolitis is inflammation of the mammary areola which is more frequently encountered during breast-feeding and is related to nipple dysplasia (a flat or inverted nipple), incorrect child put to the breast, insufficient skin care or baby infection.

Clinical course. Severe pain on breast-feeding, itching, fissures in the nipple area and mammary areola. There is sometimes dirty film.

Treatment. Painting of the nipple with 1% alcoholic solution of methylene blue; 2% solution of gentian violet. It is possible to influence the yeast infection with 25% soda and glycerine solution. It is also rational to treat the nipples with neutral solutions.

Mastites

Mastitis (from Greek *mastos* — breasts) is a focal inflammatory process in the mammary glands associated with initial penetration of purulent infection to its parenchyma or stroma which often has a tendency to spread, purulent destruction of the glandular body and fatty capsule, retromammary cellular tissue.

As a rule, mastitis develops in the lactous mammary gland, often postpartum, especially in breast-feeding women (80–85% of all mastites). The

rest of postnatal mastites are observed in nonfeeding women and in 0.5–1% of cases it develops in pregnant ones. Sometimes mastitis rarely develops in newborns against a background of initial activation of proliferative processes in the gland with intake of lactogenic hormones with mother's milk.

The rate of postnatal mastites ranges from 2 to 6% among all parous women. Mastitis usually develops in one gland, more often in the right one. Bilateral mastitis rarely develops though nowadays bilateral damages are 9–11% of all mastites. Mastitis more often develops in young women (more than 80% at the age under 30).

Inflammation in mastitis is caused by agents of purulent infection, which vegetate or get into the skin in the region of the nipple or areola. The main representatives of such agents are staphylococci which are often isolated like monoculture (80–82%), rarely in association with gram-negative intestinal flora (up to 10–12%) and very rare — streptococci, bacillus proteus, pathogenic fungi (2–3%).

The source of infection which looks like intrahospital one is bacterium-carriers from the stuff and recently confined women with obliterated forms of pyo-inflammatory diseases and whose microbes are spread through everyday objects, bed linen, etc. A newborn with postnatal infection — navel sepsis, pyodermia — may also be a source of infection. However, the infection on the skin and nipples of the mother or in the oral cavity of the baby does not always cause mastitis. Local anatomical and systemic functional causes are necessary for its development.

Mastopathy, scarring in the gland after the severe forms of mastitis or benign tumours, anatomical peculiarities of the systemic causes such as flat, inverted, lobular nipple are the local causes. Pathology of pregnancy (delayed labour, threat of miscarriage, preterm delivery, toxicosis of the second part of pregnancy), labour pathology (the first labour with big fetus, trauma of the maternal passages, profuse bleeding, hand expulsion of afterbirth) are systemic causes. In such cases decreased activity of antiinfection resistance, postnatal fever, insomnia, overstrain of a parturient woman are important along with allergization, exhaustion during pregnancy and labour.

Major cases of affection of the gland of primipara are associated with poorly developed gland parenchyma, underdeveloped nipple or nipple erects badly, a mother has lack of experience in baby feeding and expression of breast milk. Ample amount of patients with affected right mammary gland can be explained by the fact that right-handed women feel more comfortable to decant left gland while the right one is less available in this case.

Pathogenesis. There are two main varians of mastitis development, which determine infection penetration to the glandular body. In cases when nipple fissures and areolae are the source of infection entry, the lymphogenic way

of the agent penetration by lymphatic vessels, which are in the intralobular connective tissue, layer is a characteristic feature. Under conditions of increased blood filling and lymphogenesis in the lactic gland, lymphogenic type of pathogenesis causes development of interstitial mastitis.

Another variant is galactogenic in the ductal system of the gland on breastfeeding or expression of the breast milk under the conditions of lactogenesis. Penetration of the agents into the region of terminal sections of the mammary ducts and alveoli causes milk solidification while acidic medium and the products of microbe activity causes the edema of duct walls and surrounded stroma, affects the epithelium, intensifying lactostasis with development of parenchymatous mastitis. However, differentiations of these forms of mastitis disappear soon and it is impossible to differentiate them clinically.

Under conditions of transient bacteriaemia, associated with foci of infection in the nasopharynx (tonsilitis, rhinitis, inflammation of additional nasal sinuses and others), maternal passages or other localization, the agent may penetrate the stagnant sections of the parenchyma by hematogenic way. Finally, the infection may penetrate through the skin due to sudden injury or thermal injury of the latter (hot water bottle). In such cases, the inflammation is easily spread into the subcutaneus cellular tissue and fatty capsule of the gland.

Both separation of the serous oozing and inflammatory infiltration of the glandular tissue or its passage to new sections with affection of the glandular body up to panmastitis is possible depending on the pathogenetic agent, condition of local and general antiinfection resistance of the organism. Pyodestructive or necrotic process develops simultaneously. The organism allergization may be accompanied by acute local reaction according to Arthus — Sakharov phenomenon against such a background.

Pathological anatomy. Microbe invasion in the nipple fissures may be limited by inflammation of the mammary ducts, which is revealed by lactorrhea with pus admixture, or areola inflammation. During inflammatory change in the gland tissue, the phases of serous and purulent inflammation with parenchymatous changes are observed.

The tissue of the affected section of the mammary gland is infiltrated with serous substance with leucocytic infiltration around the vessels in serous inflammation. The serous infiltration is replaced by cellular infiltration of all glandular mass which is gradually eliminated or forms small foci of purulent fusion of the gland particles. According to spread of serous and then purulent inflammation to new sections of the gland body it looks like a sponge infiltrated with pus or “honeycomb”.

In other cases, because of thinning of the interlobular septa (hyperplasia of the secretory apparatus during lactogenesis), lobular abscesses may be

fused. Multichamber cavities of irregular forms and different extent, which contain pus and tissue sequestra, form in it. Pus from these cavities may get into the subcutaneous cellular tissue and retromammary space. Sometimes because of involvment of blood vessels into the inflammatory process (edema, compression of the surrounding tissues) and their thrombosis gangrenous mastitis develops that is necrosis of separate, sometimes — significant parts of the gland. In such cases, the gland tissue is grey with slight discharge sometimes of bad odour (nonclostridial anaerobes).

Atypical variant of necrotic changes is often observed (up to 5–6% of destructive mastitis). It is characterized by development of the gland destruction in separate segments not only like purulent fusion of the glandular tissue but also transfer of the inflammatory process into the cellular tissue and skin of all surface of the affected breast. Allergization confirmed by chill, urticaria, rhinitis, antibiotic intolerance, anaemia and exhaustion is the basis of such destruction.

Mastites with slow, subacute development of morphological changes, which are characterized by stable tissue infiltration, significant fibroblast development, the small foci of abscess revealed against a background of them, are distinguinshed as a separate form.

Classification. The clinical classification of mastites is based on morphologic principles (the stages of development of the inflammatory process in the gland), the rate of development of the purulent destruction, its extent and level of intoxication. That is why the stage of serous, infiltrated and pyodestructive mastitis is singled out. The slow and fast progressive forms are distinguished according to the rate of destruction development among which there is the limited destructive form with a tendency to spreading and finally the septic form.

Clinical course. The initial or serous stage of mastitis should be distinguished from lactostasis. However, in practice any coarsening of the lactic mammary glands, which is accompanied by high temperature, is considered to be mastitis. When a parturient woman complains of heaviness and tension in the gland, which gradually increases while milk is excreted fluently, and expression of milk causes pain in one or some parts corresponding to congestion, thickening with some segment borders, congestion is considered to be possible.

If thickening is not decreased in dynamics, stable subfibrilety appears and the serous stage of mastitis is diagnosed. This process is often manifested by acute development during 2–3 days. The disease starts with chill, temperature up to 38–39°C. The patient complains of general malaise, breakdown, and pain in the affected mammary gland. The gland is enlarged, but palpation is usually less painful, the affected region has unclear borders.

Expression of milk is painful and does not relieve pain. Leucocytosis develops in peripheral blood, ESR increases.

In some cases inflammatory changes subside and thickening becomes soft. In other cases temporary relief occurs, the body temperature becomes subfebrile, but local manifistations of mastitis are revealed: solid painful infiltrate with clear edges is determined in the affected gland. Local hyperthermia and skin hyperemia is absent over the infiltrate. The function of the mammary gland at the infiltrated stage is partially disturbed and galactorrhea is decreased. In 4–5 days after development of mastitis against a background of the infiltrate the softened part appears, that is the infiltrated stage changes into the destructive one. The local and general manifestations become intensified.

General malaise, sleeplessness, thirst, loss of appetite are characteristic of the destructive stage. The body temperature becomes high constantly (39–40°C) or fever becomes hectic. The affected glands are enlarged and tense. The skin is hyperemic over the affected area, cutaneous veins are dilated. Leucocytosis becomes significant. In these cases when the breast skin becomes swollen, hyperemic, cyanosed, regional lymphadenitis develops that is peculiar to phlegmonic type of acute mastitis. On pressing the affected skin, the place becomes pale and returns to its natural colour very slow. The infiltrate occupies the whole gland but there is no evidence of fluctuation.

Gangrenous mastitis is characterized by breast enlargement with tissue swelling, appearance of necrosis and subepidermal blisters, full of dark haemorrhagic fluid. The clinical signs of severe intoxication such as excitement or apathy, tachycardia up to 120–130 beats per minute, hyperleucocytosis or leucopenia, anaemia, proteinuria develop. Proteolytic blood activity increases, kalicreine-kinin system is maximally activated.

The rate of stage development of acute mastitis is individual: sometimes the pathological process may have 3 stages during a day from serous mastitis to abscess which is explained by infection virulence and suppression level of antiinfectious organism resistance, that's why temporary intervals can't be used for diagnosis of the disease stage. In uncontrolled usage of antibacterial drugs, the peculiarity of the course of destructive forms of the disease is possible. The majority of infiltrate-purulent forms, frequent diffuse damage of the gland body with development of a great length infiltrate significant and pus absorption by the gland tissue are characteristic of the destruction stage in such cases.

Even a more severe course of progressive destructive forms up to sepsis and septic shock is often observed. At the same time along with obliterated variants of destruction when the volume of changes in the gland does not correspond to the clinical course, atypical development of mastitis may occur.

Diagnosis of acute mastitis is based on the data of clinical examination. The investigation of peripheral blood with determination of hemoglobin, erythrocyte, leucocyte concentration and leucocytic formula with counting the ratio neutophils/lymphocytes, sugar level, and protein is necessary for diagnosis of this disease. The extent of damage may be determined with the help of thermography, contact electrothermometry: in destructive forms, the skin temperature over the damage centre is 1.5–2°C higher than other regions of the glands or over another gland. Bacteriologic study of milk from the affected and healthy gland, pus with bacterioscopic smear is used to specify diagnosis (lactostasis and mastitis) and choice of antibacterial therapy.

Treatment is chosen according to the inflammatory stage, rate of development of the destruction gland body and its extent. The women with acute mastitis in the serous and infiltrate stage more often come to doctor that is why it's better to pay more attention to conservative treatment. At the first stage, it must provide the decrease of exudation and swelling, evacuation of the galactoexcretory system and rational antibacterial therapy combined with treatment of the nipple fissures to prevent infection entrance.

With signs of lactostasis the mammary glands must be at rest (limitation of movements, elevated position of the breast with the help of bra or bandage, which should support but not squeeze the breast). Cold (an ice pack) should be applied to the area of inflammation for 20–30 min with an interval of 1–1.5 h. Antibradykinin drugs are used as anesthetics: analgin, baralgin. A short retromammary block by A. V. Vishnevsky may be used simultaneously, that is 100–120 ml of 0.5% solution of novocain with 10 mg chemotripsin and additional dose of effective antibiotics (ampiox, garamycin, lincomycin) is introduced under the gland body. Baby feeding is kept in lactostasis for effective evacuation of the galactoexcretory system. In ineffictiveness of these methods, evacuation may be reached by the use of breast-pump. Predominant bactericidal antibiotics are semisynthetic penicillins, cephalosporins. In case of suppuration and its quick spread to the gland, the woman should be admitted to hospital for surgical aid. Surgery in polyclinics is used only in limited suppuration, e. g. subareolar abscesses.

Operative intervention is the main component of treatment of the destructive forms of mastitis. Wide and deep incisions must be done to excise all necrotic tissues and eliminate purulent swelling. The obligatory condition of the operation is adequate anesthesia with one of the methods of intravenous anesthesia.

Intramammary abcesses in the upper quadrants are opened with radial incision proposed by a Kyiv surgeon S. G. Shalita in 1887 — from the areola

through the zone of greatest pain. A finger examines the abscess cavity, intersections are separated, and pus is removed. Then the cavity is cleaned with the solution of antiseptics (hydrogen peroxide, chlorhexidine). After the draining, the edges of incision are set apart with hooks, examining the purulent cavity, pressing the gland for not missing the junction with other deeper abscesses. Not only sequestra but also necrotic tissues connected with gland tissue are excised. Additional incisions are made to provide necessary radicalism to necrectomy and contraperturae are applied.

Bardengeyer opens retromamary abcesses in the lower quadrants of the gland with a crescent incision — the retromammary space is penetrated along the inferior transition fold, separating the mammary gland from the great pectoral muscle. Intramammary abscesses are opened in the back from the side of the smooth surface of the gland body. The surgery is combined with antibacterial, detoxication and infusion therapy, intensity of which depends on the form and level of destruction.

The wound may be managed by three variants after such intervention. There is an open management with optimal conditions for draining and local usage of the substances, which provide constant outflow from the wound: bandages with honey, ointments on water-soluble base, polyethylenglycol 400 and others. Salycilic acid, protease (chemotripsin, terylitin) combined with active antiseptics (chlorhexidine, solafur, dimexide) of local action on the infection and ultrasonic cavitation, treatment of cavity walls with laser of physiotherapeutic action are used for completing lysis of the necrotic parenchyma areas.

The early repeated sutures are indicated in the phase of regeneration when the wound is cleaned but marginal epithelization is unexpressive and has no granulations. It is possible to save breast shape, gland function and protect toxemia and wound exhaustion with their help.

Another variant of wound management after emptying abscesses and necroectomy is proposed by S. Popkirov (Bulgaria). After surgery of the destructive zone, the first suture is applied with multihole drainage for constant streaming irrigation of the purulent cavity with antiseptics.

It is possible to apply the variant of N. N. Kanshin (1977), when after surgery and excision of the affected area the purulent wound is sutured at once or in 2–3 days of temporary tamponade with introduction of double-lumen tube made of rubber, silicone, 6–8 mm in diameter to the wound from the separate incision. The irrigation of the purulent wound is provided by persistent introduction of antiseptics, fractional introduction of proteolytic enzymes through the small lumen of the double-lumen tube and constant aspiration through the great lumen with little rarefaction (20–25 mm Hg).

For successful surgery of destructive mastitis, it is important to have possibility to interrupt lactation, which has a pathogenic action on the inflammatory process in the mammary gland, and feeding with affected breast is impossible, expression of milk is difficult. It is not only that affected milk may cause intestinal infection in a baby. The milk becomes inadequate in structure because of protein and lactose decrease and related increase of fat. The usual methods of lactation restriction such as tight dressing, liquid limitation, salt laxatives, diuretics, atropine and some others are less effective, sometimes dangerous. The significant doses of folliculine (100,000 U), sinaestrol 2 ml of 0.1% solution intramuscularly for several days as well as androgens and estrogen may be utilized for medicamentous suppression of lactation.

Inhibition of hormones of the anterior part of the pituitary gland — prolactine and somatotropine — is more predominant method now. Administration of bromocriptine 2–5 mg 3 times a day decreases galactogenesis and eliminates lactostasis. Discontinuation of drug causes recurrence of lactopoiesis and interrupted breast-feeding may be continued. Bromocriptine 5 mg a day during next 10–12 days eliminates lactopoiesis. This component of therapy is indicated in gland malformation and dystrophy, mastopathy, scarring, nipple pathology. Besides, medicamentous interruption of lactation is applied in premature birth, death of a newborn.

Prevention. If surgery is performed in time and correctly, it is possible to obtain rehabilitation during 8–12 days after the destructive stage of mastitis. Inefficacy of intrusion, late and irrational surgical treatment of the destructive focus, prolonged usage of antibiotics intramuscularly or locally (into the infiltrate), massage of the inflamed glands, hand expression of milk, allergic reactions cause development of septic complications especially against a background of decrease of immunoresistance and wound exhaustion. The suppuration process progresses involving not only the glandular body but also fatty capsule and skin with formation of a big wound surface, which can be closed only with autodermoplasty.

The preventive measures of mastitis are preparation of future mother to delivery that is sanation of the infection foci, teaching the skills of breast-feeding, care for the mammary glands, hardening, and preparation of the nipples. Special attention should be paid to pregnant women from risk group (purulent infection in anamnesis, especially mastitis in previous delivery, mastopathy, abnormal course of pregnancy). In such cases, the most important is to prevent galactostasis, prophylaxis and treatment of the nipple fissures after the delivery.

Plasmocellular Periductal Mastitis

Plasmocellular periductal mastitis that is inflammatory damage of the mammary gland has a subacute course in contrast to purulent destructive damages. There are usually hyperaemia, edema and skin tenderness over one of the swollen segments of the gland occuring more frequently in external quadrants against a background of subfebrility and enlarged lymph nodes. In some days the skin manifestations dissapear and a solid infiltrate without distinct edges is left in the gland body. Inverted nipples and serous discharge are sometimes observed. Enlarged lymph nodes, which are not connected with adjacent tissues, are revealed. Infiltration of the gland stroma with a number of plasmic cells around the dilatated ducts are revealed in punctional bioptate of the infiltrate.

Diagnosis of subacute mastitis in the infiltrate stage is rather difficult; it is especially difficult to differentiate it from the tumour of the gland when the doctor failed to follow up development of the process. That is why if antiinflammatory therapy (including thermal procedures) and roentgenotherapy of this area prove ineffective during 2 weeks, the sectoral resection with histologic identification is indicated.

Tuberculosis of the Mammary Glands

Tuberculosis of the mammary glands develops rarely (0.5–1.5% of all diseases of this organ). It is characterized by such clinical manifestations as an inverted nipple, solid infiltrate without distinct borders that is spread to the skin and enlarged lymph nodes in the groin, usually on the affected side. Biopsy of the affected gland area, anamnesis and detailed examination of other organs are most important for diagnosis. During investigation of the peripheral blood, lymphocytosis and accelerated ESR are more characteristic than in chronic mastitis. The course of this type of mastitis is slow, unmarked. It is necessary to consult phthisiatrician on suspicion of tuberculosis of the mammary glands. There are 2 forms: caseous and sclerotic. The treatment is the following: intensive specific therapy and excision of the pathologic focus if it failed.

DYSHORMONAL MASTOPATHIES

Fibroadenoma and Fibroadenomatosis

Among dishormonal diseases of the mammary gland mastopathy, or fibroadenomatosis, with pathological proliferation of mammary gland's epithelium against a background of collagenous transformation of the gland body stroma and its following hyalinosis with cysts formation frequently

occurs. During routine inspections mastopathy is found out in more than a quarter of all women, but most frequently at the age of 30 up to 50 years in fibrocystic form.

Etiology. The leading factor, predisposing to the origin of mastopathy, is the disorder of regulative activity of the hypothalamo-hypophysial-adrenal system. According to the judgement of P. A. Gertsen, huge potential for future lactation in the epithelium of the mammary gland is hidden, and non-realization of this function creates conditions for pathological proliferation. Therefore a small number of deliveries, short and inconsistent lactation, abortions, inflammatory processes of the uterine appendages and follicular cysts of the ovaries become the factors predisposing to mastopathy. Such women have various disorders of the menstrual cycle, disorder of delivering function, anovular cycles. Disorder of liver function (inactivation of estrogens) under the influence of infection or intoxication, reduction of the thyroid gland function can influence the hormonal regulation.

Pathogenesis of mastopathy is generally determined by persistent action of prolactin, disorder of estrogens and progesterone corelation, increase of the level of follicle stimulating hormone and estrogens outside natural mammogenesis. Under the influence of endocrine disorders usual neurohumoral connections of the mammary gland change, cyclicity of physiological processes is perverted in it, that creates conditions for proliferation or promotes growth of stroma, therefore the drainage of gland ductal system worsens.

Pathological anatomy. The morphological picture of mastopathy is represented by combination of proliferative, dystrophic and atrophic modifications of the epithelium of passages, peculiar modifications of the myoepithelium and connective stroma of mammory gland's body. Depending on the primary influence of these modifications they select proliferative and unproliferative form of mastopathy. The proliferative form can develop in several variants, features of which are determined by the dominance of epithelial, myoepithelial or fibroepithelial proliferation and the phase of the process with intensive proliferation of the parenchyma (early phase) or sclerosis of the stroma (late phase).

In total epithelial proliferation there is adenitis, or mastoplasia, when enlarged but correctly generated lobules of the gland are revealed and small-sized cysts and cystoadenomas develop in its passages at late stages. Primary proliferation of intra-duct epithelium results in formation of multilayer basis of passages. Exactly these proliferations are inclined to malignancy. The third variant is mastopathy with proliferation of initial departments of adenogenous tissue and growth of the epithelium of acinuses and myoepithelium.

In the first phase proliferates form the fields from bands, deformed alveolar ducts and alveoli. Sclerosis of proliferation field with the development of so-called sclerotic adenosis occurs in the second phase. At last, fibroepithelial proliferation is possible, when connective tissue structures, covered by several numbers of cylindrical or cubic epithelium are formed in the milk ducts. In the late phase of this mastopathy variant multiple small-size adenomas, cysts and cystoadenopapillomas form. Separate cystoadenopapillomas — fibroadenomas, are revealed in large milk ducts.

Unproliferative form of mastopathy is characterized by the absence of early phase and is shown by the growth of connective tissue, in which atrophic lobule and cystic changed ducts are located, covered by epithelium forming small-size papillae. Lymphoplasmocytarian infiltration is revealed around the ducts.

Clinical course. The main signs of mastopathy are discharges from the nipple, breast nodularity, painful sensation. In the first phase of the process development, especially in the proliferative form, patients mark pains in one or both mammary glands appearing in the premenstrual period, which are combined with swelling of the mammary gland.

Simultaneously there can be observed the discharges from the nipple — foremilk, serous, sometimes with blood. Their character depends on the variant of proliferation. Painful sensation can be spontaneous or arise only at pressing, varying from sensation of a minor pain up to clear painfulness, provoked even by a contact with clothes to the mammary gland, it can be nagging, stabbing, irradiating to the neck and shoulder.

The third cardinal symptom is breast nodularity. It is determined during palpation in a standing position of the patient, when lobularity, granularity, banding of mammary gland tissue can be revealed. At palpation in lying position of the patient, at pressing of the gland body to the thorax this dense disappears (negative Kenig's symptom). Later, in the second phase of mastopathy, the periodicity of painful sensations becomes less clear, and changes in the mammary glands depend less on the menstrual cycle.

Two clinical forms of mastopathy are selected: diffuse, described above, and nodal. With nodal form of mastopathy the damage of not the whole gland body, but its separate segments and lobes take place. During palpation it is characterized by revealing of one or (more often) multiple, as a rule painless, indurations which are stable during the menstrual cycle. Quite often real tumours — fibroadenoma — are revealed in these nodes. With prevailing cystic regeneration of the ducts the third — cystic form of mastopathy — is distignished.

In restoration of menstrual-ovarian cycle, especially in the development of normal pregnancy and lactation, the initial forms of mastopathy dis-

appear, especially against a background of satisfactory course of this period of woman life. In the late, sclerotic phase or in unproliferative form of mastopathy, especially at the beginning of menopause, the indurations persist.

Diagnosis of mastopathy is created on the basis of clinical data, and also X-ray and morphological researches, which are possible under conditions of medical establishment. Contrast-free X-ray research of the mammary gland — mammography — allows to confirm mastopathy, to update the dominance of adenosis, cysts or fibrous modifications, to observe dynamics under the influence of treatment, to reveal tumors originating on the background of mastopathy. In revealing large cystic formations pneumocystographya is applied, which enables to update completeness of cyst draining and to reveal intracystic tumoral formation. In suspicion on excretory ducts damage ductography with application of water-soluble contrast reveals the extension of duct system terminal departments. Morphological characteristics of mastopathy can be obtained during cytologic research of discharges from the nipple, fine needle aspirative biopsy of obturating centers.

Treatment. In the diffuse form of mastopathy preference is returned to conservative methods, which are directed on the treatment of disorders of endocrine secretion glands function with the relation to pathogenesis of disease, and diseases of female genitals. At late terms androgens, methylandrostendiol, testosterone or progesterone (at insufficiency of the corpus luteum function) is indicated to women over 40 years. In the case of significant painful sensations in premenstrual period it is possible to use retromammary blockades or electrophoresis with solution of novocain or trimecaine, accupuncture.

Operative treatment is indicated in the nodal form, taking into account difficulties of differential diagnosis with malignant formations: sectoral resection of the mammary gland damaged department with an urgent histological identification.

Folia-like Fibroadenoma

Sometimes folia-like fibroadenoma is called by morphologists phylloid-cystic carcinoma, as if underlining that it is not a purely benign tumor. It is a tumour of mixed (connective and epithelial) structure, with characteristic foliate disposition of tissues, and they are considered as an intermediate link in a circuit "fibroadenomatosis — fibroadenoma — phylloid tumor — sarcoma". Actually not only its mesenchymal (connective) components can be malignizated, but also the epithelial one, that results in emerging of carcinoma (cancer) or, more often, carcinosarcoma.

The foliate form of fibroadenoma differs in the fast growth and reaches large, sometimes huge sizes. In the slit such tumors have a stratified structure, reminding sheets of a book. During histological identification the growths of connective tissue, rich cell-like elements, adenogenous ducts and cysts, covered by proliferative epithelium, sites of intracanalicular and pericanalicular fibroadenomas are revealed.

Clinically these tumors are large (more than 5 cm), sometimes they cover all the mammary gland, with the precise boundaries, with a smooth polycyclic surface. Their consistency is irregular, sometimes intense, changing by sites of softening and fluctuation. The long-term presence in the mammary gland (from several months up to tens of years) with rapid enlargement is characteristic, that is frequently the reason for surgeon consulting. These tumors occur more often at the age from 30 till 40 years, but can also be found out in the other age groups (from 15 till 65 years).

Treatment is operative. Sectoral resection quite often should be expanded to mastectomy in foliate fibroadenoma because of large sizes of the tumor.

Gynaecomastia

Gynaecomastia (from Greek *ginaeikos* — woman + *mastos* — mammary gland) — dyshormonal disease of the mammary glands in men, which consists in their increase up to the sizes of a female mammary gland because of hyperplasia of the gland ducts, which are usually reduced, and connective tissue. Magnification of the mammary gland is less often observed at the expense of redundant development of fatty tissue, a so-called false gynaecomastia connected with application of hormonal therapy in the patients with disorders of prostatic gland function. In these cases, the extension and hyperpigmentation of the areola are simultaneously observed.

The disease can develop in any age, more often after 40 years. Gynaecomastia in youth is less often. True gynaecomastia can be uni- and bilateral, that is determined by the reduction degree of the mammary gland rudiments.

Among etiologic factors the basic meaning has a decrease of androgenous activity — in cryptorchidism, atrophy of testicles, orchitis, tumors of testes, and also in tumors of adrenal glands with increased estrogen activity. Gynaecomastia is quite often found out in patients with adenoma of the prostatic gland. Modifications in mammary glands can arise in chronic diseases of the liver with permanent disorder of detoxicative function, deep disorders of metabolism (chronic protein deficiency). In some tribes of Africa it occurs so often that it is possible to speak about relative endemicity of gynaecomastia.

The disorder of physiological correlations of man's sexual hormones and estrogens produced by testicle and cortex of the adrenal glands under the influence of honadotropic hormone of the pituitary gland lies in the

basis of *pathogenesis* of true gynaecomastia. The second mechanism — disorder of estrogens inactivation in the liver even at usual level of their secretion in the blood.

Gynaecomastia is characterized by growth of the alveolar ducts, small-size and medium milk ducts, which forms small-size cysts. Real acinuses sometimes appear. Then the morphological picture reminds a structure of lobules of a non-lacting female mammary gland. The epithelium of the ducts can proliferate, forming papillomas. Hyperplasia of interlobular connective tissue is simultaneously observed.

Like in mastopathy, they distinguish diffuse and nodal forms of gynaecomastia. Appearance of painful induration which is placed behind the areola, with elastic consistency, indistinct outlines and small-grained surface characteristic for the diffuse form. The skin of the mammary gland and areola do not change. A protrusion of the nipple, serous discharges from it are sometimes observed.

The nodal form of gynaecomastia is, as a rule, an unilateral damage. A dense, painless, mobile egg-like formation with a smooth surface appears during palpation of the gland. The authentic diagnosis of true gynecomastia is made by exam of formation punctate or removed specimen.

The diffuse form of gynecomastia has mainly a cosmetic defect. If there are symptoms of sex function decrease, it can be treated by androgens. In young men with obvious cosmetic defect as a result of the disease it can be corrected with the help of amputations of the mammary gland. In the satisfactory reduction of the gland body the nipple is present. Removing of the mammary gland is indicated at the nodal form of true gynaecomastia. If the nipple is not deformed and not involved, it can be saved. A histological investigation of the removed gland, even in the absence of external indications of invasive growth, is necessary, taking into the account oncologic alarm concerning to this disease.

BENIGN TUMORS OF THE MAMMARY GLAND

Epithelial and non-epithelial benign tumors can develop in the mammary gland. There are frequently fibromas, lipomas, and lymphangiomas among non-epithelial tumors. They have a morphological structure and characteristic clinical signs irrespective of the organ, in which they develop. Most frequently, fibroadenomas — epithelial benign tumors — develop in the mammary gland. In 1978–1981 the experts of WHO offered histological classification of benign tumors of the mammary gland.

Histological classification of benign tumors of the mammary gland:

I. Epiphelial tumors:
1. Intraductal papilloma.
2. Adenoma of the nipple.
3. Adenoma:
   a) tubular;
   b) lactating.
4. Others.

II. Mixed connective and epithelial tumors:
1. Fibroadenoma:
   a) pericanalicular (periductal) fibroadenoma;
   b) intracanalicular (intraductal) fibroadenoma:
   — simple type;
   — cell-like intraductal fibroadenoma.
2. Phylloid tumor (cystic carcinoma, phylloid).

III. Mixed tumors:
1. Tumor of soft tissues.
2. Tumor of the skin.

IV. Non-classified tumors.

V. Dysplasia of the mammary gland (fibrocystic disease).

VI. Tumor-like processes:
1. Ectasia of the ducts.
2. Inflammatory pseudotumors.
3. Hamartoma.
4. Gynaecomastia.
5. Others.

Adenomas of the Mammary Gland

Adenoma of the mammary gland, in contrast to fibroadenoma, occurs in a pure state seldom and in clinical signs reminds fibroadenoma. Frequently it is combined with diffuse mastopathy. Adenoma of the nipple is very rare observed.

Treatment is surgical. It is necessary to make sectoral resection, instead of enucleation of the tumor, because enucleation can result in amplification of proliferative processes in tissues, close to adenoma.

Lipomas

Lipoma of the mammary gland is more often observed in elderly women. Unlike to epithelial tumors located in the gland body, this tumor is located in the subcutaneous fat under the gland, or retromammarily. It is soft, painless, lobular formation up to 8–10 cm in diameter. Very seldom lipomas develop

in the fatty capsule of the gland or in interlobular fat, which causes a strain of the mammary gland sometimes with painful sensation. On mammograms lipomas are seen on the background of parenchyma as the clearly limited enlightenment.

Treatment is operating. Enucleation of the tumoral node can be a method of choice in lipomas.

Papillomas. Papillomas develop in the milk ducts and colligation sinuses. Discharges from the nipple are characteristic for this tumor alongside with induration in the central zone of the gland, they are brownish, yellow-green, less often with blood. Diagnosis of papillomas is made with the help of cytologic research of discharges and ductography. Defect of filling with precise outlines is revealed in the later case.

Treatment is surgical.

MALIGNANT TUMORS OF THE MAMMARY GLAND

Breast Cancer

Breast cancer is a malignant tumor, its primary center arises in parenchyma or excretory ducts of the gland. Taking into account significant frequency of the disease many native oncologic surgeons have devoted a number of fundamental researches to this problem (P. A. Gertsen, N. N. Petrov, and others.).

According to WHO information, breast cancer (BC) alongside with tumors of the stomach and the lungs belongs to three main and most often forms of cancer. The disease more often occurs in older age groups (after 50 years), however, a rapid growth of morbidity rate is observed from 40-year age. Breast cancer in men occurs seldom, and the rate of this disease makes 1:500,000 of male population.

Etiology. Today the judgement about a leading role of dyshormonal disorders, and first of all increased estrogenous activity as one of the causes of cancer of this localization predominate. Diseases and disfunction of endocrine organs (the ovary, the adrenal glands, the pituitary gland, the hypothalamus), hormones of which influence the processes of pathological proliferation in the gland body and ducts of the mammary gland, result in dyshormonal disorders. Inflammmatory processes has an important role in female sexual organs, acute lactation mastitis, especially treated conservatively, disorders of the menstrual ovarian cycle, malignant tumors of female reproductive organs.

Unproductive phase of mastopathy, mastoplasia, plasmocytic mastitis, ductoectasia do not influence the morbidity. At the same time proliferative forms of fibroadenomatosis is a factor of risk, as well as traumas of the mammary gland, inflammatory processes and infiltrates in it, which occur due to a local use of antibiotics. Hence unfavorable influence of a primary sterility, disorder of pubescence and delay of involution (later onset of menstruation, later onset of menopause), irregularity and later onset of sexual life, application of contraceptives, late first labor (at 26 years and older) becomes clear. Low morbidity rate of breast cancer in such ethnic groups, where in connection with the tradition they seldom resort to abortion, early begin a sexual life, deliver children at young age, have more repeated deliveries, normal lactation.

A certain value has a hereditary genetic factor, which is caused by susceptibility of tissue of the mammary gland to oncogenous influences of a hormonal character. Women, among blood relatives of which on the mother's side there are those suffered from breast cancer, have a risk to become ill 9–10 times more often, than women with unburdened heredity.

As a whole the development of BC is a result of action of many factors: hereditary-genetic predisposition, unsufficient reproductive and lactative function, organic diseases of sexual and endocrine organs (obesity, hypothyroidism, diabetes), changes in functions of the central nervous system.

Each of these factors is not specific in appearance of carcinoma, but their combination can stimulate malignant growth.

Pathogenesis. Arising in one of the areas of the parenchyma or ducts of the gland, the center of a malignant growth forms a tumor, which may be non-invasive some time, but under the influence of ethiological factors, tumor cells begin penetrating the tissues of the mammary gland. With progression of the process the infiltration of next tissues of the gland body increases, the tumor spreads through intralobular slots, milk ducts, lymphatic and more rarely capillary blood vessels, and then leaves the limits of the organ. The tumoral cells invade the lymphatic plexuses and block them, changing the outflow of lymph and determining occasionally unusual ways of lymphogenous metastatic spreading. With extraorganic lymphatic system the cells come into the first lymphatic collectors more often into the inguinal sometimes subscapular, parasternal lymph nodes. With further spreading the distant lymphogenous and hematogenous metastasis of cancer of this localization arise.

Pathological anatomy. The both mammary glands are affected by cancer approximately similarly often, but simultanious bilateral involvement is very rare. Most frequently a primary tumor locates in the upper-external quadrant and external lateral half. Sometimes tumors develop outside of the gland body from ectopic adenogenous cells in additional or abberant mammary glands along the pass of the mammary lines. The origin of BC development is the milk ducts, more often small-size. More peripheral localization (lobular, apocrinic carcinoma) makes about 1–2% of all cases of BC. Epidermoid cancer or Paget cancer (2–3.5%) which arises from the transitional epithelium of the nipple immediately in outlet departments of colligation milk ducts.

Non-invasive forms of cancer (intralobular carcinoma and lobular carcinoma *in citu)* and invasive, infiltrative forms of cancer are distinguished. Solid cancer (80–85%), medullar cancer, skirrh, adenocarcinoma are related to the latter. The other forms of cancer including undifferentiated are rare. Skirrhous cancer is revealed more often in men, though adenocarcinoma or intraductal carcinoma can be revealed.

Classification. According to the International Clinical Classification of BC (according to TNM system) they distinguish four stages of the process spreading. A degree of primary tumor spreading in the gland (size, fixation with enclosing tissues, proliferation into the skin, ulceration and presence of other tumors), affection of the regional lymph nodes (their adhesion with each other or with adjacent tissues, swelling of a hand on the side of affection), presence of remote metastasis are taken into account for their definition. The extent of a tumour spread and affection of lymph nodes should be updated by histological identification.

In practical work in our country a clinical anatomic classification is widely used, according to which four stages of tumoral process are also selected:

I stage — a tumor less than 2 cm in the greatest diameter without involvement of the breast surface, adjacent tissues and without affection of regional lymph nodes;

II stage — a tumor from 2 up to 5 cm with presence of skin symptoms, affection of single lymph nodes of the first collector;

III stage — a primary tumor from 5 up to 10 cm or any size, but with affection of the skin integuments, penetration in outer fascial and muscle strata and promoted metastases into the regional lymph nodes;

IV stage — spreading of the tumoral process with dissemination throughout the skin or extensive ulceration, fixation of a tumor to the thoracic wall or

independently from condition of a primary tumoral center, but with remote metastases.

Clinical course. The manifestations of BC largely depend upon the localization and size of the primary focus, its relation to subcutaneous fat of the affected mammary glands, nipple and areola, intraorganic and extraorganic lymphatic system and blood vessels. As a whole a primary clinical investigation (examination of not only the mammary gland, but the whole upper half of the trunk, purposeful palpation of the mammary gland, lymph nodes in the inguinal hollows, neck, supraclavicular and subclavicular zones) enables to receive an idea about a character of the process and form of the tumor.

The main clinical form of cancer is nodal, characteristic presence of the tumoral node in the gland body or in the central zone. A node has a spherical or star-shaped form. Cancer is characterized by absence of precise limits of induration, gradual transition into the external tissues and cartilaginous consistency without painfulness. Only in some histological variants (pappilomatous, mucous or colloid carcinoma) the consistency of a tumor can be softer, even hard elastic.

A "wrinkled skin" symptom discribed by P. A. Gertsen is a characteristic feature for small tumors in the gland body. The symptom arises due to shortening of Couper's ligaments and allows to distinguish cancer from mastopathy. With central localization of the tumoral node due to the same circumstances there reveals a contraction of areola, nipple retraction, its deviation to the side of the node. The positive pronounced Kenig's symptom: the node does not disappear on press to the thoracic wall in a lying position.

In more significant tumoral infiltration during a primary examination the signs indicating disorders of lymphatic flow in underareolar plexus — the symptoms of umbilication, a ground, with central localization — Pribram's symptom, Crouse's symptom (edema of the areola) are revealed. The symptom of "orange peel" testifies to the advanced process, as it is a result of cancer embolism of deep lymphatic vessels with swelling of the skin. In skirrous forms of cancer tension of the gland in accordance with the growth of a tumoral node is observed. With proliferation of the tumor to the edge of the glandular disk the nipple displaces to its direction.

Diffuse forms of BC are more malignant. Swelling — in filtrative, mastitis-like, erysipelas-like, testa-like forms are recognized according to the clinical manifestations. Two first forms develop at young age, but more frequently in a period of pregnancy and lactation progress roughly. Large metastases in lymph nodes are early found out. Testa-like form is more characteristic of elderly women, and though the tumor in these cases grows more slowly, this form is unfavorable.

Paget's cancer is revealed in patients at the age of 45–70 years and is characterized by affection of the nipple causing the formation of scales and pathes or ulcers, wet eczema-like changes which are associated with pruritus, sensation of priking, unusual pains in the nipple area. During 1–3 years a primary affection of the nipple slowly progresses, papilla becomes thicker, it is deformed, retracted and may absolutely disappear. To this time in the gland body a tumoral node occurs, appearance of which depends on invasion of carcinoma into the ductal system. Sometimes such node is a single menifestation of this form of cancer, that enables to single out not only a so-called psoriasis-like form, but also a nodal form of Paget's cancer, a typical histological picture of which (rounded large light cells) is revealed only as an operational find.

BC develops more slowly in men. A tumor seldom reaches large sizes and, as a rule, locates subareolarly. Frequently the disease develops against on a background of gynaecomastia. Some men do not assume that they have cancer, and consult a doctor lately when the tumor invades into the skin and is covered with ulcers, the infiltration of the thoracal wall is observed. Unsufficient oncologic suspicion during a primary visit to a doctor may lead to diagnostic mistakes.

Clinical course. In the most often nodal form of BC up to the moment of diagnosis establishment regional metastases are revealed in 50% of women, and in 6–12 months — in all of them. In slowly developed cancers and especially on intraductal localization of the tumor the regional metastases arise later. The diffuse form metastazes more intensively, whereas the course of Paget's cancer (up to the moment of appearence of a palpated tumor) in the gland is favorable and metastases are observed seldom. With the appearence of a node in the gland body Paget's cancer has a rapid course with a simultaneous appearence of metastases. To the moment of detection of a tumoral node in men metastases in the regional nodes are found out more often than in women (57–76% of all patients).

A gradual development of a tumoral blockade of metastases in lymph nodes results in compression of vessels and nerves in the inguinal area with painful neuralgiae and edema of the hand. The blockade may change a route of lymph outflow and result in additional metastatic spreading into the skin and lymph nodes of the contralateral gland.

Remote metastases may appear both in early terms after removal of a primary tumor, and in 15–20 years. However, most frequently they arise in the first 2–3 years after the establishment of a diagnosis. Most often metastases are observed in the bones (the vertebrae, the femoral, pelvic, shoulder bones, the sternum) less often in the lung and the pleura (13–14%), the liver (9–11%), the brain (about 3%). Remote metastases have their own symptoms, due to their localization.

Diagnosis. Progress in revealing of early forms of BC is largely associated with introduction in clinical practice of special methods of research: X-ray examination, various kinds of biopsies and cytologic researches. However, it does not mean that traditional methods of clinical diagnosis of tumors on relatevily early stages of their development lose the significance. In the most cases even in "minimum" forms of BC (a tumor less than 1 cm) correctly and completely conducted clinical inspection gives a doctor sufficient information for establishment of diagnosis or planning of further diagnostic researches. An experienced physician on careful examination is able sometimes to diagnose indurations in the mammary glands sized 3–5 mm.

On diagnosis of BC the objective indications have decisive significance, but it should not neglect the family and history data. It is necessary to pay special attention to a presence of pathogenetic features considered as the factors of risk of BC development. Today, it is established that factors which characterise a function of reproductive system, hereditary-constition features, accompanying and preceded disease, endocrine-metabolic changes play not similar role in patients with BC of different age.

Factors of risk in women younger than 35 years are as follows:

1. Euthyroid and hypothyroid enlargement of the thyroid gland.
2. Late (after 28 years) first delivery.
3. BC in blood relatives.
4. Preceding or accompanying dyshormonal hyperplasia of the mammary glands (cystic fibroadenomatosis with ductal or lobular proliferation of the epithelium, intraductal papilloma, cystic adenopappillomas).

In women of 35–49 years old the most essential factors of risk of development of BC are:

1. Early (till 12 years) beginning of menstruations.
2. Disorders of the menstrual function (irregularity, abundant menstrual bleeding, and painful menstruation).
3. Disorders of a sexual function (absence of orgasm, frigidity).
4. Disorders of the delivery function (primary, secondary sterility, late first or last delivery).
5. Inflammatory and hyperplastic processes of the uterine appendages.
6. Chronic hepatopathy.
7. Cancer of the mammary gland in the family history.
8. Dyshormonal hyperplasia of the mammary glands.

In women of 50–59 years old, who are in pre-or menopause, the increase of risk in development of BC is associated with persistent increase of function of the adrenal glands and appropriate clinical signs

of hypercorticoidism. Here the following features have a great importance:

1. Hypertension (10 years and more).
2. Obesity of the III degree (5 years and more).
3. Atherosclerosis (5 years and more).
4. Diabetes mellitus.
5. Birth of a large (4 kg and more) fetus.
6. Late (after 50 years) beginning of menopause.
7. Fibromyoma of the uterus.

For the women aged 60 years and older, being in deep menopause, the factors of risk are following:

1. Combination of early (under 12 years) beginning of menstruation and their late termination (after 50 years old).
2. Late first and last delivery (after 40 years).
3. Long-term (more than 10 years) absence of sexual life in the reproductive period.
4. Combination of hypertension, obesity, atherosclerosis, diabetes mellitus.
5. Malignant tumors of female sexual organs and colon in the family history.

Women who have three features of appropriate age group are refered to a "group of risk" of BC. According to the experience, about 20% of the whole group primary examined women are usually referred to a "group of risk".

Correctly taken anamnesis allows also to receive an idea about the first signs of disease and rates of its development. Cyclical changes of sizes and consistency of a tumor and pronounced painful sensation in the mammary glands before menstruations are more characteristic for dyshormonal modifications. The complaints of the patients to a sudden rase in growth of a tumor, change of its consistency, deformity of the areola, retraction of the nipple, change of the skin above induration, appearance (or cnange) of the colour of discharges from the nipple are rather typical for cancer. At the same time the absence of symptoms in the history, characteristic for malignant tumors, in any case does not exclude the presence of a tumor, especially at the early stage. So, in patients of elderly age small carcinomas of the mammary gland (up to 0.5 cm) may long time exist without visible changes of their size and form (so-called involute forms), that sometimes serves as a condition for mistaken explaining process as benign.

Diagnosis. The examination of the mammary glands is necessarily carried out in well illuminated room at some distance from an undressed to the waist patient first with hanged hands, and then hands are put on the head. On such conditions, pathognomic features for BC may be noticed easier:

1) change in disposition of the nipple;
2) deformation of the areola and outline of the gland;

3) retraction of the skin;

4) more developed subcutaneous vessel network, a possible asymmetry of the affected gland position.

In "minimum" forms of BC changes in position of the nipple may be hardly noticed and found out only in comparison with a healthy mammary gland. The nipple to a greater or lesser extent deviates to the side of a tumor, quite often is flat. Sometimes the skin of the nipple and areola as well as ajacent areas are transformed into the wet surface with rough, slightly flat roller-like edges or is covered with dry peels (cancer of Paget's type).

With localizations of the tumor in the para-areolar zone the features of its deformation may be revealed — shortening the radius on the one hand, appearance of flat areas, protrusion of the areolar circle. Retraction of the skin above the tumor is more expressed in cancers of skirrous type or located closer to the skin. This symptom is better revealed on examination of the mammary glands, when a patient keeps his hands lifted.

Palpation is carried out in vertical and horizontal positions of a patient that allows defining the localization, sizes, boundaries of the tumor, its form, consistency and character of the surface, as well as interconnection with surrounded tissues. First it is carried out by easy touches the tips of fingers, and then to a deeper palpation.

Tumor consistency change in transition of the patient from a vertical position into horizontal, softening the edges on preservation of hardness of the central part of the tumor, pronounced increase of the tumor from periphery to the centre are characteristic of cancer. Small flattenig and retraction of the skin above the tumor invisible for approaching eye, become more pronounced and marked on easy compression of the skin on the borders of the tumor and approaching to the centre (symptom of "wrinkling"). This sign is rather frequently revealed in cancer and considerably less often in infected cysts of the mammary gland. In superficial (close to the skin) located "minimum" cancers the symptom of a "platform" and "umbilication" is possible.

Palpation of the mammary gland in a horizontal position can significantly facilitate the diagnosis of "minimum" tumors, in particular the differential diagnosis with dyshormonal hyperplasia. In this position the whole mammary gland, as a rule, becomes softer, that allows better to reveal small area of induration in it. Besides, in a horizontal position of a patient the area of hyperplasia either become noticeably softer on touch or is not palpated at all, while a tumoral node does not change its consistency in any position.

An obligatory component of examination of the patients with a pathology of the mammary glands is careful observation of the zones of regional metastatic spreading, such as: the axillary, subclavicular, supraclavicular and neck lymph nodes. In "minimum" cancers (up to 1 cm) the affection of

regional lymph nodes by metastases is found out in 15–20% of the patients. Just in such patients the detection of regional metastases serves as a starting-point for diagnosis of BC.

The clinical detection of the "minimum" forms of BC (especially on early stages), when there is not any persistent, obligatory symptom as well as the absence of any specific indication of a malignant tumor do not prove that it is absent. The absence of typical symptoms, characteristic for initial stages of BC, forces to involve some additional diagnostic techniques: biopsy, cytologic studies of discharges from the nipple, mammography, ductography and echography.

A regular self-inspection of the mammary glands is one of the important components of diagnosis, but it cannot detect some forms of BC, especially in young women.

Indications of pretumor and cancerous diseases of the mammary gland are: painful swelling of the mammary glands some days prior to the beginning of menstruation, dense node in the mammary gland, discharges of droplets from the nipple of yellowish colour or bloody liquid (appearance of spots on the bra), change of the form of the mammary gland, retraction or protrusion of skin covers and retraction of the nipple.

The best time for self-inspection is the first week after the end of the menstruation. The women who have no menstruations, should inspect their mammary glands every month at the same day.

Treatment of BC is carried out by three methods: operative, combined and also by a combination of surgery and radiation, or surgery and chemotherapy, or by a complex treatment including all kinds of medical effect. The program of treatment is created according to the stages of disease, age of the patient, general condition, character of accompanying diseases, menstrual-ovarial function. Therefore with suspicion on BC a further inspection and treatment at the specialized in-patient department appears to be necessary.

There are four principles lying in the basis of surgical treatment, among them: radicalism, ablastism, antiblastism and rational prevention of relapses and metastases.

The development of the principles of operative treatment has been started by N. I. Pirogov (1847), who regarded that it was necessary to combine removing an affected mammary gland with removing fascia of the greater pectoral muscle, lymph nodes and a fat of the axillary crease. Three main variants of radical surgery are used today. The first variant is Halsted — Meyer's radical mastectomy — removing the mammary gland together with the greater and minor pectoral muscles, fat and regional lymph nodes of axillary and subscapular areas. In a cancerous node in medial quadrants the ex-

panded radical mastectomy — removing the mammary gland with the greater and minor pectoral muscles, inguinal, subscapular and peripectoral lymph nodes may be applied.

In limited nodal tumors in some cases Paty — Dison's mastectomy — removing the gland and the minor pectoral muscle with the first main three lymphatic collectors (apical, axillary, subscapular) with a preservation of the grearter pectoral muscle is proposed.

With mences preservation in women and during the first 5 years after the beginning of menopause ovarioectomy with consequent long-term use of androgens (Sustanone-250) or prednisolone in women with high contents of sexual chromatin in cells of a tumor is necessary. The operative treatment as a unique method may be recommended only in I–II stages of cancer and less often in Paget's cancer. In the rest cases it should be combined. In rapidly growing tumors of an infiltrative type a preoperative irradiation with the purpose of devitalization of cancerous cells and prevention the threat of their dissemination as well as implantation in tissues of the operative wound are indicated.

BC is peculiar not only by high frequency of remote metastatic spreading, but also involvement of various organs that may result in death. There is inversive relation between the stage of the process, at which the treatment began and general life-span of the patients. In widespread forms of cancer, even with complex treatment, the majority of lethal outcomes take place during the first 2 years of treatment, five year survival rate in such patients is no more than 50%. In the first stage of Paget's cancer and the absence of a tumoral node in the gland within 5 years up to 92% of the patients live, 10 years — up to 87% of the patients.

Preventive therapy of BC should be carried out in two directions. In women of a dangerous age (40 years and older) it should be the observation at the dispensary with the use of mammography, macroframe photoroentgenography of the mammary gland, especially in the presence of the major factors of the risk: disorder of ovarial-menstrual activity, preceding benign tumors of the mammary gland, family predisposition. The second direction is a duly treatment of precancerous diseases, including surgery, for example unilateral nodal gynecomastia in men of mature and elderly age.

Sarcoma of the Mammary Gland

Sarcoma of the mammary gland develops from the elements of the connective tissue and occurs rather seldom, making from 0.6 up to 2% of all malignant formations of the mammary gland. More than 50% of cases of this disease occur in the third-fourth decade of life.

The origin of sarcoma is frequently connected with a previous trauma of the mammary gland, which is considered to be a provoking factor which acts against a background of genetically determined predisposition.

Predisposing factors, apparently, are dyshormonal disorders, exhibited in absence of reproductive function in women with diseases of female sexual organs, obesity, and diabetes mellitus.

Classification. Sarcomas of the mammary gland are divided into: the tumors arisen from stromal elements of fibroadenomas and phylloid tumors by way of malignization, and into tumors developed from the stroma of the intact tissue of the mammary gland.

The first group is characterised by the presence of epithelial structures among the fields of sarcomatoseous tissue. Such structures in tumors of the second group are not determined.

According to WHO classification of tumors of the mammary gland, sarcomas are divided into those arisen from cells of intraductal fibroadenoma (that is foliate tumor) and other kinds of sarcomas. Angiosarcomas (25.9%) and hematosarcomas (22.2%) occur more often.

Stromal sarcomas due to their biological essence are sarcomas of soft tissues located in the mammary gland.

The tumors of the first group arise more often in preclimacteric period and are revealed at the age of 24–50. In girls they occur seldom. Stromal sarcoma mainly arises during a menopause at the age of 49–70. On appearance a sarcoma represents tuberous nodes, usually precisely limited. In some cases the capsule is saved. On a section a sinuous character of the structure is evident. Tissue of fibrosarcomas is pale pink. Fusiformcellular and polymorphocellular sarcomas on a section are motley, due to the presence of areas of bluish, red colours, browish and yellowish shades. In the centre of the node areas of necrosis may be found.

The specific symptoms of the disease are absent. Its signs depend on the type of sarcoma.

As it was already specified, sarcoma frequently arises due to trauma of the mammary gland, during pregnancy and lactation, against a background of accompanying gynecological diseases: primary sterility, uterine fibromyoma, cervical erosion, polyposes of the endometrium.

The main clinical symptom, being a defining complaint of the patients, is appearence of a dense tumoral formation of various size with clear outlines, usually painless, with expansive growth. More often large tumors (more than 10 cm in a diameter) are revealed, which results in change of the organ or enlarged gland if neoplasm involves the whole organ.

In late periods of the disease course there can occur the thinning of skin integument with a blue-red shade, extension of superficial veins, change of

the skin above the tumor in a shape of “orange peel”, “umbilication” or protrusion of the nipple.

For hematosarcomas the early affection of separate or all groups of regional lymph nodes is characteristic. In other histological variants the nodes are rarely palpated and metastases are not found out in them on histological examination.

Clinical course. Clinical manifestations and growth rate of a tumor depend on its histological appearence. For sarcomas that develop from fibroadenomas a more favorable course, long-term anamnesis (2–24 years), rare proliferation into the skin and tissues of the anterior thoracic wall are characteristic. Accordingly the skin symptoms occur rarely (about 30% of the patients). Metastatic spreading and affection of the regional lymph nodes are not typical.

Stromal sarcomas are characterised by rapid growth (for 7–8 months up to 8–10 cm), short anamnesis, proliferation into the skin and tissues of the anterior thoracic wall, affection of the lymph nodes and metastatic spreading. The skin symptoms are revealed in 90% of the patients.

A rapid increase and growth of a tumor after thermal and physiotherapeutical procedures is characteristic for both groups. Metastatic spreading into the lungs, liver, sometimes bones happens mainly by hematogenous way.

Differential diagnosis is carried out with fibroadenoma, nodal form of mastopathy, cancer of the mammary gland — a long-term preservation of mobility of a tumor in relation to surrounding tissues is characteristic for sarcoma. On study of anamnesis one should pay attention to a possible genetic predisposition to malignant neoplasms, and also to a trauma or inflammatory diseases of the mammary gland in the past; a long-term existence with further rapid growth of formations or primary rapid growth of the tumor and enlargement of its sizes.

On a mammogram sarcoma looks like a massive round formation of a various form, more than 10 cm in size with precise outline. Stromal tumors have the appearance of a conglomerate with indistinct tuberous outlines and tuberous surface. The intensity of a shadow is high, structure is heterogeneous.

Sarcomas, developed from fibroadenomas, on a mammogram look like indurations with sinuous, sometimes, tuberous outlines. Intensity of the shadow is high, the structure is homogeneous.

On diagnosis of the disease US, galactography and scintigraphy are used. High-informative method is paracentetic, aspirative biopsy with further histological, cytological exam of paracentetic and aspirative material, including urgent intraoperative exam.

Treatment depends on the type of the tumor. The main method is surgery. The majority of the authors consider a simple mastectomy with axillary lymphadenectomy to be an operation of choice in sarcoma developed from fibroadenomas. The operation of choice in stromal sarcomas is Hallsted's radical mastectomy.

The operation is added by radiation therapy or chemotherapy (cyclophosphamide).

A sectoral resection should not be applied because of high frequency of local relapses (up to 76.1%).

Organopreserving operations are possible in small sizes of a tumor without affection of the lymph nodes. The prognosis depends on histological type and stage of development of sarcoma.

A medium life-span after operation in hematosarcoma is 22.3 months, in angiosarcoma — 11.2 months, in other types — 62.5 months. It is minimal in rabdomiosarcoma and maximal in fibrosarcoma.

Bleeding mammary gland. Bleeding mammary gland is a symptom which is characterised by pathological discharges from the mammary gland irrespectively of the menstrual cycle, pregnancy and period of lactation.

The cause of appearence of pathological discharges may be extramammary and intramammary factors. Extramammary causes are traumas and bruises of the mammary gland. The most often intramammary cause is benign or malignant tumors of the mammary gland or intraductal papilloma, isolated or multiple fibroadenoma. Hyperplastic proliferation of the endothelium with the formation of granulated tissue in passages and acinuses develops.

Pathological discharges may be unilateral, less often — bilateral, liquid, serous, milk-like, sometimes dense, greaselike. Secretion may be discharged out spontaneously or on hard pressing, from one or several orifices or may be discharged out in a jet.

The discharges may occur in unchanged sizes and form of the mammary gland, on growth of the adenogenous tissue.

Standard diagnostic researches are:

— mammography;

— galactography;

— histological analyses.

On cytologic research of pathological secretion, two groups of cells are distinguished:

1. Non-tumorous:

— intraductal epithelial cells (in intraductal papilloma);

— foamy (most frequently occur in a secretion);

— the cells of the colostrum (in pregnant women, feeding mothers, even in discharges from mammary glands of children);

— macrophages;

— cells of blood (a grand number of polymorphonuclear leucocytes and lymphocytes in case of inflammation or tumors in great amounts);

— epithelial cell (are determined accidentally as keratosic, polygonal cells).

2. Tumorous cells.

The positive result confirms the diagnosis of breast cancer, the negative one does not exclude it.

Pathological discharges from the mammary gland specify a pathological process, more often papilloma, cystoadenoma, advanced mastopathy or other kind of epithelial prolifiration. It has a precancerous character. Bloody or like "meat slops" discharges — it is the extremely serious symptom, a sign of alarm which manifestates any tissue change. A drop of blood, spontaneously or on pressing appearing from the nipple, quite often may be the only indication of latent non-palpated malignant growths. The first discharges with bloody secretion occur, as a rule, after the trauma of the mammary gland, but only in 75% of cases in bleeding mammary glands on palpations a tumor is revealed.

Colourless liquid discharges from the mammary gland wth hypertrophies are connected with the period of beginning a sexual maturing or in pregnancy.

Serous secretion which is discharged out in the premenstrual period, usually specifies mastopathy.

Treatment of bleeding mammary gland consists first of all in treatment of the main disease, manifested by this symptom. In a palpated tumor or grounded suspicion its removing and histological investigation are indicated.

Lecture 5
INTESTINAL OBSTRUCTION

Intestinal obstruction is a syndrome complicating the course of a series of diseases and pathological condition. A pathological chain of the intestinal obstruction is a complete or partial arrest of passage of a chyme within intestinal tube, due to mechanical obstruction or a disorder of motor function in the intestines.

Acute intestinal obstruction occupies one of the leading places in urgent abdominal surgery and makes up to 10% from all urgent surgical diseases of organs of abdominal cavity. More often it arises in men (55%) and less often in women (45%). More than 35% of the patients with acute intestinal obstruction are older than 60 years with a lethality reaching 20% (V. R. Petrov, I. A. Yeryukhin, 1989).

Classification of intestinal obstruction according to

Origin:
1. Congenital.
2. Acquired.

Course:
1. Acute.
2. Chronic.

Mechanism of development:

I. Mechanical:
1. Obturative (occlusion from inside and prelum from the outside of intestinal tube by tumours, helminths, fecaliths and gall-stones).
2. Strangulative (incarceration of the intestine, volvulus, nodulation).
3. Mixed (adhesive, strangulated hernias, invagination).

II. Dynamic:
1. Spastic.
2. Paralytic.

— localization of a mechanical obstruction:
a) Small bowel: high and low.
b) Colonic.

The cardinal difference of mechanical intestinal obstruction from paralytic is that in the first case there is a mechanical obstacle of the intestinal passage, as the lumen of intestine is blocked either from inside, or from the outside, and the second case — the lumen of the intestine is free. The dynamic intestinal obstruction makes from 1 up to 2% from all kinds of this pathology.

The division of mechanical intestinal obstruction into strangulative and obturative is based on that in strangulation intestine and its mesentery involved into the pathological process, and in obturation — only the intestine. The strangulative intestinal obstruction occurs approximately in 30% of cases and 70% are obturative and mixed kinds of obstruction. From 60 up to 70% of all kinds of acute intestinal obstruction are obstructions of the small bowel, and more than in 75% of cases the pathological process being localized in the ileal intestine (low small bowel obstruction), rather frequently in its terminal department.

Malignant tumours of the colon are frequently (in 26%) complicated by development of obturative obstruction, that makes 80–90% of all kinds of colonic obstruction. In 10–16% colonic obstruction is caused by volvuluses of its various departments, more often of the sigmoid intestine.

On research of mesenterial microcirculation in acute intestinal obstruction it was established, that both in obturation and strangulation the bloodflow in the intestinal wall is damaged and then is blocked at all at the place located higher of the mechanical obstacle. The similar changes of microhaemodynamics are marked as well as in paralytic obstruction. The strangulative intestinal obstruction from these positions may be regarded as a combination of all kinds of obstruction because both obturative and paralytic components are formed above the place of strangulation.

Adhesions in the abdominal cavity may make "a double-barrelled gun" from the intestine, promoting obturation of its lumen in this place, and the intestine may be "strangulated" by adhesions, therefore adhesive obstruction is considered to be mixed. Strangulated hernias belong to the same group because the incarceration of the intestinal wall (Richter's) causes obturative intestinal obstruction, and strangulation of a segment of intestine with the mesentery — strangulative. Invaginations of small areas of the intestine do not damage the mesenterial bloodflow, that is necessary to regard as obturation, at the same time a mesentery is involved in the pathological process on long invaginated segments of the intestine.

The chronic form of intestinal obstruction is admitted by not all researchers, but it is necessary to notice that in some diseases (tumours of the intestine, multichamber postoperative ventral hernias, adhesive disease etc.) the conditions for partial (incomplete) blocking of the intestinal lumen are formed. In this case the disease is characterised by attacks, which is similar

to obstruction in its clinical picture. After conservative treatment the period of remission take place.

Etiology. In etiology of acute intestinal obstruction factors which promote the development and directly result in obstruction are distinguished. Anatomo-morphological changes in the abdominal cavity causing the development of intestinal obstruction are:

— adhesions and adnations in the abdominal cavity, various dense formations in the intestinal lumen (fecal and gall-stones, helminths, tumour etc.);

— congenital malformations of the intestine (Meckel's diverticulum, malrotations, superfluous length of the intestine and its mesentery, pockets in the parietal peritoneum etc.);

— superfluous mobility of various departments of the intestines.

The causes which directly result in obstruction are: excessive motor activity of the intestine, its spastic conditions, food particularly rich in hard digested fat, a sharp rising of intra-abdominal pressure and other causes. These and other causes may realize the contributing factors and result in development of the acute intestinal obstruction syndrome.

The leading place among the reasons of occurrence of acute intestinal obstruction belongs to adhesive process in the abdominal cavity developing after inflammatory diseases and after operative measures, that occurs much more often (M. I. Lytkin et al., 1982), leading position of which belongs to appendectomy as the most often transabdominal operations, gynecologic surgery takes the second place.

Separated adhesions in the abdominal cavity are capable to press a segment of the intestine with its mesentery, causing a strangulative intestinal obstruction. Around these adhesions the intestine can be also turned. Volvulus of the intestinal loop with mesentery occurs around the longitudinal axis and magistral vessels by 360–720°. Mesenteric vessels thrombosis, ischemia of the intestine with its further necrosis develop. Most frequently there is a volvulus of the sigmoid intestine in the presence of cicatrical changes in its mesentery.

Nodulation, the reasons and mechanism of which have not been found out yet up to now occurs more often between the small intestine and sigmoids, but the nodes may be formed between the loops of the small intestine with a long mesentery which are squeezed on overlaping. Quickly there comes a gangrene of both sites of the intestine which participates in nodulation.

Discoordinative motor function of the intestine in the presence of adhesions, helminths, tumours, inflammatory diseases results in squeezing of the spasmodic area of the intestine into the adjacent one causing invaginative intestinal obstruction. Invagination may occur in various sites of the

intestinal tube, but most frequently it occurs in a terminal department of the ileal intestine as "thin in thin" and also in the area of the ileocecal angle — "thin in thick".

In proportion to oncologic diseases incidence the number of the patients with acute intestinal obstruction increases on these grounds, and quite often before complete blocking a lumen of the intestine there being some period when the features of chronic (incomplete, partial) obstruction are marked.

Hernias are the frequent cause of intestinal obstruction as the contents of the hernial bag on squeezing in more than 60% of the patients are various departments of the intestine.

Acute infectious diseases (typhoid, enterocolites etc.) have a course with phenomena of paralytic ileus.

Paresis of the intestine is an obligatory component of peritonitis, quite often with intra-abdominal abscesses and other infectious centers in the abdominal cavity. Besides, paralytic intestinal obstruction is a mirror which reflects an unfavourable course of postoperative period after abdominal surgery.

Disorders of homeostasis, in particular, electrolyte and albuminous, promote a progression of paresis of the intestine in operated patients.

Severe pareses of the intestine are observed in patients with thromboembolism of the mesenterial vessels, the longer ischemia of the intestine, the more severe course of dynamic intestinal obstruction.

Congenital maldevelopments of the intestine and its nervous apparatus are the factors causing both chronic and acute intestinal obstruction. Chilaiditi's syndrome — location of the right flexure of the colonic intestine between the diaphragm and the liver: Hirschprung's syndrome — a hereditary megacolon, Jurasek — Zueltzer — Wilston syndrome — aganglionar (absence of Auerbach's plexus) megacolon, Ledd's syndrome — hereditary abnomality of the intestines and pressure of the duodenum by cicatrical chordas, Marfan's syndrome — an extremely long intestine, Ogilvie's syndrome — disorders of sympathic innervation of the intestines, Piulachs — Hederich's syndrome — a combination of dolichosigma and narrowing of various departments of the intestinal tube.

Pathogenesis. The existence of numerous theories of pathogenesis (the theory of water-electrolyte and protein balance disorder, infectious, intoxicative, nervous-reflectious etc.) on the one hand, testifies about the complexity of mechanisms in development of acute intestinal obstruction, and on the other hand — about insufficient research of this pathological process.

The study of mesenterial bloodflow on macrolevel has allowed assuming that the main role in pathogenesis of acute intestinal obstruction syndrome belongs to disorder of regional mesenterial haemodynamics.

Both on obturation and strangulation above the place of the mechanical obstacle in the intestine the zone of increased intra-intestinal pressure is formed. This mechanical force is transferred to the wall of the intestine stretching it and creating additional intramural pressure. In microvessels of the intestinal wall the rate of blood flow is reduced and stasis of formal elements of blood occurs. The similar changes are marked as well as in paresis of the intestines. Up to a certain moment such damages of microhaemodynamics are compensated, convertible, but in progression of pathological process decompensation of microcirculation appears and only since that period it is possible to speak about the formation of the acute intestinal obstruction syndrome.

