Bogomolets National Medical University
Department of Surgery N2

# Study Guide of Surgery for Medical Students ($6^{th}$ year)

Edited by
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The content of the textbook covers the new surgical curriculum for students of the 6th year and includes issues of history of surgery, organization of surgical care in Ukraine, ethics and deontology in surgery, systemic inflammatory response syndrome in surgical patients, critical conditions in surgical patients, the main pathological syndromes of acute abdominal pathology. Issues of clinical and instrumental diagnosis and treatment of surgical diseases of the chest, abdominal cavities, blood vessels, including traumatic injuries of the organs are covered in detail. (10 topics). The presented material consists of the main part, terminology, clinical signs, diagnostic methods, conservative and surgical treatment tactics, used literature and test tasks, materials for self-study.

For the English language students of 6-th course of medical schools of IV accreditation level.

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**CONTENT**

## PREFACE

In recent years of global challenges for Ukraine and the world (the territorial invasion of Ukraine by russia, the Covid-19 pandemic), great progress has been made in our understanding of a wide range of issues of surgical care, systemic surgical pathology, diseases of the digestive, cardiovascular, respiratory systems and injuries of internal organs. Modern diagnostic methods and minimally invasive techniques have become routine in these branches of surgery and have fundamentally changed diagnostic and treatment tactics. Innovative technical approaches such as robotic surgery are becoming a reality today. Several important consensuses have been reached on complex surgical issues. However, students and young surgeons are faced with a huge amount of information that is difficult to systematize and understand.

The purpose of this study guide is to facilitate the preparation of 6th year students for the cycle of surgery, which currently has 10 topics. More than 10 years have passed since the publication of the first edition of our study guide. Taking into account that during this time the leading specialists of Ukraine in cardiovascular, thoracic and military field surgery were involved in the Department of Surgery №2 of Bogomolets National Medical University, it became necessary to highlight the modern principles and new technical aspects of these areas of surgery. The presented material and test tasks should help students prepare for classes, as well as the final module control. It is obviously impossible to achieve detailed coverage of every aspect of surgery in one textbook. However, our goal was to create a study guide that would be useful for students and could be used as a reference for surgeons in various specialties. Ultimately, surgery remains a discipline in which the surgeon's knowledge and skill are combined for the well-being of our patients.

We hope that this manual will be useful and understandable to English-speaking students who will continue to study, undergo training or practice in surgical clinics around the world.

**Head of the Department of Surgery №2 of the Bogomolets National Medical University, academician of the Ukrainian Academy of Sciences, laureate of the National Academy of Sciences of Ukraine award, Doctor of Medical Sciences, professor** **Ihor Kolosovych**

## THEMATIC PLAN OF PRACTICAL STUDIES

| № | THEME | Amount of hours |
|---|---|---|
| **Modul 6. Content module 5: General issues of surgery and clinical manifestations of surgical diseases** | | |
| 1 | General issues of surgical care в in Ukraine. New technologies in surgery. Abdominal pain syndrome. | 5 |
| 2 | Systemic inflammatory response syndrome in surgical patients. Critical conditions in surgical patients. | 5 |
| 3 | Dysphagia syndrome, vomiting and defecation disorders. Intestinal obstruction syndrome. | 5 |
| 4 | Acute abdominal syndrome. Syndrome of local and widespread purulent and inflammatory peritoneal process. | 5 |
| 5 | Mechanical jaundice syndrome in tumor and non-tumor lesions of the biliary tract. | 5 |
| 6 | Bleeding syndrome from the digestive tract. Surgical aspects of gastric ulcer and duodenal ulcer. | 5 |
| 7 | Closed and open abdominal trauma. Wounds and closed injuries of the pelvis and pelvic organs. | 5 |
| 8 | Syndromes of chest pain, respiratory and heart failure in the diagnosis and differential diagnosis of diseases of the chest cavity. Modern methods of diagnosis and surgical treatment of cardiovascular diseases. Surgical pathology of the lungs and pleura. Damage to the chest cavity organs. | 5 |
| 9 | Limb ischemia syndrome. Thrombosis and embolism of the vessels. Thromboembolism of the pulmonary artery. | 5 |
| 10 | Iatrogeny in surgery. | 5 |
| Total | | 50 |

## OBLIGATORY MINIMUM LIST OF PRACTICAL SKILLS

1. Perform artificial respiration, indirect cardiac massage*.
2. Fixing the tongue*.
3. Stop external bleeding*.
4. Carry out primary wound care*.
5. Rinse out the stomach, intestines*.
6. Administer injections of medicinal substances (i.m., i.v., p.c.)
7. Measure blood pressure
8. Perform bladder catheterisation with a soft catheter*.
9. Perform a finger examination of the rectum*.
10. Performing a pleural puncture*.
11. Determine blood groups, Rh blood type
12. Know the name and purpose of the surgical instruments of the general surgical kit (scalpels, tourniquets, abdominal mirrors, cornets, tweezers, etc.)
13. Know the name, purpose and demonstrate the use of instruments for proctological examination

## TOPIC 1. GENERAL ISSUES OF SURGICAL CARE B IN UKRAINE. NEW TECHNOLOGIES IN SURGERY. ABDOMINAL PAIN SYNDROME

### 1.1. General issues of surgical care в in Ukraine. History of surgery in Ukraine. Organization of surgical care in Ukraine. Ethics and deontology in surgery.

**Relevance of the topic.**

The history of surgery is an important section of the history of medicine, which demonstrates fundamental discoveries and events that significantly influenced the development of both surgery itself and all medicine as a whole. The development of surgery is considered through the prism of the progress of science, the activities of individual prominent personalities, and historical circumstances. Knowledge of this history allows students to understand the mechanisms of the development of the discipline that determine the progress of surgical science and practice, the emergence of new technologies for treating patients and ways to improve the results of patient treatment. The emergence of surgery dates back to the very origins of human society. Having begun to hunt, fight, and work, man was faced with the need to heal wounds, remove foreign bodies, stop bleeding, and perform other surgical manipulations. Surgery is the oldest medical specialty, which is unthinkable without the use of the latest achievements of human thought, the progress of science and technology.

Organ and tissue transplantation is truly the greatest achievement of the 20th century. People have dreamed of replacing a lost or diseased part of the body with an organ taken Organ transplantation began in the 20th century with the development of the vascular suture technique and the first experimental kidney transplants [Ullmann E., 1902] and heart transplants [Carrel A., 1905]. During the period from 1902 to 1912, A. Carrel, D. Guthrie and their colleagues performed a series of organ transplants on animals, including kidney, heart, spleen, ovaries, limbs and even the head. A. Carrel wondered why an organ taken from another animal is rejected, while an organ taken from an animal and replanted into the same animal takes root and functions normally despite the use of the same surgical technique. He suggested that this phenomenon is explained by the biological properties of the recipient. The study of the biological phenomenon of rejection of a transplanted organ, predicted by A. Carrel, took several decades and continues to this day. The most significant successes have been achieved in the last 10-15 years. By the end of the 1990s, more than 230 organ transplantation centers had been organized in the world, the conditions for organ removal from donors, and the relationships between the donor, recipient, relatives, and medical personnel were legally regulated.

The shortage of donor organs and the difficulties associated with financing limit the performance of these expensive surgical interventions. The number of patients waiting for transplantation is 3 times greater than the number of patients who have received an organ from a donor. Tissue transplantation does not require a donor with a beating heart, so the transplantation of heart valves, corneas, skin, bone and connective tissue products is not limited.

**Educational aims:**

- Define the concept of "Discipline of Surgery".

- To study the main historical milestones in the development of surgery in Ukraine.
- To learn the principles of organizing surgical care in Ukraine.
- To form deontological ideas when working with patients, to master the ability to establish psychological contact with patients and their relatives, to develop a sense of responsibility for the timeliness and correctness of professional actions.

**A student must know:**

- One of the founders of the first surgical schools in Ukraine.
- Definition of the concept of "medical ethics".
- Definition of the concept of "deontology".
- What are the features of surgical deontology?
- The scope of surgical care at the outpatient stage
- The scope of surgical care at the inpatient stage

**The student must be able to:**

- Apply the principle of collegiality in relationships with colleagues
- Guided by moral and ethical principles, inform the patient about his condition, treatment plan, and possible complications.
- Fill out the inpatient card.
- Collect and evaluate complaints of patients requiring organ transplantation, medical history, and conduct a physical examination;
- Determine the rational volume of laboratory and instrumental research methods;
- Be able to interpret the collected diagnostic information, analyze it correctly, and establish a diagnosis based on an integrated assessment of the collected data;
- Determine treatment tactics depending on the stage of diseases and the presence of complications in patients requiring organ transplantation

**Terminology.**

| Term | Definition |
|---|---|
| Ethics | a science whose subject is determining the ethical value of human aspirations and actions. |
| Deontology | the doctrine of the duty of a medical professional (from the Greek "deon" - duty, "logos" - study, science), the principles of medical behavior, which should be aimed at creating the best conditions for the patient's speedy recovery. |

**Content:**

**History of surgery in Ukraine**

The history of surgery consists of five main periods:

- the period of ancient times from the 6th-7th millennium BC to the Renaissance, that is, until the 16th century AD;

- anatomical-morphological period - 16th - 19th centuries;
- the period of great discoveries - the 19th and early 20th centuries;
- physiological period - surgery of the 20th century;
- modern surgery of the late 20th and early 21st centuries.

The study of ancient manuscripts, mummies, and excavation materials has allowed us to form an idea of surgery, starting from the 6th-7th millennium BC. Ancient people knew how to stop bleeding by applying vascular compression, tight bandages, pouring hot oil over wounds, and sprinkling ashes. There is information about the performance of quite complex operations at that time: trepanation of the skull, amputation of limbs, removal of stones from the bladder, removal and prosthetics of teeth.

The most famous surgical school of the ancient Indians. The manuscripts of that time describe the clinical picture of many diseases (smallpox, tuberculosis, erysipelas, anthrax). Ancient Indian doctors used more than 120 surgical instruments, which allowed them to perform complex interventions, including cesarean sections. Plastic surgery became popular in ancient India. The method of rhinoplasty (Indian plastic surgery) of those times entered the annals of surgery and is still used today. The achievements of the ancient Egyptians in anatomy and surgery are also widely known.

One of the most famous doctors of the ancient world was Hippocrates (460-377 BC). He was an outstanding man of his time, from whom all modern medicine originates. That is why the Hippocratic oath is taken by young doctors who are ready to devote their whole lives to the medical profession. Hippocrates distinguished between wounds that heal without suppuration and wounds complicated by a purulent process. He considered air to be the cause of infection. When dressing, he recommended cleanliness, used boiled rainwater and wine. When treating fractures, Hippocrates used peculiar splints, stretching, and gymnastics. Hippocrates' method for correcting a shoulder dislocation is still known.

In ancient Rome, the most famous followers of Hippocrates were Cornelius Celsus (30 BC, 38 AD) and Claudius Galen (130-210). Celsus created a thorough treatise on surgery, which described many operations (removal of bladder stones, trepanation of the skull, amputation), treatment of dislocations and fractures, and methods of stopping bleeding. He was the first to suggest ligating a bleeding vessel. In addition, Celsus was the first to describe the classic signs of inflammation: calor (heat), dolor (pain), tumor (swelling, swelling), rubor (redness). Galen collected extensive material on anatomy and physiology, introduced the experimental method of research. He proposed surgery for a defect in the development of the upper jaw ("cleft lip"), and used the method of twisting a bleeding vessel to stop bleeding.

The most famous representative of ancient Eastern medicine was Ibn Sina, better known in Europe under the name Avicenna (980-1037). Ibn Sina was an encyclopedic scientist, knowledgeable in philosophy, natural science and medicine, the author of about a hundred scientific works. Ibn Sina wrote the "Canon of the Medical Art" in five volumes, where he outlined the issues of theoretical and practical medicine.

In the Middle Ages, the development of surgery, especially in Europe, slowed down. The rule of the church made scientific research impossible, operations associated with "bloodshed" and autopsy were prohibited. Medical faculties were

opened in many universities in Europe, but official medical science did not include surgery. Surgeons were formed in the circle of barbers, craftsmen, and artisans.

The achievements of some surgeons of the Middle Ages were quite significant. In the 13th century, the Italian surgeon Lucca used special sponges impregnated with substances for anesthesia, the inhalation of which vapors led to loss of consciousness and pain sensitivity. In the same 13th century, Bruno de Langoburgo discovered the fundamental difference between primary and secondary wound healing, introduced the terms "healing by primary tension" and "healing by secondary tension". The French surgeon Mondeville proposed applying early stitches to the wound. The stagnation of the Middle Ages was replaced by the flowering of the Renaissance - a time of rapid development of art, science and technology. In medicine, a struggle began against religious canons, the authorities of ancient scientists. The empirical approach to surgery ended, and the anatomical-morphological era of surgery began.

Although knowledge of anatomy is a basic requirement of surgery, its study was forbidden by the church until the mid-1500s. Only after the permission of Pope Sixtus IV (1414-1484) and Clement VII (1478-1534) did anatomical studies of cadavers become possible. The first prominent anatomist-researcher of the structure of the human body was Andreas Vesalius (1514-1564). Many years of research on human corpses, reflected in his work "De Humani Corporis Fabrica Libri Septem" (1543), allowed him to refute many dogmas of medieval medicine and to lay the foundation for a new stage in the development of surgery. A great contribution to the development of surgery of that time was made by the Swiss physician and naturalist Paracelsus (Theophrastus Bombast von Hohenheim, 1493 - 1541) and the French surgeon Ambroise Paré (1517-1590). Paracelsus, participating in many wars, significantly improved the methods of treating wounds, using astringents and various chemicals for this. A. Pare developed a technique for amputations and introduced a new obstetric manipulation - turning the fetus onto its feet. He proved that gunshot wounds are not poisonous, but are a type of bruised wounds.

The most important event in Renaissance medicine was the discovery in 1628 by William Harvey (1578-1657) of the laws of blood circulation. Based on the research of A. Vesalius and his followers, W. Harvey established that the heart is a kind of pump, and the arteries and veins are a single system of vessels. In his classic work "Exertitatio anatomica de motu cordis et sanguinis in animalibus" he first distinguished the large and small circles of blood circulation. The successes of physiology, chemistry and biology were of great importance for the development of surgery. First of all, it is necessary to note the invention by A. Leeuwenhoek (1632-1723) of a magnifying device, the prototype of the modern microscope, the description by M. Malpighi (1628-1694) of capillary blood circulation and his discovery in 1663 of "blood corpuscles". An important event of the 17th century was the first human blood transfusion, performed by Jean Denis in 1667.

One of the reasons for the development of surgery was the huge number of wars in Europe at that time. For example, the French surgeon, Napoleon's personal physician D. Larre, personally performed two hundred limb amputations in one day after the Battle of Borodino. Nikolai Ivanovich Pirogov (1810-1881) performed such operations as amputation of the breast or dissection of the bladder in 2 minutes (!). And

osteoplastic amputation of the foot - in 8 minutes (!). Such speed was forced and was due to the impossibility of full anesthesia during surgery.

During the 18th and first half of the 19th centuries, the use of knowledge of anatomy in surgery allowed to improve the results of surgical interventions. Each country had its famous surgeons. In the Netherlands there were Govard Bidloo (1649-1713), Bernhard Siegfried Albinus (1697-1770), Pieter Camper (1722-1789); Albrecht von Haller (1708-1777), August Richter (1742-1812) and Johann Friedrich Meckel (1781-1833). In Germany, Antonio Scarpa (1752-1832) worked, in Italy and France, Pierre-Joseph Desault (1744-1795), Jules Cloquet (1790-1883), and Alfred Armand Louis Marie Velpeau (1795-1867), in Great Britain - William Cowper (1666-1709), William Hunter (1718-1783), brothers John Bell (1763-1820) and Charles Bell. (1774-1842).

Three main problems became obstacles to the further development of surgery: 1. Ignorance of methods of combating infection. 2. Lack of methods of adequate anesthesia. 3. Impossibility of complete stopping of bleeding and correction of blood loss. All these problems were fundamentally solved in the late 19th and early 20th centuries. In particular, the development of surgery during the period of great discoveries of the late 19th and early 20th centuries is associated with three fundamental discoveries:

- Introduction of asepsis and antiseptics into surgical practice.
- The development of the science of anesthesia.
- Discovery of blood groups and the possibility of blood transfusion.

The powerlessness of surgeons in the face of infectious complications in the 19th century was terrible. Thus, in M.I. Pirogov's case, out of 400 patients operated on in 1850-1852, 159 died from infection. Five stages are distinguished in the development of asepsis and antiseptics:

- empirical period;
- Listerian antiseptics of the 19th century;
- Lister's antiseptic;
- occurrence of asepsis;
- modern asepsis and antiseptics.

An empirical approach to the use of "antiseptic" methods can be found in doctors of ancient times. Thus, ancient surgeons considered it mandatory to remove a foreign body from a wound. Hippocrates preached the principle of cleanliness of the doctor's hands, demanded that nails be cut short; used boiled rainwater and wine to treat wounds; shaved the hair from the surgical field, and spoke about the need for cleanliness of the dressing material. In the middle of the 19th century, even before the works of J. Lister, some surgeons began to use methods to destroy infection in their work. A special role in the development of antiseptics during this period was played by I. Semmelweis and M. I. Pirogov.

Lister's antiseptic. In the 60s of the 19th century in Glasgow, the English surgeon Joseph Lister, having become acquainted with the works of Louis Pasteur, came to the conclusion that microorganisms enter the wound from the air and from the hands of the surgeon. In 1865, having become convinced of the antiseptic effect of carbolic acid,

which in 1860 the Parisian pharmacist Lemer began to use, he used a bandage soaked in carbolic acid in the treatment of an open fracture. At the same time, he sprayed carbolic acid into the air of the operating room. In 1867, Lister published an article in the journal "Lancet" "On a new method of treating fractures and abscesses, taking into account the causes of suppuration." It outlined the basic principles of the antiseptic method he proposed. The method included: 1) spraying carbolic acid into the air of the operating room; 2) treatment of instruments, sutures and dressings with a 2-3% solution of carbolic acid; 3) treatment of the operating field and the surgeon's hands with a 2-3% solution of carbolic acid; 4) use of a special dressing after the operation: the wound was closed with a multilayer bandage impregnated with carbolic acid. A special role in the spread of Lister's antiseptics in Tsarist Russia was played by M. I. Pirogov, P. P. Pelekhin and I. I. Burtsev. In particular, M. I. Pirogov used the healing properties of carbolic acid in the treatment of wounds.

The successes of microbiology have put forward new principles for the prevention of surgical infection. The main one is to prevent bacterial infection of the surgeon's hands and objects in contact with the wound. Thus, surgery included: processing the surgeon's hands, sterilization of instruments, dressings, linen, etc. These simple postulates formed the basis of asepsis. The development of the aseptic method is associated with the names of two outstanding scientists: E. Bergman and his student K. Schimmelbusch. At the X International Congress of Surgeons in Berlin in 1890, the principles of asepsis in the treatment of wounds received general recognition. By the mid-90s, antiseptic and aseptic methods had found their way to most European and American surgical clinics. Asepsis and antiseptics, as fundamental postulates of clinical surgery, were successfully tested on the battlefields of the First World War. In 1897, Jan Mikulicz-Radecki (1850-1905), a Polish-Austrian surgeon, developed a gauze mask to be worn during surgical operations, as microbiologists had shown that bacterial discharges from the mouth and nose increased the likelihood of wound infection. Therefore, silence and appropriate dress in the operating room became a cardinal feature of surgery.

Pain control. Until the middle of the 19th century, surgeons could not cope with pain during surgery, which significantly slowed down the development of surgery. In the middle and late 19th century, a number of turning points occurred that contributed to the rapid development of anesthesiology. In particular, in 1800, Davy discovered the peculiar effect of nitrous oxide, calling it “laughing gas”. In 1818, Faraday discovered the intoxicating and blocking effects of diethyl ether. And in 1844, dentist Horace Wells (1815-1848), a dentist from Connecticut, used nitrous oxide for pain relief during tooth extraction. The very first operation under anesthesia (ether) was performed in 1842 by the American surgeon Long. On October 16, 1846, in Boston Hospital, Harvard University professor John Warren removed a tumor from the submandibular region of a 20-year-old patient, Gilberto Abbott, under anesthesia. M. I. Pirogov and A. M. Filomafitsky played a major role in the development of anesthesiology in Russia. M. I. Pirogov used anesthesia on the battlefield, studied various ways of administering diethyl ether (into the trachea, blood, gastrointestinal tract), and became the author of rectal anesthesia.

The emergence of endotracheal anesthesia. An important achievement in anesthesiology was the use of artificial respiration, in which the main merit belongs to R. Mackintosh. He also became the organizer of the first department of anesthesiology at Oxford University in 1937. During operations, curare-like substances began to be used to relax muscles, which is associated with the name of Griffiths (1942).