In capillaries, arterioles and venules the bloodflow rate is reduced up to the complete stopping, the units from elements of blood are formed, the quantity of not functioning microvessels is progressively increased. The permeability of their wall rises, in venous and lymphatic vessels the pressure grows, the consequence of which is intestified output of liquid part of the blood and formal elements outside the vascular wall.

Intestinal edema, diapedetic hemorrhages, inhibition of the intestinal wall by blood occur. Then a transudate occurs in the abdominal cavity, first of yellowish colour due to transuding a liquid part of the blood and lymph, and later it gets hemorrhagic.

The damages of microhemodynamics are transferred on the macrolevel, in the arteries and veins the pressure gradually decreases reaching the critical point — systolic pressure is lower than 40 mm Hg.

Haemodynamic disorders in the intestinal wall cause a disturbance of oxidation-reduction processes, hypoxia develops. Strangulated sites of the intestine are exposed to necrosis. In suprastenotic departments, above the place of obturation and in the paretic intestine acute ulcers are formed in the beginning, which quite often are complicated by intra-intestinal bleeding and later the local necrosis with perforation of the wall of the hollow organ is possible.

Increase of permeability of the vascular wall and rising pressure in blood and lymphatic vessels cause a loss of liquid, electrolytes and proteins both in the intestinal lumen, and in the peritoneal cavity, that results in deficiency of these components in an organism.

The intestinal wall in hypoxia loses its barrier function. The microorganisms from the intestinal lumen penetrate into the abdominal cavity, inseminate a transudate, promoting development of one of the most serious complications of acute intestinal obstruction — peritonitis.

Besides, a favorable medium for intensive reproduction of microflora, occurrence of dysbacteriosis and development of pathogenic properties by

conventional-pathogenic flora are formed in the intestinal lumen. In such conditions microflora and the products of its vital activity getting into the blood begin to occupy a dominant position in development the endogenic intoxication syndrome and may serve as the reason for sepsis.

The disorder of oxidation-reduction processes in the intestinal wall results in occurrence of nonoxidate products, which is one of the components of a complex mechanism of intoxication. Due to hypoxia of the intestinal wall and pathological condition inside its lumen parietal digestion is disturbed that also promotes development of intoxication in a high degree.

The flow of nervous impulses from a strangulated segment of the intestine with mesentery or its suprastenotic department directly through the nervous centers influences negatively the regulation of blood and lymphatic vessels, motor and secretory functions of the intestines. The role of the nervous system is noticed in strangulation of large segments of the intestines or nodulation, when a great number of pain receptors of mesenteric nerves are involved in pathological process that may result in shock.

Strangulation, the volvuluses of large segments of intestines or nodulation cause a sequestration of great amount of blood in the intestine, involved in pathological process, that influences common hemodynamics negatively, damages homeostasis roughly and cause anemia in the patient.

A distinctive feature of obturative intestinal obstruction is that the pathological process develops slowly, exhaustion of compensative mechanisms occurs not so rapidly as in strangulative ileus. Internal medium of organism still is damaged to a lesser degree in chronic intestinal obstruction in which during the periods of remission up to the next exacerbation the damaged homeostasis is restored completely or partially.

Clinical course. A classical and the most constant feature of acute intestinal obstruction is spasmodic pains. In obturative forms their intensity and frequency increase gradually. For strangulation (volvulus, nodulations) owing to irritation of numerous receptors of the mesentery a typical onset is persistent, extremely intensive pains which gradually weaken and change into spasmodic. Paroxysmal character of pains is necessary to regard as protective reaction, because by amplified hyperperistalsis the organism attempts to overcome a mechanical obstacle. On exhaustion of the nervous-muscular aparatus of the intestines the intensity of paroxysmal pains is reduced, they become constant without precise localization — paresis of the intestines occurs.

Reflex protective reaction of an organism is also vomiting, which happens one time or repeatedly and is expressed in all patients differently (in patients with obturation of the intestine more often repeatedly). After vomiting some relief quite often occurs, but the intensity of pain sensations

is almost not changed. On accumulation of contents and elevation of intraintestinal pressure a vomitive attack may be repeated. In severe obstruction vomiting occurs in early term after the onset of disease, in mild (the large intestine) — more lately. In case of complication of intestinal obstruction by peritonitis frequent vomiting does not relieve the general state.

Acute intestinal obstruction is frequently accompanied with stools and gases retention. At the beginning of the disease the patient may undertaken repeated attempts of evacuation of feaces, and due to emptying the intestine under the obstacle a defecation may be scanty but later due to development of deep paresis of the whole intestines which becomes deeper. Digital rectal examination may reveal the empty ampoule of the rectum and gape sphincter of anus (Obukhov's hospital sign).

Due to progression of pathological process and accumulation of contents and gases in the intestine abdominal distention may occur. If on initial stages of the disease in subtile patients hyperperistalsis and conturation the loops of the intestine may be detected through the abdominal wall, in later terms the abdomen is even inflated in all departments, tympanitis with a metal shade is revealed by percussion — Kivul's sign.

Certain dynamics of changes of peristaltic function of the intestine is characteristic for mechanical intestinal obstruction. With development of the disease the increase of intestinal murmurs reaching a maximum at height of spasmodic pains is defined on auscultation. Gradually intestinal murmurs acquire a sonorous musical shade. Then the intensity of intestinal murmurs is reduced till complete disappearance paresis of the intestines occurs. From this moment Sklyarov's sign is most clearly detected — capotement in the intestine on slight concussion of the abdominal wall. In strangulative intestinal obstruction the positive Shchotkin — Blumberg' sign is determined locally in a projection of the pathological center. At the same time in paralytic and obturative intestinal obstruction this sign is either unconvincingly expressed or negative.

The temperature of the body in the patients with acute intestinal obstruction, as a rule, is subfebrile, and only on complication by peritonitis and serious paresis it exceeds 38°C. The reaction of white blood is detected by a moderate leucocytosis $9–12 \cdot 10^9$/l with a left shift. Higher leucocytosis is usual in strangulative forms of acute intestinal obstruction, and there occuring a directly proportional dependence — the larger sizes of the intestinal segments involved in pathological process the higher leucocytosis.

Hemodynamic parameters in the certain degree reflect gravity of the syndrome of acute intestinal obstruction. Tachycardia, typical of this disease, within the limits of 80–100 beats per minute in serious forms and in advanced cases exceeds these figures and the arterial pressure decreases.

Diagnosis. A high-informative method of diagnosis of intestinal obstruction is roentgenologic. For confirmation of the diagnosis general X-ray examination of the abdominal cavity in a standing position or lateroposition is carried out. At the initial stages of the disease the gas in inflated loops of the small intestine where it must not be in the norm is determined roentgenologically. The detection in the intestine the levels of fluid and gas above them serves as ("Kloiber's cups") basis for the establishment of diagnosis. In doubtful cases, especially in adhesive form of intestinal obstruction contrast roentgenography is carried out: the patient is given to drink or he is introduced through the gastric tube 150–200 ml of barium suspension and with 2 hour interval a passage of barium is observed. Retention of the passage, contrasting the isolated loop, detection of obstacle in the intestine confirm the diagnosis of the intestinal obstruction.

A perspective and faster method of research is enterography, when through a probe curtained off the Treitz's ligament barium suspension is intensively injected and after this roentgenography is carried out. However, under conditions of discoordinated peristalsis and in paresis the installation of a probe behind the pyloric sphincter is extremely difficult.

The reccurence of attacks reminding on their clinical manifestations acute obstruction is typical for chronic (partial, incomplete) intestinal obstruction, but the signs are usually weakly expressed and gradually regress. Such patients are indicated the examination under in-patient conditions with keeping the principles of oncologic suspicion.

The phrase "the longer the patient lives before the surgery the less after it" is quite right for a syndrome of acute intestinal obstruction. Firstly, according to the modular statistics of the various authors within the first 6 hours from the beginning of the disease only 9–40% of the patients are hospitalized, during 24 hours — 19–46%. Secondly, the lethality among the patients hospitalized till 24 hours from the beginning of the disease makes 3–16%, and after 24 hours — 17–35%.

Among the main causes of lethal outcomes more than 50% belong to peritonitis developing both before the operation and during postoperative period and complicates the course. 24–34% patients die because of endogenic intoxication, severe disorders of water-electrolyte exchange. In other cases the patients die from pneumonia, cardiovascular failure and tromboembolic episodes of the postoperative period.

Treatment. Diagnosis of acute intestinal obstruction should be an indication for urgent operation, therefore the basic task of the first medical aid is a prompt delivery of the patient to a medical establishment. The serious condition of the patient is indication for the beginning of infusion therapy at the

prehospital stage, including probable continuation of it during transportation to a medical establishment.

With precise diagnosis at the stage of qualified surgical aid, differential-diagnostic researches with the purpose of establishment of a kind of intestinal obstruction and its localization should not be carried out because it may take a lot of time and has no essential importance for definition of indications for operative intervention.

Preoperative preparation lasts no more than 2 hours from the moment of admitting the patient to a hospital. This time is required to confirm the diagnosis (in uncertain cases) and for differential diagnosis with other acute diseases of abdominal organs.

Preoperative preparation includes general hygienic measures. A gastric tube is obligatory inserted, contents are evacuated, and with a tube the patient is moved to the operationg room.

The volume and contents of preoperative infusion therapy directed first of all on correction of water-electrolyte disorders should be determined with an anaesthesiologist. With a condition of moderate severity the patient is intravenously introduced about 1,500 ml of Ringer-Lock's solution, 40% solution of glucose with insulin, spasmolythics, cardial medicines, vitamines B and C. In severe cases particularly in nodulation, blood transfusion and introduction of protein preparations may be necessary.

Cleansing enema is carried out on filled distal departments of the intestine. In the most cases, contrary to the wide-spread recommendations of conservative treatment of acute intestinal obstruction the effect of it relatively liquefaction of the ileus is rather arguable. Sometimes it is possible to avoid the operative intervention with conservative therapy only in fecal blockage and volvulus of the sigmoid intestine. The operation in acute intestinal obstruction is carried out under endotracheal narcosis with myorelaxants.

A surgical access is more often medline laparotomy, and after inspection and topical diagnosis of obstruction incision is made upwards or downwards. In strangulated hernias an access is carried out on the site of localization of the pathological center.

Tasks of operative interventions are elimination of intestinal obstruction, creation of conditions for treatment of postoperative paresis and prophylaxis of relapses of the disease

As a rule, extent of operative intervention is determined by a degree of ischemic changes in the intestine involved in pathological process. The small intestine with obvious feature of inviability (absence of peristalsis, glisten of serous integument, pulsation of vessels) is subjected to resection with the keeping a rule: resection should be carried out at a distance of 30–40 cm proximally and 15–20 cm distally from a visible border of necrosis,

as at such distances an adequate blood supply of the intestinal wall is kept. In suspicious cases after elimination of the causes of obstruction the intestine is subjected to "reanimation": 100 ml of 0.25% solution of Novocainum with a spasmolytic are injected in the mesenterial root, the intestine is covered by hot napkins. The occurrence of peristaltic waves, pulsation of parietal and intraparietal vessels, a reddened strangulated loop of the intestine testifies to its viability.

With resection of the small intestine anastomosis for a type "end to end" is considered to be more physiological and which should be preferrable meanwhile other kinds of anastomosis — "end to side", "side to side" are possible.

In severe volvuluses, nodulations and evident features of necrosis of the intestine a resection is carried out without preliminary elimination of a mechanical obstacle, because unscrewing, untying of the intestine may cause endotoxic shock due to rapid and volumenous discharging out of toxic substances from necrotic intestine into the blood stream.

Extent of operative intervention in obturation of the intestine with foreign bodies more often does not exceed the opening of the lumen, removal of the body which obturates and suture of the wound.

Oncologic obturative colonic obstruction of the right canal is eliminated by hemicolectomy with applying ileotransversalanastomosis, and transversocolon and the left canal — resection of the intestine with a tumour, suture of a distal part and exteriorization of a proximal one — on the anterior abdominal wall in a form of monotrunk anus (Gartmann's operation). Under favorable conditions (absence of rough microcirculatory disorders in a zone of resection of the intestine and peritonitis) applying a primary anastomosis is permitted. In case of advanced inoperable oncologic obstruction it is recommended to use bypass anastomoses — ileotransversal, transversosigmoanastomosis and others. Intra-operative intubation of the small intestine has a gtreat importance in treatment of acute intestinal obstruction. It is carried out according to the following indications:

— severe paresis of the intestine;

— acute intestinal obstruction complicated by peritonitis,

— promoted adhesive process in the abdominal cavity.

Among a great number of available techniques of drainage of the small intestine preference is frequently given to a nonfistulous method — nasogastrointestinal intubation, but when this method is not possible to perform the fistulous methods, which are the most wide-spread in surgical practice, such as Dederer's gastroenterostomy, Zhitnuk's enterostomy, cecoenterostomy and others are used.

In pronounced overstraining of the intestine by gas and liquid contents after laparotomy first of all the intubation of the intestine and evacuation of

its contents is carried out with the purpose of improvement the conditions for inspection of full value and for operative intervention as well.

A surgical approach with paralytic intestinal obstruction is based on active conservative treatment of intestinal paralysis. In absence of effect within 3 days the operative treatment is started with the purpose of intestinal intubation. If organic substrate in intestinal paralysis (intra-abdominal abscess, peritonitis, postoperative pancreonecrosis and others) is diagnosed, the operation is performed at once after the establishment of diagnosis.

The conservative treatment of the intestinal paralysis should be complex, directed on all branches of pathogenesis. It includes:

— evacuation of gastric contents with a constant probe;

— restoration of damaged homeostasis;

— detoxicative therapy (hemo- and lymphosorption, plasmopheresis, enterosorption and others);

— antibacterial therapy;

— stimulating intestinal peristalsis (peridural anaesthesia, neostigmine methylsulfate, pituirin, saline enemas, combination of various methods);

— novocaine blocks (of round ligament of the liver, sacrospinal).

The same complex of measures should be carried out during the postoperative period after elimination of mechanical intestinal obstruction.

The patients with chronic intestinal obstruction are undertaken to urgent planning hospitalization, meticulous inspection with the purpose to detect the cause, its elimination, including usage of operative methods.

Lecture 6
ACUTE APPENDICITIS

Acute appendicitis is the most common surgical disease of the abdominal organs. Annually from 2 to 5 of 1,000 people suffer from it. This disease can arise at any age, but the greatest morbidity is marked in juvenile and young age.

Etiology. The acute appendicitis is an acute nonspecific inflammation of the vermiform appendix caused mainly by causative agents of purulent infection, living in the lumen of this organ (colonic rod, enterococcus, staphylococcus, streptococcus etc.). Disease is more common among people consuming mainly meat or meat products, that's why it is intended that putrid infection plays a leading role of pseudotuberculous rod in etiology of acute appendicitis. Lately, the high etiological role of pseudotuberculosis rod in occurrence of acute appendicitis has been proved.

Pathogenesis. The mechanism of occurrence and development of acute inflammation of the vermiform appendix is rather various and in many respects it is still unsufficiently investigated. The most probable way of invasion of the agent is enterogenic, due to which an infectious process in the vermiform appendix arises owing to penetration of agents into the wall of appendage from the contents of its lumen. However, in experiment on animals the pathogenic microbes introduction into the lumen of the appendage does not result in changes in its wall, which are observed in patients in clinic. Only under conditions of blood circulation disturbances in appendage caused by introduction of epinephrine in its mesentery or mechanical squeezing of vessels, pathogenic flora introduction into the lumen of causes changes peculiar to acute appendicitis.

An acute inflammation of the vermiform appendix under conditions of the experiment can be caused also by introduction of a culture of pathogenic microbes into its lumen in complete obturation of the latter, or after previous mechanical trauma of its mucous wall. Other mechanisms of develop-

ment of acute pathological changes in the vermiform appendix are also known. So, for example, acute appendicitis may develop as an infarct of the vermiform appendix owing to clottage of blood vessels of its mesentery. In these cases gangrene of its wall is found out during first hours from the onset of the disease, and the zone of necrosis is usually circumscribed to a territory of blood supply by a thrombosed vessel. In such a mechanism of destruction purulent inflammation of adjacent tissues is secondary, and it should be considered as a manifestation of protective reaction as a demarcation inflammation, which always arises in the sites of microbe-contaminated areas of necrosis with alive tissues. Such mechanism of gangrene of the vermiform appendix most frequently arises at elderly age and in organic defeats of the vessels.

Thus, the enterogenic infection of the vermiform appendix is possible only in certain degree of its mucosa epithelium barrier function decrease, that can be due to a disorder of regional circulation (spastic stricture, paresis, clottage of vessels of the mesentery of the vermiform appendix), elevation of intraluminal pressure in its lumen (appendicular obstruction), mechanical trauma of the mucosa (foreign bodies, helminthic invasion, etc.) and, probably, for others, yet unclear reasons. Much less common pathogenic agents penetrate in lymphoid apparatus of appendage through the hematogenous way, the tonsils (tonsilitis) are the most probable and clinically proved primary origin of infection in these cases. Hematogenous infection of the vermiform appendix from other distant centers of a purulent inflammation arises rare, and even in septicopyemia the purulent metastases in this organ do not develop.

Probably, the cases of penetration of pathogenic agent by lymphogenous route due to spread of infectious process from adjacent organs and tissues are more uncommon, as the signs of inflammation of the vermiform appendix, marked in these cases (secondary appendicitis), both according to its localization, and mechanism of occurrence essentially differ from pathological changes in the wall of the vermiform appendix in acute appendicitis. Modern recognition of a leading part of autoinfection in etiology of acute appendicitis does not mean that the appearence and outcome of inflammatory process in the appendage is caused by increased virulence of enterogenic microflora, as it was proved by a German pathologist Ashoff. The recent clinical and experimental researchers have shown that in appearence, course and outcome of acute inflammation of the vermiform appendix a great and, probably, determining meaning has the functional condition of immunological systems and a character of protective reactions of an organism. Apparently, a stage-life character of morphological changes is not obligatory, as the analysis of numorous clinical observations does not find

out essential changes in the structure of morphological characteristics of the vermiform appendixes, removed at different terms from the onset of a disease. Accorging to many great statistic researches the correlation of catarrhal, phlegmonous and gangrenous forms of appendicitis comprises 1:3:1 correspondingly.

The dependence of character of morphological changes in the vermiform appendix is traced more clearly as compared with the parameters of reactivity of an organism: in considerable decrease of activity of systems of anti-infectious protection gangrene of the vermiform appendix is found out three times more often, than catarrh in it, and in absence of such decrease catarrh in the vermiform appendix is marked as 1.5 times more often than a deep destruction like gangrene (I. G. Leshchenko et al., 1974). Obviously, various pathoanatomical forms of acute appendicitis not always are the consequence of a stage development of the process, and their formation depends on the ratio of the forces of aggression (pathogenic agents) and the forces of protection (functional condition of reactivity and immunological systems of an organism). The morphological changes in the vermiform appendix in various forms of acute appendicitis can develop according to various "programmes": gangrene of the vermiform appendix can arise without a previous catarrh, but a catarrhal form can not be transformed into a phlegmon and gangrene of the vermiform appendix even during its long-term existence.

Thus, the term "acute appendicitis", accepted for a designation of a separated nosological form of the disease of the vermiform appendix, includes actually different according to the etiology and pathogenesis inflammatory and destructive changes in this organ found out together with the identical clinical signs and characterized by the common diagnostic and tactical principles of treatment of these patients.

Pathological anatomy. Acute appendicitis is characterized by appearance in the vermiform appendix of a circumscribed focus of purulent inflammation of the mucosa. The defect of the epithelium is determined in histopathological investigation, under which there is an area of tissues of the vermiform appendix with a leucocytic infiltrate which penetrates the submucous layer. Quite often the inflammation arises in two or more areas. In some cases the primary affect can not be found out, and then pathomorphologic changes in the vermiform appendix have the appearance of superficial serous-purulent inflammation. Acute destructive appendicitis arises in phenomena of purulent-necrotic changes.

The vermiform appendix is enlarged in volume, serous coat with small hemorrhages becomes covered by fibrosal applyings. On a cross section the wall of the vermiform appendix is thinned, its layers are indiscernible. The

mucosa is acutely plethoric, pus accumulates in the lumen of the vermiform appendix. The diffuse leucocytic infiltration extending on all layers of the vermiform appendix is determined at pathohistological research. A plenty of polymorphonuclear leucocytes and lowered cells of the mesothelium are found out in fibrinous applyings. Such inflammatory process is called phlegmonous appendicitis with the phenomena of a periappendicitis (initial stage of circumscribed appendicitis). Sometimes in the wall of the vermiform appendix purulent decomposition begins and abscess occurs — apostematous appendicitis. The abscess can open in the lumen of the vermiform appendix or perforates its wall and excretes its content in the peritoneal cavity.

The most rare form is gangrenous appendicitis. Macroscopically gangrenous appendicitis is displayed by circular necrosis of the wall of the vermiform appendix on several vascular segments or the vermiform appendix. The wall becomes flabby, impregnated with blood, thin, it has dirtygray-red colour, with fecal odour. Occurrence of such a form of appendicitis is connected to disorder of circulation caused by spastic stricture of small vessels and their clottage.

Classification. Clinical necessity in classification of acute appendicitis is caused by essential differences of the contents of operative intervention and outcomes of treatment depending on the character of pathological changes in the vermiform appendix and adjacent organs. Besides, the classification is necessary for the objectivating of statistical analysis of various aspects of acute appendicitis, without which the further improvement of diagnosis and treatment of this disease is impossible.

There are some classifications that reflect the difference of signs of classification and polymorphism of disease. The classification, in the basis of which the morphological criteria of changes in the vermiform appendix and the feature of the clinical course are put, satisfies the demands of practical surgery and needs of scientific analysis of clinical aspects of acute appendicitis to a great extent.

Clinical course. In spite of that the inflammation of the vermiform appendix belongs to a group of acute surgical diseases, actually acute appendicitis in its clinical manifestations frequently begins not so acutely as many doctors and even surgeons establish. Abdominal pain is the first and mandatory sign of acute appendicitis. Most frequently from the very beginning it is localized in the right iliac area, but sometimes disease begins with anorexia, nausea; cutting pains in the abdomen without certain localization which are usually seem for the patients as the common menifestations of meteorism. Quite often vomiting, headaches, lethargy join these symptoms, but soon pains, which are localized in the epigastrium, in

the area of the gallbladder, in the right hypochondrium, less often in the area of the pelvis and the loin become leading in the clinical picture of the disease. But in several hours the epicenter of the pain is more clearly displaced into the right iliac area of the abdomen.

Thus, quite often it is possible to distinguish the initial and final localization of pain in acute appendicitis. Localization of pain in the right iliac area is the most often and characteristic feature during a typical attack of acute appendicitis.

The localization of initial pain in the epigastric area independently on locating the vermiform appendix is peculiar, mainly, to destructive forms of acute appendicitis. Pains in acute appendicitis are usually persistent, which are increased on movements of the patient, cough and physical exertion. In obstructive acute appendicitis they quite often have a spasmodic character and are marked by excessive intensity and suddenness of appearence as an acute painful attack. Along with spontaneous pains the provoked pains are characteristic for acute appendicitis which arise in the area of inflamed vermiform appendix on cough ("a cough sign"), palpation of this area, percussion of the anterior abdominal wall (Razdolsky's symptom), on sliding forward movements along the anterior abdominal wall (Voscresensky's symptom), concussion and moving the caecum and adjacent intestinal loops etc. The spontaneous and provoked pains may be unique clinical symptoms of acute appendicitis. A peculiarity of the pain syndrome at acute appendicitis is the absence of irradiation of pains, excepting cases of a pelvic, subhepatic and retrocecal locating of the vermiform appendix. In these cases the pains can irradiate to the right scapula, inguinal area, scrotum, right leg, that, apparently, is caused by reaction of nearby organs (ureter, gallbladder, urinary bladder) on the arisen focus of inflammation.

Other clinical symptoms of acute appendicitis — nausea, vomiting, retention of stool and gases — arise rather frequently, but are not constant for this disease, and the presence of these signs indicates the presence of destructive changes in the vermiform appendix.

The further inspection of the patient with acute appendicitis helps to find out the other most essential objective signs of disease. The careful examination of the anterior abdominal wall sometimes allows determining some retardation or immobility of its right half on respiratory movements, the presence of asymmetry.

Palpation of the anterior abdominal wall is carried out according to the principle of comparative research, and it should be performed with a gentle superficial palpation allowing determining the slightest changes in muscular tonus of its various areas, imperceptible by usual means of research. The

palpation is carried out as follows: first the degree of tension of muscles in the area more distant from the site of painful sensations (the left iliac area) is defined and received sensations are compared with the data of the same palpation in the "epicenter of pains" (for example, the right ileal area). The examination in epigastric and subcostal areas is carried on in the same way. The detection of muscle tension of the anterior abdominal wall specifies the presence of inflammatory process in the abdominal cavity. This tension needs to be distinguished from spontaneous muscle rigidity, testifies to the involving into the inflammatory process of the parietal peritoneum and the development of peritonitis.

The tension of the anterior abdominal wall in acute appendicitis may be absent in the following cases:

— if the patient is examined immediately after the onset of the disease, that is during the earliest period of the development of acute appendicitis;

— in pelvic locating of the vermiform appendix;

— in retrocecal and retroperitoneal locating of the vermiform appendix;

— in elderly and exhausted patients;

— immediately after the perforation of gangrenous "obstructive appendicitis", when hypertension disappears, and the patient insists on that he feels much better;

— in superficial catarrhal appendicitis.

After superficial palpation the signs considered as characteristic for acute appendicitis are studied. Today, there are more than one hundred of such symptoms. However, only few of them have the greatest importance and are used in medical practice.

Shchetkin — Blumberg's symptom. Slow press on the anterior abdominal wall by all fingers and take them away quickly. The sign is considered to be positive with the appearance or reinforcing of pains at the moment when the fingers are taking away. In retrocecal location of the vermiform appendix or its limitation by pronounced adhesions the sign can be negative or slight-positive even in destructive appendicitis.

Rovsing's symptom. Press on anterior wall of the abdomen in the left iliac area by fingers of the left hand according to the site of location of the descending intestine, the fingers of the right hand pressing on the area above the descending intestine. The sign is considered to be positive if on pressing by the right hand the pain in the right iliac area arises or increases.

Sitkovsky's symptom. Appearance or reinforcing of pains in the right iliac area when the patient is turned out from his back to the left side and in a position on the left side.

V. M. Voscresensky's symptom: a doctor, which carries on the examination, settles down to the right of the patient and pulls a patient's shirt for

its edges with his left hand; the tips of his II, III, IV fingers of the right hand he puts on the epigastium area and during the inspiration of the patient when anterior abdominal wall is the most weakened, with a moderate pressing on the patient's abdomen he makes a sliding rapid movement slantwise downwards to the area of the cecum there he stops his hand leaving it on the patient's abdomen as it is done in Blumberg's symptom. At the moment of the completion of such "sliding" the patient notices a sharp increasing of appendicular pains and quite often expresses it with a facial expression. For the control the same movement may be carried out in a direction from bottom to the top and such increasing of the pains is not marked.

I. Ya. Razdolsky's symptom. Pain in the right iliac area on percussion of the anterior abdominal wall with percussion hammer.

O. P. Krymov's symptom. Tenderness of the right inguinal canal on introduction of a finger through external opening in the area of the posterior wall.

A. S. Chuguyev's symptom. Palpation of tense "bands" in external oblique abdominal muscle on palpation of the right iliac area. More often these "bands" are defined on palpation of the patient in the left lateral decubitus position.

Bartomier — Mikhelson's symptom. Tenderness on palpation of the right iliac area in the left lateral decubitus position of the patient.

A lumbar symptom (V. N. Varlamov). Pain occurs in the right ileal area on percussion of the XII rib or on the area of lumbar muscles on the right and on the left.

The importance of above mentioned symptoms in diagnosis of acute appendicitis is variously regarded by the authors. However, almost all of them are unanimous in that tenderness, tension of muscles and the positive sign of Shchotkin — Blumberg in the right iliac area are cardinal signs of a typical course of acute appendicitis.

"Cough sign"— appearing or increasing the pains in the right iliac area on cough — is also of great value in diagnosis. Therefore each patient with the suspicion on acute appendicitis should be undertaken a careful medical examination. Analyses of blood and urine as well as taking the temperature in the groin area and rectal thermometry are obligatory in making a diagnosis if doubts occur.

Differential diagnosis. Such purposeful inspection is necessary not only for a detailed evaluation of general condition of the patient but also for differential diagnosis, the need in which arises on inspection of each patient with acute appendicitis.

Depending on clinical manifestations acute appendicitis is necessary to differentiate from the diseases of the stomach and duodenum (acute gastritis, stomach and duodenum ulcers, food intoxication etc.), extrahepatic bil-

lary ducts (acute cholecystitis, cholelithiasis), acute pancreatitis, acute enteritis, coloenteritis, inflammation and perforation of Mekkel's diverticulum, acute intestinal obstruction, diseases of female sexual sphere (acute inflammation of the endometrium, acute inflammation of the uterine appendages, disturbed extrauterine pregnancy, ruptures and hemorrhages of the ovaries) as well as with nephrolithiasis and right-hand renal colic.

ATYPICAL FORMS OF ACUTE APPENDICITIS

Obstructive Appendicitis

Acute appendicitis owing to obstruction of the lumen of the vermiform appendix is mainly marked at the young age. According to the clinical data, 59.7% of the patients with this form of acute appendicitis were younger than 29 years and only 3.7% were after 60 years. The disease begins suddenly with sharp colic-like pains in the abdomen, frequently in the area of umbilicus, and is usually accompanied by vomiting, quite often by repeated one. Pains are so intensive that cause a physical suffering to the patient. Dull pain persists during the period between the attacks in the right iliac area or in the area of the umbilicus. During the examination of the patient within the first hour of the disease the objective signs of disease (tension of the abdominal wall, high temperature, leucocytosis) are usually absent and only tenderness on deep palpation in the area of the vermiform appendix localization indicates the possibility of pathological process in this organ. However with each hour clinical picture of the disease becomes more and more troubled owing to development of the center of infection in the stretched and intense vermiform appendix. X-ray film of the right iliac area demonstrates the vermiform appendix inflated by gas. Pains decrease in gangrene of the vermiform appendix, but the general condition of the patient does not improve. This deceptive period of "imaginary well-being" may persist for some hours, untill a perforation of the vermiform appendix develops. If "appendicular colic" persists more than 3–4 hours, operative intervention (appendectomy) is indicated.

Retrocaecal Appendicitis

Retrocaecal appendicitis is acute inflammation of the vermiform appendix located behind the caecum. If the vermiform appendix with such a localization is not circumscribed by adhesions but lies in the retrocecal pocket that is freely connected with the peritoneal cavity, clinical manifestations of acute inflammation of the vermiform appendix usually do not differ from a typical course of this disease. The features of the retrocecal appendicitis

clinical course are evident, mainly, in those cases when the vermiform appendix located in the retrocecal pocket is circumsribed from a free abdominal cavity by adhesions and closed in them (V. I. Kolesov).

The disease begins usually with appearance of moderate pains in the right half of the abdomen and lumbar area, sometimes above ileal crest and quite often is accompanied by vomiting. Pain may irradiate to the inguinal area, right femur, and to the area of the pelvis. The inspection of the patient gives scanty data: the abdominal wall tension quite often is not present, Shchotkin — Blumberg's sign is frequently negative, and there may occur a moderate tenderness on deep palpation in the right ileal area. In progression of the process in the vermiform appendix the general condition of the patient becomes worse: the signs of a purulent intoxication arise, leucocytosis increases, the body temperature rises. Fresh erythrocytes, leucocytes, protein, individual hyaline cylinders appear in urine frequently. In these cases in diagnosis local signs which are characteristic to retrocecal appendicitis take a great importance:

Yaure — Rosanov's symptom: pain on a finger pressure in the area of Petit's triangle on the right.

B. V. Lunin's symptom: tenderness on a finger pressure on the area of the transversal process of the II–III lumbar vertebra.

A. V. Gabay's symptom: press by a finger on the area of the right Petit's triangle and take away a finger quickly. The sign is considered to be positive if at the moment of finger taking away the pain arises or reinforces in this area (by analogy with Shchotkin — Blumberg's sign).

V. I. Varlamov's positive symptom: (see above).

Obraztsov's symptom: in occurrence of a purulent process in periappendicular tissues "psoas symptom" arises — tenderness during extension of the right lower extremity, bent in a hip joint.

Acute Appendicitis in Mesocecal Disposition of Appendix

If the distal part of the vermiform appendix is located between the loops of the small intestine, its acute inflammation is usually characterized by rough development of clinical menifestations. It is due to high resorbtive capability of inflammatory exudate formed in a free peritoneal cavity. The pains in the area of the abdomen in mesocecal appendicitis more diffuse, but their epicenter is localized in the area of the umbilicus or in the right iliac area. Tension of the anterior abdominal wall and Schotkin — Blumberg's symptom are usually clearly marked and defined on its almost whole right half. As a rule, abdominal distension is more marked owing to frequently arising paresis of the intestine on this localization of appendix, than in other forms

of acute appendicitis. Besides the symptoms of acute appendicitis on mesocaecal localization of the vermiform appendix described above, Gorn's symptom (pain in the right iliac area on moderate tension of spermatic cord); A. M. Gurevich's symptom (tenderness on cough after the introduction of an index finger into the outer space of the right inguinal canal) can be positive.

Acute Appendicitis in Pregnancy

About 80% of all cases of acute appendicitis in pregnant women arise during last 6 months of pregnancy. Clinical course of disease in these cases is polymorphic and it is necessary to differentiate from other conditions that have similar clinical menifestations. The epicenter of abdominal pain in pregnants is localized higher and more laterally than usually (owing to displacement and, probably, some pression of the vermiform appendix). Very important for recognition of acute appendicitis in pregnancy is the presence of similar pain attacks in anamnesis. Blood analyses help little in diagnosis, as increase of the number of leucocytes may be due to pregnancy. In determination of indications to the operation it is necessary to mean that against a background of pregnancy appendicitis has a particularly severe course. Therefore the vermiform appendix should be removed as soon as possible after the onset of the disease, when possibility of the development of complications is minimal. According to the modern views, appendectomy in acute appendicitis is indicated irrespective of terms of pregnancy.

Acute Appendicitis with Atypical Clinical Picture

In all cases, when a patient with the complaints of abdominal pains of uncertain character and localizations is examined, first of all it is necessary to exclude acute appendicitis with atypical clinical course. Most frequently this atypical picture is formed under the influence of symptoms chatacteristic for the diseases of other organs.

Acute Appendicitis with Dysuric Disorders

Dysuric disorders in acute appendicitis arise when the vermiform process is located in the small pelvis. If an inflamed apex of appendage directly adjoines the wall of the urinary bladder or purulent exudate arising owing to decomposition of appendix, adjoins the wall of the urinary bladder, the imperative, painful and frequent vesical tenesmi appear. Dysuric disorders may be so evident, that keep themselves in the foreground in a common clinical picture. However, on careful examination of the patient it is always possible to determine that the disease has begun with the pains below the

abdomen of the lower part or the right iliac area, accompanied with common malady and rising temperature. The tension of the anterior wall of the abdomen in these cases, as a rule, is not present, but tenderness and positive "cough" sign quite often are defined on a deep palpation above the pubic symphysis on the right. With the digital examination of rectum tenderness and at the late stages as well as infiltrate in the area of its anterior wall are usually revealed. In doubtful diagnosis taking temperature not only in the inguinal area, but also in the rectum is obligatory in such cases. The rising of the rectal temperature in comparison with axillary one by more than 1°C is a sign of inflammatory process in the small pelvis.

Acute Appendicitis with Diarrhea

Usually the appearance of acute appendicitis is accompanied with retension of stool and gases. However, sometimes the first and most evident sign of this disease is diarrhea. Loose stool in acute appendicitis may be due to a severe intoxication by products of purulent-gangrenous disintegration of appendix as well as the result of irritent action of peritoneal exudate on location of inflamed vermiform appendix in the small pelvis or its closed location with intra-abdominal portion of the rectum. In the latter case there may occur tenesmi and mucus in stool. Diarrhea is accompanied by febricula and pains below the abdomen or in the right iliac area immediately above the inguinal ligament. On localization of inflammatory process within the limits of the vermiform appendix tension of the abdominal wall and Shchotkin — Blumberg's symptom may be absent, but a "cough sign" is usually expressed clearly enough.

In such cases the great large importance in diagnosis of acute appendicitis has the digital investigation of the rectum during which tenderness and at the late stages infiltrate or hanging of its anterior wall may be revealed.

Due to impossibility to exclude acute appendicitis with ordinary clinical methods of examination the combination of these signs even with the poor apparent tension of the anterior abdominal wall in the right ileal area and other signs of inflammation (fervescence, leucocytosis) are sufficient base for operative intervention.

Acute Appendicitis with Hyperpyrexia

The body temperature in acute appendicitis is usually increased but insignificantly and does not exceed 38°C in the beginning of disease. Its increasing above 38°C arises later and quite often testifies to the development of complications (periappendicular abscess, perforation of appendix, peritoni-

tis). However, sometimes disease begins with the chill and rising of the temperature up to 40°C and more. In some cases in such hyperpyrexia there may occur the signs of serious purulent intoxication — tachycardia, a dry coated tongue, high leucocytosis. Quite often in such cases the presence of pyelitis, pneumonia is assumed mistaken, but these assumptions which exclude acute appendicitis, every time should be very convincingly proved. Acute appendicitis may not be excluded only when the patient has very high (or very low) temperature of the body. The careful examination of the patient is necessary, and if the clinical signs of acute appendicitis will be found out or this diagnosis will not be convincingly refused, it is necessary to start the operative intervention.

Acute Appendicitis with the Signs of Gallbladder Disease

On high location of the apex of the vermiform appendix it may reach subhepatic space, and on occurrence of inflammation in appendix the clinical manifestations of appendicitis may remind acute cholecystitis, less often hepatic colic. On a subhepatic locaton of the vermiform appendix acute appendicitis quite often begins with acute pains in the epigastric area in the right half of the abdomen, lumbar area, the changes of localization of the pains in the process of the disease course not occurring, and wide-known signs of acute appendicitis may be absent. Only with progression of inflammatory process the zone of maximal tenderness and muscular tension in the right half of the abdomen or in the right hypochondrium may be designated, but, usually without irradiation typical for the gallbladder disease. Unfortunately, this differential-diagnostic sign is hardly detectible. As a rule, there is leucocytosis and fervescence.

Due to displacement of the vermiform process, and sometimes a cecum, evident adhesions with adjacent organs appendectomy is usually connected with considerable technical difficulties and it should be performed under general anesthesia.

Treatment. The contents and volume of medical aid for the patients with acute appendicitis under conditions of a polyclinic are determined by the basic tactical principles of treatment of this disease. According to these principles the early diagnosis, early hospitalization to a surgical hospital and early operation are major factors which determine the success of medical management. Therefore not only a diagnosis of acute appendicitis, but also a well-grounded suspicion on this disease should be considered as the indication to urgent evacuation (lying, in ambulance) to a surgical hospital. The observation of the patient in home conditions with the purpose of specification of a diagnosis of acute surgical disease of the organs of the abdomi-

nal cavity is inadmissible, because a rapid relieve of pain is not persuasive evidence of the disappearance of pathological changes in the vermiform appendix, but is more often due to a restriction of inflammatory process, by formation of the inflammatory infiltrate or gangrene of the vermiform appendix. The severity of morphological changes in appendix frequently appears much greater than it may be assumed on the basis of clinical data. Therefore slight evidence of symptoms may not be justification for the deviation from tactical principles of rendering a medical aid in acute appendicitis. The lethality in this disease is in a direct dependence on the terms of operative treatment. So, according to the data of many clinics, among persons operated within the first 6 hours from the beginning of the disease it comprised 0.17%, from 6 up to 24 h — 0.22%, and more than 24 h — 0.75%.

For these reasons the first medical aid should be limited (in presence of indications) by introduction of cardiac agents and spasmolytic preparations. Conterraindications in acute appendicitis and suspicion on this disease are as follows:

— local heat (hot water bottle) on area of the abdomen;
— narcotic analgetics and other anaesthetics;
— laxative;
— enema.

Enumerated medical measures in acute appendicitis are not only vain, but are extremely dangerous due to elimination of pain and other signs of the disease, that complicates its detection, and also the opportunity of a rapid progression of the process and appendix perforation.

A qualified surgical aid in acute appendicitis usually consists of appendectomy and operative treatment of surgical complications of the disease. The operation is indicated in any terms from the onset of the disease, except for some kinds of appendicular infiltrates, detection of which makes it more expedient to avoid urgent operative intervention. The operative intervention is justified and in those cases when there is only a well-grounded suspicion on the possibility of acute appendicitis. The doubts as for the presence of acute appendicitis (in absence of other symptoms of disease being the indication for laparotomy, and in satisfactory general condition of a patient) under conditions of a surgical hospital should not last more than 24 h. During this period the diagnosis of acute appendicitis must be rather argumently rejected or appendectomy should be performed. Clinical well-being of the patient in presence even of mild tenderness in the right iliac area should not be a circumstance justifying the avoidance from operative intervention. Appendectomy is the most often operation concerning acute appendicitis. Today, in overwhelming majority of cases it should be performed under the general anesthesia, which is especially indicated in a compli-

cated appendicitis (peritonitis, early progressing infiltrate), atypical forms of appendicitis, in a very developed hypodermic fatty tissue, in uneasy patients and children, and also when it is difficult to clearly differentiate acute appendicitis with other acute surgical diseases of the organs of abdominal cavity (perforative ulcer of the stomach and duodenum, destructive cholecystitis, acute pancreatitis etc.)

An operative approach through Volcovich — Dyakonov — Mc-Burney's incision in most cases is sufficient for appendectomy, inspection of organs of the small pelvis and the right lateral canal of peritoneal cavity. Lennander's incision and a transverse section are used more rarely, but it is necessary to remember about some advantages of these accesses during the operation under conditions of appendicular infiltrate and local peritonitis.

In acute appendicitis, complicated by diffuse peritonitis, the most rational is midline laparotomy; efficiency of anesthesia, pedantic definition of a position and the sizes of a dermal incision in these cases in many respects determine the course, and sometimes the outcome of operation.

Sometimes it is useful to dissect a parietal peritoneum along the outside border of the intestine when it is difficult to remove a cupola of the caecum in the operative wound.

If the vermiform appendix is without evident external pathological changes, it is necessary to examine a terminal department (with the extent not less than 50 cm) of the ileal intestine, the area of the gallbladder and duodenum, uterine appendages (in women), and in absence of inflammatory changes in these organs, about which the clinical manifestations testify, to performe appendectomy.

In presence of exudate in the abdominal cavity it should be carefully removed, to carry on bacteriological research with the definition of sensitivity to antibiotics, and in phlegmonous and gangrenous appendicitis it is necessary to introduce a drainage into abdominal cavity for evacuation of inflamatory exudate and further intra-abdominal introduction of antibiotics not less than 2 times per day. The cavity of the small pelvis should be especially carefully examined and drained.

In peritonitis the parenteral introduction of antibiotics is necessary to begin before the operation. The treatment of the abdominal cavity and removal of exudate in these cases are performed according to the modern principles of treatment of peritonitis.

The abdominal cavity is sewn up tightly, except for cases, when its drainage and introduction of tampons are justified, namely:

— impossibility of stopping of bleeding by usual means;

— when it is impossible to remove completely a pathologically changed vermiform appendix or the other center of infection in this area;

— uncertainty as for reliability of peritonization of a stump due to pathological changes of the wall of the caecum.

In gangrene and purulent decomposition of the vermiform appendix located retocecally or in subhepatic space, for a drainage of infected bed of appendage or abscess cavity contraperture in the lumbar area is performed, through which the active (vacuum) drainage of the center of infection with a double-lumen silicon tube in a diameter not less than 8 mm is carried out.

For the prophylaxis of infectious complications in the wound the decisive importance has a pedantic keeping the rules of aseptics and antiseptics during the operation, in particular, changing the instruments, careful handling with tissues, reliable and careful hemostasis in the process of operation, application of primary delayed sutures on the skin in case of uncertainty as for asepsics of the operational wound.

Complications of acute appendicitis. Among complications of acute appendicitis it is necessary to distinguish such complications which are due to the features of inflammatory process course, and those which have not specific signs inherent to this disease, but due to operative interventions or which appeared due to accompanied diseases. The last group of complications (peritonitis, subphrenic and interintestinal abscesses, bleedings, inflammatory infiltrates, pyesis of the operational wound, pneumonia, intestinal obstruction etc.) frequently have not any features due to acute appendicitis.

Appendicular Infiltrate

Appendicular infiltrate is a conglomerate of organs and tissues which are formed around the inflamed vermiform appendix and usually consisting of area of an infiltrated large omentum, cupola of the cecum and the loops of the ileal intestine connected with each other and surrounded with fibrinous adhesions. As the local inflammatory process, the appendicular infiltrate independently or under the influence of treatment on the stage of infiltration of tissues may undergo a reverse development or may transit into the following phases of sequestration of necrotic tissues and their purulent decomposition with the formation of abscess.

The appearance of appendicular infiltrate is promoted by some causes: the character of inflammatory process and anatomic features of a vermiform appendix, age and immunological features of an organism, the character of the pathogenic agent etc. According to the clinical course it is possible to single out the early infiltrates arising during the first 2 days from the onset of the disease (their appearance is usually connected with acute destructive changes in the vermiform appendix), and infiltrates which arise on the 3rd–5th days from the onset of acute appendicitis. The most often outcome of early infiltrates is the formation of periappendicular abscess.

The clinical symptomatology of such infiltrate does not have any essential features in comparison with the clinical course of acute destructive appendicitis, but on examination of the patient, as a rule, there is a tension of the abdominal wall in the right iliac area, which prevents the detection of infiltrate. Only in case of absence of protective tension of the abdominal wall the infiltrate may be determined as a painful dense formation, sometimes mobile, without precise borders.

On appearance of infiltrate at later terms from the onset of the disease (3–5 days) the initial pains usually decrease and assume the character of dull, bagging pains, and are localized in the right iliac area. A "cough sign" and tenderness on palpation in the right iliac area are preserved, where a painful, without precise borders formation is defined. The features of irritation of the peritoneum are absent or they are weakly pronounced only in this area. The temperature of the body is normal or subfebrile. A moderate leucocytosis with insignificant shift to the left is observed in the blood.

At favorable course, the late infiltrate is soon precisely limited and its sizes gradually decrease since the 7–8th day of the disease; depending on the rate of involution of inflammatory process in 3–5 weeks the infiltrate is not defined. Tenderness at the deep palpation in the right ileal area frequently disappears by this time. Augmentation or stability of the sizes and too slow resorption of the infiltrate with a high probability specify destruction of the appendix and formation of abscess or tumoral nature of a palpated formation.

With infiltrate, which has arisen on the 3–5th days from the beginning of the disease, conservative treatment (bed regimen, antibiotics, local thermal procedures) is usually applied. However, it is necessary (especially at the patients of elderly age) to determine if the palpated formation is not a tumour before applying physiotherapeutic procedures. The patient should be in the hospital until complete disappearance of infiltrate and in doubtful (concerning a tumoral nature of formation), cases can be discharged only after X-ray examination of the gastrointestinal tract. Appendectomy is recommended to all patients exposed to successful conservative treatment concerning an appendicular infiltrate, or immediately after a complete resorption, or in 4–6 weeks after discharging from the hospital. At an early infiltrate or infiltrate which has arisen in later terms, but mobile and loose, appendectomy is admissible with resection of the infiltrated appendix and swelling site of the large omentum fused to the appendix. In these cases the operation comes to an end by introduction of microirrigator in the right ileal area, through which intraperitoneal introduction of antibiotics is carried out during 3–4 days.

If in the case of clinical complications (rising of temperature, sometimes with chill, intensifying pain with augmentation of the sizes of infiltrate,

the rising of leucocytosis and increase of shift to the left in the leucocyte formula) abscess is supposed, the operation (opening of abscess, erasion from the purulent cavity of fibrinotic-necrotic applyings and its active drainage through the operational wound or additional lumbar incision) is indicated. Such drainage is better to carry out with double-lumen drain, ensuring an opportunity of irrigation of the purulent cavity by antiseptic solutions and long conservation of drainage function system.

Infiltrates and Abscesses in the Area of the Small Pelvis

The most probable causes of pelvic infiltrates and abscesses with acute appendicitis are:

— destructive appendicitis with pelvic location of the appendix;

— in purulent exudate flowing with acute appendicitis complicated by peritonitis, under conditions of usual location of the appendix;

— bacterial contamination of exudate or blood, which has got into the small pelvis at appendectomy;

— delimitation of purulent exudate as an outcome or complication of diffuse purulent peritonitis.

Clinical picture of pelvic infiltrate and abscess consists of inflammatory process in the region of the small pelvis: pain in the inferior part of the abdomen and above the pubis, subfebrile or increased body temperature up to 38°C and more, tenesmuses, liquid frequent stool with an admixture of mucous, tendency to meteorism, urinary retention or, on the contrary, painful frequent micturated urges.

The large importance in diagnosis of this complication has digital investigation of the rectum: tonus decrease of the anus sphincter, tenderness, induration with the subsequent softening and hanging of the anterior wall of the rectum with a high probability indicate the presence of pelvic infiltrate and formation of an abscess. Therefore digital investigation of the rectum in men and vaginal research in women is indicated to all patients, operated on destructive appendicitis (irrespective of a locating of the appendix), as well as adverse course of the postoperative period. It allows not only diagnose pelvic infiltrate in time, but also estimate its course, the efficiency of treatment, and to determine the indications to an operation.

The conservative treatment of the patients with a pelvic infiltrate consists in application of local thermal procedures (microclyster of temperature 38–40°C), antibiotics and obligatory bed regimen in Fovler's position. At occurrence of signs of abscess (softening of dense infiltrate at digital investigation of the rectum, hanging of its anterior wall, fever with chill, the pains are more often in the left iliac area etc.) the operation is indicated.

After a local or general anesthesia and stretching of the anus sphincter, examination of the rectum with the help of a mirror and its processing with alcohol specifies a place of softening and the puncture with a needle is made. If there is pus in the syringe at aspiration, without taking out a needle, a small vertical incision is made carefully on an average line of the anterior wall of the rectum, and then this aperture is dilated by a packer or finger. Drainage tube is introduced into a purulent cavity, it is fixed by a bandage or sutures to the skin of the perineum. "Blind" diagnostic puncture of the softening, outside the operation room and absence of readiness to make the abscess opening is dangerous and should not be applied.

At correct diagnosis after the opening and excretion of the purulent cavity, the condition of the patient is quickly improved. For the period of drainage, which proceeds until the purulent discharge disappears, the patient is administered: tinctura araliae or opium (5–7 drops 2 times a day), the mild diet, the same regimen and position of the patient as at conservative treatment of pelvic infiltrate.

CHRONIC APPENDICITIS AND OTHER DISEASES OF THE APPENDIX

Morphological basis of chronic appendicitis is usually the consequences of acute inflammation of appendix (cicatrical deformations and strictures sometimes extending to all layers of its wall, causing a complete or partial obliteration of the lumen, etc.). Besides pathological changes in the appendix, essential importance in occurrence of painful sensations and functional disorders of the intestine has also multiple, flat adhesion and adnations in the ileocecal area, which frequently are found out at operations concerning chronic appendicitis. Sometimes proliferation of elements of connective tissue in its wall is so pronounced that the appendics becomes firm, enlarges in a diameter, and this fibroplastic process can extend to the caecum cupula ("fibroplastic appendicitis"). At segmental obliteration of the appendix lumen, the mucous may be accumulated in its distal parts, that results in formation of the mucous cyst or liquid accumulation and formation of hydrops of the appendix. Purulent inflammation and accumulation of a purulent exudate with formation of empyema of appendix may develop in the closed spaces (at segmental obliteration). In rare cases, the main reason of chronic appendicitis is the foreign bodies, helminths, and lamblia penetrating its lumen or wall.

Clinical course of chronic appendicitis is rather original, but the most frequent complaint is the pain in the right iliac area as short-term

attacks or constant, that is often intensified under the influence of physical load, walking at the end of a working day. This pain is frequently accompanied by a delay of stool, heavy sensation in the inferior part of the abdomen and right iliac area.

According to the clinical course we differentiate three forms of chronic appendicitis:

1. Chronic relapsing appendicitis characterized by attacks of acute appendicitis against a background of unpleasant or painful sensations in the right iliac area;

2. Chronic residual appendicitis, when the painful sensations and other signs of this disease after acute appendicitis are still present;

3. Primary-chronic appendicitis, when the clinical signs of disease and morphological changes in the appendix developed gradually without any signs of attack of acute appendicitis in anamnesis.

Diagnosis of various forms of chronic appendicitis is based on the data of anamnesis and clinical manifestations of the disease. In some cases a rather useful information (deformation of the appendix, the local narrowing of its lumen, foreign bodies etc.) can be received at X-ray examination of the appendix and ileocecal area.

Differential diagnosis should be performed with diseases accompanied with the same painful symptoms (tuberculosis of the ileocecal area, intestinal form of lambliasis, mesadenitis of different character etc.). Besides, other diseases of the appendix can cause painful sensations in the right iliac area and pain irradiation (except pelvic, retrocecal location of the appendix when pain may be irradiated to the right scapula, groin, scrotum, right leg that is caused by response of adjoining organs — the ureter, gall bladder, urine bladder to the formation of inflammation focus) — diverticula, carcinoid, benign and malignant tumours.

Treatment of chronic appendicitis consists in appendix removal like in acute appendicitis. However, some painful sensations and other signs of the disease may be kept after the operation (appendectomy).

Lecture 7
ACUTE PANCREATITIS

Acute pancreatitis is aseptic inflammation of the pancreas of the demarcative character based on the processes of necrobiosis of pancreocytes and enzymic autoaggression with the following development of necrosis, gland degeneration and joining of secondary infection. The notion "pancreonecrosis" introduced in the clinical practice by A. N. Bakulev and V. V. Vinogradov (1951) most exactly corresponds to modern concepts about pathogenetic essence of the disease.

Pancreatitis takes the third place among the main forms of acute surgical diseases of the abdominal organs (9–12%) and does not have any tendency to stabilization and reduction. General mortality rate is 4–21% and reaches 50–85% in its destructive forms and 98–100% — in the fulminant form. Undoubtedly, social significance of the problem of acute pancreatitis is great as nearly 70% of patients are active and working people and the highest age level of the disease is 30–50. Women fall ill 2.2 times more often than men.

Etiology. It is generally accepted that acute pancreatitis is polyetiological but monopathogenic disease. Different authors report about 140 etiological factors of acute pancreatitis. The most frequent causes are cholelithiasis (40–80%), chronic alcoholism (13–39%), trauma of the pancreas including intrasurgical one (up to 10%), etc.

However, in variability of the etiological factors of acute pancreatitis the place of their affection is a structural unit of the gland — the acinus. Any damage to the pancreatic acinus or its secretory hyperactivation with the following release of active anzymes of the pancreas or enzymes that become self-activated into the interstitial tissue results in development of acute pancreatitis.

There are three interconnected groups of causes of acute pancreatitis (Savelyev V. S., 1983):

1) mechanical;
2) neurohumoral;
3) toxico-allergic.

All factors causing primary mechanical damage of different anatomical pancreatic structures are related to mechanical group. They are, first of all,

factors that cause intraductal hypertension, biliary-pancreatic reflux, duodenal-pancreatic reflux, dyskinesia of the duodenum. Of importance is direct trauma of the gland including a surgical one. All stress situations make the second group of causes including hormone therapy and different neurohumoral effects. Toxico-allergic group includes infection, allergy, immunologic disorders, and drug intolerance.

Pathogenesis. S. A. Shalimov et al. (1990) formed clinical and pathochemical conception of pathogenesis: the triggering mechanism of the disease is the cause that brings about damage of the acinous cells of the pancreas; as a result the cells release an active substance — cytokinase (prokinase) that activates tripsinogen even in a small amount transforming it in tripsin; the formed active tripsin activates callycreinogen, chimotripsinogen, proelactase, procarboxipeptidase and tripsinogen. Activation of tripsinogen by tripsin and its transformation into active tripsin is the beginning of the autocatalytic process. Further development of the autocatalysis process depends on correlation of components in the system "tripsin-tripsin inhibitor". In prevalence of the inhibitor (in sufficient compensatory mechanisms), inhibitors inactivate the activated tripsin and balance in the system is restored. In the inhibitor deficiency, the balance between proteolytic and antiproteolytic activity is disturbed. It results in progress of the autocatalytic process and development of the clinical picture of acute pancreatitis. Because of activation of the proteolytic enzymes in the pancreatic tissue and their release in the blood, there occurs activation of the callycreinkinine system accompanied by decrease of the vascular tone and disturbed permeability of the vessels. The activated pancreatic enzymes penetrate into the vena cava and thoracic lymphatic duct conditioning the first period of endogenic intoxication and affection of other organs and systems. Accumulation of the exudate in the abdominal cavity, which is rich in activated proteolytic enzymes, causes a considerable reaction of the peritoneum, enhances intoxication and aggravates general state of an organism. A strangulated, swollen head of the pancreas compresses the distal part of the common bile duct and may lead to development of jaundice. Dissolution of the tissues and organs takes place at the places of accumulation and retention of the active pancreatic juice. It results in formation of retroperitoneal phlegmons, gastric and intestinal fistulas, erosion of vessels.

Peculiarities of pathogenesis of the main clinical and morphological forms of acute pancreatitis (edematous pancreatitis, fatty and haemorrhagic pancreonecroses) are the followimg. In edematous form, that develops near the foci of pancreocyte destruction, hyperergic, inflammatory, mainly microvascular plasmorrhagic reaction results in considerable interstitial edema of the pancreas — the basic morphological sign of this form of the

disease. The plasmic systems of inhibitors are revealed in the interstitial space and become one of the decisive factors preventing enzymatic autoaggression. Recovery is spontaneous or under the influence of conservative therapy.

A progressive course of pancreonecrosis is conditioned by pathological influence of the system of activated pancreatic enzymes on the gland tissue, at first from the interstitial lymphatic flow and later through the general blood flow. In cases when lipolytic group of enzymes is mainly pathologically activated both pancreatic (phospholipase A, lipase) and tissue (lipase of the lipid tissue) fatty pancreonecrosis is formed. In prevailed activation of the system of the pancreatic proteolytic enzymes (elactase, tripsin) haemorrhagic pancreonecrosis develops. The same factors condition accelerated disease development. Fatty pancreonecrosis makes slow progress. The inflammatory process involves peripancreatic tissues and organs within the fascial sheaths of the abdomen. It rapidly results in formation of the parapancreatic effusion and development of pancreatogenic peritonitis. Haemorrhagic pancreonecrosis is characterized by rapid progressive course of the disease with formation of hemorrhagic effusion-aseptic enzymatic peritonitis. Simultaneously there is usually spread of the exudate in the retroperitoneal and pericolon space and development of aseptic dystrophic retroperitoneal phlegmon. Thus, in fatty and haemorrhagic pancreonecrosis intensive conservative therapy of the disease is necessary as well as application of surgical methods of treatment.

Etiological classification. At present, it is generally accepted that acute pancreatitis is a polyetiological disease. A great number of causes of acute pancreatitis can be united in three groups: mechanical, neurohumoral and toxical-allergic.

Mechanical causes are pancreatic traumas including surgical ones, all kinds of occlusion of the hepatic-pancreatic ampoule and pancreatic ducts: calculi, cicatricial stricture, tumour, inflammatory edema of the large duodenal papilla, stable spasm of the sphincter of the hepatic-pancreatic ampoule, duodenal dysfunction — dyskinesia in stomach ulcer, duodenostasis, duodenal hypertension, duodeno-pancreatic reflux. Causes that bring about mechanical trauma of the gland are of greater importance in the clinical practice, especially those leading to intraductal hypertension. These causes may be both organic and functional.

Neurohumoral causes include disorders of lipid metabolism, systemic diseases of the vessels, functional diseases of the stomach, secondary disorders of blood circulation in the pancreas, diseases of the liver, heart, pregnancy and postpartum period, surgery on the heart and lungs and other organs.

Toxical-allergic causative factors include food and drug allergy, foci of acute and chronic infection, alcoholism, poisoning.

Of practical value is division of etiological factors into the predisposing and producing factors. The predisposing causes are constitutional-anatomical, hereditary, seasonal-alimentary and toxical-allergic factors. The producing causes are provoking food loading (alcohol, fatty and spicy dishes), neurohumoral influences, blood circulation disorder in the pancreas.

The producing factors determine clinical-morphological form of acute pancreatitis, the course and character of complications. Study of the producing causative factors allowed to distinguish the following etiological forms of acute pancreatitis: 1) ductogenic (duodenobiliary, i. e. refluxogenic and genuine); 2) cholecystogenic; 3) contact; 4) angiogenic; 5) posttraumatic; 6) criptogenic; 7) pancreatitis as a complication of another disease.

Ductogenic forms of acute pancreatitis are observed most frequently (about 50%). Detection of these forms is of practical value as obstructive processes in the secreto-excreting system may be eliminated only by surgery. Necessity of purposeful diagnosis of other etiological forms of acute pancreatitis is also of great importance.

Clinical and morphological classification. The morphologists have considered for a long time that acute pancreatitis is a combined concept including a number of quite different diseases of the pancreas. A morphologic characteristic proposed by A. I. Abrikosov (1954) is the closest to modern classification; he distinguished acute haemorrhagic necrosis, fatty necrosis of the pancreas and acute catarrhal and purulent pancreatitis. The concept "pancreonecrosis" was first introduced in the clinical practice by A. N. Bakulev and V. V. Vinogradov (1951). They distinguished "edematous pancreatitis", "different kinds of pancreonecrosis" and "purulent pancreatitis".

S. O. Shalimov (1990) made the following classification of acute pancreatitis:

According to morphological changes:

1. Edematous:
— serous;
— serous-haemorrhagic.
2. Necrotic (pancreonecrosis):
— haemorrhagic (microfocal, macrofocal, subtotal, total);
— fatty (microfocal, macrofocal, subtotal, total);
— mixed (microfocal, macrofocal, subtotal, total).
3. Purulent:
— primary-purulent;
— secondary-purulent, exacerbation of chronic purulent pancreatitis.

According to severity:

1. Mild.
2. Moderate.
3. Severe.
4. Grave (fulminant).

According to the clinical course:

1. Regressive.
2. Progressive.
3. Recurrent.

According to the presence of complications:

1. Local complications (complications in the gland itself).
2. Intraabdominal complications.
3. Retroabdominal complications.

Of greatest importance in the practical activity are three clinical-morphological forms of acute pancreatitis: edematous pancreatitis, fatty pancreonecrosis and haemorrhagic pancreonecrosis.

It is also expedient to distinguish periods of destructive pancreatitis (pancreonecrosis) accepted at the V All-Russian Congress of Surgeons in 1978:

— period of haemodynamic disturbances and pancreatogenic shock;
— period of functional insufficiency of the parenchymatous organs;
— period of dystrophic and purulent complications.

According to the international classification of diseases of the X revision (ICD-X) there are:

— acute pancreatitis, edematous form;
— acute pancreatitis, destructive form.

Clinical diagnosis. Acute pancreatitis is characterized by plurivisceralіty, and the most permanent symptoms are pain localized in the epigastric area, multiple vomiting with duodenal contents, meteorism. In other words, acute pancreatitis does not have clear typical clinical picture and diagnosis of this disease, especially in plurivisceral period, is frequently a difficult task.

The onset of the disease is often sudden.

The most significant symptom of acute pancreatitis is pain. The pain syndrome in acute pancreatitis is manifested by different ways and ranges from insignificant to unbearable pain but it is not characteristic of forms of pancreonecrosis. There are no painless forms of pancreonecrosis observed. The pain syndrome cannot be determined in pancreatogenic shock, delirious syndrome or pancreatogenic coma.

Pain is localized in the epigastric area in most patients (95%), in half of cases pain is in the left subcostal area. Combination of the pain syndrome in the epigastrium and pain in the right subcostal area is observed much rarer, thus localization of pain in the epigastric area with its spreading to the left

subcostal area is an improtant differential sign. In acute pancreatitis pains are frequently irradiate transversally, encircling (65%), rarely they irradiate in the shoulder girdle, substernally and in the heart area. As a rule, intensity of the pain syndrome corresponds to severity of the disease course. In pancreonecrosis moderate pains are observed only in 6% of patients, severe pains — in 40%. In most cases (up to 50%) pains are very severe unbearable and accompanied by collapse in 10%.

One may make a conclusion about topics of the main changes in the pancreas by pain localization. Encircling pains are characteristic of total affection of the gland, in the right subcostal area — affection of the head, in the epigastrium — affection of the body, pain in the left subcostal area — localization of the process in the tail of the pancreas. In localization of the process in the tail of the gland pains irradiate into the left costal-spinal angle (Mayo-Robson's symptom). However, the objective pathognomic symptom of acute pancreatitis is pain in the epigastric area and left subcostal area.

The other significant symptom of acute pancreatitis is vomiting. Not infrequently, vomiting precedes pains and it gave V. M. Voskresensky grounds to consider it a cause of development of acute pancreatitis in people with alcoholic intoxication and after overeating. During vomiting, the patient suddenly feels severe unbearable pains in the abdomen and they are so acute that one may think of perforative stomach ulcer. However, repeated vomiting and absence of other signs of perforation give a possibility to refuse this diagnosis very rapidly. Vomiting is explained by irritation of the stomach and duodenum, solar plexus, paresis of the intestine and reflex effect.

In acute pancreatitis vomiting is peculiar. V. M. Voskresensky wrote that vomiting may be repeated and painful. Sometimes it is of uncontrollable character. Some patients under our observation were racked more from vomiting than from pains. During a short period free from vomiting patients made an attempt to sip some water but again it caused long-term and painful attack of vomiting. The picture is so severe and characteristic that we may suspect acute pancreatitis only by frequent, painful vomiting. As it is seen vomiting does not give relief to a patient, on the contrary, his state worsens. At first vomiting corresponds to the stomach contents, then bile admixtures and sometimes blood (in haemorrhagic and necrotic pancreatitis) appear.

From the first hours of the disease most of patients experience acute abdominal distension, retention of defecation and gases, i. e. signs of acute intestinal obstruction, which is supplemented by vomiting. In most patients, the whole abdomen is distended or its upper part and this symptom is more frequent in mild forms of pancreatitis. Abdominal distention may be consi-

derable but it cannot be always determined as many patients with pancreatitis have a thick abdominal wall. Therefore, it is necessary to follow up this symptom in dynamics by measuring abdominal circumference with a tapeline and writing down the figures obtained. In some patients, the abdomen is flattened at the expense of muscular tension. The abdomen does not participate in breathing in most patients due to severe pains in the abdomen, distension, tension of the abdominal wall.

A very frequent symptom of acute pancreatitis is icteric skin and scleras because of affection of the liver and disintegration of erythrocytes.

The patients' behaviour is restless, even with disturbance of the mental state (signs of acute psychosis). Sometimes such patients are directed to the mental hospital (especially in presence of signs of alcoholic intoxication).

Superficial palpation reveals tension of the muscles of the anterior abdominal wall, specifies localization and expressiveness of tenderness and detects possible pathological formations in the abdomen. Tension of the muscles of the anterior abdominal wall is more frequently marked in the epigastric area, rarer — all over the abdomen when the peritoneum is involved in the process. In considerable tension the value of palpation is reduced. However, one can establish expressiveness of tension, localization and intensity of tenderness; it helps in making the diagnosis. When muscular tension does not interfere examination, palpation may reveal pasty state, "filling" of the epigastrium along the pancreas and sometimes there may be found painful infiltrate stretched in the transversal direction. In formation of a cyst or abscess that may occur in several days after the onset of the disease, it is possible to find a round or oval formation of different size, which occupies the whole epigastric area. While examining the abdomen one should have an idea about projection of the pancreas on the anterior abdominal wall.

Percussion is made very carefully determining intensity, spreading and localization of the percussion tenderness. Drastic edema and haemorrhages in the pancreas with involvement of the transversal colon mesentery will give dullness of the percussion sound in the epigastrium and sometimes — dull sound along the whole length of the abdomen, which is especially marked in formation of the cyst or infiltrate that push the stomach and transversal colon aside. Presence of the exudate in the abdominal cavity is marked by shortening of the percussion sound in the flat places and shifting of the dullness border in change of the body position. On percussion, one may hear a high tympanic sound in the epigastric area or all over the abdomen when the stomach and the intestine is sharply inflated by gases in paresis.

Auscultation reveals drastic weakening of the intestinal sounds or their complete disappearance. It depends on expressiveness of the process and paresis of the intestine.

There is a series of special symptoms for diagnosis of acute pancreatitis, but their value is different.

Sarfert's symptom — cyanosis of the skin.

Mondor's symptom — violet spots on the face and trunk.

Lagerlöff's symptom — sharp cyanosis of the face.

Halsted's symptom — cyanosis of the abdominal skin.

Turner's symptom — cyanosis of the lateral walls of the abdomen.

Cullen's symptom — yellow colour of the skin around the umbilicus.

Joston's symptom — yellow-blue colour of the umbilicus.

Development of this group of symptoms is explained by action of the enzymes and hemodynamic disturbance.

Körte's symptom — painful resistance as a transversal stripe in the epigastric area 6–7 cm above the umbilicus.

Voskresensky's symptom — absence of pulsation of the abdominal aorta in the epigastric area.

Mayo-Robson's symptom — tenderness on pressing of the finger tips in the left costal-spinal angle.

Razdolsky's symptom — percussion tenderness over the pancreas.

Makov's symptom — hyperesthesia of the skin above the umbilicus.

Katsch's symptom — hyperesthesia of the skin along the left paravertebral line at the level of D_7–D_9.

Shchetkin — Blumber's symptom — is a nonspecfic symptom expressiveness of which depends on intensity of the peritoneal phenomena.

Chukhrienko's symptom — tenderness in the epigastric area in pushing movements of the abdominal wall from downward to upward and from the front to back by the doctor's hand placed crosswise below the umbilicus.

The diagnostic value of special symptoms increases when they are detected in a whole complex.

Fatty pancreonecrosis is characterized by fast formation of the inflammatory infiltrate in the epigastric and left subcostal area. Generally the infiltrate is clearly revealed in 3–5 days from the onset of the disease. In recurrect course of the process there may be formation of the infiltrate in much later period.

Severity of the clinical manifestations of hemorrhagic pancreonectrosis is conditioned by acceleration of the pathological process with involvement of the retroperitoneal cellular tissue that oozes out hemorrhagic exudate and is transformed into detritus-like mass under the influence of enzymes; accelerated development of serous-hemorrhagic enzymatic peritonitis. Progressive hemorrhagic pancreonecrosis is characterized by very intense pain

of the encircling character, multiple vomiting, intestinal distension, drastic weakness or absence of peristaltic sounds. Free liquid is rapidly enough detected in the abdominal cavity as well as positive symptom of Shchetkin — Blumberg. The symptom of Mayo — Robson is usually observed, which expressiveness progresses in general, and not infrequently, it is accompanied by tension of the transversal muscle on the left. Presence of puffiness of the transversal areas is a sign of severe retroperitoneal phlegmon.

Mixed forms of pancreonecrosis — fatty with hemorrhages and hemorrhagic with fatty necroses — are characteristic of signs of the corresponding forms of necrosis: in the first case — early development of the inflammatory infiltrate in the epigastrium; in another case — fast development of signs of disturbances of the peripheral and central hemodynamics (circulatory shock) and enzymatic peritonitis.

Of great significance are manifestations of toxemia in diagnosis of forms of acute pancreatitis. A group of symptoms having diagnostic and prognostic value characterizes the pancreotogenic toxemic syndrome. The most constant symptoms are change of the skin colour, dry tongue, tachycardia, dysfunctions of the kidneys, lymphocytopenia.

Changed colour of the skin is manifested by different variants: pallor, icterus, cyanosis, marble skin, vascular spots, etc. Progress of toxemic disturbances causes acrocyanosis, vascular spots and marble pattern on the trunk skin, increased dryness of the skin — in 35–40% of patients and on the contrary, increased moisture, cold stick sweat — in 20% of patients. Dryness of the tongue is a constant symptom of the toxemic syndrome.

The cardinal symptoms of pancreatogenic toxemia are tachycardia (90–120 bpm) in normal or subfebrile temperature and changes of the arterial pressure. Relative hypertension often observed at the onset of the disease transforms into relative hypotension and even collapse in deepening of toxemia. Practically all patients are observed to have decreased diuresis, marked oliguria is found in 60–65% of patients. Thus, pancreatogenic toxemic syndrome is a reliable clinical criterium for diagnosis of the progressive course of the disease that have limited symptomatology.

There are mild, moderate and severe degrees of toxemia, which criteria are clinical symptoms of toxemia, data of some special methods that characterize microcirculation and rheology of the blood: capillaroscopy, level of erythrocyte aggregation, etc.

The mild degree is characterized by pallor of the skin, moderate tachycardia. There is dryness of the tongue, relative hypertension, reduced diuresis, moderate lymphocytopenia. Capillaroscopy reveals pericapillary edema, spasm of the arteriolar and some dilatation of the venular parts of the capillaries, slowing down of blood flow velocity. Rheologic properties of blood are not disturbed in this period.

In toxemia of moderate degree of severity, there are hyperemias of the facial skin, sometimes with icterus, increased dryness of the skin, moderate dyspnea. Hemodynamic disorders are manifestated by tachycardia with inconsistent temperature by 20–30 bpm, relative hypotension, oliguria, lethargy and euphoria. The capillaroscopic picture is characterized by marked pericapillary edema, marked dilatation of the venular part of the capillaries and some dilatation of the artriolar part, drastic slowing down of the blood flow, stasis in the venular part, development of signs of sludge-syndrome. There are moderate aggregations of erythrocytes, lymphocytopenia.

Toxemia of severe degree is characterized by: acrocyanosis, vascular spots or marble pattern of the trunk skin, increased moisture of the skin, cold sweat, very dry tongue, marked dyspnea, tachycardia — 120 bpm, hypotension and collapse, delirium, significant oliguria, hematuria, cylindruria. There is always pronounced lymphocytopenia. On capillaroscopy, considerable microcirculation disorders are observed: dilatation, stasis and microthromboses of the capillaries, pericapillary edema, diapedesis of erythrocytes, and significant reduction of the number of functioning capillaries. There is marked worsening of haemorheologic indices: the degree of erythrocyte aggregation and blood viscosity increases 305 times as compared with the norm, there are severe disturbances in the system of blood coagulation.

Pancreatogenic toxemia arises and becomes most pronounced in the first period of the disease, i. e. in the first 5 days from the onset of the disease.

PROGRESSING PANCREONECROSIS

Clinical symptoms of both slowly and rapidly progressing pancreonecrosis are variable and change depending on the disease period.

The progressive course of pancreonecrosis is characterized by totality of sequential pathomorphological changes in the gland as well as pathophysiological changes in an organism and may be divided into three clearly outlined clinical periods:

1) haemodynamic disturbances and pancreotogenic shock;
2) functional insufficiency of the parenchymatous organs;
3) postnecrotic dystrophic and purulent complications.

The Period of Haemodynamic Disturbances and Pancreatogenic Shock

Duration of the first period ranges from some hours to 3–5 days. Pancreatogenic toxemia arises and reaches its maximum during this time. The period is characterized by generalized microcirculation disorders, increase of general peripheral resistance, reduction of the volume of the

circulating blood, worsening of indices of the central hemodynamics. Pancreatogenic circulatory shock is based on these haemodynamic disturbances.

Disturbances of peripheral circulation and central haemodynamics characteristic of the first period depend on clinical and anatomical forms of pancreatitis, extension and course of the process.

In edematous pancreatitis, disturbances of haemodynamics are the least marked. They are often observed only in the first hours and minutes of the disease and then rapid adaptation takes place. In slowly progressing course characteristic of fatty pancreonecrosis, haemodynamic disturbances are more marked and their maximum is observed on the 2nd–3rd day of the disease.

Haemodynamic disturbances are eliminated before formation of parapancreatic infiltrate in most patients with fatty necrosis. In rapidly progressing haemorrhagic pancreonecrosis when there are very severe pains in the epigastric area with typical irradiation, of the encircling character, signs of drastic disturbances of the central and peripheral haemodynamics prevail in the clinical picture. These disturbances result in pancreatogenic circulatory shock in 20% of patients when the patients' condition is severe or grave. The skin is cold, moist, and pale, not infrequently of spotty or marble colour. There are acrocyanosis, dyspnea, frequent and thready pulse, compliance of the superficial veins, hypotension with acute reduction of the pulse presssure, minute's volume of the heart and volume of the circulating blood, reduction of the central venous pressure, changes on the ECG by the ischemic type. Laboratory studies reveal metabolic acydosis. Such disturbances of haemodynamics, especially when they are accompanied by irradiation of pain in the left side of the chest, ischemic type of ECG in insignificant abdominal symptomatology complicate considerably the identification of haemorrhagic pancreonecrosis. Differential-diagnostic errors are most frequent in such patients towards hyperdiagnosis of myocardial infarction. Clinical interpretation of ECG in pancreonecrosis is difficult, for in many cases the disease develops against the background of coronary heart disease. On the other hand, some patients develop myocardial infarction against the background of progressive haemorrhagic pancreonecrosis. It drastically worsens the course of the disease and may be a cause of diagnostic mistakes.

Many patients may have mental disorders in the first period: anxiety, excitement, delirium, long-term prostration, delirious state that may make the diagnosis of pancreonecrosis difficult.

The Period of Functional Insufficiency of the Parenchymatous Organs (Plurovisceral Period)

The second period begins on the 3rd–7th day of the disease and is mainly manifested by dysfunction of the vitally important organs: the lungs, liver, kidneys, heart, adrenal glands and affection of the CNS.

The degree of expressiveness of these disorders is different. Abdominal manifestations of pancreonecrosis are most pronounced in this period; however, polyorganic insufficiency may cause complications in differential diagnosis. The degree of affection of the parenchymatous organs is directly proportional to the degree of pancreatogenic toxemia. Severe respiratory failure rapidly develops in toxic affection of the lungs.

The main symptom of respiratory failure is dyspnea, which is accompanied by signs of arterial hypoxemia. X-ray examination usually reveals disc-like atelectases, focal pancreatogenic pneumonitis, and effusion in the pleural cavities. Changes in the lungs in unexpressed abdominal symptoms of pancreonecrosis may be interpreted as primary by mistake. In haemorrhagic pancreonecrosis there are observed severe forms of respiratory failure with a clinical course resembling the picture of "shock lungs".

In acute pancreatitis dysfunction of the liver does not usually have adequate clinical manifestation, only 4.2% of patients develop toxic hepatitis, in severe cases it changes into toxic dystrophy of the liver. Signs of pancreatogenic hepatargia are mental disorders, dryness of the skin, skin subicterus, rarer — jaundice, tachycardia, stable hypertension, diminution or enlargement of the liver, tendency to bleeding. Laboratory studies reveal thrombocytopenia, bilirubinemia, hyperglycemia, hyporpoteinemia.

Functional failure of the kidneys is observed in most patients with pancreonecrosis. It is manifested by oliguria, marked proteinuria, increasing azotemia and creatinine. In severe pancreatic toxemia, there may be development of acute renal failure.

Results of numerous studies give evidence of dysfunction of the adrenal glands in acute pancreatitis. No doubt, in progressing pancreonecrosis, the adrenals participate in general syndrome of adaptation and their dysfunction depends directly on the degree of toxemia. The manifestations of changes of the adrenal functions are the same as in other acute surgical diseases (peritonitis, intestinal obstruction, etc.).

One can judge about the functional state of the adrenals in patients with pancreonecrosis only after determination of the adrenal hormone contents in blood and urine. In pancreonecrosis there may be development of acute adrenal failure as a result of direct involvement of the adrenals

in the pathological process (in pancreatogenic retroperitoneal aseptic phlegmon and its extension to perirenal cellular tissue).

60% of patients with progressive haemorrhagic pancreonecrosis are observed to have mental disorders mostly manifested by excitement or lethargy, behaviour inadequacy. One third of the patients are observed to have severe changes in the CNS — delirium and coma. Pancreatogenic delirium and coma, especially developed prior to hospitalization are not infrequently mistaken for alcoholic ones; therefore, most patients have indication of alcohol abuse in their history. There are no clear differential-diagnostic, clinical and laboratory criteria for alcoholic and pancreatogenic delirium syndromes till present.

The Period of Postnecrotic Dystrophic and Purulent Complications of Pancreonecrosis

The third period usually begins on the 7th–14th day from the onset of the disease and is characterized by development of local postnectrotic processes. These processes may be of reparative character and have an aseptic course with formation of parapancreatic infiltrate, cyst (untrue one is more often) of the pancreas or have a septic way of development when combination with secondary infection results in purulent and apostematous pancreatitis, formation of parapancreatic abscesses and phlegmon of the retroperitoneal cellular tissue, destruction of the walls of the organs and blood vessels.

As to diagnosis, the third period is the most severe. Semiotics of this period is determined by totality of such complications as infiltrate, cyst, abscesses of the pancreas and omental bursa, purulent peritonitis, fistula, arrosive bleedings, compression and thrombosis of the splenic vein and large vessels adjacent to the pancreas.

The postnecrotic infiltrate is pathognomic of fatty and mixed pancreonecrosis. On palpation the infiltrate may be found early enough, up to 3rd–5th day of the disease. It is more frequently detected before subsiding of the acute process. Solid, moderately painful inflitrate without clear borders is easily palpated in the epigastric area, in the left, rarer in the right subcostal region, sometimes in the whole upper abdomen. In absence of complications (abscess formation, suppuration) the infiltrate is slowly resolved. In aseptic involution of the infiltrate there are no hectic temperature, septic changes in the leucogram. However, there are moderate leucocytosis, accelearted ESR and α-amylase clearance. Duration of infiltrate involution is variable and may persist from 2 to 8 weeks.

Involvement of the cellular tissue of the right paracolic space into the pathologic process may lead to mistaken diagnosis of acute appendicitis. It may be a cause of hyperdiagnosis of malignant tumour as the acute process subsides before the formation of the infiltrate. Roentgen-contrast examination of the stomach made at this period may lead to mistaken diagnosis of tumorous infiltration because of deformed and pushed forward posterior wall of the stomach fixed in the infiltrate.

Purulent complications usually develop in 14–30 days after development of acute pancreatitis. The main sign of starting purulent complications of pancreonecrosis is classic purulent-resorptive fever having alternative character at first and then being hectic. As a rule, hyperthermia is accompanied by chill, increased sweating, tachycardia. The degree of expressiveness of purulent-resorptive fever depends on spreading and localization of the purulent process as well as on virulence of the microbic flora. Sometimes the CNS disturbances are brought to the forefront; they are excitement, delirious syndrome. Some patients may not have purulent-resorptive fever, and the main symptom of pronounced purulent intoxication is tachycardia and considerable disparity between pulse rate and body temperature. These "scissors" are unfavourable prognostic sign.

Local symptomatology of purulent complications is frequently unexpressed, and concealed by general symptoms of intoxication in a number of patients. Purulent phlegmon of the retroperitoneal cellular tissue is characterized by pain intensification in the upper part of the abdomen, lumbar area, and iliac fossa. Palpation may reveal "burning" tenderness in the costal-spinal angle, along the left and right lateral canals. Later on one may detect infiltration and edema of the subcutaneous fatty tissue and skin in the transversal area, fluctuation, peritoneal symptoms. Acute pancreatitis and other purulent-septic complications are characterized by high leucocytosis, shift of the Schilling's formula to the left, development of juvenile forms of leukocytes, plasmic cells and toxic granules of neutrophils, moderate anemia and significant elevation of ESR, increased intoxication leucocytic index.

Visceral complications in the postnecrotic period are extremely variable: interal and external fistula of the pancreas, stomach and intestine, external and internal arrosive bleedings, compressions and thromboses of the splenic and portal veins.

Clinical diagnosis of dystrophic and purulent complications of pancreonecrosis is difficult and therefore it is necessary to apply special methods of investigations for their topic diagnosis. The main methods of diagnosis are roentgen-contrast investigation of the stomach, relaxion duodenography, laparoscopy, gastroduodenoscopy, angiography, ultrasonography, thermography and others.

Enzymic diagnosis. Excretion and tissue enzymes of the pancreas as diagnostic criteria may be divided into two groups — indicators (amylase, transaminidase) and pathogenic (lipase, tripsin). The former are only signals of affection of the pancreas and the latter directly participate in pathogenesis of acute pancreatitis. Determination of the indicator enzymes are of greatest importance in the clinical practice.

In progressive pancreonecrosis (in contrast to edematous pancreatitis), there are four pathobiochemical syndromes that occur in turn:

1) damage of integrity of pancreocytes;
2) pancreostasis;
3) pancreonecrobiosis;
4) pancreonecrosis.

The syndrome of damage of pancreocyte integrity is characterized by appearance of tissue enzymes in blood — elactase and transaminidase.

The syndrome of pancreostasis arises due to increased pressure in the secretion excreting tracts of the pancreas because of edema of its stroma as well as rupture of the ductoacinar compounds that promotes diffusion of enzymes into the intercellular place. This syndrome is most expressed in fatty pancreonecrosis and first of all is characterized by hyperamilsemia and increased α-amylase clearance as well as increase of general activity of esterase and lipase activity in the blood serum.

The syndrome of pancreonecrobiosis is most characteristic of progressive forms of acute pancreatitis. Necrobiosis arises in considerable part of the acinar tissue of the gland and is manifested by general hyperenzymemia and disenzymemia — disturbance of normal interrelationships between activity of blood enzymes.

The syndrome of pancreonecrosis is most typical of hemorrhagic pancreonecrosis. Necrosis affects a considerable part of the acinar tissue of the gland; it results in rapid reduction of activity of excretive enzymes that was high at first and then reduced to hyperenzymemia and even to anenzymemia. Determination of α-amylase activity in the blood and urine is of greatest diagnostic value. Within first hours of the disease α-amylase activity is 1.5–2 times exceeds the normal level disregarding the following course of acute pancreatitis.

Marked differences of α-amylase activity levels are observed on the 3rd day of the disease when the process takes pronounced abortive (edematous pancreatitis) or progressive character. In the abortive course the upper border of the reliable interval of enzymatic activity is 2.5 times higher than the norm, in fatty pancreonecrosis — 3.05 times higher, in hemorrhagic one — 6–9 times higher. In edematous pancreatitis enzymatic activity decreases to the normal level on the 5th–9th day.

Total amilolytic activity of urine is determined by a classic Wolgemuth's method. This method allows to get precise results, but it is necessary to note that the method of Wolgemuth is not informative enough as to pancreatic α-amylase as it reflects only total amylolytic activity of all glycolytic enzymes that are present in the biological medium.

The lipase enzymatic test is the most reliable criterion in diagnosis of acute pancreatitis. The application of this test is limited due to wide range of fluctuation of enzymic activity, which depends on degree of lipase participation in local and general pathobiochemical processes.

Edematous pancreatitis is not accompanied by lipasemia. In fatty pancreonecrosis there is stable lipasemia within first two weeks of the diseases; in haemorrhagic — lipasemia is of short-term character and is usually marked on the 3rd–5th day of the disease. It is clearly within the concept of pathobiochemical syndromes in acute pancreatitis: pancreostasis — long-term lipasemia, pancreonecrobiosis — stable hyperlipasemia, pancreonecrosis — a sharp reduction of lipase activity.

Studies of tissue enzymes of the pancreas are of great diagnostic value. Activity of the tissue enzymes of the pancreas in blood in acute pancreatitis — elastase is usually determined by Keller and Mandle (1971) and transmidinase — by Bergmayer (1970). It is established that activity levels of these enzymes are moderately high in all forms of the disease. Activity of elastase and transminidase is not determined in the blood of healthy people. We can observe the inverse ratio in dynamic follow-up during the first 5 days of the disease: increased activity of elastate and reduced activity of transminidase in the abortive course, and reduced activity of elastase along with increased activity of transminidase in the progressive course of the disease.

Thus, the character of correlation between the activities of tissue enzymes allows establishing the character of acute pancreatitis. The fact of appearance of the tissue enzymes of the pancreas in the blood is evidence of pancreocyte destruction and forms a concept of the pathobiochemical syndrome of the same name. Most statistically reliable differences of blood enzymograms are observed on the 3rd day of the disease.

The programme of examination:

1. Obligatory laboratory studies:

— blood count, general analysis of urine;

— blood sugar;

— urine amylase;

— blood group, Rh-factor;

— biochemical blood analysis (bilirubin, ALT, AST, AP, GGT, thymol test, electrolytes, urea, protein, creatinine, urea nitrogen, uric acid);

— protein and protein fractions;

— coagulogram;
— midline molecules;
— calcium of blood serum;
— C-reactive protein (in quantitative units);
— gas compounds of blood;
(All these studies are made within 2 hours of patient's hospitalization)
2. Antibioticogram.
3. Blood culture for sterility.
4. Biochemical, bacteriological, cytologic analysis of the exudate from the pleural cavity and abdominal cavity (when there was pleural puncture or laparocentesis).
5. Obligatory instrumental examinations:
— review roentgenography of the abdominal organs;
— ultra-sound examination of the abdominal organs.
(All these examinations are made within 2 hours of patient's hospitalization).
— CT of the abdominal organs (early CT within 72 hours in severe acute pancreatitis, suspicion on infected necrosis, pseudocyst or abscess of the pancreas, determination of the amount of sterile necroses);
— laparocentesis, laparoscopy, pleural puncture — according to indications.
6. Additional laboratory studies:
— LDH;
— immunogram (including determination of interleukines (IL) 1, 2, 6, 8, 10; tumour necrosis factor (TNF-α);
— procalcytonine;
— malonic dialdehyde;
— ceruloplasmin, α1-antitripsin, polymorphonuclear elastase, phospholipase A_2 of I type;
— amylase, blood lipase.
7. Additional instrumental examinations:
— EFGDS;
— CT with contrast increase;
— spiral CT with bolus contrast increase;
— ERPCG (endoscopic retrograde pancreocholangography;
— aspiration biopsy through a thin needle with bacteriologic study of the aspirate;
— angiography.
It is necessary to consult an expert in resuscitation on admission and hospitalization of patients with severe acute pancreatitis at the intensive care unit.

It is obligatory to take into consideration the criterium of evaluation of severity degree in patients with acute pancreatitis by APACHE II, Ranson, Glasgow; assessment of polyorganic insufficiency according to MOF, as well as determination of the criteria by Balthazar with Bradly addition on the basis of CT data.

The differential diagnosis of acute pancreatitis should be made with acute cholecystitis, perforative ulcer of the stomach and duodenum, acute appendicitis, renal colic, intestinal obstruction, thrombosis of the mesenteric vessels, food intoxication, pneumonia of the lower lobe, pleurisy.

Characteristics of Therapeutic Methods

Treatment of patients with acute pancreatitis is urgent at the in-patient department. When the patient with suspicion on acute pancreatitis is detained in the diagnostic room of the reception department because of some reasons he should be given prehospital treatment. Being at the emergency surgical department and during examinations treatment should not be stopped, and the surgeon according to the clinical signs determines its character and scope. The patients with pronounced endogenic intoxication may be directed to the intensive care unit, and their treatment should be given with obligatory participation of the anesthesiologist-expert in resuscitation. Results of rapid laboratory and special examinations allow determining more precisely treatment even in cases when the patient was admitted to the in-patient department after the early period of the disease.

Treatment of the patients with acute pancreatitis depends on clinical and morphological variant of the disease.

Patients with edematous pancreatitis are treated conservatively (except biliary pancreatitis when surgical intervention is performed on the bile ducts without any manipulations on the gland).

Conservative therapy of acute edematous pancreatitis:

1. It is expedient to administer fasting in the first 1–3 days of the disease (to provide physiological rest of the gland) and intake of alkaline solutions every 2 hours (for example, sodium bicarbonate — 0.5–0.6 mg for a glass of water or mineral water “Borzhomi”).

2. Aspiration of the gastric contents through the probe to prevent ingress of the hydrochloric acid into the duodenum.

3. Analgesics — to fight with pain syndrome (preferably nonnarcotics, in some cases — tramal, tramadol, moradol).

4. Myotripic spasmolytics (no-spa, papaverin, buscopan, galidor) — to restore outflow of the pancreatic juice.

5. M-choline blockers (atropin, platiphilin, gastrocepin) — to suppress secretion of the pancreas and remove spasm of its ducts.

6. Infusion therapy (colloids, crystalloids), their volume is determined by the clinical situation; the main task of the infusion therapy in this case is fight with endogenic intoxication.

7. Antioxidants (ascorbinic acid, ceruloplasmin, tocoferol) — to reduce peroxide oxidation of lipids.

8. In gastrogenic pancreatites — H_2-blockers, antacides, sucralfat (decrease of gastric juice secretion to prevent pH reduction in the duodenum).

Duration of hospital treatment is 3–7 days.

Requirements to treatment results: control of the pain syndrome, absence of changes in blood count indicating the inflammatory process, normalization of the body temperature.

Peculiarities of treatment of biliary pancreatitis (edematious form):

1. Cholecystectomy within 24 hours from the moment of patient's admission to hospital.

2. External draining of the choledoch is indicated only in presence of expressed biliary hypertension, choledocholythiasis, and purulent cholangitis.

3. Basic therapy is the same as given above with inclusion of hepatoprotectors, antibiotics (preferably chlorchinolon, cephalosporins of III generation) in the complex of treatment.

Duration of hospital treatment is 5–14 days.

Requirements to treatment results: control of the pain syndrome, absence of changes in blood indices showing presence of the inflammatory process, normalization of the body temperature, elimination of cholekinesis disorders and active inflammation of the biliary tract.

Patients with destructive pancreatitis should be given conservative therapy, surgery is indicated only in development of purulent complications.

Conservative treatment of destructive pancreatitis:

I. In I and II periods (the period of hemodynamic disorders and pancreatogenic shock and the period of polyorganic insufficiency)

Basic therapy should be started immediately after establishment of the diagnosis of "severe pancreatitis" (within 2 hours of hospitalization):

1. Fasting and intake of alkaline solutions for 3 days and longer.

2. Decompression of the stomach with the probe with periodic aspiration of the gastric contents.

3. Suppression of the external secretory activity of the pancreas for correction of release of the pancreatic enzymes in blood and adjacent cavities and tissues is one of the main components of intensive treatment of patients with acute disease and damage of this organ. Suppression of the external secretory activity may be achieved by several ways:

— introduction of antienzymes in blood or locally, first of all antiproteases (contrical, aprotinin, trasilol, gordox, pantripin, 2-macroglobulin);

— chemical inhibition of pathological hyperactivity of pancretocytes (cytostatics-antimetabolites: 5-fluorouracil, flourafur, ribonuclease, etc.);

— application of regulatory peptides (somatostatin, calcitonin);

— physical methods with application of intragastric or external (mostly zonal) therapeutic hypothermy.

At present there is a method of choice consisting in application of blockers of the pancreas secretion of the regulatory peptide group, sometimes artificial pancreatic hypothermy is made along with them. Reserve methods (when regulatory peptides and hypothermy are ineffective) are cytostatic drugs. Nowadays inhibitors of proteolytic enzymes of the antiprotease group are used only in treatment of DIC-syndrome (it often develops in acute pancreatitis).

Blockers of the pancreas secretion:

a) somatostatin (sandostatin R (NOVARTIS) 0.1 mg 3 times a day subcutaneously for 5–7 days;

b) calcitonin (myacalcic R (NOVARTIS) 300 IU/24 hr i/v for 6 days;

c) 5-lay-encephalin (dalargin) 10 mg i/v dropwise, then 4 mg 3 times a day/i.m.

Blockers of the pancreas secretion should be administered only in the first 5–7 days of the disease, and drugs of somastatin — in development of arrosive bleedings and fistula (pancreatic, intestinal).

4. Cytokine blockade (pentoxiphilin 20 ml a day i/v dropwise for 5–7 days).

5. Antioxidants:

— ascorbinic acid — 5% solution 10–20 ml a day for 5 days.

— α-tocoferol 2 ml a day for 5 days.

— ceruloplasmin 100 mg a day for 5–7 days (as a stimulator of haemopoiesis may be used in anemia in patients with purulent-necrotic complications).

6. Analgesics for controling pain syndrome: nonnarcotic (baralgin, ketanov), narcotic (except morphine, which causes spasm of the Oddi's sphincter and may aggravate the patient's state), peridural anesthesia or other types of blockade with local anesthetics (paranephral blockade, blockade of the round hepatic ligament, sacrospinal blockade, intracutaneous blockade of the abdominal plexus by Capisse).

7. Spasmolytics for facilitation of pancreas secretion outflow: myotropic (no-spa, papaverin, buscopan, galidor), M-cholinoblockers (atropin, platiphilin, gastroceptin), the latter also reduce secretion of the pancreatic juice.

8. Stimulators of the intestinal motility — fight with intestinal paresis: ubretid 0.5 mg, repeated introduction not earlier than in 24 hours; benzohexamenthonium 2.5% 0.5 ml twice a day; peridural anesthesia.

9. Infusion therapy under the control of laboratory indices (the volume depends on the clinical situation; colloids are more preferable than isotonic solutions of crystalloids). The aim is to fight with hypovolemia, electrolyte disorders and acid-alkaline disbalance, disintoxication, sometimes — parenteral feeding. At the initial stages of the disease when there are signs of massive early dehydration, the infusion therapy should be intensive, with introduction of up to 65–100 ml of solution per 1 kg of the body weight per day, the ratio of colloids and crystalloids should be 1:1. In reduction of the dehydration degree, the volume of the introduced liquid is decreased and the amount of colloids in it increases. Correction of the acid-alkaline balance is required. For detoxication, the infusion therapy should contain such drugs as neohaemodesis, gelatinol as well as a method of forced diuresis. For parenteral feeding the patient is an intravenously introduced solution of aminoacids — aminosol or alvesin by 500 ml dropwise from the 2nd day of fasting.

10. Physical methods of detoxication. In emergency pancreotology there are widely used plasmopheresis, haemo- and plasmosorption, external introduction of lymph and lymphosorption, peritoneal irrigation and peritoneal dialysis. Therapeutic methods that replace and model the processes of biotransformation of endogenic toxins are less developed and introduced into the clinical practice of management of patients with acute pancreatitis. They may include isolated homo- and xenoorgans (the liver, the kidneys, the spleen). Therapeutic methods of active detoxication also include methods aimed at direct change of the internal medium by the so-called hemocorrection. Hemocorrection is achieved at the expense of oxigenation, magnetic treatment or irradiation and photomodification of blood that are made both extracorporally, after blood taking and intracorporally, by the intravascular way.

11. Treatment of the syndrome of disseminated intravascular coagulation (DIC-syndrome) — heparinotherapy (10–20 thousand U per day subcutaneously or i/v), inhibitors of proteases i/v (see above) in combination with rheopolyglucine, nicotinic acid, euphyllin (10 ml, 2.4% solution i/v slowly in 10–20 ml of the isotonic solution).

12. Antibioticotherapy in acute pancreatitis is aimed at prevention of development of purulent-septic complications:

— carbopenems (tienam 500 mg 3–4 times a day i/v dropwise; meronem 500–1,000 mg i/v dropwise once a day).

— fluochinols (ciprofloxacin 500–750 mg twice a day, ofloxacin (Tarivid) 400–800 mg twice a day i/v dropwise, pefloxacin (abactal) 400 mg twice a day i/v dropwise;

— cephalosporins of II–IV generation — cephtriaxon, cephobid, cephotaxim, cephtisoxim;

— synthetic penicillins — piperacillin, meslocillin.

All antibiotics (except carbopenems) should be combined with antianaerobic drugs that are introduced parenterally (metragil, trichopol).

The course of antibioticotherapy of not less than 14 days is always complemented by antimycotic therapy (ketoconasol (nisoral) 200 mg per day, fluconasol (diflucan) 50–400 mg per day.

13. Prevention of erosive-ulcerous affections of GIT, as well as fight with oxidation of the duodenal contents which is a stimulator of the pancreatic secretion (H_2-blockers, antacids, inhibitors of H^+, sucralfate).

14. Prevention of translocation of the intestinal microflora (i. e. barrier dysfunction of the intestinal wall):

— detoxication (intraintestinal lavage, enterosorption);

— correction of metabolic disorders and restoration of the barrier function of the intestine (glutamin, arginin, antioxidants);

— immunocorrection (ω–3, ω–6, polyunsaturated fatty acids "Tecom", arginin, glutamin);

— enteral feeding as early as possible (insertion of the probe in the initial section of the small intestine intraoperatively (when the patient has been operated on) or endoscopically;

— selective decontamination of the intestine (polymyxin, norfloxacin, amfotericin B).

15. Correction of hyperlipidimia (lipostabil 10–20 ml a day i/v).

16. Hepatoprotectors: essentiale, carsil, legalon (the former is not administered in cholestasis, the latter two ones — in chronic active hepatitis).

In presence of affection of target-organs (respiratory-distress syndrome, acute renal failure, hepatic insufficiency therapy is polysyndromic).

II. Treatment in the III period (dystrophic and purulent-necrotic complications) in formation of the pancreatic infiltrate.

Basic therapy:

— antibioticotherapy (antibiotics of choice are given above);

— antiacidic therapy: antacids, sucralfate, H_2-blockers;

— immunomodulating therapy (sodium nucleinate, timalin, T-activin, levamisol);

— analgesics, spasmolytics, antiemetics;

— infusion therapy (if necessary);

— parenteral feeding (if necessary).

III. Treatment in the III period (dystrophic and purulent-necrotic complications) in presence of purulent-necrotic complications.

1. Surgical treatment.

2. Conservative therapy (in the postoperative period):

a) antibioticotherapy;

b) immunomodulating therapy;

c) hyperalimentation — intensive feeding (enteral in combination with parenteral if necessary).

Surgical treatment. There are different methods of surgical treatment of acute pancreatitis. Most surgeons do not support the necessity of gland resection and pancreatectomy in pancreonecrosis, motivating the reasons for refusal to perform "radical" surgery by difficulties of the intraoperative diagnosis of pancreonecrosis spreading, difficult surgical technique, impossibility to prevent necrosis of the remaining part of the pancreas. High postoperative mortality, threat of the endocrine and exocrine pancreatic insufficiency after surgery does not promote wide application of such operations. Closed operations are most widely used in our country — draining of the omental bursa with following peritoneal perfusion, omentopancreatopexia — wrapping of the anterior surface of the pancreas in a strip of the greater omentum.

Omentopancreatopexia promotes separation of the process, improves blood supply to the pancreas, and accelerates organization and incapsulation of the necrotic areas. Omentopancreatopexia is indicated in fine and single macrofocal forms of fatty pancreonecrosis.

In pancreonecrosis complicated by pancreatogenic peritonitis omentopancreatopexia is complemented by insertion of microirrigators and drainages in the abdominal cavity for introduction of intraabdominal infusion of isotonic solutions with antienzymic drugs, antibiotics and simultaneous active evacualtion of the peritoneal exudate and excess of the infused solution in the postoperative period.

In pancreonecrosis, especially haemorrhagic one, the exudate that contains enzymes and decomposition products easily penetrates into the cellular tissue of the retroperitoneal space, mesenteries of the large and the small intestine, sometimes reaching the small pelvis. Draining of the abdominal cavity, omentopancreatopexia are aimed at prevention of peritonitis development, do not provide dissociation of the process on the posterior surface of the gland, and do not stop the process of infiltration and dissolution of the retroperitoneal space. The operation of "abdominization of the pancreas" is indicated in such cases. The principle of the operation is in conduction of the omentum on the pedicle behind the mobilized gland, it prevents entrance of the exudate into the retroperitoneal space.

The operation is indicated in fatty pancreonecrosis and mixed forms when there is no sequestration and dissolution of the gland tissue.

To prevent development of purulent-septic complications in large and total necroses early radical operations are most promising: sequestrectomy, necrectomy, gland resection, and pancreatectomy. The last two operations should be performed in patients in whom complex treatment proves to be ineffective and the necrotic process progresses rapidly.

Sequestrectomy, excision of the necrotized part of the gland within dead tissues, may be performed by the dull way, more frequently by digitoclasia with the following draining of the necrotic area.

Necrectomy is excision of the necrotized part of the gland limited by the blood supply of the tissues. The necrotized area is separated by the acute way: gland tissues are dissected along the necrosis line, the bleeding vessels are ligated. The gland area is draining.

Resection of the pancreas is excision of the organ with its transversal transection within the unchanged tissues. Corporocaudal resection of the gland is usually combined with excision of the spleen as thrombosis of the splenic vein occurs in pancreonecrosis. On the other hand, preservation of the spleen complicates the operation and leads to considerable blood loss in separation of its vascular pedicle from the gland tissue.

Usually the necrotized gland is easily mobilized by the dull way. The operation is bloodless as all fine vessels supplying the gland are thrombozed. Treatment of the gland stump is made by different ways: both manual and mechanical suturing is used. Closure of the suturing line on the gland stump may be achieved by using medical adhesive. Canulation and external draining of the pancreatic duct is used only in obstruction of the distal part of the duct confirmed by intraoperative pancreatography.

Resection of the gland should be performed carefully as there may be thrombi in the splenic vein that may be pushed into the portal vein. Embolectomy is indicated in such cases.

Special attention is paid to draining of the pancreatic bed and parts of the abdominal cavity after radical surgery as results of the operation depend on reliable evacuation of the exudate and possibility of constant irrigation of the operated zone.

Radical and closed opeartions in pancreonecrosis are usually completed by decompression of the bile-excreting ducts (cholecystostomy, external draining of the common bile duct).

In development of purulent complications, treatment is surgical; they are apostematous purulent pancreatitis, abscesses of the gland, abscesses of the lesser omentum, retroperitoneal phlegmon. The operation consists in opening of the abscess, removal of its contents and draining of the cavity with drainages to provide adequate outflow of the contents and possibility of the cavity irrigation with solutions of antiseptics.

In abscess of the lesser omentum its opening and draining is accomplished through the upper midline incision. Opening and draining is made through the oblique incision in the transversal area on the right in the abscess in the region of the head with spreading of phlegmon along the right paracolic space. When the abscess is located in region of the gland tail and

body and spreads to the left paracolic space, it is approached through the oblique incision in the transversal area on the left. In cases of bilateral spread of phlegmon, the incisions are made on either side. After removal of the exudate, sequestra of the cellular tissue and gland the cavity is drained by two-three drainages, one of them is intended for introduction of solutions, the other — for evacuation.

Even timely opening of phlegmon has frequently unfavourable outcome. Patients die from arrosive bleeding and fistula of the cavernous organs.

Indications for early surgical treatment of patients with destructive pancreatitis:

1. Progressive course of biliary pancreatitis (the operation is performed within 24 hours).

2. Acute traumatic pancreatitis (in "fresh" rupture of the gland — resection of the distal part of the gland, in developed pancreonecrosis — pancreaticostomy with the following pancreatointestinal anastomosis or occlusion of the part of the organ).

3. Progressive polyorganic insufficiency that does not respond to intensive therapy during 48–72 hours.

Enzymatic peritonitis is not indication for surgery, evaculation of the liqud should be achieved by laparoscopy or laparocentesis with the following draining of the abdominal cavity.

Development of purulent complications (infected necrosis, abscess of the pancreas) is indication for surgery. The operation is performed on the 15th–21st day from the onset of the disease.

In pancreatic abscess (pus with minimal amount of necroses), the following is made:

1. Opening of the abscess with closed draining.

2. Different kinds of endoscopic and noninvasive draining operations.

The following is made in infected necroses:

1. Pancreatonecrsequestrectomy with closed draining.

2. Pancreatonecrsequestrectomy with prolonged irrigation.

3. Pancreatonecrsequestrectomy with laparostomy.

4. Programmed relaparotomy.

5. Different kinds of resection of the gland (very rare).

If necessary these interventions are accompanied by surgery on the bile-excreting ducts and splenectomy.

Peculiarities of treatment of ***biliary pancreatitis (necrotic form):***

1. Basic therapy (see above).

2. Indications to ERGPCG + endoscopic papillosphingotomy:

— wedged stone of the distal part of the choledoch;

— expressed cholestasis;

— more than 4 Ranson's criteria.

3. Early cholecystectomy does not give any advantages, the operation should be performed as soon as the symptoms of acute pancreatitis subside.

Duration of hospital treatment is individual depending on the character of the disease course, presence of complications.

Requirements to treatment results: satisfactory general state, absence of changes in blood indices showing presence of the active inflammatory process, normalization of the indices that characterize function of the liver, the kidneys, external breathing, possible formation of the cyst, presence of the infiltrate at the stage of dissolution in absence of the signs of active inflammatory process.

Lately results of treatment of acute pancreatitis have considerably improved. In edematous forms of acute pancreatitis the outcome and prognosis is favourable. Lethality in pancreonecrosis ranges from 10 to 15% mainly at the expense of pyo-septic complications. Thus, application of modern methods of conservative and surgical treatment yielded good results in fight with such severe disease as acute pancreatitis, especially its destructive form.

Lecture 8
PERITONITIS

Peritonitis is an inflammation of the visceral and parietal peritoneum, which is accompanied by severe general symptoms of the disease, and results in serious frequently irreversible affection of vitally important organs and systems for a very short time.

There is no so ancient, more important, more difficult and fatal problem in surgery as a problem of peritonitis. Peritonitis is an evil genius of abdominal surgery. It causes a lot of sleepless nights and inexpressible anxiety in surgeons. It may develop in any complication and disease of the abdominal organs and penetrating wounds of the abdominal wall, and in case of delay or sluggishness in fight with it peritonitis may rapidly take the power over the protective forces of the organism and leads to a number of victims. Among them are a newborn or a person in the prime of life, a happy young mother or an elderly person.

Information about peritonitis has been known since ancient time. In 1st century, A. D. Soranus of Ephes substantiated the necessity of incision in the groin to evacuate pus that accumulated between the intestine and peritoneum. But vigirous struggle against peritonitis began only at the end of the last century, and before and especialy at the beginning of the 19th century the literature gave only description of the clinical picture of peritonitis, therapeutic treatment and very restricted surgical tactics reflected in works of X. X. Salomon (1840), N. I. Pirogov (1852) and many foreign surgeons.

A Russian doctor A. Shabanov was the first who made the clinical description of peritonitis in 1816 and Hancock (1848), Merten (1861), Keith (1861) and Buchanan (1871) first accomplished surgical treatment of peritonitis. 100 years ago, Wegner (1876) published his classic work, which described a great resistance of the peritoneum to infection and significance of three factors in development of peritonitis: a large area of the peritoneum, its great absorption ability and property to respond to any irritation by exudate. In Russia, the first successful operation for purulent peritonitis was performed by A. I. Schmidt in 1881.

Classification. There are many classifications of peritonitis according to its different signs. Most frequently peritonitis is divided by etiologic signs, ways of infection penetration into the abdominal cavity, character of infection, character of exudate, extension or limitation of inflammation, stage of the process development and clinical course.

Peritonites are divided into primary and secondary.

Primary peritonites are caused by primary affection of the peritonium by microorganisms that penetrate by hematogenous, lymphogenous way or involvement *(per continuitatem)* the mucous membrane of the uterine tubes. This type of peritonitis rarely occurs (about 1%).

Secondary peritonites are caused by spread of infection from the abdominal organs that have undergone inflammatory changes (the appendix, the gall bladder, etc.) or organs which were perforated (the stomach, the duodenum, the small and large intestine) or injury of the mentioned organs externally (wounds, incompetence of the intestinal suture, etc.). Secondary peritonitis occurs more often.

While making the diagnosis of peritonitis the primary cause is usually found that results in inflammation of the peritoneum (postoperative, posttraumatic, appendicular, biliary, gynecological, pancreonecrotic, after perforation of the cavernous organ, etc.).

According to the clinical course, there are acute and chronic peritonites. Acute peritonitis is the most frequent and threatening disease; this form is meant when the term "peritonitis" is used without any attributes.

According to the kind of the causative agent there are peritonites caused by:

1. Microflora of the gastrointestinal tract (GIT) — strains of the colibacillus (ischerichia), streptococci; aerobic (enterococci) and anaerobic (peptococci), gas-forming (clostridia) and nongas-forming (bacteroids), anaerobs, clebsiella, protea, bacillus pynocyaneus, etc.

2. Bacteria that are not related to the GIT: gonococci (neiseria), pneumococci, hemolytic streptococci, mycobacterium tuberculosis, etc.

There are also *aseptic* (abacterial) peritonites. They arise due to penetration of blood, chylous liquid, bile, pancreatic enzymes, meconia, and urine in rupture of the abdominal part of the bladder into the abdominal cavity. Aseptic necrosis of the inner organs is also the cause of peritonitis. All the so-called toxic-chemical forms of peritonitis become infectious during several hours due to penetration of infection from the intestinal lumen because of increased permeability of the intestinal wall during development of the peritoneal inflammation.

Depending on the character of ascites in the abdominal cavity, there are serous, fibrinous, purulent, hemorrhagic, putrid peritonites and others as well as their combinations (serous-fibrinous, fibrinous-purulent, etc.).

Classification of peritonites according to spread of affection of the peritoneal surface is of great importance for clinicians. There are peritonites:

1. Local:
   a) limited (inflammatory infiltrate, abscess);
   b) unlimited (there are restricting adhesions but the process is located only in one of the peritoneal pockets).
2. Extensive:
   a) diffuse (the peritoneum is affected but the process involves only less than two anatomic zones of the abdominal cavity);
   b) generalized (over two anatomic zones of the abdominal cavity is affected, almost the whole peritoneum);
   c) total (total inflammation of the whole serous covering of the abdominal organs and walls)*.

During the clinical development, peritonitis has several stages depending on the time, which has passed from the onset of the disease, and pathophysiological changes that took place in the patient's organism for this time. It is accepted to distinguish the following stages:

1) reactive (the first 24 h);
2) toxic (24–72 h);
3) terminal (over 72 h).

Expressiveness of each stage depends on etiologic factors that resulted in peritonitis. In peritonites caused by perforation of the organs, for example, reactive and toxic stages are shorter when the treatment was started untimely.

Local peritonitis occurs in 40% of cases (limited — 30%, unlimited — 10%); extensive — in 60% (diffuse — 30%, generalized — 30%). In the reactive stage acute pancreatitis is observed in 30%, toxic — 50%, terminal — 20%.

Anatomical and physiological data. The peritoneum is a thin serous membrane of blue-pink colour, smooth and glistening. Its parietal and visceral leaves cover all walls of the abdomen and a considerable part of organs of the abdominal cavity and pelvis. The area of the whole peritoneal surface corresponds to the skin area and makes approximately 21,000 cm^2. In men, the abdominal cavity is closed and does not contact with external environment. In women, it contacts through the uterine tubes with the uterus, vagina and external environment. It is one of the ways of infection penetration and development of pelvic and generalized peritonites in women. All fissures of the peritoneal cavity are filled with a small amount (nearly 20 ml) of the capillary layer of the serous liquid (*liquor peritoneale*). In surgery, the peritoneal cavity has been called abdominal for a long time.

*Not all authors distinguish this form (it is often included in the concept of generalized peritonitis).

It is wrong anatomically. According to R. D. Sinelnikov (1963), the abdominal cavity (*cavum abdominale*) is a peritoneal cavity (*cavum peritoneale*) and retroperitoneal space (*spatium retroperitoneale*).

The thickness of the peritoneum ranges from 0.7 to 1.1 mm.

The peritoneum is richly supplied with blood, lymph vessels and nervous elements. B. V. Ognev established that there were 75,000 capillaries in each square milimetre of the peritoneum. Capacity of the peritoneal blood vessels and abdominal organs is tremendous. In peritonites blood, congestion in the abdominal cavity become a depot for a large amount of blood sharply reducing the volume of the circulating blood and is a serious threat to hemodynamics.

The peritoneum is richly innervated with a thick net of nervous plexuses that originate from the sympathic, vagus and diaphragm nerves. The innervation peculiarity is development of severe shock in perforative peritonites and pain irradiation. It should be taken into consideration in manipulations in the abdominal cavity and switch off the reflexogenic zones by the novocaine solution.

The peritonium has great absorptive and exudative properties revealed by Wegner 120 years ago. About 70 l of liquid (in the norm) are released and absorbed in the peritoneal cavity for 24 hours. The peritoneum of the diaphragm and the large intestine has the biggest absorptive property and the small intestine peritoneum has biggest ability for transsudation.

A pronounced ability of the peritoneum to resorb the contents of the peritoneal cavity is of great importance in pathogenesis of peritonitis. The least resorptive abilities are characteristic of the pelvic peritoneum. It probably explains more favourable course of pelvioperitonitis.

Exudative ability of the peritoneum plays a significant role in pathogenesis of peritonitis as well as its ability to react to any damage to discharge of excessive amount of the peritoneal liquid (rich in leucocytes and fibrin) having a bactericidal effect.

Great plastic abilities of the peritoneum also influence development of the inflammatory process and are quite variable. Deposits of fibrin in response to damage, formation of adhesions and fusions may rapidly separate the pathologic focus and stop progress of the process, they are characteristic features of the peritoneum. The greater omentum plays an indispensible role in accomplishment of this protective reaction. It is clear that its size, form, anatomical, and functional value are of great importance in organization of protection against damage. All properties explain great resistance of the peritoneum.

Etiology. Inflammation of the peritoneum is most often conditioned by infection factor. The main source of infection in peritonitis is microorga-

nisms that are present in the lumen of the cavernous organs and oral cavity. The number of bacteria in 1 ml of the intestinal contents ranges from 10^6 to 10^{12}. Flora changes depending on the age, feeding conditions and hygienic conditions of life. The most numerous and pathogenic flora is of the large intestine.

Peritonitis is based on any disturbances of the integrity or permeability of the wall of the cavernous organs, inflammation or rupture of organs of the abdominal cavity and retroperitoneal space, ruptures of cysts and diverticuli, injury of the abdominal cavity and closed damage of them.

The most frequent cause of peritonitis is appendicitis (perforative, gangrenous, phlegmonous). Peritonitis of the appendicular origin takes the first place among all peritonites. Diseases of the stomach and duodenum (perforative ulcers, cancer, wound, diverticulitis, tuberculosis, syphilis, etc.) take the second place among the causes of peritonitis. The causes of peritonitis may also be:

1) diseases of the gall-bladder (gangrenous, perforative, phlegmonous cholecystitis, peritonitis of the gall-bladder without its perforation) and pancreas (acute pancreatitis, cysts, injuries, pancreonecrosis, abscesses);

2) pathological processes in the small and large intestine (diverticuli — their inflammation, ulcer perforation — simple, abdominal typhoid, tuberculous, syphilitic, tumour perforation, diverticulum rupture, disturbed blood supply due to strangulation or occlusion of the mesenteric vessels, foreign body, wounds);

3) diseases of the genitalia in women: salpingites of different etiology (purulent, tuberculous, gonorrheal), inflammation and cysts of the ovaries, abdominal pregnancy (tubal abortion, rupture of the tube), uterus perforation during abortion, endometritis and parametritis;

4) pathological processes in the liver, spleen (injuries, ruptures, cysts that became suppurative, parasitic and nonparasitic, rupture of the superficially located fine intrahepatic bile ducts);

5) rupture of the part of the bladder covered by the peritoneum, its injury is also a source of peritonitis;

6) damage of the starting part of the pectoral lymph duct is accompanied by development of chyloperitoneum followed by inflammation of the peritoneum;

7) transition of the inflammatory process from abscesses located retroperitoneally (paranephritis, diseases and damage of the kidneys, pelvic cellular tissue) or in the anterior abdominal wall.

Postoperative peritonites develop due to inadequacy of the anastomosis suture, intraoperative abdominal infection, ligation of the large areas of the omentum and mesentery with subsequent necrosis of the tissue distal to the

ligature. Mechanical damage of the peritoneum and its dryness, hemorrhage into the free abdominal cavity in insufficiently thorough hemostasis creates conditions for development of peritonitis.

Hemoglobin, which got into the abdominal cavity, is of importance in genesis of peritonitis as hemoglobin produces highly toxic products in the process of disintegration that enhance bacterium aggressiveness. Hemoglobin slows down natural cleansing of the abdominal cavity from microbes as if protecting them from influence of natural protective mechanisms of the organism (phagocytosis, antibody influence, etc.). It is shown in experiments on dogs and rats that introduction of clean culture of colibacillus into the abdominal cavity, as a suspension does not bring about lethal peritonitis. All animals survive. When 30–40 g/l hemoglobin is added to this dose of microbic bodies, 90% of animals develop lethal peritonitis. The mechanism of increasing microbe virulence in presence of hemoglobin is insufficiently studied. It is extremely important as hemoglobin is frequently revealed in the effusion in peritonitis.

Irritation of the peritoneum with chemical agents that suddenly penetrated into the peritoneal cavity in large quantity (urine, gastric juice in perforation of the gastric wall, bile, blood, chylous liquid from large lymphatic collectors) causes aseptic toxical-chemical peritonitis that rapidly becomes infectious at the expense of increased permeability of the intestinal wall.

The most frequent causative agents of peritonitis are colibacillus (58.0–65.2%), staphylo- and enterococci (11.3–12.5% correspondingly), protea (8.2%), streptococci (7.1%), blue pus bacillus (bacillus pynocyaneus) (4.3%), nonclostridial anaerobes (2.7–1.7%). Combination of two-three causative agents is observed in over 30% of cases. Only lately more attention has been drawn to anaerobic causative agents of peritonitis, namely nonclostridial anaerobes of the bacteroid group. They are gram-negative rod-shaped bacteria with rounded edges that do not produce spores; they are conditionally pathogenic and vegetate in the lower section of the intestinal tract. It is difficult to detect them by ordinary technique of flora isolation from the exudate as they are strict anaerobes, grow slowly (over 48 h) despite adequate anaerobic conditions in the apparatus and special culture. The bacterium cultures are not kept in many laboratories after 48 h therefore the slowly growing flora is not revealed. Bacteroids are stable to penicillin and streptomycin, highly susceptible to derivatives of metronidasole (trichopol, flagil, facigin, etc.) as well as to lincomycin.

Pathogenesis. Despite numerous works dealing with clearing-up the pathogenesis of acute peritonitis in modern surgery, there is no clear notion about mechanisms of the pathological reactions in inflammation

of the peritoneum. Influence of microbes and their toxins, chemical irritative substances, mechanical and thermal factors (trauma, dryness of the serous membranes during surgical intervention) on the peritoneum results in disturbed integrity of the serous covering, damage and desquamation of the mesothelium cells, irritation of the large receptor of the peritoneum nervous apparatus. In response to this influence the peritoneum reacts by inflammation, hyperemia in the area of affection, increased permeability of the vessels, edema in the peritoneal thickness, discharge of the exudate and fibrin in the free abdominal cavity, fibrinous deposits on the peritoneum. Absorption from the abdominal cavity is somewhat accelerated at the beginning of inflammation, it allows the microbes and toxins get into the blood flow already in the early period. Later on, as edema and disorder of the capillary blood circulation, absorption ability of the peritoneum sharply decreases. Along with increased exudation, it leads to accumulation of the exudate in the abdominal cavity.

There was firmly established presence of the inflammatory focus in the abdominal cavity, paresis of the intestine and intoxication at the expense of bacterial exo- and endotoxins, products of disturbed metabolism in the organism, toxic compounds of the intermediate metabolism (aldehydes, acetone, isopropanol, ethanol, etc.), death of leucocytes and release of abundant lisosomal proteases, death of smooth cells with release of histamine and serotonine in peritonitis. The bacterial toxins influence the myocardium, precapillaries of the cardiovascular system that results in insufficient oxigenation of the tissues, accumulation of suboxidated products — polyptides, similar to indol-containing amines and lactic acid that cause tissue acidosis. Opening of arteriovenous (precapillary) shunts, reduction of the blood flow, peripheral resistance in the intestine and abdominal organs also result in massive liquid diffusion from the vascular flow into the abdomen and exhaustion of the functional reserves of the cardiovascular system. Energy consumption increases, plasma protein is lost and hypovolemia develops. Acute renal failure develops. The desamine role of the liver is disturbed; ammonium is accumulated with metabolic alkalosis. Due to loss of sodium and potassium, the mineral metabolism is disturbed. There are considerable changes of acid-base balance — subcompensated forms of acidosis and alkalosis in the reactive stage, 2/3 of patients are observed to have subcompensated alkalosis in the toxic stage and in 1/3 — there is subcompensated acidosis and decompensated metabolic acidosis or alkalosis in the terminal stage. The immunity is reduced. Development of polyorganic insufficiency is the main cause of lethal outcome in acute peritonitis.

On the whole, taking into account the stage development of peritonitis the following phases of changes in the organism can be roughly distinguished:

1. The reaction to the local inflammatory process that is composed of local and general mechanisms of protection against the aggression and nonspecific response of the system — hypophysis — adrenal cortex to the stress effect. These changes are characteristic of inflammation of any localization; they are characteristic of early reactive stage of peritonitis and diseases of the abdominal organs that precede it.

2. Response to toxins in the general blood flow (bacterial exo- and endotoxins; enzymes produced by bacteria; products of protein origin formed during cell disintegration — lysosome enzymes, proteases, polypeptides; toxic substances accumulated in the organism due to metabolic disorders in the tissues, etc.), that is characterized by signs of endotoxic shock in some cases. All this corresponds mainly to the toxic stage of peritonitis.

3. Response of an organism to a complex of factors characteristic of the terminal stage of peritonitis which has a course with signs of septic shock and polyorganic insufficiency.

Clinical course is determined by the source of peritonitis (primary disease), time of the process development (stage), degree of the process spreading as well as intoxication, paresis of the intestine and presence of polyorganic disturbances. Symptoms of the extensive form of peritonitis are most typical as well as clinical and laboratory indices. Peritonitis is characterized by a number of symptoms, a part of them are found constantly, some are rare.

Permanent symptoms of peritonitis (by V. Ya. Shlapobersky):

— pain in the abdomen;
— tenderness on palpation of the abdomen;
— Shchetkin — Blumberg's symptom;
— muscular tension of the anterior abdominal wall;
— tachycardia;
— decreased arterial pressure;
— changed character of respiration (accelerated, costal type of respiration, absence of respiratory movements of the abdomen);
— elevated temperature, the temperature is behind the pulse;
— dry and furred tongue;
— nausea, vomiting, thirst;
— abdominal distension (develops a little later);
— paresis of the intestine, retention of gases and stool;
— tenderness on rectal examination;
— leukocytosis in the blood, left shift of leukocytes;

— anemia, increased ESR;
— changes in the urine (protein, indican, cylinders);
— Hippocrates' face (during later stages of the disease).

Nonpermanent symptoms of peritonitis (by V. Ya. Shlapobersky):

— chill;
— exudate in the abdominal cavity (is not always found); diarrhea (in 3% of cases) in pneumococcal puerperal and septic peritonites;
— gas in the abdominal cavity;
— signs of disuria.

Not all enumerated symptoms are of the same value. The principal symptoms among them are muscular defense (*defence musculare*), pain, and Shchetkin — Blumberg's symptom and inability of abdominal breathing with participation of the abdominal wall.

The leading symptoms in the reactive stage (24 h) are sharp constant pains that are enhanced during change of the body position, cough and movements. Usually the patient is in the supine position or on the sick side with legs bent to the abdomen, spares his abdomen in breathing, cough, and avoids unnecessary movements because they increase pain. There may be irradiation of the pain in the back, in the supraclavicular area in localization of the inflammatory process in the upper part of the abdominal cavity.

As a rule vomiting with gastric content does not give relief, it may be non-permanent or infrequent. The pulse is a little accelerated (90–100 bpm), the arterial pressure is normal or decreased. In some diseases that promote peritonitis there may be shock (perforation of the cavernous organs, injury, thrombosis and embolism of the mesenteric vessels, strangulated intestinal obstruction, acute pancreonecrosis, etc.).

In localization of the inflammatory focus in the small pelvis there may be false urges to defecation, disuric symptoms, irradiation of pain into the sacral area, perineum. There is no defecation but sometimes it may be scarce, there may be tenesmus due to irritation of the large intestine.

One of the important symptoms of peritonits is often detected on objective examination of the abdomen — absence of abdominal breathing. Many experienced surgeons begin patient's examination by examination of the abdomen and ask the patient "to breathe with the abdomen". Absence of abdominal respiration or lagging of one or another part of the abdominal wall during breathing is often evidence of localization of the pathological process in this area. Sometimes asymmetry of the abdomen is revealed on examination due to local paresis and bloating. To avoid mistakes it is expedient to bare the whole abdomen (up to the nipples upwards and to the middle of the thighs downwards), it allows to examine all parts of the abdo-

men in detail. It is necessary to examine those sites of the abdominal wall where hernial gate and strangulated hernias are localized.

On superficial palpation, there may be determined defensive tension of the muscles (defence musculare) according to the zone of the parietal peritoneum of the anterior abdominal wall affected by the inflammatory process.

The muscular defense is the most probable sign of peritoneal irritation. All authors who have studied pathology of the abdominal organs indicate value of this symptom. G. Mondor wrote that when a young doctor got acquainted with the symptom of contracted abdominal musculature for the first time he obtained an imcomparable working instrument. He saves people from lethal danger with the help of this instrument.

This sign is one of the best clinical methods. We can hardly find more correct, more precise, more useful and saving index.

Expressiveness of the muscular tension may be different and depends not only on character of the pathological process but also on patient's age, character of his reactivity and degree of muscular development of the abdominal cavity. The sharpest tension (wooden belly) is observed in sudden perforation of the cavernous organ namely in perforation of gastric or duodenal ulcer. Defense tension is absent in localization of the process in the small pelvis, in the omental bursa when the parietal peritoneum of the anterior abdominal wall is not involved in the process. It may be absent in old and cachectic patients, in presence of *tabes dorsalis*, in alcohol intoxication and shock, in patients with typhoid fever, when visceromotor reflexes are inhibited or absent. In single cases, muscular defense without peritonitis may be observed in traumas of the spine with damage of the corresponding endings of the spinal nerves as well as in some forms of myocardial infarction (abdominal form).

Thus, the muscular defense is a sign of all abdominal disaster; its presence should exclude any doubts. It is useless to indicate normal temperature, a good patient's pulse, and rare episodes of vomiting. As H. Mondor considered it would result in waste of time so appropriate for surgical intervention.

Pain is an important symptom of peritonitis (except for very weak and cachectic elderly patients). Intensity and character of pain is different and depends on character and place of localization of the source of peritonitis in the first hours. In perforative ulcer the pain is burning, severe and very acute. The patient in rupture of the intestine or acute pancreatitis feels approximately the same pains when not infrequently the patients develop pain shock. In acute appendicitis, cholecystitis, pyosalpinx the intensity of pain is considerably lower, though its persistence makes us think about severe inflammatory process in the abdominal cavity. The character of pain

depends largely on the patient's age and peculiarities of his nervous system. Some patients are calm even in severe pains; others show motion restlessness, cry and ask for help in insignificant pains. The examining doctor should take all of these into consideration but in all cases intense pains are evidence of intraabdominal disaster.

Shchetkin — Blumberg's symptom is also an important sign of peritonitis, which is evidence of the irritated peritoneum. The authors of this symptom interpreted the mechanism and its importance in different ways. Blumberg suggested to consider this sign as a symptoms that was evidence of irritation of the peritoneum of any origin including inflammatory one in peritonitis. Shchetkin considered this symptom to be characteristic of generalized purulent peritonitis. In the course of time this symptom began to be used widely both for diagnosis of peritonites of different etiology and for diagnosis of acute appendicitis, cholecystitis. It was found out that besides peritonitis it may be in abdominal pregnancy and ovarian apoplexy when there is outflow of blood in the abdominal cavity. Thus, combination of tension of the abdominal wall with Shchetkin — Blumberg's symptom is evidence in favour of peritonitis, and combination of this symptom with soft abdomen is a sign of gynecological diseases that are accompanied by abdominal bleeding.

Percussion may determine a zone of percussion tenderness. Usually it is most expressed in the area, which was the starting point of the inflammatory process (right iliac area in appendicitis, right subcostal area in acyte cholecystitis, etc.). A thorough percussion allows determining accumulation of the liquid in the free abdominal cavity or presence of gas under the diaphragm and disappearance of percussion dullness over the liver. Palpation may reveal inflammatory infiltrate or abscess in localized peritonitis, displaced or enlarged organ, invagination, tumour, etc. Vaginal and rectal examination helps to diagnose peritonitis and reveal infiltrate (abscess) in the small pelvis, acccumulation of liquid, tenderness on palpation of the inflammatory peritoneum in Douglas cul-de-sac area, changes in the uterine appendages, uterus and rectum.

In the reactive stage, blood tests usually show increased amount of leukocytes, left leukocytic shift, elevated ESR. There is frequent dependence of leukocytosis and left leukocytic shift on severity of the process. Some patients have indices of hemoglobin and hematocrit within the norm. Elevation of these indices is evidence of blood clotting and hypohydratation. The amount of protein in the reactive stage of peritonitis is usually normal. However, disproteinemia begins already in short time manifested by decreased amount of albumins and increased amount of globulins. Moderate elevation of blood sugar and amilase may be

sometimes observed because of inflammation and activation of the sympathicoadrenal system. Investigation of the coagulation system of blood like in all inflammatory process usually shows signs of the started hypercoagulation.

The toxic stage of peritonitis (24–72 h) is characterized by the same symptoms. However, the local signs are a little smoothed, pain and local tenderness decreases and defense tension of the muscles is a little weakened due to exhaustion of the visceromotor reflexes. The symptom of Shchetkin — Blumberg becomes less pronouced. The intestinal peristaltic sounds disappear ("deathly" silence), pronouced distension and retention of defecation and gases develop.

General manifestations of peritonitis are enhanced because of increased intoxication. The pulse is sharply elevated (120), the arterial pressure falls down. The body temperature is high (38–39°C) and has a hectic character. The respiration is accelerated, there are rales in the lungs, oliguria, and the contents of creatinine and urea in the blood are elevated. The elevation of creatinine is especially unfavourable sign. Prognosis is unfavourable in its content of 265–442 mcmol/l. There are protein and cylinders in the urine. All these are evidence of the beginning of the renal failure. In absence of appropriate correction of disorders in an organism, marked imbalance in water-electrolyte and acid-base state develops. Dryness of the mucous membrane of the cheeks and tongue marks hypohydratation. Leucocytosis and left shift increase. In this period, the clinical picture of peritonitis is not infrequently accompanied by septic shock.

It is expedient to determine the degree of hypohydratation by laboratory methods as well as degree of blood clotting (increase of hematocrit, hemoglobin, and amount of erythrocytes). It allows making a thorough plan of volemic disorders compensation.

Weakening of the cardiac activity, functions of the lungs, liver and kidneys at this phase of the disease is closely associated with hypovolemia, disseminated intravascular coagulation, and desorganization of microcirculation. At this period of the disease, the clinical picture corresponds to septic shock.