An important event in the development of surgery was the scientific justification and practical implementation of the idea of blood transfusion from one person to another. The most important events of this period are as follows:

- 1901 - the discovery by the Viennese bacteriologist Karl Landsteiner of three human blood groups (A, B, C) - due to the properties of the serum and erythrocytes of their blood, they cause the phenomenon of isohemagglutination (gluing of erythrocytes);
- 1907 - Czech scientist J. Jansky proved that the new blood group is independent and that all people are divided into four groups according to the immunological properties of their blood, not three, and designated them with Roman numerals (I, II, III and IV);
- 1910-1915 - discovery of a method for stabilizing blood. In the works of V. Yurevych and N. Rosengart (1910), Gusteau (1914), Levinson (1915), and Agota (1915), a method for stabilizing blood with sodium citrate was developed;
- 1919 - V. N. Shamov, N. N. Elansky and R. R. Negrov received the first standard serums for determining blood group and performed the first transfusion
- 1940 - discovery by K. Landsteiner and A. Wiener of the Rh factor - the second most important antigenic system, which plays an important role in immunohematology. Later, in addition to the known erythrocyte antigens, platelet antigens were discovered in 1953, leukocyte antigens in 1954, and antigenic differences of blood globulins in 1956.

The current period of development of surgery at the beginning of the 21st century. can be called a technological period. Since the progress of surgery is determined not only by the development of certain anatomical and physiological concepts, or the improvement of manual surgical skills, but also by more advanced technical support, powerful pharmacological support and impressive capabilities of biotechnology. Vivid examples of the rapid development of surgery are the successes of transplantology, cardiac surgery, vascular surgery and microsurgery, endovideosurgery, experimental and clinical immunology.

It is worth noting the surgeons who won the Nobel Prize in Medicine and Physiology.

| Surgeon | | Country | Problem, award year |
|---|---|---|---|
| 1 | Theodor Kocher (1841–1917) | Switzerland | Thyroid disease (1909) |
| 2 | Allvar Gullstrand (1862–1930) | Sweden | Eye diopter (1911) |
| 3 | Alexis Carrel (1873–1944) | France and the USA | Vascular surgery (1912) |
| 4 | Robert Bárány (1876–1936) | Austria | Vestibular disease (1914) |

| | | | |
|---|---|---|---|
| 5 | Frederick Banting (1891–1941) | Canada | Insulin (1922) |
| 6 | Walter Hess (1881–1973) | Switzerland | Physiology of the midbrain (1949) |
| 7 | Werner Forssmann (1904–1979) | Germany | Cardiac catheterization (1956) |
| 8 | Charles Huggins (1901–1997) | USA | Oncology (1966) |
| 9 | Joseph Murray (1919-2012) | USA | Organ transplantation (1990) |

In the territory of Ukraine, back in the times of Kyivan Rus (9th-13th centuries), the first professional doctors (so-called artisans) began to appear, who provided medical care in large cities. Some of them had a special talent for treating wounds, fractures, and bloodletting. The basis of their knowledge was the centuries-old experience of folk empirical medicine. Later, in the 14th century, when guild medicine emerged, artisan doctors, who were already called "barbers" by that time, united in guilds. It was the guilds that became the basis for the creation of both the first hospitals and schools of medical personnel. Individual barbers possessed extraordinary skill in treating wounds, pulling teeth, performing amputations, operations to remove stones, and, especially, in the very common means of treatment at that time - bloodletting. The largest guilds existed in Lviv, Kamianets-Podilskyi, and Kyiv.

In the 15th century, brotherhoods emerged in Ukraine - organizations of the Ukrainian Orthodox bourgeoisie. They were engaged in educational activities and also organized brotherhood hospitals, which, like schools, were maintained at the expense of parishioners. In hospitals, bloodletting was done, abscesses were opened, teeth were pulled, wounds were bandaged, dislocations were set, and healing plasters were made.

At this time, a number of higher educational institutions appeared in Ukraine: collegiums were opened in Ostroh, Lviv, and Kyiv. The Kyiv collegium, established in 1632 by Petro Mohyla, acquired the status of an academy during the hetmanship of I. Mazepa.

The organization of surgical care in the Zaporizhzhia army was unique. During campaigns, the Cossacks treated themselves: they covered their wounds with a small amount of earth, which they had previously rubbed on their palms with saliva. To get rid of fever, they diluted half a charge of gunpowder in a glass of vodka, drank this mixture and went to bed. The Zaporizhzhia army maintained a number of hospitals at its own expense, the most famous of which were Trakhtemyrivsky and Mezhyhirsky.

In the 18th century, a number of hospital schools were established in the Russian state. One of them was in Ukraine, in Elisavetgrad. The students of these institutions were mainly from Ukrainian brotherhood schools, colleges and the Kyiv Academy. The best graduates were sent abroad to obtain a doctorate. The first nine doctors who received the title of Doctor of Medicine in Leiden were Ukrainians, graduates of the Kyiv Academy. Here are a few names worth mentioning in this regard.

Buyalsky Ilya Vasilyevich (1789-1864) - the son of a village priest from Chernihiv region. He was a wonderful anatomist and a virtuoso surgeon. He published "Anatomical and Surgical Tables" with a text about topography and operations on vessels and removal of stones. The atlas was translated into all European languages. I.V. Buyalsky was the first to successfully perform an operation to resect the upper jaw, tied the innominate artery twice, and developed an original method of draining

inflammatory processes in the pelvis through the obturatorium foramen, which in surgery bears his name to this day. He introduced many different instruments into surgical practice, of which Buyalsky's spoon and curette have survived in surgical sets to this day. The first major work on military field surgery, entitled "Military and Campaign Medicine", in 5 parts (1836-1837), was written by Yakym Charukivskyi (1798-1848), who was originally from the Poltava region.

The Medical Faculty at Lviv University operated from 1784 to 1805. The second year of this university taught general surgery, and the fourth year - special surgery. In 1795, several clinics were opened at the university. Surgery was taught by August Kriegel, Fried, France, and Mazel; the Medical Faculty had no Ukrainian professors at that time. In 1805, instead of the Medical Faculty in Lviv, a two-year medical and surgical school was founded, and in 1833 it was reorganized into a three-year school. Surgery was taught in the second and third years of this school. In 1894, the Medical Faculty was again created at Lviv University, and L. Ridiger has been working there as a professor of surgery since 1897. Professor Ridiger was the first in the world to perform a gastric resection for duodenal ulcer; he is a recognized authority in emergency surgery, urology, orthopedics, and proctology.

The pioneer of antiseptics in Russia was Pavel Petrovich Pelekhin (Pelekh) (1842-1917). A few months after the publication of the works of the English scientist Lister on antiseptics, P.P. Pelekhin went to England and studied this method with the author himself, and after returning home he published his first work on antiseptics "The Success of New Ideas in Surgery in the Treatment of Wounds, Complex Fractures and Purulent Accumulations". Using the antiseptic method, the scientist reduced the mortality rate from surgical interventions from 50% to 7%. In 1898, P.P. Pelekhin transferred his family's savings (70 thousand Russian rubles in gold) to the T.G. Shevchenko Scientific Society in Lviv in order to organize the "Pyotr Pelekhin Department of Surgery" at the first Ukrainian university.

The bodies of two prominent 19th-century surgeons, Mykola Ivanovich Pirogov (1810-1871) in the village of Vyshnia, near Vinnytsia, and Mykola Vasilyovich Sklifosovsky (1836-1904) in the village of Yakivka in the Poltava region, are buried on Ukrainian soil.

In the second half of the 19th century, surgical clinics of medical faculties in Kharkiv and Kyiv became real centers of surgical science. Thus, at Kharkiv University, the Department of Surgery existed initially at the Department of Medicine and Medical Sciences, and since 1835 - at the Medical Faculty. Since 1814, a small surgical clinic has been opened at this educational institution. Honorary members of the Council of Kharkiv University were Petro Zahorsky and Ilya Buyalsky, and the first professor of surgery was Pavlo Shumlyansky.

Pavlo Mykhailovych Shumlyansky (1750-1824) was born in Poltava region, studied at the Kyiv-Mohyla Academy, the St. Petersburg Hospital School at the General Land Hospital, in 1779 he received the title of doctor, after which he worked in the army and studied abroad. In 1789 he defended his dissertation for the degree of Doctor of Medicine at the University of Strasbourg on the topic: "De proxsma topicae inflammationes causa", from 1790 p. he was a teacher at medical and surgical schools in St. Petersburg and Kronstadt, from 1795 - professor of pharmacology and surgery at

the Moscow Medical and Surgical School, and from 1799 - professor of surgery at the Moscow Medical and Surgical Academy. In 1805 he was elected professor of surgery and dean of the medical faculty of Kharkov University.

From 1821 to 1833, the Department of Surgery at Kharkiv University was headed by M.I. Yellinsky. Mykola Ivanovych Yellinsky (1789-1834) was born in Ukraine, graduated from the Medical Faculty of Kharkiv University in 1817, then worked at the St. Petersburg Medical and Surgical Academy, where he improved his knowledge of surgery and anatomy. In 1821, he was elected Professor of Surgery at the Medical Faculty of Kharkiv University (during 1830-33, he also worked as the rector of the university. M.I. Yellinsky is the author of the first manual on desmurgy in two volumes, which describes in detail the use of gypsum in the treatment of fractures.

In 1858, Wilhelm Fedorovych Grube (1827-1898) was elected to the Department of Surgery at Kharkiv University. He was of Estonian origin and graduated from Yuriev (Tartus) University in 1850. In his "Essays and Observations from the Optional Surgical Clinic", the scientist reported that he had used carbolic acid as early as 1865. In 1871, V.F. Grube performed the first operation under nitrous oxide anesthesia in Kharkiv and published a number of works in the 1880s on the use of chloroform and morphine.

An outstanding Ukrainian surgeon of the 19th century who devoted his life to Kharkiv University was Professor A.G. Podrez. Apollinariy Grigorovich Podrez (1852-1900) was born in the Kharkiv region. In 1875 he graduated from the medical faculty of Kharkiv University, in 1878 he defended his doctoral dissertation on the topic "On stretching nerves", then worked as a resident at a military hospital in Kharkiv. A.G. Podrez was the first in the world in 1897 to remove a foreign body from the heart, in 1887 he was the first in Ukraine (and in Russia) to perform a successful splenectomy, wrote the first textbook on urology in Ukraine and Russia, proposed original methods of colostomy, gastroenterostomy, treatment of narrowing of the urinary canal, proposed a method of intraperitoneal connection of the ureter with the bladder, etc.

M.F. Grube's student - Mykola Petrovich Trinkler (1859-1925) - graduated from the Medical Faculty of Kharkiv University in 1883, later worked at the Department of Surgery, in 1889 he interned at the clinic of E. Bergmann in Germany, in the same year he defended his doctoral dissertation "On the Surgery of Transverse Fractures of the Patella". Since 1905 he was a professor of the Departments of Surgical Pathology and Therapy of Kharkiv University, and since 1913 - a professor of the Faculty Surgical Clinic. M.P. Trinkler is the author of 78 scientific works devoted to the issues of oncology, neurosurgery, abdominal surgery, and traumatology.

The first professor of surgery at Kyiv University was V.O. Karavaev - one of the best surgeons of the 19th century. Vladimir Opanasovich Karavaev (1811-1892) in 1831 graduated from the medical faculty of Kazan University, worked for two years in St. Petersburg, then was on a foreign mission in Germany, in 1836-38 worked in Dorpat (Tartu) under the leadership of M.I. Pirogov, in 1838 defended his dissertation on the topic "De phlebitide traumatica". In 1841 p. he was invited to Kyiv University to the position of dean of the medical faculty and professor of surgery. V.O. Karavaev's services to Ukrainian medicine are very significant. He was one of the organizers of

the medical faculty of Kyiv University, a talented teacher, an outstanding clinician and scientist. Being a good expert in topographic anatomy, operative surgery, masterfully mastering surgical technique, he achieved brilliant success in performing many complex operations, in particular in ophthalmology, paid much attention to the work of students and doctors with patients, in the operating room, in the outpatient clinic. Under his influence, many hundreds of doctors became surgeons and worked in Ukraine not only in cities, but also in villages. His students were such outstanding surgeons as M.M. Volkovich, K.M. Sapezhko, I.F. Sabaneev, O.T. Bagaevsky, Ya.B. Zilberberg and others. Scientific works of V.O. Karavaev were devoted to ophthalmology, rhinoplasty, ovariotomy, amputation. He wrote manuals on operative surgery: "Course of Operative Surgery" (Kiev, 1858) and "Operative Surgery" (Kiev, 1886), which for a long time were desk books of doctors and students. He was the first in the world to perform an operation to drain the pericardial sac, developed a technique for performing such an operation in cases of effusion pericarditis, applied his own method of cataract removal, cleft lip surgery, etc. The Kyiv City Council elected V.O. Karavaev an honorary citizen of the city, named the street where he lived after him, a bronze bust of the scientist stands in the auditorium of the faculty surgical clinic, and his grave is carefully looked after at the Baykovo cemetery.

Among the prominent Ukrainian surgeons, the following names are worth mentioning.

**VOLKOVYCH Mykola Markiyanovych** (11.28.1858-07.11.1928) - Academician of the Academy of Sciences of the Ukrainian SSR (1928), Doctor of Medicine, Honored Ordinary Professor (1914), Head of the Department of Hospital (1903-1911) and Faculty (1911-1923) Surgery. He was the first in Ukraine to combine otology and laryngology into a single clinical discipline and from 1889 to 1903 he taught a course in otorhinolaryngology. A student of V.O. Karavaev and F.K. Bornhaupt. He graduated from the Medical Faculty of Kyiv University (1882). In 1889 he defended his dissertation for the degree of Doctor of Medicine on the topic "Rhinoscleroma with clinical, pathological and bacteriological side effects". Author of over 80 scientific works, including 3 monographs. He discovered and studied the causative agent of rhinoscleroma (1888), which was called the Volkovich-Frisch stick. He described the symptom of chronic appendicitis - Volkovich's muscular symptom (1911), developed methods of treating bone fractures (Volkovich's splint), and proposed a number of new methods of plastic surgery. He was one of the first in Russia to perform laminectomy (1894). M.M. Volkovich created a domestic school of surgeons and orthopedists (O.S. Delens, M.Ya. Kharshak, P.S. Babitsky, A.F. Gorbachovsky, etc.). The scientist was the founder (1908) and permanent chairman of the Kyiv Scientific Surgical Society.

**KRYMOV Oleksiy Petrovich** (31.07.1872-11.12.1954) - Academician of the Academy of Medical Sciences of the USSR (1945), Doctor of Medicine, Professor, Honored Scientist of the Ukrainian SSR (1940), Professor of the Hospital Surgical Clinic of the Medical Faculty of the Kyiv University of St. Volodymyr (1912), Head of the Department of Hospital (1913-1923), Faculty (1930-1954) Surgery. From 1923 to 1928 - Head of the Surgical Department of the 3rd, and then the 2nd Working Hospital of the city of Kyiv. At this time, he participated in the organization of the

Ukrainian Medical Institute. O.P. Krymov - Graduate of the Medical Faculty of Moscow University (1898), Student of A.A. Bobrov and S.I. Spasokukotsky, author of over 135 scientific papers, including 10 monographs devoted to the problems of clinical surgery: skull and brain injuries, brain infection, gunshot aneurysms, lung abscesses and hernias. A participant in five wars, O.P. Krymov is the author of over 30 scientific papers on wartime surgery, including lectures on military field surgery. His works devoted to injuries of blood vessels deserve special attention. He proposed a method of radical surgery for inguinal hernias, surgery for dilation of the veins of the spermatic cord, the method of nephropexy. O.P. Krymov is a laureate of the I.F. Bush and S.P. Fedorov prizes, chairman of the Ukrainian Society of Surgeons since 1936. Awarded the Orders of Lenin, the Red Banner of Labor, the Red Star, the medal "For Valiant Labor in the Years Great Domestic " Wars " (1941-1945). Under his leadership, 35 dissertations were prepared, including 15 doctoral dissertations. The scientist's students are M. Ishchenko, O.O. Fedorovsky, Y.G. Turovets, V.D. Bratus. The name of Academician O.P. Krymov was assigned to the faculty surgical clinic of the Kyiv Medical Institute.

In the 1920s and 1930s, S.S. Bryuhonenko and Kyiv pathophysiologist V.D. Yankovsky created the world's first artificial circulatory system (ACS). This invention largely determined the entire subsequent development of cardiac surgery on a global scale.

**AMOSOV Mykola Mikhailovich** (06.12.1913 - 13.12.2002) - Academician of the NAS of Ukraine (since 1969) and the AMS of Ukraine (since 1993, corresponding member of the RAMS (1961), Honored Scientist of the Ukrainian SSR (1959), Hero of Socialist Labor (1973), laureate of the Lenin Prize (1961) and three State Prizes, writer. Graduated from the Arkhangelsk Medical Institute (1939) and the All-Union Correspondence Industrial Institute (1940). In 1952, he defended his doctoral dissertation on the topic "Pneumonectomy and lung resection in tuberculosis". Head of the Department of Surgery of the Kyiv Research Institute of Tuberculosis and Thoracic Surgery (1952-1968), Department of Surgery of the Institute for Advanced Training of Doctors (1955-1970), Department of of Biological Cybernetics of the Institute of Cybernetics of the Academy of Sciences of the Ukrainian SSR (1960). Since 1983, Mykola Mikhailovich has held the position of Director of the Research Institute of Cardiovascular Surgery, and since 1988, he has been its Honorary Director. Mykola Mikhailovich is the author of over 450 scientific papers, including 19 monographs devoted to thoracic surgery, biocybernetics, etc. M.M. Amosov is one of the founders of pulmonary and cardiac surgery in the USSR. He developed new methods of lung operations and surgical treatment of heart defects. He was the first in the world to create and implement antithrombotic heart valve prostheses. M.M. Amosov is the author of original artificial blood circulation devices, the organizer of the Ukrainian Center for Cardiovascular Surgery, the founder of the Ukrainian School of Biomedical and Psychological Cybernetics, a member of the board of the Ukrainian Society of Surgeons and Cardiologists, the International Association of Surgeons, the International Society of Cardiovascular Surgeons. Mykola Mikhailovich is the author of the books "Thoughts about the Heart", "Notes from the Future", "PPG 2266". He

was awarded two Orders of Lenin, the Order of the Patriotic War of the 1st degree, two Orders of the Red Star and medals.

**Danylenko Mykhailo Vasylevich** (1918 - 2002). Corresponding Member of the National Academy of Sciences (1992) and the Academy of Medical Sciences (1993) of Ukraine, Corresponding Member of the Russian Academy of Medical Sciences (1975). Honored Scientist of Ukraine (1968), Honorary Doctor of Sciences of the Medical University of Pecs (Hungary, 1974), Full Member of the International Society of Surgeons (1978), Doctor of Medical Sciences (1959), Professor (1960), Scientific Consultant of the Department of Hospital Surgery of the Lviv State Medical University. A well-known scientist-surgeon, teacher. The main areas of his activity are: heart and vascular surgery, general surgery and anesthesiology, resuscitation and intensive therapy, history of medicine and higher education. Author of 376 works, including 10 monographs and manuals. Under his supervision, 15 doctors and 62 candidates of sciences were trained. Author of scientific works: "Paragonimosis" (1963); "Mitral stenosis and its surgical treatment", (1970); "Trichlorethylene analgesia" (1971); "Corrective therapy in modern surgery" (1974); "Instructions for surgery" (1979); "Intensive postoperative therapy" (1984).

**Knyshov Gennady Vasylevich**, (1934 – 2015) Academician of the National Academy of Medical Sciences of Ukraine (1994), Corresponding Member of the National Academy of Medical Sciences of Ukraine, Doctor of Medical Sciences (1975), Professor (1984), Laureate of the State Prize of Ukraine (1988), Honored - Worker of Science and Technology of Ukraine (1994), Director of the Institute of Cardiovascular Surgery of the National Academy of Medical Sciences of Ukraine (since 1988), Head of the Department of Cardiovascular Surgery of the Kyiv Medical Academy of Postgraduate Education (since 1992). The main areas of scientific research are: improving the surgical treatment of acquired heart defects, ischemic heart disease and complex cardiac arrhythmias. He developed a number of original operations for the treatment of mitral stenosis, prosthetic heart valves. He was the first in Ukraine to introduce and improve coronary artery bypass grafting in the treatment of ischemic heart disease, complex operations for the treatment of cardiac tachyarrhythmias. Under his leadership, the most complex emergency operations for the correction of severe congenital heart defects in newborns were introduced into clinical practice. Author of 300 scientific papers, 9 inventions, 7 monographs. Under his leadership, more than 20 doctoral and candidate theses were defended.

**ISCHENKO Ivan Mykolayovych** (22.06.1891-22.11.1975) - Corresponding Member of the Academy of Sciences of the Ukrainian SSR (1945), Doctor of Medical Sciences (1941), Professor, Honored Scientist of the Ukrainian SSR (1942), Major General of the Medical Service, Head of the Department of General (1944-1955) and Faculty (1955-1968) Surgery, Professor-Consultant until 1975. Ivan Mykolayovych graduated from the Medical Faculty of the Kyiv Medical University in 1917, a student of O.O. Bogomolets and O.P. Krymov. In 1931-1941 and in 1943-1954 he held the positions of Consultant Physician, Chief Surgeon of the Kyiv Military District. In 1937-1941 worked as a scientific director of the Kyiv Institute of Emergency Surgery and Blood Transfusion. I.M. Ishchenko is the author of over 100 scientific works, including 3 monographs and 2 textbooks, which are devoted to the issues of urology,

abdominal surgery, clinic and therapy of craniocerebral trauma. He was a pioneer of neurosurgery in Ukraine, one of the first to recommend the use of intubation potentiated anesthesia. Ivan Mykolayovych - Chairman of the Board of the Republican, Kyiv City and Regional Society of Surgeons (1954-1966), member of the Board of the All-Union Society of Surgeons. Participant of the Great Patriotic War. Awarded two Orders of Lenin, two Orders of the Red Banner, Orders of the Red Star, "Badge of Honor" and numerous medals.