At the terminal stage of peritonitis, the patient's state becomes more aggravated, both general and local manifestations of peritonitis increase. Not infrequently, the patients develop hemodynamic noneffective tachycardia up to 120–140 beats per min; the arterial pressure is reduced, as a rule. Respiratory dyspnea up to 30–40 respirations per min develops due to progressive tissue acydosis; on ausculatation, breathing is rough, not infrequently with moist rales and weakened respiration in the lower parts with development of associated effusion in the pleural cavities. At the terminal

stage, there are signs of hepatic-renal insufficiency manifested in some lethargy of the patient (hepatargia), mild icterus and oliguria. The biochemical blood analysis reveals increased level of hepatic transpherases, bilirubin, urea, creatinine, reduced prothrombin. In severe cases of hepatic insufficiency the level of choline esterase and urea is reduced, which is evidence of irreversible destructive disorders in hepatocytes. The clinical analysis of blood may show some reduction of hemoglobin and erythrocytes due to intoxication. Not infrequently, the number of blood leucocytes is normal or even decreased along with considerable inflammatory deviation in the leucogram and presence of toxic granulosity in leucocytes. This so-called phenomenon of consumption is evidence of marginal tension of the bone marrow and system of mononuclear phagocytes and is of unfavourable prognostic value. The presence of toxic changes in the urine: protein, erythrocytes, cylinders, renal epithelium is of unfavourable prognostic value too.

Local changes in the terminal stage of peritonitis are manifested in considerable abdominal distension, reduction of its respiration excursion, rigidity of the abdominal wall and generalized tenderness of the whole abdomen with presence of marked symptom of Shchetkin-Blumberg. On percussion, dullness in the flat places of the abdomen along with tympanitis is characteristic of accumulation of the liquid. Peristalsis is not heard. Presence of painful accumulation of the liquid in the Douglas cul-de-sac may be revealed on vaginal and rectal examination.

The *diagnosis* of peritonitis is based on clinical and laboratory data, results of X-ray and ultrasound examination, endoscopy and laparoscopy.

X-ray examination of the abdominal and chest cavities is an important stage; it is made with contrast agent in the vertical and left lateral position. It allows revealing accumulation of gas in the abdominal cavity in perforation of the cavernous organs that contain gas (stomach, appendix) or its appearance in inflation of the stomach or the large intestine via the probe. This procedure is quite safe and allows detecting perforative ulcer without dynamic follow-up in severe cases difficult for diagnosis. Roentgenologic survey may reveal a paretic, gas-inflated intestine that is adjusted to the focus of inflammation — the source of peritonitis. In extensive peritonitis, the picture of paralytic intestinal obstruction with Kloiber's cups in the loops of the small and large intestine is found in the toxic and terminal phases. During lateroscopy the inflated loops shift taking the highest position. It is a generally known confirmation of dynamic nature of obstruction. In localization of peritonitis and its source in the upper part of the abdominal cavity one may reveal limited mobility of the diaphragm characteristic of inflammation of organs of this area and its high cupula on the side of affection, disc-like atelectasis of basal segments of the lung, associated pleuritis as large or small accumulation of effusion in the costodiaphragm sinusis.

In severe extensive peritonitis in the toxic and terminal stages slight spotted shadowing is sometimes determined in both lungs which is evidence of interstitial edema of the lungs (the so-called watery or shock lung). Some patients are revealed to have bronchopneumatic foci. Early manifestation of changes in the lungs is conditioned by presence of direct lymphatic connections between abdominal and chest cavities; it facilitates rapid penetration of microbes and their toxins from the abdominal cavity into the chest one.

When speaking about instrumental methods of investigation it is expedient to use laparoscopy for diagnosis of severe cases of peritonitis. It allows revealing inflammation of the peritoneum, sometimes to see the inflamed organ. There may be used laparocentesis with introduction of the searching catheter. In small volume of the accumulated liquid in the abdominal cavity, sterile solution with further aspiration may be introduced through the instrument (catheter, laparoscope). Macro- and microscopic investigation of the aspired solution allows detecting signs of pus or blood in the abdominal cavity, thus making correct diagnosis faster and helps to administer early treatment. Laparocentesis is strongly recommended in polytrauma with possible injury of the internal organs. Shock, unconsciousness (combination with cerebro-spinal injury) sharply reduces possibilitites of early diagnosis of the abdominal organ rupture and peritonitis.

Bacterial express-diagnosis of purulent peritonitis is made with the help of luminescent phase-contrast microscopy that allows identifying culture in 6–12 h after taking the exudate as well as tetrasolil-chloride test is used.

Differential diagnosis of extensive peritonitis in the toxic and terminal stage when all typical symptoms are present is not difficult. In early (reactive) stage, it is more difficult to recognize peritonitis, as its signs are identical to the main disease. Peritonitis should first of all be differentiated with all diseases in which surgery is not indicated: hepatic and renal colic, stenocardia, pneumonia and basal pleurisy, myocardial infarction (abdominal form), etc. In cases when surgery is not necessary the dynamic follow-up helps to reveal progressive signs of the disease and make a correct diagnosis.

Treatment of extensive peritonitis is given taking into consideration clinical forms and stages of the process, character of the causative agent, spreading of inflammation, degree of disturbance of metabolic processes and functions of the vitally important organs.

General principles of treatment of peritonites are as follows:

1. Early elimination of foci of infection during surgical intervention (in secondary peritonitis).

2. Suppression of infection in the abdominal cavity and beyond it with the help of antibacterial drugs, lavage and adequate drainage wuth tube drainages.

3. Elimination of paralytic intestinal obstruction by aspiration of the content through the nasogastral probe, decompression of the gastrointestinal tract, application of medicines.

4. Correction of volemic, electrolyte, protein deficiencies and acid-base state by adequate infusion therapy.

5. Restoration of function of the kidneys, liver, heart and lungs and its support at the optimal level.

Surgical treatment plays a leading role in peritonitis, indications to it is absolute when the patient's condition allows it. The operation should be performed as early as possible. Surgical treatment is not indicated in primary peritonitis, comparatively rapid improvement of the patient's condition and localization of the inflammatory process, in pelvioperitonitis conditioned by a gynecological disease as in this case conservative treatment usually results in recovery and at last in gravely ill patients who are dying, the state of which does not improve despite intensive therapy.

Surgical treatment of peritonitis can't be standardized because of variety of its causes. The principal aims of the operation are:

— removal of the exudate and infected material;

— elimination of the source of infection (surgery on the affected organ or its resection);

— decompression of the intestine;

— adequate drainage by tube drainages;

— reliable closure of the wound.

Not always, it is possible to succeed in radically removal of the primary focus of infection (for example, in postoperative peritonitis). In this case, the surgeon has to perform additional resection, when spread of peritonitis permits, forming artificial fistulas or new anastomoses, palliative drainage of the source of peritonitis is especially frequent.

In peritonitis, the operation should be urgent, under general anesthesia with a short-term preoperative preparation. The surgical approach is rather wide for excision of the source of infection and sanation of the abdominal cavity. Medial laparotomy is preferable: upper, middle line or lower.

When there is much turbid liquid in the abdominal cavity, its aspiration is a primary task of a surgeon. Usually most of the liquid is aspirated with electroaspirator but it is no use to aspirate all of it. The liquid that remain will be evacuated by big gauze napkins inserted in the abdominal cavity by three on each side: under the subcostal area, the lateral canal and in the cavity of the small pelvis. After insertion of the napkins, revision of the abdominal cavity is performed to find the focus of the destructive inflammation or in other words, the cause of peritonitis.

It is generally known that destructively changed organ, if it is not vitally important, should be resected (the gall bladder, appendix, a loop of a necrotic intestine, etc.).

After elimination of the destructive focus, wet gauze napkins should be replaced in the abdominal cavity and the surgeon begins the important stage of the operation — influence on the inflamed peritoneum. It is necessary to excise fibrous layers and toxic substances on them from the abdominal cavity without damaging underlying serous layers. K. S. Simonyan (1971) proposed the following method. The mesentery of the small intestine is infiltrated with at least 100 ml of 0.25% solution of novocaine, and then about 200 ml are introduced under the peritoneum along the whole length of the abdominal cavity. Then having removed the napkins from the abdominal cavity the surface of the peritoneum and intesinal loops are treated with soap-suds with the help of soft brush. The soap is a highly dispersed alkaline solution that easily irrigates and absorbs toxic substances from the fibrous layers with which they are slightly connected. Besides, soapsuds have a disinfective effect on microflora. The remnants of soapsuds are washed off by the isotonic solution (1.5–2 l) and the gauze napkins dry the abdominal cavity again. Then the abdominal cavity is irrigated by a great amount of antiseptic solutions (10–20 l) that contain novocaine, tripsin, limesid, heparin, fibrinolysin, hidrocortisone, levomycetin or canamycin, sodium succinate or 0.12% solution of sodium hyposulphate.

Modern physical methods during the operation are as follows: ultrasound low frequency cavitation of the abdominal cavity of 2 Wt/cm^2 for 2–7 min; ultraviolet irradiation of the abdominal cavity for 1 min; laser irradiation with outlet power of 3–5 mWt/cm^2 with exposure for 10 min; vacuumization; stream-aerosol ultrasound treatment with a mixture of solution of chlorhexidine and hydrogen peroxide (9:1), pulsating stream of antibiotics.

To prevent intestinal paresis one may use the method of permanent aspiration of the intestinal content by the probe that is introduced to the patient by the anesthesiologist through the nose into the stomach during the operation. Having felt the olive with the starting part of the probe by the hands, the surgeon leads the olive through the pylorus and pushes the probe through by 20–30 cm lower than the Treitz ligament. Active aspiration of the intestinal content is made in the postoperative period until complete restoration of the motor activity of the intestine. The rubber tube of about 2 m in length is retrograde conducted through the ileostomic window up to the Treitz ligament and loops of the intestine are strung on the end of the tube. Thus, even in complete paralysis of the intestine its evacuation is achieved thanks to the tube.

When the task of unloading the intestine is solved, one should decide whether it is necessary to drain the focus of obstruction and the whole abdominal cavity. The following variants are possible in such case:

1. Closure of the abdominal cavity tightly with drainage or without it with insertion of several microirrigators of 2–3 mm in diameter for local antibacteriotherapy in the early postoperative period. The microirrigators are inserted through the punctures of the abdominal cavity made by a special thick needle.

The closure of the abdominal cavity with microirrigators (or without them) is indicated in reliable removal of the source of infection, in the early stage of peritonitis, in serous or serous-purulent character of the exudate, after complete intraoperative lavage of the abdominal cavity. The microirrigators are used for introduction of antibiotics fractionally (2–3 times a day) or dropwise constantly using up to 500–1000 ml of the solution per day. The liquid introduced in such a way is absorbed during 24 h.

2. Draining of the abdominal cavity by one-two tube drainage of 8–10 mm in diameter for efflux of the exudate and blood. The drainages are inserted in inflammation of the retroperitoneal cellular tissue when hermetic closure of the intestinal suture is doubtful, in operations on the liver, bile ducts, and pancreas as well as for draining the lower parts of the abdominal cavity. This method of draining may be combined with fractional irrigation of the abdominal cavity. When the capillary bleeding from the retroperitoneal cellular tissue is not stopped, tampons are sometimes applied for confining the inflammatory process and in abscesses. However, their application becomes rare as the tampons promote retention of the exudate but not its evacuation.

Unsatisfactory results of treatment of extensive forms of peritonitis promoted search for new methods of theraputic effect. One of them is long-term irrigation of the abdominal cavity postoperatively. Indication to this method is severe extensive purulent peritonitis in the toxic and terminal stage of development.

There are three methods of long-term irrigation of the abdominal cavity:

1. Closed irrigation: at the end of the operation four drainage tubes are inserted in the abdominal cavity (two ones — for liquid introduction and two one — for efflux) and irrigation is constantly accomplished for 4–6 days after tight closure of the laparotomic wound.

2. Half-closed irrigation: the wound of the anterior abdominal wall is not tightly closed, only its edges are brought together; some part of the irrigation liquid penetrates into the wound through the dressing.

3. Open, the so-called ventrodorsal irrigation: the liqud from the depth of the abdominal cavity fluxes through the laparotomic wound.

Closed irrigation (synonyms: lavage, peritoneal dialysis) of the abdominal cavity became the most commonly used procedure. The drainages are inserted at the end of the operation. The most commonly used tubes are made of silicon of 8–10 mm in diameter. The drainages are inserted through the punctures in the anterior abdominal wall in its lateral parts (along the anterior inguinal lines) and thoroughly fixed to the skin. Two drainages are inserted through the puncures in the subcostal areas and fixed under the right and left diaphragm cupula. Two more drainages are inserted through the punctures in the ileac areas, placing the end of one of them in the Douglas cul-de-sac (this drainage may be inserted by posterior colpotomy in women) and another — in the left lateral canal of the abdominal cavity. Sometimes the fifth drainage is inserted for facilitation of the liquid efflux from the mesentery root area and along it. Other arrangement of drainages is possible depending on the spread of the process. One-luminal and two-luminal tubes may be used. The latter are preferable as they are less clogged with fibrin. The laparotomic wound is sutured by layers up to the skin and retension sutures are applied. The patient is placed in the Fowler's position (semisitting position with elevated head end of the bed) to facilitate the liquid efflux.

The liquid is introduced into the abdominal cavity through the upper tubes and it fluxes through the lower ones. The irrigation is started immediately when the operation is over. It is necessary to look after the amount of liquid: the amount of the liquid that fluxes should be equal to that introduced as retention of liquid in the abdominal cavity or reduced amount of the fluxed one due to its absorption through the peritoneum may cause unfavourable signs. Fractional irrigation is sometimes used instead of constant irrigation of the abdominal cavity. All drainages are used for introduction of solutions for some time and then all tubes are open for efflux. This procedure is repeated many times during a day using approximately the same amount of liquid as in the constant irrigation. In recirculatory irrigation of the wound the liquid received from the lower drainages is passed through the sorbent and then again introduced in the peritoneal cavity through the upper tubes.

The drainages are not recommended to be connected with the aspiration system as the intestinal wall may get adhered to the drainage opening and damaged.

Constant long-term irrigation of the abdominal cavity with special solution for peritoneal dialysis may be used for fight with renal failure that is developing or elimination of some toxic substances from an organism. In this case the peritoneum plays the role of dialysis membrane.

The drawbacks of the long-term irrigation method are the following: possible slowing down of surface adhesion in the area of suture and anasto-

moses; decreased defense mechanisms due to washing out of macrophages, protein, polysaccharides, etc.; threat of uncontrolled absorption of drugs and water from the solutions and manifestations of their side toxic effect; comparatively rapid cessation of flow system functioning due to confining of drainages by the omentum, the intestine, that are adhered, clots of fibrin; threat of infection penetration along the drainages. The enumerated drawbacks may be prevented as they are, to some extent, exaggerated. Lately a method of planned (programmed) repeated lavage and revision of the abdominal cavity has been used in the surgical clinics in severe and grave diffuse extensive purulent peritonitis. At the end of the operation the abdominal cavity is drained by four plastic wavy drainages (like in lavage), the intestinal loops are covered by perforated polyethylene film, which in its turn is covered by gauze liniment dressing with proteolytic enzymes. Sutures are applied to the laparotomic wound that only brings the edges together and there is a possibility to efflux. Then strictly according to the program the abdominal cavity is opened under the anesthesia daily or in a day, a thorough revision is made, all puffy adhesions are separated among the intestine and all parts are carefully irrigated. The wound is again closed by retension sutures. This procedure is repeated several (up to 5) times until the patient stop being in the critical state and the abdominal cavity is clean and free from purulent exudate. Only after that a usual suture tightly closes the laparotomic wound.

With the help of planned (programmed) lavage and revision of the abdominal cavity, doctors may prevent formation of abscesses and early mechanical intestinal obstruction due to formation of adhesions between the intestinal loops. Threat of suppuration and eventration decreases as open management of the wound allows fighting with infection better. Contraindication to application of the method is tumours of IV stage. Success of the surgical treatment depends on purposeful general treatment, the aim of which is compensation of pathophysiological disorders and fight with infection and intoxication. General treatment starts in the preoperative period, is given during the operation and is of main importance in the postoperative period. Primarily it is necessary to replace plasma deficiency by its substitutes: aminoacids, blood, solutions of electrolytes. Manitol (100 ml, 20%) is used to maintain diuresis. Diuresis should be equal to 30–50 ml/h. Besides, to restore the volume of circulating blood (VCB) it is necessary to introduce drugs for treatment of polyorganic insufficiency, give parenteral feeding and enteral feeding through the probe when peristalsis is restored, and in presence of defecation — feeding per os. In respiratory failure, the patient may need tracheostomy, artificial ventilation in synchronous regimen with moderately positive pressure at the end of expiration.

One of the most important stages of treatment of peritonitis is elimination of paralytic intestinal obstruction. Evacuation of gastric contents is accomplished with the help of a gastrointestinal probe inserted during the operation through the stomach or ileostoma to unload the intestine. First of all, treatment of paralytic intestinal obstruction should be aimed at elimination of nervous-reflex sympathicotonic block of the contractive ability of the intestinal muscular membrane. To achieve this novocaine paranephral blockade, intraoperative infiltration novocaine in the mesentery of the small intestine, epidural enesthesia, intramuscular introduction of sympathicolytics (aminasin, chlorpromasin) are made.

Introduction of aminasin is possible in stable hemodynamics, absence of VBC deficiency and mechanical nature of intestinal obstruction. The drug is of central and peripheral effect, blocks α-adrenoreceptors, facilitates blood flow (perfusion) and metablolism in the tissues. Parasympathomimetics (proserin, neostigmin, ubretid, etc.) are introduced in 30–40 min after appearance of intestinal peristalsis (intestinal sounds), that is after elimination of the sympathicotonic blockade. Enema is made for greater efficacy. Intestinal sounds appear in 20 min, flatus — in 75 min, and defecation — in 90 min after introduction of sympatholytic. In general, reduction of the arterial pressure due to introduction of aminasin does not exceed 10–20 mm Hg.

To restore and enhance peristalsis electrostimulation of the intestine may be successfully applied with a special apparatus or Bernard impulse diadynamic streams. Electrostimulation is combined with introduction of proserin and enemas. There may be also used diathermy on the solar plaxus, compresses on the abdomen, sorbitol, sorbit, xylite, pituitrin, etc.

The fight with intoxication includes: infusion therapy, forced diuresis, hemosorption made by arteriovenous or venovenous type through the carbon, metabolic plasmapheresis, lymphosorption, ultraviolet irradiation of blood, connection with xenoliver and xenospleen by the appartus with perfusion volume of 40–50 ml/min for 45 min, peritoneal dialysis, draining of the pectoral lymphatic duct, endolymphatic introduction of antibiotics, enterosorption with polyphepan or vaulen, selective endogenic detoxication with polybiotin, cryoprecipitate, blood plasma, estradiolpropionate. Vitamins and hyperbaric oxigentation are used to normalize tissue metabolism.

Antibiotic therapy should be started from the preoperative period when the antibioticogram is still unknown and bacteriologic causative agent is not established yet. The most applicable antibiotic in acute peritonitis is chloramphenicol. In predictable infections and presence of antibioticogram, aminoglycosides, cephalosporins in addition to metronidasole are more frequently used.

At the first days after the operation, it is better to combine local application of antibiotics (through irrigators, drainages) with intravenous one using

different apparatuses. In 4–5 days when the drainages or microirrigators stop functioning effectively, the drugs are introduced mainly intravenously and intramuscularly.

While introducing antibiotics during lavage it is necessary to bear in mind the possibility of their retention and do not exceed therapeutic dose, especially of aminoglycosides. There will be a necessity to change one drug for another in 6–7 days according to changes in flora and its susceptibility to antibiotics.

The immune stimulating therapy is given by stimulation of specific immunity (passive immunization by globulins, sera, introduction of leucocytic suspension) and nonspecific immunity (drug therapy with levamisole, decaris, tymalin, T-activine, sodium nucleinate).

Prognosis depends on the main disease that caused peritonitis, patient's age, character of concomitant diseases, and time from the onset of the disease before surgery, full value surgery and other factors. When the operation for acute disease of the abdominal organs is performed in the early period and with observance of all rules of prevention and treatment of arising peritonitis, the prognosis is favourable in most cases. In cases when presence of diffuse peritonitis was underestimated during the operation and the exudate was not completely evacuated and the prophylactic measures were not not taken in full value, there may be a short-term improvement after surgery, but in 2–3 days it is replaced by the picture of generalized peritonitis. A repeated operation is required in such cases — midline relaparotomy, evacuation of the exudate, and all measures necessary for treatment of peritonitis. Prognosis in the terminal stage is extremely severe and as a rule such patients die without urgent therapeutic measures.

During the process of peritonitis regression, some patients may have local abscesses in different areas of the abdominal cavity under the influence of treatment: subdiaphragmatic, subhepatic, intraintestinal, pelvic, etc.

Even nowadays peritonitis is a very important problem especially in cases when patients are brought to hospital in negluted stages or when the surgeon missed the onset of the peritoneal process. Scientific and technical progress, achievements in surgery and other medical and biological sciences do not decrease lethality in purulent peritonitis, which makes from 23 to 83% according to the data presented. Acute purulent diseases of the abdominal cavity are complicated by peritonitis in 20% with lethality in 40–50%. The words of S. I. Spasokukotsky (1926) are still significant nowadays: "Operation in peritonitis during the first hours gives recovery in up to 90%, at the first day — 50%, over 3 days — 10%".

Lecture 9
GASTRIC AND DUODENAL ULCER

Ulcer is a widespread disease all over the world. The statistic data presented in the special literature at the end of the 20th century are evidence of millions of sick people in the economically developed countries, emphasizing tendency to the increase of this disease. The prominent gastroenterologists think that ulcer is a national problem. Thus, the social price of this disease in the USA is more than 1,000 mln dollars; total money spent on treatment of patients suffering from ulcer is 480 mln krones in such a small country as Sweden.

Anatomy. The stomach is located in the epigastric area, mainly in the left subcostal region. The stomach is composed of four parts:

1. Pars cardiaca ventriculi is an area of the stomach located 5 cm lower and circumflex the gastroesophageal junction.
2. Fundus ventriculi is an area located to the left from the cardiac part and above the cardiac notch.
3. The body of the stomach is located between the pars cardiac a ventriculi and fundus on one side and pyloric antrum on the other side.
4. Pyloric antrum is a distal quarter of the stomach from the angular notch of the lesser curvature.

According to the body type, the stomach can be located:

1. Vetrically — in asthenics.
2. Horizontally — in hypersthenics.
3. Transversally — in normosthenics.

The stomach has sphincter mechanisms.

The gastroesophageal sphincter is a circular muscle, located in the inferior part of the esophagus. On swallowing, the sphincter is relaxed for the food to enter the stomach. In contraction, the sphincter prevents food from passing back to the esophagus.

The pyloric sphincter is a muscle which regulates food passage from the stomach to the duodenum and prevents duodenal contents from passing back to the stomach.

The stomach wall is composed of four layers:

1. The serous membrane is a layer of the peritoneum covering the stomach except lesser and greater curvatures along the line of fixation of the superficial ligaments (gastrocolon, gastrosplenic, gastrodiaphragmatic, gastroesophageal, diaphragmoesophageal, hepatogastric, hepatopyloric and deep gastropancreatic).

2. The muscular membrane is composed of smooth muscular cells located in 3 layers: external or ileac, middle or circumflex and profound or oblique.

3. Submucous membrane. The mucous membrane forms folds and is movable thanks to this membrane.

4. The mucous membrane is of different cellular structure according to the stomach part. There is an area of cardiac glands in the cardia and parietal-cellular area and area of the antral section in the body.

Blood supply of the stomach. The right gastric artery comes from the general hepatic artery and comes along the lesser curvature upwards. It anastomoses with the left gastric artery at the level of the middle third of the stomach body.

The left gastric artery comes from the abdominal trunk, passes in the pancreatogastric ligament near the upper edge of the pancreas and enters the stomach wall near the cardiac section.

The right gastroepiploic artery (from the system of the general hepatic artery) is located along the greater curvature at the level of the body and antral section.

The left gastroepiploic artery comes from the splenic artery along the greater curvature.

Short gastric arteries supplying the stomach fundus with blood may come from the splenic artery or left gastric one.

Venous outflow takes place both in the system of the inferior vena cava and portal vein. There are numerous anastomoses in the inferior third of the esophagus, with the help of which blood may be pushed out into the system of the inferior portal vein in portal hypertension. As a result varicosis of the vessels of these anastomoses and threat of bleeding develop.

Lymphatic outflow takes place along the rami of ventral trunk: along the left gastric artery from the upper part of the stomach; along the splenic artery from the greater curvature; along the hepatic artery from the antral section.

Stomach innervation is accomplished by parasympathetic and sympathetic nervous fibers. Parasympathetic innervation is accomplished by vagus nerves. The anterior (left) vagus nerve innervates the anterior part of the stomach and pyloric sphincter and contains motor fibers. The posterior (right) vagus nerve is located mainly on the posterior wall of the stomach

and contains secretory fibers. Sympathetic innervation is accomplished by the fibers of the abdominal plexus in combination with major abdominal nerves with formation of the gastric plexuses: the superior one along the left gastric artery, the inferior one — along the gastroduodenal artery. The pain impulse of the stomach (visceral pain) is transfered by sympathetic fibers.

The duodenum is a begining of the small intestine. It is located horseshoe shaped in the retroperitoneal area, including the head of the pancreas. The common bile duct (choledoch) runs into it as well as the main pancreatic duct (Wirsung's) through the Vater's papilla. There are the following parts in the duodenum: the superior horizontal part is located immediately after the pyloric sphincter, is of 4–5 cm long; crosses the midline at the level of T_{11}–L_1. The descending part of 7–10 cm in length is located to the right from the spine to the level of L_3. There is the major duodenal **(**Vater's**)** papilla and minor one is on its medial wall. The additional duct of the pancreas (Santorini's duct) is opened through the minor duodenal papilla. The inferior horizontal part of 10–12 cm in length crosses the midline in the horizontal direction. The ascending part of 2.5–5 cm in length, rises by one transverse vertebra upwards and gets over to the jejunum at the level of the Treitz ligament, forming the duodenojejunal flexure.

The wall of the duodenum is composed of 4 membranes: serous, muscular, submucous and mucous. The duodenum is located extraperitoneally except the primary section of the duodenojejunal flexure, located intraperitoneally.

There is the anterior wall, which has four membranes, and the posterior one located in the retroperitoneal area, which has no serous membrane. The muscular membrane is composed of two layers: internal — ileac and external — circular.

The *arterial blood* comes by the superior pancreatoduodenal artery (from the system of the common hepatic artery) and inferior pancreatoduadenal artery (from the superior mesenteric artery).

The anterior and posterior pancreatoduodenal arch in the system of the portal and superior mesenteric veins accomplishes the venous outflow.

The *lymphatic outflow* is closely related to lymphatic vessels of the pancreas and is discharged in the lymph nodes by its superior and inferior edges and in the retropancreatic lymph nodes. It flows from the anterior wall to the pylorus and from the posterior wall to the base of the superior mesenteric artery.

Innervation of the duodenum is accomplished by the sympathetic branches from the superior mesenteric plexus and by the parasympathetic fibers of the vagus nerve.

Functions of the stomach:

— Reserve function. Initially processed food gets into the stomach.

— Secretory function. Gastric juice is secreted in the stomach. The gastric juice is composed of hydrochloric acid which is produced by the parietal cells of the stomach fundus and body, pepsinogen, produced by the main cells of the fundic glands. The synthesis of antianemic Castle's factor, the formation of mucobicarbonate barrier as a result of production of mucosa and bicarbonate ions by the surface mucous cells take place in the stomach.

— Food passes to the duodenum during digestion owing to the motor function. The regulation of secretion and motor activity is characteristic of incretory function by means of synthesis of gastrointestinal hormones (gastrin, produced by G-cells of the antral part of the stomach, histamine, secreted by fatty cells of the layer of the mucous membrane of the parietocellular area).

— Absorption function includes a capacity to absorb water, alcohol and some other substances.

— The excretory function includes the secretion of toxic substances in the stomach lumen.

— The barrier function includes bactericidal action of the gastric juice and presence of mucobicarbonate barrier.

The process of digestion finishes in the duodenum and food oxidation is accomplished there; food is influenced by bile, pancreatic juice and secretion of Brunner's glands located in the proximal part of the duodenum. Synthesis of the hormones occurs here, they take part in digestion regulation (gastrin, cholecystokinin, secretin, enterogastron, bulbogastron and others).

Stimulation of hydrochloric acid secretion is accomplished by the following scheme:

1. Reflex phase: encephalon → vagus efferent innervation → parietal cells → main cells → G-cells.

2. Gastric phase: food passage into the stomach → gastrin from the G-cells of the antral section → parietal and main cells.

3. Enteral phase: food passage into the small the intestine → parietal cells of the stomach fundus.

The inhibition of secretion of hydrochloric acid is accomplished by the following scheme:

1. Passage of chyme saturated with acid into the duodenum → bulbogastron → inhibition of acid secretion.

2. Fat passage into the duodenum → enterogastron → retention of hydrochloric acid secretion and weakening of gastic peristalsis.

The control of gastric evacuation is accomplished as a reaction to osmolarity and pH by the type of feedback. The pH decrease of the antral section below three results in inhibition of gastrin production.

Stomach ulcer is a disease which is characterized by areas of destruction of the mucous membrane. The mechanism of ulcer formation both in

the stomach and duodenum is disorder of interaction of aggression factors and protection (resistance) of the mucous membrane of the gastroduodenal area, which is manifested by shifting of the first link of the given relationship and weakening of another one.

The mechanisms of gastric and duodenal ulcer despite the ample amount of common features are different. In pathogenesis of duodenal ulcer formation aggressive action of the acid-peptic factor and bacteria Helicobacter is the most important. This disease is sometimes called peptic ulcer. Gastric ulcer develops in normal and even decreased acidity of the gastric juice. Much attention is paid to infection and decrease of resistance of the mucous membrane among other causes of its development. That is why, according to the International Classification of Diseases, accepted in 1992, there are gastric ulcer and duodenal ulcer.

DUODENAL ULCER

Localization. Most of ulcers are located in the initial section of the duodenum (at the bulb). About 5% of ulcers are located postbulbarly.

Etiology and pathogenesis. The factors of ulcerogenesis are divided into non-specific (exogenic) and specific (endogenic). Non-specific (exogenic) — malnutrition, bad habits (smoking, alcoholism), stress, professional factors and life mode, remedies (corticosteroids, antibacterial drugs, digoxin, reserpin, iron and potassium preparations) and others.

Endogenic factors of ulcerogenesis:

a) hyperproduction of hydrochloric acid and pepsine;
b) *Helicobacter pylori*;
c) chronic gastritis of B type and metaplasia of the gastric epithelium into the duodenum;
d) disorder of gastroduodenal motor activity;
e) genetic susceptibility (pariecytosis, excessive gastrin release, increase of pepsinogen-I in serum, deficiency of tripsin inhibitor, deficiency of mucoproteins, disorder of IgA production, blood group O(I), Rh(+), HLA antigens B_5, B_{15}, B_{35});
f) age and sex.

The Role of *Helicobacter Pylori* in Ulcerogenesis

Helicobacter pylori plays an important role in pathogenesis of such diseases as chronic gastritis of B type, duodenal ulcer, gastric ulcer, gastric adenocarcinoma, malignant gastric lymphoma, Ménétrier's disease.

Helicobacter pylori is revealed with the help of invasive methods of diagnosis: bioptates of the mucous membrane of the antral section of the

stomach with the subsequent light microscopy, microbiological, urease investigation, polymerase reaction which determines bacterial pRNA and non-invasive — serologic ones which determine the levels of IgG and IgA in the blood and saliva; respiratory ones — tests with uria labelled with radioactive carbon and subsequent determination of breathed out air radioactivity.

Pathogenesis of ulcerogenesis in the duodenum caused by *Helicobacter pylori* is the following one. As a result of *Helocobacter pylori* colonization on the mucous membrane of the antral section of the stomach there is production of urease enzyme by bacteria which causes the medium oxidation on the surface of the mucous membrane followed by transformation of uria into ammonia (with preservation of low pH indices in the gastric lumen), stimulation of G-cells with acidic medium by the feedback mechanism. Excessive release of gastrin results in the increase of mass and activity of the parietal cells, hyperproduction of hydrochloric acid, gastric metaplasia into the duodenum, colonization of the gastric type cells by *Helicobacter pylori* bacteria with development of antral gastritis, duodenitis, duodenal ulcer.

Diagnosis. The patients complain of pain in the epigastric area, arising in 2–3 hours after meal, sometimes pain is relieved after meal but in 2–3 h develops again. Pain often develops on an empty stomach and at night.

Dyspeptic signs are characteristic features of duodenal ulcer, they are heartburn, nausea, vomiting, sensation of heaviness in the epigastric area after meal. The epigastric area is tender to palpation.

Esophagogastroduodenoscopy (EGDS) is the most reliable method of ulcer diagnosis, which must be accompanied by biopsy for revealing helicobacterial infection and possible ulcer malignization. Local treatment is possible. This method allows to diagnose ulcer complication by bleeding, make endoscopic hemostasis as well as control objectively the process of treatment.

X-ray examination of the stomach and duodenum with barium sulphate in duodenal ulcer shows circular defect of filling with smooth edges (ulcer niches), deformation of duodenal bulb, fold convergence to the ulcer, X-ray transparent cushion of the edema around the ulcer (Hampton's line).

Increase of basal secretion of the acid (BSA) more than 2 mekv/g and maximal stimulated secretion (MSS) more than 20 mekv/g, gastrin level of blood urea (norm is 50–100 pg/ml, in Zollinger — Ellison syndrome it reaches 600 pg/ml and more), pH decrease of the gastric contents — are positive symptoms of ulcer.

Conservative therapy. Conservative drugs for ulcer therapy are divided into:

I. Antisecretory drugs
  1. Anticholinergic drugs:
     a) blockers of muscarine receptors (M-cholinilytic): non-selective (atropin, platyphillin, metacyn), selective (gastrocepin), of predominantly central action (amysil);
     b) ganglioblockers (benzohexonium).
  2. Blockers of H_2-histamine receptors: cymetidin, rhanitidin, phamotidin, nizatidin, roxatidin.
  3. Blockers of proton pump: omeprasol, lansoprasol, pantoprasol (controloc), nexium.
  4. Antagonists of gastrin receptors — proglumid.
  5. Antacides — non-absorbed (maalox, megalac, phosphalugel), absorbed (sodium bicarbonate).

II. Gastrocytoprotectors
  1. Stimulators of mucin-production — synthetic prostoglandines (misoprostol, ziprostil), carbenoxol.
  2. Drugs forming protective layer — colloid bismuth subcitrate (de-nol), sucralphate, smekta.
  3. Protracted and astringent drugs — bismuth preparations (vikalin, vikair).

III. Antihelicobacterial drugs — antibiotics (amoxicillin, tetracyclin, and claritromycin), metronidasol, de-nol.

IV. Drugs stimulating reparative processes — solcoseril, methyluracil, sea-buckthorn oil.

V. Drugs influencing the motor function of the stomach and duodenum — gastrokinetics (cerukal, cizaprid), spasmolytics (papaverin, buscopan, no-spa).

VI. Drugs of the central action — eglonil, sedatives and tranquilizers.

Each drug has its point of usage.

Modern conservative therapy of duodenal ulcer provides destruction of *Helicobacter pylori* (eradication) and decrease of the secretion of the hydrochloric acid.

The following schemes of treatment are used:

1. Bicomponent therapy: controloc 40 mg in the morning + amoxicillin 500 mg 4 times a day (for 2 weeks) (eradication 50–90%) or controloc 40 mg in the morning + claritromycin 500 mg 3 times a day (for 2 weeks) (eradication 60–80%).

2. Tricomponent therapy: controloc 40 mg 2 times a day + amoxicillin 500 mg 4 times a day + metronidasol 500 mg 3 times a day (for 2 weeks) (eradication 92–95%) or controloc 2 times a day + claritromycin 500 mg

2 times a day + metronidasol 400 mg 2 times a day (for a week) (eradication 97%).

Therapy of metronidasol-resistant infection: controloc 40 mg 2 times a day + tetracycline 500 mg 4 times a day + de-nol 120 mg 4 times a day (eradication 90%).

Successful eradication decreases the recurrence rate of the ulcer from 60–80% to 5% a year and complication of peptic ulcer by bleeding.

Reinfection rate in the developed countries is 0.5–1.5% a year while in the developing countries it is up to 30% a year.

The failure of eradication and frequent recurrences is indication to the operation.

Indications to the operation: absolute — profuse bleeding, perforation, pyloroduodenal stenosis; relative — failure of conservative therapy, frequent relapses, penetration into the adjacent organs.

Types of surgery in duodenal ulcer: truncal vagotomy with drainage operation (pyloroduodenoplasty by Zhabuley, Finney, Heineke — Mikulicz, gastroenteroanastomosis) significantly suppresses gastric secretion. The motor-evacuation dysfunction developed after truncal vagotomy is compensated by extension of the gastric outlet made by pyloroduodenoplasty or gastroenteroanastomosis.

Truncal vagotomy with antrumectomy is suppression of gastric secretion by parasympathetic denervation is accompanied by excision of the part of the stomach, which contains gastrin-producing cells. It is finished with gastroduodeno- or gastroenteroanastomosis by Billroth-I or Billroth-II.

Selective proximal vagotomy is performed per se or in combination with drainage surgery. All acid and pepsin-producing cells with preservation of the hepatic branches and Latarget nerve are denervated, which allows saving the function of the pylorus and the bile ducts.

Resection of 2/3 of the stomach with subsequent gastroduodeno- or gastroenteroanastomosis by Billroth-I or Billroth-II is performed. Now it is rarely used in duodenal ulcer.

GASTRIC ULCER

Classification (by Johnson):

Type I — mediogastric ulcers, located in the stomach body.

Type II — combined gastric and duodenal ulcer

Type III — prepyloric ulcer and ulcer of the pyloric canal.

Etiology and pathogenesis. Etiologic factors which cause gastric ulcer are similar to those of duodenal ulcer. The acidopeptic factor has less im-

portance in pathogenesis of gastric ulcers (possible ulcergenesis in normal or even reduced acidity). Counteraction to the decreased protective properties of the mucous membrane develops in colonization of *Helicobacter pylori* on its surface, disorder of gastroduodenal motor activity (gastroduodenal reflux), antacid and other functions of the stomach.

The second type of gastric ulcer (by Johnson) is defined by development of ulcerous defect in the duodenum, after this retention of the acid contents occurs in the stomach in scarring of ulcer and disturbance of pyloroduodenal passage. As a result, the conditions for intensive influence of acidopeptic factor on the mucous membrane are created.

The ulcers of the third type are more similar to duodenal ulcers in their etiology, pathogenesis and clinical course.

The principles of diagnosis and conservative therapy of gastric ulcer are similar to those of duodenal ulcers. More attention is paid to increase of the protective properties of the mucous membrane by gastrocytoprotectors, drugs, stimulating regeneration and normalization of gastroduodenal motor activity.

The indications to surgery in gastric ulcer are divided into:

1. Absolute indications — perforation, profuse bleeding, malignization, stenosis of the gastric outlet.

2. Conditionally absolute indications — large callous ulcers, penetrating or multiple ulcers which recur, moderate bleeding, ulcer recurrence after conservative treatment. It is relative efficacy of complex conservative therapy during 1 year and more (a prolonged conservative therapy is dangerous because of possible development of ulcer malignization).

The main methods of surgery of gastric ulcer are resection ones. They are directed at both removal of the morphologic substrate (ulcer itself with adjacent part of the stomach) and correction of the acidopeptic factor as well as renewal of food passage.

Types of operations:

1. Resection of 2/3 of the stomach together with gastroduodenoanastomosis by Billroth-I (in presence of technical possibilities), gastroenteroanastomosis by Billroth-II, Y-like anastomosis by Roux.

2. Truncal vagotomy with antrumectomy (in localization of the ulcer in the pyloro-antral part of the stomach in II and III type of gastric ulcers by Johnson).

3. Resection of the stomach with ulcer over the portal vein allows to save the pyloric sphincter with its nervous-regulation apparatus, that minimally interferes in the natural passage of the food.

4. Subtotal resection of the stomach is used in high (subcardial) ulcers.

Perforation is a severe, life threatening complication, it continues to take the leading position in lethality in duodenal ulcer. For the last decades the

number of operations has been at the level of 7. 5–13. 0% per 100,000 persons and lethality is from 5 to 17.9%. Modern statistics of the last decade emphasizes the growth of this dangerous complication. It develops sevenfold more frequently in men than in women.

Classification (by V. S. Savelyev et al., 1976)

1. According to etiology:
   a) ulcerous;
   b) hormonal.
2. According to localization:
   a) gastric ulcer: of the lesser curvature, of the anterior wall, of the posterior wall;
   b) duodenal ulcer: of the anterior wall, of the posterior wall.
3. According to the course:
   a) perforation into the free abdominal cavity;
   b) covered perforation;
   c) atypical perforation.

Pathophysiology. Peritonitis, accompanying perforation, is one of the factors that influences both the mentioned disorders and management of surgical treatment. It is necessary to mark "peculiarities" of peritonitis in perforation. As a rule, there is transparent or turbid exudate with threads and pieces of fibrin in the abdominal cavity up to 6 h from the moment of perforation; parietal and visceral peritoneum is brightly hyperemic. In most cases, paralytic ileus is absent. The degree of bacterial contamination does not exceed 1,000–10,000 microbic bodies per 1 ml of exudate. In most cases gram-positive cocci, lactobacilli and Candida fungi represent the microbic spectre. It should be noted that in 30% of cases bacterial contamination is absent at this period. The previously mentioned allows choosing a radical method of surgical intervention.

In the later period of perforation, pus-like exudate appears in the abdominal cavity as well as fixed deposits of fibrin, paralytic ileus with increased bacterial contamination. Therefore, it is expedient to choose minimally traumatic way of intervention using one of the kinds of abdominal sanation.

Diagnosis. Perforation of ulcer develops acutely though the prodromal period can be revealed in 20% of patients — aggravation of pains, nausea, vomiting 3–4 days before. Classic triad of signs accompanies perforation: stab-like pain (95%), muscle guarding ("wooden belly") (92%), anamnesis of ulcer (80%).

On physical examination there are acute percussion and palpation pains in the abdomen, muscular tension first in the upper part and then all over

the abdomen, positive symptom of Shchetkin-Blumberg, diappearance or considerable reduction of the hepatic dullness, dullness in the right side canal and right iliac fossa (Kerven's symptom), positive phrenicus-symptom.

There are three phases in the course of the disease: shock, imagined improvement and peritonitis. The picture given above is characteristic of the phase of shock (in 5–6 h after perforation). Then the picture of shock becomes as if obliterated and imaginary improvement comes. Muscular tension of the abdominal wall decreases, on palpation moderate pain, positive symptoms of peritoneal irritation. Peristalsis is weakened, hepatic dullness is absent. On rectal examination overhang of the rectal anterior wall and tenderness can be found. Then in 6–12 h after perforation the patient's condition progressively worsens, pulse pressure subsides, the abdomen is distended, there is no peristalsis. Clinical picture of peritonitis develops.

X-ray examination of the abdominal cavity in perforation of the cavernous organ allows to reveal free gas under the diaphragm cupula in 76% of cases. In doubtful cases, it is necessary to make pneumogastrography that increases reliability of the method up to 95%.

Esophagogastroduodenoscopy allows to make diagnosis more precisely, give exact localization of ulcer and its size, and find concomitant inflammations (stenosis, presence of a combined form of ulcer with another ulcer, bleeding or its threat). Besides, the material can be obtained for morphological verification of *Helicobacter pylori.*

Laparoscopy helps to make more exact diagnosis and choose an adequate plan of treatement in obscure clinical picture, and in a number of cases it allows to be ascertained in possibility to perform one or another type of operation. It is especially necessary for patients with a so-called covered perforation of ulcer.

Laboratory studies of blood allow finding rapidly growing leucocytosis with left shift. Urianalyses in peritonitis are distinguished for appearance of blood corpuscles, protein and cylinders.

Surgical managemen. The first to form a conception of conservative management of such patients were Wangestin (1935), Bedfort — Turner (1945) and Taylor (1945) who used constant nasogastral aspiration. Today it is clear that this method is not perfect and is used forcedly — in agonal state of the patient or in absence of possibility to perform surgery.

The pioneers of surgical treatment are Mikulicz — Radecki (1892) and Hausner (1892) who sutured perforative ulcer. Until nowadays, suturing is one of the most common method of interventions that is characterized by simplicity of performing and little traumatizing effect. Laparoscopic

variant of such operation is especially attractive (Nathanson et al., 1990). An English surgeon Keetley in 1902 first performed stomach resection in perforative ulcer, which was, as a matter of fact, discovery of radical methods of treatment. The staunchest propagandists of using stomach resection were S. S. Yudin in Russia (1929) and Odelberg in Sweden (1927). Nevertheless, having reliaved the patients from further sufferings from ulcer disease, stomach resection resulted in development of a number of postoperative disorders. It made surgeons work out new, organ-saving, pathophysiologically substantiated operations. These are operations with vagotomy combined with drainage of the stomach or antrumectomy. World experience since the beginning of the 60s of the last century has shown that for the last 40 years the operations of truncal vagotomy with pyloroplasty lead to excellent immediate results (postoperative lethality is 0–1%) with low figures of relapses of stomach ulcer and insignificant figures of postoperative dysfunctions. Development of noninvasive technologies affected the performance of these interventions in perforative ulcer, though their number is not high at present.

According to pathophysiologic peculiarities of stomach ulcer, the radical operation is still antrumectomy with vagotomy or resection within hemigastrectomy.

A Conception of Urgent Surgical Treatment of Perforative Gastroduodenal Ulcers with Individual Choice of the Operation Type

Suturing of gastric or duodenal perforative ulcer remains a saving method of treatment, especially in patients with diffuse “late” peritonitis or at high degree of operative-anesthesiologic risk.

Laparoscopic suturing is indicated to young patients when a so-called silent ulcer perforates, unexpressive or favourable course of the disease is determined in anamnesis and preoperative diagnostic program gives evidence of absence of other concomitant complications of stomach ulcer.

Truncal vagotomy with excision of ulcer and pyloroplasty is a gold standard in perforative duodenal ulcer and allows eliminating other complications of the disease that accompany perforation and create conditions for prevention of recurrences. Selective proximal vagotomy (SPV) with ulcer suturing, which is technically more difficult should be used in some cases.

Laparoscopic truncal vagotomy with excision of ulcer and pyloroplasty with minilaparotomic approach should be performed first in complications diagnosed before the operation combined with perforation (com-

pensated stenosis, bleeding or its threat). The patients with long-term ulcer anamnesis and stable recurrent course are related to the same group.

Vagotomy with antrumectomy are used in perforation when there are other complications (late stage of pyloroduodenal stenosis) as well as in combined form of stomach ulcer.

Stomach resection (hemigastrectomy) is indicated in perforative gastric ulcer in patients at low operative-anesthesiologic risk.

Postoperative period. Management of patients after suturing of ulcers and operations with vagotomy should be carried out according to requirements of today's surgical gastroenterology.

After suturing of perforative ulcer that does not influence on pathogenesis of ulcer formation the patients are administered the course of antiulcerous therapy immediately after the operation is over. Taking into account modern concepts of pathogenesis of stomach ulcer the administration includes antisecretory drugs (the last generation of H_2-blockers or inhibitors of protone pump) as well as one of the schemes of antihelicobacterial therapy.

Later on, the patients who have undergone suturing of perforative ulcer are followed up by the gastroenterologist who determines the necessity of supporting pharmacotherapy of stomach ulcer with antisecretory drugs as well as indications for eradication antihelicobacterial therapy. These measures allow reducing considerably recurrences of stomach ulcer in this group of patients.

Prevention of motor-evacuation disorders of the operated stomach is necessary after organ-saving operations with vagotomy. Drugs of prokinetic group are applied from the first day. In the intraoperative diagnosis of pronounced stenosis or technical difficulties in performing pyloroplasty the probe is inserted behind the area of the Treitz ligament using it afterwards for enteral feeding. In the early postoperative period, these patients should also solve a question of necessity of supporting antisecretory therapy with their gastroenterologist and eradication antihelicobacterial therapy.

ULCEROUS GASTRODUODENAL BLEEDINGS

Morbidity rate. Importance of the problem of treatment of acute ulcerous gastroduodenal bleedings (UGDB) is first of all determined by high level of general lethality, which makes 10–24%. Despite generally accepted efficiency of modern antiulcerous methods, the number of patients with UGDB increases every year and is 90–103 per 100,000 of adult population per year. There are many people of elderly and senile age among these

patients with advanced age and concomitant pathology. The average age of patients is 54.1±2.5, and practically the third part of the patients is over 60. In men UGDB is observed 3 times more frequently than in women.

Classification. Gastric and duodenal ulcers complicated by bleeding make 42–47% of all cases of acute gastrointestinal bleedings. The so-called secondary ulcers are also often encountered, the cause of which is intake of nonsteroid antiinflammatory drugs.

Classification of bleeding sources by J. A. Forrest (1974):

F Ia — long-term stream bleeding; F Ib — long-term capillary bleeding as diffuse leakage; F IIa — large thrombosed vessel is seen; F IIb — tightly fixed clot-thrombus to the ulcer crater; F IIc — fine thrombosed vessels as stained spots; F III — absence of bleeding stigma in the ulcer crater.

According to the degree of bleeding severity, the classification of three-degree scale is most rational: mild, moderate and severe degree of bleeding taking into account the volume of the blood loss and state of the patient.

The patient's reaction to the blood loss on the one hand, is determined by the amount of bleeding, i. e. volume of the blood loss and its duration; on the other hand, by the state and reaction of the patient's main systems to the blood loss. Development of the doctrine about disseminated intravascular coagulation (DIC) as a universal mechanism of realization of massive blood loss syndrome and triggering mechanism of polyorganic insufficiency synrome became a significant factor for understanding the pathophysiologic bases of this process and thus for formation of expert infusion-transfusion therapy. It should be emphasized that hypercoagulation phase of DIC-syndrome and microcirculatory disorders that result in reduced oxygen supply and nutritional substances to the tissues develop in every patient with clinically significant UGDB. It is understandable that functional or organic disorders of the patient in the cardiovascular, respiratory, exretory systems (the so-called aging concomitant diseases) make the patint's state more severe and require appropriate correction and are taken into consideration in making a decision of surgical treatment or preparation to it.

One of the tasks of modern surgical gastroenterology is not only to ensure good and excellent immediate results but also to form conditions for faster rehabilitation of patients in the early postoperative period and prevention of ulcer recurrence.

The diagnosis should answer three main questions: what was the source of bleeding; whether bleeding is still present and its rate; what are consequences of bleeding.

Clinical course. Clinical manifestations of acute, especially massive (profuse) gastroduodenal bleedings are quite expressive and consist of general symptoms characteristic of blood loss (acute weakness, faint, loss of con-

sciousness) and manifestations characteristic of bleeding into the lumen of the gastrointestinal tract (vomiting with fresh or altered blood, melena or hematemesis).

The data of physical examination allow determining the degree of severity of bleeding and probably its source. Confusion, acute pallor of the skin, accelerated deficient pulse of weak filling, reduced arterial and pulse pressure, presence of large amount of blood and clots in the stomach, black scarce or with blood admixture rectal contents on rectal examination are signs of acute profuse bleeding.

Diagnosis. No doubt, urgent esophagogastrodudenoscopy (EGDS) is a principal method of diagnosis of the source, type and character of bleeding.

X-ray examination of the upper parts of the digestive tract as a method of urgent diagnosis of UGDB is not so important; it is mainly used after control of bleeding as a method of additional diagnosis of the state of roentgen-anatomy and motor-evacuation function of the gastrointestinal tract.

Selective angiography in UGDB is of limited use and is applied in specialized establishments with necessary equipment in cases when the diagnosis cannot be established by other methods or as a diagnostic stage of endovascular intervention aimed at embolization of the bleeding artery.

Laboratory methods of diagnosis (hemoglobin, hematocrit, deficiency of globular volume and volume of the circulatory blood, hemocoagulation, biochemical indices) give significant data about the degree of severity of bleeding and disturbances caused by it.

Treatment. The nonoperative treatment of UGDB is based on combination of methods of therapeutic endoscopy with modern methods of conservative therapy of stomach ulcer.

The methods of therapeutic endoscopy are applied for temporary and in some cases final control and prevention of bleeding. The most common methods used before are monoactive diathermocoagulation, injections of absolute ethanol and its solutions. Lately the possibilities of full scope local hemostasis have considerably widened at the expense of using videosystems, ultra-wide chanelled endoscopes, clipping methods, argoplasmatic coagulation, laser photocoagulation with AIG-laser of 1,06 mcm wave length; application of "endoforceps". The efficacy of therapeutic endoscopy in long-term methods of therapeutic endoscopy makes 95,3%.

Modern methods of drug treatment include antisecretory drugs from the group of protone pump inhibitors and blockers of H_2-histamine receptors; antihelicobacterial drugs; remedies that enhance regenerative processes and have cytoprotective effect.

They allow forming favourable conditions for cessation of destructive processes in the ulcer crater and together with endoscopic methods, help to achieve adequate hemostasis without surgical intervention in 60–65% of all cases of ulcer bleedings and the operation becomes a planned surgery. Temporary hemostasis allows winning time and preparing patients who are subject to urgent surgery. Non-operative hemostasis with the use of active dynamic endoscopy allows preventing recurrent bleeding in patients who are not subject to surgical treatment being at high operative-anesthesiological risk.

Surgical management in patients with UGDB is based on differential approach that combines active character of diagnostic and therapeutic methods with differential determination of indications to urgent surgical intervention.

Indications to urgent surgical intervention are based on impossibility to obtain reliable hemostasis with the help of non-surgical methods.

Urgent operation is performed to patients with profuse long-term bleeding; patients with massive bleeding when conservative methods including endoscopic methods proved to be ineffective as well as to patients with recurrent bleeding.

An urgent operation is indicated to patients with ulcer bleeding which can't be relaibly stopped with conservative methods and there is a high risk of bleeding recurrence. As a rule, patients of this group are performed surgery within 24–48 h from the admission — the time necessary for preparation of a patient to the operation.

A choice of the operation technique and decision to perform emergency surgery depends first on severity of the patient's state, degree of operative-anesthesiological risk, and naturally on localization and character of a bleeding ulcer.

Truncal vagotomy with pyloroplasty in combination with suturing (excision) of a bleeding ulcer and extraduodenisation (exteriorization of the ulcer crater from the intestinal lumen) in penetration is considered a method of choice in the bleeding duodenal ulcer including patients at high operative risk.

Laparoscopic truncal vagotomy and pyloroplasty in combination with excision of a bleeding ulcer from the mini-approach is indicated in the source of bleeding in the duodenal bulb.

Antrumectomy with vagotomy in duodenal ulcer is performed in patients with comparatively low risk of the operation.

Stomach resection (hemigastrectomy) is performed, as a rule, in stomach localization of the ulcer.

The postoperative period in patients who have had excision of a bleeding ulcer and organ-saving operation with vagotomy has a number of peculia-

rities and should be planned according to modern requirements for surgical gastroenterology.

After excision of the bleeding ulcer without vagotomy (this group consists, as a rule, of those at high operative-anesthesiological risk) the patients are administered a course of antiulcer therapy in the nearest postoperative period including H^+-inhibitors or the last generation of H_2-blockers as well as a complex of antihelicobacterial therapy. After discharge from the inpatient department the patients are subject to regular medical check-ups at the gastroenterologist who determines a necessity of supporting drug therapy of stomach ulcer aimed at reduction of recurrences, prevention of repeated bleedings.

The peculiarity of the postoperative period after organ-saving surgery with vagotomy is appropriate prevention of motor-evacuation disorders in the operated stomach. A thin probe for enteral feeding is inserted already at the stage of surgical intervention in diagnosis of sub- or decompensated stenosis or technical difficulties while making pyloroplasty beyond the area of the Treitz ligament. Besides, drugs of prokinetic group are given in the postoperative period. Supporting antisecretory therapy is of importance in the postoperative management as well as eradicative antihelicobacterial therapy.

ULCEROUS PYLORODUODENAL STENOSIS

Morbidity rate. Pyloroduodenal stenosis arises in 5–47% of patients who suffer from stomach ulcer. Reduced rate of stenoses requiring surgery may be associated with active introduction of new antisecretory and antihelicobacterial drugs in the clinical practice.

Classification. There are clinical, anatomical and functional criteria for division of different stages of pyloroduodenal stenosis. The stage of pyloroduodenal stenosis may be determined only in comparison with clinical, roentgenological and endoscopic data and results of gastric motor function study.

Classification of pyloroduodenal stenoses by Pantsirev (1979):

I stage — stenosis in the process of formation.

II stage — compensation.

III stage — subcompensation.

IV stage — decompensation.

Pathophysiological disturbances in an organism. Deformation and narrowing of the pyloroduodenal area develops due to cicatrization of numerous recurrences of ulcer causing one or another degree of its obstruction,

disturbed gastric evacuation. While stenosis is progressing, the stomach loses its compensatory properties, gets extended, enlarges, its peristalsis becomes weakened resulting in greater dirturbance of gastric evaculation. To compensate evacuation of the stomach its muscular membranes become hypertrophic and motor activity increases. However, such work is not limitless and gradually signs of its decompensation develop. Vomiting with gastric contents, slowing down of food evacuation from the stomach disturb feeding and vital activity of the patient. In sub- and decompensated stenosis, there is progressive metabolic alkalosis and water-electrolyte and protein imbalance.

Diagnosis. In I stage of stenosis clinical manifestations of the main disease — stomach ulcer — take place. The patient's general state is not disturbed.

The II stage is characterized by constant feeling of overfullness in the epigastrium after meal. Occasionally there may be vomiting that gives relief for some time. While probing the stomach relatively large amount (200–500 ml) of the gastric contents is pumped out with sour, slightly unpleasant smell and admixture of recently taken food mass. The patient's general state is not disturbed.

In III stage, the patients feel a constant feeling of weight and overfullness in the epigastric area combined with pain and belching with air. There is profuse vomiting several times a day at once or in 1–2 h after meal with the food eaten the day before without signs of putrid fermentation. There are large amounts of gastric contents in probing on an empty stomach. At this stage the patient has weight loss.

In IV stage progressive gastric stasis develops and stretching of the stomach. The patient's state aggravates; there is sharp dehydration. A sallow complexion is typical with loss of turgor. Malaise, apathy and flaccidity suppress other symptoms of the patient. A bursting feeling in the epigastium makes the patient cause vomiting. The vomit contains fetid contents in a large amount that disintegrates, with remnants of food eaten many days ago.

Physical examination is carried out. At the initial stages of stenosis the general state of the patients is not changed, the gastric size remains normal and it is impossible to reveal symptoms specific of stenosis. In the later stages weight loss progresses, the patient becomes cachectic; on examination enlarged percutory borders of the stomach are determined, auscultatively there is sound of splash. In probing congestive gastric contents with unpleasant odour is aspirated from the stomach in a large amount (over 500 ml).

Diagnosis. X-ray examination: at the early stages of stenosis (I and II) there is cicatricial-ulcerous deformation in the pyloroduodenal area, moderate enlargement of the stomach, there may be delay of evacuation up to

12 h. In the later (III and IV) stages there are signs of decompensation of the gastric motor activity — its distension and weakening of peristalsis with delayed evaculation for over 12 h in acute narrowing of the pyloroduodenal canal.

Esophagogastroduodenoscopy: at the early stages of stenosis there is rough cicatricial-ulcerous deformation in the pylorobulbar area with moderate narrowing of the area that does not interfere in introduction of the endoscope in the lower parts of the duodenum. At the late stages of stenosis, there is a sharp narrowing of the pyloroduodenal canal that makes introduction of the endoscope impossible and the lower parts of the duodenum cannot be examined; moreover, marked enlargement of the stomach is determined with signs of disturbed evacuation from it.

Studies of the motor function by the method of ionometry helps to reveal the tone, amplitude of the stomach contraction on an empty stomach and after intake of food irritant, allows determining time of delayed initial evacuation. At the early stages of stenosis the gastric tone is normal or increased, with characteristic inversion of amplitudes and contraction intervals with slowing down rhythm of active contractions.

In the later stages there are decreased gastric tone, rare contractions of reduced amplitude, delay of the initial evacuation.

Laboratory diagnosis (at the late stages of stenosis) determines signs of metabolic alkalosis, exicosis, hypoglicemia, hypoproteinemia.

Treatment. Conservative therapy in pyloroduodenal stenosis is, actually, preoperative preparation of patients to elective surgery. It is directed at healing of ulcer with application of modern antisecretory drugs and antihelicobacterial drugs.

Besides, in apparent disorders treatment should be aimed at correction of metabolic processes and renewal of the body weight. The latter is achieved by parenteral introduction of salt and protein solutions as well as probe enteral feeding in marked stages of stenosis (the probe is inserted lower than the stenosis area with the help of endoscopic equipment).

Surgical treatment. It should be noted that first operations of vagotomy with pyloroplasty in pyloroduodenal stenosis were not evaluated properly (J. A. Weinbergital, 1956; R. O. Kraft et al., 1964; Harper, 1966). The surgeons pointed out the possibility of gastric atonia and disturbed gastric evacuation in the postoperative period. However, next works (Z. Rachlin, 1970; N. P. Davis, J. A. Williams, 1971) confirmed the possibility of surgery with vagotomy in pyloroduodenal stenosis and the authors emphasized that threat of evacuation disturbances was exaggerated, and the postoperative period might have a smooth course.

One of the first information about vagotomy with drainage gastric operations in stenosis of the outlet part of the stomach was published in our country in 1973 (Yu. M. Pantsyrev et al.). A monograph of V. A. Ageichev et al., 1985 is one of the latest investigations that gave a clinical and pathophysiological substantiation of the surgical management in ulcerous pyloroduodenal stenosis. Leading French and American surgeons (J. Mouiel, K. Katchkhouda, 1989) performed good operations with vagotomy in noninvasive technique with the help of laparoscopic methods.

Modern surgical management takes into consideration that the process of cicatrization of the pyloroduodenal area is of irreversible and progressive character. It is clear that treatment of this complication can be only surgical regardless severity of the clinical manifestations, degree of the stomach distension, disorders of its motor-evacuation function. While choosing the method of surgical intervention, one should take into account the stage of stenosis and degree of disorder of the motor function of the stomach and duodenum as well as peculiarities of gastric secretion and degree of the operative risk.

Truncal vagotomy in combination with stomach drainage operations is most common surgery in compensated stenosis that is being formed and partly in subcompensated pyloroduodenal stenosis when patients have adequate preoperative preparation (restoration of the gastric tone and gastric evacuation rate).

Laparoscopic truncal vagotomy with pyloroplasty from mini-approach is performed in patients of young age in compensated stenosis and stenosis that is being formed.

Selective proximal vagotomy in combination with pyloroplasty is practically performed only in I stage of stenosis.

Vagotomy with antrumectomy is performed in pyloroduodenal stenoses with signs of sub- and decompensation as well as in combined form of stomach ulcer.

Stomach resection (hemigastrectomy) is performed in patients with decompensated stenosis with reduced acid production of the stomach.

Postoperative period. Management of patients after the operation for stenosis with vagotomy has its own peculiarities — first it is associated with necessity to create conditions for prevention of gastric evacuation disorder. 13% of patients develop gastric stasis in the postoperative period after truncal vagotomy. It may be both of functional and mixed character (narrowing of the pyloroplasty area by inflammatory changes of the mucous membrane). In most patients, gastric stasis is of mild degree. Intraoperatively patients at high risk of gastrostasis are inserted nasojejunal probe for enteral feeding, are administered prokinetics from the first day, and are made physiotherapy (amplipulse).

Lecture 10
CARCINOMA OF THE STOMACH

Carcinoma of the stomach is the most often malignant neoplasm of the human and occupies the first place among the reasons of death of the oncologic patients. Japan, Chile, Iceland, Russia also belong to the countries with the highest morbidity with carcinoma of the stomach. The morbidity rate in these countries is 5 times as higher as in USA. Canada, Greece, Indonesia, Nigeria belong to safe countries in this respect.

Most frequently carcinoma of the stomach arises in the age from 50 till 75 years, and the men fall ill twice more often than women. In all countries, the highest frequency of carcinoma of the stomach is marked among the people who are least provided for.

Pathological anatomy. In most cases cancer arises in the antral department of the stomach (60–70%), less often at the small curvature (10–15%) and in the cardial department (8–10%) and even less often in other parts of the stomach: the anterior and posterior wall of the stomach (2–5%), large curvature of the stomach (1%), fundus of the stomach (1%). The total damage of all the stomach by a cancer tumour is marked at 3–5% of the patients. The numerous classifications of macroscopic forms of carcinoma of the stomach are offered, but the most widespread one was suggested by Bormann (1926). Its signs have prognostic importance. Four forms of carcinoma of the stomach are distinguished:

1. Polypiform cancer (clearly limited, can be ulcerated), has a rather favorable prognosis.

2. Ulcerating carcinoma ("cancer — ulcer"), precisely limited, quite often it is difficult to differentiate macroscopically from a benign ulcer (biopsy is required). The prognosis is rather good.

3. Ulcerating and infiltrating cancer, when the borders of a tumour are determined unclear, there is infiltration in submucous structures extending usually up to a subserous layer. It is the most often type of carcinoma of the stomach, and it has a rather adverse prognosis.

4. The diffuse infiltrating tumour is notable for early metastatic spreading and has the worst prognosis from all forms of carcinoma of the stomach.

Microscopical changes at carcinoma of the stomach are rather various, as they are determined not only by degree of differentiation of cellular elements of the mucosa, but also reaction of tumour growth and other changes. On histogenesis, carcinoma of the stomach is adenocarcinoma consisting of cylindrical or cubic cells with a various degree of their differentiation. A planocellular carcinoma of the stomach (cancroid) as well as mixed glandular and planocellular cancer (acanthomas) are seldom.

According to histological attributes there are two types of carcinoma of the stomach: an intestinal type looking like differentiated and rather clearly limited tumour, and diffuse — a low-differentiated carcinoma. It is considered that the intestinal and diffuse type of carcinoma of the stomach can have different etiology and these differences have a certain practical meaning.

Etiology and pathogenesis. Numerous researches which have been carried out in our country and abroad have revealed that the carcinoma of the stomach usually arises against a background of available pathological changes of the mucous coat of the stomach. The normal functional condition of the stomach is one of the basic conditions determining resistance of the mucosa of this organ to influence of carcinogenic substances. The changes before occurrence of a cancer tumour are more often shown as various forms of chronic gastritis, polyps or ulcer of the stomach. In occurrence of these changes action of toxins (alcohol, smoking), predilection for spices, and rasping disorders of dietory habits have a doubtless meaning. Many researchers have noted high frequency of atrophic gastritis among the population of the countries with cancer morbidity, and the transformation of cells of the mucosa into a malignant tumour was proved by regular gastrobiopsies in these patients. However, character and mechanism of starting influence in a chain of arising morphological and biological changes are still unknown. Gastrotropic cancerogen is not revealed till now, but all known gastric cancerogens have genotoxic properties, actively influencing on DNA of the cell. It was shown under experimental conditions that carcinoma of the stomach can be caused by influence of nitrosamins, which can play a role of an etiological factor at the man, being formed from nitrates. An intermediate product in these transformations are the nitrites formed from nitrites at participation in this process of bacteria and enzymes, which are blasted and die under action of a hydrochloric acid. It is considered that with hypochlorhydria the formed nitrites are quickly combined with amins of a gastric juice and form nitrosamins, their cancerogenic properties are well known. Submitted above data up to a known degree explain higher frequency of carcinoma of the stomach (approximately 2–3 times as higher

than in healthy people) with diseases accompanying with hypochlorhydria and achlorhydria (atrophic gastritis, pernicious anemia, Ménétrier's disease, polyps of the stomach, especially adenomatous etc.). However, listed above diseases are considered nowadays only as facultative precancerous, so each of these diseases can exist long time and not always turn into cancer, in spite of the fact that the morphological changes of the mucous membrane of the stomach at these diseases are irreversible. Carcinoma of the stomach, as well as stomach ulcer, more often arises in the people with A (II) blood group.

The ways of stomach cancer spreading are rather diverse. The tumoral cells can penetrate immediately through the wall of the stomach and spread into neighboring anatomic structures, sprouting in the pancreas, lien, abdominal department of the esophagus and initial department of the duodenum, gallbladder, liver, colon etc. A conception about impossibility of stomach carcinoma spread to the terminal department of the esophagus and the duodenal intestine owing to structural-morphological differences of the mucous membrane of these organs and the stomach is out-of-date now. The infiltration of the submucous layer by tumoral cells can reach up to 5–7 cm from a determined border of a tumour, spreading to a neighboring part of the esophagus (duodenum). With the exophitic form of tumour (I and II type) the cancer infiltration is spread at a distance of 3–4 cm, and at infiltrative and mixed forms — up to 5–7 cm, from the macroscopically identified border and are spread mainly in a proximal direction. Lymphatic ways of metastasing is found out more than in 60–70% of the patients, and estimation of their condition is rather important from the clinical point of view. It is considered that the lymphogenous dissimination occurs first of all in organic lymph nodes of the stomach located subserously. Then the lymph nodes, located along the direction of the main gastric arterias, and nodes of the general lymphatic collector, locating in the area of the celiac trunk, are affected. This collector is basic and collects lymph from the stomach of body, small curvature and neighbouring to it parts of anterior and posterior walls, cardial part, medial part of the fundus and abdominal department of the esophagus (A. V. Melnikov). The largest lymphatic vessels and nodes are located along the pathway of the left gastric artery and in gastropancreatic ligament. A sequence and the prevalence of a lymphogenous metastatic spreading on lymphatic collectors can be various depending on localization of a tumour in one or another department of the stomach. The lymphatic system of the stomach differs by a plenty of anastomoses between various ways of lymph drainage and at a slightest obstacle to transport of the lymph, it begins to go in lymphatic vessels of the next area that also changes direction of ways of

a metastatic spreading. For the same reason quite often there is a defeat of lymph nodes of all listed ways of lymph drainage.

Removed lymphogenous metastases are found out mainly in the left subclavial lymph nodes, lymphatic node in the area of connecting of the left thoracal lymphatic duct to a venous angle (metastasis of Virchov), and lymphatic node posed in a medial department of the supraclavicular triangle, in a place of connection of internal jugular and subclavial veins (Troirsier's node). The defeat of Troirsier's node usually specifies presence of metastases of a tumour in parasternal or mediastinal lymph nodes. The hematogenous metastases most frequently arise in the liver, less often in other organs. Women can have metastatic spreading of carcinoma of the stomach in ovaries (Krukenberg's tumour), and both ovaries are usually affected.

Spreading of tumoral formations on the peritoneum can be diffuse, accompanied with ascites, or focal with formation of cancеromatous nodes in the large omentum, on a parietal peritoneum in rectovesical space (in men), and in women — in a rectovaginal fold (Shnitzler's metastasis).

Classification. All kinds of carcinoma of the stomach by the degree of spreading are divided into 4 stages.

Tumours, which do not fall outside the limits of mucous layer, are precisely limited and have no regional metastases, belong to the first stage. The second stage — a large tumour spread to all layers of the stomach wall, except for serous one, the stomach is mobile and not connected with neighboring organs. Isolated mobile metastases are present only in the nearest regional lymph nodes. The third stage — a tumour sprouts through all the layers of the stomach wall, is connected with surrounding tissues, has multiple metastases in regional lymph nodes. The fourth stage — a tumour of any size, there are multiple distant metastases.

Under the decision of the WHO the international classification of carcinoma of the stomach by TNM system was accepted, according to which three departments are distignished in the stomach: proximal (inferior third), stomach body (medium third) and antral (distal third). This classification is based on the data of clinical, roentgenologic, endoscopic researches and morphological study of the removed organ.

Element T— diffusion of a tumour:

T1 — tumour affects only mucous and submucous layers;

T2 — tumour penetrates deeper, but occupies no more than half of one anatomic departments of an organ;

T3 — tumour with a deep invasion, occupies more than half of one anatomic departments of an organ, however there is no defeat of the next departments;

T4 — tumour affects the gastric wall on the total extent and penetrates the next organs.

Element N reflects condition of regional lymph nodes:

N0 — metastases in the regional lymph nodes are not determined;

N_{xa} — only perigastric lymph nodes are affected;

N_{xb} — those lymph nodes are affected which can be removed by a surgical way (along the way of the left gastric artery, celiac trunk, common hepatic, splenic arteries);

N_{xc} — those lymph nodes are affected which cannot be removed by a surgical way (along the way of the aorta, mesenteric and ileal arteries).

Element M characterizes remote metastases:

M0 — remote metastases are not determined;

M1 — there are remote metastases.

The American Joint Committee on Cancer Staging recommends a more detailed classifications of staging carcinoma of the stomach (1988).

The detection of metastases in the cervical lymph nodes, lymph nodes of Virchov, Troirsier, area of the umbilicus, peritoneal metastatic centers (Krukenberg's tumour, Shnitzler's metastasis) irrespectively of parameters of other criteria of staging, testifies about IV stage of disease.

The exact definition of the stage of carcinoma of the stomach can be made only after inspection of the abdominal cavity during the operation and histological research of a removed specimen. The depth of penetration of tumoral elements in the stomach wall is essentially important in definition of the stage, that in comparison with size and localization of the primary tumoral center has a prior importance in definition of the prognosis.

Clinical course. The occurrence of carcinoma of the stomach is more probable among the people with increased oncologic risk. Facultative precancerous diseases of the stomach (chronic gastrites, chronic stomach ulcer, polyps of the stomach, chronic gastritis of the stomach stump after its distal resection, smoking, predilection for alcoholic drinks, action of professional harmfulnesses, stomach carcinoma in a family anamnesis) belong to the factors of increased risk.

Clinical picture of carcinoma of the stomach is various and depends on the anatomic form of a tumour, its localization, rate of growth, stage of tumoral process and character of metastatic spreading. Depending on a background, on which the carcinoma of the stomach develops, three clinical types of disease are distinguished:

— cancer developed in a healthy stomach, without signs of previous disease;

— cancer developed against a background of a chronic ulcer of the stomach;

— cancer developed against a background of gastritis and polyposis.

The signs which are found out at questioning of the patient, are possible to divide into early and late. In the early stage of carcinoma of the

stomach which has arisen against a background of complete health, the revealing of "a syndrome of small signs" (A. I. Savitsky) has important meaning.

It includes:

— change of health condition of the patient, groundless weakness, decrease of working ability, fast fatigability;

— motiveless steady decrease or complete loss of appetite;

— gastric discomfort (loss of physiological feeling of satisfaction from meal reception);

— progressing weight loss for no reason;

— mental depression (loss of pleasure of life, interest to work and environment).

Late signs of carcinoma of the stomach are dull, "pressing" pains of various intensity in the epigastric area. They can amplify after reception of coarse fare. With cancer, which has arisen against a background of chronic stomach ulcer, the earlier pain becomes constant, but less intensive. Quite often pains in the epigastric area can irradiate in the back, lumbar area and get surrounding character, which specifies penetration of a tumour to the pancreas. The pain signs proceeding with attributes of a partial intestinal obstruction specify an opportunity of penetration of a tumour in the mesentery of the transverse colon or metastatic spreading to the lymph nodes and the mesentery of the small intestine.

Dyspeptic disorders — sialosis, eructation, unpleasant taste in the mouth, nausea, vomiting specify localization of cancer in the antral department of the stomach with disorder of permeability of the pylorus. After reception of food, the patient has feelings of gravity in the epigastric area. By the end of the day quite often there is vomiting by food eaten on the eve, with a fetor odour. Frequently on an empty stomach capotement is defined.

Sensation of difficulty of food passage — dysphagia, hypersalivation, regurgitation of food and hiccup (owing to reflex contraction of the diaphragm) specify localization of cancer in the cardial department of the stomach. In these cases along with progression of tumoral process and compression of the esophagus lumen, dysphagia becomes constant and its gravity intensifies. Sensation of pressure and pains behind the breastbone or in the interscapular space owing to esophagectasia above a tumour and stagnation of nutrition are observed. General manifestations of carcinoma of the stomach are expressed in weakness, weight loss, adynamia caused both by disorders of digestion and nutritional factors, and cancer intoxication. The fervescence is connected to adsorption of albuminous products of disordered metabolism from a tumour, microbic contamination and infection of

a tumoral tissue, especially at ulceration of a tumour, and centers of inflammation in neighboring tissues and in the field of metastases, that is observed, as a rule, in late stages of the disease.

Carcinoma of the stomach can reveal itself without any local signs for a long time at localization on the anterior and posterior walls, large curvature and fundus of the stomach and conducting displays of disease in these cases can be weight loss and anemia, subfebrile temperature, groundless increased fatigability.

The clinical symptomatology of carcinoma of the stomach depends not only on localization but also from the form of tumour growth. The exophitic tumours are shown by attributes of anemia, latent and obvious bleedings, increased fatigability, weight loss, feverish condition, while the local signs are weakly expressed. An ulcero-infiltrative form of carcinoma of the stomach are shown first of all by signs of local injury — pains in the field of the epigastrium, gastric discomfort, vomiting or dysphagia.

Complications. The most often complications of carcinoma of the stomach are bleeding, perforation and infection of a tumour, and also pathological condition caused by its penetration in the next tissues. The development of carcinoma of the stomach is usually accompanied by hemorrhage, which results in anemia of the patient. More often there is a regular loss of small quantity of blood, but rather massive bleedings can arise, concerning which an urgent operation can be necessary. A clinical picture of "acute abdomen" and necessity in emergency surgery accompany perforation of a tumour in a free abdominal cavity. The penetration of carcinoma of the stomach in the next organs complicates the disease course, as the signs of affected organ join. Feature of acute surgical complications of carcinoma of the stomach (as well as other digestive tract's organs) is immunodeficiency caused by oncologic disease.

Diagnosis. Anamnesis in details assembled and well investigated has decisive meaning in establishment of the diagnosis of carcinoma of the stomach. Revealing of the probable factors of the increased oncologic risk (precancerous diseases, genetically determined predisposition etc.) is rather important also. The appearance of the patient at early stages of disease, as a rule, does not change. Grey colour of the face, mild yellowness of scleras, xeroderma, sharp decrease of the turgor, appreciable disappearance of hypodermic fat specifies the late stage of disease. Palpation of the stomach should be carried out in four positions of the patient: on the back, on the right and left side and standing. It allows to determine the sizes of a tumour, character of its surface, displaceability in relation to other organs of the abdominal cavity.

It is necessary to investigate places of their frequent occurrence: the left supraclavicular fossa (Troirsier's node), intercrural space of the left stemocleidomastoid muscle (Virchov's node), area of umbilicus, digital research of the rectum (Shnitzler's metastasis), research of the ovaries (Krukenberg's tumour) for revealing of the remote metastases. For recognition of metastases in parenchymatous organs the roentgenologic inspection of the lungs is obligatory, US of the liver, the kidneys, the pancreas, if it is necessary — computer roentgenography of this area.

The laboratory blood analysises at carcinoma of the stomach usually find out decrease of amount of haemoglobin and erythrocytes, increase of ESR. During researching gastric secretion achlorhydria and hypochlorhydria, quite often — positive Gregersen's reaction are revealed. Assay on occult blood in feces (Gregersen's reaction) is positive at 90% of the patients with carcinoma of the stomach. From other methods of diagnosis, the X-ray inspection is basic for early and well-timed revealing of a carcinoma of the stomach and allows distinguishing this disease approximately in 90% of the patients. The complex roentgenologic inspection under conditions of a double contrast study and hard filling of the stomach allows establishing localization, extent and form of a tumoral defeat.

Fibrogastroscopy with a directed biopsy now as the most precise method of diagnosis of the early forms of cancer is regarded. It allows not only to establish character of a tumoral defeat, localization, form of growth, prevalence, but also to specify morphological structure of a tumour by means of histological and cytologic research of material (slices of the tissue, scraping from the surface mucous layer) taken at endoscopy. These two methods (gastroscopy and gastrobiopsy) do not compete among themselves, and supplement each other, that considerably raises accuracy of diagnosis and allows essentially to reduce terms of establishment of the diagnosis.

The accuracy of morphological diagnosis of benign and malignant tumours of the stomach reaches 99% at gastrobiopsy.

The laparoscopy (peritoneoscopy) at carcinoma of the stomach allows to examine a parietal and visceral peritoneum in the field of research, organs of the abdominal cavity, to determine penetration of a tumour to the next organs, to find out metastases in the liver, carcinomatosis of the peritoneum and to exclude an opportunity of vain explorative laparotomy.

Radioisotope diagnosis with the help of various methods of scanning is based on an ability of more intensive accumulation of some radioactive isotopes in cells of malignant tumours. The application of these methods allows to differentiate malignant neoplasms from benign ones and to reveal the remote metastases in various organs.

At clinical suspicion on carcinoma of the stomach, when with the help of modern complex methods of research it fails to establish a true nature of disease, diagnostic laparotomy is indicated to the patient.

Differential diagnosis of carcinoma of the stomach is necessary first of all with those precancerous diseases of the stomach on which background a malignant tumour frequently develops (chronic gastritis, chronic ulcer of the stomach, polyps and polyposes of the stomach). Carcinoma of the stomach is necessary to differentiate also with tuberculosis of the stomach, which more often is shown in the form of cicatrical stenosis or ulcer, solitary breaking up granuloma or hyperplastic process quite often arising at the serious forms of tuberculosis.

At syphilis of the stomach, there are series of characteristic clinical features: men fall ill more often than women, the disease develops in earlier age and achlorhydria is always marked.

Pernicious (malignant) anemia, achlorhydria and digestive disturbances are peculiar. Study of the blood analyses and roentgenologic inspection help to establish the correct diagnosis.

Treatment. The basic kind of radical treatment of the patients with a carcinoma of the stomach is operative erasion of a tumour within the limits of healthy tissues and regional lymph nodes.

Radicalism of operation is provided with keeping of the following oncologic principles:

— ablastics of operative intervention achieved by the use of a complex of measures, preventing contact of tumour with healthy tissues of a surgery field;

— complex erasion of the affected stomach or its part and three groups of lymph nodes (which most frequently affected by metastases) together with the small and large omentum and ligamentous apparatus of the stomach;

— dissection of the esophagus, the stomach and the duodenum within the limits of healthy tissues, which is proved by urgent histological research of oral and aboral section of a specimen.

The following basic types of radical operations are applied:

— a distal subtotal resection of the stomach according to one of Billroth's ways (at cancer of the pyloroantral department of the stomach);

— gastrectomy with applying of the esophagojejunoanastomosis, in same cases — esophagoduodenoanastomosis (at cancer of the stomach body or its total defeat);

— subtotal proximal resection of the stomach with applying of esophagogastric anastomosis (at cancer of the upper third of the stomach).

The palliative operations (palliative resection of the stomach, collateral anastomoses — gastroenteroanastomosis, esophagojejunoanastomosis, eso-

phagofundoanastomosis, gastrostomy and jejunostomy) are applied in case of occurrence of complications (obstruction of the cardial part, pylorus, inoperable bleeding tumours etc.), grave condition of the patient and impossibility of radical operation. These operations can be complemented by the subsequent chemotherapy (5-fluoruracil, phthorafurum). However, as experience of many establishments shows, the immediate effect of such combined treatment is insignificant and practically is not reflected in the remote results. The radiation therapy of a carcinoma of the stomach is in the stage of scientific development. In cases of late carcinoma of the stomach, when the general condition of the patient does not allow making any palliative intervention, the symptomatic treatment is carried out.

The most favorable prognosis and the positive remote results of surgical treatment of a carcinoma of the stomach are marked at the early forms of a cancer. So, five years' survival rate after operative treatment at 0, I stage makes 80–90%, II — about 50%, III — about 20%. On all stages the medium five years' survival rate after subtotal distal resection of the stomach makes 42–45%, after a proximal subtotal resection — about 24%, after gastrectomy — about 30%.

Thus, early diagnosis of carcinoma of the stomach is in the basis of improvement of remote results of treatment by way of regular routine inspections, first of all among groups of the increased risk.

Prophylaxis of carcinoma of the stomach consists in organization of a balanced diet, healthy life style, well-timed diagnosis and treatment of precancerous diseases.

Lecture 11
HERNIAS OF THE ABDOMINAL WALL

There are external and internal hernias of the abdomen. External hernias of the abdomen are protrusion of internal organs of the abdominal cavity covered with parietal layer of the peritoneum through natural and acquired defects of a musculoaponeurotic layer of the abdominal wall.

It is necessary to differ eventration and abaissement (prolapse) of internal organs from external hernias of the abdomen. Eventration is a protrusion of internal organs through defect in the abdominal wall and the parietal leaf of the peritoneum. The eventration is usually associated with an acute trauma of the abdominal wall and most frequently arises because of wounds, traumas and operative interventions. It can be closed (hypodermic) or open (complete). Prolapse is a protrusion of separate internal organs or their parts through natural openings due to inversions of their internal wall (prolapse of the vagina, uterus, and rectum).

Internal hernias of the abdomen are a protrusion of organs of the abdominal cavity into the various folds and pockets of the peritoneum, mesentery of the intestine, and into the thoracic cavity through the natural or acquired apertures and clefts of the diaphragm. The internal hernias of the abdomen belong to infrequent kinds of pathology. In series of cases, they can be the cause of development of intestinal obstruction and, as a rule, are recognized during operative intervention.

The external hernias of the abdomen are one of the most frequent surgical diseases. Among the diseases, undergoing to surgical treatment, they make 8–20%, or about 3–4% of all population have the abdominal hernias. External hernias of the abdomen occur more often in men than in women. Inguinal hernias develop more often in men, other localization of external hernias of the abdomen occur mainly in women. Hernias of the abdomen are observed at any age, but the greatest morbidity rate is marked in preschool age and after 50.

Etiology and pathogenesis. As the basic condition of occurrence of abdominal hernias it is necessary to consider a long term disbalance be-

tween force of the intra-abdominal pressure and resistance of certain sites of the abdominal wall. Under the influence of repeated considerable increase of intra-abdominal pressure stretching of tissues arise in these places and the hernial ring is formed. Relaxation of the parietal leaf of the peritoneum and its protrusion occur. From inside a recess is formed according to a locating of hernial ring, and from the outside it represents a protrusion of the parietal peritoneum which is subsequently transformed to the hernial sac — an element of an external abdominal hernia.

It is possible to divide the causes, promoting to the formation of hernias into local and general.

The local causes contributing to occurrence of external abdominal hernias are associated with anatomic features of the abdominal wall structure, especially in its "weak" places. To such places, or "hernial points", of the abdominal wall the following places belong: a zone of the inguinal canal, zone of the oval fossa on the femur, aponeurosis of the white line of the abdomen, the umbilical area, a zone of Spigel's line, Pty's triangle, Grunfeld — Lesgaft's quadrangle, obturator canal and some others.

The general causes are the following:

1. Contributing causes are features of constitutional character (general malformations, weak body build, insufficient physical development), heredity, sex, age etc.

2. Causes which lead to the increase of the intra-abdominal pressure and decrease of biomechanical properties of the abdominal wall.

Now the role of hereditary predisposition in the formation of hernias is beyond all question and observed in 20–25% of the patients. It tells not only about the hereditary transfer of features of the abdominal wall anatomic structure, but also about true, constitutional predisposition to the formation of hernias. Inherent weakness of connective tissue lays in its basis, having a character of a systemic pathology which is revealed as multiple hernias, for example, combination of hernia of the esophageal aperture with an inguinal hernia and so on. In such patients malformations of sceletal musculation, platypodia, varicosis, and atonic constipations are often observed.

To some degree, the type of a body build contributes to occurrence of hernias of one or another localization. So, the people of hypersthenic (brachimorphic) type have anatomophysiological precondition for occurrence of inguinal, epigastric hernias, and at asthenic (dolichomorphic) body build umbilical and femoral hernias are more often observed. The features of occurrence of hernias in men and women are connected with the features of sexual differences of the body build in many respects. The most frequent development of external abdominal hernias in men is explained by signifi-

cant prevalence of inguinal hernias, due to the features of development and structure of the inguinal canal. In men the inguinal canal is shorter and wider, than in women, it is worse strengthen by the muscular and tendinous layers, however diaphragmatic, umbilical, femoral, postoperative, obturator, perineal hernias and hernias of Spigel's line occur more often in women. This fact allows approving that constitutional features of a woman to the greater degree contribute to the formation of appropriate kinds of hernias.

An important etiological factor causing weakening of the abdominal wall in women is pregnancy, in which significant thinning and expansion of the white line of the abdomen, expansion of the umbilical ring, overstretching of the muscles take place.

Ascites, sharp attrition of the elementary character or due to prolonged illnesses are factors weakening the abdominal wall and contributing to occurence of abdominal hernias. Such patients have flaccidity of the tissues of the abdominal wall, their elastance decreased. Disappearance of fat from the tissues is accompanied by increase of those clefts and apertures in the abdominal wall which were filled with it before. As a result the resistance of the abdominal wall to the action of intra-abdominal pressure is reduced and the favorable conditions for the formation of excavation (fossa) in the parietal peritoneum are formed, i. e. the first and necessary condition of herniation. The occurrence of ventral hernias is caused also by obesity, leading to the degenerative changes in the muscles and overstretching of tendinous and muscular layers, which surround structural formations of the abdominal wall, filled with the fatty tissue. Absence of the regular physical exercises, hypodynamia results in weakening of the abdominal wall, therefore an early flaccidity of muscles develops. Atrophy of the muscles of the abdominal wall arises in case of damage of their nervous trunks (intercostal nerves) because of traumas, operative interventions or diseases (poliomyelitis). The cases of hernias development after damage of the backbone and spinal cord are known.

The important cause of development of hernias of the abdominal wall is the significant stable increase of the intra-abdominal pressure: serious physical work, especially in absence of labour skills; frequent cry in infantile and children age; acute and chronic diseases of respiratory organs accompanying by unbearable cough; long constipations and diarrheas; diseases complicating emiction (adenoma of prostate); frequent vomiting; play wind instruments etc.

Structure and pathological anatomy. In the structure of each external hernia of the abdomen it is necessary to distinguish its following elements: hernial ring, hernial sac, hernial environments and hernial sac contents.

The hernial ring — is a defect in musculoaponeurotic layer of the abdominal wall, through which protrusion of internal organs and the parietal leaf of the peritoneum occurs. The hernial ring can arise in each congenital or acquired weakened place of the abdominal wall. Congenital weakened places are caused by features of localization of muscles, fascias, aponeuroses of the abdominal wall promoting to the occurrence of clefts interspaces between their edges, which are initial item for formation of the hernial ring. The musculoaponeurotic tissues forming the edges of the hernial ring are movable and elastic at the initial stage of the hernia formation, but due to the pressure of enlarged hernial protrusion, which increases, they can be exposed to atrophy and fibrous degeneration, transformed into inflexible and rigid ones. The hernial ring can be round, oval, triangular, fissural or indefinite. The size is also variable a lot: from 1–2 cm in umbilical hernias and hernia of the white line; up to 20–30 cm and more in postoperative hernias.

The hernial sac is formed by protrusion of the parietal leaf of the peritoneum through the hernial ring. The ostium, the neck, the body and the fundus of the hernial sac are distinguished. An opening of the hernial sac is a place, where its cavity is connected with the peritoneal cavity. The neck of the hernial sac is its proximal department, which is located in the hernial ring. Usually the neck has a little bit extended, narrow, tubular shape; its walls are thicker in comparison with the other part of the hernial sac and are less moveable. The neck of the hernial sac passes into the body, its largest part. The most distal part of the hernial sac is the fundus.

The shape of the hernial sac can be various: piriform, cylindrical, spindle-shaped, etc. Besides, the hernial sacs can be simple — unicameral, and complex — double, and polychamber. In case of the formation of multiple lateral diverticula the hernial sac can remind the fingers of a rubber glove. Sometimes under the influence of the undergone inflammation and subsequent cicatrization one of the cavities of the hernial sac appears separated from the other cavities and gets character of a cyst, in other cases it transforms into a so-called sacculated hernia. This term means a hernia, in which the hernial contents are located in the general cavity of the hernial sac not freely, but inside the second cavity or capsule, which is a pocket of the hernial sac, separated by adhesions from a general cavity of the ostium. The thickness of the hernial sac is various and depends on the time of occurrence and degree of its traumatization. In old hernial sacs the significant thickening of the wall and development of cicatrical connective tissue is observed. The size of the hernial sac widely varies. Like the size of the hernial ring it can be small at initial stages of hernia formation and reaches large and vast sizes while its long existence and development.

Sometimes through the hernial ring simultaneously with the hernial sac the organ is protruded only partially covered with the peritoneum or placed in the retroperitoneal fat tissue, for example, urinary bladder, colon and so on. In these cases, one of the walls of the hernial sac is partially covered with peritoneum of a "glided organ". Such hernias are called sliding ones.

The hernial sac can be congenital (congenital protrusions, or embriologic rests of former pockets, peritoneum) and acquired. Therefore, for example, in case of delay of obliteration of the vaginal process of the peritoneum in the inguinal canal it can transform into the hernial sac of a congenital inguinal hernia.

The hernial environments — are tissues covering outside the hernial sac and locating between it and the skin. In various hernias, the character and the quantity of such environments are different. There are preperitoneal fat, stretched and thin plates of fasciae, fibers of the muscles; elements of the spermatic cord also belong to them.

Internal organs of the abdominal cavity, which are moved constantly or periodically through the hernial ring in the hernial sac are called hernial sac contents. The hernial sac contents can be any organ of the abdominal cavity, but more frequently those organs which are located closer to the place of the hernial ring and which have the greater mobility. Due to the frequency of property to the structure of the hernial sac contents they can be located according to the following order: loops of the small intestine; the omentum; parts of the intestine supplied with the mesentery; a vermiform and fatty appendages of the large intestine; the uterine tubes and ovaries, sometimes the uterus, even pregnant; folds of the mesentery of the intestine; organs located retroperitoneally or partially covered with the peritoneum, as a so-called sliding hernia (the urinary bladder, the caecum, ascending and descending departments of the colon, the ureter, the kidney, the pancreas); at last, the stomach, the spleen, the gallbladder, Meckel's diverticulum. The hernias, which contents were all the organs of the abdominal cavity, occur.

If the hernial sac contents are freely replaced into the abdominal cavity, such hernias are called free, or reducible. Under the influence of various mechanical irritators, for example, while long use of a bandage or often incarcerations, there is an aseptic inflammation in the walls of the hernial sac, which entails the formation of adhesions between the hernial sac and hernial sac contents. The adhesions more often develop in the area of the fundus and neck of the hernial sac. They fix contents of the hernial sac to its walls, their reductions interfering into the abdominal cavity. The fixed or irreducible hernia develops. The sudden prelum of the hernial sac contents

in the area of the hernial ring, excluding an opportunity of its free reductions, is called a strangulated hernia.

Classification. Abdominal hernias are classified by anatomic, etiological and clinical signs.

According to localization of the hernial ring inguinal, femoral, umbilical hernias, hernia of the linea alba, Spigel's line, and also lumbar, ischiadic, diaphragmatic and perineal hernias are distinguished.

According to etiological signs all hernias depending on their origin are divided into two groups: congenital and acquired. The majority of external congenital hernias of the abdomen is inguinal and umbilical hernias. They can occur at once after the birth of a child or much later. Acquired hernias are divided depending on the causes, which have resulted in occurrence of defect of the abdominal wall. Most frequently between all acquired external hernias of the abdomen there are hernias which have arisen in typical "weak" places of the abdominal wall. The major group is made with postoperative ventral hernias arising in various areas of the abdominal wall after laparotomies. If these operations were performed concerning hernias arising again in the same area, they are called recurrent.

Hernial protrusions of the abdominal wall in various areas after traumas without skin damage, but with defect of the other anatomic layers, except of the peritoneum, are called traumatic hernias. Hernial protrusions due to the weakening of the abdominal wall (congenital weakness of the abdominal wall, malformation, residual phenomena after poliomyelitis) are called neuropathic hernia. After various pathological processes in the abdominal wall with the damage of separate layers and its subsequent weakening hernial protrusions develop, called pathological hernias. And at last, there are artificial hernias developing after severe damage of the external inguinal ring (mutilation), which present only historical interest.

According to clinical classification, hernias can be single or multiple. According to their course they can be complicated and uncomplicated. The uncomplicated hernias are divided in reducible, or free, and irreducible (some authors consider irreducible hernias as chronically complicated). Basic complications of hernias are incarceration, coprostasis, inflammation of both hernial sac contents, and hernial environments, damage and neoplasm of the hernias.

UNCOMPLICATED HERNIAS

The most typical complaints of the patients are pains and presence of a protrusion in the area of the abdominal wall. The pains caused by abdominal hernias, can be in the area of the hernial ring, in the lumbar area, according to a projection of the localization of the small intestine mesentery root, and diffuse abdominal pains without certain localization.

At first pains are usually insignificant or moderate and even not strictly localized. Sometimes these pains are characterized by patients as insignificant, but in the process of hernia formation they become localized and pronounced. The character of pain is rather individual and various, and the extent of pain is not proportional to the increase of the sizes of the hernial protrusion. Frequently on the contrary, the pains disturb the patient more during the hernia formation, and with its transfer into the stage of complete one they weaken considerably. The aggravation of pains while walking, on exertion, lifting of loads, defecation of the patient is typical. Sensations of pain can be single complaints during the initial periods of the hernia formation, and indeterminacy of their localization and their atypical irradiation can serve the cause of the erroneous diagnosis. Sometimes pain sensations are absent or are so insignificant that practically do not disturb the patient. The basic complaint in these cases is presence of a protrusion of different sizes in the area of the abdominal wall. In a reducible hernia the protrusion changes in sizes depending on the position of the body of the patient (being enlarged in a vertical position and decreasing in supine position), under condition of the intra-abdominal pressure (being enlarged in tension, coughing) and so on. It does not occur in case of irreducible hernia. In large hernias, especially irreducible, sometimes there are complaints of dysuric and dyspeptic disorders, constipations that can be an indirect sign indicating a possible presence of a sliding hernia. Sometimes complaints are practically absent and hernia appears as a casual finding at physical examination.

The examination of the patient is necessary to carry out in vertical and horizontal positions, in rest and in tension.

A characteristic objective sign of an external hernia of the abdomen is the presence of a protrusion of the anterior abdominal wall in the area of the hernial ring. The shape and size of the hernial protrusion are various and depend on the localization and the stage of hernia formation. At the initial stages the protrusion of the abdominal wall can be absent and then in recognition of hernia an anamnesis is of great importance. The following two signs, characteristic for external abdominal hernias, are caused by the presence of the connection between the hernial sac and the abdominal cavity. The first sign is the change of the size of the hernial protrusion depending on the position of the body and the change of intra-abdominal pressure: in a vertical position the hernial protrusion is enlarged, and in supine position it decreases or completely disappears. The increase of sizes of a hernial protrusion is observed in coughing tension, and depends on the moving of the hernial sac contents from the abdominal cavity into the hernial sac and the back. In irreducible hernia this sign is negative. The second

cardinal sign is the presence of "a cough shock", which is felt with a finger introduced in the place of hernial protrusion to the location of the hernial ring. The sign is considered positive if at the moment of coughing the shock pressure on finger, caused by sharp short-term increase of intra-abdominal pressure, is felt. A characteristic sign of the external uncomplicated hernia is also determination of defect in musculoaponeurotic and fascial layers of the abdominal wall in the place of a protrusion, set in the abdominal cavity. In an irreducible hernia, the detection of this sign happens difficult. Its signs are: conservation of the sizes of the hernia while the change of a position of the body of the patient and attempts of a cautions reductions, difficulty of digital examination of the hernial ring, morbidity on palpation of the hernial protrusion in conservation of a sign of the cough move symptom.

The local inspection should be complex and include all obligatory methods of clinical examination of the patient. On examination it is necessary to pay attention to the condition of the skin above the protrusion: presence of venous network dilatation, intertrigoes, maceration, cracks and ulcerations. On palpation a consistency, character of the surface of the hernial protrusion, its relation to surrounding tissues and nearby anatomic structures are estimated. The consistency of the hernia depends on the character of hernial sac contents: if it contains the omentum, the consistency is soft, with the intestinal loop — it is soft-elastic, if the contents is liquid, the hernia is rigid-elastic. Then, if it is possible, the cautious reduction of the hernial protrusion in the abdominal cavity is made, and it is better to ask the patient to make it by himself. In free hernias it is easily made if contents appear to be the omentum, the reduction occurs soundless, if the intestinal loop is set — with ham. Examination of the hernial ring is carried out after hernia reduction. Thus the form and sizes of the hernial ring, condition of surrounding tissues, durability, flaccidity, tonus of the muscles, mutual relation with hernial sac contents are estimated. On percussion of the hernial protrusion and the presence in the hernial sac of a caval organ thympanitis is determined, and in the presence of the omentum or liquid — a dull percussion sound. Peristaltic hums, auscultated above the hernial diverticulum, indicate the presence of the small intestine loop in hernial contents.

On suspicion on a sliding hernia, it is necessary to add irrigoscopy, cystography, bimanual gynaecologic examination. If there is a suspicion on the involvement of the ureter or the kidney, the infusion urography is necessary. In case of differential diagnosis between an inguinal hernia and tubercular abscess, the large help can be rendered by roentgenography of thoracic and lumbar departments of the backbone. In some cases, for example during differential diagnosis between an indirect inguinal hernia and hydrocele, diaphanoscopy is applied.

Treatment. Hernial self-recovery is not practically observed; therefore, the presence of hernia is the indication for peforming operation. The use of bandage is administered only in the case when there are absolute contraindications or during preparation for operative treatment (in large hernias). In irreducible hernias the use of bandage is contraindicated.

Contraindications for the operative treatment:

a) absolute: acute infectious diseases, malignant tumours of the III–IV stages, severe accompanying diseases in the stage of decompensation, acute period of disorder of cerebral and coronary circulation, presence of pustular diseases of the skin in the area of a prospective surgical intervention;

b) relative: early children age, very advanced age of the patient in the presence of chronic diseases, late terms of gestation.

The aim of the operative treatment is elimination of the hernial sac and closing of defect of the abdominal wall.

The basic stages of operation:

1) anatomic section of tissues level-by-level above the hernial protrusion;

2) isolation of the hernial sac from the hernial environment;

3) hernial sac dissection, liberation from the adhesions of the hernial sac contents and its reduction to the abdominal cavity;

4) dissection and ligation of the hernial sac at the level of its neck, with its further removal;

5) plastic repair of the hernial ring.

The features of an operative intervention depend on the localization and the structure of the hernia, clinical course of the disease.

Besides of traditional methods of operative interventions, hernioplasty is possible at the place of laparotomic approach. During this operation, the hernial ring is closed from the part of the abdominal cavity more often with the help of various alloplastic materials: ampoxen, polypropilen, grid etc. Performance of similar operative interventions, as a rule, is carried out in uncomplicated reducible hernias. It is considered to be expedient the performance of these operations as simultant, when laparotomy is carried out due to the other surgical disease.

Recently the endoscopic surgery of the abdominal cavity has become available and widespread. Various methods of laparoscopic hernioplasty are developed and applied in clinical practice. The modern laparoscopic techniques provide good examination and an opportunity of surgical manipulations in the area of the hernial ring internal surface. Intra- and extraperitoneal approaches in hernioplasty are applied. In small and uncomplicated hernias, complete removal and dissection of the hernial sac are possible.

The cases of successful performance of a laparoscopic hernioplasty in irreducible inguinal hernias are described in literature. The closing of the hernial ring is carried out both with the help of alloplasty, and the abdominal wall defect suture ligation. In certain cases, laparoscopic hernioplasty can become alternative to traditional methods of the abdominal hernias surgical treatment.

Prognosis in the operative treatment of uncomplicated hernias, as a rule, is favorable. The relapses rate after hernioplasty ranges from 0.8 to 16%. They are observed mainly in large hernias, at the elderly and senile age, in postoperative wound infection and in disorder of a working regimen and on physical exertion stresses. The character of operation is also important. The basic danger in patients with external abdominal hernias is incarceration. The well-timed surgical treatment is its reliable prophylaxis.

Inguinal Hernias

Inguinal hernias leave the abdominal cavity into the inguinal area. It is the most frequent localization of external abdominal hernias and occurs mainly in men. Among all external abdominal hernias inguinal hernias make up 70–80% (N. I. Kukudzhanov, 1969). The borders of inguinal area are: from above — a line connecting spina iliaca anterior superior, from below — the inguinal ligament, medially — external edge of the musculus recti abdominis. The part of the inguinal area in which there is the inguinal canal, is called the inguinal triangle. The borders of the inguinal triangle are: from above — the horizontal line, which has been carried out from the border between the external and medium third of the inguinal ligament, from below — the inguinal ligament, medially — the lateral edge of the musculus recti abdominis. The inguinal canal with the length of 4–5 cm is located obliquely, passes from above, outside and behind — downwards, inside and forward. The anterior wall of the inguinal canal is formed by aponeurosis of the musculus obliquus externus abdominis, the upper wall — by the lower edges of the musculus obliquus internus abdominis and the musculus transversus abdominis, lower one — by the inguinal ligament. The internal aperture is limited by the fibers of the transversal fascia and is on the posterior wall of the inguinal canal. Through it from the retroperitoneal fat tissue in the inguinal canal the spermatic cord in men, the round ligament of the uterus — in women enters, making its contents. Passing through the inguinal canal, they go through its external aperture located on the anterior wall. The external aperture of the inguinal canal is limited by two pedicles of aponeurosis of the musculus obliquus externus abdominis, which are

radiated, and from the middle part they are limited by transversal fibers. In the inguinal canal they observe a so-called inguinal space — a room between the inguinal ligament from below, the lower edges of the musculus obliquus internus abdominis and the musculus transversus abdominis from above, by the external edge of the musculus recti abdominis medially, that corresponds to the posterior wall of the inguinal canal. The shape and the sizes of the inguinal space is of great importance in the development of inguinal hernias.

Structure of the spermatic cord includes the deferent duct, blood, lymphatic vessels and nerves of the duct and testicle, a rest of the vaginal process of the peritoneum. All these formations are placed into the fascial environment limited by the transversal fascia, and from above they are covered with the fibers of the muscle, which lift the testicle. Outside of the inguinal canal the spermatic cord is covered from above with the fascia spermatica externa, representing the thin continuation of Tompson's plate of the proper fascia of the abdomen and aponeurosis of the musculus obliquus externus abdominis. The round uterine ligament passes into the inguinal canal near to the adherent vaginal process of the peritoneum. In its non-closure a so-called Nuck's canal is formed, at the expense of which there can be cysts or inherent inguinal hernias.

For understanding the mechanism of occurrence of inguinal hernias it is necessary to know the features of location of the peritoneum, which forms the posterior surface of the abdominal wall in the lower part of the abdomen. Here between the peritoneum and the transversal fascia the vessels and rest of the urinary duct pass, above which the peritoneal folds are formed. From the apex of the urinary bladder to the navel the plica umbilicalis midlinea is located, which contains the obliterated urinary duct. From the lateral surface of the urinary bladder in the direction to the navel from each side plica umbilicalis medialis goes, which is formed mostly by obliterated umbilical artery. The plica umbilicalis lateralis passes externally from this one, in which the artery and *vena epigastrica inferior* are placed. There are excavations, or fossae, of the peritoneum above the inguinal ligament between these plicas. The fossa supravesicalis is formed between the plica umbilicalis midlinea and the *plica umbilicalis medialis;* between the plica umbilicalis medialis and the *plica umbilicalis lateralis* — the *fossa inguinalis medialis,* and externally from the *plica umbilicalis lateralis* — *fossa inguinalis lateralis.*

The medial inguinal fossa according to its position approximately corresponds to the superficial inguinal ring and is projected externally from the pubic tubercle; the lateral inguinal fossa corresponds to the deep inguinal ring and is projected above the middle part of the inguinal ligament.

For understanding the development of oblique inguinal hernias, especially congenital, it is important to know embryogenesis of the lower part of the abdominal wall, testicle or uterine ligaments. For the first 3 months of the intrauterine development the testicle is located into the lumbar area, laterally from the backbone, adjoining to the primary kidney. The peritoneum covers it from three sides and adheres with tunica albuginea of the testicle. From the lower pole of the testicle behind of the peritoneum there is a special connective chord, a so-called conductor of the testicle. It penetrates at the level of the future internal aperture of the inguinal canal into the scrotum. There the transversal fascia and parietal leaf of the peritoneum are stuck out, which forms a vaginal process of the peritoneum. The testicle, lawering into the scrotum, passes through the inguinal canal. It pushes out the leaf of the peritoneum, which covers the testicle, ahead and densely adheres to its tunica albuginea. In the scrotum the testicle is covered with two layers — visceral and parietal, between which there is a small fissural cavity. The vaginal process is usually obliterated higher than the testicle to the moment of birth of the child. If it does not occur, the abdominal cavity is connected with an unadherent vaginal process of the peritoneum, which can be the hernial sac of inherent inguinal hernia. In this case there will be the testicle due to the structure of its contents, in women — the round uterine ligament.

One differs oblique, or lateral, and direct, or medial, inguinal hernias. Supravesicular hernias occur rarely. Oblique inguinal hernias are formed as a result of a protrusion of the hernial sac through the internal inguinal ring located according to the external inguinal fossa. In this case the hernial sac repeats the way of the spermatic cord, and is placed in the middle part of the common vaginal tunic, surrounded from the different directions with its elements. At the initial stage of development oblique inguinal hernia remains within the limits of the inguinal canal, and in the process of enlargement leaves through its external aperture and then following to the spermatic cord penetrates into the scrotum (in women — into the fat tissue of the large pudendal lip). In case of unadherence of the vaginal process of the peritoneum the inherent inguinal hernias develop. Congenital inguinal hernias are always oblique.

The direct inguinal hernias develop as a result of a protrusion of the hernial sac through the medial inguinal fossa. The hernia passes into the inguinal canal through its medial parts, just in the area of the external aperture. The hernial sac in this inguinal hernia is located externally of a common vaginal tunic of the spermatic cord. The direct inguinal hernias do not lower into the scrotum. A supravesicular hernia is pushed out through the same fossa.

Inguinal hernias, especially frequently direct, can be sliding. Sometimes there is a combination of direct and oblique inguinal hernias. Such hernias are called combined. Sometimes an oblique inguinal hernia while enlarging does not leave the inguinal canal through the external aperture, and begins to go between the layers of the muscles and aponeuroses. Such hernia is called interstitial. In men an oblique inguinal hernia can be combined with cryptorchism, varicocele, cysts of the spermatic chord, periorchitis, perifuniculitis; in women near to the hernial sac a cyst of Nuck's canal can be located.

Local reasons for inguinal hernias appearence are connected with individual features of the inguinal area structure. The condition of the posterior wall of the inguinal canal, transversal fascia, and expansion of the internal inguinal ring in the inguinal hernias pathogenesis are especially important. The weakening of the posterior wall of the inguinal canal is a cause of not only direct but also oblique inguinal hernias development. The enlargement of the hernial protrusion while the formation and the development of an oblique inguinal hernia results sometimes in considerable expansion of a deep inguinal ring, its combination with the superficial one and the formation of the hernia with a straightened canal.

The condition of the posterior wall of the inguinal canal in many respects depends on the shape of the inguinal space. The fissural and triangular forms of the inguinal space are distinguished. At the fissural form the *musculus obliquus internus abdominis* covers well the internal aperture of the inguinal canal and strengthens the posterior wall. At muscular contraction the valve compresses the spermatic cord. At the high triangular form of the inguinal space the posterior wall of the inguinal canal on the large extent is not covered with the muscles, its internal aperture extends, the valval function of the muscles is absent. The relaxation of aponeurosis of the *musculus obliquus externus abdominis,* the extension of the aperture of the inguinal canal are of great importance.

Classification of inguinal hernias:

I. According to the localization:
   1. Unilateral inguinal hernias.
   2. Bilateral inguinal hernias.

II. According to the type:
   1. Direct inguinal hernias.
   2. Oblique inguinal hernias:
      a) congenital;
      b) acquired;

III. According to the stages of development:
1. Inguinal hernia which develops.
2. Hernia of the inguinal canal, or canal hernia.
3. A complete oblique inguinal hernia.
4. An inguinoscrotal hernia.
5. A giant hernia.

IV. According to the course:
1. Uncomplicated inguinal hernias:
a) reducible;
b) irreducible;
2. Complicated (strangulated, coprostasis, inflammation etc.).

V. According to the complexity:
1. Simple inguinal hernias.
2. Transitive.
3. Complex.

Clinical picture and diagnosis. Oblique hernias occur more frequently than direct ones. Direct inguinal hernias make only 5–10%. Oblique hernias usually arise at young and middle-aged people, direct — at elderlies. Direct hernias develop especially with a so-called soft, pliable, "weak" groin. It is no considence that they have been called hernias due to the "weakness". Direct hernias are more frequently bilateral. The characteristic objective sign of inguinal hernias is the protrusion of the anterior abdominal wall into the inguinal area. In an oblique inguinal hernia it is placed following to the inguinal canal, frequently lowers into the scrotum. In large hernias in men half of the scrotum laterally of the protrusion is sharply enlarged, its skin becomes stretched, the penis deviates into the opposite side, and in giant hernias disappears in the folds of the skin. In direct inguinal hernia the protrusion has a spherical or oval shape; it placed in the medial part of the inguinal ligament, near to the external edge of the pubis. When a hernial diverticulum is located above a projection of the external aperture of the inguinal canal, it is necessary to suspect the presence of a periinguinal or interstitial hernia.

On palpation it is necessary to estimate the relation of the hernial protrusion to the pubic tubercle. The palpation is made with an index finger, which is introduced into the external aperture of the inguinal canal. In inguinal hernias the tubercle is easily palpated outside from the protrusion; in femoral hernias it is not possible to find out (Kuper's sign). After palpation of the protrusion in free hernias it is set into the abdominal cavity. The palpation of the ilioinguinal area is made immediately after the reduction of the hernial protrusion. The presence or absence of testicles in the scrotum, their

size and shape, the presence of varicocele are determined. Lymph nodes of the inguinal area and area of Skarpovsky's triangle are examined simultaneously.

Then the examination of the inguinal canal is begun. In men the dermal fold of the scrotum is invaginated with a medium or index finger. In women the introduction of a finger into the inguinal canal isn't always possible, but it is possible only in case of flabby skin and considerable extension of the external aperture. Therefore it is necessary to be limited by the inspection and palpation of the hernial protrusion which in a direct hernia is located above the inguinal ligament, and in oblique one lowers into the large pudendal lip. The examination of the inguinal canal is carried out both in vertical, and horizontal position and is subdivided into a number of subsequent moments. At first they palpate a pubic tubercle, then advance a finger forward and out in the direction of the external aperture of the inguinal canal. Normally the external aperture passes the end of a finger. In hernia, depending on its size, the diameter of an aperture is enlarged up to 2–3 cm and more, passing up to 2–3 fingers and more. In a direct inguinal hernia the finger entered into the hernial canal along the lower edge of the canal in its initial part the upper surface or the tubercle of the pubic bone is clearly palpated. It is not present in an oblique inguinal hernia: the finger does not feel the presence of the bone on its moving into the inguinal canal, as it is prevented by musculoaponeurotic elements of the lateral part of the inguinal triangle. Then, leaving the finger, the patient is asked to cough determining a cough jork symptom.

For definition of a location of the hernial protrusion in relation to the spermatic cord it is grasped at the root of the scrotum by the other hand and slightly tightens, and the patient is asked to cough. If there is an oblique hernia, the protrusion occurs outside of the spermatic cord, and if a direct one, the finger easily goes on following to a hernia. If a hernia is oblique, the extended course repeats the direction of the spermatic cord, and if a direct one, the finger is referred directly, penetrating into the inguinal space. Sometimes it is possible to palpate pulsation of the artery epigastrica inferior. In a direct hernia the pulsation of the artery is felt outside from the examining finger, and in oblique one — inside. In cases of very acute extension of the hernial ring with a finger introduced into the inguinal canal, it is possible to palpate the defect of its posterior wall, and sometimes to pass into the abdominal cavity. At a wide hernial ring in some patients a soft, dense fold, enlarged on exertion of the patient is palpated the wall of the large intestine (I sign of Lototsky) in the inguinal canal. In slided cystoceles, while examining the hernial ring the patient can have vesical tenesmus (II sign of Lototsky).

Differential diagnosis should be carried out with the femoral hernias, hydrocele, periorchitis, cyst of the spermatic cord and Nuck's canal, varicocele, lipomas of the spermatic cord and area of the external inguinal ring, inguinal lymphadenitis, tumours of the root of the scrotum, the large pudendal lip and the abdominal wall in the area of the inguinal canal, tuberculous abscess and some other diseases.

Treatment is operative. The general principles and sequence of stages of operation do not differ from those in other localizations of hernias. The peculiaritis is the treatment of the hernial sac in congenital oblique inguinal hernia. In this case, having isolated a sac, they dissect its wall along its length, stitch with a purse-string suture from the middle part and ligate the sac at the neck at the level of its adhesion with the preperitoneal cellular tissue ("by a fatty plug of Pirogov"). The superfluous, free edges of the hernial sac are cut off, and its stump is turned by the back side around of the testicle and spermatic cord, as at Winkelmann's operation in hydrocele.

The main differences of operations concerning inguinal hernias consist of the features of closing of the hernial ring. It is offered more than 200 ways of restoration and reconstruction of the abdominal wall in inguinal hernias. Theoretically they can be divided into the methods directed mainly on the strengthening of the anterior or posterior wall of the inguinal canal. Choosing a method of hernioplasty, it is necessary to remember that in genesis of the formation of inguinal hernias not so much weakness of aponeurosis of the *musculus obliquus externus abdominis* and expansion of the external inguinal ring, as the weakening of the posterior wall, i. e. the transversal fascia, and the enlargement of a diameter of a deep ring of the inguinal canal play the leading role. The aim of this stage of operation is not only the elimination of defect in the abdominal wall and the restoration of normal anatomic ratio in all its layers, but also the restoration of function of the inguinal canal, its valval mechanism.

To choose a proper management of the operation it is expediently to subdivide all inguinal hernias into simple, transitive and complex one. The oblique inguinal hernias of the small sizes, with a diameter of the internal inguinal ring no more than 2 cm belong to simple ones. Oblique inguinal hernias with a diameter of the internal inguinal ring no more than 3.5 cm belong to transitive ones. Direct hernias, recurrent and oblique inguinal hernias of large and very large sizes, with a diameter of the internal inguinal ring more than 3.5 cm belong to complex ones. Complex inguinal hernias make about 42% from all inguinal hernias and in patients of advanced age 67% (Yu. A. Nesterenko, 1982). The plasty of the external wall of the inguinal canal can be made only in simple hernias. If there is an extension of the inner opening of the inguinal canal during an operation, it

is necessary to reduce the normal sizes by applying sutures on the edges of the transversal fascia from the medial side. Only after that the plasty of the external wall of the inguinal canal is made. For strengthening of the external wall many ways of operation are offered: Martynov's, Girard's, Spasokukotsky's, Kimbarovsky's etc.

In transitive and complex hernias it is necessary to strengthen the posterior wall of the inguinal canal. The most widespread in practice "the classical" way of strengthening of the posterior wall of the inguinal canal was offered in 1888 by Bassini. The posterior wall of the inguinal canal becomes stronger with suture appliance to the inguinal ligament of the internal oblique and transversal muscles of the abdomen behind the spermatic cord. The anterior wall of the inguinal canal is formed by means of aponeurosis duplicature of the *musculus obliquus externus abdominis*. If in transitive hernias it is enough to have a monolayer plasty of the posterior wall with the formation of the normal sizes of the internal inguinal ring, in complex hernias it is necessary to perform plastic operations directed at complete reconstruction of the inguinal canal.

The most physiological method, which corresponds to modern principles of operations for complex hernias, is N. I. Kukudzhanov's technique (1938). In case of such operation the strengthening of the posterior wall in a medial part of the inguinal space is made by means of suture appliance of the external edge of a sheath of the musculus rectus abdominis to Kuper's ligament with 3–4 sutures, but in a lateral part at the expense of the creation of a duplicature of the transversal fascia (1st way) or suture appliance after a dissection of hyperinflate superfluous sites (2nd way). The plasty of the posterior wall is finished by the restoration of the normal sizes of the internal inguinal ring. The strengthening of the anterior wall is carried out by the creation of a duplicature of aponeurosis of the musculus obliquus externus abdominis. The basic advantage of this operation is as follows: besides the mechanical strengthening of the posterior wall of the inguinal canal, the lowering of the inferior edge of the lateral muscles of the abdomen is reached, due to which the inguinal space is narrowed and the valval function of the inguinal canal is restored.

A similar, technically more difficult method of the inguinal canal plasty was offered in 1939 by Guanar and was modified by Mc-Wey in 1941. It consists in the strengthening of the posterior wall of the inguinal canal by way of suture appliance of the superior edge of transversal fascia to Kuper's ligament along the wall length of the inguinal space. The method provides release of femoral vessels, that makes it more complex and more dangerous.

For last 20 years a method of Shouldice is applied with success. With this method of hernioplasty the strengthening of the posterior wall of the

inguinal canal is made at the expense of the creation of a duplicature of the transversal fascia along the wall length of the inguinal space with continuous multi-raw suture by a metal-like thread. The author attaches great importance to the strengthening of the internal inguinal ring. To carry out denudation better before the reconstruction he recommends to cross fibers of *musculus cremaster* above the spermatic cord. The plasty is finished by the creation of a duplicature of aponeurosis of the *musculus obliquus externus abdominis*.

The plasty of the posterior wall of the inguinal canal with the help of "classical" technique of Bassini is performed by the method described above, as it provides only its mechanical strengthening sutured muscles atrophyed and are cicatrially regenerated, their valval function doesn't not restore. For these reasons the operation of Bassini loses the supporters ever more.

Femoral Hernias

Femoral hernias are those which leave through the femoral canal. According to the frequency of their formation they make up 5–6% from all abdominal hernias and occur mainly in women (4:1–5:1).

The femoral canal is a short interspace with the length of 1–2 cm of a triangular form, connecting the internal femoral ring with the oval fossa on the femur. Its walls are: in front — a crescent-shape edge of the broad fascia of the femur, from behind — a pectineal fascia, from outside — the femoral vein and its fascial sheath. There is no femoral canal in healthy people; it is formed while the formation of the femoral hernia. The internal aperture of the femoral canal (internal femoral ring) is a part of the vascular lacuna and is medially from the femoral vessels. The internal femoral ring is limited in front by the inguinal ligament, from the middle — by the lacunar ligament, from behind — by the pectineal ligament, from the outside — by the femoral vein and its sheath. On the part of the peritoneum the femoral fossa corresponds to the femoral ring, which is located under the inguinal ligament in the same vertical as the internal inguinal fossa. The external aperture of the femoral canal is the oval fossa on the femur limited by a crescent-shaped fold of the wide fascia of the femur. Anomaly of the artery epigastrica inferior is of practical importance. If it leaves not from the artery iliaca externa but from the artery obturatoria, it can be located near the posterior-internal surface of the neck of the hernial sac, forming with the femoral vein a vascular ring of the hernia — "a death crown", the damage of which is dangerous by the development of a profuse bleeding.

Typical femoral hernias are formed as a result of the protrusion of the hernial sac through the internal femoral ring. Much less often the hernial

ring is located in the muscle lacuna, in a zone of the femoral vessels or Gimbernat's ligament.

Femoral hernias more often arise at elderly age, from the right side they quite often are sliding. Femoral hernias are inclined to strangulation. Frequently an irreducible hernia develops. The hernial swelling in these hernias, as a rule, being of small sizes, oval or ball-shaped form is placed below the inguinal ligament (in a zone of the oval fossa of the femur). Sometimes it is difficult to find out the protrusion and to determine its relation to the inguinal ligament in obese women. Femoral hernias can be manifested by the pains in the groin irradiating to the leg, accompanied by dyspeptic disorders. With a small protrusion the establishment of the exact diagnosis can be difficult. In rare cases in femoral hernia the edema of the lower extremity can develop as a result of the strangulation of the femoral vein by the hernial protrusion.

Differential diagnosis should be carried out with lymphadenitis, metastases of a malignant tumour in the lymph nodes, tuberculous abscess in spinal tuberculosis, varix dilatation in an oval fossa, aneurysm of femoral vessels, inguinal hernias.

Treatment. It is offered about 100 methods of operative treatment. They use mainly two operative accesses for closing of the hernial ring: femoral and inguinal.

The femoral access is characterized by the approach to the femoral canal laterally from the external aperture. Advantages of this approach are the smaller traumatism and a relative simplicity of operation technique. However, this method shows the greatest number of relapses and does not provide a sufficient closing of the hernial ring. It is carried out in case of femoral approach by the suture appliance of the inguinal ligament in the area of an internal femoral ring to the periosteum of a pubic bone — Lockwood's method and the additional suture appliance of semilunar edge of the large fascia of the femur in the area of the oval fossa to the pectineal fascia (Bassini's method).

In case of inguinal approach they open the inguinal canal, the transversal fascia. and in a retroperitoneal fatty tissue. The neck of the hernial sac is isolated near the internal aperture of the femoral canal. The hernial sac is taken out into a wound and after the opening it is ligated and removed. The hernial ring is closed by the suture appliance of the inguinal ligament with the Kuper's ligament (Ruggy's method) or the edge of the internal oblique and transversal muscles with the periosteum of the pubic bone and Kuper's ligament (Parlavecco's method). Then the integrity of the inguinal canal is restored. Technically the inguinal approach is more difficult, but it provides more reliable closing of the hernial hilus.

Hernias of the Linea Alba

The white line of the abdomen is formed at the expense of crossed tendinous fascicles of three steams of wide muscles of the abdomen. It lasts from the xiphoid process of the breast bone down to the symphysis, wider above the navel. In the white line of the abdomen there are fissural interspaces, through which the vessels and nerves pass, connecting preperitoneal fatty tissue with hypodermic one. These clefts are places of the hernial exit.

Mainly there are hernias of supraumbilical part of the white line of the abdomen, so-called epigastric, but can arise paraumbilical. Originally the site of the preperitoneal fatty tissue penetrates into the defect of aponeurosis; a so-called "preperitoneal lipoma" is formed. The fatty tissue can contain the nervous branches, diverging from the umbilico-hepatic ligament, which is in turn connected with a solar plexus. The prelum of fatty tissue can cause the occurrence of the pronounced pain syndrome, sometimes reminding a clinical picture of abdominal organs diseases: ulcer of the stomach, cholecystitis etc. Then the cleft in the white line of the abdomen enlarges gradually, and the parietal leaf of the peritoneum penetrates there and a hernia is formed.

Hernias of the linea alba are seldom large; their palpation is usually painless. In a reducible hernia, it is possible to determine the defect of the white line of the abdomen. In case of muscle tension of the abdominal prelum the presence of diastase of *musculus recti abdominis,* which frequently occurs in epigastric hernias is determined. Definition of size of muscles divergence is made at a middle distance between the xiphoid process and the navel. There distinguish three stages of diastasis of the *musculus rectui abdominis:* I stage — up to 5–7 cm, I stage — more than 5–7 cm, III stage — the large diastases combined with hanging abdomen.

Differential diagnosis is carried out for the diseases of the abdominal organs which can have a similar clinical picture.

Treatment is operative. Aponeurosis duplicature is made for closing of the hernial ring (Yukhelson's method), or they reformed the white line from the xiphoid process up to the navel by M. I. Napalkov's method, creating more favorable conditions of antiaction of *musculus recti abdominis* to the wide (lateral) muscles traction.

The henial ring is the umbilical ring in umbilical and paraumbilical hernias.

The navel is involved with a cicatrix, which forms at the place of the umbilical ring. This ring is an aperture limited by aponeurotic fibers of the white line of the abdomen, through which during intrauterine development two umbilical arteries, the umbilical vein and the urinary duct pass. Then

the vessels and the duct are transformed into the ligaments. After the umbilical cord falling-away the umbilical ring is tightened by a cicatrical tissue (a so-called umbilical cicatrix). The layers forming a navel consist of the skin, connected with a cicatrical tissue of the umbilical fascia, the peritoneum. There is neither hypodermic cellular tissue, nor preperitoneal one here.

One distinguishes:

1) embryonal umbilical hernias;

2) umbilical hernias in children;

3) umbilical hernias in adults.

Embryonal umbilical hernias are a consequence of delay of formation of the anterior abdominal wall of the fetus. In these cases, the hernial sac is an amniotic membrane of the umbilical cord with a malformed internal membrane, covering it from the middle part (conductor of the peritoneum). Embryonal umbilical hernias belong to inherent defects of the development.

Umbilical hernias in children is a result of insufficient closing of the umbilical ring during its cicatrization, so the child is born with a defect of the anterior abdominal wall, in the area of which a hernial protrusion, as a rule, develops.

Umbilical hernias in adults make 3–5% from all external hernias of the abdomen. They arise mainly in women after 30 years having a hypersthenic type of the body build. The important etiological factors of umbilical hernias are pregnancies, especially repeated, and obesity.

The sizes of umbilical hernias can vary from several centimeters in diameter up to huge ones. The hernial ring may be narrow, even with large hernia, or reach considerable sizes. Frequently the umbilical hernias are multichamber; they are inclined to the incarceration. There are pains in the area of the protrusion, constipations, sometimes, nausea and even vomiting in the large hernias. The pain syndrome and dyspeptic disorders are especially pronounced in irreducible hernias.

Treatment of umbilical hernias is operative. In large hernias it is necessary to remember that the fast reposition of contents into the abdominal cavity can result in considerable increase of intra-abdominal pressure and can cause disorders of respiration and cardiovascular activity. In these cases the preliminary special preparation for surgery is necessary by bandaging of the abdomen and gradual increase of intra-abdominal pressure. The closing of the hernial ring is carried out by the creation of aponeurosis duplicature in a transversal direction (Meyo — Stone's method) or in a longitudinal direction (Sapejko's method), or in case of small defect by a suture appliance with its purse-string suture (way of Lexer's method), that prevents a relapse to a lesser degree. In case of large defects, the methods of allo-, homo- or autoplasty are applied.

Rare Forms of Hernias

Hernias of the xiphoid process (xiphoid), lateral hernias of the abdomen, lumbar, obturator, ischiadic, perineal and others belong to rare forms of hernias.

A hernia of the xiphoid process occurs seldom. It arises at the presence of inherent aperture in the xiphoid process of the breastbone. Its basic signs are pain in the area of the xiphoid process, the presence of protrusion there, and then it is possible to palpate an aperture after its reduction.

Treatment consists in dissection of the hernial sac, removal of the xiphoid process and plasty of defect of the anterior abdominal wall.

Lateral hernias of the abdomen arise on its lateral surface as a complication of casual traumas, operations or disorders of embryogenesis. In case of malformation of any muscles of the abdominal wall, there are inherent hernias of the abdomen, which can be revealed clinically at any age.

One distinguishes three kinds of lateral abdominal hernias: hernias of sheath of the *musculus recti abdominis,* a hernia of Spieghel's line and hernia of the muscular lateral part of the abdominal wall arising due to its development delay.

Hernias of sheath of the musculus recti abdominis occur more often in the lower part of the abdomen, where there is no posterior wall of the sheath, and also in case of mechanical damages of the *musculus recti abdominis.*

Hernias of Spieghel's line most frequently arise on the border of the medium and lower third of sheath of the *musculus recti abdominis,* in the area of transition of a muscular part of the transversal muscle of the abdomen into the tendinous stretching. They are usually located along the anterior line of Duglas, extending from a distal part of the white line, up to long extensors of the back.

As a rule obese women who delivered many times and those who were engaged in physical labour, have lateral hernias of the abdomen. The basic signs of a lateral hernia of the abdomen are pain and hernial protrusion of the various sizes depending on the width of the hernial ring. The lateral abdominal hernias are more often interstitial and preperitoneal; rarely hypodermic ones. Therefore, their diagnosis, especially in obese patients, is difficult. The hernial hilus can be of various sizes, their edges, as a rule, are a dense immobile fibrous ring that promotes frequent incarcerations of these hernias. Contents of the hernial sac can be the small intestine, omentum, caecum and colon. Frequently it is a partial enterocele of the intestine in lateral hernias.

Treatment of lateral abdominal hernias is only operative. In small hernias after removal of the hernial sac, the hernial ring is sewn up level-by-level applying sutures on the transversal and oblique muscles, and also on aponeurosis of the *musculus obliquus externus abdominis*. Plastic methods are applied in large hernias.

Lumbar hernias can be formed in the area of Pty's triangle (hernia lumbalis inferior), Grynfeltt — Lesshgaft's (hernia lumbalis superior) and in aponeurotic clefts.

Pty's triangle is formed behind by the external edge of the wide muscle of the back, in front — by the posterior edge of the musculus obliquus externus abdominis, from below — by crest of the ileal bone. The fundus of the triangle is the internal oblique and transversal muscle of the abdomen located more deeply. In this place, they form a rather thin muscular plate, which is weakened by perforating it with iliac-epigastric nerve and the 4th lumbar artery and vein.

The Grynfeltt — Lesgaft's space is limited by the lower edge of the *musculus serratus posterior inferior,* the medial edge of the *musculus obliquus internus abdominis,* by the XII rib and the lateral edge of an extensor of the back. The fundus of the space is aponeurosis of the transversal muscle of the abdomen, through which the twelfth intercostal nerve, artery and vein pass. From behind this interspace is closed by the wide muscle of the back.

The lumbar hernias are inherent or acquired after repeated servere labors, damages of the lumbar area, purulent paranephritis, etc. They occur more often in women. Upper lumbar hernias are located under the wide muscle of the back or under *musculus obliquus externus abdominis*. Lower lumbar hernias are located subcutaneously and that's why are diagnosed more easily. The basic signs are pains in the lumbar area and occurrence of the protrusion, especially on physical exertion. Contents of lumbar hernias are more often loops of the small intestine, the omentum, less often the caecum. Their incarceration belongs to the complications of lumbar hernias. The extensive strangulations of the small intestine in the lower lumbar hernia (O. S. Makhmurov, 1955) are described.

Treatment of lumbar hernias is operative. The closing of the hernial ring is made by a suture appliance of intermuscular defect in the lumbar area.

Obturator hernias occur through the obturator canal, which passes under the pubic bone. Its inlet is located in the cavity of the small pelvis, and the outlet — in the bed of adducting muscles of the femur. The outlet opening of the canal is projected at 2–2.5 cm out from the pubic tubercle and at 1.5–2 cm lower from the inguinal ligament. It is covered with the pectineal muscle. The canal is formed: outside and from above — by the osteal obturator sulcus of the pubic bone, and inside and from below

— by a superexternal edge of *membrana obturatoria* with the beginning from it by the internal and external obturator muscles. The obturator vessels and the nerve pass in the canal, surrounded with the cellular tissue.

V. S. Mayat marks two forms of obturator hernias: postspinal, locating in the obturator canal, when the hernia at exit from the canal is lying under the pectineal muscle and anterospinal, when the hernial sac passes between pectineal and adducting muscles of the femur, being localized either under the wide fascia or in the hypodermic fatty tissue. The contents of the hernial sac is more often the small intestine, along or together with the omentum.

Obturator hernias occur mainly in women of the elderly age. Their diagnosis is very difficult, especially if a hernial protrusion does not fall outside the limits of the obturator canal or is covered with a potent pectineal muscle. As a rule, the patients suffer from the pains in the inguinal area and internal surface of the femur, intensified at walking. Obturator hernias are inclined to incarceration. The strangulation of an obturator hernia is manifested by acute pains in the inguinal area according to the localization of the hernia. The pains intensify at coughing and walking, irradiate to the internal surface of the femur (Howship — Romberg's sign). The leg is given and is a little bent in coxofemoral and knee joints. Abduction and rotation outside of the femur are acutely painful (Treves's sign). In large hernias it is possible to palpate a spherical tender formation of an elastic consistency locating below a horizontal branch of the pubic bone under the pectineal and long adducting muscles.