**KOLOMIYCHENKO Mykhailo Isidorovych** (07.11.1892-30.05.1973) - professor (1936), Honored Scientist of the Ukrainian SSR (1954), head of the Department of General Surgery (1955-1973), deputy director of the Kyiv Medical Institute for scientific work (1930-1935), dean of the Faculty of Dentistry (1931-1934).

Mykola Isidorovych - a graduate of the Kyiv Medical Institute (1919), a student of E.G. Chernyakhivsky, N.M. Volkovich, O.P. Krymov, author of over 130 scientific works, including 5 monographs devoted to various issues of surgery of the esophagus, stomach, pancreas and problems of emergency surgery, in particular pathology of the esophagus. He was the first in Ukraine to perform complex plastic surgeries to create an artificial esophagus from the small and large intestines. M.I. Kolomiychenko - a participant in the Great Patriotic War - was awarded two Orders of Lenin, two Orders of the Red Banner of Labor, Orders of the Patriotic War of the 1st and 2nd degree, the "Badge of Honor", the October Revolution and medals.

**FEDOROVSKY Oleksiy Oleksandrovych** (03.15.1897-08.28.1981) Doctor of Medical Sciences (1951), laureate of the State Prize of the Ukrainian SSR (1959), professor of the Department of Faculty Surgery (1949), founder and first head of the Department of Surgery of the Pediatric Faculty (1953-1969), dean of the Pediatric Faculty (1957-1958), consulting professor (1971-1981).

O.O. Fedorovsky - a graduate of the Kharkiv Medical Academy (1921), a student of N.P. Trinkler - in 1949 defended his doctoral dissertation on the topic "New blood stabilizer Natrog". On his initiative in 1958, the first burn center of the Ukrainian SSR was created. The scientist was one of the organizers, and later the director of the Kyiv Institute of Blood Transfusion (1935). Back in 1941, O.O. Fedorovsky developed and proposed a new domestic blood preservative "NATROG", for which in 1952 he received the Academician N.N. Burdenko Prize. Under his leadership, 4 doctoral and 29 candidate theses were defended. Alexey Alexandrovich is a member of the International Society of Transfusionists and an honorary member of the All-Union and Ukrainian Societies of Surgeons, a holder of the Orders of the Red Star, the Patriotic War II degree, the "Badge of Honor", and has been awarded numerous medals.

**KARAVANOV Georgy Grigorovich** (20.10.1899-04.01.1982) - Soviet surgeon, professor, head of the Department of Faculty Surgery of the Lviv Medical Institute (1944-1973), author and co-author of over 400 scientific papers. G.G. Karavanov developed methods of surgical treatment of elephantiasis, adrenal glands, biliary tract and pancreas. Permanent head of the Lviv Scientific Society of Surgeons, trained 12 doctors and 52 candidates of sciences.

**SYTKOVSKY Mykola Borisovich** (20.08.1916-31.07.2003) - Doctor of Medical Sciences (1964), Professor (1969), Honored Scientist of the Ukrainian SSR, Laureate of the State Prize of the Ukrainian SSR (1982), Head of the Department of

Pediatric Surgery and Orthopedics (since 1968), First Dean of the Faculty of Advanced Training of Doctors organized by him (1968-1976), Chief Pediatric Surgeon of the Ministry of Health of Ukraine (1969-1995). In 1941, M.B. Sytkovsky graduated from the 2nd Kyiv Medical Institute, in 1963 he defended his doctoral dissertation on the topic "Anomalies of the development of the rectum in children and their treatment". Author of over 340 scientific works, including 10 monographs. Participant of the Great Patriotic War. Awarded the Orders of the Red Star, the Patriotic War, the Badge of Honor, the Polish Order of Merit Cross, and medals.

**KOVALEV Mikhail Markov** (08.11.1916-14.04.1990) - Doctor of Medical Sciences, Professor (1961), Honored Scientist of the Ukrainian SSR, laureate of the State Prize of the Ukrainian SSR (1986), Head of the Department of Hospital Surgery (1962-1985), since 1985 - Professor of the Department. Graduated from the Donetsk Medical Institute (1948). Author of about 400 scientific works, including 8 monographs devoted to the issues of urology, surgical endocrinology, gastroenterology. Developed and implemented the method of sparing resection of the thyroid gland. M.M. Kovalev is a member of the International Society of Surgeons, the boards of the All-Union and Republican Scientific Societies of Surgeons and Endocrinologists. Under his leadership, 17 doctoral and 53 candidate theses were completed. M.M. Kovalev - a participant in the Great Patriotic War, awarded the Orders of Lenin, the Patriotic War of the 1st and 2nd degrees, the Red Star, two orders: the Red Banner of Labor and medals.;

**Vasyl Dmytrovych BRATUS** (26.12.1916-09.10.2008) - Corresponding Member of the National Academy of Sciences and the Academy of Medical Sciences of Ukraine (1972), Doctor of Medical Sciences (1963), Professor (1963), Laureate of the O.O. Bogomolets Prize of the Academy of Sciences of the Ukrainian SSR (1969) and the State Prize of the Ukrainian SSR (1981), Honored Scientist of Ukraine (1989), Head of the Department of Surgery of the Faculty of Dentistry (1964-1984), of Surgery of the Faculty of Pediatrics (1984-1992), Professor of the Department (since 1992). He held the positions of Minister of Health of the Ukrainian SSR (1954-1956 and 1968-1975), rector of the Kyiv Institute for Advanced Training of Doctors (1957-1967), then of the Medical Institute (1959-1966).

In 1940, V.D. Bratus graduated from the Kuibyshev Military Medical Academy, and in 1962 he defended his doctoral dissertation on the topic "Surgical treatment of thermal burns." V.D. Bratus is the author of over 320 scientific papers, a participant in the Great Patriotic War - awarded the Orders of Lenin, the October Revolution, the Red Banner of Labor, the Red Star, the Patriotic War, and medals.

**SHALIMOV Oleksandr Oleksiyovych** (01.20.1918-02.29.2006) - founder of the modern Ukrainian surgical school. A man with a capital letter, a surgeon with a world name, an outstanding scientist, a full member of the NAS and AMS of Ukraine, Doctor of Medical Sciences, Professor, Hero of Socialist Labor, Hero of Ukraine, Honored Scientist and Technician of Ukraine, Honored Doctor of the Russian Federation, laureate of the State Prizes of the USSR and Ukraine, Honorary Director of the Institute of Surgery and Transplantology of the AMS of Ukraine, Honorary Chairman of the Board of the Association of Surgeons of Ukraine, member of the Board of the Association of Surgeons named after M.I. Pirogov, member of the

International Association of Surgeons, full member of the New York Academy of Sciences, editor-in-chief of the journal "Clinical Surgery".

In 1941, O.O. Shalimov graduated from the medical faculty of the Kuban Medical Institute, in 1965 he headed and reorganized the Kharkiv Research Institute of General and Emergency Surgery, in 1970-1971 he headed the Department of Thoracoabdominal Surgery of the Kyiv Institute for Advanced Training of Doctors, in 1971-1972 he held the position of director of the Kyiv Research Institute of Hematology and Blood Transfusion. In 1972, O.O. Shalimov created the Kyiv Research Institute of Clinical and Experimental Surgery (now the Institute of Surgery and Transplantology of the NAMS of Ukraine), which he headed for 16 years. Since 1988, Oleksandr Oleksiyovych was elected honorary director of the Institute of Surgery and Transplantology of the Academy of Medical Sciences of Ukraine. From 1980 to 2004, O.O. Shalimov held the position of Chief Surgeon of the Ministry of Health of Ukraine. The scientific and practical activities of O.O. Shalimov were devoted to the development of various areas of modern surgery: reconstructive and restorative treatment of pathologies of the esophagus, stomach, intestines, bile ducts, liver; correction of portal hypertension, methods of surgical treatment of pancreatic diseases, cardiac surgery, reconstructive and restorative vascular surgery, microsurgery, endovascular surgery, cryosurgery, as well as other issues of modern surgical science. Shalimov developed and implemented into practice numerous original methods of surgical interventions: esophageal plasty, gastric resection, selective proximal vagotomy, bile duct plasty, pancreatoduodenal resection, methods of operations for acute and chronic pancreatitis, obliterating atherosclerosis of the vessels of the extremities, created an original artificial blood circulation apparatus, dilators for mitral commissurotomy, many other devices and instruments. During his bright scientific activity, O.O. Shalimov received 112 copyright certificates and patents for invention, published more than 870 scientific works, including 37 monographs. Under the leadership of O.O. Shalimov, 46 doctoral and about 100 candidate theses were defended. For the fruitful many years of work of O.O. Shalimov was awarded two Orders of Lenin, the Order of the October Revolution, two Orders of the Red Banner of Labor, the Orders of Merit of the I-III degrees, and medals. For 22 years, O.O. Shalimov was elected a deputy of the Supreme Soviet of the Ukrainian SSR, where he headed the Permanent Commission on Health Care. The International Chamber of the American Biographical Institute elected O.O. Shalimov "Person of the Year" in 1997. Shalimov was awarded the Honorary Diploma of the International Biographical Center of the University of Cambridge for achievements in medicine of the 20th century.

**Ivan Ivanovich SUKHAREV** (05.06.1939-05.07.2003) - Doctor of Medical Sciences (1981), Professor (1991), Honored Worker of Science and Technology of Ukraine (1992), Laureate of the State Prize of Ukraine (1994). I.I. Sukharev - founder of vascular surgery in Ukraine, chief vascular surgeon of the Ministry of Health of Ukraine (1992-2003), founder and first president of the Association of Vascular Surgeons of Ukraine (2001-2003), member of the European Association of Vascular Surgeons, head of the Department of Vascular Surgery (1972-2003), deputy director for scientific work (1995-2003) of the Institute of Surgery and Transplantology of the National Academy of Medical Sciences of Ukraine.

The main areas of scientific and practical activity of I.I. Sukharev were: surgery of obliterating atherosclerosis of the abdominal aorta and its branches, surgery of atherosclerosis of extracranial arteries, repeated reconstructive interventions on the abdominal aorta and arteries of the lower extremities, surgery of purulent-septic complications after reconstructive operations on vessels, surgery of diseases of the superior and inferior vena cava, problems of reperfusion syndrome. Under the leadership of 1.1. Sukharev, 5 doctoral and 18 candidate theses were defended. He published 308 scientific papers, received 42 author's certificates for patents. 1.1. Sukharev was awarded 4 medals, a diploma of the Verkhovna Rada of Ukraine "For Services to the Ukrainian People".

**DANYLENKO Mykhailo Vasylevich** (10.08.1918-17.10.2002) - Corresponding Member of the Academy of Medical Sciences of the USSR (1975), the National Academy of Sciences (1992) and the Academy of Medical Sciences of Ukraine (1993), Head of the Department of Thoracic (1964-1981) and Hospital (1981-1991) Surgery, Head of the Center for Cardiovascular Surgery and Rector (1964-1981) of the Lviv Medical Institute. M.V. Danylenko graduated from the Saratov Medical Institute (1946), worked as an advisor to the Minister of Health in the DPRK (1953-1955), and headed the Departments of Surgery at the Vinnytsia and Ternopil Medical Institutes (1959-1964). M.V. Danylenko is the author (co-author) of over 370 works on heart and vascular surgery, general surgery and anesthesiology, resuscitation and intensive care. Having significantly developed and expanded the Institute, he contributed to its "internationalization", displacing the Ukrainian language from the educational process.

Ukraine should be considered one of the leading countries in the establishment of both experimental and clinical developments in the problem of organ and tissue transplantation. V.P. Filatov was the first to perform a cadaveric cornea transplant, and V.M. Shamov was the first to perform a cadaveric blood transfusion. In April 1933, a student of Professor V.M. Shamov - Dr. Yu.Yu. Voron was the first in the world to perform a kidney transplant from a male corpse to a woman who had been poisoned with sulema. The kidney, removed from the corpse 6 hours after death and transplanted into the vessels of the recipient's thigh, began to secrete urine. The concentration of sulema in the blood decreased 10 times, but the patient's serious general condition still caused her death.

In 1969, under the leadership of BC Karpenko, a team of urologists, nephrologists, immunologists, electrophysiologists, microbiologists and morphologists began to study experimental and clinical problems of kidney transplantation, which allowed on May 16, 1972 BC Karpenko with his colleagues E.Ya. Baran, P.S. Vukalovych, O.S. Pereverzev, Y.E. Serhiychuk, M.T. Terekhov and P.S. Chernenko to perform the first successful kidney transplant in Ukraine from a living donor to his sick brother with a positive result.

**KARPENKO Viktor Stepanovych** (1923-2003). Laureate of the State Prize of Ukraine (1983), Honored Scientist of Ukraine (1976), Doctor of Medical Sciences (1968), Professor (1969), Director of the Kyiv Research Institute of Urology and Nephrology (1969), Head of the Department of Plastic and Reconstructive Urology of the Kyiv Research Institute of Urology and Nephrology (1987), Chief Urologist of the

Ministry of Health of Ukraine (1970-1987). Leading scientist-urologist, teacher, organizer of the urological service in Ukraine. The main areas of his activity are: kidney transplantation and autotransplantation, prostate surgery, ureterohydronephrosis, pathology of a single functioning kidney, bladder cancer. Author of over 250 scientific works, including 12 monographs, reference books, manuals. Prepared 15 doctors and 34 candidates of sciences, author of 11 inventions.

**NIKONENKO Oleksandr Semenovich,** (born September 9, 1941) Doctor of Medical Sciences, Professor, Honored Worker of Science and Technology of Ukraine. Main areas of scientific research: problems of cardiovascular surgery, organ transplantation. Since 1992, kidney transplantations have been performed in the clinic of hospital surgery of the Zaporizhia Medical Institute, in 1994 - the first liver transplantation was performed in Ukraine, in 1998 - kidney and pancreas transplantation for diabetes mellitus. Author of 189 scientific papers. Prepared 4 doctors and 18 candidates of sciences. By the decision of the Cabinet of Ministers in 1984, the Coordination Center for Organ, Tissue and Cell Transplantation was organized under the Ministry of Health of Ukraine. A group of specialists prepared a draft Law on the transplantation (transplantation) of organs and (or) tissues in Ukraine.

The founder of endocrine surgery in Ukraine is I.V. Komisarenko.

**KOMISARENKO** Igor Vasilyevich (1933-2013), Corresponding Member of the National Academy of Medical Sciences of Ukraine (1997), Doctor of Medical Sciences (1978), Professor (1981), Laureate of the State Prize of Ukraine (1976, 1988), Honored Worker of Science and Technology of Ukraine (1997), Founder and Head of the Surgical Department of the V.P. Komisarenko Institute of Endocrinology and Metabolism of the Academy of Medical Sciences of Ukraine (since 1965), Founder and Director of the Ukrainian Scientific and Practical Center of Endocrine Surgery, Organ and Tissue Transplantation (since 1995). Author of 240 scientific papers. I.V. Komisarenko's scientific research is devoted to the study of the relationship between the endocrine glands and the higher departments of the central nervous system, problems of surgical endocrinology and endocrine oncology, endovascular surgery, auto- and heterotransplantation in endocrinology. For the first time in the world, he developed combined methods of treating adrenal cortex tumors using steroidogenesis inhibitors, tumor embolization and metastases; methods of treating thyroid tumors, especially in children and people affected by radioactive contamination. Under his leadership, a school of endocrinological surgeons was created.

**Dmytro Fedorovych SKRIIPNYCHENKO,** (1921-1994), Laureate of the State Prize of Ukraine (1986), Doctor of Medical Sciences (1956), Professor (1957), Chief Surgeon of the Ministry of Health of Ukraine (1959). A prominent scientist-surgeon, teacher, specialist in thoracic, general and emergency surgery, vascular surgery, endocrinology and the organization of emergency surgical care. Author of more than 300 scientific works, including 15 monographs. Prepared 10 doctors and 40 candidates of sciences. The main scientific works of D.F. Skrypnychenko are: "Emergency abdominal surgery" (1986); "Surgical treatment of toxic goiter" (1976); "Malignant tumors of the thyroid gland" (1969); "Surgical treatment of myasthenia gravis" (1982).

**ZEMSKOV Volodymyr Sergiyovych** (09.09.1939-21.02.2002) - Doctor of Medical Sciences (1981), Professor (1983), Head of the Department of Surgery of the

Pediatric Faculty (1980-1982), General Surgery (since 1982), Head of the Kyiv Center for the Treatment of Liver, Biliary Tract and Pancreas Diseases (since 1982), President of the International Public Holding Company "Zemskov Clinic", Laureate of the USSR State Prize (1985), Honored Scientist of the Ukrainian SSR (1988), Academician of the G. Marconi World Academy of Rome (1995), Member of the International Society of Surgeons, Member of the International Association of Pathology of the Biliary Tract and Pancreas, Member of the European Union Charity Society and Medical Societies of Russia, Austria, Belgium, Laureate of the International Rating "Golden Fortune 2000". In 1962, V.S. Zemskov graduated from the Lugansk Medical Institute, and in 1980 he defended his doctoral dissertation on the topic "Surgical treatment of acute pancreatitis and its complications". Author of over 500 scientific papers, including 5 monographs, as well as new methods of diagnostics, conservative and surgical treatment, confirmed by 125 patent certificates. He introduced into practice various methods of detoxification (thermo-, plasma-, enterosorption), used an artificial liver. He created a school of surgeons: among his students there are 16 doctors and 42 candidates of medical sciences. He was awarded the "Order of Merit" (European Parliament), the Order of the Belgian-Spanish Crown (Belgium), and the Albert Schweitzer Medal.

**PAVLOVSKY Mykhailo Petrovich,** (1930-2013), academician of the National Academy of Medical Sciences of Ukraine (1997), laureate of the State Prize of Ukraine (1987), Honored Worker of Higher Education of Ukraine, Doctor of Medical Sciences (1970), Professor (1971). International recognition was received for his work on allotransplantation of pancreatic β-cells in diabetes mellitus. For the cycle of research "Development, theoretical substantiation and clinical implementation of new methods of surgical treatment, detoxification and rehabilitation of patients with diseases of the liver and bile ducts" M.P. Pavlovsky and a group of colleagues were awarded the State Prize of Ukraine in the field of science and technology for 1987. Author of more than 370 works, 9 monographs, 10 inventions. He trained 7 doctors and 20 candidates of sciences. Main areas of scientific research: surgical endocrinology, surgery for liver cirrhosis, surgical aspects of diabetes mellitus, surgical correction of genetically determined anomalies of the genital organs.

**MATYASHYN Hnat Mykhailovych** (11.11.1925-31.10.1979) - Doctor of Medical Sciences, Professor, Honored Scientist of the Ukrainian SSR (1979), Head of the Department of Faculty Surgery (1968-1979). Graduated from the Donetsk Medical Institute (1950). Author of over 250 scientific papers. Chief Surgeon of the Ministry of Health of the Ukrainian SSR (1970-1979), Chairman of the Republican (1973) and Honorary Member of the International Society of Surgeons (1976). For his work in reconstructive surgery, he was awarded the Jubilee Medal "100th Anniversary of N. A. Bogoraz" and the Medal "For Merit in the Development of Reconstructive Surgery". Participant of the Great Patriotic War. Awarded the Orders of the Red Banner of Labor, the Red Star and medals.

**BONDAR Hryhoriy Vasylevich** (1932-2014) - General Director of the Donetsk Regional Anti-Tumor Center, Doctor of Medical Sciences, Professor, Academician of the National Academy of Medical Sciences of Ukraine, Head of the Department of the Donetsk National Medical University named after M. Gorky, Hero of Ukraine. Born

on April 22, 1932. Since 1959 - Head of the Surgical Department of the Donetsk Regional Clinical Hospital named after. Kalinin. Since 1962 - Assistant Professor of the Department of General Surgery; since 1967 - Associate Professor of the Department of Surgical Diseases of the Faculty of Dentistry. In 1975, he created the Department of Oncology on the basis of the Donetsk National Medical University, which later became the basis of the Donetsk Regional Anti-Tumor Center. Deputy of the Donetsk City Council (since 2006). Under the leadership of Academician Bondar, 14 doctoral and 40 candidate theses were defended. Bondar G.V. - Author of over 800 scientific works.

**HUSAK Volodymyr Korniyovych** (1939-2002), Corresponding Member of the National Academy of Medical Sciences of Ukraine, Doctor of Medical Sciences, Professor. Since 1966 - Assistant Professor of the Department of Surgery of the Faculty of Dentistry of the Donetsk Medical Institute. Since 1973 - Associate Professor, since 1983 - Professor of the Department of General Surgery; since 1984 - Head of the Department of Hospital Surgery named after Vladimir Bogoslavsky. Since 1991 - General Director of the Donetsk Regional Medical and Clinical Association. In 1999 he created the Institute of Emergency and Reconstructive Surgery of the National Academy of Medical Sciences of Ukraine, was its first director. Author of over 400 scientific papers, 2 monographs, 20 inventions . He trained 3 doctors and 20 candidates of medical sciences . He was the first in Donbas to master open-heart surgery, the cardiac conduction system, and limb reimplantation surgery, created a modern burn center, and organized a scientific laboratory for growing skin cell cultures.

**ZOZULYA Yuriy Panasovich** (b. 1927) Academician of the National Academy of Medical Sciences of Ukraine (1994), Corresponding Member of the National Academy of Medical Sciences of Ukraine (1991), Full Member of the New York Academy of Sciences, Doctor of Medical Sciences (1966), Professor (1968), Laureate of the State Prize of Ukraine in Medicine. Vice-President of the Academy of Medical Sciences of Ukraine (since 1993), Director of the Institute of Neurosurgery of the Academy of Medical Sciences of Ukraine (since 1993), where he has been working since 1950. The main areas of scientific research are neuro-oncology, clinical pathophysiology of cerebral circulation, vascular neurosurgery, problems of neurotransplantation, development of methods of surgical interventions for various types of brain pathology. Author of 360 scientific works, including 9 monographs, 21 inventions. Under his supervision, 28 doctoral and candidate theses were completed. Scientists of the Institute of Neurosurgery are developing one of the new areas of neurosurgery - reconstructive surgery. The organizer of this area is Academician V.I. Tsymbalyuk.

**TSYMBALIUK Vitaliy Ivanovych** (b. 1947) - Honored Worker of Science and Technology of Ukraine (1997). Laureate of two State Prizes of Ukraine (1996, 2002), the Prize of the Academy of Sciences of Ukraine (2003) and the Yaroslav the Wise Prize National Academy of Sciences of Ukraine (1998). Laureate of 2012 Prize of the NAS of Ukraine named after O.O. Bogomolets for the cycle of works “Physiology and pathology of afferent brain systems : disclosure of mechanisms and development of new methods of medical correction”. Awarded the Orders “For Merit” II and III degrees, “St. Volodymyr” III degree, as well as 5 medals. President of the NAMS

(2016). Main areas of scientific activity: restorative neurosurgery, functional and stereotactic neurosurgery, neurosurgery of the peripheral nervous system, neurotransplantation, nosocomial infections, history of neurosurgery. Organizer and head of a new direction in neurosurgery - restorative neurosurgery. Developed a technology and first performed neurotransplantation in patients with organic lesions of the nervous system (infantile cerebral palsy, apalic syndrome, epilepsy, consequences of craniocerebral and spinal trauma, degenerative diseases, etc.). He introduced domestic electrostimulation systems to the clinic for the treatment of pain syndromes, spasticity, and epilepsy. He is the author of about 1,000 scientific works, including 28 books (monographs, textbooks, manuals, reference books), 46 copyright certificates and patents. He has trained 11 doctors and 51 candidates of medical sciences.

**BONDARENKO Viktor Oleksandrovych** (b. 10.18.1931) Academician of the National Academy of Medical Sciences of Ukraine (1993), Doctor of Medical Sciences (1968), Professor (1969), Honored Worker of Science and Technology of Ukraine (1991), Director of the Kharkiv Institute of Emergency Surgery, People's Deputy of Ukraine 1st convocation. One of the leading specialists in the field of emergency surgery. Scientific interests are mainly focused on the surgical treatment of inflammatory-destructive injuries, acute diseases of the respiratory and abdominal organs. He proposed methods for correcting functional disorders in surgical patients, developed effective methods for treating diseases of the lungs, stomach, intestines, and biliary tract. Author of over 250 scientific papers, including 11 monographs. He has prepared 4 doctors and 26 candidates of sciences.

The surgery of the future will be equipped with the latest and most advanced technology that will allow for quick and bloodless tissue joining. In this aspect, laparoscopic, endoscopic, endovascular, endolymphatic, X-ray surgery, laser surgery, cryosurgery, lithotripsy, microsurgery, and transplantology will be widely used. The development of gnotobiological tools that will ensure the maintenance of operated patients in a germ-free environment will be of great importance. All this will contribute to improving the results of surgical operations.

## ORGANIZATION OF SURGICAL CARE IN UKRAINE

The organization of medical care, including surgical care, for citizens of Ukraine is based on their right to free state medical care of all types, enshrined in Article 49 of the Constitution of the country. Surgical care is one of the most widespread forms of medical care. Its peculiarity is due to the wide spread of surgical diseases, congenital and acquired defects and injuries and the significant possibilities of surgery in the treatment of various diseases, correction of defects and replacement of functionally defective organs and tissues (organ transplantation and xenoprosthesis).

The basis of the system of organizing surgical care in Ukraine is still the system inherited from the former USSR. This care was organized according to the territorial principle and the stages of its provision to the population of the country with the lower stage being subordinated to the higher. The organization provides for the maximum proximity of medical care in general and surgical care in particular to the patient's place of residence, and is provided by the relevant territorial medical institutions. The organization of surgical care includes, in ascending order, primary medical care, qualified and specialized surgical care. Surgical care is divided into emergency, or

urgent, which is needed by patients with acute diseases and injuries, and planned, which is provided to patients with chronic ailments.

Primary emergency medical care for patients with acute surgical diseases and injuries is provided in outpatient clinics of paramedical and obstetric posts and in district rural hospitals - in villages, and in cities and in settlements equated to them - by surgeons of polyclinics, trauma center doctors and ambulance stations teams. For patients with minor acute injuries that do not require surgical interventions or can be performed by doctors of these stages, and for patients with acute diseases that do not require hospitalization, the primary care provided at these stages is actually qualified and ends there. The essence of primary medical care for surgical patients with acute illnesses and injuries in rural outpatient clinics and district hospitals, as well as care in city polyclinics, which by its nature exceeds the scope of care programmed for the surgeon in the polyclinic, consists in examining the patient with available means to establish a preliminary diagnosis and referring the patient to the surgical department of the district or central district hospital with the determination of the transport by which the patient should be transported.

In most cases, he is transported by an ambulance station car, or by an ambulance of the district hospital itself, to which the patient is sent. Less often, medical aviation may be invited if the patient's life is in mortal danger. Patients from surgical offices of city polyclinics are sent to the corresponding surgical departments of district or city hospitals, or through the ambulance station (by calling from the last car) - in case of acute diseases of internal organs, or - by city or own transport in case of mild acute diseases and injuries.

In polyclinics of cities and settlements, which have surgical offices and departments, patients with minor superficial injuries and uncomplicated acute diseases (small wounds of soft tissues of the body, limited burns, boils, abscesses, subcutaneous panaritium, etc.) are provided with qualified surgical care.

Qualified emergency and planned surgical care for patients with the most common acute diseases of the abdominal cavity (acute appendicitis, strangulated hernia, acute cholecystitis, perforated gastric and duodenal ulcer, gastric bleeding, pancreatitis, acute intestinal obstruction, etc.) and with injuries of the abdominal organs, soft tissue injuries, purulent processes, as well as patients with chronic diseases of the abdominal cavity and some other organs is provided in general surgical departments of central district hospitals, city and regional hospitals, where there are surgical departments and appropriate conditions for such interventions (highly qualified surgeon, means for accurate laboratory and instrumental diagnostics and anesthesia support). In large cities, in particular regional centers, in addition to district and city hospitals that provide qualified surgical care to city patients, the latter is also provided by regional hospitals.

The development of modern surgical technologies (laparoscopic, endoscopic, endovascular) has led to the need for differentiation, division of surgery into separate disciplines and sections. Thus, specialized surgical care was initiated from separate sections of surgery and specialized surgical institutions emerged, providing patients with specialized surgical care.

Thus, traumatology and orthopedics, oncology, urology, neurosurgery have long been separated from surgery into independent disciplines. Even earlier, ophthalmology, otorhinolaryngology, and dentistry became independent branches. In the post-war period, surgery underwent further differentiation. Lung and bronchial surgery, esophageal surgery, cardiac surgery, vascular surgery, rectal surgery (proctology), surgical gastroenterology, and surgical endocrinology were separated into separate disciplines. The process of differentiation of surgery into separate sections continues even now. For example, herniological and other clinical departments already exist. In almost all regional and large city hospitals, there are surgical departments from all major sections of surgery (thoracic, neurosurgical, surgical gastroenterology, maxillofacial, otorhinolaryngological, ophthalmological, burn, vascular, or even cardiovascular departments), which provide specialized surgical care to patients from the relevant territories.

A large role in providing qualified and specialized care to the population is played by surgical clinics of medical universities and academies of Ukraine, which operate on the bases of general surgical departments of city and regional hospitals of university cities. Highly qualified staff of teaching surgeons (professors, associate professors, assistants) provide a large amount of specialized care for various diseases. Many of these clinics are city centers of specialized surgical care (liver, biliary tract and pancreas surgery; stomach and intestinal surgery; surgical treatment of bleeding; lung surgery; endocrine surgery, rectal and colon surgery, esophageal surgery, etc.).

Specialized assistance to citizens of Ukraine, regardless of their place of residence, is provided by research institutes of a surgical profile, which are scientific, methodological and organizational centers for the development and implementation of the latest diagnostic, treatment and prevention tools for diseases and injuries of certain systems or organs. Among them are the National Institute of Surgery and Transplantology named after O. O. Shalimov of the Academy of Medical Sciences of Ukraine, the Kharkiv Research Institute of General and Emergency Surgery of the Ministry of Health of Ukraine, the Kyiv Research Institute of Neurosurgery named after A. P. Romodanov of the Academy of Medical Sciences of Ukraine, the Kyiv Research Institute of Cardiac Surgery of the Academy of Medical Sciences of Ukraine, the Kyiv Research Institute of Otorhinolaryngology, the Kyiv Research Institute of Endocrinology and Metabolism of the Academy of Medical Sciences of Ukraine, the Odessa Institute of Ophthalmology named after V. P. Filatov, the Dnipropetrovsk Research Institute of Gastroenterology, the Kyiv Research Institute of Hematology and Blood Transfusion, the Kyiv Research Institute of Tuberculosis and Pulmonary Surgery, etc. Along with the institutes, there are a number of specialized republican centers in certain areas of surgery (burn, proctology centers, thyroid surgery center, etc.).

In the last two decades, specialized hospitals for emergency surgical care have been established in Ukraine in megacities and large industrial centers (Kyiv, Kharkiv, Donetsk, Dnipropetrovsk, Zaporizhia, Odessa, Lviv, Kryvyi Rih, Kherson, etc.), which include departments of the main areas of surgery (neurosurgical, vascular, gastroenterological, polytrauma department, etc.), the activities of which provide

effective specialized care for patients with various acute diseases and conditions (gastric bleeding, perforated ulcer, acute pancreatitis).

Specialized traumatology (bone and joint injuries) and orthopedic care is provided by trauma centers in cities, as well as relevant departments of central district, city and regional hospitals, such scientific and methodological centers as the research institutes of traumatology and orthopedics (Kyiv, Kharkiv, Donetsk).

Oncological care for patients is provided by oncological city and regional dispensaries and cancer centers, together with oncology departments of medical universities and academies located on the bases of these dispensaries, as well as scientific and research institutes of oncology, in particular the Ukrainian National Cancer Center with its numerous multi-profile clinics (thoracic, gastroenterological, gynecological, proctological, maxillofacial, breast tumor clinic, etc.).

A huge role in providing emergency surgical care is played by city, regional, and republican "Ambulance" stations and specialized teams created within them in various fields of emergency surgery (shock, thromboembolic, burn, etc.). Thanks to this, medical care is provided to victims quickly and professionally from the scene of the injury or illness.

Thanks to the development of new technologies, the appearance of television and video equipment and computers, in particular laparoscopic devices, in recent years new, non-traditional forms of surgical interventions have appeared, which are performed endoscopically (laparoscopic removal of some abdominal organs in case of their chronic lesions - gallbladder, appendix, fallopian tubes, segments of intestines, etc., plastic closure of abdominal hernias, etc.) or endovascularly (balloon and laser dilation of the opening of narrowed coronary arteries, etc.). Such types of specialized surgical care are provided in specialized medical institutions.

In Ukraine, new forms of financing medical care are being developed and implemented, which also applies to the provision of surgical care to citizens of Ukraine, when, along with budgetary medical care, an important place belongs to insurance medicine, social security funds, and the population's own funds.

**Organization of outpatient care for surgical patients**

Preservation, restoration and strengthening of the health of the population is the most important task of the health care system. The organization, content and quality of surgical care in outpatient settings represent one of the important sections of the daily activities of the medical staff of outpatient and polyclinic institutions. The basis of this work is:

- knowledge of the peculiarities of the work activities of the population of the service area, everyday life, and specific pathologies of the region that adversely affect health;
- analysis of the results of medical monitoring of the health of the population and the effectiveness of preventive measures carried out in the region;
- results of statistical analysis of injuries and illnesses, their causes and the nature of the associated loss of working capacity.

The work of a polyclinic surgeon consists of the following elements:

- organization and provision of surgical care to the population;

- treatment of surgical patients in outpatient clinics and day hospitals;
- organization of treatment of the population at home;
- organizing the evacuation of patients to appropriate surgical hospitals and other medical institutions;
- observation of patients in dispensary groups and persons who have undergone surgical interventions and injuries;
- medical monitoring of the health of the population, organization of medical and labor examinations and medical check-ups;
- development and implementation of measures to reduce surgical morbidity and trauma in the region.

To provide surgical care, the clinic deploys:

- a clean dressing room, which also serves as a clean operating room;
- purulent dressing, where purulent operations are also performed;
- procedure room equipped for emergency surgical care;
- jobs for an ophthalmologist and an otolaryngologist.

A special place in the surgical work of a polyclinic doctor is occupied by the organization and content of emergency medical care for surgical diseases and injuries. The organization of such care is ensured by a clear division of responsibilities of medical workers at all its stages - from the site of the disease (injury) to the stage of qualified or specialized medical care inclusive. In general, the scheme of organizing emergency care in a polyclinic is presented in the table.

In this system, a special place is occupied by the organization, content and quality of medical care in the polyclinic, for which the doctor bears personal responsibility. Medical equipment must be in constant readiness for use to perform:

- temporary or permanent stop of external bleeding;
- immobilization and pain relief of fractures;
- elimination of asphyxia using tracheostomy, intubation, tongue fixation;
- applying a sealing bandage in case of open pneumothorax;
- puncture and drainage of the pleural cavity for tension pneumothorax, hemothorax;
- indirect heart massage;
- removal of foreign bodies from the conjunctiva of the eye;
- intravenous administration of medications;
- primary surgical treatment of wounds;
- suturing wounds of the skin and subcutaneous tissue;
- applying aseptic dressings;
- tamponade of the nasal passages during bleeding.

**Principles of organizing emergency care in a polyclinic**

| Place of assistance | Volume of diagnostic tests | Volume of emergency medical care | Who provides | Equipment |
|---|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| At the site of injury, illness | Examination of the patient, monitoring of pulse, blood pressure, breathing, body temperature, studying the circumstances of the injury (disease) | Temporary stop of external bleeding, restoration of upper airway patency, artificial respiration, indirect heart massage, application of bandages, splints, administration of medications | Doctor, paramedic | Medical first aid kit, special sets of medical equipment and medicines, splints, tourniquets, bandages, etc. |
| Transportation by ambulance | Monitoring the patient's general condition, pulse, blood pressure, breathing | The same | _ | Medical first aid kit, medical containers, tire set, resuscitation equipment |
| Clinic | Medical examination of the patient, laboratory, instrumental diagnostics | Stopping bleeding, mechanical ventilation, indirect heart massage, tracheal intubation, analgesia, intravenous infusions and injections of medicinal substances, novocaine blockades, transport immobilization, tracheotomy, surgical wound treatment. Bladder catheterization, suprapubic puncture, pleural puncture, insertion of drains for tension pneumothorax | Doctor | Respiratory equipment, aspirator, set of splints, medical dressings, anti-shock solutions for transfusions, equipment for the procedure room and clean dressing room |

The second extremely responsible section of surgical care is the organization of outpatient admission and treatment in outpatient settings. The organization of outpatient admission should allow the allocation of the following groups of patients.

- requiring emergency care;
- requiring inpatient treatment in a day hospital or hospital;
- requiring additional outpatient and inpatient examination;
- who continue treatment in the outpatient clinic.

The scope of surgical care in the outpatient clinic should include:

- sanitation of superficial phlegmons and abscesses;
- removal of skin sutures and changing dressings during outpatient wound treatment;
- suturing of small skin wounds in the absence of contraindications to their use;
- removal of superficial soft tissue tumors in the absence of indications for their urgent histological examination.

The day hospital examines and treats patients who require hospitalization and constant medical supervision for up to 10 days. These usually include patients with:

- uncomplicated cutaneous, subcutaneous panaritium and paronychia;
- phlegmons and abscesses of the subcutaneous tissue;
- infected superficial soft tissue wounds and abrasions;
- boils;
- local forms of streptoderma and pyoderma;
- burns and frostbite of the 1st - 2nd century.

The day hospital accommodates patients who require referral to a hospital after providing them with primary medical care and patients who cannot be transported until their condition improves, allowing for safe evacuation. Examination and treatment of patients in the polyclinic are carried out by the polyclinic's doctors.

Home treatment can be carried out for limited and uncomplicated purulent diseases of the skin and subcutaneous tissue (except for boils of the face and scalp, carbuncles regardless of the localization and severity of the clinical course). At home, treatment can be continued for patients discharged from medical institutions who require rehabilitation measures. Home treatment requires extensive and systematic clinical training from the doctor, since this contingent may include people with a variety of diseases.

If during the treatment at home the patient's condition worsens and hospitalization becomes necessary, the polyclinic doctor refers the patient to a hospital.

To provide this amount of medical care, the clinic doctor must:

- know and be able to recognize the symptoms of acute surgical diseases and injuries to internal organs;
- by venipuncture, and if impossible - venesection, to perform intravenous drip and jet infusions of medications, transfusion media, including blood products;
- perform novocaine infiltration anesthesia and novocaine blockades of fractures and the spermatic cord;
- perform primary surgical treatment of superficial soft tissue wounds;
- carry out transport immobilization;
- stop external bleeding by tamponade, ligation of vessels in the wound, application of a tourniquet to the bleeding vessel, application of a tourniquet;
- perform catheterization and bladder puncture;
- perform a puncture of the pleural cavity with the introduction of drainage with a valve in case of pneumothorax;
- apply an occlusive dressing for a penetrating chest wound;
- perform artificial respiration in various ways;
- perform indirect heart massage;
- provide first aid for burns, frostbite, electrical injuries, and treat limited burns and frostbite of the 1st - 2nd degree in the clinic;
- perform operations on subcutaneous, subungual and periungual panaritiums, superficial abscesses, ingrown nails, remove some benign formations of subcutaneous tissue (atheroma, lipoma, etc.);

- to provide treatment in the clinic for victims with superficial injuries (abrasions, subcutaneous hematomas), bruises, and uncomplicated injuries to the ligamentous apparatus of the extremities.

Wide access to medical documentation creates certain deontological difficulties in ensuring medical confidentiality. Therefore, all information about the patient's health should be communicated to the patient only by the attending physician. It is prohibited for the average medical staff to provide such information.

Referral of patients for inpatient treatment to other institutions may be for urgent and routine indications, but in any case, the polyclinic doctor is obliged to:

- determine the preliminary diagnosis of the disease and indications for hospitalization;
- assess the patient's transportability and take necessary measures to ensure safety during transportation;
- determine the type of transport by which the patient can be evacuated, and the patient's position along the route;
- prepare all documentation that must be issued at the clinic when referring for inpatient treatment to another medical institution;

Patients and victims with the following symptoms and conditions are subject to urgent hospitalization in a surgical hospital:

- external and internal bleeding;
- acute injury to major blood vessels or nerve trunks;
- penetrating wounds;
- bone fractures of any location;
- concussions and brain injuries;
- shock;
- pneumothorax, hemothorax of any origin;
- damage to the genitourinary organs;
- acute diseases or injuries of the abdominal cavity and retroperitoneal space, even in cases of suspicion;
- complicated superficial and deep panaritiums;
- deep abscesses and phlegmons, carbuncles of any location, head boils;
- acute osteomyelitis and arthritis;
- foreign bodies of any organs;
- malignant neoplasms of any localization;
- dysphagia of any origin;
- hematuria, pyuria, acute orchitis, epididymitis, prostatitis;
- burns and frostbite of the 2nd-3rd degree.

The organization of donation requires great attention and care, which has not only medical but also great educational significance. The medical staff of the polyclinic carries out all the preparatory work. Blood sampling is carried out only in specialized departments.

A very important section of surgical work is the dispensary observation of patients who have undergone surgical interventions, the detection during medical examinations

of diseases that are subject to surgical treatment (hernias, neoplasms, etc.). The polyclinic doctor performs significant expert work as a member of the LCC, LTEK. This activity requires not only appropriate clinical training, but also knowledge of certain legal documents.

**ETHICS** since the time of Aristotle, the science whose subject is the determination of the ethical value of human aspirations and actions is called. According to this, actions that have a positive ethical value are called ethical (ethical, worthy, positive), and actions devoid of it are called unethical (immoral, unworthy). Ethics includes a set of norms of behavior and morality, regulates the sense of professional duty, honor, conscience and dignity of a doctor. One who has received the title of doctor voluntarily takes on the obligation to flawlessly fulfill these norms throughout his life.