The patients are usually operated in case of incarceration occurrence of obturator hernia manifested by acute intestinal obstruction. During operation the intestinal loop penetrating into an internal aperture of the obturator canal is usually revealed. A section of a restraining ring of the hernial hilus is made by incision of an obturator membrane to the bottom. Hernial content is released by the blunt way laterally from the abdominal cavity. After the liberation of a strangulated loop of the intestine they monitor its condition; if it is necessary, the resection of damaged sites is made too. With small sizes of the hernial hilus they are closed by a simple method of invagination (Kocher): the hernial sac is turned inside out the back of the abdominal cavity and is ligated at the neck. A "plug", arising as a result, closes the hernial hilus. If it is necessary, in case of large hernias hernioplasty is performed from the retropubic preperitoneal approach by Chatle (1920), Henry (1936). After dissection of the skin and the anterior leaf of the sheath of the *musculus recti abdominis,* parietal peritoneum is separated and moved upwards. The hernial sac is removed, ligated and cut off. The internal aperture of the obturator canal is closed with the sutures, covering the edges of cruses of the internal obturator muscle.

Ischiadic hernias passes through the posterior surface of the pelvis through the large or small ischiadic apertures. There distinguish three types of ischiadic hernias: hernia passing through the piriform muscle (hernia suprapiriformis), under the piriform muscle (hernia infrapiriformis) and through the small ischiadic aperture (hernia spino-tuberosa). More often, there are hernias passing through small ischiadic aperture.

Ischiadic hernias are revealed very seldom. The elderly women with a wide pelvis and large ischiadic apertures mainly suffer from them. Following the data of the literature, the ischiadic hernias occur more often to the right. It is rather difficult to reveal an ischiadic hernia. The hernial sac is usually covered with a thick layer of tissues consisting of the gluteal muscle and subcutaneous fatty tissue, and only sometimes, with its large sizes, it can be lowered on the posterior surface of the femur along the sciatic nerve. The patients complain of pain in the gluteal area, intensified on movements, the occurrence of a protrusion is very typical. In some cases an ischiadic hernia is wrongly considered as a gluteal abscess.

The *treatment* is operative, and consists in removal of the hernial sac through the incision in the gluteal area. Walstein's approach is usually applied: the dermal integuments along the line connecting the coccygeal bone and large ischiadic tuber are widely dissected, the muscles are bluntly stratified and the hernial sac is removed. The operation has large technical difficulties in connection with the danger of damage of the gluteal vessels and the sciatic nerve. The closing of the hernial hilus consists in the piriform muscle anchoring to the small gluteal muscle. Sometimes a fascial plasty is applied.

The most often complication of an ischiadic hernia is its strangulation. A strangulated ischiadic hernia is recommended to operate with in a combined way, beginning with laparotomy, and then to pass to approach in the gluteal area.

Perineal hernias are rare leave in the area of the perineum through the urinogenital septum or between the fibers of the *musculus levator ani,* or between it and the other muscles of the perineum.

There distinguish the anterior and posterior perineal hernias. The border between them is arched convex overhead line connecting the ischiadic tubers. Thus, the anterior perineal hernias pass through the urinogenital area, and the posterior ones — in the anal area of the perineum. In the anterior perineal hernias, the hernial sac is connected with the anterior Douglas' space and passes through the perineum in the area of the large pudendal lips in women, between the scrotal root and the anal aperture in men. The anterior perineal hernias pass through the urinogenital diaphragm. In posterior hernias the hernial sac is connected with the posterior Douglas' space and passes through the perineum into the ischiorectal fossa. The back peri-

neal hernia can pass between the muscle lifting the anus, both iliococcygeal and coccygeal muscles, and also through clefts in *m. levator ani*. The urinary bladder or its diverticulum, female internal sexual organs can be contents of perineal hernias, the intestine and the omentum are more often in the posterior hernias.

The basic signs of disease are the pains and the occurrence of the protrusion in the area of the perineum. The protrusion is enlarged on tension, especially in a squat position. In women, it is necessary to differentiate anterior perineal hernias lowering in the large pudendal lip from an inflammation or cyst of Bartholin's glands. The presence of the posterior perineal hernia can be an occasion for the erroneous diagnosis of a paraproctitis.

Treatment of perineal hernias is operative. Approach through the perineum, according to hernia localization is usually applied. The hernial hilus plasty consists in closing the defect of a musculoaponeurotic layer of the perineum. With hernia strangulation and absence of exact preoperative diagnosis, the operation is begun with laparotomy.

Internal Abdominal Hernias

Internal abdominal hernias are the protrusion of the abdominal organs into the various folds and pockets of the peritoneum, the intestinal mesenteries, and also in the thoracal cavity through the natural or acquired apertures and clefts of the diaphragm.

They distinguish the following:

a) retroperitoneal internal hernias in the area of the duodenal intestine and the caecum, the intersigmoid fossa and in the peritoneal diverticula;

b) intraperitoneal — in the apertures of the large and the small omentums, mesentery of the small intestine and the colon, the mesentery of the vermiform process, the gastrocolic ligament and the falciform ligament of the liver, in pockets and apertures of the wide uterine ligament; hernias of Winslow 's aperture and Douglas' pouch;

c) preperitoneal (supravesical, paravesical, etc.);

d) diaphragmic.

Internal hernias in the area of *recessus duodenalis superior* are called Treitz hernias, or periduodenal hernias. They make 29.9–40% of all internal abdominal hernias (D. F. Skripnichenko, 1974). They distinguish a right and left-hand periduodenal hernia. In right-hand Treitz hernias the hernial sac is located in the right half of the abdominal cavity, below the transversal colon, in left-hand — to the left from Treitz ligament behind the abdomen. Right-hand hernias occur more often. The hernial sac of these hernias can reach the vast sizes, containing the most part of the small intestine.

Lecture 12

DISEASES OF THE RECTUM

Diseases of the rectum are haemorroids, cancer, sarcoma, melanoma etc.

HAEMORRHOIDS

Haemorrhoids are one of the most wide-spread diseases of a man. More than 10% of all adult population suffer from haemorrhoids. These patients make 15–28% from general number of the proctologic patients. Haemorrhoids appear to be in 3–4 times more common in men than in women; age of the patients is 30–50 years.

According to modern representations, the basis of disease is the illness of cavernous bodies of the submucous layer of the distal part of the rectum. A vertical position of a man, dietary habits of alcohol abuse, sedentary work, and hypodynamia — are the main factors, determining wide spreading of haemorrhoids in a modern society. A certain importance in disease rate have the regular violation of hygienic rules of the anal area care; it is noticed that in the countries where this care is determined by religion the morbidity is lower.

Healthy people have the vestigial cavernous bodies in distal parts of the rectum as well. Thus, a symptom-complex of haemorrhoids is connected not with the presence of cavernous bodies but with their illness. The cavernous bodies are posed in the area of the basis of Morgagni's columns diffusely or more often are grouped basically in three zones: on left lateral, right anteriolateral and right posteriolateral walls of the anal canal (according to a position of 3, 7 and 11 o'clock of a clockdial, in a supine position).

Works of P. V. Starkov (1912), L. L. Kapuller (1969) have proved that haemorrhoids are not the varicosely dilated veins, but hyperplastic changes of the cavernous tissue of the rectum caused by amplified inflow of the arterial blood in the cavernous bodies by arteries and its complicated outflow by veins.

Classification. Among a variety of classifications the most simple one is division of haemorrhoids into acute and chronic forms. Each of them is subdivided into external, internal and mixed (combined) haemorrhoids.

Depending on severity acute haemorrhoids are divided into mild, moderate and severe degrees:

— the mild degree is characterized by a moderate inflammation, thrombosis, sometimes bleedings; the pains are enough intensive, the stool is not disordered, the prolapsed haemorrhoids are reduced independently. The general somatic signs of disease are absent;

— the moderate degree: edema and inflammation of haemorrhoids of the perianal skin, the prolapsed edematous haemorrhoids are not reduced independently. Signs of thrombosis and bleedings from external or internal haemorrhoids are present. There are constipations, acute painful act of defecation, general rising of the temperature, weakness, malaise, decrease of appetite;

— the severe degree: strangulation of the prolapsed internal haemorrhoids, their necroses or acute purulent inflammation, acute edema of the perianal skin and the anus, general somatic disorders with a high fever, chill, pains, not only in the area of the anus and rectum but also in the abdomen. The independent defecation is impossible.

Three stages are distinguished in process of chronic haemorrhoids.

Haemorrhoids are defined in the area of the anus *at the first stage*, they are enlarged on tension during the act of defecation, reminding "a mulberry". Rectum bleedings are not abandant, more often with filaments of scarlet blood, anal itching, episodes of inflammation arise rather seldom (2–3 times a year). The anal sphincter of the rectum functions well.

At the second stage, during stools there are prolapsed haemorrhoids, quite often with signs of a thrombosis. The prolapsed haemorrhoids are not reduced independently, and for their reduction a manual assistance is necessary. Bleeding from haemorrhoids is regular, quite often rather intensive, though there is no pronounced anemia. Exacerbations are often, almost every month; the anal itching is pronounced and, as a rule, precedes episodes of anal bleedings. Sphincter tonus of the rectum is decreased, but retaining of intestinal contents is sufficient.

At the third stage there are prolapsed haemorrhoids and sites of mucous membrane on mild tension, physical work and vertical position of the body. The prolapsed haemorrhoids are not reduced independently, and there are constant hemorrhages. Severe anemia and long-term anamnesis are typical, when alongside with frequent inflammation or strangulation of haemorrhoids progressing decreases of closing function of an outside sphincter are noted, there is insufficient retaining of gases and intestinal contents, inhibition and deformation of emotional and mental sphere.

Acute and chronic haemorrhoids are divided also into primary and secondary, in etiopathogenesis of the latter the large importance is given to a portal hypertension, tumours and phlebothrombosis in the area of the small pelvis.

Clinical course. The basic complaints of the patient with haemorrhoids are caused by prolapse or protrusion of a dense, painful haemorrhoids from the anus during exacerbation, strangulation of haemorrhoids and signs of their acute inflammation. A rectal bleeding with a scarlet blood from small drops and filaments up to extremely plentiful discharges of blood is a rather frequent sign of haemorrhoids, and the blood usually covers fecal masses, not being admixed with them. These complaints, as a rule, are connected with the act of defecation and are accompanied by feeling of pressure in the rectum, anal itching, which quite often precedes episodes of bleedings. The listed signs especially amplify after consumption of alcohol, plentiful, spicy food, in absence of a neccesary care around the anal area and during hard physical work. The exacerbations of haemorrhoids are provoked also by all factors promoting intensifying of cavernous bodies filling with blood, including alcoholism, sexual life disorders. In chronic haemorrhoids without exacerbation the main sign is bleedings with scarlet blood. Proceeding for a long time, they quite often cause development of severe anemia.

In anamnesis of the patients with haemorrhoids a certain sequence of occurrence of the listed complaints is usually noted. So, the anal itching occurs in the earliest stages of disease. Subsequently the patients begin to mark a protrusion and prolapse of haemorrhoids, quite often with predilection to their inflammation or strangulation. The bleedings, as a rule, occur later, after the month-and-years-long disease course. They mark transition of disease into a new phase and testify to the beginning of morphological changes in the structure of rectum cavernous bodies. Bleeding is frequently persistent, long-term and intensive, sometimes causing severe anemia. In anamnesis hereditary predisposition, predilection for taking acute food, alcohol, hard physical work is frequently traced. It is necessary also to take into consideration diseases causing secondary haemorrhoids (portal hypertension, pelvic tumours etc.).

During inspection of the patient, which usually begins with examination of the anal area, enlarged or condensed inflamed haemorrhoids (at 3, 7 and 11 o'clock according to a clockdial) are found out. In some patients haemorrhoids are not clearly grouped, that testifies to diffuse character of the rectal cavernous bodies. At the level of Hilton's white line haemorrhoids are devided, determining border of internal and external ones. Internal haemorrhoids which remind a "mullberry" easily bleed. During their extensive enlargement and tension of the patient the haemorrhoids get out and

even prolapse. Prolapse of the mucosa characterizes subsequent, later phases of disease. During palpating the anal area it is possible to define haemorrhoids, which during exacerbation become dense and painful. Tonus decrease of the rectum closing apparatus is rather characteristic in long-term haemorrhoids that promotes the protrusion and prolapse of haemorrhoids, which becomes constant even during a slight physical exercise, vertical position or walking. Alongside with palpation rectoromanoscopy is obligatory, allowing to estimate the forms and stages of the pathological process and to exclude other rectal diseases. So, a majority of late diagnosing of rectal cancer, delayed detection of a tumour are consequences of pathological changes during superficial inspection for haemorrhoids. In acute haemorrhoids the application of tool research is undesirable, as alongside with pronounced painfulness of procedure an intensive bleeding may occur.

Diagnosis usually does not represent considerable difficulties. It is necessary to specify character of the process (primary, secondary), degree of rectum disturbances, severity, kind and stage of disease.

Differential diagnosis of haemorrhoids is carried out first of all with the prolapse of the rectum, in which the diverticulum from the anus always has a circular character, while in haemorrhoids only one or several areas of the mucous rectum get out or prolapse. In the acute phase of disease the differential diagnosis should be carried out with anal cracks, paraproctitis and rectal cancer, taking into condition, that rectal bleedings in cancer can never be with scarlet arterial blood. It is also necessary to remember an opportunity of combination of several diseases, when against a background of long-term haemorrhoid course other rival diseases (proctitis, polyps, and cancer) take place.

Treatment. The conservative treatment of haemorrhoids is applied in the acute phase of disease. It includes a sparing diet; alcohol, spicy, greasy and fried food are forbidden; local application of heat (sitting warm baths with antiseptics); local use of ointments and suppositories having haemostatic, antiinflammatory, antibacterial and analgetic action (for example, "Anesthesol", "Betiol", "Neoanusolum"), general antiinflammatory, antibacterial, analgetic treatment in a combination with sedatives. The application of narcotic agents in haemorrhoids is undesirable, as drugs of this group provoke constipations. The operative treatment in these cases is expedient not earlier than after 5–6 days of conservative therapy.

The operative treatment of haemorrhoids is necessary during frequent inflammations, intensive and persistent bleedings, prolapse and strangulation of haemorrhoids. Enlarged haemorrhoids without clinical signs are not the indication to operation. Functional results of the operations, performed in

clinically pronounced haemorrhoids, are always better than in asymptomatic or slightly-symptomatic disease.

The operation concerning haemorrhoids consists in removing of the rectal cavernous bodies. The ligation of haemorrhoids pathogenically is not proved. Therefore the most proved is haemorrhoidectomy of Parks, Milligan — Morgan and their modifications, which provide the minimal relapses rate. The operation of Milligan — Morgan consists of a successive dissection of all three groups of cavernous bodies (at 3, 7 and 11 o'clock) with the ligation of the vascular pedicle and applying of a primary indistinct suture on the operative wound.

Introduction of sclerosing substances as well as haemorrhoids ligation are carried out mainly in connection with gravity of a patient's condition due to severe accompanying diseases. The method of sclerosing therapy consists in the following. With the help of the anoscope or the rectal speculum internal hemorrhoids are exposed. They inject 1–2 ml of solution (alcohol-novocainic mixture, admixture of carbolic acid with novocain and sunflower-seed oil and so on) in it, causing damage of intima of the vessels, aseptic clottage with a subsequent occurence of thrombus and obliteration of the vessels lumen. Unfortunately, this method is accompanied by the greater relapses rate than removal and ligation of haemorrhoids.

One more non operative way of treatment of haemorrhoids is possible (it is used in the patients with a high degree of operative risk in connection with severe accompanying diseases of internal organs) — ligation of haemorrhoids by rubber or latex rings. In comparison with a surgical method this method is less radical and gives greater number of relapses.

The surgical treatment of haemorrhoids is contraindicated in portal hypertension and hypertension of the 3rd stage. Complications of haemorrhoids are clottage and strangulation of haemorrhoids.

Thrombosis of an external hemorrhoid node more often arises after malnutrition, physical exercises, and stress. There are acute pains in the area of the anus intensifying during any physical exercises, cough; sensation of a foreign body. During examination in the area of the anus cyanotic haemorrhoids are very painful on palpation. The rectal exam does not give the additional essential information.

Treatment is more often conservative: a diet without spicy dishes and alcohol; laxatives, antiinflammatory preparations (Butadion, Rheopyrin, Acetylsalicylic acid, locally — lead water) are expedient. Since the 3rd–4th day — sitting warm baths with a weak solution of potassium permanganate are used. A more expedient surgical treatment is a section of thrombosed haemorrhoids, removal of thrombotic masses. The treat-

ment allows to reduce duration of patient's disability, to eliminate the pain syndrome.

The strangulation of internal haemorrhoids is provoked usually by a diet, physical exercise nonobservance, and constipations. The internal haemorrhoids prolapse and strangulate in the anus. A clottage of strangulated haemorrhoids and their necrosis may occur. The patients have acute aching pains in the anus and sensation of a foreign body. On examination along all perimeters of the anal canal prolapsed dark cherry haemorrhoids are visible. In necrosis of the mucosa bleeding and paraproctitis are possible. The rectal exam in the acute period is impossible.

Treatment. The laxative diet, antiinflammatory agents, lead water, presacral blockade with 0.25% novocain solution renders good effect, then the patient is put to bed with a raised foot end. In such a position some patients have haemorrhoids reduced. After acute phenomena, subside the surgical treatment is indicated to the patients in accordance with the plan.

RECTAL CANCER

Rectal cancer rate is 4–6% of all malignant tumours of a man, and in many industrial countries rectal cancer morbidity increase. They believe that genetic factors play an insignificant role in etiology of this disease. Much more influence is rendered to nutritional factors, determining bacterial flora of fecal masses and periodicity of defecation. A diet with prevalence of easy-assimilable products and high-caloric fats contributes to slow passage of intestinal contents, dysbacteriosis and, hence, to longer contact of the rectal mucosa to oncogenic substances containing in foodstuff. An adverse influence is rendered also by decrease of physical activity, hypodynamia that negatively influence the gastrointestinal tract motility, provoking a delay of defecation and constipations.

Etiology. In etiology of malignant neoplasms of the rectum, as well as a number of other localizations of tumours, a great importance is given to precancerous diseases (polyps, villiferous tumours, proctitis, nonspecific ulcerative colitis, Crohn's disease, anal fissures, chronic paraproctitis, haemorrhoids etc.), on background of which the foci of tumoral growth occur. The rectal cancer rate is increased among the men with homo- and heterosexual behaviour, the connection of cancer of the anal canal with previous sexually transmitted diseases (herpetic viruses, papillomaviruses of a man, etc.) is observed.

Pathological anatomy. As a rule tumours occur in the ampullar area (up to 70%), less often — in the supraampular (15–20%) and the anal ones (3–6%). The exophitic and endophitic forms of a tumour have an almost equal rate. Exophitic tumours at a certain stage of development ulcerate, expose

to disintegration, getting a plate-like. Endophytic tumours, spreading deep into the intestinal wall, cause deformation and narrowing of the intestinal lumen. The cancer invasion spread along the intestinal circle considerably pass ahead growth of a tumour on length of the intestine, that is connected to features of blood vessels architectonics of this organ.

Intramural invasion of cancer cells is defined on an extent up to 5 cm and more from a tumour's border.

The sizes of a tumour depending on the stage of its development can be from several millimeters up to 5–10 cm in diameter that has the large importance in definition of the prognosis of disease. Tumours with a diameter up to 2 cm and invading not deeper than the submucous layer belong to early (initial) stages. Glandular polyps and villiferous tumours also belong to the initial forms of rectal cancer, if malignisation areas are found out in them, but without involving into the muscular layer of the intestinal wall. Multiple tumours, as synchronic, and metachronic, are found out in the rectum in 1–2% of the patients with malignant neoplasms of this organ.

According to histological structure among cancer tumours of the rectum adenocarcinomas (up to 90–95%) with various degree of differentiation of their cellular elements prevail; the mucous and solid cancer is observed much less often. Planocellular (epidermoid) cancer makes about 2–4%, and these tumours are usually located on the border of the ampullar area and the anal canal.

Metastatic spreading of rectal cancer occurs by lymphogenous, hematogenous and implantative ways. In supraampular localization of a tumour its metastatic spreading goes on in paraaortal lymph nodes along a course of the superior rectal and inferior mesenteric artery. In tumours of the ampullar area, besides of these ways of metastatic spreading, there are also metastases along the medial rectal artery, in sacral lymph nodes and along the internal and external ileal arteries. With anal cancer metastatic spreading occurs in the sacral lymph nodes, along the common ileal artery, in the femoral and inguinal lymph nodes. Most frequently hematogenous metastases are found out in the cerebrum, bones of the skeleton, the liver. Implantative metastases are possible owing to direct carrying of tumoral cells along the peritoneum (carcinomatosis of the peritoneum) or surface of the mucosa.

Classification. According to the accepted clinical staged classification of the process four stages of rectal cancer are differentiated:

I stage:

a) small, precisely limited, quite mobile tumour or tumoral ulcer localized on a small area of the mucosa and submucosa;

b) metastases in regional lymph nodes are not present.

II stage:

a) a tumour or an ulcer occupies a half of the intestinal circle, without leaving its limits; metastases are absent;

b) a tumour of the same or smaller size, but there are single, mobile metastases in regional lymph nodes.

III stage:

a) a tumour occupies more than a semicircle of the intestine, invades all the wall, united with adjacent tissues or organs;

b) tumour of any size with multiple metastases into regional lymph nodes.

IV stage:

a) the extensive breaking up motionless tumour involving adjacent organs and tissues with metastases into the regional lymph nodes or remote metastases.

The characteristic of prevalence of cancer rectum according to *TNM system* is made by the following criteria:

T1 — a tumour occupying 1/3 or less of the rectal circle which does not sprout the muscular layer;

T2 — a tumour occupying more than 1/3, but no more than 1/2 length of the circle involving in the muscular layer, but it does not disturb a mobility of the intestine;

T3 — a tumour occupying more than half of the intestinal circle, distorbs mobility, but not extending to the adjacent organs and tissues;

T4 — a tumour occupying all the circle of the intestine or extending on the adjacent structures.

The condition of the lymph apparatus (N) in rectal cancer can finally be estimated only after histological research of the removed organ, therefore instead of the digital characteristic of sign N a designation N_X is used which in subsequent is supplemented by N1 — on detection of metastases and N0 — at their absence. The presence of the remote metastases is designated M1, and their absence — M0.

Besides of the listed above symbols, the criterion P is introduced in classification, reflects depth of germination of a tumour by results of histological research that is designated additionally in digits at this symbol:

P1 — cancer infiltrating only the mucosa;

P2 — cancer infiltrating the submucosa without affection of the muscular layer;

P3 — cancer infiltrating the muscular layer without germination of the serous layer;

P4 — cancer involving the serous layer of the intestine and leaving limits of the organ.

Other additional histological criterion is the parameter G, describing a degree of differentiation of tumoral cells that is designated by digital symbols:

G1 — adenocarcinoma with a high differentiation of cells;

G2 — adenocarcinoma with a moderate degree of tumoral cells differentiation;

G3 — anaplastic forms of cancer.

Clinical course. Early stages of neoplasm development proceed asymptomatically, and the tumour quite often is found out casually during proctologic or routine inspections. Later on, in the process of a tumor growth, the local (depending on the size of a tumor) and the general (depending on intoxication and complications) signs occur. One of initial symptoms of rectal cancer is pathological discharges (slime, blood) from the anus. At first mucus discharges, sometimes rather plentiful occur, and then they can became mucopurulent. Later in mucus discharges of blood streaks, and quite often blood mixed with fecal masses are found. Blood is usually dark; less often it is scarlet and skanty. The profuse rectal bleedings in rectal cancer take place seldom. In the exophitic forms of cancer pathological discharges arise earlier than in the endophitic one and usually they are more plentiful. Stools are often irregular, diarrheas alternate with constipations, and quite often painful tenesmi occur especially in supraampular localization of tumour. The patients do not have feeling of satisfaction after defecation and complain of sensation of a foreign body in the rectum. The act of defecation can be repeated, with "sheep's" stool, or the feces can be of a ribbon-like form, constipations usually have persistent character. With anal cancer pains in the area of the anal canal arise often during and after defecation, which is more characteristic for endophitic tumours, which, spreading into adjacent tissues can cause dysuric disorders (friquent urodynia), abdominal pains and pains in the lumbosacral area.

Common signs of cancer (unmotivated weight loss, appetite loss, weakness, increased fatigability, loss of vital interests and so on) in rectal cancer occur at the late stage of disease.

The objective sign of rectal cancer is detection of a tumour in the rectum on digital investigation or proctosigmoidoscopy. A tumour usually has a dense consistency, tuberous surface, it easily bleeds at touch, its mobility is often limited.

Complications. On localization of cancer in the supraampular area at late stages of disease development the tumour can invade to the uterus, vagina, wall of the urinary bladder with formation of vesicorectal, urinary and vaginorectal fecal fistulas. In cancer of the lower ampullar area the tumour involves the urethra with formation of rectourethral and rectovaginal fistulas. Characteristic feature of rectovesical and rectourethral fistulas is occur-

rence of a fecal odour of urine, gas and parts of fecal masses excretion with urine, fever with a hectic chill. The cancer of the anal canal can become complicated by paraproctitis and formation of pararectal fistulas, functional incompetence of anal sphincter, fecal and gas incontinence; rectal obstruction. The rectal lumen narrowing with development of obturative intestinal obstruction, as a rule, is preceded by long-term, persistent constipations, on background of which there can be intestinal wall perforation which locates more proximally than the tumour. The occurrence of remote metastases changes the clinical course of disease depending on their localization (the liver, the brain, the lung, the skeletal bones etc.). A rather rapid clinical course of disease develops in the patients of young age (up to 40 years). Rectal cancer which has arisen against a background of nonspecific ulcerative colitis, chronic paraproctitis and Crohn's disease has an especially unfavourable prognosis.

Diagnosis is based on clinical data and results of special researches. An asymptomatic course of the early stage of disease and late visit to a doctor are the basic reasons of its late diagnosis. The other reason of late diagnosis with a well-timed visit to a doctor is the neglect of a diagnosis method, which is necessary in these cases. Digital research of the rectum should be carried out in three positions: a genucubital position, position on the left side and "squat" position. The digital research of the rectum should be preceded by careful examination of the area anal (maceration of the skin with liquid fecal masses, swelling and clottages of haemorrhoidal veins, insignificant openning of the anus).

Sometimes tumour masses have ulcerations with attributes of a perifocal inflammation, incarceration and acute painfullness of adjacent tissues. On detection of a tumour during digital research of the rectum its size is determined, consistency, localization, mobility and displaceability in relation to adjacent structures, and character of pathological discharges are determined too. In female in a series of cases a combined, rectovaginal research is carried out, at which it is possible to estimate more precisely prevalence of a tumoral process. The second obligatory exam on suspicion of the rectal tumour is the proctosigmoidoscopy allowing estimating a condition of the rectum and rectosigmoid proximal areas and carrying out biopsy in necessary cases. Irrigoscopy is used more often than other special methods (for estimation of condition of higher parts of the colon), at which the defects of filling, deformation, sites of deposition of contrast mass can be found out. Last years selective adenography, pelvic phlebography, lymphography, echolocation (USE) with cavitary gauges, computer X-ray tomography and research with the help of MRI are applied for more exact topical diagnosis of tumours and possible metastas-

es. The application of modern methods of research allows making the exact etiological and topical diagnosis in 95% of the patients before operation.

Treatment. The basic method of treatment of rectal cancer is surgical in a form of radical operations: sphincteropreserving (anterior resection and abdominal-anal resection with lowering of the sigmoid intestine) and connected with removing of anal closing apparatus and applying of an unnatural anus as an abdominal-perineal extirpation of the rectum and obstructive resection (operation of Hartmann). During palliative operations (colostomy) primary tumour can be removed, but its metastases are left. Electrocryolysis of a tumour, elimination of complications (obstruction, bleeding, perforation) without removing the primary tumoral center are palliative operations too.

Adjuvant therapy concerning cancer of the rectum is applied as radiation therapy before operation and in the postoperative period and chemotherapy (general and local), in a combination of these methods and in connection with other influences (general hyperglycemia, a controlled local hyperthermia). However, influence of adjuvant therapy has been still under intensive study, as 5-years' life survival of the patients with cancer of the rectum makes 45–55% from number of radically operated persons.

RECTAL SARCOMA

Non epithelial malignant tumours (sarcomas) of the rectum make about 1% among neoplasms of this organ. A source of their development is connective-tissue structure (simple spindle-shaped and round-cell sarcomas), lymphatic tissues (lymphosarcomas) and endothelium of blood vessels (haemangioendotheliosarcomas). Originally these tumours represent nodes of various size covered with a non-struck mucosa. These formations are painless and are not fallen down on palpation for a long time.

Lymphosarcoma of the rectum is met rather seldom and proceeds from lymphoid elements of the submucous layer (lymphoid follicles). Localized and diffuse forms of lymphosarcomas are distinguished, and the latter form represents a local display of a common lymphosarcomatosis. Unlike cancer lymphosarcoma remains in the thickness of the wall for a long time and does not cause any complaints, however, later on stools with mucus and blood admixtue, deterioration of general condition, weight loss, general weakness, fervescence can appear.

The prognosis in diffuse forms of lymphosarcoma is unfavourable, in localized forms surgical treatment (dissection of a tumour, ablation of the rectum) in a combination with radiation therapy is quite justified.

Spindle-shaped and round-cell sarcomas proceed from the connective tissue of the submucous layer and are usually covered with unchanged mucosa. Their sizes reach 8–10 cm in diameter, but, locating in the thickness of the rectal wall, they do not cause any anxieties for a long time. Treatment is surgical (dissection of a tumour within the limits of healthy tissue). In the presence of metastases the prognosis is unfavourable.

Haemangioendotheliosarcomas are described in single cases. But when integrity of the epithelium covering them is broken, these tumours can cause rectal bleedings.

Taking into account rather long absense of signs in course of rectal sarcomas, it is necessary to undergo each patient with proctologic complaints to digital research of rectum, proctosigmoidoscopy with biopsy and urgent histological research when a tumour or ulcer is detected in this organ.

RECTAL MELANOMA

Melanoma of the anorectal area is met very seldom, and differs by malignant course and in late diagnosis and wrong treatment results in lethal outcome.

Among melanomas of various localization it makes about 0.7%. In most cases patients complain of anal bleeding, quite often combined with pain and itching in the anal area and perineum. The tumour usually is small, covered with normal mucosa and has dark cherry or dark blue colour. It can be assumed to be haemorrhoids or polyp by its appearance and position, especially when the tumour has a pedicle and can prolapse like a polyp or haemorrhoids, being easily set behind the rectal sphincters. As a rule melanoma localizes in the anal canal, less often it is in the area of the transitive fold and even less often — in inferioampular part of the rectum. For a long time melanoma remains as a local process and does not metastasize. The correct diagnosis and the wide dissection of melanoma at this stage can give the good remote results. If it has not taken place, subsiquently a tumour grows, becomes ulcerated and can metastasize by lymphatic ways to the inguinal lymph nodes, and haematogenously to the lungs, the liver, the kidney. The choice of a treatment mode depends on localization of a tumour and extensiveness of metastatic spreading. They consider laparotomy an expedient diagnostic method and at absence of metastases in the mesenteric lymph nodes and the liver in melanoma of the anorectal area the abdominal-perineal extirpation of the rectum with subsequent radial and chemotherapy (Vincristin + Dactinomycin + nitrosomethilurea) is used. The prognosis is unfavourable at late stages.

INFLAMMATORY DISEASES OF THE RECTUM

Acute and Chronic Proctitis

The inflammation of the rectum — proctitis — is more often formed as a part of common disease of the colon (proctocolitis, proctosigmoiditis). Such proctites are observed, for example, in ulcerative colitis, dysentery. Other reasons of an inflammation of the rectum are helminthiases, gonorrhea, tuberculosis, inguinal lymphogranuloma and lues. The radial and allergic proctites belong to the special forms.

The isolated inflammation of the rectum is observed less often. Its occurrence is usually caused by microtrauma of the mucosa with dense fecal masses, foreign bodies (for example, fish bone) with the subsequent development of a nonspecific inflammation. A certain role is played by a regular irritation of the rectal mucosa owing to abusing purgative, medical clysters, suppositories.

Acute and chronic proctites are distinguished according to clinical picture. Among local inflammatory processes in the rectal wall diverticulitis, cryptitis, papillitis, perineal proctitis, pectenosis are defined.

Clinical course and diagnosis. In an inflammation of the rectal mucosa the patients complain of pain in the anus at defecation, tenesmus; quite often blood and mucus admixture is marked in stool. Edematous inflamed mucousa does not provide hermetism of the obturator apparatus of the rectum, that causes mucus discharge from the anus with development of maceration of the perianal skin, itching. Sometimes sphincter failure and incontinence of liquid fecal masses and gases take place. During rectal exam the painfullness of the walls of the anal canal; changes of a sphinter tonus are marked at the acute stage. In chronic course of proctitis the rectal exam allows to reveal the thickening of the rectal wall, its cicatrical changes.

The clinical forms of proctitis are diagnosed by the data of ano(procto)-scopy. The swollen bright red mucosa of the rectum easily bleeds at contact with a radiographic cone of a proctoscope, has fibrinous layers. In series of cases erosive changes or ulcers are found out. Clinical forms of proctitis are diverticulitis, cryptitis, perineal proctitis, papillitis, and pectenosis.

Diverticulitis

The inflammation of a diverticulum in the rectal wall is shown by pains in depth of the pelvis, delay of stool, fervescence, leucocytosis and other attributes of infectious intoxication. A limited painful infiltrate in the wall of the intestinal is defined on rectal exam; endoscopically in this zone

the mucosal hyperemia is found out. At the further disease course a purulent diverticulitis develops, which perforation results in development of acute paraproctitis; perforation of a diverticulum localized in the intraabdominal part of the rectum is like to clinical course of pelvic peritonitis. Diverticula of the rectum belong to infrequent diseases, and their diagnosis is difficult.

Cryptitis

Cryptitis is inflammation of the Morgagni's crypts' mucosa. This disease develops, as a rule, as a result of a chronic trauma of the mucosa in constipations, but can be also secondary (in haemorrhoids, fissures, anal itching, chronic proctitis and paraproctitis). The patients complain of a pain and sensation of a foreign body in the anus. At anoscopy hyperemia of one or several Morgagni's crypts and mucus layers is defined.

Perineal Proctitis

Perineal proctitis is a limited inflammation of the skin and mucosa of the perineal part of the rectum. Its basis is the affection practically of all cryptes along the circle of the anus.

Papillitis

Papillitis is a thickening of papillas along the pectineal line, which hypertrophy occurs under influence of chronic inflammatory process (owing to traumas in constipations and haemorrhoids). Quite often dwarfed papillas are seen in the anus as cone-like formations. In exacerbation of the inflammatory process they become tender and are felt as a foreign body.

Pectenosis

Pectenosis is a thickening and cicatrical degeneration of tissues at the basis of Morgagni's crypts and banks at the upper part of the transitive fold, in which replacement of muscular elements of the submucous layer of the anal canal by connecting or cicatrical tissue occurs. The reasons of pectenosis are chronic inflammatory processes: perineal proctitis, anal fissure, and also chronic paraproctitis with a circular locating of fistulous channels. The loss of elasticity of the anal canal walls and retaining function lays in the basis of clinical displays of pectenosis. The patients complain of incontinence of gases, liquid fecal masses. In accompanying proctosigmoiditis the maceration and itching of the perianal skin are observed. In severe

cases, with development of narrowing of the anal canal (circular pectenosis), patients become disturbed by difficulties of firm feces evacuation from the intestine; the cases of the intestinal obstruction owing to pectenosis are described. On rectal exam a rigid cicatrical chord is defined in the lumen of the anal canal, posed in the longitudinal or circular direction.

Diagnosis of proctitis provides obligatory research of other parts of the colon (proctosigmoidoscopy, irrigoscopy, and fibrocolonoscopy), histological research of bioptates with changes of the rectal mucosa. These data and specific serologic tests allow differentiating isolated proctites, common diseases of the colon, tumoral and specific affections (tuberculosis, lues, and gonorrhea).

Basis of proctites *treatment* is a complex of conservative measures. A sparing diet (giving up of spices, smoked meal, greasy nutrition and fried dishes) is indicated. Toilet of the anal area, sedentary baths, perineal showers are obligatory. Warm clysters with addition of weak antiseptics are applied. Resolvents are expedient in a form of rectal suppositories. The antibiotic therapy is indicated in an inflammatory infiltration of the rectal wall locating deeper than the submucous layer and in development of common signs of intoxication.

Surgical indications arise rarely. In diverticulitis the operation is recommended in case of increasing of an inflammatory infiltrate with transition to the pararectal tissues; opening of the infiltrate through the rectal lumen is usually made. For treatment of relapsing papillitis under a local anaesthesia a dissection of enlarged papillas with the subsequent sutures applying is made; this operation should be performed when there isn't exacerbation of disease. The narrowing of the anus owing to pectenosis is the indication to devulsion of the sphincter or dosed sphincterotomy. Other operative measures in patients with proctitis are undertaken for treatment of the diseases which caused proctitis development or in development of pyo-septic complications (for example, paraproctitis and pelvic peritonitis).

Rectal Ulcers

Rectal ulcers more often are the results of specific inflammatory processes (tuberculosis, lues, soft chancre etc.). A simple ulcer of the rectum belongs to rare diseases.

A tuberculous ulcer is one of three forms of the rectal and anal tuberculosis (fistulous, ulcerative, and warty). The patients complain of mucus, blood or purulent discharges from the anus, false urge to defecate. A longtime tuberculous ulcer can become complicated by perforation of the intestine wall or its cicatrical narrowing. The diagnosis of rectal tuberculo-

sis is based on laboratory data (histological exam of a bioptate from the ulcer, serologic tests, pus inoculation of special mediums). A thorough examination for revealing other tuberculous foci is necessary. The patients are directed to specialized establishment (antituberculous dispensary), where along with specific antituberculous therapy the treatment of proctitis is carried out.

A syphilitic ulcer of the rectum is observed at II or III stages of the disease. The multiple syphilitic ulcers are met rarely, they tend to merge. In a circular affection of the rectum its stenosis can develop. Rectoscopy reveales an ulcer with undetermined edges, the tissues have cartilage-like density, the fundus of ulcer is covered with dirty-grey deposit, and granulations are absent. These objective data in many respects are similar to endoscopic attributes of a malignant affection of the rectum; the diagnosis is made by results of histological exam of ulcer bioptate and serological tests for lues. The specific treatment is carried out; surgical indications are usually absent.

Ulcers of the rectum in case of a soft chancre are formed as a result of spontaneous opening of pustules. The patients mark an admixture of blood and pus in stool, pain on defecation. Rectoscopy reveales multiple painfull ulcers with uneven contours; the fundus is covered with mucopurulent separations, the edges are undermined. Inguinal lymphadenitis having predilection to purulent decomposition with formation of purulent fistulas develops quite often. The diagnosis proves to be true by detection of Peterson — Ducrey's streptobacilli in pus. Differential diagnosis with lues is necessary. The treatment provides a course of antibiotic-and sulfanilamide-therapy (within 10 days), clyster with weak solutions of antiseptics. The Healing of ulcers takes place usually during 8–9 days.

ACUTE AND CHRONIC PARAPROCTITIS

Paraproctitis is a bacterial inflammation of the perirectal fat. The morbidity to a certain degree depends on social-hygienic conditions of life, chronic diseases of the rectum. Paraproctitis appears to be 2 times more common in the male than in the female. In structure of proctologic diseases paraproctitis occupies the fourth place after haemorrhoids, anal fissures and colites and makes 15–40% of all the rectal diseases.

Etiology. Paraproctitis is caused by multi-infection, which structure can include intestinal rod, anaerobes, golden and white staphylococcus, streptococcus, and enterococcus. More than 90% of observations are associated

with intestinal rod, staphylococcus or streptococcus; such paraproctitis is called usual or banal. Clostridial anaerobes are allocated less often. Extremely seldom specific (tuberculous, actinomycotic, syphilitic) paraproctites are met (in 0.1–0.5% of cases).

Pathogenesis and pathological anatomy. The causative agent invades the perirectal fat through the anal glands, damaged mucosa of the rectum, from the adjacent organs, affected both by inflammatory process and by haematogenous and lymphogenous ways.

The anal glands, which ostiums open in the basis of Morgagni's crypts, are considered to be the basic infection entrance in paraproctites. This point of view in 1878 has been stated by H. Chiari for the first time, and it has received an anatomic substantiation by researches of O. Hermann and L. Desjosses (1880). Getting into the anal glands from the intestinal lumen, the infection breaks them and penetrates the pararectal space. The other variant of the inflammatory course takes place as follows: a retention cyst of the anal gland forms as a result of edema and occlusion of the duct, cyst infection causes abscess. The subsequent development of an abscess cyst, haematogenous or lymphogenous diffusion of an infection results in formation of an abscess in the pararectal fat.

The microtrauma of the rectum and anal canal mucosa is considered to be the other basic reason of acute paraproctitis. It is caused by dense fecal masses in constipations, which are marked in 2–20% of patients with paraproctitis, or dense and acuminate inclusions in fecal masses (fruit, fish, meat bones, sharp cereals etc.). Microtraumas (microtearings) of the mucosa are likely to be in diarrhea, proctitis, and proctosigmoiditis too.

Acute paraproctitis can be developed owing to damage of the intestinal wall during medical manipulations (blockade, clysters, endoscopic researches etc.). The cases of paraproctitis as a result of erroneous introduction in the rectum of ammonia alcohol, solutions of chloramin, potassium permanganate, etc., are known. Severity of mechanical damage of the rectal wall, necrosis of tissues owing to influence of aggressive liquids result in a massive contamination of the pararectal fat, pronounced intoxication, which leads to extremely severe course of iatrogen paraproctites with high lethality (up to 50% and more).

Exogenous infection of the pararectal fat may occur as a result of wounds (including gunshot wounds) of the perineum, alcohol-novocainic blockade, injections of non sterile solutions, and during operative interferences. Posttraumatic paraproctitis makes less than 1% and proceeds more severely, if there was a trauma of the rectum.

Paraproctitis quite often arises against background of other rectal diseases. Acute proctitis (cryptitis) is a ground of occurrence of acute paraproc-

titis as a rule. On the contrary, chronic proctitis is extremely seldom complicated by inflammation of the pararectal fat. Acute paraproctitis is met in 6–8% of the patients with a nonspecific ulcerative colitis, in 15–25% of the patients with Crohn's disease. A certain role in occurrence of paraproctitis in these diseases is apparently caused by decrease of a barrier function of the inflamed rectal mucosa and dissemination of infection through ulcerative defects of the mucosa.

Paraproctitis is also observed against a background of haemorrhoids. However, in spite of the fact that haemorrhoids are present approximately in 30% of the patients with paraproctitis, occurrence of paraproctitis is caused by accompanying proctitis; pararectal fistulas in the patients with haemorrhoids have an internal aperture in the crypts, but they practically never open into mucosa covering the haemorrhoids.

In 0.7–6% of cases the reason of paraproctitis can be an anal fissure. It is necessary to notice that a chronic anal fissure, limited from surrounding tissues by a strong granular barrier, is rather seldom complicated by paraproctitis.

Paraproctitis can arise as complication of a disintegrating malignant tumour of the rectum.

Paraproctites as a result of pathological process spreading from the adjacent organs are allocated in a separate group. The inflammatory process can involve the pararectal fat in diseases of the prostatic gland (acute prostatitis, abscess of the prostatic gland), the urethra, the paraurethral, Kouper's glands, female reproductive organs (perimetritis, bartholinitis), less often in osteomyelitis of the pelvic bones, spinal tuberculosis, urinary phlegmons, juxtaspinal abscesses. The development of paraproctitis as complications of Douglas' space abscess opening through the rectum is possible. The inflammation of the pararectal fat is met in spread of pus of the epithelial coccygeal channel, abscessing dermoid cyst.

Inflammatory infiltrate spread from the adjacent organs or areas is the main mechanism of specific paraproctites occurrence: tuberculous, actinomycotic, and syphilitic. Less often specific paraproctites arise owing to a primary affection of the rectum, when the infection penetrates the pararectal fat through mucosal ulcerations.

The lymphogenous or haematogenous metastatic spreading of a nonspecific and specific infection as the reason of paraproctitis is found out extremely seldom, but the cases of paraproctitis are described in connection with previous panaritium, angina, influenza and purulent diseases of other areas.

Sometimes it is difficult to establish the origin of paraproctitis.

The further development of the penetrated infection in many respects depends on the type of pararectal fat infection and is shown by various

forms of paraproctitis. The anal glands are localized in submucous or intermuscular layers, in some people they are in the supralevator space. Breaking them, the pus penetrates the submucous, hypodermic ischiorectal, pelviorectal or retrorectal fatty spaces. Formation of the most complex by structure purulent cavities and fistulas of the rectum, which further form, is connected with branching of anal glands in the supralevator space.

Infection spread through the damaged mucosa is possible by the intertissue clefts, the paravasal fat, and also by lymphogenous and haematogenous way. The submucous inflammatory infiltrates are easily spread upwards, detaching the mucosa. Spread along vascular branches explains affection of intermuscular spaces between circular and longitudinal layers of muscles of the intestinal wall.

The stage of serous-infiltrative inflammation of the pararectal fat is short (2–3 days), and the process turns into the pyo-necrotic stage. The typical phlegmonous inflammation develops, predilection of which to limitation is connected to a virulence of microflora and reactivity of an organism: banal paraproctites are more inclined to an abscess formation (limitation); in virulent, especially anaerobic flora the inflammation is quickly spread by lymphatic ways or owing to lysis of tissues to the pelvis fat, gluteal area and femur. In especially severe cases there can be necrosis of tissue without formation of pus. The prevalence of the purulent process depends also on conditions of outflow. In wide connection with the rectal lumen or fast reaching the skin and evacuation of pus outside, the purulent cavity is relatively insignificant and has a simple structure. The worse spontaneous evacuation of the purulent exudate, the more extensive scales of destruction of the pararectal fat, and the cavity formed has more and more complex, broad structure with numerous purulent edemas.

The acute purulent inflammation in paraproctitis reaches the maximum, as a rule, for 5–8 days. Further with the absence of adequate treatment there can be a spontaneous evacuation of the purulent cavity in the rectal lumen or outside. The external fistulous apertures in paraproctitis are more often posed in the perineum, but they are also met on the scrotum, femur and forward abdominal wall. Spontaneous opening of an abscess in the intestinal lumen at the level of the ampullar part and even in the abdominal cavity is possible.

Through a spontaneously opened fistula the purulent cavity is cleared slowly, the infectious-inflammatory process is inclined to exacerbations, and the disease practically always passes in the chronic form.

The histological structure of the pararectal fistula is various, but a chronic inflammation with development of the granular tissue in the wall of the fistula is usually determined. Almost 26% of the patients have the attributes

of cuticularization of fistulous channels, which excludes an opportunity of their independent closing. Besides, chronic paraproctitis results in appreciable change of muscular structure of the perineal part of the rectum, sphincter, it causes cicatrical atrophy of the muscular tissue of the pelvic fundus.

Classification. Two basic forms of disease are distinguished: acute paraproctitis, which basic sign is a purulent cavity in the pararectal fat, and a chronic paraproctitis which is the most often result of the previous form, the basic substrate of which is a rectal fistula.

According to classification of Scientific-Research Institute of Proctology paraproctites are divided into the following classification signs:

I. According to etiology:
— nonspecific;
— specific;
— posttraumatic.
II. According to activity of inflammatory process:
— acute;
— relapsing;
— chronic (fistulas of the rectum).
III. According to localization of the inflammatory focus:
— hypodermic (50–75%);
— submucous (2–6%);
— ischiorectal (15–40%);
— pelviorectal (2–7%);
— retrorectal (1–3%).
IV. According to location of an internal fistulous openning:
— anterior;
— posterior;
— lateral.
V. According to relation of fistulous channel to sphincter fibers:
— intrasphincteral (subcutaneously — submucous);
— transsphincteral;
— extrasphincteral (1–4 degrees of complecacy).
VI. Presence of external and internal apertures of the fistula:
— complete;
— incomplete (internal).

Clinical course. As a rule, acute paraproctitis begins with occurrence of malaise, weakness, headache, unpleasant sensations in defecation. Then chill, fever and increasing pains in the rectum, the perineum or in the pelvis join. This first period of disease, which does not exceed three days, quite often is called prodromal; it is more correctly to consider, that it corresponds to development of serous-infiltrative inflammation of the pararectal fat. With transition of inflammation in the stage of purulent-necrotic decomposition of tissues, pains become intensive, constant, get pulsating

character, their prevalence is enlarged; in some cases the pains are marked in the abdominal area. The higher inflammatory process in fat is localized, the more prevalence of pains.

Quite often patients complain of tenesmus, delay of stools, dysuric disorders. However, reassessment of these signs, as a matter of fact secondary, reflecting a degree of functional disorders, can direct diagnosis to a wrong way.

In the area of the anus there is an edema at the beginning on the side of the affection; in 2–3 days it is displaced to the other side. The skin in the zone of swelling becomes smooth, glistening. The palpation of the area damaged at the height of the inflammatory process is acutely painful.

As the process of inflammation spreads and turns into the stage of purulent decomposition of tissues, the syndrome of endogenic intoxication progresses: weakness, unhealthy condition, loss of appetite, sleeplessness appears. Fever, chill, hyperhidrosis join. The pulse becomes frequent, leucocytes number in the blood rises, neutrophilic shift of the leucocytic formula occurs etc.

In a phlegmonous inflammation or anaerobic infection the signs of intoxication become leading in the clinical picture. The early signs of anaerobic paraproctitis are tachycardia, decrease of arterial pressure, hectic fever, cyanosis, euphoria, and other signs of severe endogenic intoxication. Quite often there are signs of sepsis. The severity of condition is explained by both high virulence of anaerobes, and extensive disintegration of ischiorectal and pelviorectal fat with the process spread on the hypodermic fatty tissue and muscles of the gluteal area. The development of an ascending anaerobic lymphangitis with an affection of the anterior abdominal wall and the peritoneum can cause occurrence of signs of peritonitis.

The local signs of pararectal abscesses are caused by their localization. The submucous abscess is localized above Morgagni's crypts or anorectal line. The digital research of the rectum is painful, the site of an infiltration of the rectal wall is defined; a vague infiltrate of the intestine wall testifies to diffusion of inflammation to the intermuscular space. The inflammatory infiltration can be spread to the perineum and perianal skin. An attribute of purulent submucous paraproctitis is softening of the infiltrate or spherical elastic swelling (abscess) locating under the mucosa. In some cases the pyo-inflammatory process spreads along the submucous layer downwards, reaches the hypodermic fat; such an abscess is called subcutaneously-submucous.

Hypodermic paraproctitis localized near to the anus causes acute pains especially strong on defecation. This abscess is seen from outside: asymmetry and swelling of the perianal area are pronounced because of that the anus loses its typical stellate form. The skin is hyperemic. The palpation of an infiltrate is acutely painful, the strain of tissues is determined. Revealing of fluctuation is late and not in all cases. In neglected cases, at sponta-

neous opening of paraproctitis, it is revealed the site of dermal excoriation with an aperture with discharges of pus, quite often with an admixture of blood and feces.

An ischiorectal abscess, located in deep layers of the ischiorectal fossa fat, can be spread up to the prostatic gland, pelvic fat or behind the rectum to the other side. During the first days of disease common signs usually prevail. The outside local attributes of an ischiorectal abscess are shown, as a rule, at the end of the first week as not-pronounced edema and skin hyperemia that testify to transition of an inflammation to the hypodermic fatty tissue. The palpation of the perineum allows revealing a deep infiltrate posed more medially than tuber of the ischium, which is acutely painful with an irradiation of pains to the rectum. The signs of softening and fluctuation are usually late revealed, when the suppurative focus reaches great sizes.

A pelviorectal abscess is posed higher, behind the pelvic fundus, in the levators' sheath. It has no outward signs in the area of the anus, the superficial pain is absent. The patient is disturbed with malaise, fever, chill, pains in the lower part of the abdomen, pelvic areas; the cutaneous edema above the pubic joint is possible. The dysuric disorders, irradiation of pain to the sacrum and suprapubic area are frequent. The palpation of the perineum does not allow revealing the pathological center, but on examination of the abdomen a deeply located infiltrate without precise contours can be palpated in the suprapubic area. The inflammatory infiltrate is defined on digital rectal examination, especially bimanual. Only when the purulent process spreads downwards, into the ischiorectal fat, external signs of inflammatory affection of tissues occur.

A retrorectal abscess is shown by a severe pain in the rectum with irradiation to the sacrum; edema of the sacrococcygeal area is possible. On palpation: tissues of the perineum are not practically changed; on examination of the abdomen deeply located infiltrate without precise contours can be palpated. The local attributes of disease occur late, in spread of the purulent process into the ischiorectal and hypodermic fat. On rectal examination a painful protrusion of the posterior wall of the rectum is defined; the pressure the coccygeal bone strengthens pain.

Burst of an abscess towards the skin or its evacuation into the lumen of the rectum leads to a temporary improvement of a common condition.

However, unsufficient evacuation of pus through a formed fistulous channel causes the further progression of infection.

Local complications of acute paraproctitis are following: breaking out of pus into the retroperitoneal space, into the abdominal cavity, into the urinary tract, spread of purulent process to the pelvic bones. The evacuation of pus in the rectal lumen above the serrate line is possible. In usual forms of acute paraproctitis these complications are extremely rare.

After opening of acute paraproctitis three outcomes of disease are possible:

1) formation of a rectal fistula (chronic paraproctitis);

2) temporary heading with the subsequent relapse of inflammation;

3) convalescence.

The formation of a rectal fistula in spontaneous opening of acute paraproctitis is marked more than in half of patients. The reason is that the internal fistulous opening remains open after the burst (infection entrance).

If after opening of acute paraproctitis the internal fistulous opening is covered with granulations, and then with a cicatrix, purulent discharge stops, the wound heals without a burrowing. The convalescence begins when the infection entrance has undergone purulent destruction, and a strong cicatrix has been formed; such outcome of acute paraproctitis is observed in each third patient. When the internal aperture persists, a cicatrix on its place appears to be fragile and it is easily injured, then the internal aperture opens, fat is re-infected, paraproctitis recurs

Paraproctitis, appeared against a background of already available fistulas, is called chronic relapsing one; if in repeated paraproctitis the functioning fistulas are absent, the illness has the name of acute relapsing paraproctitis.

High percentage of changing of acute paraproctitis into chronic forms is connected with the late reference of the patients with acute paraproctitis for medical help (30% — after spontaneous burst), insufficient volume of an operative measure (without elimination of the internal fistulous openning).

The clinical course of chronic paraproctitis is caused by presence of the aperture in the intestine, fistulous channel, perifocal inflammatory and cicatrical changes in the intestinal wall and fatty spaces of the pelvis.

The disease has an undulating course: the periods of exacerbation are replaced by more or less steady remissions. The internal fistulas are shown by tenesmus and pains. In external fistulas the opening is visible, soaking and itching are observed; the pains are not characteristic for them. Pains occur or increase after delaying of purulent discharge from the fistulous channel; in these cases they are accompanied by signs of intoxication. The constant pains can be observed in malignization of the fistula.

The character and quantity of discharge from the fistula depend on activity of the inflammatory process, size of the purulent cavity. The more volume of the cavity, the more active the inflammatory process, the more discharge volume. Gases and intestinal contents discharge are observed in complete fistulas. In a banal intestinal microflora discharge is moderate, in tuberculous infection they are plentiful, in actinomycotic paraproctitis scanty, sometimes crumblike.

The common condition of the patients is usually normal, but during long-term cause of paraproctitis there are irritability, sleeplessness, headaches; working ability is reduced, the development of neurosis, impotency is possible.

Complications of chronic paraproctitis are caused by intoxication from a suppurative focus and changes of adjacent tissues and organs. Common complications are asthenia (fast fatigability, irritability, common weakness, decrease of working ability), neurotic reactions and amyloidosis of internal organs. The most often local complications are maceration of the perianal skin (at absence of a due care); an accompanying proctosigmoiditis (70% of the patients), manifested by mucus discharge unstable stools with abundance of gases, abdominal distention, anal itching; pectenosis (50% of the patients with extrasphincteral fistulas).

The most dangerous, but very rare (0.3% of the patients) complication is malignization of a fistula. Malignancy begins from the internal fistulous opening, less often it is at the side of an external fistulous opening. Adenocarcinoma, less often planocellular cancer is usually revealed. The malignization is possible when a fistula exists more than 3 years. The most often attributes of malignancy of a fistula are constant pains in the perineum and rectum, change of character of fistulous discharge, discharge of dark blood from the rectum, false urge to defecate. These signs are not pathognomonic, but they are the indications for biopsy and histological exam of bioptate. In 60% of cases malignization is diagnosed later on, when the radical operation is already impossible.

Diagnosis of acute paraproctitis is based on data of clinical exam, cautious digital research of the rectum and laboratory data. It is necessary to analyse probable reasons of disease occurrence, and also to pay attention to diseases of other organs, complicated course which can cause acute paraproctitis (abscess of Douglas space, atypical forms of acute appendicitis, parametritis, and pelvioperitonitis).

In most cases diagnosis of acute paraproctitis does not represent difficulties. The digital research of the rectum is enough for recognition of hypodermic and submucous paraproctites. For revealing deep abscesses the bimanual research of the rectum is necessary: the finger of one hand enters the rectum, and the other hand outside reveales indulation of the pararectal fat. In pelviorectal abscess it is impossible to reach the top border of an inflammatory infiltrate that distinguishes it from ischiorectal paraproctitis. In doubtful cases at absence of a strong pain the proctosigmoidoscopy is probable, during which hyperemic easily bleeding mucosa, located above the infiltrate, is found out; the diagnostic puncture can be made. It is necessary to notice that for diagnosis of acute paraproctitis it is not necessary to resort to instrumental researches because of acute painfulness and labored preparation of the intestine; informativity of these methods is insignificant in acute paraproctitis.

Acute paraproctitis should be differentiated with osteomyelitis of the pelvic bones, abscess formation of presacral teratomas, pyesis of epithelial coccygeal channels and cancer of the anus. The revealing of purulent de-

composition of the rectal wall, absence of X-ray attributes of bones destruction testifies to acute paraproctitis.

Diagnosis of chronic paraproctitis usually does not cause difficulties, as the patient visits a doctor concerning a fistula in the field of the perineum. The examination of the patient, alongside with revealing of the reason of fistula occurrence, accompanying diseases, has the purpose of exact topical diagnosis of a fistula: the relation to fibers of the sphincter, localization of internal fistulous opening, presence of purulent cavities in the pararectal fatty spaces, degree of fistulous channel branching, development of cicatrical process in the wall of the anal channel and along the fistula channel, complication of chronic paraproctitis.

If a fistula has internal (on the rectal mucosa), and external (on the skin) openings, this is a complete pararectal fistula. In presence of one aperture we speak about incomplete (internal or external) fistula.

The external fistulous opening is revealed on examination of the perineum. The multiple fistulous openings are suspicious of specific process. If the perineal asymmetry is revealed, it is necessary to think of the inflammatory or degenerative process. The pronounced cicatrical process around the external fistulous openings allows thinking of pectenosis and failure of the anal sphincter.

Palpation of the perianal skin in the perineal fat allows to detect a dense chord, referred to the side of the rectal wall. On bimanual rectal exam it is possible to reveal close connection of this chord with the intestinal wall to palpate incanceration of tissues and in the anal channel at the region of the internal fistulous opening.

The probing of a fistulous channel with a simultaneous rectal exam allows specifying a direction of the fistulous channel; it's branching in the perineal tissues, presence of purulent cavities in the pararectal fatty spaces, relation of the fistula to fibers of the rectal sphincter. The following manoeuvre is used: during probing the fistula with rectal exam the patient is offered to compress the anus, owing to which the anal channel's walls densely fit the finger. In the intrasphincteric fistula the probe, entered into the fistulous channel, is pressed to the finger and begins to be palpated more clearly; in the extrasphincteral locating of the fistula the sensation of the probe as a result of sphincter reduction is completely lost.

The internal fistulous opening is revealed with the help of anoscopy and proctosigmoidoscopy, which should be supplemented by chromofistuloscopy (introduction of a methylene blue solution in the external fistulous aperture). The endoscopic research allows also revealing accompanying diseases of the rectum.

Radiopaque fistulography is obligatory in complex fistulas, enables not only to characterize prevalence, localization and branching of the fistula in detail, but also to diagnose diverticulitis, Crohn's disease, rectal tumour,

to reveal relation of the fistula to nearby organs (the vagina, the urethra, the urinary bladder).

For diagnosis of a specific etiology of chronic paraproctitis (tuberculosis, actinomycosis and lues) bacteriological, histological and serologic researches are undertaken.

Treatment of acute paraproctitis in the initial stage consists in conservative measures: antibiotic therapy, resolvents, UV. However, in most cases patients address already at the stage of purulent-necrotic inflammation, when the urgent operative measure is indicated. The performance of operation in early terms promotes decrease of complications and relapses.

Tasks of operation are erasion of the purulent-necrotic substrate in the center of inflammation, an adequate drainage of the purulent cavity, revealing and possible dissociation of the purulent cavity with the rectal lumen.

Minimal volume of operation — opening of an abscess by a linear incision — is sufficient only in submucous paraproctitis. The operation is carried out transanally, and the wound of the mucosa heals by a repeated intention during 5–7 days.

In all the other forms of acute paraproctitis the preference is given to arcuate (semilunar) incision, which is put on a circle of the anal ring, receding from it not less than 2 cm. In small abscess with superficial locating in ischiorectal paraproctitis the application of a radial incision is possible, that allows to avoid an inadvertent damage of the external sphincter. An open purulent cavity is examined by the finger, all pockets are liquidated, necrotic tissues and pus are carefully deleted, a wound is repeatedly properly washed out by a solution of antiseptics. In a deep locating of an abscess there is a danger of the rectal wall and the anal urethra damage, and the radical surgical treatment is often impossible. In these cases there is a special need in intraoperation application of ultrasound.

In anaerobic paraproctitis when an affection is extensive the additional incisions in the gluteal area, on the anterior abdominal wall can be required.

A peculiarity of operative measures in acute paraproctitis is the stage of dissociation of the purulent cavity with the rectal lumen. Its absence, i. e. the performance of operation especially on the suppurative focus, is one of the essential reasons for disease transition into a chronic form. The integral condition of performance of this stage is the detection of infection entrance during operation, i. e. an internal opening of the passage, connecting the rectal lumen with a purulent cavity in the pararectal fat.

There are some variants of operation:

1) sphincterotomy (except for a deep portion of the external sphincter) on the depth of 12–15 mm through the internal opening (Rizhikh — Bobrova's method);

2) the dissection of the crypt, in which the internal opening in hypodermic paraproctitis is localized (according to type of Gabriel's operation);

3) dissection of an affected crypt and realization of Hippocrates' ligature in transsphincteral or extrasphincteral locating of the passage, connecting the rectum with a purulent cavity, and deep paraproctitis.

Under conditions of a specialized department with exception of anaerobic character of infection, a complete surgical treatment of a purulent cavity is performed, the operation can be finished by an active flowing drainage of the wound with applying sutures, that considerably reduces the term of wound healing and facilitates patients care. In other cases a wound is left open, gauze draine with an ointment on a water-soluble basis, or applying of proteolytic enzymes, solutions of antiseptics are used, and the treatment of a wound is carried out by standard rules.

The radical treatment of chronic paraproctitis is possible only by operative way. In account of undulating course of disease various surgical modes are selected. In exacerbation of disease (acute or chronic relapsing paraproctitis) an urgent intervention is indicated with an appropriate treatment of acute paraproctitis. In presence of a functioning pararectal fistula an elective radical operation is performed, thus in case of subacute course of a paraproctitis, in presence of inflammatory infiltrates in the fistulous circle the operation is postponed for 1–3 weeks; this period is necessary for reducing inflammation by conservative methods (antibacterial therapy, lavage of a fistulous channel by solutions of antiseptics and antibiotics, treatment of accompanying proctitis). At last, during remission, when the internal fistulous opening and purulent channel are not defined, the operative measure is not performed.

Tasks of surgery are syringectomy, erasion of cicatrix-changed tissues and liquidation of the internal aperture (source) of a fistulous opening. The choice of the operation method is determined by the relation of the fistula to fibers of the sphincter, complexity and prevalence of fistulous channels. The sex, the age of the patient, quantity of external fistulous openings, their distance from the edges of the anal canal, length and width of the fistulous channel, locating of an internal fistulous opening do not influence results of treatment.

There are 5 basic types of operations:

1) section of fistula and syringectomy to the intestinal lumen;

2) syringectomy of a fistula with sphincterotomy;

3) syringectomy of fistula with displacement of the mucosa of the distal part of the rectum;

4) ligature methods;

5) other methods.

In intrasphincteric fistulas in overwhelming majority of cases (up to 80%) syringectomy in the rectal lumen (Gabriel's operation) is carried out.

It consists of syringotomy along a probe from the internal opening up to the external one. The wound heals by a secondary intention. The relapses after this operation are marked in 1.7% of cases, failure of anal sphincter is in 1.2%. If necessary Gabriel's operation is supplemented by drainage of purulent cavities of the pararectal fat. Fistulotomy in the rectal lumen is applied less often.

In transsphincteric fistulas fistulotomy in the rectal lumen is supplemented by sphincterotomy. However, the higher from the anus the fistula perforates into the sphincter, the more is a threat of development of anal incontinence after operation. In such cases either sphincterotomy is carried out and at the end of the operation the fundus of the wound is partially cloused, restoring integrity of the sphincter, or a fistula is excised up to the sphincter, and through its transsphincteric part a ligature is carried out (Hippocrates' method). In transsphincteric fistulas it is necessary to supplement operation of syringectomy by opening and drainage of cavities in the pararectal fat.

The operations in extrasphincteral fistulas are the most difficult. Such a fistula, as a rule, can not be excised radically, as it leads to irreversible incontinence. As a rule, an extrasphincteral fistula is excised up to the rectal wall. The operative methods with the purpose of elimination of an internal fistulous opening in this situation are the most popular. The stump of a fistula is taken in the perineal wound by a Rudykh's method, then a back dosed sphincterotomy is carried out. The operation of Aminyev provides a dissection of the internal opening on the part of the rectal lumen with its closing by П-like mucous graft. The methods of invagination of the fistula stump in the rectal lumen are offered. Approximately in 25% of the patients the most ancient method of Hippocrates is applied successfully, it consists in performing a ligature through the internal fistulous opening, during the gradual pulling up of which cutting of intersection of tissues from the sphincter and fat occurs.

The relapses after operation concerning an extrasphincteral fistula are observed in 2–7% of patients depending on a degree of fistula complexity.

In the postoperative period during 3–5 days the patient receives low-residue free diet (before occurrence of stool). Treatment of the wound, if necessary — antibacterial, detoxicative and immune-corrigating therapy is carried out in volume depending on the extent endotoxicosis and disorder of vital functions.

Lecture 13
CHOLELITHIASIS

Cholelithiasis belongs to wide-spread diseases. Gallstones were found out during research of the Egyptian mummy (XV century BC). Now it is called disease of the century, disease of the civilization. There is information that in Europe and in Northern and Southern America cholelithiasis is revealed in 10–15% of the adult population, at the age of 40 and older — in 15–20%. According to some statistical data in the USA almost 15 mln people are carriers of gallstones and about 6 thousand die annually of the reasons connected with complications of cholelithiasis, including operative measures on this occasion. In Western Europe among the adult population diseases of the gallbladder and bilary tracts affect almost each fifth woman and each tenth man. In the European countries the lowest morbidity is marked in Ireland (5%), and the highest — in Sweden (32%). Among the native population of Africa cholelithiasis is rare — less than 1%. In Russia annual consulting concerning cholelithiasis makes up on the average 5–6 men per 1000 of population, so about 1 mln people per one year. At pathologoanatomic autopsy, stones in the gallbladder are found in 10–20% by chance. It is possible to judge about prevalence of cholelithiasis on the data of the International Society of the World Union of the Surgeons. They testify that in the world more than one million cholecystectomies are annually carried out, only in the USA — 400–500 thousand.