The increasing technocratization of medicine and the struggle for its economic efficiency are making the issues of biological ethics and deontology more relevant. At the same time, the behavior of a doctor at the patient's bedside, the manner of communicating with patients, and the interaction with relatives of patients, colleagues, and junior medical staff are becoming extremely important.

It should be understood that the success of treatment directly depends on the doctor's ability to find not only the necessary professional solutions, but also to establish an individual approach to the personal problems of each patient, to each of his fears, to each hope. All this becomes especially relevant in the conditions of general accessibility of reference medical literature on the Internet, which often leads to the formation of the patient's own understanding of the course of the disease, his tendency to self-medicate, increased interest in "alternative" methods of treatment. In such conditions, such concepts as mercy, the doctor's ability to empathize, establishing emotional contact with the patient should be considered especially important and are the most important component of successful treatment of the patient. It should be remembered that a smile, an open, friendly look, an encouraging tone have a positive effect on the process of treatment and recovery. On the contrary, doubts on the face, gestures of uncertainty, anxiety in the doctor's gaze can in themselves provoke a state of stress and fear in the patient.

Thus.

- Ethics is a system of moral principles that determine what is right or wrong in professional activities. Medical ethics concerns the moral obligations of doctors to patients, colleagues, society and themselves. It is based on four basic principles:
- The principle of beneficence - the desire to benefit the patient.
- The principle of do no harm - avoiding actions that may worsen the patient's condition.
- The principle of autonomy - respect for the patient's right to make decisions about their health.
- The principle of justice - equal access to medical care for all patients.

Deontology is a section of ethics that studies the professional duties of a doctor. In a medical context, it determines how a doctor should behave with patients,

colleagues, other medical professionals. The main goal of deontology is to build trust between the doctor and the patient and ensure a high level of professional responsibility.

The main principles of medical deontology:

- Confidentiality: the doctor is obliged to maintain medical confidentiality.
- Respect: adherence to ethical norms in communicating with patients, even in difficult situations.
- Professionalism: a doctor's actions must meet the standards of medical practice.
- Empathy: the ability to understand the emotional state of the patient and support him morally.

Ethics and deontology are the basis of a doctor's professional activities. They help create a safe and comfortable environment for patients, maintain high quality medical care, and maintain the reputation of the medical profession at the appropriate level.

**Hippocratic Oath**

I swear by Apollo, Asclepius, Hygiene and Panacea, and all the gods and goddesses, taking them as witnesses, to fulfill honestly, according to my strength and understanding, the following oath and written commitment: to respect the person who taught me the art of medicine, on a par with my parents and, if necessary, to help her in everything; to consider her descendants as my brothers, and this art, if they wish to study it, to teach them gratuitously and without any contract; to transmit instructions, oral lessons and everything else in science to my sons, the sons of my teacher and students, bound by obligation and oath according to medical law, and to no one else. I will direct the regime of the sick for their benefit, according to my strength and understanding, and I will refrain from causing any harm or injustice. I will not give a deadly remedy to anyone who asks me for it, nor will I show the way to such a plan; nor will I give any woman an abortive pessary. I will lead my life and practice my art in a pure and blameless manner. In no case will I perform an operation on patients with stone disease, leaving it to people who are engaged in this business. In whatever house I enter, I will enter for the benefit of the patient, far from everything malicious, unjust and harmful, especially from love affairs with women and men, free and slave.

Whatever I may see or hear during the treatment - and also without the treatment - about the life of a person, which should never be disclosed, I will keep silent, considering such things a secret. To me, who unwaveringly fulfills the oath, may there be happiness in life and in art and glory among all people for eternity; to him who breaks or gives an insincere oath, may the opposite befall him.

In 1948, the WHO adopted the "Geneva Oath" of the doctor, which is based on the Hippocratic Oath. Ukrainian doctors have adopted the best principles of medical ethics, and students also make a solemn promise upon graduation when receiving their diploma.

On June 15, 1992, the President of Ukraine issued a Decree on the Oath of a Doctor of Ukraine

**Oath of a doctor of Ukraine**

Having acquired the profession and realizing the importance of the duties entrusted to me, in the presence of my teachers and colleagues, I solemnly swear: to

devote all my knowledge, strength and skills to the cause of protecting and improving human health, treating and preventing diseases, to provide medical care to all who need it, to be invariably guided in my actions and thoughts by the principles of universal morality, to be selfless and sensitive to patients, to admit my mistakes, to worthily continue the noble traditions of world medicine; to maintain medical secrecy, not to use it to the detriment of a person, to observe the rules of professional ethics, not to hide the truth if this does not harm the patient; to constantly deepen and improve my knowledge and skills, if necessary, to seek help from colleagues and never refuse them myself, to be truthful with colleagues; To contribute by one's own example to the upbringing of a physically and morally healthy generation, to affirm the ideals of mercy, love, harmony and mutual respect between people.

I swear to be faithful to this oath throughout my life.

A component of medical ethics is deontology - the doctrine of the duty of a medical worker (from the Greek. "deon" - duty, "logos" - study, science), the principles of medical behavior, which should be aimed at creating the best conditions for the speedy recovery of the patient. Medical deontology regulates the activities of a doctor, paramedic, midwife, nurse, medical students, taking into account the interests of the patient or victim and has ethical and moral principles, elements of legal norms, the relationship between a doctor and a patient, the concept of medical confidentiality, and issues of collegiality. The task of surgical deontology is to stabilize and preserve the patient's psyche in the preoperative and postoperative periods. The surgeon, together with the average medical workers, must instill in the patient hope for recovery, show compassion and high moral qualities, highly professional performance of medical and diagnostic measures, and elimination of negative psycho-emotional factors. All principles of medical ethics and deontology must be observed by students - future nurses, midwives, paramedics, doctors. After all, while in a medical institution during classes, they directly communicate with medical workers, patients, performing some manipulations, procedures. Therefore, students must be very careful when communicating both with each other and with patients, so as not to accidentally violate medical confidentiality, not to disturb the psychological balance of the patient, since the work of a significant number of medical workers can be nullified.

**Basic literature:**


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**Tests**

1. What kind of fundamental clinical prerequisites were required for development of surgery in 19-th century?
   A. Knowledge of human anatomy
   B. Knowledge of biochemistry
   C. Knowledge in anaesthesiology

D. Knowledge of mathematics

2. The author of "Dix Livres de la Chirurgie avec le Magasin des Instruments Necessaires à Icelle" was:
   A. John Hunter
   B. Andreas Vesalius
   C. Theodor Kocher
   D. Ambroise Paré
3. Very important event in the evolution of surgical history in 19-th century was:
   A. Methods of controlling haemorrhage
   B. Working out of techniques of surgical operations
   C. Antisepsis, asepsis and understanding the nature of infection
   D. Successes in oncology
4. Who introduced methods of aseptic and antiseptic in Russia?
   A. N. Pirogov
   B. Lister
   C. T. Kocher
   D. T. Billroth
5. Who among the outstanding surgeons of 19th century introduced fundamental science achievements in practical activities of doctors of surgeons?
   A. F. Trendelenburg
   B. W. Halsted
   C. J. Murphy
   D. M. Sklyfosovskyi
6. In 1900, which surgeon presented his results on partial gastrectomy before the American Surgical Association?
   A. John Finney (1863-1942)
   B. William Mayo (1861-1939)
   C. Fedor Krause (1856-1937)
   D. William Miles (1869-1947)
7. What prominent late 19th century discovery conducted by Pulyui Ivan and Wilhelm Roentgen had an enormous impact on the evolution of surgery?
   A. X-rays
   B. Ultrasonography
   C. Magnetic Resonance Imaging
   D. Computer Tomography
8. Which surgeon is prominent for setting the scientific tone of surgery as a legitimate scientific endeavor in surgical history?
   A. Jules Peán (1830-1898)
   B. William Stewart Halsted (1852-1922)
   C. Marin-Theodore Tuffiér (1857-1929)
   D. Eduardo Bassini (1844-1924)
9. Which important Ukrainian surgeon contributed greatly to the development of thoracic and cardiovascular surgery?
   A. N. Pirogov
   B. A. Shalimov

C. I. Pavlov
D. N. Amosov

10. Which two surgeons first described the eponymic polyendocrine adenomatosis in 1955?
A. Willis Potts (1895-1968) and Charles Hufnagel (1916-1989)
B. Francis D. Moore (1913-2001) and Jonathan E. Rhoads (1907-2002)
C. James D. Hardy (1918-2003) and Rudolph Nissen (1896-1981)
D. Robert Zollinger (1903-1994) and Edwin Ellison (1918-1970)

**Keys for initial tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| A,C | D | C | A | B | B | A | B | D | D |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Inflammation<br>– Infection<br>– Pathophysiology of basic surgical diseases. | -To represent the methods of antiseptic |
| Study:<br>– The prominent discoveries in surgery.<br>– Development of the methods of diagnosis of surgical diseases.<br>– Ethics in surgery | -Last achievements in field of pathogenesis of disease |

## 1.2. New technologies in surgery. Modern methods of diagnosis and treatment.

Medical technology is today a feature of hospitals, clinics and GP surgeries. The practice of medicine has been revolutionised by computers, digitisation, new materials and good old-fashioned laboratory research. Yet this has been accompanied by increased patient anxiety about the risks and consequences of medical intervention.

The introduction of new medical technology has not been so rapid in developing countries, but the sense of ambivalence is the same – technology represents both progress and threat. In countries with multiple healing systems patients can choose whom to consult, depending on their illness. In these countries a technological approach may be chosen as a first or last resort.

**Educational aims:**

- To be acquainted with new technologies in surgery.
- To determine the great importance of microsurgical, endoscopic, endovascular, laser operations in surgery, welding of tissues, using of prostheses and implants.
- To determine the diagnostic significance of ultrasonic, endoscopic and others new diagnostic procedures.

- To determine the main advantages of new technologies in surgery.
- To estimate efficiency of new technologies in surgery.

**A student must know:**

- Foundations of microsurgery.
- Foundations of endoscopic interventions.
- Foundations of laser surgery.
- Foundations of ultrasonic procedures.
- Foundations of using of prostheses and implants
- General information about welding of tissues. Principles of using

**A student must be able to:**

- Correctly interpret the results of endoscopic, ultrasonic examinations and others new diagnostic procedures.
- Define indications for microsurgical, endoscopic, endovascular, laser operations.
- Define contra-indications for microsurgical, endoscopic, endovascular, laser operations.
- Define the rational volume of laboratory and instrumental methods of research for microsurgical, endoscopic, endovascular, laser operations.
- Perform pre-operative preparation of patients.
- Conduct post-operative care.

**Terminology.**

| Term | Definition |
|---|---|
| Endoscopic ultrasound (EUS) | is a medical procedure in endoscopy is combined with ultrasound to obtain images of the internal organs in the chest and abdomen. |
| Tomography | is imaging by sections or sectioning, through the use of any kind of penetrating wave. |
| Magnetic resonance imaging (MRI) | is a medical imaging technique used in radiology to visualize detailed internal structures that uses of nuclear magnetic resonance for imaging |
| Laparoscopic surgery | is a modern surgical technique in which operations in the abdomen are performed through small incisions (up to 1.5 cm) as compared to the larger incisions needed in laparotomy. |
| Microsurgery | is a general term for surgery requiring an operating microscope. |
| Replantation | is the reattachment of a completely detached body part. |
| Endovascular surgery | is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels. |
| Welding | is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues |

| | |
|---|---|
| | and to stop or prevent bleeding when tissue and blood vessels are incised. |

**Content:**

**Endoscopic ultrasound (EUS)** or echo-endoscopy is a medical procedure in endoscopy (insertion of a probe into a hollow organ) is combined with ultrasound to obtain images of the internal organs in the chest and abdomen. It can be used to visualize the wall of these organs, or to look at adjacent structures. Combined with Doppler imaging, nearby blood vessels can also be evaluated.

Endoscopic ultrasonography is most commonly used in the upper digestive tract and in the respiratory system. The procedure is performed by gastroenterologists or pulmonologists who have had extensive advanced training. For the patient, the procedure feels almost identical to the endoscopic procedure without the ultrasound part, unless ultrasound-guided biopsy of deeper structures is performed.

For endoscopic ultrasound of the upper digestive tract, a probe is inserted into the oesophagus, stomach and duodenum during a procedure called esophagogastroduodenoscopy. Among other uses, it allows for screening for pancreatic cancer, oesophageal cancer, and gastric cancer as well as benign tumours of the upper gastrointestinal tract. It also allows for characterization and biopsy of any focal lesions found in the upper gastrointestinal tract. This is done by inserting a needle through the stomach lining into the target.

Endoscopic ultrasound is performed with the patient sedated. The endoscope is passed through the mouth and advanced to through the oesophagus to the suspicious area. From various positions between the oesophagus and duodenum organs within and outside the gastrointestinal tract can be imaged to see if they are abnormal and they can be biopsied by a process called fine needle aspiration. Organs such as the liver, pancreas and adrenal glands are easily biopsied as are any abnormal lymph nodes. In addition, the gastrointestinal wall itself can be imaged to see if it is abnormally thick suggesting inflammation or malignancy.

The technique is highly sensitive for detection of Pancreatic Cancer (90-95% sensitivity) particularly in patients who are suspected to have a mass or present with jaundice. Its role in staging patients with pancreatic cancer is limited to local metastases; however, in combination with CT scan which provides information on regional metastases, it provides an excellent imaging modality for diagnosis and staging of pancreatic carcinoma.

Endoscopic ultrasound can also be used in conjunction with endoscopic retrograde cholangiopancreatography (ERCP). The ultrasound probe is used to locate gall stones which may have migrated into the common bile duct. This occurrence may cause obstruction of the drain shared by the liver and pancreas which may lead to lower back pain, jaundice and pancreatitis.

Echo-endoscopy can also be used for imaging of the rectum and colon, although these applications are lesser known. It is used primarily to stage newly diagnosed rectal or anal cancer. EUS guided fine needle aspiration may be used to sample lymph nodes during this procedure. Evaluation of the integrity of the anal sphincters may also be done during lower EUS procedures.

***Respiratory tract***

An endoscopic ultrasound probe placed in the oesophagus can also be used to visualize lymph nodes in the chest surrounding the airways (bronchi), which is important for the staging of lung cancer. Ultrasound can also be performed with an endoscopic probe inside the bronchi themselves, a technique known as endobronchial ultrasound.

The quality of the image produced is directly proportional to the frequency used. Therefore a high frequency produces a better image. However, high frequency ultrasound does not penetrate as well as lower frequency ultrasound so that the examination of the nearby organs may be more difficult.

**Endovascular surgery** is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels.

Endovascular techniques were originally designed for diagnostic purposes. Basic techniques involve the introduction of a catheter percutaneously or through the skin, into a large blood vessel. Typically, the blood vessel chosen is the femoral artery or vein found near the groin. Access to the femoral artery for example, is required for coronary, carotid, and cerebral angiographic procedures. The catheter is injected with a radio-opaque dye that can be seen on live X-ray or fluoroscopy. As the dye courses through the blood vessels, characteristic images are seen by experienced viewers and can assist in the diagnosis of diseases such as atherosclerosis, vascular trauma, or aneurysms.

In recent years, however, the development of intravascular balloons, stents and coils have allowed for new therapies as alternatives to traditional surgeries such as Coronary artery bypass surgery (CABG), carotid endarterectomy and aneurysm clipping. Stents and coils are composed of fine wire materials such as platinum that can be inserted through a thin catheter and expanded into a predetermined shape once they are guided into place.

Endovascular surgery is performed by radiologists, neurologists, neurosurgeons, cardiologists, and vascular surgeons. The field is rapidly growing as its minimally invasive techniques offer an immediate advantage over more traditional, yet highly invasive surgeries. However, the science of endovascular surgery and its developing techniques are so new that it is currently difficult to compare the long-term outcomes and complications of these patients. Several trials are underway, including Carotid Revascularization Endarterectomy versus Stent Trial (CREST), and International Subarachnoid Aneurysm Trial (ISAT), among others.

**Diagnostic catheterization of the heart cavities** is a minimally invasive procedure in which catheters are inserted into the vessels of the arm or leg and advanced into the heart cavities. Then, a contrast agent is injected into the catheters and an X-ray video of the chambers, valves and vessels of the heart is taken. Using catheters, the pressure and oxygen content are measured in different parts of the heart.

A few days before the procedure, the cardiologist prescribes the necessary tests (complete blood count, blood type, chest X-ray, ultrasound of the heart, ECG). On the evening before the procedure and before the procedure, all patients are prescribed sedatives (tranquilizers). For children, the procedure is performed only under anesthesia.

During the procedure, heart rate, blood pressure, and blood oxygen saturation are constantly monitored. Recovery after the procedure is very fast. Most patients after catheterization of the heart cavities require restriction of limb mobility. Patients can get up and walk the next day.

The concept of X-ray endovascular surgery. This direction of minimally invasive surgery is developing mainly in two directions - endovascular surgery and percutaneous introduction of various instruments (drainages, catheters, endoprostheses, balloon dilators, etc.) into the lumen of hollow, parenchymal organs, the abdominal cavity or excretory ducts (most often the biliary tract of the liver).

Surgical interventions performed using interventional radiology can be divided into the following groups:

- restoration of the lumen of narrowed tubular structures (arteries, biliary tract, various parts of the gastrointestinal tract);
- drainage of cavity formations in internal organs;
- occlusion of the lumen of vessels.

Endovascular surgery as a branch of modern surgery began to develop in the 60s of the XX century. The first operation for skin angioplasty of limited atherosclerotic stenosis of the popliteal artery under angiographic control was first performed by S. Dotter and M. Judkins in 1964. The expansion of the affected artery was performed using coaxial Teflon catheters. However, the operation became most widespread after the invention of A. Gruntzig in 1974 of a double-lumen balloon catheter, which allows for precise expansion of narrowed areas of affected arteries under the control of angiographic research. The essence of the method of balloon dilation (balloon angioplasty) of arterial vessel stenosis is as follows. Initially, the possibility and feasibility of using balloon angioplasty is assessed using angiographic research of the vascular bed. Usually, the operation is performed with limited narrowing of the artery, 1-3 cm in length, and good patency of the arteries distal to the stenosis site. A double-lumen balloon catheter is introduced into the stenosis zone in an uninflated state (its outer diameter is 1.5-2.3 mm). After that, using special syringes under the control of manometry, liquid is pumped into the lumen of the balloon under a pressure of 10-15 atm. At the same time, uniform pressure is exerted on the walls of the narrowed vessel along the entire circumference. In most patients, significant expansion and even complete restoration of the normal lumen of the affected artery are achieved. In case of restenosis, repeated use of balloon angioplasty is possible in the long term after the operation. The advantages of this minimally invasive operation are atraumaticity, rare postoperative complications, the absence of a number of local and general complications inherent in "open" interventions, and a short stay of the patient in the hospital. Nowadays, to improve the long-term results of balloon angioplasty, especially in patients with strictures in the draft, this procedure is supplemented with endoprosthesis (stenting) of the affected part of the vascular bed. A metal stent in a collapsed state is inserted into the affected vessel using a metal guide through a puncture in the upper part of the femoral artery under angiographic control. Then the metal openwork stent is opened in the lumen of the artery, thereby expanding its lumen to the required diameter. There are two types of stents: self-expanding after removal of

the metal guide and stents that are straightened in the lumen of the vessel using an endovascular balloon.

Most often, balloon angioplasty in combination with endoprosthesis or without it is used to treat coronary artery disease.

In many patients, angioplasty is an alternative to coronary artery bypass grafting. It is possible to use dilatation and stenting of several coronary arteries simultaneously. In acute myocardial infarction, the method is superior in its effectiveness to the results of thrombolytic therapy. Balloon dilatation and stenting of the coronary arteries, performed according to strict indications, allow you to avoid serious complications and operations associated with wide thoracotomy or sternotomy, the use of an artificial blood circulation apparatus. At the same time, the duration of the patient's stay in the hospital and the time of physical and social rehabilitation are significantly reduced.

Mitral heart defect. Minimally invasive intervention is used for mild valvular and subvalvular calcification, the absence of critical mitral valve stenosis and signs of its combined insufficiency. Balloon dilation of the narrowed mitral valve is performed under the control of X-ray television and angiographic examination. This method is not inferior in its effectiveness to the commissurotomy operation.

Balloon valvuloplasty is a medical procedure aimed at dilating a valve whose leaflets do not open sufficiently. A balloon catheter is used for this operation. The valve is opened by inflating the balloon. This type of operation is used to treat aortic stenosis, pulmonary artery stenosis, and mitral valve stenosis.

Atherosclerotic stenosis of the iliac arteries. Minimally invasive intervention is performed for both unilateral and bilateral lesions of the common or external iliac artery. The best results are obtained with unilateral pathology.

Angioplasty for vasorenal hypertension. In these cases, good patency of the iliac arteries is detected in 80-90% of patients with a 5-year observation period.

Atherosclerotic stenosis of the arteries of the femoral-popliteal segment. Indications for endovascular dilatation and stenting of vessels are non-extensive stenosis of the artery with preserved patency of the vascular bed sections located distally. However, the long-term results of the operation performed according to strict indications are very favorable: at a 5-year observation period, 60-70% of patients have good patency of the vessels in the area of the operation. Vasorenal hypertension. As is known, the most common causes of the development of vasorenal hypertension are atherosclerotic stenosis or occlusion of the renal arteries, as well as their fibromuscular dysplasia. In both the first and second cases, balloon dilatation can be effective. In all cases, angioplasty should be supplemented with stenting. The best long-term results are observed in patients with fibromuscular dysplasia. It is quite difficult to compare the results of a minimally invasive technique with the results of traditional surgery, since clear indications for its use have not been finally formulated and the surgical risk is often very high.

Carotid artery stenosis of atherosclerotic etiology in the presence of neurological symptoms, narrowing of the vessel lumen by more than 60% is usually treated with endarterectomy. The first balloon angioplasty operation was performed in 1980 (S. Kerber et al.). In most cases, stenting of the affected vessel is used after previous balloon angioplasty.

Endoprosthesis of an aortic aneurysm is performed using a special self-expanding endoprosthesis. According to most authors, balloon angioplasty should be preferred with a "high" localization of the stenosis zone (close to the entrance of the internal carotid artery into the cranial cavity). In individuals with increased surgical risk, this surgery is usually performed for restenosis of the artery after a previously undergone "open" endarterectomy.

Vascular stenting. Stents are usually placed in a narrow section of the vessel, providing the vessel with stable and long-term expansion. Stents are a metal braid made of a special material in the form of a tube. In a compressed state, its diameter is slightly thicker than the head of a match. The stent is usually placed on top of a balloon at the end of a catheter. The catheter is advanced through the vessels of the thigh, the balloon is placed in the narrow section of the vessel. X-rays help to correctly position the stent. Then, by inflating the balloon, the stent is spread, which stretches the narrow section of the vessel, creating an internal frame for it. Then the balloon is deflated and removed from the vessel, while the stent remains in its previous position. In the future, this frame can be spread to a larger diameter with a larger balloon. This is most relevant for children whose vessels are still growing.

Infrarenal abdominal aortic aneurysms. The traditional method of treating this disease is aneurysm resection followed by aortofemoral prosthesis. The first stenting operation for abdominal aortic aneurysms was performed in 1991 (J.S. Parodi et al.). Unlike the technique of minimally invasive endovascular interventions, the technique of aortic endoprosthesis is much more complicated, since it requires reliable fixation of the proximal and distal ends of the endoprosthesis to the walls of the aorta and iliac artery. The main advantage of the minimally invasive technique is its low traumatism and complications and a low number of postoperative complications. In addition, unlike the "open" operation performed through a wide laparotomy access, the function of the lungs, heart, kidneys, intestines are significantly less disturbed.

It should be mentioned about other endovascular minimally invasive interventions on the arteries. Splenic artery occlusion is performed in patients with portal hypertension with severe splenomegaly and hypersplenism at high surgical risk as an alternative to splenectomy or in the preoperative period to reduce intraoperative blood loss. Through transfemoral access, a catheter is advanced proximally and inserted into the distal splenic artery, and embolization of the main trunk or branches of the splenic artery is performed. Gianturco metal coils, special composites based on gelatin sponge or polyvinyl alcohol are used to occlude the lumen of the vessels.

Occlusion of the ductus arteriosus (arterial) in its non-ingrowth using a minimally invasive technique is a highly effective and minimally traumatic intervention, which allows you to avoid a number of complications associated with a wide sternotomy.

Selective occlusion of the branches of the hepatic artery is used to treat metastatic liver damage when radical surgical removal of liver metastases is impossible or in the general serious condition of the patient due to the underlying disease. It is known that the blood supply of metastatic nodes in the liver is carried out mainly by arteries, therefore, the blockade of arterial blood flow causes partial necrosis of the tumor node and slowing down the progress of the cancer process in the liver. When performing the operation (access - through the femoral artery), it is necessary to strive to install a

catheter in the lobar or segmental branch of the hepatic artery to avoid ischemia and necrosis of the healthy parenchyma of the liver and gallbladder. Chemoembolization of the liver vessels combines regional chemotherapy and blockade of arterial blood flow to the tumor node.

For regional chemotherapy, doxorubicin and mitomycin are most often used. Given the local fixation of the chemotherapy drug in the tumor tissue, much higher doses can be used than with systemic chemotherapy. Chemoembolization of the hepatic arteries is also used for primary hepatocellular carcinoma.

Thrombectomy of peripheral arteries is more often performed for fresh thrombosis or embolism or after reconstructive operations on the leg arteries (aortofemoral or femoral-popliteal bypass). Such a minimally invasive technique allows you to avoid sometimes quite complex surgical operations. Thrombectomy is performed using a special device "Angiojet". At the same time, its working part, introduced into the lumen of the vessel, as a result of hydrodynamic action allows you to achieve complete destruction of the thrombus and removal of its small fragments.

Minimally invasive interventions on venous vessels are used much less frequently: this is the implantation of a cava filter, transhepatic portocaval shunting. The implantation of a cava filter is carried out to prevent pulmonary embolism in phlebothrombosis of the legs. The most commonly used is the Greenfield cava filter, which resembles a spindle-shaped openwork metal basket with radially located clamps at the proximal end. The operation is performed using a transjugular approach. In a folded form, using a metal guide, the cava filter is guided into the inferior vena cava below the level of the confluence of the renal veins under the control of an X-ray screen. After opening the basket, sharp metal clamps are fixed in the vein wall. The indications for the use of a cava filter are recurrent thromboembolism of the branches of the pulmonary artery and fresh thrombi floating in the lumen of large pelvic and femoral veins.

Transhepatic portocaval bypass for the treatment of intra- and suprahepatic forms of portal hypertension syndrome has been used recently. The first such operation was performed by G.M. Richter et al. in 1990. Indications for the operation are bleeding from varicose veins of the esophagus (if other methods of treatment are unsuccessful), persistent ascites, high risk of portocaval bypass surgery. This intervention is often used in patients before liver transplantation to decompress dilated abdominal veins. The results of minimally invasive surgery are approximately the same as with traditional portocaval bypass surgery. The operation is performed as follows: the right internal jugular vein is punctured; then a guide with a plastic catheter is advanced in the distal direction and, under the control of radiography and ultrasound, a channel is formed transhepatically between the right hepatic vein and the right branch (or branches of the first order) of the portal vein. Then, after control angiography, the channel inside the liver tissue is expanded to a diameter of 8 mm using special coaxial bougies. The operation is completed by inserting a stent into the formed channel.

Closure of heart septal defects with various endovascular devices.

Heart septal defects (atrial septal defect, ventricular septal defect, patent foramen ovale) can also be treated in the X-ray operating room during interventional catheterization using various endovascular devices. These operations can be an

alternative to open heart surgery (with artificial blood circulation). All devices are folded into a thin catheter, which is inserted through the vessels into the heart cavities. All endovascular devices are made of thin metal (some of them have tissue in their composition) and have shape memory properties. This means that when they are straightened in the heart cavities, they regain their original shape. Thus, it is possible to deliver sufficiently large devices to the defect through a thin delivery system. Catheters with devices inside are advanced through the vessels into the heart cavities and close the heart septal defects. The implant acts as a kind of framework on which heart cells can grow, completely covering it over time. Therefore, for some time after implanting the device, patients need to take aspirin, which prevents blood clots, until their own cells cover the implant.

**Laser surgery**

Lasers have been used in surgery for several decades. Over time, all new operations in various fields of medicine are performed with the help of lasers. The implementation of such techniques in everyday practice depends on the production of laser devices with sufficient operating capabilities, inexpensive and convenient to use.

The word “laser” that we are familiar with is generally an abbreviation (Light Amplification by Stimulated Emission of Radiation). Lasers have found the widest application in medicine.

Laser surgery allows the use of a laser for sterile and bloodless dissection and destruction of tissues. At the same time, laser surgical units are distinguished by high precision, which allows them to affect individual cells, organs and the body as a whole. Such units, having universal properties, have wide possibilities of influencing living tissue by irradiation, excision, evaporation and coagulation (coagulation) of biological tissues with laser radiation.

Laser surgical units are used mainly in ophthalmology, otolaryngology and outpatient surgery. But every day the scope of their application is expanding.

High-intensity lasers are used in surgery, which cause irreversible changes in tissues. Using a laser scalpel, you can coagulate (cause irreversible coagulation) tissues, evaporate them, simply cut and remove them.

Advantages of laser surgery:

Lasers provide a number of advantages during surgery: under the influence of a laser beam, any infection is destroyed (prevention of purulent complications), blood vessels are sealed (absence of traditional bleeding during surgery, and therefore, swelling and pain in the postoperative period), the laser beam acts very precisely, cutting off only those tissues that are subject to removal, without affecting healthy tissues. Penetrating deep into the tissue, the laser beam activates cells, which helps to shorten the recovery period after surgery.

A big plus of laser installations is that they can be combined with endoscopic instruments during endoscopic operations (with this method, small incisions are made in the skin for the introduction of special instruments that the surgeon controls, observing the course of the operation through a monitor).

Today, small gynecological operations are performed using a surgical laser. For example, treatment of various diseases of the cervix is often carried out by coagulation of pathological tissue with a laser beam in this area. In the same way, small benign

growths (for example, condylomas) are removed. This technique is gaining increasing popularity, since when acting on the cervix it allows you to strictly dose the work of the surgical laser in depth and area, without damaging adjacent, healthy tissues. In this case, the surgical laser can be used simultaneously with a colposcope (an endoscopic device that allows you to examine the mucous membrane in detail at the site of exposure). After laser coagulation, there are no rough scars and narrowing of the cervical canal, which is especially important for women who have not yet given birth.

**Welding technology** is advancing victoriously on the ground, in the underwater world and in space. Welding is starting to advance in the medical field. It is used with success for joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised. After that the operated organs inevitably lose their functions, which are not restored when the patient recovers. The basic phenomena, which proceed in soft tissue welding, can be schematically described as follows. The tissue layers being joined are brought into contact over their surface layers by means of a welding tool. Then the surgeon clamps the tissue area to be welded by the electrodes of the welding tool and switches on the welding current source. When the welding process control program is completed and the power is turned off, the clamped tissue is released, and then the process should be repeated until the wound is closed completely.

Electrowelding of soft tissues. A method of connecting soft tissues during surgical intervention using high-frequency electric current.

The method was invented in Ukraine and is used in many clinics, more than 80 thousand operations have been performed. For the invention of the method, the team of authors was awarded the State Prize of Ukraine in the field of science and technology (2004).

The method of electrowelding of soft tissues was proposed by the Institute of Electrowelding named after E.O. Paton of the NAS of Ukraine. The idea of development belongs to academician Boris Paton, under whose leadership a team of specialists works. In 1993, employees of the Institute of Electrowelding together with surgeons of the Institute of Clinical and Experimental Surgery (now the Institute of Surgery and Transplantology) and the Okhmatdyt Hospital conducted experiments that confirmed the fundamental possibility of obtaining a welded joint of various soft tissues of animals by the method of bipolar coagulation. Research into this technology began in the experimental department of the Institute of Surgery and Transplantology in 1992.

Later, the project "Welding of Soft Living Tissues" became one of the two priority areas of activity of the International Association "Welding" of the IAW, founded in 1991 at the Institute of Electric Welding. In 1996, in accordance with the IAW program, an international team was organized with the participation of Ukrainian specialists and the American financial company Consortium Service Management Group, Inc (CSMG).

In 1997, Ukrainian specialists first demonstrated surgical operations using electric welding of soft tissues on animals in the USA.

The set of experiments on a control group of animals (pigs), as well as on removed and removed human organs using welding technology in general surgery, which

showed the reproducibility of obtaining positive results, served as the basis for the issuance by the Ministry of Health of Ukraine of certificates of state registration of the use of welding equipment in medical practice for 2001–2004, 2005–2010 and 2011–2015 (№ 9613/2010). This allowed the development of methods of welding soft tissues in more than 80 clinics in Ukraine and Russia for use in various fields of general and minimally invasive surgery, as well as the development of more than 130 surgical intervention techniques. To date, more than 80 thousand operations have been performed on various human organs using this technology. The technology is approved for clinical use in Ukraine and the United States.

In 2004, the team of project authors led by Academician Boris Paton was awarded the State Prize of Ukraine.

Using electric welding technology in clinical conditions, the following operations can be performed:

- performing plasty of the fallopian tubes;
- obtaining a strong connection with perfect tightness when closing the lumen in the ureter;
- suturing the stomach without the threat of its contents entering the abdominal cavity;
- hermetic welding of the intestine;
- restoring the continuity of the nasal septum;
- removal of tonsils;
- cosmetic operations on the mammary glands, abdominal cavity, lower and upper extremities

Principles of operation

Schematically, the process of welding soft tissues consists of:

the connected layers of tissue contact each other with their surface layers; then the surgeon compresses the welded area of tissue with the electrodes of the welding tool and turns on the current source; after executing the welding process control program and turning off the energy, the captured tissue is released, and the process is repeated until the wound is completely closed. The formation of a welded joint is based on the effect of electrothermal denaturation of protein molecules. When exposed to a low-voltage electric current, cell membranes are partially destroyed, resulting in the release of protein fluid. Due to the coagulation (clotting) of the protein, the tissues stick together - "weld". After a certain time, the morphological structure of the tissue is restored, so there is no scar in the usual sense of the word on the operated organ. In order for the restoration of the affected organ to be quick and without complications, the heat input should be minimal, but sufficient to form a joint. In this regard, the requirements for controlling the welding process are significantly increased. To simplify the surgeon's task in controlling the welding process, an automatic control system has been created. The temperature in the welding zone is 60-70 °C.

Advantages:

Unlike traditional surgery, the welding method does not require suture material, staples, clips and stapling devices, since the connection is made using the "native" material of the welded organ using special equipment. The places of the seams when

using the electric welding method heal easily, as evidenced by the difficulty of finding them during an autopsy a month after the operation.

The absence of suture material at the site of the operation, in turn, eliminates the possibility of an inflammatory process and the threat of infections. When using welding technology, according to surgeons, not a single case of postoperative complications has been recorded; complete sealing of the connection at the site of the weld is achieved and asepsis is ensured. At the same time, the welding method allows you to save on suture materials.

Another widespread method of high-frequency electrosurgery — the coagulation process — causes burns and tissue necrosis at the site of heating, while electric welding causes significantly less tissue trauma and the absence of burns. This is confirmed by morphological studies, as well as the absence of smoke and unpleasant odor during the welding process. This has a positive effect on both the health of the patient and the surgeon, especially when working with infected patients. Significantly less trauma also contributes to faster and easier healing of the tissues of the operated organ, restoration of its morphological structure and functions.

Prospects for use

Thanks to the international project "Welding of Soft Living Tissues" and financial assistance from foreign partners, electric tissue welding is already routinely used in some modern clinics in Ukraine. At the same time, the technology still has broad prospects for the development of applications in gynecology, urology, thoracic surgery, ophthalmology, oncology, etc. In the future, the technology may find application in space, on sea vessels, submarines, etc.

While the American partners of the project "Welding of Soft Living Tissues" have already patented welding equipment in 40 countries around the world, foreign experts are closely monitoring the clinical development of the method in Ukraine. Thus, in the future, a wide international implementation of Ukrainian technology is possible.

It is planned to certify and produce devices for welding living tissue in China. In particular, this direction is being addressed by the Chinese-Ukrainian Institute of Welding named after E.O. Paton, established in 2011.

**Microsurgical technologies in surgery**

- Microsurgical technique in plastic and reconstructive surgery ensures the performance of operations, involves the use of optical magnification and ultra-thin suture material (thickness 16-25 microns).
- It is used in various fields - from ophthalmology to neurosurgery
- It is important to emphasize that the use of microsurgical technique is harmoniously combined with other classical methods of plastic surgery, which allows emphasizing the advantages of some and avoiding the disadvantages of others.
- For the first time, the use of an operating microscope in otorhinolaryngology was reported by C. Nylen in 1921.
- The introduction of microsurgical techniques into general surgery began in the 1960s, when J. Jackobson and E. Suarez (1960) at the XI International Congress of Surgeons reported on the successful suturing of vessels with a

diameter of up to 1.6 mm, after which the operating microscope was used for limb injuries (R. Malt and C. McKhann 1962).

- In 1972, the first report appeared on the free transplantation of a complex tissue complex with the restoration of its blood supply by microanastomosing vessels, thus beginning a new era of organ and tissue autotransplantation.
- Microsurgical techniques are used in plastic surgery for suturing (plastics) of small-caliber vessels (about 1 mm in diameter), which allows for the transplantation of a wide variety of blood-supplied autotransplants. The use of microsurgical techniques in interventions on individual bundles of peripheral nerves has significantly increased the efficiency of their suturing and plastic surgery.

Equipment and apparatus

Microsurgery requires the use of an operating microscope, special instruments and ultra-thin suture material. The operating microscope must provide high contrast and stereoscopicity of the image, a constant working (focal) distance, significant (up to x40) magnification and the possibility of its smooth change, as well as the possibility of simultaneous participation in the operation of two or three surgeons.

Modern models use pedal control, zooms and mechanisms for coordinate movement of the tube, which allows you to smoothly and without the participation of the surgeon's hands to change the degree of optical magnification, sharpness and position of the working part of the microscope above the operating field. The operating microscope also allows you to document operations using a built-in video camera

Microsurgical instruments

The basis of special instruments is made up of microneedle holders, microscalpels, microtweezers and microscissors, their presence already allows for the application of a microvascular suture and a nerve suture.

Spring models of microneedle holders are most widely used in clinical practice. However, some surgeons believe that the accuracy of microsuture application can be increased by using microneedle holders with a hydraulic, pneumatic or electric drive, which almost completely eliminates the movements of the operator's fingers in the act of capturing or releasing the microneedle and microthread.

Microtweezers of various purposes and sizes are also a necessary part of the instruments. With their help, the surgeon captures tissues and suture material.

Microscissors provide preparation and separation of the thinnest anatomical formations.

Effective execution of a microvascular suture requires the use of other instruments. Single vascular microclamps provide bleeding stoppage and serve for vessel marking. With the help of double vascular microclamps, the ends of the vessel are fixed in the position required for suturing.

Suture material is of great importance for the successful performance of microsurgical operations. Atraumatic needles with a thickness of 70-130 microns are used with a synthetic thread with a thickness of 16-25 microns. For operations on vessels with a diameter of 0.3-0.6 mm, metallized suture material is used, which is made by applying metal to the end of a synthetic thread, which, thanks to special processing, turns into a kind of needle.

History of the development of laparoscopy.

Minimally invasive surgery (or small-access surgery) is surgical interventions through small tissue punctures or natural physiological openings using special equipment and instruments that allow you to operate in small spaces and use a video monitor to control the course of the operation; surgery aimed at minimizing the area of intervention in the body and the degree of tissue injury. The main techniques used in minimally invasive intervention are laparoscopic (endoscopic) surgery and endoscopy

- Ott in 1901 was the first to endoscopically examine the abdominal organs
- Kelling in 1901 was the first to use an optical device to perform laparoscopy
- Jacobeus in 1910 introduced laparoscopic examination into clinical practice
- Kalk in 1929 first designed a special laparoscope with optics and lighting
- Veres in 1938 was the first to design a special needle for applying pneumoperitoneum
- Jacobeus in 1910 was the first in world practice to propose the method of thoracoscopy
- Kurt Zemm 1985-1988 was the first to perform appendectomy, developed the technique of operations on the pelvic organs, developed a large number of instruments,
- Eric Müch 1985 was the first to perform cholecystectomy

In addition to laparoscopic surgery, the increasing number of other minimally invasive operations, videoendoscopic interventions or minimally invasive surgical interventions, in which surgeons use alternative accesses to internal organs, can be combined under one term - minimally invasive surgery. This term best reflects the essence of the listed surgical interventions.

Taking into account the interests of patients (i.e. minimizing the traumatic nature of the operation) and under the influence of various socio-economic factors (reducing the length of stay of patients in the hospital and returning them to a full-fledged normal life and work as soon as possible), progress in modern surgery and modern technologies has given rise to a new era in surgery - the era of minimally invasive surgery.

Methods for identifying tumor stages, modern diagnostic methods, surgical techniques, including the performance of regenerative operations, thanks to which the impact on the patient is significantly reduced, both mental, psychological and biochemical, have radically changed modern surgery.

Advantages of minimally invasive techniques:

- Cosmetic effect.
- Short postoperative period.
- Fast rehabilitation period.
- Less likelihood of postoperative hernias.
- Optical magnification.
- Better visualization of organs during revision of the abdominal organs.
- Better conditions for performing simultaneous operations.
- Less likelihood of postoperative adhesions.
- Diagnostic laparoscopy is less traumatic than laparotomy.

– Promising method.

Disadvantages of minimally invasive techniques:

– High cost and equipment cost.
– Anesthesia.
– Duration.
– Specific complications.
– Training.
– Man-made.
– Video camera.
– Impossibility of tissue palpation.
– Operating field in 2D image

Laparoscopic equipment: The set of equipment and tools necessary to perform any video laparoscopic operation can be divided into 3 main groups.