The gallstones are rather various according to a number of signs. The number of stones in the gallbladder can be from one up to several hundreds, form — round, spherical, barrel, cigar-like, berry-like. They vary in size from a gain of sand up to a chicken egg. Many stones, when they are multiple, have a facet surface. However, the surface can be smooth. With cholestasis both in the bladder, and in the biliary ducts higher than the narrowing (cicatrical structure) there can be "cholic putty" — shapeless mass usually of dark brown.

According to chemical structure, gallstones are divided into cholesterol, pigment and calcareous.

Cholesterol gallstones are formed in the gallbladder without inflammation in it. They have cyanotic-yellowish colour, float in water, burn with light-blue flame. On an incision these stones have a ray-like structure and well seen crystals of cholesterol. According to appearance, they sometimes remind a berry with a small-grained surface, but can be completely smooth. One large spherical stone is found quite often in the bladder, but they can be multiple. Cholesterol gallstones are not roentgenocontrastive.

Pigment gallstones are formed of bilirubin also in aseptic medium. They are dark brown, and black sometimes. These gallstones are formed with increased erythrocytolysis (hemolytic jaundice) with the high contents of unconjugated bilirubin in the blood. Pigment gallstones usually fill the whole bladder and have various sizes — from a gain of sand up to pea. Their structure is amorphic.

Calcareous gallstones consist of a carbonic lime. They are grey-white with rough surface, shapeless. They are formed usually in the bladder under conditions of inflammatory process.

Special researches have shown that in any gallstone it is possible to find either cholesterol, pigment, or a calcareous part. Gallstones of a mixed structure therefore are more frequently observed, but on appearance taking into consideration prevalence of one or another chemical substance, they are divided into three groups mentioned above. Cholelithiasis and its complications can be with any structure of a gallstone.

Etiology and pathogenesis. Long time ago it was noticed that in development of cholelithiasis three factors have the important meaning — inflammatory changes in the wall of the gallbladder, cholestasis and metabolic disorders of the lipid exchange. These factors are variously combined, but it is impossible to consider each of them separately absolute, and furthermore as the only reason causing cholelithiasis. Cholelithiasis in many respects is connected with disorders in the bile structure (dyscholia) and its other properties: buffer, electrical field, pH, change in the lipid structure etc.

Dyscholia can be primary, when a hepatic cell produces bile with high-lithogenic properties and secondary, depending on disorder of absorption and concentration of bile in the changed gallbladder. Primary dyscholia depends on a number of reasons, first of all on the excessive use of greasy and high-caloric feeding that causes the increased contents of lipids in the blood and high concentration of cholesterol. On increased erythrocytolysis (hemolytic jaundice), the hepatic cells produce bile with high contents of bilirubin pigment and it breaks balance in colloid suspension, which the bile is. The high content of calcium in the blood also causes the increased precipitation of bile. Primary dyscholia can develop with insufficiency of cholic acids, that very considerably breaks the properties of bile. On 1 mole-

cule of cholesterol 6 molecules of cholic acids and 2 molecules of lecithine should fall. In normal correlations the concentration of cholesterol to the contents of cholic acids makes up 1: 20–25. At the correlation lower than 1:13, the conditions for deposition of cholesterol are framed and bile becomes lithogenic. Cholic salts support cholesterol under condition of solution by formation of mycells, which give bile the property of colloid suspensions. The stability of a suspension depends on durability of an electrical charge of mycells. It is known that some animals have the high concentration of cholic acids and they do not have stones in the gallbladder. If the quantity of cholesterol, which is in bile, is divided into quantity of cholesterin, which can be dissolved at a concrete correlation of cholic acids and lecithine, it is possible to receive an index of lithogenity. If it is 1, the bile is saturated, if lower than 1 — unsaturated. Lithogenity of bile depends also on endocrine influences. Therefore, for a long time it is noticed that cholelithiasis frequently occurs at hypothyroidism, as the ovarian hormones influence the exchange of lipids and carbohydrates in the liver. On the increased contents of estrogens, the high concentration of cholesterol in bile is marked. The bile structure changes with the bile pH disorders. In the hepatic bile pH is 8.2, in the gallbladder it falls up to 7.0.

Secondary dyscholia (the change of bile properties in the gallbladder) has the greatest importance. It is known that stones form in the gallbladder. Cholestasis and the inflammatory process in the gallbladder complicate primary dyscholia into cholelithiasis. Xanthomatous cells, containing cholesterol, occur in the mucosa of the gallbladder. Macroscopically these changes are clearly visible as an impregnation of white-yellow colour in the mucosa or a gentle network. These original changes of the gallbladder are designated as cholesterosis. On appearance, they are described as a "strawberry bladder", and in view of features of histological research — as a "xanthomatous cholecystitis". Cholesterosis is found out in 43.7% of removed gallbladders in the patients with chronic calculous cholecystitis, and in case of gallstones with high contents of cholesterol it is even more often — in 77.4% of observations. Some authors consider cholesterosis to be a "prelude of cholelithiasis", as it is found out in gallbladders with absence of gallstones in them. Cholesterosis of the gallbladder is not a steady condition and with the lapse of time, it can disappear or its manifestations can become brighter.

Destabilization of a physical-chemical condition of bile with disorder of its colloid endurance is the central factor in modern representations about lithogenesis. Most completely, it is investigated on an example of formation of cholesterol gallstones (Yu. Kh. Marakhovsky, 1995). As a basis, the mycellar theory of bile cholesterol transport is used. According to this theory the formation of cholesterol gallstones is represented as follows: oversaturation of bile by cholesterol — change in correlation of cholesterol and its stabilisators concentration — crystallization (monohydrated cholesterol) —

synthesis of liquid crystals — saturation of crystals with cholesterol (cholesterol / phospholipids > 1.0) — crystallization of cholesterol — gallstones.

Thus, the development of cholelithiasis is connected with many factors and their combinations, but dyscholia and state of the gallbladder (stagnation, disorder of absorption and concentration of bile, inflammation) are of major importance.

Classification. Stones in the gallbladder are quite often found in the people who do not feel any anxiety and do not assume presence of concrements in their organism. Such people are called "stone-carriers" and we speak about asymptomatic cholecystolithiasis.

They distinguish the chronic form of cholelithiasis (chronic calculous cholecystitis) and acute (acute calculous cholecystitis).

Chronic calculous cholecystitis can become complicated by penetration of stones into the common bile duct (choledocholithiasis) with accompanying pathological changes of the pancreas (cholecystopancreatitis), the liver (satellite hepatitis), major duodenal papilla (constrictive papillitis), and development of an edema or chronic empyema of the gallbladder. They distinguish primary chronic calculous cholecystitis, residual — the residual phenomena after the acute attack, and chronic relapsing calculous cholecystitis with repeated pain attacks.

Acute cholecystitis is a dangerous complication of cholecystolithiasis, as it can cause development of very serious lethal complications. In the gallbladder, the process can get phlegmonous or gangrenous character, i. e. to become destructive. All this can cause local or diffuse peritonitis, acute cholangitis, a bedsore in the bladder with formation of a vesical-intestinal fistula and passage of a concrement along the intestine with danger of obturative intestinal obstruction development. An inflammatory infiltrate around the gallbladder can spread on hepatico-duodenal ligament and give rise to acute cholangitis or formation of the inflammatory stricture of the biliary tracts with development of a mechanical jaundice. Developed purulent cholangitis on the ground of choledocholithiasis can cause formation of single or multiple liver abscesses (cholangiogenic abscesses). All the time it is necessary to remember that the chronic inflammation in the gallbladder is considered a precancerous disease.

CHRONIC CALCULOUS CHOLECYSTITIS

Manifestations of cholelithiasis are extremely various. It is possible to single out a so-called latent form, when the carriers of gallstones do not have any complaints. Clinical manifestations of the disease sometimes develops gradually from mild pains in the upper half of the abdomen and dys-

peptic disorders, and sometimes initially from acute cholecystitis, cholangitis and jaundice, which depends on a sudden obturation of the vesical duct, penetration of stones into the choledoch, development of acute inflammation. Most typical manifestations of chronic relapsing calculous cholecystitis (CRCC) is hepatic colic. It has characteristic features. The pains arise acute, suddenly, after plentiful meal, use of greasy or spicy food, physical exertion, driving on a jolty road, emotional stresses etc. Pains are localized in the right hypochondrium, supraperitoneal region or spread to the whole zone of the epigastrium. Pains fast (20–30 min) reach its maximum, have a bursting or compressive character, become intolerant in majority of the patients. The pains radiate to the right shoulder, forearm, right half of the neck, under the right scapula, and sometimes get a surrounding character (cholecystopancreatitis). In 15–20% hepatic colic proceeds without a typical irradiation. The patients do not find a facilitating position — they change rising, walking. The pains are so intolerant, that the patients groan, quite often cry and beg for help. An attack of pains is accompanied by belching, vomiting, which does not relieve a condition. The body temperature sometimes rises.

During an attack of hepatic colic quite often pains in the field of the heart or arhythmia occurs; S. P. Botkin wrote that quite often cholelithiasis is expressed in the phenomena concentrating mainly in the field of the heart. These phenomena are designated as biliary-cardial syndrome of S. P. Botkin. Its essence is in a complex of disorders of a functional character in the myocardium (automatism, excitability, conduction), which arise against a background of metabolic and reflex influences. This syndrome is observed in 15% of the patients. It can be expressed in painful or painless form (arrhythmia, changes on the ECG). After cholecystectomy at least in 50% of the patients with syndrome of Botkin all unpleasant phenomena on the part of the heart disappear, and in others they are kept in connection with developed metabolic and miocardiotonic disorders, quite often already of age character.

The attack lasts usually for some hours, sometimes it stops itself, but more often it is necessary to resort to anesthetizing and antispastic agents, to call for ambulance. When attacks last during six hours, the patients are hospitalized in a surgical department. The mechanism and clinics of development of a pain attack in cholelithiasis in many respects depend on migration of gallstones and disorder of hydrodynamics in the biliary tracts. A highly sensitive zone is the area of the neck of the bladder and cystic duct. Small gallstones pass to the choledoch or depart into the lumen of the gallbladder, the permeability of the cystic duct is restored, the pain attack breaks. In steady obturation of the cystic duct, hepatic colic persists, the pains

accrue, the process progresses, and with activization of bacterial flora acute cholecystitis can develop.

In men, especially of young age, cholelithiasis develops with some features. The pains more often are localized in the epigastrium, instead of in the right hypochondrium, in 50% an atypical irradiation of pains (left arm, area of the heart, left subscapular area, loin) happens. The signs are more considerably expressed on the part of the gastrointestinal tract (nausea, heartburn, belching, vomiting, diarrhea). The correct diagnosis is not made for a long time, and the patients are treated for chronic gastritis, stenocardia, osteochondrosis etc. In 8–10% of cases the disease is revealed in connection with development of cardial syndrome.

The objective research of the abdomen at chronic uncomplicated calculous cholecystitis is directed on revealing painfulness in the right hypochondrium, in a projection of the gallbladder. The projection zone of the gallbladder — is the place of crossing of the outside edge of the *right musculus rectus abdominis* with a costal arch. Besides a usual palpation in the right hypochondrium, during which painfulness characteristic for cholecystitis, can be revealed, the special signs allowing more confidently revealing such painfulness are used.

Kehr's symptom is intensifying of tenderness at the top of inspiration at a usual palpation in the right hypochondrium.

Merfy's symptom is a sharp pain on inspiration with the previously entered fingers of the hand or only of large finger of the left hand in the right hypochondrium.

Ortner's symptom is a sharp pain at a mild knocking by the rib of the hand on the right costal arch. This symptom is also positive in acute hepatitis.

Mussy — Georgievsky's symptom (phrenicus-symptom) is tenderness on pressing above the right clavicle between sternal and clavicular attachments of the sternocleidomastoid muscle. This symptom is most pronounced at acute cholecystitis and hepatic colic.

Zakcharyin's symptom is pain on knocking by fingertips or pressing on the area of the gallbladder projection.

In some patients, the painfullness is found out at pressing by a finger on the right of VIII–X thoracal vertebrae.

In chronic cholecystitis, the gallbladder is not usually palpated, as it appears mostly diminished and sclerosed. The palpated enlarged gallbladder is in edema or chronic empyema.

By way of *differential diagnosis* in the patients with CRCC, the complete objective abdominal research with examination of all internal organs is carried out. The laboratory analysis of blood, urine and duodenal contents on CRCC usually does not reveal any deviations, which could confirm the

diagnosis. Hypercholesterinemia is found out quite often, but according to summary statistics no more than in 30–40% of the patients. Stones in the gallbladder are revealed with the help of USR. The condition of the gallbladder, its size and form, frame of the walls, biliary ducts — the width, presence of stones are estimated. With certain skills it is possible to diagnose cholesterosis and pseudopolyps connected with it.

In a permeable cystic duct the valuable information about cholecystolithiasis can be received with the help of cholecystography and intravenous cholegraphy. Presence or absence of stones in the gallbladder should be determined precisely today.

The development of CRCC is characterized by alternating of exacerbation with the periods of remissions. Single large stones may not manifest themselves for a long time. It is noticed that cholelithiasis is quite often combined with some other diseases. So, a well-known Saint's triad is cholelithiasis, hernia of the esophageal aperture of the diaphragm and divertuculosis of the colon. Vilk's triad is cholelithiasis, duodenal ulcer and chronic appendicitis.

ACUTE CHOLECYSTITIS

Among acute surgical diseases of the abdominal organs, acute cholecystitis occupies the second place after acute appendicitis. The patients with acute cholecystitis make up 15–20% of all patients hospitalized in surgical departments with urgent indications. In case of occurrence and development of acute cholecystitis a series of the factors play a role, but the main importance is given to cholestasis, infection, reflux of pancreatic enzymes under conditions of cholic hypertension and disorders of circulation in the wall of the gallbladder. A certain meaning can have the trauma of the mucosa of the gallbladder caused by cholic concrements.

Mechanical obstacles can cause bile stasis in the gallbladder: i. e. an obturation by concrements, mucus, parasites, cicatrical obstruction of the cystic duct, stenosis of a terminal department of the common bile duct and major duodenal papilla, duodenostasis etc. Motor dysfunction of the gallbladder owing to atony of its wall can promote these disorders, as it is observed at deficiency of intestinal hormones, (cholecystokinin — pancreozymin, secretin etc.) or dysfunctions of the vegetative nervous system. The basic consequence of stasis and cholic hypertension in the bladder is the decrease of a barrier function of the mucosa epithelium, which becomes permeable for microflora containing in the gallbladder.

The presence of microorganisms in the gallbladder is found out much more often than the attributes of acute inflammation, causative agents

of which are mostly intestinal rod, enterococci, staphylococci, streptococci, Proteus, typhoid rod and much less often representatives of nonclostridial anaerobes. The microbes can penetrate the gallbladder in enterogenous, hematogenous and lymphogenous ways. More often detection of causative agents in the wall of the gallbladder, instead of its contents, has given the basis to assume that the bile has bactericidal properties. However, this assumption has not been proved true by the subsequent researches. On the contrary, bile has appeared an excellent medium for intestinal rod and typhoid rod, which under condition of cholestasis and reflux of bile from *ductus choledochus* can become the basic way of penetration of infection in the gallbladder.

Doubtless influence in penetration of causative agents through the mucosa of the gallbladder have the mechanical damages by concrements, which are revealed in 80–90% of the patients suffering from acute cholecystitis, and acute purulent cholecystitis much more often occurs in these cases. The presence of pancreatic enzymes in bile does not cause destructive changes in the biliary tracts. However, under conditions of cholic hypertension and accumulation of pancreatic enzymes in system of the biliary ducts (that occurs, mainly, owing to organic or functional changes of the sphincteric apparatus in the field of the major duodenal papilla), the pancreatic juice destroys surrounding tissues, including the mucosa of the gallbladder, causing its necrosis and raising a porosity of other anatomic structures of the wall of the gallbladder. Uncalculus enzymatic cholecystitis with cholic peritonitis takes place in these cases.

In elderly and senile age, an important role in acute cholecystitis occurrence is played by circulation disorders in the wall of the gallbladder caused by atherosclerosis of its vessels (elastofibrosis of the intima, sclerosis of the muscular layer and thinning of adventition), disorder of the bloodflow in them which promotes clottage. With pathological changes of terminal departments of the vascular channel of the gallbladder, there are focal necrosises of the mucosa, and at clottage of large branches of the cystic artery, the diffuse ischemic necrosis of the whole wall of the gallbladder develops.

Pathological anatomy. The occurrence of acute inflammation of the gallbladder is frequently preceded by disturbed bile outflow, that results in fast overflow of the gallbladder, stretching of its walls and rising of intravesical pressure up to 500–600 mm H_2O (in normal conditions it does not exceed 200 mm H_2O). The walls of the gallbladder become hydropic, dark red colour with a cyanotic shade; there are signs of serous inflammation in them. On restoration of normal bile outflow, these changes disappear or get chronic course with periodic exacerbations. If the pathological process

progresses, serous inflammation turns into purulent one. Through Luschka's canals and lymph clefts, the causative agents of infectious process penetrate into deep layers of the wall of the gallbladder. It usually results in occurrence of a phlegmon, because of epithelial destruction, multiple ulcers penetrating in deeper layers of the wall of the gallbladder (acute phlegmonous-ulcerative cholecystitis) are formed. A purulent-inflammatory exudate mixes with bile and accumulates in the gallbladder. If the permeability of the cystic duct is not restored, the walls of the gallbladder become intense, the gallbladder is enlarged, empyema of the gallbladder is formed.

The destruction of tissues under conditions of intravesical pressure rising, results in perforation of the gallbladder and development of cholic peritonitis. However, a site of wall necrosis is more often covered with neighbouring organs (the colon, the large omentum, the liver, the loop of the small intestine, the parietal peritoneum) with formation of limiting fibrinous adhesions and perforation occurs like penetration in these organs. Purulent cholecystitis is also quite often accompanied by purulent inflammation of biliary ducts (purulent cholangitis), and also essential changes in the parenchyma of the liver.

Classification. Depending on the tasks which should be solved in different time various classifications of acute cholecystitis have been offered. The classifications taking into account morphological changes in the wall of the gallbladder, character of clinical course of disease and complications gained the greatest spreading. They distinguish calculous and uncalculous acute cholecystitis, however, in the solution of treatment-diagnostic tasks these differences have no large difference, therefore in many modern classifications they are not taken into account. Also many surgeons do not take into account whether the acute cholecystitis is primary (arisen with complete health) or secondary "as a relapse or exacerbation of disease at chronic relapsing cholecystitis".

According to the character of morphological changes in the gallbladder, they distinguish simple catarrhal cholecystitis; phlegmonous (phlegmonous-ulcerative) cholecystitis; gangrenous cholecystitis; perforating cholecystitis; empyema of the gallbladder.

According to development, they distinguish uncomplicated acute cholecystitis; complicated acute cholecystitis (peritonitis, cholangitis, hepatitis, obturative icterus, pancreatitis, paravesical infiltrate, paravesical abscess).

Clinical course. The pain localized in the field of the epigastrium or the right hypochondrium always accompanies acute inflammation of the gallbladder. The pain can be very intense, sometimes accompanying even by signs of collapse. Unlike hepatic colic, when pains are short-term or

have paroxysmal character with light intervals, in acute cholecystitis such intervals are not present; for pain syndrome in acute cholecystitis the irradiation of pains in the right shoulder, forearm, the right scapula and the interscapular space is typical. In case of the pancreas involving in the pathological process, the pain can have a surrounding character, to irradiate in the lumbar area. Sometimes pains are localized under the xiphoid process ("a symptom of the xiphoid process of Gubergrits"), sometimes, especially at localization of an occlusive concrement in the field of the gallbladder neck, the pains can irradiate to the area of the heart ("biliary-cardial syndrome of Botkin"). A slightest physical strain (cough, respiration, conversation) strengthens the pain. Simultaneously with pains quite often there is feeling of gravity in the right hypochondrium, combined with nausea and vomiting, though the latter, as a rule, does not give relief to the patient. The described above complaints can be accompanied by fever up to 38°C and more, sometimes with chill and mucous and dermal icterus marked on the 2nd–3rd day from the beginning of the disease. Unlike hepatic colic, in acute cholecystitis there are signs of intoxication, they are pronounced in dependence on gravity of morphological disorders in the gallbladder. The general condition of the patient in uncomplicated acute cholecystitis more often remains satisfactory and is worsened with complications. The pulse can be a little rapid, but of good filling, short breath, dryness in the mouth are usually marked. The superficial palpation of the abdomen finds out the increased resistance of the abdominal wall in the right hypochondrium and often acute tenderness in the field of projection of the fundus of the gallbladder (place of crossing of the right costal arch with the external edge of the *right musculus rectus abdominis*). In case of mild pressing by a palm of the hand in this area the deep inspiration owing to arising acute tenderness (Murphy's symptom) is impossible, that indicates to the inflammatory process in the gallbladder. Painfulness quite often is defined in knocking on the right costal edge (Ortner's symptom). In case of a deep palpation in this area sometimes it is possible to palpate an enlarged and acutely tender gallbladder, and at destructive forms — infiltrate is quite often defined.

In blood analysis leucocytosis with left shift is usually revealed, the increased contents of bilirubin in the blood serum and ESR acceleration are quite often defined. On perforation of the gallbladder the signs of acute diffuse limited peritonitis is observed. In doubtful cases it is used general X-ray examination of the abdominal cavity, which allows excluding other acute surgical diseases (perforated ulcer, intestinal obstruction), pneumonia. It allows to reveal indirect signs of acute cholecystitis (circumscribed inflation of the right half of the colon or its right flexure, pneumatosis of

loops of the small intestine with formation of small intestinal arches without levels of liquid in them, locating more often on the right according to the body of II–III lumbar vertebra). Sometimes on a roentgenogram concrements or homogeneous shadow in a projection of the gallbladder are visible. Intravenous cholangiography in acute cholecystitis usually reveals blockade of the gallbladder, which is not contrasted. However, this sign occurs in other diseases as well.

Recently X-ray examination in acute cholecystitis is superseded with USR allowing identifying not only concrements but also changes of the wall and sizes of the gallbladder.

CHOLEDOCHOLITHIASIS. CHOLANGITIS

Stones in the biliary ducts are found out in 10–15% of the patients with calculous cholecystitis. Choledocholithiasis is the presence stones in the general bile duct (choledoch). Really, in this department of the extrahepatic biliary ducts they are found out in overwhelming majority of cases, but with a great number of concrements they are also located in proximal departments of the biliary ducts, besides, stones easily move along them. The term "choledocholithiasis" in practice is applied in a more comprehensive sense, it means gallstones present in all departments of the extrahepatic biliary ducts.

Gallstones in the ducts can be of different size — from several millimeters up to several centimeters. Their number is different — from individual up to multiple (20–30 and more). Their form is also different. Gallstones or their parts in the ducts get from the gallbladder through the cystic duct, if it is not obliterated and not very narrow. In the biliary ducts gallstones themselves usually are not formed and this can be only in stenosis of the terminal department of the choledoch, getting of some helminths in it (ascaris lumbricoides, fasciola hepatica). It is possible also in a so-called Far East choledocholithiasis, in case of which in a certain region for unsufficiently realised reasons the bile with especially high lithogenic properties is produced and stones are formed already in the intrahepatic biliary ducts, then filling almost all the biliary system.

Gallstones can cause an occlusion of the choledoch, and then mechanical jaundice develops. Small gallstones pass in the intestine, larger ones can be restrained in the major duodenal papilla, which besides for icterus, causes acute pancreatitis. Gallstones, passing through the major duodenal papilla, cause constrictive papillitis owing to a repeated trauma. In disorder of

bile outflow its properties change, and so-called cholic substance with the subsequent formation of loose, spherical stones is formed in the ducts. Concrements getting in the choledoch from the gallbladder, usually have edges, which testifies to formation of several at once. Concrements in the ducts causing difficulties for bile outflow promote development of an inflammation in the ducts, i. e. cholangitis.

Acute cholangitis is manifested by icterus, high temperature of the body and chill (Charkot's triad). This is promoted by usually dyspeptic disorders. Repeated attacks of cholangitis can cause cholangiogenic abscesses of the liver.

According to clinical signs choledocholithiasis can be suspected in the patients with calculous cholecystitis only on occurrence of icterus during pain attack.

However almost 50% of the patients with gallstones in the ducts can have no icterus (nonicteric choledocholithiasis), that complicates diagnosis. At the same time in case of pronounced mechanical jaundice caused by occlusion of the biliary ducts by a gallstone, almost in 10% of cases there can be no pain syndrome. It is uneasy to distinguish choledocholithiasis if there is no icterus, connected with pain attack, but it is not less difficult to reveal stones of the biliary ducts in doubtless icterus, if there are no pain attacks. All this should be taken into account using objective methods of diagnosis and in some cases it is nesessary to continue investigation during operation (intraoperative cholangiography or USR etc.). Gallstones in the ducts can be also in case of concrements absence in the gallbladder (4.4%), in case of a wide cystic duct gallstones penetrate into the hepaticocholedoch in case of fistula formation between the gallbladder and the choledoch.

It is possible to suspect choledocholithiasis according to some features in the patients: when small multiple stones in the gallbladder are revealed, with mentioning about even a short-term icterus in connection with an attack of hepatic colic, with extended biliary ducts detected at USR, and, certainly, on examination of the patient under icterus condition, which has appeared at once after an attack of pains in the right hypochondrium. In such cases, even if before operation stones in the hepaticocholedoch were not found out, it is necessary to show persistence during intraoperative inspection to reveal a gallstone or convincingly prove its absence.

Choledocholithiasis leads to biliary hypertension and expansion of the biliary ducts, that quite often is surveyed as an indirect feature of probable stones in the choledoch. At the same time it was established that in 5.2% of the patients with chronic calculous cholecystitis concrements are revealed in rather narrow ducts.

All this dictates necessity of thorough examination of the patients with cholelithiasis for well-timed (before operation) revealing of gallstones in biliary ducts.

Objective diagnosis of choledocholithiasis is carried out with the help of USR, retrograde cholangiopancreatography (RCPG) and in a part of cases on cholegraphy. However, it is necessary to remember that in the icteric form of choledocholithiasis gallstones in the ducts are revealed on USR maximum in 81.1% of cases, and at nonicteric choledocholithiasis even less often — in 62.8%. Intravenous cholegraphy truthfully reveals gallstones in the ducts according to direct signs in 20.0–30.0%. A valuable method is RCP, but it is necessary to resort to it when it is impossible to reveal stones with other methods and usually in stable icterus. It is necessary to remember that at RCPG a contrast agent quite often gets in the pancreatic duct, that can cause acute pancreatitis.

Diagnosis of possible choledocholithiasis should be continued during operation (cholecystectomy). In this purpose intraoperative methods of biliary ducts condition estimation are used.

Constrictive Papillitis

The development of constrictive papillitis (CP) is connected with passage of small stones through the major duodenal papilla (MDP), that leads to fibrous and cicatrical changes down to complete stenosis of the papilla. These changes cause biliary hypertension, expansion of the biliary ducts, cholangitis. They distinguish three degrees of stenosis: I — presence of narrowing without functional disorders; II — a pronounced narrowing with insignificant (12–15 mm) expansion of the ducts; III — a pronounced stenosis with cholestasis. Clinical displays of constrictive papillitis depend on degree of stenosis. An isolated stenosis of MDP without a choledocholithiasis occurs infrequently. It is necessary to differentiate CP from organic stenosis of a terminal department of the choledoch, which chronic indurative pancreatitis leads to from papillomatosis of MDP and tumour (adenoma, cancer).

The diagnosis of CP is carried out with the help of fibroduodenoscopy, RCPG and during operation with the help of intraoperative methods of diagnosis, including intubation of the terminal department of the choledoch and MDP.

Spontaneous Biliodigestive Fistulas

Because of the gallstone's bedsore in the gallbladder and inflammation there can be anastomosis between the gallbladder and duodenum, less often — with the colon. In case of functioning and wide enough fistula it can be found out with the help of X-ray inspection with the usage of baric suspen-

sion. Narrowing or the cicatrical closing of a fistula causes cholecystitis exacerbation with the appropriate clinical manifestations. A pathognomonic sign of this complication is aerocholia (gas in the biliary tracts), determined at X-ray inspection.

Obturative Intestinal Obstruction Caused by a Gallstone

A gallstone, especially a large one, got from the bladder to the intestine, can cause obturative intestinal obstruction. Such complication is connected with retained acute process in the bladder, but can develop a bit later after stone penetration into the intestine.

Cholecystopancreatitis

The biliary and pancreatic system are closely connected anatomically and functionally. Therefore the inflammatory process in the gallbladder, and furthermore in the choledoch and the major duodenal papilla quite often leads to development of biliary pancreatitis, that complicates the disease course, is accompanied by additional signs (surrounding character of pains irradiation, enzymatic failure). After removal of the pathological center in the bladder (cholecystectomy) and ducts, in overwhelming majority of cases (90%) the signs of pancreatitis retreat.

Satellite Hepatitis

In chronic calculous cholecystitis special researches (regional biopsy of the liver during operation) reveal degenerative changes with all signs of chronic inflammatory process almost in 30% of patients. This hepatitis, accompanying pathological process in the biliary system, is designated as satellite, i. e. dependent on the process in the biliary system.

Hydrops of the Gallbladder

In chronic relapsing calculous cholecystitis in due course the obstruction of the cystic duct (stone, cicatrixes) develops almost in 60–70% of the patients. Therefore the gallbladder appears isolated from the basic biliary ducts. When bile is infected, the conditions for development of acute inflammatory process are formed. The repeated attacks of cholecystitis can result in thickening of the wall of the gallbladder, rough fibrous changes, therefore the gallbladder turns into a non-functioning cicatrical “saccule” filled with concrements. There an acute inflammation can develop at any time. In a flaccid infection course or in absence of microflora in the isolated

gallbladder, the glands of Lushka's ducts continue to function, the gallbladder is filled with serous-mucous contents. An intact wall of the gallbladder appears pliable to a stretching, that leads to enlargement of the gallbladder and hydrops. Usually a non-palpated gallbladder becomes accessible for a palpation and is palpated in the right hypochondrium as soft, elastic, painless, spherical formation with a smooth surface. Such a gallbladder can contain 250–300 ml of liquid contents. The bile elements absorb and the content becomes colourless. In case of infection, hydrops of the bladder develops. Hydrops of the bladder can be manifestation of acute cholecystitis.

Courvoisier's Syndrome

It is necessary to differentiate hydrops of the bladder with its enlargement in Courvoisier's syndrome. In case of this syndrome an intact gallbladder with a permeable cystic duct is enlarged in connection with a tumoral occlusion of a terminal department of the choledoch (cancer of the duct, head of the pancreas, adenoma or cancer of the major duodenal papilla), biliary hypertension accrues, mechanical jaundice develops, the bile loses its "staining pigments" and becomes colourless — a so-called "white bile". The clinical manifestations of cholelithiasis and its complications are diverse and a series of researchers aspired to systematize them. Some of them allocated the periods during disease — three or five — and formed medical management accordingly. The others wrote about phases of disease. The others payed attention to certain groups of the patients with stones in the gallbladder, coordinating it with a condition of the gallbladder wall, clinical and laboratory data, frequency of attacks of hepatic colic and signs of "isolated bladder".

All that was taken into account while determining the indications to surgical treatment. Striving for differential approach to cholelithiasis and its complications is clear, but excessive detailed elaboration and desire to "divide" development of disease into the periods and phases with transition from one condition into another is not practically justified. A point of view is right that even the most benign course of disease unexpectedly can develop the worst way. It is well known that asymptomatic cholecystolithiasis unexpectedly can be manifested by acute, destructive cholecystitis. Therefore, phases or periods of disease can concern only some part of the patients and naturally, without a guarantee of reliability of consecutive transition from one phase to another, from period to period. It is obviously important for us to know whether there are stones in the gallbladder or not, whether they can be responsible for clinical signs or not, what condition of the gallbladder is (isolated bladder), whether complication

of disease are obvious or latent (nonicteric choledocholithiasis). If the presence of gallstones is proved and there are no clinical manifestations of the disease, it is necessary to remember that at any unpredictable moment the danger of serious complications can become a reality. It is impossible also to forget that a prolong chronic inflammation in the gallbladder should be considered a precancerous condition, that is proved to be true by many observations.

Differential diagnosis in many respects depends on the stage of inspection of the patient. Now methods of objective diagnosis have improved so much that without appreciable loss of time it is possible to answer the basic question — whether there are concrements in the gallbladder or not. However, doctor should always remember that quite often stones in the gallbladder could be found by occasion like a sign of asymptomatic cholecystolithiasis. Therefore one should estimate attentively clinical signs and in necessary cases to continue examination of the patient. The list of diseases which is necessary to pay attention to on the preliminary stage of diagnosis is rather large and various. They are gastroenterologic diseases — chronic gastritis, gastroduodenitis, peptic ulcer of the stomach and the duodenum, hiatal hernia, reflux-esophagitis, chronic colitis, hepatitis and cirrhosis of the liver etc. Always it is necessary to remember about oncologic diseases of the stomach and pancreas as well as the liver and gallbladder.

When the disease develops with recurrent pain attacks, an opportunity of chronic relapsing pancreatitis, nephrolithiasis, intestinal colics due to spastic colitis or other diseases of the intestine, including neoplasms may be possible. The doctor should always remember about difficulties of revealing one of the common complications of calculous cholecystitis — choledocholithiasis. Stones in the biliary ducts long time can develop no symptoms, so choledocholithiasis can be asymptomatic and without icteric manifestation. Therefore, it is always necessary to take into account the width of the biliary ducts, as their dilatation (on USR more than 7–8 mm) means biliary hypertension and mechanical jaundice. But in doubtless cholangitis with jaundice, besides of such a common reason as choledocholithiasis, it is necessary to take into consideration constrictive papillitis, sclerosing (obliterating) choledochitis, ascaridosis of the biliary ducts, capitate pancreatitis, failure of the Oddy's sphincter, neoplasms (cancer, adenoma, papillomatosis) of the major duodenal papilla, more rarely of the head of the pancreas. At the same time it has been established that sometimes cholangitis can be the independent and only reason of the disease.

It is no necessity to list those methods of the objective diagnosis which should be known by the doctor working on the inspection of patients suffering from cholelithiasis, but one can not manage without USR, fibrogas-

troduodenoscopy and in some cases of roentgenoscopy of the stomach and the intestine. In many cases, and especially in icteric patients, RCPG is a highly informative method.

One must remember that it is not necessary to carry out differential diagnosis by the principle "or — or". In patients suffering from cholelithiasis accompanying diseases are rather frequently revealed, they must be detected in time, and if necessary treated as after cholecystectomy they can cause a so-called postcholecystectomic syndrome. The frequency of combination of cholelithiasis with other diseases is, for example, in mentioned above Sent's triad (cholelithiasis, hiatal hernia, and divertuculosis of the colon) and Wilk's triad (cholelithiasis, duodenal ulcer, and chronic appendicitis).

In case of differential diagnosis of cholelithiasis in patients with jaundice it is necessary to take into account some diseases of the liver.

Treatment of Cholelithiasis and Its Complications

In treatment of cholelithiasis, different methods are applied: cholelithotherapy (dissolution of stones), extracorporal lithotripsy (destruction of stones), cholecystectomy (surgical removal of the gallbladder), choledochotomy with removing calculi, biliodigestive anastomoses, endoscopic or open papillosphincterotomy.

The dissolution of stones is based on action of cholic acids (chenodesoxycholic and ursodesoxycholic) which are active ingredients of the preparations used in practice: Chenofalk (Germany), Chenochol (Yugoslavia), Ursan (Czech Republic), Ursofalk (Czech Republic), Oktaglin (Russia) etc. These preparations reduce the absorption of cholesterol from the small intestine, decrease synthesis of cholesterol in the liver, and probably stimulate the destruction of endogenic cholesterol-contaning lipoproteins. The treatment is carried out at the functioning gallbladder, permeable cystic duct, only at cholesterol stones, which should not fill more than a half of volume of the gallbladder. According to summary statistics, the favourable effect may be expected no more than in 10–15% of cases. In case of success, the treatment should be carried out permanently.

The devices creating a shock wave, very precisely focused on the revealed stone are used in extracorporal lithotripsy (ECLT). The necessary conditions the permeable cystic duct, a functioning gallbladder, cholesterol stones with the total size up to 30 mm or one stone of 20–30 mm. Fragments, formed during the destruction of the stone, leave the gallbladder for the common bile duct and then the intestine. The relapse of cholecystolithiasis and accompanying damages to the kidneys are probable.

Operative treatment. At the beginning of surgical treatment of cholelithiasis there was a hope of "ideal cholecystectomy", that means section of the gallbladder, erasion of a stone or stones from its cavity with subsequent stitchinging of the wall of the gallbladder tightly. Due to the almost inevitable relapse of cholelithiasis (sometimes in 10–20 years), they have refused this operation everywhere.

Cholecystectomy is recognized and is widely applied as a basic operation in treatment of calculous cholecystitis. The removal of the changed gallbladder with stones saves the patients from suffering the relapse of the disease and does not cause any new disorders in connection with erasion of the gallbladder.

Cholecystectomy is performed through the midline, subcostal, right transrectal or pararectal incisions, usually, from the neck, with separate ligation and cutting both the cystic duct and the cystic artery. The knowledge of Calot's triangle helps to be competent in anatomic structures. When the identification of the basic anatomic structures in subhepatic area is difficult due to several variants of development or considerable infiltrate, the gallbladder is removed "from the fundus", but it is always necessary to carry out cholecystectomy subserously. The bed of the gallbladder is coagulated with laser, plasma or electrothermal installations. On favorable conditions, the edges of serosa are stitched up. Sometimes it is necessary to stitch the bed of the gallbladder by noose sutures if it fails to stop bleeding. The operation is finished up by inserting a drainage into the subhepatic area.

During the operation in many cases of acute cholecystitis it is expedient to drain the extrahepatic biliary ducts through the stump of the cystic duct for relieving biliary hypertension and possible subsequent realization of cholangiography to control absence of gallstones in the ducts. The important and crucial stage of the operation is the intra-operative estimation of the condition of extrahepatic biliary ducts and first of all regarding to positions of probable choledocholithiasis and stenosing papillitis. During the operation, the ducts are examined to establish their width. Different measuring instruments, such as a caliper, small rulers are used. Compasses with blunt needles at the end may be also used. After gauging an outside diameter on the ruler, the outside width of the duct is determined. Characteristics of bile, excreted through the cystic duct, are also estimated. A series of special methods of research — intraoperative cholangiography, transillumination of the extrahepatic biliary ducts, choledochoscopy, intubation with plastic, metal as well as acoustic probes, roentgenotelevision choledochoscopy, USR of the biliary ducts are used. Palpation of the ducts has to be considered useful in the diagnosis, especially on the probe inserted through the cystic duct. Some surgeons attach a particular importance to this method in intra-operative diagnosis of choledocholithiasis. However, it is necessary to remember that the distal part of the common biliary duct passing in the corre-

sponding sulcus of the head of the pancreas and overlapped by the duodenum, is hardly accessible to palpation, and stones in this part of the duct can not be found out.

Intraoperative cholangiography (ICG) is of the greatest importance due to simplicity of its execution and high informativity. Through the cystic duct by means of a special cannula, the radiopaque substance (Cardiotrast, Urotrast etc.) is introduced and the photos are taken. The intraoperative cholangiography is a valuable method of diagnosis of stones in the biliary ducts. It may not be carried out only in case of undoubtedly revealed concrements on preoperative USR as well as in patients with the narrow ducts (on USR — 4 mm and less, external diameter is no more than 7 mm), if attacks of hepatic colic are not accompanied by jaundice. The well-grounded refusal from ICG is possible almost in 50% of patients. Recently the intraoperative inspection of the ducts has been simplified due to use of USR during the operation. Revealed stones (one or several) or proved suspicions are the reason for choledochotomy and removal of concrements; fenestrated forcepses, gall-stone scoops, tweezers are used.

The intubation of the major duodenal papilla is sure to be perfomed to estimate its permeability. Its width shouldn't be less than 3 mm. After erasion of all stones from the bile duct and sufficient permeability of the major duodenal papilla, the operation is finished up by outside drainage or stitching up the common bile duct. The best way to carry out drainage is by means of a T-like drain prepared for introducing into the biliary ducts. In its absence O. V. Vishnevsky's drainage is used. The drainage is inserted in a proximal direction. Blanket suture of choledochotomic aperture with the obligatory unwide unloading drainage in the absence of cholangitis and if calculi are sure to have been removed from the ducts is used. This drainage is necessary during the first 3–5 days after the operation, while the spastic stricture and edema of the major duodenal papilla and following hypertension in the biliary ducts persist. The advantage of external drainage of the biliary ducts is obvious in case of cholangitis, and consists in opportunity to remove residual stones, if they are revealed by control cholangiography after the operation. In case of obstruction of the major duodenal papilla, the operation is completed by choledochoduodenal or choledochojejunal anastomosis. The transduodenal papillosphincterotomy is also used.

Recently *laparoscopic cholecystectomy* (LCE) has been widely practiced, when a pneumoperitoneum is induced and troacars (usually four) are inserted. The laparoscope allows to examine the organs of the abdominal cavity with imaging the picture on the monitor. The gallbladder is removed by means of special instruments. The cystic duct and the cystic artery are clipped. It is possible to carry out cholangiography and if necessary, to remove a concrement through the cystic duct. If it is necessary, the operation is finished by drainage of the abdominal cavity.

The advantages of LCE are obvious: a reduced rate of serious postoperative complications, the spreading of adhesive process in the abdominal cavity is diminished, the period of postoperative treatment and rehabilitation is reduced, and danger of postoperative hernia development is decreased.

Indications for LCE are the same as those for laparotomic cholecystectomy. Contraindications for LCE are following: lung and heart disorders, disturbances in blood coagulation, diffuse peritonitis, inflammatory changes of the anterior wall of the abdomen, cases of late pregnancy, obesity of II–III degree. Local contraindications for LCE are evident cicatricial infiltrative changes in the neck of the gallbladder and hepatoduodenal ligament areas, mechanical jaundice and cholangitis, suspicion on malignant lesions of the gallbladder. In all these cases laparotomy should be planned.

Endoscopic papillosphincterotomy (EPST) is performed for removing a calculus from the common bile duct and drainage of the hepaticocholedoch in case of purulent cholangitis. Urgent EPST is carried out in the patients with mechanical jaundice caused by choledocholithiasis, before cholecystectomy, as the first stage of the operation.

A wide variety of surgical operations for treatment of patients with cholelithiasis requires to take into account specific individual indications in view of individuality in every case.

Establishing the indications for operative treatment of cholelithiasis one must take into consideration a real danger of severe complications development, impossibility and unsafety to dissolve or destroy stones in the gallbladder in the majority of patients. It is also necessary to remember that in 60–70% of cases of chronic relapsing calculous cholecystitis the gallbladder tends to be unfunctioning due to obstruction (blockade) of the cystic duct. Nowadays all the cases of calculous cholecystitis, which have left a latent phase, are subject to operative intervention. It is necessary to remember that an elective operation should be considered as prevention of possible complications and particularly of cholecystitis being still widespread.

With asymptomatic cholecystolithiasis some surgeons consider that cholecystectomy is admissible if there are no contraindications due to danger of the development of many complications. But one must remember that possible complications and even a lethal outcome may be due to the operation. Some surgeons consider that the operation is more acceptable after occurrence of clinical signs of the disease, even minimal. In case of numerous fine asymptomatic stones in the gallbladder, when a threat to migration of concrements through the cystic duct into the choledoch resulting in jaundice and cholangitis is very serious, one should insist on elective operative treatment. Active surgical treatment proved to be correct in case of asymptomatic cholecystolithiasis.

Chronic relapsing uncomplicated calculous cholecystitis is the indication for the operation. Long-term conservative therapeutic and sanatorium the-

rapy results in remission, but without full recovery. With the age and the increase of gravity of accompanying diseases the risk of the operation rises, threat of development of acute cholecystitis and urgency of operative intervention are always actual. Postoperative mortality in the individuals under 60 is 1.0–1.5%, and over 60 is 4–6%. It is also necessary to remember about Botkin's biliocardial syndrome and in the presence of cardiac complaints the indications for the operative treatment must be established urgently.

In case of permeable cystic duct and under appropriate conditions the choleolytic therapy and extracorporal lithotripsy are possible, but it is necessary to remember about unsafety of such treatment for the majority of patients, an actual relapse of the disease and strictly established indications and restrictions on these methods of treatment.

In case of chronic relapsing uncomplicated calculous cholecystitis cholecystectomy is performed. If there are no contraindications, it may be laparoscopic. Recently cholecystectomy through a mini-access is widely introduced, for this purpose the special instruments are created.

In case of hydrops of the gallbladder, even without the pain syndrome, an elective operation is also indicated due to danger of empyema development.

Chronic complicated calculous cholecystitis (choledocholithiasis, cholangitis, pancreatitis, constrictive papillitis, inflammatory stricture of the hepaticocholedoch, mechanical jaundice, biliocardial syndrome) may cause an aggravation of present complications and development of new ones due to a delay in operative treatment. Cholecystectomy is supplemented with interventions on the biliary ducts or the major duodenal papilla (choledochotomy, choledochotomy with removing calculi, biliodigestive anastomoses, papillosphincterotomy).

The patients with acute cholecystitis or suspicion on it are hospitalized to surgical departments where the diagnosis is confirmed, treatment management and first indications for an urgent operative intervention are determined.

At the in-patient department the treatment of acute inflammatory process is carried out and at the same time the preoperative preparation for a possible operative intervention begins: the use of spasmolytics (nospa, platyphyllin, halidor etc.), detoxicative therapy (intravenously: 5% solution of glucose, haemodesum, osmodiuretics, Ringer's solution etc.), antibiotics. The condition of the patient is estimated by clinical and laboratory findings: the total blood test (leucocytosis, the formula haemoglobin, erythrocytes, ESR), bilirubin, transaminases, thrombinogen, filtrate nitrogen, level of glucose etc. The patients are taken ECG, if necessary they are examined by the therapeutist, and chest fluoroscopy is made. The diagnosis is specified by means of USR allowing not only to find out stones in the gallbladder, but also to characterize its wall, which looks differrently in simple and destructive cholecystitis. The width of the hepaticocholedoch and probable pre-

sence of stones in it are determined. Recently a number of indications for early surgery, including operations at height of an acute attack tend to grow.

They distinguish emergency, urgent and early operations. It is important to determine a group of the patients, who should be operated on urgently — in some hours after admission, but not more than in six hours. During this period the diagnosis is clarified, the general condition of the patient is estimated, the presence of accompanying diseases is established, and a short-term preoperative preparation is carried out. This group includes the patients with acute cholecystitis complicated by peritonitis, when destruction of the gallbladder can not be excluded. These patients develop the symptoms of serious purulent intoxication commonly in case of gangrene of the gallbladder and its perforation.

The patients with obvious signs of acute cholecystitis even if uncomplicated by peritonitis, but with manifestation of aggravation of symptoms and intensifying pains in the abdomen, tenderness on palpation, febrile body temperature, increasing leucocytosis in the blood (10–12 thousand and more) and leucocytic index of intoxication are operated urgently as well. In such cases the operation should not be postponed in the hope that the symptoms may relieve, and it must be performed during the next 1 or 2 days (an urgent operation). Urgent and immediate operations are performed in 70% of cases. In other cases, the patients are operated on during the next 2–3 weeks after relieving an acute process (early operations) or they are hospitalized for surgery departments again after having been discharged from the hospital.

The operative treatment consists of removal of the inflamed gallbladder, inspection of the extrahepatic biliary ducts, including if necessary intraoperative cholangiography, and in the presence of indications (concrements in the hepaticocholedoch, cholangitis, jaundice, dilatation of the duct) choledochotomy is performed for removing calculi. The intervention is usually finished by external drainage of the biliary ducts or formation of biliodigestive anastomosis in case of persisting stenosis of the distal part of the common bile duct. In the absence of indications for choledochotomy, cholecystectomy in case of acute cholecystitis is recommended to be finished by drainage of the cystic duct after Holsted for clearing it up and relieving probable inflammation in the ducts. In the individuals of elderly and senile age, acute cholecystitis often has a symptom-free course, despite destructive process in the gallbladder. The indications for urgent operations in them should not be reduced.

For patients with acute cholecystitis accompanied by serious diseases, cholecystectomy may be extreemly dangerous. In these cases cholecystostomy or puncture of the gallbladder by means of a laparoscope, laparocentesis or laparotomy is carried out. This low-traumatic intervention relieves tension in the gallbladder. Its contents are evacuated, the gallbladder is washed out, on laparotomy, stones are removed and the drainage is inserted into the gallbladder. In many cases, cholecystostomy is the first stage of intervention, which is followed by cholecystectomy in favourable conditions.

Lecture 14
DISEASES OF ARTERIES

Anatomy and physiology. The aorta is divided into the ascending part *(pars aortae ascendens)*, the arch *(arcus aortae)* and the descending part *(pars descendens aortae)*, consisting of thoracal and abdominal departments.

The arteries participating in the blood supply of the brain and upper extremities branch off from the aortic arch (the brachiocephalic trunk, left common carotid and left subclavial arteries). The brachiocephalic trunk is divided into the right common carotid artery and the subclavial artery. The vertebral arteries branch off from the subclavial artery which pass into the axillary arteries. The axillary artery passes into the humeral artery, whose terminal branches are the radial artery and the ulnar artery. They in their turn are the place of origin of smaller branches providing blood supply of the hand.

The branches supplying the gastrointestinal tract (the celiac trunk, superior and inferior mesenteric arteries), the kidneys (renal arteries) and the lower extremities (the right and the left common ileac arteries) with blood, pass from the abdominal aorta. The common ileac artery is divided into the internal and external iliac arteries. The external iliac artery turns into the femoral artery, whose largest branch is the deep artery of the thigh passing from its lateral surface below the inguinal ligament. In the popliteal space area, the femoral artery passes into the popliteal artery, which is divided into the anterior and the posterior tibial arteries. The last is the place of the fibular artery origin. The terminal branches of these three arteries of the crus provide blood supply of the foot.

The walls of the arteries consist of three layers: the external layer, or the adventitia (*tunica extema)*, the medium layer *(tunica media)* and the inner layer *(tunica intima)*. The adventitia is formed by loose connective tissue — longitudinal fascicles of collagen fibers including elastic fibers, which are particularly noticeable on the border with the medium layer. The medium layer is composed of several parts of circularly disposed smooth muscular fibers, among which there is a network of elastic fibers forming along

with elastic elements of the adventition and the intima a general elastic framework of the arterial wall. The endothelium, basal membrane and subendothelial layer including thin elastic fibers and stellate cell form the intima of the artery. Behind it the network of thick elastic fibers forming the internal elastic membrane is posed. Depending on prevalence in the walls of vessels of one or another morphological element, the arteries of elastic, muscular and mixed types are distinguished.

Blood supply of the walls of the arteries is provided by arterial and venous vessels (vasa vasorum). Branches of small periarterial vessels provide supply of the arterial wall. They penetrate through the adventitia and, reaching up the medium layer, form a capillary network within it. The intima has no blood vessels.

The sympathetic and parasympathetic nervous system provides innervation of arteries. Chemo-, baro- and mechanoreceptors located within the walls of arteries play an important role in the regulation of vascular tonus.

Lymph drainage from the walls of the arteries is carried out through their own lymphatic vessels (vasa lymphatica, vasorum).

Immediate continuation of the arterial network is the system of microcirculation, uniting vessels with the diameter of 2–100 microns. Each morphological unit of the microcirculatory system includes 5 elements: 1) arteriole; 2) precapillar arteriole; 3) capillary; 4) postcapillar veinule and 5) veinule. In the microcirculatory channel, transcapillar exchange takes place, prividing vital functions of the organism. It is carried out on the basis of filtration, reabsorption, diffusion and microvesicular transport. Filtration takes place in the arterial department of a capillary, where the sum of values of hydrostatic pressure of blood and osmotic pressure of plasma on average is 9 mm Hg more than the value of oncotic pressure of tissue fluid. In the venous department of the capillary there is a reverse ratio of values of mentioned above pressures that supports reabsorption of interstitial liquid with products of metabolism. Thus, any pathological processes accompanied by hyperpermeability of the capillary wall for proteins, lead to the decrease of oncotic pressure, and consequently, to the decrease of reabsorption.

METHODS OF EXAMINATION

Inquiry

Usually even during inquiry of the patient can reveal symptoms related to circulatory insufficiency of one or another organ. Therefore, complaints about occurrence of pains in the legs when walking and their disappearance at rest (alternating lameness) allow assuming stenosis or occlusion of the arteries of

the lower extremities. Muscular weekness, sensation of numbness are manifestations of the peripheral circulation. Headaches can be a consequence of failure of cerebral circulation, especially when accompanied by giddinesses or episodic loss of sight, and abdominal pain at height of digestion — a sign of chronic disorder of the visceral circulation.

Physical Methods of Examination

The examination gives valuable information about the character of the pathological process in the majority of cases. In case of chronic ischemia of the lower extremities muscular hypotrophy usually develops, the filling of cutaneous veins decreases, the colouring of the skin changes (paleness, mottling), there are trophic disorders such as hair shedding, xeroderma, the thickenings and fragilities of the nails etc. In case of pronounced peripheric ischemia blisters filled with a serous liquid, dry (mumification) or wet (wet gangrene, necrosis of the distal segments of the extremity) are formed. The presence of pulsation synchronously with the pulse of the formation in one or another area (the neck, the stomach, the extremity) allows to suspect aneurysm of the vessel.

Palpation of pulse on the arteries is the major clinical exam in estimating condition of the arterial circulation in affected part of the body. The volume and force of the pulse are determined on symmetric sites of the head and the neck (temporal and common carotid arteries), on the upper extremity (the humeral and the radial artery), on the lower extremity (the femoral, the popliteal and the posterior tibial arteries, the dorsal artery of the foot). On palpation of the abdomen it is necessary to pay attention to pulsation of the abdominal aorta. The pulse examination is carried out at the following places: on the temporal artery — to the front from the tragus of the auricle; on bifurcation of the common carotid artery — behind the angle of the mandible, on the radial artery — on the palmar surface of the radial part of the forearm 2–3 cm more proximal than the line of the radiocarpal joint; on the humeral artery — in the internal sulcus of the biceps; on the dorsal artery of the foot — between I and II metatarsal bones; on the posterior tibial artery between the lower edge of the internal malleolus and Achilles tendon; on the popliteal artery — in depth of the popliteal fossa in lying position on the abdomen and with bent knees at the angle of 120°; on the femoral artery — below the inguinal ligament in 1.5–2.0 cm medially from its middle; on the abdominal aorta — on the medium line of the abdomen higher and at the level of the navel.

Auscultation of the vessels is an obligatory component of inspection of the patients. Normally the tone of a pulse wave is heard above main arteries, systolic murmur arises in case of stenosis or aneurysmal arteriectasia.

Auscultation is carried out above a projection of the carotic and subclavial arteries, the brachiocephalic trunk, the vertebral arteries, the ascending and abdominal aorta, the celiac trunk, the renal, the iliac and the femoral arteries. Murmurs from the left subclavial artery are auscultated behind the sternocleidomastoid muscle at the place of its attachment to the clavicle; on the right in the same point it is possible to detect a murmur from the brachiocephal trunk. Murmurs from the vertebral arteries are projected 2 cm more proximal than the middle of the clavicle, from the ascending aortae — in the second intercostal space to the right of the breastbone. On the abdominal midline under the xiphoid process, murmur from the celiac trunk of the aorta is auscultated in case of its stenosis. On the pararectal line in the middle of the distance between the horizontal lines imaginarily marked through the xiphoid process and the navel, murmur from the renal arteries is auscultated. On the medium line at the level of the navel and higher murmurs from the abdominal aorta are localized. Murmur from the iliac arteries is projected on the line connecting the abdominal aorta with the point disposed on the border of internal and medium third of the inguinal ligament. Auscultation of the femoral and common carotid arteries is performed in places where their pulsation is determined. On auscultation of the cervical vessels it is necessary to distinguish murmur due to artery stenosis from cardiac murmurs, whose intensity increases with approaching to the heart. In case of vessel aneurysms and arteriovenous fistulas, the maximal intensity of murmurs is detected in places of their localization.

The arterial pressure should be taken on all four extremities. Measuring the arterial pressure in the leg, a cuff of a mercurial sphygmomanometer is applied on the femur and in position on the abdomen Korotkov's sounds above the popliteal artery are auscultated. It is possible to determine only the systolic pressure, while palpating the first pulse fluctuations of one of foot arteries after exhaling air from a full cuff. Normally the pressure in the lower extremities is 20 mm Hg higher than in the upper ones. The difference in pressure in symmetric extremities of more than 30 mm Hg is a sign of arterial permeability disorder. The high systolic pressure in the upper extremities with its acute decrease or absence in the lower extremities is characteristic for aortal coarctation.

Functional Tests

Among the functional tests used in diagnosis of chronic arterial failure of the lower extremities Opel's symptom, Goldflam's test, Samuels's test, Panchenko's knee phenomenon, prelum of the nail bed symptom are the most wide-spread.

Oppel's symptom (plantar ischemia): paling of the sole of the affected extremity lifted up at an angle of 45°. Depending on speed of getting pale

the degree of circulatory disorder in the extremity can be established: in case of severe ischemia it comes during the next 4–6 sec.

Goldflam's test: a position of the patient on his back with his legs, raised above the bed. He carries out flexions and extensions in the talocrural joints. With disorder of circulation the patient feels fatigue in the leg at least in 10–20 movements. The observation of colouring of the plantar surface of the foot (*Samuels's test)* is simultaneously made. With severe failure of blood supply within several seconds there comes paling of the feet.

Panchenko's knee phenomenon is determined in a sitting position. The patient, having thrown back the sick leg on the healthy one, soon begins to feel pain in the gastrocnemius muscles, feeling of numbness in the foot, insensibility in the tips of the toes of the affected extremity.

The sign of prelum of the nail bed means that at prelum of the tip phalanx of the toe in the anterior or posterior direction during 5–10 sec in healthy people the formed paling of the nail bed is immediately replaced by normal colouring. At disorder of the circulation in the extremity the paling keeps for a long time. When the nail plate is changed, the colouring not of the nail bed, but of the nail platen is observed. In patients with the disordered peripheric circulation the white stain on the skin, formed as a result of compression, is disappearing for a long time.

Special Instrumental Methods of Examination

Ultrasonic methods of exam, computer tomography, rheovasography, thermography and angiography have the greatest informativity.

Ultrasonic flowmetria (dopplerography) is based on the effect of Doppler and consists of registration of fascicle of ultrasonic fluctuations reflected from the surface of the blood, flowing in the vessel. Dopplerogramms, written down from the peripheric arteries, give valuable information on the size of regional arterial pressure, linear rate of the bloodflow, that allows to judge about the degree of vessels affection and condition of the collateral circulation. With the help of advanced devices of new generation equipped with microcomputers, spectral analysis of doppler signals is carried out, the image (colour or black-and-white) of vessels is received on the screen of the display, the diameter of their lumen and outside contour, volumetric rate of the bloodflow are determined.

Computer tomography is based on reception of the level-by-level transversal images of a human body with the help of the X-ray tube, rotating around it. It allows to make visible the transversal sections of the aorta and ostiums of its branches (ileal, mesenteric, renal arteries, celiac trunk, bra-

chiocephal arteries), to estimate the condition of their walls, interrelation with surrounding tissue frames.

MR-imaging enables to carry out researches of the vessels in some mutually perpendicular planes without introduction of contrast agents and receive a detailed representation of the vascular channel condition.

Rheography is based on registration of fluctuations of electrical resistance of tissues, which varies depending on the blood volume in the extremity. A rheographic curve in the norm is characterized by abrupt and fast rising of the pulse wave, a precise top, the presence of two additional waves in the descending part (catacrotism). With its help it is possible to determine the transmission time of the pulse wave, the speed of the maximum blood containing the examined segment and a series of other parameters, among which the most informative is the size of the geographical index — a derivative from the relation of the amplitude of the basic wave of the rheographic curve to the height of the caliber signal.

Thermography is based on registration of the own infra-red radiation of one or another site of a human body and its transformations in electron pulses. With the help of the modern equipment it is possible to receive objective representation of heat intensity.

Angiography is necessary for exact topical diagnosis of pathological processes. There are the following kinds of angiography:

1. Closed arteriography, when a contrast agent is injected immediately into one of the peripheric arteries (femoral, humeral) by their puncture through the skin.

2. Aortoarteriography according to Seldinger, when a special radiopaque probe is inserted into one or another department of the aorta retrogradely through the peripheric artery (femoral, humeral), through it a contrast agent is injected and a series of snapshots are carried out, allowing to study changes of the aorta and its branches.

3. Translumbar aortography, when the aorta is punctated with a special needle at the level of XII thoracal or I lumbar vertebras, a contrast agent is injected and X-ray films are carried out.

With the help of angiographic installations of a new generation it is possible to receive digital, three-dimensional images of arteries after an intravenous injection of rather small doses of a contrast agent.

In most cases of vascular diseases it is possible to establish a correct diagnosis with the help of a routine clinical inspection. Special methods, as a rule, only detail it. Therefore at certain stages of inspection with a correct usage of clinical methods it is possible to refuse a series of exams. Instrumental diagnosis has doubtless priority during a preoperative preparation and a subsequent postoperative observation.

THROMBOSES AND EMBOLISMS

Thrombosis is a pathological condition described by formation of a clot of blood at any site of the vascular channel.

Etiology and pathogenesis. Essential conditions for occurrence of arterial thromboses are disorders of integrity of the vascular wall, changes in the system of hemostasis and retardation of the bloodflow. It explains high frequency of thromboses in the persons suffering from obliterating atherosclerosis, endarteritis, and diabetes mellitus. Quite often development of thromboses is promoted by damages of arterial walls in the places of bruises of soft tissues, dislocations and fractures of bones of the extremities, compression of the vascular fascicle by a tumour or hematoma. Acute arterial thromboses can be preceded by angiographic exam, reconstructive operations on the vessels, intraarterial hemotransfusions. Thromboses arise also against a background of some hematological and infectious (sapropyra) diseases.

In all the specified cases adhesion and subsequent agregation of thrombocytes is a corresponding reaction to damages of the endothelium of the vascular wall. The formed aggregates tend to further growth, that is connected with the influence of physiologically active substances liberated from thrombocytes (prostaglandins G2 and H2, thromboxan A2, ADP, serotonin, epinephrine). At the same time the intensity of formation of thrombocytic aggregates depends on ability of the endothelium to produce inhibitors of the agregation, in particular prostaglandin 12 (prostacyclin). Thrombocytic factors, liberated from platelets, and biologically active substances not only promote thrombocytes agregation but also lead to activation of the coagulating system of blood, reduction of its fibrinolytic activity. As a result fibrin threads adsorb on the surface of the unit, a net-like frame forms, which, detaining cellular elements of blood, turns into bloodclots. With significant oppression of a lytic link of the hemostasis system thrombosis can have a wide-spread character.

Embolism is occlusion of the blood vessel lumen by embolus, which is usually presented by a part of a thrombus which has come off from the basic source and migrates with the bloodflow in the blood channel.

Etiology and pathogenesis. In 92–95% of the patients the reasons for arterial embolism are heart diseases and first of all myocardial infarction (especially the first 2–3 weeks of the disease), complicated with serious disorders of the heart rate, acute or chronic aneurysm of the left ventricle.

The reason for embolism can be a rheumatic combined mitral defect of the heart with prevalence of stenosis complicated with intraatrial thrombosis owing to cardiac fibrillation. Arterial embolism arises also at subacute septic endocarditis and inherent defects of the heart.

Sources of emboluses can be thromboses formed in aneurysms of the abdominal aorta and in the greater main arteries (3–4% of patients with embolisms), ateromatous ulcers of thoracal and abdominal departments of the aorta.

As a rule, emboluses are localized in the field of branching or narrowing of the arteries. Embolism is accompanied with pronounced reflex spastic stricture of the arteries, that leads to formation of an oblong thrombus, which blocks the collateral network.

In cases of thromboses and embolisms of the main arteries of extremities and appropriate vascular pools there comes acute hypoxia of tissues, which basic reason is disorder of bloodflow in the vessels of the microcirculatory channel. In the affected tissues the excess of underoxidated metabolic products is formed, that promotes development of metabolic acidosis. The latter causes adhesion of thrombocytes and formation of thrombocytic aggregates in the lumen of capillaries, which increases severity of ischemia and creates favorable conditions for intravascular thrombogenesis. The increase of hypoxia has a negative effect for oxidation-reduction processes in the tissues, in which the contents of membranotoxins — histamin, serotonin, kinins, prostaglandins raising the permeability of cellular and intracellular membranes is enlarged, therefore a subfascial muscular edema develops. The changes of the cellular metabolism and destruction of cells conduct to disintegration of lysosomes with remission of hydrolyzing enzymes lysing tissues. The consequence of it is development of necrosis of soft tissues, underoxidated metabolic products causing metabolic acidosis, toxiferous products, potassium, myoglobin come into the general bloodflow from the ischemised tissues. There are serious morphological disorders of the cardiovascular system accompanied with deterioration of parameters of cardiac activity, disorders of the rhythm of cardiac contractions, rasping changes of the central hemodynamics. Circulatory hypoxia accrues; renal filtration is reduced.

Clinical picture and diagnosis. The signs of acute arterial obstruction have the greatest manifestation with embolisms. The beginning of the disease is characterized by pains in the affected extremity, which arises suddenly and becomes intolerable at embolism. In their parentage a paramount importance has spastic stricture of both the main artery, and the collaterals. After 2–4 h spastic stricture decreases and the intensity of pains is reduced a little. The feeling of numbness, coldness and sharp weakness in the extremity join pains. Dermal integuments of the affected extremity get pale colouring, which is further replaced by specific marbleness. The veins get empty (sign of a flute). Pulsation of the artery more distally from the localization of the embolus is absent, it is usually ampli-

fied higher than an embolus. The dermal temperature is considerably reduced, especially in the distal departments of the extremity. The pain and tactile sensitivity is simultaneously broken; and in the beginning superficial, and then also deep sensitivity is reduced. In the patients with serious ischemic disorders complete anaesthesia develops quite often, the function of the extremity is broken down to flaccid paralysis. In severe cases there comes sharp restriction of passive movements in the joints, muscular contracture sometimes develops. There is subfascial edema of the muscles, which is the reason of pain sensations during palpation. With progression of local signs the general condition of patients is also worsened. It is connected with entering of underoxidated toxic products from the ischemised tissues in the general bloodflow rendering pernicious influence on functions of the vital organs.

The essential influence on the clinical picture of the disease is rendered with the level of the occlusion, intensity of the arterial spastic stricture, degree of obturation of the arterial lumen with embolus, features of the collateral circulation and sizes of an oblong thrombus. An extremely serious clinical symptomatology is observed with embolism of aortal bifurcation. Suddenly megalgias arise in the lower extremities and hypogastric area irradiating in the lumbar area and the perineum. The "mottled" skin during the first 1–2 days spreads to the buttocks skin and lower departments of the anterior abdominal wall. In connection with dysfunction of the pelvic organs dysuric phenomena and tenesmi are possible. Pulsation on the femoral arteries is not determined, and the zone of disordered sensitivity reaches the lower departments of the abdomen. The motor function of the extremity quickly disappears, muscular contractures and irreversible changes in the tissues develop.