1. Optical equipment. This set includes a 10-millimeter laparoscope with a miniature video camera attached to it, a light source attached to the laparoscope, one or two video monitors, to which the image of the operating field is transmitted using the video camera. All surgical manipulations are performed by the operator and his assistants, focusing on the screen image on the monitors. In addition, a video recorder is usually used to record the entire operation from the monitor

2. Equipment for providing access to the abdominal cavity. This includes a gas insufflator into the abdominal cavity, connected to a gas cylinder, and trocars of various diameters, through which instruments are inserted. Pneumoperitoneum is applied using a special atraumatic needle (Veresch needle). The insufflator provides automatic gas supply to the abdominal cavity depending on the intra-abdominal pressure. If it exceeds a certain value, the gas injection is stopped. After the application of pneumoperitoneum, for which carbon dioxide is used, a laparoscope is inserted into the abdominal cavity and the organs in the abdominal cavity are examined. Only after this are trocars inserted under visual control. There is a special ball valve in the lumen of the trocar, which allows various instruments to be inserted through its lumen without hindrance, but prevents gas from escaping from the abdominal cavity.

3. A set of laparoscopic surgical instruments. It usually includes miniature clamps, a dissector, a hook for dissecting and coagulation of tissues, scissors, atraumatic needles and a needle holder, a tool for applying metal clips to blood vessels, endoscopic stapling devices, a plastic bag for placing the removed organ in it, and an electric suction tip. Depending on the type of operation, the number of instruments in the set can be expanded or, conversely, reduced.

Types of laparoscopic surgical interventions

As already noted, the range of surgical interventions performed laparoscopically is quite wide. They can be divided into 2 main groups. The first group includes conventional operations that are used more often than "open" ones.

Cholecystectomy. In most medical institutions, laparoscopic cholecystectomy has replaced the "traditional" one. It is performed not only in chronic but also in acute cholecystitis, including destructive ones. The mortality rate for planned interventions ranges from 0 to several hundredths of a percent; in experienced hands, the operation

ends with laparotomy in approximately 3% of cases (due to technical difficulties). In emergency operations, this figure is several times higher. According to world statistics, in the early 90s, the incidence of extrahepatic bile duct injuries was about 0.6%, which is 3-7 times higher than with open "traditional" surgery; in the late 90s, this figure became almost the same for both methods of surgical intervention. During laparoscopic surgery, if choledocholithiasis is suspected, intraoperative cholangiography is performed using a special cannula. If the diagnosis is confirmed, endoscopic papillotomy is performed with removal of stones using instruments. Laparoscopic biliary-digestive anastomoses are under development.

Appendectomy is a fairly simple laparoscopic operation. Its use is permissible for local diffuse peritonitis limited to the right iliac region, with mandatory washing and drainage of this area at the end of the operation. In comparison with the "open" method, laparoscopic appendectomy is accompanied by a smaller number of suppurations of the postoperative wound. Other advantages of laparoscopic operations in this type of intervention are small, which is due to the short length of the laparotomic access.

Inguinal hernioplasty is technically significantly different from traditional methods. Its meaning is to cover the medial and lateral inguinal fossa with a synthetic mesh. Given the relatively short terms of remote observations after the intervention, it is quite difficult to judge the frequency of hernia recurrences. According to preliminary data, when using laparoscopic implantation of a synthetic mesh, recurrences occur in no more than 2% of cases.

Fundoplication for diaphragmatic hernias combined with severe esophagogastric reflux is performed in various modifications and its essence is not fundamentally different from the traditional method. The Nissen method is most often used, the results of which are not inferior to the results of "open" surgery. Contraindications to performing laparoscopic surgery are shortening of the esophagus and previous interventions on the stomach. Vagotomy. Of the various vagotomy options, laparoscopic selective proximal vagotomy is the most complex, and therefore the risk of incomplete transection of the vagus nerve branches and ulcer recurrence is very high. Therefore, posterior trunk vagotomy is usually used in combination with anterior selective proximal vagotomy (selective transection of the secretory branches of the anterior vagus nerve that go to the secretory zone of the stomach), or with anterior seromyotomy. According to preliminary data, the frequency of ulcer recurrence after various variants of laparoscopic vagotomy does not exceed 5%. However, the final opinion on the effectiveness of the method can be made with the accumulation of a significant number of observations of patients in the distant (more than 5 years) terms after surgery, since in some patients, ulcer recurrences occur in the late stages.

Splenectomy is used as a radical or palliative method of treatment of a number of hematological diseases. The main contraindications are a significant increase in the spleen and pronounced abnormalities in the coagulogram. The spleen is removed from the abdominal cavity by performing a minilaparotomy with an incision about 5-6 cm long, or using a special device that allows you to grind the organ to a homogeneous mass, and then place it in a plastic bag and remove it.

Adrenalectomy is usually performed in specialized surgical endocrinological centers. Since the mid-1990s, the operation has become firmly established in the

surgical practice of these clinics: access is transabdominal or retroperitoneal using a plastic expander bag (dilator) to create space for gas injection.

Colon resection in benign and malignant diseases (diffuse familial polyposis, diverticulosis, Crohn's disease, cancerous tumors) is performed in two variants: completely laparoscopic with the imposition of an interintestinal anastomosis using a stapling device inserted through the anus, or with the formation of an interintestinal anastomosis through a small (3-7 cm) incision on the anterior abdominal wall using the usual method. In early stages of colon cancer, laparoscopic removal of the colon is quite acceptable. If the tumor has grown beyond the serous cover, this method is probably impractical, since there is a very high risk of tumor dissemination throughout the peritoneum, as well as implantation of tumor cells in the places of trocar insertion. Adhesiolysis - dissection of adhesions in acute adhesive intestinal obstruction. The operation is performed in the absence of pronounced swelling of the small and large intestine loops. Adhesions are also a contraindication. In this case, the risk of intestinal damage during the imposition of pneumoperitoneum or the introduction of trocars into the abdominal cavity increases many times.

In inoperable malignant neoplasms of the esophagus and the cardiac part of the stomach, laparoscopic gastrostomy and jejunostomy have become quite widespread.

The second group includes a fairly wide range of laparoscopic interventions. Suturing a perforated gastric or duodenal ulcer is most appropriate in young patients with "asymptomatic" ulcers. Adequate drainage of the abdominal cavity is performed similarly to drainage with the "open" method. Distal resection of the pancreas is performed for benign neoplasms (most often for neuroendocrine tumors, less often for the initial stages of cancer). Gastric resection for peptic ulcer disease and early gastric cancer is performed as in the "open" method, with the removal of all regional lymph nodes. Gastrojejunal anastomosis is formed endocorporeally, but with the help of stapling devices. To remove the resected stomach, a mini-laparotomy is used. Esophagocardiomyotomy for achalasia cardia is performed according to the usual method. For fundoplication, the Nissen method is used (prevention of gastroesophageal reflux). Laparoscopic techniques are used for rectal excision for cancer. This provides an even more complete examination of the pelvic organs than with the traditional method. The rectum is removed through the perineal approach. The colostomy is formed by performing a minilaparotomy.

As modern technologies develop, the list of laparoscopic operations is increasing; liver resection and extensive pancreatic resection in cancer are already performed laparoscopically.

Specific complications of laparoscopic operations include damage to internal organs caused by defects in the technique of trocar insertion, as well as gas embolism, hypercapnia, respiratory acidosis, and subcutaneous emphysema. The second group of complications can develop when using the so-called gas-free technique of laparoscopic interventions. In this case, adequate air space in the abdominal cavity, which provides various manipulations on the abdominal organs, is created using special abdominal wall lifts (laparolifting). The disadvantages of laparoscopic surgery are the high cost of equipment, as well as the problem associated with the training of surgeons.

The concept of minimally invasive endobiliary surgery Almost all surgical interventions in this group of patients are performed in a special operating room with X-ray equipment.

Liver abscesses. For a long time, the only way to treat bacterial liver abscesses was laparotomy and drainage of purulent cavities. In recent years, abscess drainage has been carried out by puncture and drainage under the control of ultrasound or computed tomography with subsequent washing of the contents of the abscess with antiseptic solutions. At the same time, antibiotic therapy is prescribed, taking into account the sensitivity of the microbial flora of the abscess contents to antibiotics. In case of multiple abscesses (most often cholangiogenous), the largest of them are punctured and drained. Such tactics allow more than 2 times to reduce postoperative activity and the number of complications.

Nonparasitic (simple) liver cysts. Currently, the operation of choice in the treatment of patients with simple liver cysts is their puncture and drainage under the control of ultrasound or computed tomography with subsequent sclerotherapy, the purpose of which is to induce aseptic necrosis of the epithelial lining of the cyst for gradual obliteration of its cavity against the background of through-skin drainage. Absolute alcohol is used as a sclerosant. For small cysts (diameter no more than 3-4 cm), a single puncture and exposure to the sclerosing solution for 10 minutes is sufficient. For cysts of larger diameter, external drainage is necessary. A similar technique is used for giant liver cysts (diameter 15-20 cm and more), which allows you to avoid technically quite complex "open" surgical interventions.

Liver echinococcosis. As is known, the generally accepted method of treating liver echinococcosis is traditional surgery. In recent years, a minimally invasive technique has been used. Its essence is as follows. First, under ultrasound or CT control, the cyst is punctured, its contents are aspirated and sent for urgent microscopic examination, during which live protoscolex of the parasite are detected. Then, an antiparasitic drug is injected into the cyst cavity (20-30% sodium chloride solution or concentrated glycerin solution). After a 10-15-minute exposure, the solution is aspirated and again subjected to microscopic examination. The detection of dead protoscolex in the solution indicates adequate antiparasitic effect of the drug. The operation is completed by external drainage of the cyst cavity. After a few days, drains of a larger diameter are sequentially inserted into the cyst cavity through a metal guide for complete aspiration of the fragmented chitinous membrane through a choledochoscope inserted through a drainage tube. In the case of a dead echinococcus containing thick viscous detritus, as well as in the case of calcification of its walls, a minimally invasive surgical technique is contraindicated.

Primary and secondary liver cancer. For the treatment of patients with primary hepatocellular carcinoma or metastatic liver damage, cutaneous alcoholization of tumor nodes is used. For this purpose, absolute alcohol (3-5 ml) is injected into the peripheral parts of the tumor under ultrasound control - the tumor tissue, together with the vessels that feed it, necrotizes. Later, necrosis spreads to the central areas of the neoplasm, and partial tumor regression occurs. Often, this technique is combined with chemoembolization of the branches of the hepatic artery, which increases the life expectancy of patients.

Acute cholecystitis. Ultrasound-guided cholecystostomy has become widespread in elderly and senile patients. It allows you to quickly eliminate the inflammatory process and prepare the patient for a planned operation. Scarring stricture of the distal part of the common bile duct. In most cases, endoscopic papillosphincterotomy is effective. However, with extensive strictures (more than 2 cm), this operation is not suitable. In case of mechanical jaundice and contraindications to reconstructive surgery, a minimally invasive intervention is resorted to - external-internal drainage of the biliary tract. For this, the dilated intrahepatic bile duct is punctured under ultrasound control and cholangiography is performed. Then, gradually, under the control of an X-ray screen, a metal guide is passed through the narrowed section of the duct into the duodenum, along which a plastic drainage with multiple side holes is passed at its distal end in such a way that part of the holes remains in the dilated part of the common bile duct, the other part in the duodenum. This ensures normal outflow of bile through the drainage into the lumen of the intestine. Through the proximal end, the drainage is fixed to the skin, and in the postoperative period it is washed with antiseptic solutions to prevent occlusion of the side holes. After a few months, the drainage can be removed if a well-formed channel is formed in the area of the previously narrowed common bile duct.

Scarring stricture of the hepatocholedochal or biliodigestive fistula. In these situations, both traditional treatment (reconstructive surgery) and a minimally invasive method of dilating the bile duct (biliodigestive fistula) can be used. Reconstructive operations are quite technically complex and are accompanied by complications in the postoperative period. After puncture of the intrahepatic duct, balloon dilation of the narrowed section of the extrahepatic bile duct (or biliodigestive fistula) is performed under the control of ultrasound and cholangiography. The effectiveness of the procedure is monitored by fluoroscopy and then the bile ducts are drained with an external-internal drainage. A few months after the formation of a sufficiently wide channel, the drainage is removed. In addition, external-internal drainage of the biliary tract is used in the treatment of a number of diseases accompanied by mechanical jaundice (cancer of the extrahepatic bile ducts, head of the pancreas, major duodenal papilla).

New advances in laparoscopic surgery

The major limitations of standard laparoscopic techniques have been the following:

The human hand is a wonderful structure and provides multitude of different functions during open surgery. This function is absent during standard laparoscopic surgery since the abdomen is closed and the procedure is performed with long surgical instruments inserted from the outside into the abdomen.

Two-dimensional image of the laparoscope: The image transmitted by the laparoscopic camera that surgeon utilizes as his eyes is a two-dimensional image. For some procedures this is a major limitation because of the poor depth perception that is associated with two dimensional images.

Retraction of internal organs: During open surgery insertion of the hand into the abdomen allows the surgeon to move the intestine and other organs away from the site of the surgery. During standard laparoscopic surgery the hand is not introduced into

the abdomen and introducing long thin instruments into the abdomen performs the surgery. Retraction of internal organs is often a major problem for some procedures.

Limitation of instruments: the standard instruments in laparoscopic surgery are long thin instruments. These instruments are poorly suited for many complex laparoscopic procedures.

New technologies for advance laparoscopic surgery

Two new technologies that are particularly promising are: hand access devices and robotic surgery.

Hand access devices. The human hand performs many functions during surgery that are difficult to reproduce with laparoscopic instruments. The loss of the ability to place the hand into the abdomen during traditional laparoscopic surgery has limited the use of laparoscopy for complex abdominal surgery on the pancreas, liver and bile duct. New laparoscopic hand-access devices that allow the surgeon to place a hand into the abdomen during laparoscopic surgery and perform many of the different functions with the hand that was previously possible only during open surgery. We have utilized this new device to develop a variety of laparoscopic pancreatic, liver and biliary procedures such as the Whipple operation, distal pancreatectomy and liver resection that were not possible previously by standard laparoscopic techniques.

**Robot-assisted surgery utilizing the Da Vinci computer robot system**. Da Vinci is a computer-assisted robotic system that expands a surgeon's capability to operate within the abdomen in a less invasive way during laparoscopic surgery. Da Vinci system allows greater precision and better visualization compared to standard laparoscopic surgery.

The operations with the Da Vinci System are performed with no direct mechanical connection between the surgeon and the patient. The surgeon is remote from the patient, working a few feet from the operating table while seated at a computer console with a three-dimensional view of the operating field. The physician operates two masters (similar to joysticks) that control the two mechanical arms on the robot. The mechanical arms are armed with specialized instruments with hand-like movements which carry out the surgery through tiny holes in the patient’s abdomen. The arms eliminate any hand tremor by the surgeon and offer motion scaling – allowing extremely precise movements within the patient.

The history of robot-assisted surgery has more than twenty-five years. Experience and technologies previously used for military purposes have resulted in the emergence of robot assistants, which have enabled the surgeon to perform a number of specific manipulations with the utmost precision. In 1985, the first surgical robotic system was introduced - the Puma 560, which was used in neurosurgery

Later, the arsenal of surgeons was replenished with the PROBOT manipulator for transurethral prostate resection, and in 1992 the RoboDoc system appeared, which was used in orthopedics for joint prosthetics.

All of these systems were highly specialized installations for providing stages of surgical operations and were not full-fledged robotic systems. In 1993, the Aesop robotic system (Aesop) from Computer Motion Inc. appeared - an automatic arm for holding and changing the position of the video camera during laparoscopic operations. This installation can still be found in a number of clinics around the world.

Currently, the only universal robotic system with remote control in the world is the DaVinci system. The robot-assisted surgical system "da Vinci" (English: Da Vinci Surgical System) is a device for performing surgical operations. It is mass-produced by Intuitive Surgical. It is used in several hundred clinics around the world.

The da Vinci robot consists of:

- Surgeon's console
- Stereoviewfinder
- Touch panel
- Patient stand
- Instrument manipulators
- Camera manipulator
- Endoscopes
- Video stand

Surgeon's console

The surgeon is outside the sterile field, he controls the three-dimensional endoscope and instruments using two main manipulator controllers and foot pedals

The stereo video finder provides images to the surgeon's console operator. The presence effect is provided by an optical system consisting of two parallel cameras that transmit an isolated image for each eye.

The touch panel is built into the armrest of the surgeon's console and is a tool for selecting various system functions.

The patient stand is a working component of the da Vinci system, its main purpose is to support the instrument manipulators and the camera manipulator.

Three manipulators with instruments attached to them, as well as one manipulator with a camera, are connected to the surgeon's console using a computer interface. During preparation for surgery, the patient's trolley and all manipulators are dressed in special sterile covers and remain in them throughout the operation.

To perform the robotic surgery, EndoWrist instruments are used, modeled after the human wrist and have seven degrees of freedom of movement, which exceeds the range of motion of the human hand. The EndoWrist instrument set includes a variety of clamps, needle holders, scissors, monopolar and bipolar electrosurgical instruments, scalpels and other specialized instruments (more than 40 types in total). EndoWrist instruments can have a diameter of 5 or 8 mm. An important feature is a clear limitation on the use of the instrument set. Each instrument can be used only 10 times, and when changing instruments, the interface recognizes the type of new instrument and the number of times it has been used

Main advantages:

- Three-dimensional stereoscopic image with the possibility of its increase and precision, ensuring high accuracy of surgical manipulations with minimal tissue trauma
- Minimization of blood loss
- Degrees of freedom of instruments exceeding those of the human hand
- The possibility of performing interventions that are difficult to perform or impossible with traditional and endovideosurgical methods

Operations using the Da Vinci robot:

- Hysterectomy and myomectomy
- Radical prostatectomy
- Mitral valve repair
- Myocardial revascularization
- Ablation of heart tissue
- Installation of an epicardial electronic pacemaker for biventricular resynchronization
- Gastric bypass
- Nissen fundoplication
- Spine surgeries, disc replacement
- Thymectomy - surgery to remove the thymus
- Lobectomy lungs
- Esophagectomy
- Mediastinal tumor resection
- Pyeloplasy
- Pyelophagoectoplasty
- Bladder removal
- Radical nephrectomy and kidney resection
- Ureteral reimplantation
- Hydroconcatenation of brain tissues
- Brain lobotomy
- -hyroidectomy
- Tonsillectomy

**The Cyber Knife** is a frameless robotic radiosurgery system invented by John R. Adler, a Stanford University Professor of Neurosurgery and Radiation Oncology, and Peter and Russell Schonberg of Schonberg Research Corporation.

The CyberKnife system is a method of delivering radiotherapy, with the intention of targeting treatment more accurately than standard radiotherapy. The CyberKnife system is used for treating benign tumors, malignant tumors and other medical conditions.

Several generations of the CyberKnife system have been developed since its initial inception in 1990. There are two essential features of the CyberKnife system that set it apart from other stereotactic therapy methods.

**Robotic mounting**

The first is that the radiation source is mounted on a general purpose industrial robot. Mounted on the Robot is a compact X-band linac that produces 6MV X-ray radiation. The linac is capable of delivering approximately 600 cGy of radiation each minute - a new 800 cGy / minute. The radiation is collimated using fixed tungsten collimators (also referred to as “cones”) which produce circular radiation fields. At present the radiation field sizes are: 5, 7.5, 10, 12.5, 15, 20, 25, 30, 35, 40, 50 and 60 mm. Mounting the radiation source on the robot allows near-complete freedom to position the source within a space about the patient. The robotic mounting allows very

fast repositioning of the source, which enables the system to deliver radiation from many different directions without the need to move both the patient and source as required by current gantry configurations.

**Image guidance**

The image guidance system is the other essential item in the CyberKnife system. X-ray imaging cameras are located on supports around the patient allowing instantaneous X-ray images to be obtained.

**6D skull**

The original (and still utilized) method is called 6D or skull based tracking. The X-ray camera images are compared to a library of computer generated images of the patient anatomy. Digitally Reconstructed Radiographs (or DRR's) and a computer algorithm determines what motion corrections have to be given to the robot because of patient movement. This imaging system allows the CyberKnife to deliver radiation with an accuracy of 0.5mm without using mechanical clamps attached to the patient's skull. The use of the image guided technique is referred to as frameless stereotactic radiosurgery. This method is referred to as 6D because corrections are made for the 3 translational motions (X, Y and Z) and three rotational motions. It should be noted that it is necessary to use some anatomical or artificial feature to orient the robot to deliver X-ray radiation, since the tumor is never sufficiently well defined (if visible at all) on the X-ray camera images.

**6D Skull tracking**

Additional image guidance methods are available for spinal tumors and for tumors located in the lung. For a tumor located in the spine, a variant of the image guidance called Xsight-Spine is used. The major difference here is that instead of taking images of the skull, images of the spinal processes are used. Whereas the skull is effectively rigid and non-deforming, the spinal vertebrae can move relative to each other, this means that image warping algorithms must be used to correct for the distortion of the X-ray camera images. A recent enhancement to Xsight is Xsight-Lung which allows tracking of some lung tumors without the need to implant fiduciary markers.