Clinical picture of acute arterial thrombosis reminds that one in embolisms, however, it is specified with more gradual development of signs. It especially concerns the patients suffering from obliterating diseases of peripheric arteries, at which quite often thrombosis of vessels arises against a background of advanced network of the collaterals. Only in the process of progression of thrombosis there occur special signs of intractable ischemia of the affected extremity.

There are three degrees of ischemia of the affected extremity at acute arterial failure, each of them is divided into two forms — "a" and "b" (V. S. Savelyev). With ischemia of 1a degree feeling of numbness and coldness, paresthesia occur; at 1b degree pains join. Disorders of sensitivity and active movements in the joints of extremities from paresis (IIa degree) up to paraplegia (IIb degree) are specific for ischemia of IIa degree. Ischemia of IIIa degree is characterized by necrobiotic phenomena, which are

indicated by subfascial edema at the IIIa degree and muscular contracture at ischemia of IIIb degree. The final result of ischemia can be gangrene of the extremity. Precise anamnesis data enable to put a correct preliminary diagnosis. If the patient with heart disease complicated by cardiac fibrillation or aneurysm, suddenly has megalgias in the extremity, diagnosis of arterial embolism is doubtless. At the same time acute disorder of circulation in the affected extremity in the patients suffering before from one or another obliterating disease of the vessels, allows to assume arterial thrombosis.

The most informative methods of diagnosis of acute arterial obstruction are ultrasonic flowmetry and angiography, which allow to determine the level and the extent of occlusion. Angiography gives additional information about the pathological process (embolism, clottage), the condition of the collaterals. Absence of contrasting of the affected site of the main artery having equal smooth contours and a weakly expressed network of the collaterals belongs to angiographic attributes of embolism. With incomplete obturation of the artery an embolus is traced as oval or spherical formation, streamline with contrast agent. Attributes of organic defeat of the arteries (roughness, change of contours etc.) are revealed in case of acute thrombosis.

Treatment. Thromboses, embolisms of the abdominal aorta of the main arteries of the upper and lower extremities are the absolute indication to surgical treatment. It is caused by inability of the conservative therapy to result in a complete lysis of thrombus and embolus, which frequently represents thrombus with the phenomena of organization. The conservative treatment can be administered only to the patients who are in an extremely serious condition but with sufficient compensation of circulation in the affected extremity. It should be directed on elimination of the factors promoting ischemia progression. It is necessary to include preparations having thrombolytic, anticoagulant, desagregative and spasmolytic action in a complex of medical measures.

At the same time medical preparations improving microcirculation and central hemodynamics as well as eliminating metabolic disorders should be simultaneously prescribed.

Now such thrombolytic preparations as fibrinolysin, streptokinase and its analogues: streptase, streptoliase (avelysin), streptodecase etc., as well as urokinase are used.

Fibrinolysin represents profibrinolysin of the blood plasma activated by trypsin. The preparation is dissolved in the isotonic solution of sodium chloride and is injected intravenously droply 20,000–30,000 U 2 times per day with a 12-hour interval. Simultaneously heparin is prescribed, which is added

to the solution at the rate of 10,000 U to every 20,000 U of fibrinolysin. Heparin is injected in breaks between infusions of fibrinolysin. Alongside with usual unfractioned heparin, a more effective low-molecular heparin is used last years, which is injected 1–2 times per day. Treatment is carried out under coagulogramm control, and if impossible, they use blood coagulation time, which should exceed the top level 2–2.5 times. In 3–5 days in case of achievement of clinical effect, combined application of fibrinolysin and heparin is finished, only injections of heparin, and then indirect anticoagulants are continued. The efficiency of fibrinolysin is low, as it is quickly neutralized by antiplasmin contained in the blood plasma. Using the preparations, immediately activating profibrinolysin (plasminogen) — streptokinase and urokinase — is much more effective.

Streptokinase (the product of vital activity of hemolytic streptococcus) is an indirect activator of plasminogen, urokinase — a direct activator of plasminogen liberated from urine. The preparations are injected intravenously droply (systemic thrombolysis) or intraarterially (selective thrombolysis) during several days till the clinical effect is achieved. The disadvantage of the existing technique of systemic thrombolysis is great expenses for fibrinolytic agents and often hemorrhagic complications. The technique of selective thrombolysis is more perspective, it consists of arterial puncture, installation of the infusion catheter above the thrombus or in the thrombus with a subsequent infusion of small doses of thrombolytic. Unlike to streptoand urokinase a Ukrainian drug Streptodecase requires only unitary intravenous or intraarterial injection in a dose of 3,000,000–9,000,000 U.

One of the most effective anticoagulants of direct action, used for treatment of acute arterial obstruction, is heparin. It interferes with formation of thrombin, blocking process of coagulation of blood. Anticoagulant effect of heparin comes at once after intravenous and in 10–15 min after intramuscular injection and proceeds during 4–5 h. The daily dose of heparin is 30,000–50,000 U. Heparinotherapy is effective if the blood clotting time exceeds an initial level 2–2.5 times. At overdosage of heparin there can be the hemorrhagic syndrome, which is eliminated by injection of 1% solutions of protamine sulfate, 1 mg of which neutralizes effect of 100 U of heparin. Heparin therapy continues during 7–10 days and 2 days before withdrawal, indirect anticoagulants are prescribed. Thus, a daily dose of heparin gradually reduces 1.5–2 times at the expense of decrease of its single dose.

Indirect anticoagulants (neodicumarin or pelentan, syncumar, phenilin etc.) suppress biological synthesis of thrombinogen in the reticuloendothelial system of the liver. Their action begins in 18–48 h from the beginning of the usage and is kept during 2–3 days after the drug withdrawal. The criterion

of efficiency of the used therapy is the size of the prothrombin ratio. The optimum dose of the preparation is the dose, which reduces the prothrombin ratio up to 35–40%. The earliest sign of overdosage is microhematuria. An antidote of indirect anticoagulants is vitamin K (vicasol).

The increasing of adhesive-agregative functions of thrombocytes in the patients with acute arterial obstruction makes necessary prescribing of drugs having desagregative action, in particular of trental and curantyl. Specified agents render the greatest effect with intravenous introduction. It is expedient also to use a low-molecular dextran — rheopolyglucin, improving microcirculation, having desagregative properties and intensifying fibrinolysis.

It is more preferable to inject antispasmodics (nospa, papaverine, halidor) intravenously. For removal of pains and psychomotor exaltation analgetics, fentanyl, droperidol, sodium hydroxybutyrate are prescribed.

Metabolic acidosis, which quite often develops in the patients, requires the control after acid-base condition indices and their well-time correction with injection of sodium carbohydrate 4% solution. For the improvement of metabolic processes in the tissues, vitamins, complamin, solkoseril are expedient. With certain indications cardiac glycoside and antiarrhytmic drugs should be prescribed.

Surgical treatment of acute arterial embolism consists of removing embolus and thrombotic masses from the arterial lumen with the help of Fogarty's catheters. The catheter is a flexible elastic conductor with the diameter of 2.0–2.5 mm with graduations on it. On one end there is a pavilion for connection of a syringe, on the other — a latex cylinder with a thin director. At lesion of the arteries of the lower extremities they expose bifurcation of the femoral artery, and at obturation of the arteries of the upper extremities — bifurcation of the humeral artery. Then they carry out transversal arteriotomy and advance a catheter to the place of the occlusion of the vessel, getting it through the thrombotic masses. Then with the help of a syringe they inject liquid, fan the cylinder and take the catheter out. The full cylinder carries away behind itself the thrombotic masses, and at restoration of permeability of the artery from the arteriotomic aperture there is a jet of the blood. The incised artery is sutured. With the help of a cilinder catheter the thrombotic masses can be removed not only from the peripheric arteries but also from aortal bifurcation. With severe ischemia accompanied with edema of the muscles (IIIa degree) or muscular contracture (IIIb degree), with the purpose of decompression and improvement of the tissue bloodflow an additional fasciotomy is administered.

With acute thrombosis developed against a background of organic defeat of the arterial wall, simple thrombectomy is usually noneffective, as rethrombosis of the vessel comes quickly. Therefore it should be supple-

mented by a reconstructive operation. Ablation is indicated with gangrenosis of the extremity.

Prognosis. With late diagnosis of acute arterial obstruction and delayed rendering of qualified medical care the prognosis is adverse.

EMBOLISM OF THE PULMONARY ARTERY

Etiology and pathogenesis. This term designates a syndrome caused by a complete or partial occlusion of the pulmonary artery or its branches by embolus, a clot of blood formed in the venous channel with thrombosis of veins (thromboembolism), drops of adeps (fatty embolism) or bubbles of air (air embolism). Thromboembolism of the pulmonary artery or its branches caused by moving of thrombus which has come off by the bloodflow in the pulmonary artery occurs the most often. It is one of the most frequent reasons of a sudden death. According to autopsy data, its rate ranges within 4.4–14.7%. Thrombosis of veins of the shin, femur and pelvis is the most often source of emboluses at thromboembolism of the pulmonary artery. Much less often the reason of formation of emboluses in the venous system is thrombosis of veins of the upper extremity or formation of thrombuses in the right departments of the heart.

In most cases (89%) a thrombus begins to form in the shin — rather large cavities, blindly ending in the gastrocnemius muscles, connecting with deep veins of the shin. At movements as a result of contraction of the gastrocnemius muscles the sunuses are passively filled with blood and get empty. When the patient lies without any movements, congestion occurs in these sinuses and thrombuses form. They enlarge, spread to the veins of the shin, and under adverse conditions — to the veins of the femur and ileal veins. Thrombi localized in the shin and veins of the shin in most cases (up to 80%) are exposed to spontaneous lysis and only in 20% they spread to the veins of the femur and higher. Therefore thrombi localized only in the veins of the shin, seldom result in clinically important embolism of the pulmonary artery.

Thrombogenesis in the tibial veins begins already on the operating table in most cases. This is promoted with venous congestion as a result of muscles relaxation and the motionless position of the patient; hypercoagulation arising as a result of tissues trauma and tissue thromboplastin liberation; damage of the endothelium of the veins owing to overstraining of their walls (in connection with decrease of the tonus of the muscular membrane) and exposure of collagenous fibers of the venous wall.

The emboli can close only branches of the pulmonary artery or its basic trunks. Depending on it, the large or small part of the vascular channel of the lung ceased its function in circulation. Accordingly there are distinguished small, submassive, massive (two and more lobar arteries) and fulminant, or lethal, embolism, when occlusion of the basic trunks of the pulmonary artery takes place with exclusion from circulation more than 50–75% of the vascular channel of the lung. After embolism of branches of the pulmonary artery, pulmonary infarct or infarct-pneumonia develops in 10–25% of cases.

Occlusion of the pulmonary artery and its branches results in sharp rising of pressure in the pulmonary artery owing to rising of resistance to blood flow. It entails overloading of the right ventricle of the heart and right ventricular failure. At the same time cardiac emission and arterial pressure decrease, the gas exchange in the lung is broken and anoxemia occurs. Specified disorders reduce the coronary bloodflow, that may result in left ventricular failure, pulmonary edema and death.

Clinical picture and diagnosis. According to the size of occlusion of branches of the pulmonary artery (according to the data of angiography) and clinical signs there are 4 degrees of gravity of thromboembolism of the pulmonary artery.

The small and submassive thromboembolism of the pulmonary artery (I and II stages) is shown by insignificant clinical signs sometimes as pneumonias or pleuritis. Massive thromboembolism (III stage) is accompanied by serious depressed condition, and fulminant (IV stage), when more than 50% of the lung arterial channel is expelled from the circulation, is finished by death within several minutes. Classical signs of thromboembolism of the pulmonary artery are: sudden sensation of air shortage (tachypnea, dyspnea), tussis, pneumorrhagia, tachycardia, stethalgias, cyanosis, moist rale, pleural rub; enlargment of the cervical veins; fever and collapse. Depending on degree of severity the pulmonary-pleural, cardial and cerebral syndromes are allocated. The pulmonary-pleural syndrome more often arises with small and submassive thromboembolism, i. e. in occlusion of peripheric branches of the pulmonary or the lobar artery. It is shown by dyspnea, stethalgias (more often in its lower departments), tussis sometimes accompanied with expectoration with an impurity of blood. The cardial syndrome is more typical for massive thromboembolism: pains and feeling of gravity behind the breastbone, tachycardia, enlargement of the cervical veins, amplified cardiac beat, accent of II tone on the pulmonary artery, rising of the central venous pressure, shock, loss of consciousness. The cerebral syndrome is concerned with hypoxia of the brain, which is observed more frequently at elder patients, is detected by loss of consciousness, cramps, hemiplegia, involuntary excretion of urine and feces. The mentioned syndromes and

signs can be observed in different combinations depending on massivity of thromboembolisms.

Diagnosis of pulmonary artery thromboembolism is difficult. Life-time diagnosis is carried out only in 30–40% of observations, so a correct diagnosis is more often an exception than a rule. X-ray examination is unsufficiently informative. The most important signs are high location of the diaphragm, a shadow in basal segments, (atelectasis, infarct-pneumonia), pleural exudate. A normal roentgenogram of the lungs does not exclude embolism. Computer tomography can give more information, especially with infarct-pneumonia, exudate in the pleura.

ECG can reveal signs of ischemia of the right ventricle, shift of heart electrical axis to the right, right crus of His' bundle blockade, disorder of rhythm. Ascent of ST segment above the isoelectric line, negative wave in III abduction and decrease of the segment ST in I and II abductions are considered typical. Absence of the listed data does not exclude embolism of the pulmonary artery.

Selective angiography of the pulmonary artery allows diagnosing most authentically as this method is high sensitive and specific. On roentgenograms in anterioposterior and lateral projections the direct signs of thromboembolism of the pulmonary artery are revealed: the image of a thrombus, intravascular defects of fillings caused by its presence, complete obturation of the vessel with its expansion more proximal than occlusion and absence of vessel contours — more distally. Indirect signs are important for diagnosis too: decrease of blood contaning peripheric sites of the lung more distally than occlusion, elongation of arterial phase owing to rising of peripheric resistance of the lungs vascular channel, asymmetry of vessels filling. During angiography it is possible to measure the pressure in the pulmonary artery and to destroy a thrombus by the end of the catheter, i. e. to make recanalization and begin treatment with anticoagulants and thrombolytic drugs. Even the seriously ill patients endure angiography well.

The perfused and inhalating scintigraphy allows to find out changes concerned with embolism of the pulmonary artery in 90% of cases. However, these methods are not so informative as angiography. Detailed instrumental research at suspicion on pulmonary artery thromboembolism is expedient after a preliminary injection of 10,000–15,000 U of heparin.

Treatment. The basic purpose of treatment is to save the patient's life, restoration of bloodflow in the pulmonary artery.

Extent of medical measures is determined by massivity, severity of embolism. For the first time it is necessary to place the upper part of a patient's body in a raised position, to begin oxygen insufflations through the nasal catheter (not less than 3 L/min), to inject anesthetizing drugs and

sedatives, to establish a catheter in the subclavial vein for immediate introduction of heparin (10,000 U instantly and further up to 40,000 U per day), intensive antishock therapy and measurement of the central venous pressure. In submassive embolism (II degree) it is expedient to prescribe cardiac and antiarrhytmic agents, antibiotics for prophylaxis of infection. In massive thromboembolism (III degree) with pronounced manifestations and tendency to aggravation of condition it is expedient to supplement heparinotherapy by fibrinolytic therapy (streptase, streptokinase, urokinase, kabikinase etc.). Angiography and recanalization of embolus by a catheter are indicated. Antishock measures should be complemented by introduction of corticosteroids (prednisolon, hydrocortisone etc.). All medicinal substances are injected droply intravenously together with solutions of polyglucin, rheopolyglucin, glucose-novocainic admixture.

In massive and especially fulminant embolism progressing against a background of a severe shock alongside with fibrinolytic therapy operative erasion of embolus — embolectomy — is possible (Trendelenburg's operation). The operation is carried out in case of inefficiency of antishock therapy and presence of contraindications to fibrinolytic therapy. Embolectomy (including conditions of artificial circulation application) is accompanied by high lethality. Removal of embolus by a special catheter with a sucker on the end, which allows to remove embolus through the caval vein and the chambers of the heart, is safer.

Prognosis. At I and II degree of embolism and adequate treatment prognosis is favorable, at III and especially IV degree lethality is high, as an adequate help, as a rule, is late.

Relapsing chronic embolism of the pulmonary artery is treated by indirect anticoagulants is indicated, and in case of microembolism reoccurrence a special cava-filter, which detains emboli, is implanted in the inferior caval vein. The filter is entered through the bulbar vein. It is possible to block the inferior caval vein with the help of a special device (a cava-clip) which divides the lumen of the vein into narrow canals, which do not pass emboli to the heart.

Prophylaxis of thromboembolism development consists in prophylaxis of phlebothromboses of the lower extremities. Preventive measures should be directed on prevention of venous blood stasis in the veins of the lower extremities, elimination of hypercoagulation, and decrease of thrombocyte aggregation at their contact to collagenous frames of the vein wall.

Venous stasis is removed by early physical exercises after getting up, transition of the patient on a common regimen, application of dosed elastic compression of the shins and feet with the help of elastic bandages, socking

by intermittent pneumatic compression of the shins and femurs with the help of special apparatuses with inflatable cuffs, which are put on the legs.

Early getting up, physical exercises in the bed (flexion and extension of the feet imitating walking, movement in the knee and hip joints etc.) have received the greatest spread, they improve bloodflow in the extremities and prevent congestion.

At the same time the legs are bandaged by elastic bandage or elastic sockings fit by size are used specially. Intermittent pneumocompression of the legs is applied less often. As a rule this kind of influence takes place in the operation room and continued after operation in patients with the increased risk of deep vein thrombosis (operation on the hip joint), and in oncologic patients with episodes of vein thrombosis in the disease hystory. The efficiency of elastic compression is caused by prevention not only venous congestion in the cruses, but also overdistension of vein walls, damage of their endothelium. This method is frequently combined with other ones, for example with decreasing of blood hypercoagulation.

For decrease and elimination of hypercoagulation the drugs which reduce intensity of thrombocyte aggregation are used — antiaggregants (acetylsalicylic acid, indomethacin, curanthyl etc.), anticoagulants (unfractionated and low-molecular heparin, polyglucin). Heparin is usually introduced subcutaneously in small doses (up to 2,500 U 4 times per day). Low-molecular heparin (fragmin, fraxiparin etc.) is highly effective, as well as unfractionated heparin. The dose of the drug comes to 5,000 U/day. A single application of the drug is preferable, because it is no need in the special control of blood coagulability. Preventive injection of heparin proceeds for 5–9 days. Polyglucin with molecular mass of 70,000 and 40,000, that influences immediately on fibrin formation, prevents from cross-bonds in its molecule. As a result, the formed fibrin is easealy exposed to lysis under the action of natural fibrinolytic activity of blood. At application of polyglucin it is necessary to remember about side effects (allergic reaction, renal failure, hypervolemia with a possible pulmonary edema).

The combined way of prophylaxis of pulmonary artery embolism and deep vein thrombosis allows reducing up to a minimum the rate of deep vein thrombosis of the lower extremities and lower the risk of pulmonary artery embolism.

Lecture 15

DISEASES OF THE VEINS

VARICOSIS OF THE LOWER EXTREMITIES

Etyology and pathogenesis. Among numerous etiological factors of varicosis development heritable predisposition plays the leading role. Two known factors are inheritally transmitted, one of which can prevail: disorder of collagen ratio and elastin in morphological structure of the vein wall and poor providing of veins by valves. Inherent anatomic inferiority, structural-morphological feature of the venous wall and the valval apparatus cause poor resistance of these structures to rising of intravascular pressure: the walls of veins are excessively stretched, that results in failure of valves and retrograde reflux of the venous bloodflow.

With combination of the both factors the disease can progress with early occurrence of its first signs and prompt development. According to the first sign, the disease stands closer to group of collagenosis, according to the second one — to the category of angiodysplasias. Collagenosis and angiodysplasia are hereditary diseases. However, the affinity of varicosis to these versions of inherent pathology can not be the reason to prefer one of them as the basic etiological factor. Varicosis arises only in a human; the morbidity among other representatives of fauna is not observed. Until now, it was not possible to frame a pilot model of varicosis. In a human, the veins of the legs are mainly injured, and the relationship of the disease with vertical position and orthostatic venous hypertension is obvious. Orthostatic hypertension is a physiological phenomenon; pathological manifestations arise with disorder of the venous valves function, when pathological retrograde bloodflow arises in superfical and deep veins.

Pathological manifestations of dynamic venous hypertension can arise owing to shunt of the arterial blood in the venous system (inherent arteriovenous fistulas) at the precapillary level (arterial types of varicosis).

Descending and ascending types of varicosis can be distinguished (V. S. Savelyev, 1972). With the descending type of disease, descending bloodflow has the basic pathogenetic role, caused by disorders of hemodynamics in the system of the vena cava inferior connected with its morphofunctional specificities.

The ascending type is generally caused by disorders of bloodflow because of "pumps" function disorganization of the foot and the shin. In these cases, the damaging influence of venous hypertension mostly should be shown in the area over the shin, zone of normal bloodflow from deep to superficial veins along the overshin communicant veins. The strikes of retrograde bloodflow waves on zones of microcirculation in the area of the communicant veins result in sharp disorders of microcirculation and trophic changes of tissues under condition of hypertension.

Retrograde bloodflow causes overdistension of vessels and progress of relative incompetence of venous valves creating a zone of hypertension above the valve. Obviously, valval cusps also change in the process of retrograde bloodflow, and the damaging influence of venous hypertension on condition of microcirculation amplifies, microcirculation disorders underlie pathological changes in the hypodermic fat and dermal integuments.

The evolution of disorders of venous hemodynamics in varicosis can be submitted as a series of changes increasing after a cascade type. Two pathogenetic mechanisms are leading in development of varicosis: the shunt of blood in superficial veins through the ostium of the large and small hypodermic veins that was described by B. K. Brodie (1846) and through the perforant veins. It may promote structural changes of deep veins (ectasia, valval failure), occurrence of not only of "chambers of strain" but also vertical reflux of blood as well in the system of deep veins. Development of pathological veno-venous shunts with reflux of blood through the sapheno-femoral, sapheno-popliteal anastomosis and through perforant vein causes formation of total venous hypertension within all the leg. The last stage of varicosis is characterized by development of microcirculatory disorders, mainly in the venous part of the capillary bed, arteriolo-venous shunting, autosensibilization, dermatosclerosis, eczema, dermatitis, formation of trophic ulcers and other complications.

Morphological changes. The morphological examinations with the help of light microscopy allow retracing the development of pathological changes in various layers of the venous wall in varicosis. The majority of the authors allocate three stages of their development:

1) compensation;

2) initial or uncomplicated decompensation;

3) complicated decompensation.

At the stage of compensation, compensative, adaptative mechanisms work in reply to rising hydrostatic pressure inside the vessel: morphological

changes on this stage are characterized by swelling, hypertrophy and hyperplasia of smooth muscular cells, by moderate growth of connective elements, plethora and hypertrophy of the venous wall. Later there is a partial destruction of smooth muscular and connective elements and growth of fibrillar connective tissue with replacement of elastic fibers on collagenic ones.

The opportunities of electronic microscopy have allowed to estimate the role of internal environment of veins in occurrence and development of varicosis, as the structure of the vessel internal surface substantially allows to consider the changes occurring in the vascular wall. According to the data of electronic microscopy, the internal surface of a vessel in varicosis is not a smooth-wall cylinder, but a complex relief formation. The damage of the endothelium owing to chronic venous stasis and hypoxia of the venous wall results in pathology of smooth muscular cells and further development of pathological process (I. A. Tchalisov, 1988).

Transformation of smooth muscular cells function under conditions of varicosis is of importance in pathogenesis of the disease. These cells get properties of phagocytes capable to blast and absorb some of collagenic fibers.

Clinical classification. Depending on varicosis localization expansion in the pool of the great saphenous vein, varicosis in the pool of the small saphenous vein, varicosis of the lower extremities lateral surface and combined defeats of the listed above areas can be allocated.

The following stages of venous outflow disorders are distinguished (V. S. Savelyev et al., 1972).

1. Compensated — varicosis without clinical attributes of circulation disorder.

2. Subcompensated, which is characterized by occurrence of pains, edemas.

3. Decompensated "A" is constant pallor of the shins, pain, edemas, pronounced trophic disorders of the skin (induration, pigmentation, eczema).

4. Decompensated "B", when the trophic ulcers join the above-mentioned clinical attributes.

A. N. Videnskiy (1978) allocates the following criteria of the clinical characteristic of disease:

I. Form (localization of veins morpho-functional changes):

— deep (deep veins);

— superficial (subcutanious veins);

— combined.

II. Stage:

— 0 — without distension of veins surface;

— 1 — without trophic disorders;

— 2 — with trophic changes;

— 3 — with presence of trophic ulcers.

III. Localization: the femur, the shin, and the foot.

In addition to listed above classifications individual clinicopathologic variants of disease can be distinguished:

1. Varicosis with prevalence of high veno-venous shunt. This form of disease is a result of blood shunt into superficial venous system through sapheno-femoral anastomosis or insolvent perforants, bridging the large hypodermic and femoral veins.

2. Varicosis is characterized by blood shunt from the deep venous system into the superficial one at the level of the shin through insolvent perforants or through the ostium of the small saphenous vein.

3. Atypical forms of varicosis: the posterior external surface of the femur, the area of the perineum and external reproductive organs. From practical point of view it is expedient to distinguish these forms of varicosis, as their diagnosis and treatment have certain specificities and distinctive features.

Clinical course. Clinical manifestations of varicosis change during its development and depend on the stage of disease.

Some patients have complaints of increased fatigability, gravity in legs at the end of a working day, local pain sensations in any department of the shin even before occurrence of superficial veins distension. More often, these sensations occur in typical zones of the greatest localization of communicant veins, where later on a local distension of subcutanious veins takes place. Quite often external signs of the disease, such as phlebectasias, precede occurrence of unpleasant sensations in this area.

At the compensation stage, some pronounced disorders of venous outflow in the extremity are not revealed. For a long time varicosis can have an asymptomatic course, that serves one of the reasons of late patients' reference for a medical help.

The complaints of temporary pains in the shin, transient edemas arising at long standing and disappearing in a horizontal position are typical for the subcompensation stage.

At the decompensation stage the patients complain of constant sensations of gravity, swelling of legs, fatigability, dull aches. In a horizontal position, especially during sleep, quite often there are cramps in the gastrocnemius muscles accompanying by short-term acute pains. The occurrence of trophic disorders is frequently preceded with excruciating dermal itching appearing in the evening.

The beginning of the disease is manifested as mild or moderately pronounced varicosis, which arises more often in the upper third of the shin, but can primary occur in any departments of one or simultaneously of both lower extremities. The extended veins are soft on touch, easily fallen down, the dermal integuments above them are not changed. Approximately 80% of the patients experience phlebectasias in the pool of the great saphenous vein, and frequently these changes begin in the area of any communicant vein. Isolated phlebectasias in the pool of the small saphenous vein occur in 5–7% of observations. Simultaneous changes in systems of the large and the small saphenous veins are observed in 20% of patients with varicosis.

In the subcompensation stage, small edemas or pasty skin are observed in the area of the talocrural joints of the injured extremities. These symptoms, appearing in the evening, disappear within the night. The extent of edema severity depends on the size of static load on the extremity. The stage of decompensation is characterized by the pronounced phlebectasia and global disorder of venous hemodynamics in the extremity, when plenty of blood deposites in it, which can result even in episodes of arterial pressure fall and syncopal condition. Varicosely extended veins are strain, have tight-elastic consistency. The walls of the large veins are quite often sclerosed, closely adhesed with the skin. At emptying of such nodes the excavations form on the skin surface, that testifies to old periphlebitis.

Pigmentation of dermal integuments occurs and progresses by the course of extended veins caused by repeated small hemorrhages into the skin and formation of hemosiderin in these places. In zones of skin pigmentation aseptic cellulitis develops with subsequent sclerosis and inspissation of hypodermic fatty tissue. In these departments, mainly on the internal surface of the lower third of shin, the skin can not be taken in fold, it becomes motionless, dry and rough. Besides, skin pigmentation, pronounced cyanosis or separate blue-crimson stains arise.

Functional neurologic disorders are shown as dyshydroses. Anhydrosis occurs more often than hyperhydrosis. Constant congestion in distal departments of the extremity, sharp rising of pressure in the venous system, sclerotic changes of the hypodermic fat, disorders of capillary circulation cause occurrence of trophic disorder. Especially frequently trophic changes in dermal integuments develop on the anteromedial surface of the lower third of the shin, which is in especially adverse conditions of blood- and lymphflow. The centers of atrophy of the skin, hemosiderosis and dermatosclerosis tend to diffusion and sometimes circularly cover distal third of the shin. Later on dry or moist eczema develops, trophic ulcers of the shin usually form on its background.

Diagnosis. The disorders of venous hemodynamics can rather reliably be diagnosed by modern (clinical, roentgenological, physiological, radio-

nuclidic, ultrasonographic) methods of investigation allowing not only to recognize but also to estimate their severity.

Examination of the patient includes tests for receiving necessary data about the lower extremities venous system function.

Brodie — Troyanov — Trendelenburg's test. It is necessary to lift the examined extremity approximately on 45–60° in a laying position for emptying of the varicosely extended superficial veins. After that the area of connection of the great saphenous vein with the femoral vein is pressed with fingers, or a soft rubber garret is put on the upper third of the femur under the pressure about 100 mm Hg, then the patient stands up. At first the subcutanious veins remain closed. However, during 30 sec they are gradually filled with blood from periphery. If one removes a prelum from the great saphenous vein on the femur immediately after the patient has raised, the varicosely extended veins of the femur and the shins are filled by return blood flow within several seconds, that is shown as galloping pulse wave. The moment of retrograde filling of the superficial venous system can be defined also by palpation, because a typical tremor is sometimes observed above the node. Such a result of the test is the evidence of failure of the ostium valve and axial valves of the great saphenous vein. At more profound inspection, four variants of Brodie — Troyanov — Trendelenburg's test results estimation are used.

Positive result of the test specifies failure of the great saphenous vein valves at its fast retrograde filling.

Negative result: superficial veins are filled fast (for 5–10 sec) before stopping of prelum of the great saphenous vein in the area of oval fossa, and their filling is not increasing at removing of the prelum. In such patients, the filling of superficial venous system occurs from deep veins due to incompetence of perforant veins valves.

Double positive result: the superficial veins of the examined extremity are quickly filled until squeezing of the great saphenous vein in the area of oval fossa stops and after that the strain of venous nodes walls grows sharply (combined failure of perforant viens valves, valves of the ostium and the trunk of the great saphenous vein).

Zero result: the veins are filled slowly (during 30 sec) and both the squeezing of the great saphenous vein and its stopping does not influence the degree and rate of this filling (valval failure of superficial and perforant veins is absent).

Gackenbruch's symptom. Cough causes diaphragm contraction with some decrease of the lumen of the lower cava vein and sharp rising of intraperitoneal pressure, intracaval venous pressure, that in failure of valves is quickly transferred on the common and external ileal vein into the femo-

ral vein, the basic trunk of the great saphenous vein and varicose node on which the fixed fingers feel an obvious jerk.

Barrow — Scheinis test. The patient lies on his back. After emptying the subcutanious veins by raising of an examined extremity by 40–60° in this position, 3 garrots squeezing only the subcutanious veins are put on: in the upper department of the femur above the oval fossa; above the knee joint; under the knee joint in the upper third of the shin. Then the patient gets a vertical position; fast swelling of varicose nodes at any piece of the extremity between garrots specifies the presence of communicant veins with unsufficient valves in this area. The fast filling of shin nodes can take place only through the communicant veins with unsufficient valves in its lower department. The exact localization of this perforant can be established by moving the lowermost garrot from downwards and repeating the test untill filling of the nodes won't stop more distal from the garrot.

Pratt's test. It also allows establishing presence of the communicant veins valves failure and in a horizontal position the patient lifts the examined extremity, which subcutanious veins are made empty by arm massage from periphery to the center. Rubber bandage is dressed from the basis of fingers up to the inguinal so tightly, in order to completely squeeze only the subcutanious veins. Then above the bandage under the oval fossa, a thin rubber garrot is placed to squeeze subcutanious vein and prevent reverse blood flow along it. The patient is placed in a vertical position, and the investigator begins to undress the bandage slowly by one turn from above, pulling this bandage so that a lower laying coil will continue to squeeze the veins laying under it. As soon as a palm-size interval opens between the garrot and bandage, the second rubber bandage is hardly dressed under the garrot, which gradually twists around the extremity downwards, as the first elastic bandage is taken out turn by turn. In this case between bandages the interval of 5–6 cm should be left. As soon as filled and intense varicose node or the same area of a varicose vein opens after putting off the turn of the first bandage, it is marked on the skin at once (by 2% of brilliant green or iodine tinctura) as the place where the communicant connection vein with insufficient valves flows into. In such a manner all the extremity is observed.

Delbet — Perthes's test serves for definition of permeability of deep veins and is carried out as follows. A patient being in a vertical position at with maximally filled superficial veins a rubber garrot or cuff of a sphygmomanometer is dressed on the upper third of the femur, the pressure of which is up to 50–60 mm Hg. After that the patient is offered to move by a fast step during 5–10 min. With good permeability of deep veins full value of their valval system and good condition of perforant veins valves, emptying of the superficial veins comes within one minute. If the filled superficial

veins after 5–10 min of intensive walking do not decrease, and on the contrary, even more pronounced strain of nodes and arching pain appear, it testifies to obstruction of deep veins.

However, it is necessary to emphasize that the listed above functional tests have relative informativity.

Therefore, despite of growing interest to noninvasive methods of research in varicosis, phlebography continues to be the leading way of diagnosis, determining indications for surgery, conducting results and complications of the operative treatment of varicosis. The developed techniques of functional phlebography have opened new opportunities, and modern contrast drugs have practically removed danger of complications.

Retrograde phlebography of the femur and shins in a vertical or inclined position of the patient allows revealing the degree of superficial and deep veins pathological dilatation. The presence or absence of pathological bloodflow through the deep veins, location and condition of the valval system are also established.

The following indications for phlebography in diseases of the lower extremities veins are determined now:

1. Specification of localization and condition of valves in deep and superficial veins.
2. Definition of pathological changes in deep veins and extent of their permeability.
3. Revealing reasons for postoperative relapses, as well as signs of the disease without presence of varicosis of the superficial veins.
4. Differential diagnosis with postthrombotic disease and lymphostasis.
5. Revealing of venous angiodysplasia and atypical forms of varicosis of subcutanious veins with hemangiomas.

Phlebomanometry also strongly has been introduced in daily practice and is considered to be a significant diagnostic method in phlebopathology of the lower extremities. The venous pressure is the most accurate parameter, making objective at certain degree work of the muscular-venous pump. In a calm vertical position in any form of chronic venous failure, the venous pressure in the lower extremities cannot be higher than normal hydrostatic one.

Nowadays for studying hemodynamics in varicosis new noninvasive methods are used (colour and common dopplerography, occlusion plethysmography, thermography, polarography and others), which allow to receive complete information about functional condition of varicose veins, valves of deep and perforant veins, to estimate condition of bloodflow in the large and small saphenous veins.

Venous occlusive plethysmography gets wide application due to large informativity of received parameters (bloodfilling of veins and volumetric rate of bloodflow) based on change registration of the extremity volume.

Recently new prospects of diagnosis of venous hemodynamics disorders have been opened due to occurrence of noninvasive methods, which give an opportunity to quantitatively estimate central hemodynamics and peripheric circulation, among them integrated rheography of the body after M. I. Tishchenko (1971). Quite informative is the way of active capacity definition of valval apparatus of the lower extremities deep veins based on changes of stroke volume (SV) during the performance of Valsalva test. Application of this noninvasive method allows estimating condition of bloodflow in veins of the lower extremities without resorting in some cases to phlebography, which is necessary only for definition of falured valves. SV augmentation with rising of intra-abdominal pressure is possible only in functional sufficiency of valval system of the femoral veins. Otherwise pronounced retrograde flow of blood into the venous system of the lower extremities arises, that sharply reduces venous return to the heart and, hence, results in decrease of SV.

Full-value valves of the great saphenous vein have less importance, as capacity of its pool on initial stages of disease development in moderate distension of superficial veins makes only about 10% of venous system capacity of the extremity and in the isolated defeat of the valval system in this vein the volume of retrograde flow is rather insignificant and is not reflected essentially on venous return.

Differential diagnosis. The signs of varicosis are usually easily distinguished: however, its external signs are met in various diseases. The earliest stages of varicosis development are difficult for diagnosis, because its basic sign is absent — distension of superficial veins. The careful analysis of the disease complaints and anamnesis (feeling of gravity and moderate pains in the lower extremities, edemas which subside after the period of night rest), data of objective and phlebographic examination allows to establish the correct diagnosis.

The distension of superficial veins is met at venous dysplasias, multiple inherent arteriovenous fistulas, as versions of anomaly of arteriovenous anastomoses development. Angiodysplasias are usually revealed in early children's age. The presence of arteriovenous shunts results in accelerated growth of the extremity. Local dermal temperature happens to be increased. Vascular stains are often observed on the skin of the extremity and trunk from pink-red to crimson-blue colour — they are so-called capillary hemangiomas. The distension of the subcutanious veins in angiodysplasia occurs owing to arterial blood shunting in veins.

For realization of differential diagnosis, usually special methods of exam are not required. Posttrombotic disease is hardly recognized because it develops against a background of varicosis and has bright signs of venous outflow disorder. Diagnosis becomes easier by anamnestic data on the old deep vein thrombosis. Wide-spread edemas of the lower extremities, induration of hypodermic fatty tissue, which grasps considerable area on the internal and external surfaces of the lower half of the shin are typical signs in posttrombotic disease. Unilateral thromboses of iliofemoral localization are recognized under the presence of delated veins in the lower departments of the abdominal wall, edemas of all extremity. The final diagnosis is established with the help of phlebography.

The syndrome of the inferior vena cava caused by its occlusion is revealed by moderate swelling of distal departments of the lower extremities, distension of superficial veins, trophic changes of tissues, like in varicosis at the decompensation stage. The typical sign of syndrome of the inferior vena cava is the distension of subcutanious veins of the anterior abdominal wall. Phlebography of the pelvis helps in diagnosis.

The necessity for differential diagnosis of varicosis can arise in femoral hernias, lymphadenitis, abscesses, lymphedema, obliterating atherosclerotic defeats of arteries, diabetic and hypertensive ulcers.

It is necessary to remember also that the pains in the lower extremities in combination with edemas can be in diseases of the joints, platypodia, parasynovites, osteochondrosis of the backbone and other diseases of the locomotor system and that is why a careful study of reasons for their development and forms of manifestation with taking into account accompanying diseases is required.

Treatment. The conservative therapy of varicosis should not be opposed to operative methods of treatment. Its centuries-old experience allows to determine the most rational principles of such treatment.

The elastic compression of the extremity with application of medical stocking or elastic rollers usually brings relief to the patients. It provides compression of varicosely extended veins, increases efficiency of pump function of the calf muscles, slows down process of varicosis of superficial veins, the developments of trophic changes of the shin soft tissues and improves outflow of the blood and lymph.

Elastic bandages may be recommended in cases when the operative treatment is contraindicated because of general condition of the patient or for other reasons.

To dress the bandage or to put on stockings is necessary in the morning before getting up from bed, when there is no swelling. Bandaging begins from the base of foot fingers. One turn of bandage is placed on the previous

one in order to let it cover no less than half of it. The bandage should completely close all the foot and the crus up to the knee joint.

Sometimes elastic bandage is combined with a local compression of trophic ulcer. For this purpose, the gauze bandage is dressed on the area of the ulcer, on the top of which a rubber sponge is located and press to the area of the ulcer in a subsequent applying of elastic bandage. The combination of elastic bandaging of the foot and shins with amplified local squeesing of the trophic ulcer area allows to speed up its healing.

In treatment of trophic ulcers developed owing to varicosis a zink-gelatinous bandage is the most effective method of treatment. Such bandage in single or repeated application causes healing in not less than 90% of trophic ulcers.

The maximal stay of the patient in bed with the raised extremity renders beneficial effect on trophic ulcer healing. In a raised position of the extremity in bed pathological retrograde bloodflow is absent, the venous hypertension is eliminated and edemas disappear.

The method of sclerosing therapy is applied in varicosis of superficial veins more than 100 years. 15–20% solutions of the cooking salt, 50–60% solutions of a glucose, 25–40% solutions of salicylic sodium were used as sclerosing agents; varicocid and 1–3% solution of thrombovar have recently received wide spread. Majority of modern surgeons and phlebologists negatively think about sclerosing therapy as an independent method of treatment, owing to the large number of early relapses of varicosis and danger of thrombus spread to communicant and deep veins of the extremity. The injection of sclerotic substances is contraindicated in case of varicose nodes location close to the joints. The application of this method is allowed as additional to surgical intervention for obliteration of small varicose nodes outside the zone of operation.

The basic pathogenic principles of surgical treatment of varicosis were determined in the beginning of the previous century. The operation on removal of superficial pathologically extended veins and ligating of falured perforant veins is available for each surgeon. It is made in all hospitals. However, neither of surgical pathologies has so much technical and tactical mistakes as operative treatment of varicosis has.

Varicosis is pathology of venous circulation of the extremity, which to some extent can be eliminated only by surgical method. The indications for operative interventions concerning varicosis should be determined before development of serious complications (trophic ulcers, thrombophlebitis and others). However, not earlier than 3 years after occurrence of the first signs of disease. This tendency is based on inevitable progression of venous hypertension and impossibility of early diagnosis of character and degree of

venous bloodflow disorder. The purpose of operation in these cases is normalization of bloodflow through the deep veins.

Besides, the operative intervention intended to the following:

1. Elimination of veno-venous shunts at all levels of the extremity venous system.

2. Removing of varicosely extended veins on the femur, the shin and the foot.

3. Correction of valval failure of deep veins.

4. Elimination of pathological bloodflow from deep tibial veins to veins of the foot.

5. Restoration or improvement of the muscular pump function and other mechanisms of venous return.

Keeping to principles of individual approach of not only the extent but also the methods of venous hemodynamics disorders correction is the basic condition of operative treatment success.

For correction of venous bloodflow it is necessary to break pathological connections between superficial and deep veins on the shin. Intra-operation local phlebograms in this situation help to choose the cuts and extent of operative intervention. Especially large number of insolvent perforant veins is revealed in the lower half of the shin. Usually these ectasias of veins are caused by pathological bloodflow from perforant veins of the crus and veins of the foot. After removing of the large and the small saphenous veins there comes difficulty of blood outflow from the feet and distension of its superficial veins.

Thus, the revealing and break of pathological connections of deep and superficial tibial veins should be combined with intervention on the superficial and perforant veins of the feet.

In pronounced failure of communicant veins and trophic changes of the shin skin the posterior tibial veins obturation is applied for elimination of pathological bloodflow in the area of trophic disorder. Simultaneously with the obturation, distant occlusion of perforant veins opening takes place in the area of trophic changes of soft tissues of the shin internal surface. This kind of the operative intervention is applied, as a rule, in combination with other interventions on the superficial and deep veins of the lower extremities.

Literature data analysis and clinical experience allow to define concretely the indications to operative treatment of varicosis:

1. Pathological distension of the subcutanious veins.

2. Varicosis of the subcutanious veins combined with increased fatigability of extremities, waxy pallor or swelling of its distal departments.

3. Signs of blood outflow disorder: sense of gravity, swelling, increased fatigability of the lower extremity without presence of varicosis of the superficial veins.

4. Signs of increasing trophic changes of soft tissues on the internal surface of the crus which are not treated conservatively irrespective of presence or absence of varicosis of subcutanious veins.

5. Acute thrombophlebitis of the varicosed veins irrespectively of their localization.

Alongside with the indications to the operation concerning varicosis, it is necessary to estimate contraindications to surgical treatment, which can be general and local. General contraindications are following: condition after myocardial infarction or insult, late stages of hypertension, ischemic heart disease etc. These contraindications can be relative, and therefore need individual estimation. The elderly age of the patients is a relative contraindication, but it should be taken into account while defining volume and character of operation. Local contraindications are the putrefied-necrotic trophic ulcers, widespread eczema, pyodermia, erysipilatous inflammation etc.

Operations on Superficial and Perforant Veins

In the majority of the patients operation concerning varicosis of the superficial veins include various variants of phlebectomy after the technique of Troyanov — Trendelenburg, Babcock — Narath etc. with ligation and closure of veins after different modes.

As a rule, the operative intervention begins with inspection of saphenotibial anastomosis, where pronounced conglomerates of the varicosely extended veins occur quite often. If the extravasal correction of a relative incompetence of the deep veins valves is not planned, a small incision is made in parallel and a little bit above the inguinal fold of the skin. Then sapheno-tibial anastomosis is examined, the ligating and erasion of the great saphenous vein in the place of its connection to the femoral vein and all venous branches running into it at this level are made. Then from a separate oblique-longitudinal incision on the femur the conglomerates of the extended veins are excised, and the fragments or trunk of the great saphenous vein after Babcock's method are left with probe. The varicosely dilated branches of main veins on the shin and foot are removed in combination with ligating of falured communicant veins.

Extravasal Correction of Deep Veins Valves

Operative interventions on deep veins are proved pathogenetically, as the restoration of functions of the valval system and normalization of blood outflow are reached. For correction of failured venous valves, many methods are offered, but the most popular now are extravasal cor-

rectors of venous valves developed and offered by A. M. Vedensky. They are lavsanic spiral of various diameter, ensuring basis, preventing dilatation of the vein or its extravasal sqeezing. The indications to correction of failured valves of the femur and the shin are defined depending on the degree of pathological reflux of the contrast agent according to the data of separate retrograde phlebography and do not depend on the stage of disease.

Moreover, in a series of cases the extravasal correction is carried out for preventive reasons on the ostial valve of the great saphenous vein in initial signs of superficial veins ectasia. The indication for correction of popliteal and tibial veins valves is presence of pathological reflux of the contrast agent up to middle of the shin, determined in retrograde phlebography of the shin. The degree of efficiency of valves correction of the popliteal or shin veins is defined in the same way as in the femoral vein with the help of control retrograde phlebography.

The extravasal correction of the valve of the femoral, popliteal or shin veins usually is carried out in combination with other kinds of operations on superficial and perforant veins.

Therapeutic-preventive measures in patients with varicosity in the postoperative period may be divided into general and individual. General measures consist in early movement activity of patients and include the influence of physical activity at blood outflow and activation of blood circulation. Patients are recommended to turn in bed, bend their legs, move the ankles within the first postoperative hours. Simple elevation of the lower part of the bed by 8–10 cm considerably improves venous hemodynamics. The elevated position of the extremity combined with elastic dressing allows to improve activity of the "muscular pump" in active movements, contributes to decrease of venous hypertension and facilitates lymph outflow. The following day patients begin to sit up in the bed. A dosed walking is allowed, the first dressing is made with application of the elastic bandage to both extremities beginning from the tips of the toes up to the knee joints. After bandaging patients begin to walk around the ward but elastic bandage on the leg is obligatory. Wearing elastic bandage is necessary for at least two months after discharge from the in-patient department. Active motion regimen in the early postoperative period and dosed walking from the first postoperative day form optimal conditions for prevention of slowing down of the venous outflow of blood and postoperative thromboses. Different disaggregants (aspirin, trental, curantil) in usual doses are used to prevent thrombus formation in the postoperative period.

THROMBOSES AND THROMBOPHLEBITES

Venous thrombosis (phlebothrombosis) is acute disease based on formation of thrombus in the vein lumen and disorder of venous blood outflow.

Thrombophlebitis is a complete or parietal obturation of the vein by thrombus that was formed against a background of inflammation of the venous wall.

Separation of thrombophlebitis and phlebothrombosis into different forms is artificial as pathological changes that are formed are the link of the same process established by R. Virchow in 1856 (Virchow's triad): damage of the vascular wall, change in blood composition (hypercoagulation) and slowing down of the blood circulation.

The causes of venous thromboses and thrombophlebitis are the following: infection, trauma, surgical intervention, delivery, varicously changed veins, allergic diseases, etc.

Special risk factors are oncologic and endocrine diseases, advanced age, operations on organs of the small pelvis, orthopedic-traumatological operations and intravasal aggressions (investigations, surgery).

Most authors consider that deep tibial veins are the beginning of true ascending thrombotic process in any localization of the thrombus in the system of the inferior vena cava. In many cases thrombosis begins in the pelvic veins and later on gets a descending character. A so-called "floating thrombus" is important for understanding the process of thrombus formation and mechanism of thromboembolic complications. It is formed in transition of the thrombotic process from the vein of smaller diameter into the larger one. Such thrombus does not obturate the vein, and does not give a clinical picture of thrombosis of the venous trunk. Its length may be 15–20 cm. The blood circulation in the main vein is preserved but in this situation it is a high possibility of tearing off the thrombus or its fragment and development of embolism of the pulmonary artery.

Classification. According to localization thrombophlebites are divided into thrombophlebites of the superficial and deep veins. Phlebothrombosis occurs more often in deep veins but it is very difficult to verify this process.

1. Superficial veins:

a) tibial segment;

b) acute ascending thrombophlebitis of the middle third of the thigh and opening of the great saphenous vein.

2. Deep veins — the system of the inferior vena cava:

a) thrombophlebitis of the deep tibial veins;

b) popliteal-femoral segment;

c) ileofemoral segment;

d) glomerular segment (thrombophlebitis of the glomerular veins);

e) infrarenal, renal and suprarenal segments of the inferior vena cava;

f) combined variant of affection of the cavaileofemoral segment;

g) total thrombosis of the whole deep venous system of the lower extremity resulting in venous gangrene of the extremity.

According to the clinical course:

1. Acute
2. Subacute
3. Chronic

According to the stage of the thrombotic process:

1. Inflammation (3–10 days)
2. Thrombus formation (10 days — 3–6 months).
3. Recanalization (3–6 months, development of postthrombophlebitic disease).

According to O. O. Shalimov and I. I. Sudarev duration of the acute stage is 7–14 days. Then the process becomes subacute and the chronic process begins in 3 months.

Occlusions of the main veins should be considered according to three segments:

1. Lower segment — thrombosis of the main veins of the lower extremities.
2. Middle segment — thrombosis of the external and general glomerular veins.
3. Upper segment — thrombosis of the inferior vena cava.

It is expedient to make such division as each of the segments has its own anatomical peculiarities, each segment is characterized by definite pathogenetic mechanisms of thrombus development; colateral blood circulation in occlusion of different segments has its own characteristic peculiarities; thrombosis of each segment is manifested by peculiar symptomocomplex; choice of treatment method is also determined by localization.

Acute Thrombophlebitis of the Superficial Veins of the Lower Extremities

Diagnosis of superficial thrombophlebitis is not difficult. This pathology frequently arises against a background of the varicosis of the lower extremities. Patients complain of burning pain in the extremity. Pain syndrome is pronounced on dependence on involvement of the adjacent tissues

into the inflammatory process. On palpation a painful dense strand is determined along the greater or smaller saphenous vein. The skin is hyperemic, with marked hyperesthesia and local increased temperature over a thrombosed vein. The general state changes a little. Patients feel moderate general weakness, chill, the body temperature increases from subfebrile to 38–40°C.

It is no need in additional methods of investigation to make a diagnosis.

The so-called migrating thrombophlebitis should be considered as a special form of superficial thrombophlebitis which arises in the zone of the great saphenous vein. The peculiarities of the clinical course of this disease is that dense formation of round or oval shape with signs of inflammation is formed along this vein (tenderness, hyperemia of the skin, increased local temperature). There are no general clinical symptoms. In progressive course of the disease the formation is often spread along the internal surface of the femur upwards, to the place where the saphenous vein flows in the femoral one. In such a situation there is a real threat of formation of the floating thrombus with its tearing off and embolism of the pulmonary artery. The process of migration of the thrombus formation may also result in ileofemoral thrombosis.

Superficial thrombophlebitis is frequently the first symptom of obliterating thromboangiitis (Burger's disease), the clinical course of which is characterized by skin hyperemia along the fine veins and segmental indurations. The latter are often localized on the foot and ankles but they may arise in the areas of fine veins except the main trunks of the greater and smaller saphenous veins. Painful edema is formed around the indurations. Clinical symptoms disappear in 2–3 weeks, but the induration may be kept in the vein projection much longer.

Treatment of superficial thrombophlebitis is started with conservative therapy. There are used nonsteroid antiinflammatory drugs (pyrabutol, reopyrin, indomethacin, voltaren, ibubrufen), anticoagulants (fraxiparin, heparin, sincumar, phenilin, aspirin), desensitizing drugs (suprastin, tavegil, pipolfen, diasolin), vein protectors (troxevasin, venorutol, exusan, anavenol), local treatment (compresses with dimexide, Vishnevsky ointment, troxevasin, heparin, butadion).

Bed regimen is obligatory in the acute stage of the process.

Surgical treatment is used for prevention of spreading of the process and thromboembolic complication in acute ascending thrombophlebitis.

The operations of Troyanov-Trendelenburg are performed, ligature of the great saphenous vein at the place where it flows in the femoral one, excision of conglomerations of the thrombosed veins until blood circulation in areas of the perforated veins is restored.

In pyo-septic complications of acute thrombophlebitis of superficial veins opening and sanation of abscess is made.

Acute Thrombophlebitis of Deep Veins of the Lower Extremities

The most frequent localization of the thromboses of deep veins of the lower extremities is thromboses of deep tibial veins. Clinical symptoms are general for all levels of affection. They include the following clinical manifestations: sudden acute pain in the leg, especially in the shin, bursting sensation and feeling of gravity in the whole leg, edema of the extremity with its maximum in 3–5 days. Sometimes external outline of the thigh or shin may be by 5–15 cm larger in comparison with a normal one. The skin is pale, glistening, tense on palpation. The skin temperature of the foot and toes is 1.0–1.5°C lower as compared with the healthy extremity.

Pulsation of the foot arteries is weakened and palpation along the deep venous trunks is painful due to reactive spasm of the artery and edema of the tissue.

The body temperature may reach 38–39°C.

The disease may have a course without considerable hemodynamic disorders, therefore its manifestations are slightly marked. Symptomatology may be limited by patient's complaints on pain in the tibial muscle that is enhanced on walking, feeling of gravity and periodic spasms in the tibial muscles. Edemas may be slightly pronounced.

Specific signs of acute thrombophlebitis of deep tibial veins are Hohmann's symptom — tenderness in the calf muscle in sharp, severe, passive flexion of the foot; Moses's symptom — pain in clutching of the shin in anterior-posterior direction; Lovenberg's test — the sphigmomanometer's cuff is applied on the middle third of the shin and pressure is increased in it to 80–100 mm Hg. When pain develops in the tibial muscles, the test is considered positive.

In acute thrombophlebitis of the popliteal and fibial veins edema involves the area of the shin, knee joint and distal parts of the thigh. The outlines of the knee joint become smooth, the joint is enlarged, flexion and extension causes sharp increase of pain. It's difference in outlines of the thigh as compared with the healthy one.

Acute thrombophlebitis of the glomerular-femoral segment (ileofemoral thrombosis) arises mostly on the left, because the left glomerular vein is contracted by the right general glomerular artery. The patient's state is severe. The onset of the disease is acute. There is a severe pain in the lower parts of the abdomen, inguinal area, at the side of affection, and the area of the anterior-posterior surface of the thigh. The body temperature is high, there are chill, flaccidity, adynamia. Edema of the extremity progressively

grows, involves the buttock, the anterior abdominal wall, the perineum. The skin is rense, infiltrated, distended subcutaneous veins are seen.

There are two variants of ileofemoral thrombosis: white phlegmasia and blue phlegmasia when thrombosis of the main veins of the lower extremities and pelvis is combined with thrombosis of the colateral ways of blood outflow. There is regional arterial insufficiency in it. Blue phlegmasia may result in venous gangrene of the foot, the shin, the thigh as well as lethal outcome.

Diagnosis. Laboratory and instrumental methods of diagnosis are following:

1. Clinical and biochemical blood analysis.
2. Coagulograms.
3. Sonography (determination of the level of occlusion and spreading of the process).
4. Phlebography as a method of choice.
5. Clinical urine analysis.

Treatment of thrombosis of any localization and severity begins with conservative therapy. Methods of such treatment may be divided into active and passive. The bed regimen should last for days. This time is needed for fixation of thrombus to the vascular wall and decreased risk of embolism. Drug therapy begins with administration of anticoagulants of direct action. They are low molecular heparins: heparin, flaxiparin, clexan, fragmin. Heparin is used intravenously or subcutaneously in the dose of 5–10 thousand U in every 6 hours for 7–21 days. 2–3 days before withdrawal of direct anticoagulants, indirect anticoagulants are administered (pelentan, sincumar, fenilin, etc.). The dose of both direct and indirect anticoagulants should be chosen individually under the control of coagulogram. To improve rheologic properties of blood there may be administered rheopolyglucin, rheogluman, polyglucin. To activate microcirculation there may be used nicotinic acid and trental. It is expedient to administer patients drugs of antiinflammatory effect (butadion, rheopyrin, pyrabutol, vobenzim). This therapy influences on the processes of blood coagulation and does not influence the already formed thrombus.

Fibrinolytic drugs are applied to influence the formed thrombus, the most effective in the first 4–5 days of the disease are fibrinolysin, urokinase, streptokinase.

Fibrinolytic therapy is considered inexpedient after a surgical interventions.

The main methods of treatment of ileofemoral thrombosis and thrombosis of the inferior vena cava is surgery. The main task of the surgical treatment is prevention of embolism, restoration of the venous circulation

and prophylaxis of postthrombophlebitic syndrome of the lower extremity and the pelvis.

The technique of the surgical intervention is as follows. The femoral vein is isolated. Longitudinal venotomy is made over the place of inflowing of the femoral deep vein. Measures are taken aimed at prevention of embolism in the right heart before removal of the thrombus. For this purpose a ballon cathether is inserted in the inferior vena cava through the vein of the healthy extremity, it blocks its passage; or the vena cava is taken on the tourniquet through the retroperitoneal approach. Thrombi are removed by Fogarty's catheter. It is important to achieve satisfactory retrograde circulation.

ABBREVIATIONS

UGDB — ulcerous gastroduodenal bleeding
DIC — disseminated intravascular coagulation
EGDS — esophagogastroduodenoscopy
ECLT — extracorporal lithotripsy
EPST — endoscopic papillosphincterotomy
IL — interleukins
LCE — laparoscopic cholecystectomy
OChG — operational cholangiography
RChPG — retrograde cholangiopancreotography
ESR — erythrocyte sedimentation rate

REFERENCES

1. *Александров В. Б.* Рак прямой кишки / В. Б. Александров. — М. : Медицина, 1987. — 231 с.

2. *Аминев А. М.* Руководство по проктологии / А. М. Аминев. — Т. 1, изд. 2-е. — Куйбышев, 1971.

3. *Баженова А. П.* Рак молочной железы / А. П. Баженова, Л. Д. Островцев, Г. Н. Хаханашвили. — М. : Медицина, 1985. — 269 с.

4. *Белый И. С., Десятерик В. И.* Деструктивный панкреатит / И. С. Белый, В. И. Десятерик. — К. : Здоров'я, 1980. — 237 с.

5. *Березов Ю. Е.* Рак пищевода / Ю. Е. Березов. — М. : Медицина, 1979. — 178 с.

6. *Василюк М. Д.* Варикозная болезнь нижних конечностей у женщин / М. Д. Василюк, М. Г. Шевчук. — К. : Здоров'я, 1992. — 182 с.

7. *Введенский А. Н.* Варикозная болезнь / А. Н. Введенский. — Л. : Медицина, 1983. — 254 с.

8. *Введенский А. Н.* Посттромботическая болезнь / А. Н. Введенский. — Л. : Медицина, 1986. — 239 с.

9. *Гешелин С. А.* Неотложная онкохирургия / С. А. Гешелин. — К.: Здоров'я, 1988. — 267 с.

10. *Гешелин С. А.* TNM-классификация злокачественных опухолей и комплексное лечение онкологических больных / С. А. Гешелин. — К. : Здоров'я, 1996. — 184 с.

11. *Гостищев В. К.* Общая хирургия / В. К. Гостищев. — М.: Медицина, 1996. — 575 с.

12. *Григорьев П. Я.* Диагностика и лечение язвенной болезни желудка и 12-перстной кишки / П. Я. Григорьев. — М. : Медицина, 1986. — 346 с.

13. *Даценко Б. М.* Комплексная диагностика в неотложной абдоминальной хирургии / Б. М. Даценко, Ш. Ф. Ибишев. — К. : Здоров'я, 1991. — 261 с.

14. *Дульцев Ю. В.* Парапроктит / Ю. В. Дульцев, К. Н. Салемов. — М. : Медицина, 1981. — 168 с.

15. *Дедкова Е. М.* Профилактика послеоперационных тромбоэмболий / Е. М. Дедкова, С. И. Лукомский. — М. : Медицина, 1989. — 365 с.

16. *Ковалев М. М.* Комплексное лечение больных перитонитом / М. М. Ковалев, Л. П. Чепкий. — К. : Здоров'я, 1981. — 186 с.

17. *Королев Б. А.* Экстренная хирургия желчных путей / Б. А. Королев, Д. Л. Пиковский. — М. : Медицина, 1990. — 369 с.

18. *Лащевкер В. М.* Острый панкреатит (клиника, диагностика, лечение) / В. М. Лащевкер. — К. : Здоров'я, 1982. — 167 с.

19. *Мариев А. И.* Хирургическое лечение паховых грыж / А. И. Мариев. — Петрозаводск : Медицина, 1989. — 234 с.

20. *Маслов В. И.* Лечение эмпием плевры / В. И. Маслов. — Л. : Медицина, 1976. — 190 с.

21. *Неотложная* хирургия брюшной полости / под ред. В. Т. Зайцева. — К. : Здоров'я, 1989. — 272 с.

22. *Нифантьев О. Е.* Механическая непроходимость / О. Е. Нифантьев — Красноярск : Медицина, 1989. — 367 с.

23. *Онкологія:* підручник для студентів / за ред. Б. Т. Білинського, Ю. М. Стернюка. — Львів : Світ, 1992. — 287 с.

24. *Панцырев Ю. М.* Клиническая хирургия / Ю. М. Панцырев. — М. : Медицина, 1988. — 496 с.

25. *Петров В. М.* Кишечная непроходимость / В. М. Петров, И. Н. Ерюхин. — М. : Медицина, 1989. — 359 с.

26. *Петровский Б. В.* Хирургические болезни / Б. В. Петровский. — М. : Медицина, 1980. — 592 с.

27. *Ривкин В. Л.* Геморрой. Запоры / В. Л. Ривкин, Л. Л. Капуллер.— М. : Мед. практика, 2000. — 158 с.

28. *Родионов В. В.* Калькулезний холецистит (осложненный механической желтухой) / В. В. Родионов, М. И. Филимонов, В. М. Могучев. — М. : Медицина, 1991. — 319 с.

29. *Руководство* по неотложной хирургии органов брюшной полости / под ред. В. С. Савельева. — М. : Медицина, 1986. — 667 с.

30. *Русанов А. А.* Аппендицит / А. А. Русанов. — Л. : Медицина, 1973. — 234 с.

31. *Русанов А. А.* Рак желудка / А. А. Русанов. — Л. : Медицина, 1973. — 198 с.

32. *Рыжих А. Н.* Хирургия прямой кишки / А. Н. Рыжих. — М. : Медицина, 1956. — 569 с.

33. *Сидоренко М. Н.* Мастопатия / М. Н. Сидоренко. — Л. : Медицина, 1991.— 264 с.

34. *Скрыпниченко Д. Ф.* Неотложная хирургия брюшной полости / Д. Ф. Скрыпниченко. — К. : Здоров'я, 1986. — 400 с.

35. *Теория* и практика местного лечения гнойных ран: проблемы лекарственной терапии / под ред. Б. М. Даценко. — К. : Здоров'я, 1995. — 382 с.

36. *Томашук К. П.* Билиарный острый холецистопанкреатит / К. П. Томашук. — К. : Здоров'я, 1992. — 183 с.

37. *Томашук К. П.* Острый аппендицит / К. П. Томашук. — К. : Здоров'я, 1994. — 72 с.

38. *Тоскин К. Д.* Грыжи брюшной стенки / К. Д. Тоскин, В. В. Жебровский. — М. : Медицина, 1990. — 368 с.

39. *Федоров В. Д.* Проктология / В. Д. Федоров, С. В. Дульцев. — М. : Медицина, 1983. — 569 с.

40. *Филин В. Й.* Неотложная панкреатология / В. Й. Филин, А. Л. Костюченко. — СПб. : Питер, 1994. — 416 с.

41. *Факультетська* хірургія / за ред. В. О. Шідловського, М. П. Захараша. — Тернопіль : Укрмедкнига, 2000. — 544 с.

42. *Хирургические* болезни / под ред. М. И. Кузина. — М. : Медицина, 1995. — 687 с.

43. *Хирургия* печени и желчевыводящих путей / А. А. Шалимов, С. А. Шалимов, М. Е. Ничитайло, Б. В. Доманский. — К. : Здоров'я, 1993. — 509 с.

44. *Хирургия* поджелудочной железы / А. А. Шалимов, С. А. Шалимов, М. Е. Ничитайло, А. П. Радзиховский. — Симферополь : Таврида, 1997. — 553 с.

45. *Чухриенко Д. П.* Непроходимость кишечника / Д. П. Чухриенко. — К. : Здоров'я, 1968. — 456 с.

46. *Чухриенко Д. П.* Наружные кишечные свищи / Д. П. Чухриенко, И. С. Белый. — К. : Здоров'я, 1975. — 192 с.

47. *Чухриенко Д. П.* Заболевания прямой кишки / Д. П. Чухриенко, А. В. Люлько, Я. С. Березницкий. — М. : Медицина, 1976. — 158 с.

48. *Шалимов А. А.* Хирургия желудка и двенадцатиперстной кишки / А. А. Шалимов, В. Ф. Саенко. — К. : Здоров'я, 1972. — 498 с.

49. *Шалимов А. А.* Хирургия кишечника / А. А. Шалимов, В. Ф. Саенко. — К. : Здоров'я, 1977. — 564 с.

50. *Шалимов А. А.* Хирургия пищеварительного тракта / А. А. Шалимов, В. Ф. Саенко. — К. : Здоров'я, 1987. — 568 с.

51. *Шалимов А. А.* Острый панкреатит и его осложнения / А. А. Шалимов, А. П. Радзиховский, М. Е. Нечитайло. — К. : Здоров'я, 1990. — 521 с.

52. Шпитальна хірургія / за ред. Л. Я. Ковальчука. — К. : Укрмедкнига, 1999. — 549 с.

53. *Язвенная* болезнь или пептическая язва / за ред. В. Г. Передерия. — К. : Здоров'я, 1987. — 158 с.

CONTENTS

Бібліотека студента-медика

Провідний редактор серії
В. М. Попов
Художнє оформлення серії
О. А. Шамшуріна

Навчальне видання

Б. С. Запорожченко, В. В. Міщенко, А. Л. Дехтяр, О. Б. Зубков, Ю. В. Грубнік, А. В. Снісаренко

ФАКУЛЬТЕТСЬКА ХІРУРГІЯ

Курс лекцій

Навчальний посібник

Англійською мовою

Провідний редактор ***В. М. Попов***
Редактор ***Р. В. Мерешко***
Художній редактор ***О. А. Шамшуріна***
Технічний редактор ***А. В. Попов***
Коректор ***О. В. Титова***
Поліграфічні роботи ***І. К. Каневський***

Підп. до друку 3.09.2009. Формат 60x84/16.
Папір офсетний. Друк різографічний. Ум. друк. арк. 19,21.
Обл.-вид. арк. 28,0. Тираж 50. Зам. 1279.

Видано і надруковано Одеським державним медичним університетом.
65026, Одеса, Валіховський пров., 2.
Свідоцтво ДК № 688 від 13.11.2001