**Fiducial**

For soft tissue tumors, a method known as fiducial tracking can be utilized. Small metal markers (fiducials) made out of gold for bio-compatibility and high density to give good contrast on X-ray images are surgically implanted in the patient. This is carried out by an interventional radiologist, or neurosurgeon. The placement of the fiducials is a critical step if the fiducial tracking is to be used. If the fiducials are too far from the location of the tumor, or are not sufficiently spread out from each other it will not be possible to accurately deliver the radiation. Once these markers have been placed, they are located on a CT scan and the image guidance system is programmed with their position. When X-ray camera images are taken, the location of the tumor relative to the fiducials is determined, and the radiation can be delivered to any part of the body. Thus, the fiducial tracking does not require any bony anatomy to position the radiation. Fiducials are known however to migrate and this can limit the accuracy of the treatment if sufficient time is not allowed between implantation and treatment for the fiducials to stabilize.

**Synchrony**

The final technology of image guidance that the CyberKnife system can use is called the Synchrony system. The Synchrony system is utilized primarily for tumors that are in motion while being treated, such as lung tumors and pancreatic tumors. The synchrony system uses a combination of surgically placed internal fiducials, and light emitting optical fibers (markers) mounted on the patient skin. Since the tumor is moving continuously, to continuously image its location using X-ray cameras would require prohibitive amounts of radiation to be delivered to the patient's skin. The Synchrony system overcomes this by periodically taking images of the internal fiducials, and predicting their location at a future time using the motion of the markers that are located on the patient's skin. The light from the markers can be tracked continuously using a CCD camera, and are placed so that their motion is correlated with the motion of the tumor.

A computer algorithm creates a correlation model that represents how the internal fiducial markers are moving compared to the external markers. The Synchrony system is therefore continuously predicting the motion of the internal fiducials, and therefore the tumor, based on the motion of the markers. The correlation model can be updated at any time if the patient breathing becomes in any way irregular. The advantage of the Synchrony system is that no assumptions about the regularity or reproducibility of the patient breathing have to be made.

To function properly, the Synchrony system requires that for any given correlation model there is a functional relationship between the markers and the internal fiducials. The external marker placement is also important, and the markers are usually placed on the patient abdomen so that their motion will reflect the internal motion of the diaphragm and the lungs.

**RoboCouch**

A new robotic six degree of freedom patient treatment couch called RoboCouch has been added to the CyberKnife which provides the capability for significantly improving patient positioning options for treatment.

**Frameless**

The frameless nature of the CyberKnife also increases the clinical efficiency. In conventional frame-based radiosurgery, the accuracy of treatment delivery is determined solely by connecting a rigid frame to the patient which is anchored to the patient's skull with invasive aluminum or titanium screws. The CyberKnife is the only radiosurgery device that does not require such a frame for precise targeting. Once the frame is connected, the relative position of the patient anatomy must be determined by making a CT or MRI scan. After the CT or MRI scan has been made, a radiation oncologist must plan the delivery of the radiation using a dedicated computer program, after which the treatment can be delivered, and the frame removed. The use of the frame therefore requires a linear sequence of events that must be carried out sequentially before another patient can be treated. Staged CyberKnife radiosurgery is of particular benefit to patients who have previously received large doses of conventional radiation therapy and patients with gliomas located near critical areas of the brain. Unlike whole brain radiotherapy, which must be administered daily over several weeks, radiosurgery treatment can usually be completed in 1-5 treatment

sessions. Radiosurgery can be used alone to treat brain metastases, or in conjunction with surgery or whole brain radiotherapy, depending on the specific clinical circumstances.

By comparison, using a frameless system, a CT scan can be carried out on any day prior to treatment that is convenient. The treatment planning can also be carried out at any time prior to treatment. During the treatment the patient need only be positioned on a treatment table and the predetermined plan delivered. This allows the clinical staff to plan many patients at the same time, devoting as much time as is necessary for complicated cases without slowing down the treatment delivery. While a patient is being treated, another clinician can be considering treatment options and plans, and another can be conducting CT scans.

The delivery of a radiation treatment over several days or even weeks (referred to as fractionation) can also be beneficial from a therapeutic point of view. Tumor cells typically have poor repair mechanisms compared to healthy tissue, so by dividing the radiation dose into fractions the healthy tissue has time to repair itself between treatments. This can allow a larger dose to be delivered to the tumor compared to a single treatment.

**Gamma Knife**

One of the most widely known stereotactic radiosurgery systems is the Gamma Knife. The Gamma Knife was originally developed by Lars Leksell, remains the gold standard method for delivery of stereotactic radiosurgery to the brain. The GammaKnife system uses 201 Cobalt-60 sources located in a ring around a central treatment point ("isocenter"). The Gamma Knife system is equipped with a series of 4 collimators of 4mm, 8mm, 12mm and 16mm diameter, and is capable of submillimeter accuracies. The Gamma Knife system does however require a head frame to be bolted onto the skull of the patient, and is only capable of treating cranial lesions. As a result of frame placement, treatment with Gamma Knife does not require real time imaging capability as the frame does not allow movement during treatment. This is the reason that the Gamma Knife system is likely to be more accurate than Cyber Knife. The Cyberknife Society and Accuray maintain that there are no peer-reviewed published papers that establish Gamma Knife as being more accurate than CyberKnife.

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**Tests**

1. Endovascular surgery is:

A. Is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised.
B. is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels
C. it is the reattachment of a completely severed body part.
D. is a modern surgical technique in which operations in the abdomen are performed through small incisions (up to 1.5 cm) as compared to the larger incisions needed in laparotomy.
E. is a general term for surgical procedures that require an operating microscope.

2. Replantation:
A. is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised.
B. is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels
C. is the reattachment of a completely severed body part.
D. is a modern surgical technique in which operations in the abdomen are performed through small incisions (up to 1.5 cm) as compared to the larger incisions needed in laparotomy.
E. is a general term for surgical procedures that require an operating microscope.

3. Microsurgery:
A. is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised.
B. is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels
C. is the reattachment of a completely severed body part.
D. is a modern surgical technique in which operations in the abdomen are performed through small incisions (up to 1.5 cm) as compared to the larger incisions needed in laparotomy.
E. is a general term for surgical procedures that require an operating microscope.

4. Laparoscopic surgery:
A. is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised.
B. is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels
C. is the reattachment of a completely severed body part.
D. is a modern surgical technique in which abdominal surgeries are performed through small incisions compared to the larger incisions required in laparotomy.
E. is a general term for surgical procedures that require an operating microscope.

5. Welding:

A. is joining damaged human tissues and restoration of the functioning of human organs. It is used to dissect tissues and to stop or prevent bleeding when tissue and blood vessels are incised.
B. is a form of minimally invasive surgery that was designed to access many regions of the body via major blood vessels
C. is the reattachment of a completely severed body part.
D. is a modern surgical technique in which abdominal surgeries are performed through small incisions compared to the large incisions required in laparotomy.
E. is a general term for surgical procedures that require an operating microscope.

6. What statement about GammaKnife is not correct?
A. The GammaKnife system uses any kind of penetrating wave.
B. The GammaKnife system uses 201 Cobalt-60 sources located in a ring around a central treatment point ("isocenter").
C. The Gamma Knife system is equipped with a series of 4 collimators of 4mm, 8mm, 12mm and 16mm diameter, and is capable of submillimeter accuracies.
D. The Gamma Knife system does however require a head frame to be bolted onto the skull of the patient, and is only capable of treating cranial lesions.
E. Gamma Knife system is likely to be more accurate than Cyber Knife.

7. What statement about Cyber Knife is not correct?
A. The Cyber Knife is a frameless robotic radiosurgery system.
B. The element of the CyberKnife is radiation produced by a small linear particle accelerator
C. The element of the CyberKnife is a robotic arm which allows you to direct energy to any part of the body from any direction.
D. The CyberKnife system is a method of delivering radiotherapy, with the intention of targeting treatment more accurately than standard radiotherapy.
E. Treatment with CyberKnife system does not require real time imaging capability as the frame does not allow movement during treatment.

*8.* What statement about Robot-assisted surgery (Da Vinci) is not correct?
A. Da Vinci is a computer-assisted robotic system that expands a surgeon's capability to operate within the abdomen in a less invasive way during laparoscopic surgery.
B. Da Vinci system allows greater precision and better visualization compared to standard laparoscopic surgery.
C. The operations with the Da Vinci System are performed with direct mechanical connection between the surgeon and the patient.
D. The surgeon is remote from the patient, working a few feet from the operating table while seated at a computer console with a three-dimensional view of the operating field.
E. The physician operates two masters (similar to joysticks) that control the two mechanical arms on the robot. The mechanical arms are armed with specialized instruments with hand-like movements which carry out the surgery through tiny holes in the patient’s abdomen.

9. What statement about Magnetic resonance imaging (MRI) is correct?

A. Is a medical procedure in endoscopy is combined with ultrasound to obtain images of the internal organs in the chest and abdomen.
B. Is slice or cross-section imaging using any type of penetrating wave.
C. is a medical imaging technique used in radiology to visualize detailed internal structures that uses of nuclear resonance for imaging.
D. Magnetic resonance imaging provides good spatial resolution (the ability to distinguish between two separate structures at tiny distance from each other)
E. It uses ionizing radiation in the radio frequency range.

10. What statement about CT is not correct?
A. CT provides good spatial resolution (the ability to distinguish between two separate structures at tiny distance from each other)
B. CT provides comparable resolution with much better contrast resolution (the ability to distinguish differences between two relatively similar, but not identical, tissues).
C. It uses ionizing radiation
D. CT scanning is not contraindicated in case of cardioverter-defibrillator.
E. CT scanning is not contraindicated in case of insulin pumps.

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Principles of using of ultrasonic waves<br>– Principles of using of radiological waves<br>– Principles of using of endoscopic techniques<br>– Principles of transplantation | - To represent consecution and value of the diagnostic methods for abdominal pathology as a table<br>- To represent merits and demerits of the Laparoscopic methods as a table |
| Study:<br>– Types of radiological waves.<br>– Types of endoscopic techniques. | -To determine the best radiological methods depending on types of pathology<br>-To select more suitable mini-invasive methods depending on types of pathology |

## 1.3. Abdominal pain syndrome. Features of pain syndrome in acute diseases of the abdominal cavity. Its significance for differential diagnosis.

**Overview.**

Abdominal pain accounts for 5% to 10% of all emergency department visits or 5 to 10 million patient encounters in the United States annually. Another study demonstrates that 25% of patients presenting to the emergency department complained of abdominal pain. Diagnoses vary according to age group: pediatric, geriatric, and everyone else who deals with abdominal pain in children. Appendicitis is more common in children, whereas biliary disease, colonic diverticulitis, and intestinal infarction occur more commonly in the elderly. Hospitalized patients may develop abdominal pain during the course of their illness, making diagnosis and treatment more difficult.

**Educational aims:**

- To collect the anamnesis and to make clinical inspection on patients with abdominal pain.
- To find the etiology and pathogenesis of abdominal pain.
- To understand the clinical pictures of abdominal pain.
- The complications of abdominal pain.
- To make plan of inspection on patients with abdominal pain.
- To carry out the analysis of data received from laboratory and tool methods of inspection on patients with abdominal pain.
- To define indications for surgical treatment on patients with abdominal pain or to appoint conservative treatment.
- To treat patients with abdominal pain.
- To estimate work capacity for patients with abdominal pain.

**A student must know:**

- Anatomical-physiological information about organs of the abdominal cavity.
- Classification of acute abdominal pain.
- Clinical picture of acute abdominal pain and acute abdomen.
- Methods of observation patients with abdominal pain
- Giving first aid for abdominal pain.
- Medical program (conservative and surgical treatment) for patients with abdominal pain.
- Question of rehabilitation patients with acute abdomen.

**A student must be able to:**

- Anatomical-physiological features of innervations of organs of abdominal cavity
- Etiology and pathogenesis of acute abdominal pain.
- Methods of inspection of the abdominal cavity organs.
- Clinical examinations and tests.
- Ultrasonography, computered tomography, peritoneal lavage and endoscopic ultrasound to establish the correct diagnosis.
- Clinical symptomatic of acute abdomen and its complicated forms.
- Differential diagnostic of acute abdominal pain.
- Testimony and contra-indication to surgical and conservative methods of treatment.
- Methods of surgical treatment of different forms of acute abdomen.

**Terminology.**

| Term | Definition |
|---|---|
| Pain | the focal issue in the evaluation of the patient suspected of having an acute abdomen. |
| Parietal pain | associated with intra-abdominal disorders may be more intense and precisely localized. |
| Referred pain | perceived at a site distant from the source of stimulus. |

| | |
|---|---|
| Visceral pain | dull and poorly localized, usually in the epigastrium periumbilical region, or suprapubic region |
| Colicky pain | this pain is assumed as crescendo-decrescendo character |
| Guarding | the detection of increased abdominal muscle tone during palpation |

**Content:**

**ANATOMY AND PHYSIOLOGY**

**Developmental Anatomy**

The developmental anatomy of the abdominal cavity and of its viscera determines normal structure and influences the pathogenesis and clinical manifestations of most abdominal diseases. Peritoneal attachments and visceral sensory innervations are particularly important to the evaluation of acute abdominal disease. After the 3rd week of fetal development, the primitive gut divides into foregut, midgut, and hindgut. The superior mesenteric artery supplies the midgut (the fourth portion of the duodenum to the midtransverse colon). The foregut includes the pharynx, the esophagus, the stomach, and the proximal duodenum, whereas the hindgut comprises the distal colon and the rectum. The afferent fibers accompanying the vascular supply provide sensory innervations to the bowel and associated visceral peritoneum.

Thus, disease in the proximal duodenum (foregut) stimulates celiac axis afferents to produce epigastric pain. Stimuli in the cecum or appendix (midgut) activate afferent nerves accompanying the superior mesenteric artery to cause periumbilical pain, and distal colon disease induces inferior mesenteric artery afferent fibers to cause suprapubic pain. The phrenic nerve and afferent fibers in C3, C4, and C5 dermatomes accompanying the phrenic arteries innervate the diaphragmatic musculature and the peritoneum on its undersurface. Stimuli to the diaphragm therefore cause referred shoulder pain. The parietal peritoneum, abdominal wall, and retroperitoneal soft tissue receive somatic innervations corresponding to the segmental nerve roots. The richly innervated parietal peritoneum is particularly sensitive. Parietal peritoneal surfaces sharply localize painful stimuli to the site of the stimulus. When visceral inflammation irritates the parietal peritoneal surface, localization of pain occurs. Maneuvers that exacerbate this irritation then intensify the pain. The many “peritoneal signs” useful in the clinical diagnosis of the acute abdomen originate in this fashion. Dual-sensory innervation of the abdominal cavity by both visceral afferents and somatic nerves produces clinical pain patterns that aid in diagnosis. For example, the pain of acute appendicitis originates with poorly localized periumbilical pain progressing to sharply localized right lower quadrant pain when the inflammation involves the parietal peritoneal surface.

Peripheral nerves mediate sharp, sudden, well-localized pain. Sensory afferents involved with intraperitoneal abdominal pain transmit dull, sickening, poorly localized pain of more gradual onset and protracted duration. The vagus nerve does not transmit pain from the gut. Small, unnamed sympathetic afferent nerves transmit pain from the esophagus to the spinal cord. Afferent nerves from the liver capsule, the hepatic ligaments, and the central portion of the diaphragm, the splenic capsule, and the pericardium enter the central nervous system from C3 to C5. The spinal cord from T6

to T9 receives pain fibers from the periphery of the diaphragm, the gallbladder and the stomach, the pancreas, and the small intestine. Pain fibers from the colon, appendix, and pelvis viscera enter the central nervous system at the 10th and 11th thoracic segments. The sigmoid colon, rectum, renal pelvis and capsule, ureter, and testes pain fibers enter the central nervous system at $T_{11}$-$L_1$. The bladder and the recto-sigmoid corner are innervated by $S_2$- $S_4$ spinal nerves.

Cutting, tearing, crushing, or burning usually do not produce pain in the abdominal viscera. However, stretching or distention of the peritoneum produces pain. Bacterial or chemical peritoneal inflammation produces visceral pain, so does ischemia. Cancer can cause intra-abdominal pain by invading sensory nerves. Abdominal pain may be visceral, parietal, or referred. Visceral pain is dull and poorly localized, usually in the epigastrium periumbilical region, or suprapubic region, and it usually does not lateralize well. Patients with visceral pain may also experience sweating, restlessness, and nausea.

*The parietal or somatic pain* associated with intra-abdominal disorders may be more intense, precisely localized. Referred pain is felt in an area that is distant from the primary source. Disease in the bile duct or gallbladder may produce shoulder pain.

Dilation of the small intestine can cause pain that radiates to the back. During the 5th week of intrauterine development, the intestine outgrows the peritoneal cavity, protrudes through the umbilical cord and undergoes a 180-degree counterclockwise rotation. During this process, the bowel remains outside the peritoneal cavity until approximately the 10th week, when it returns to the abdomen, and an additional 90-degree counterclockwise rotation occurs. This embryologic rotation places the viscera in their adult positions, and subsequent fusion of the portions of the colonic and duodenal mesenteries with the mesothelium of the posterior abdomen forms the normal ultimate peritoneal attachments. Knowledge of these attachments is clinically important during the evaluation of patients with the acute abdomen because of variation in the exact position of the viscera (e.g., pelvic or retrocecal appendix) and the compartmentalization of the abdomen by mesenteric attachments. The latter, for example, may channel duodenal or gastric contents from the site of a perforated ulcer to the right lower quadrant.

**Peritoneal Pathophysiology**

Mesothelial cells cover the visceral and parietal peritoneal surfaces. Openings into radials arranged lymphatics penetrate the diaphragmatic peritoneal surface.

The entry of bacteria into the abdominal cavity can cause the penetration of fluid through the peritoneum. This loss of fluid from the circulation may lead to dehydration and may produce the clinical signs of resting or orthostatic hypotension and tachycardia. Diaphragmatic lymphatics are the major route for the clearance of bacteria and cellular debris from the abdominal cavity. This process leads to an intraperitoneal circulation of fluid toward the subdiaphragmatic regions bilaterally. Fluid not cleared in this fashion tends to accumulate in the deep end of the pelvis. Thus, subdiaphragmatic, subhepatic, paracolic, or pelvic fluid collections can accompany visceral perforation. The peritoneal surfaces localize bacteria and the products of inflammation. In the peritoneum, as a result of inflammation, blood flow increases, its permeability increases, fibrin and exudate are formed. The bowel responds to

inflammation with paralysis. Fibrin restricts intestinal movement, causes the formation of adhesions that limit the area of inflammation. Peritonitis can affect both part of the peritoneum and all of it.

Transudation can produce an increase in the peritoneal fluid, which is rich in protein and leukocytes that facilitate the formation of fibrin on peritoneal surfaces.

Peritonitis denotes peritoneal inflammation from any cause. Primary or spontaneous peritonitis can occur as a diffuse bacterial infection without an obvious intra-abdominal source of contamination Pneumococcus or streptococcus more often cause primary peritonitis in children, and Escherichia coli and Klebsiella in adults. The infection enters the abdominal cavity by hematogenous, lymphogenous means or by contact (through the fallopian tubes in women).

The more common secondary peritonitis results from perforation, infection, or gangrene of an intra-abdominal organ, usually of the gastrointestinal tract.

Gastrointestinal secretions, pancreatic secretions, bile, blood, urine, and meconium cause chemical peritonitis when in contact with the peritoneum. A common form of chemical peritonitis follows perforation of a peptic ulcer. Bile peritonitis may result from perforation of the gallbladder or leakage from the bile ducts. Ordinarily, slow bleeding into the abdominal cavity produces relatively few signs of inflammation; the addition of bacteria to blood produces suppuration. The sickest postoperative patients may have tertiary peritonitis that kills 30% to 64% of affected patients. The syndrome of poorly localized intra-abdominal infection, an altered microbial flora, progressive organ dysfunction, and high mortality define tertiary peritonitis.

Peritonitis causes abdominal pain, either generalized or localized, depending on the disease. Appendicitis usually causes localized pain. Perforated peptic ulcer usually produces generalized abdominal pain. Acute cholecystitis causes right upper quadrant pain referred to the right scapula or shoulder. Physical findings of patients with peritonitis are abdominal tenderness, guarding, and rebound tenderness.

**Site of abdominal pain in relation to suspected pathology**

| Whole abdomen | Generation peritonitis and mesenteric infraction |
|---|---|
| Right upper quadrant | Acute cholecystitis<br>Cholangitis<br>Hepatitis<br>Peptic ulceration |
| Left upper quadrant | Peptic ulceration<br>Pancreatitis<br>Splenic infarct |
| Right lower quadrant | Appendicitis<br>Ovarian cyst<br>Ectopic pregnancy<br>Pelvic inflammatory disease<br>Meckels diverticulum<br>Mesenteric adenitis<br>Ureteric colic<br>Rectus sheath haematoma |

| | |
|---|---|
| | Right-sided lobar pneumonia |
| Left lower quadrant | Sigmoid diverticular disease<br>Ovarian cyst<br>Ectopic pregnancy<br>Pelvic inflammatory disease<br>Ureteric colic<br>Rectus sheath haematoma<br>Left-sided lobar pneumonia |
| Radiating pain<br>Back<br>Groin | Peptic ulcer<br>Pancreatitis<br>Aortic aneurysm<br>Dissecting aneurysm<br>Ureteric colic<br>Testicular torsion |

## CLINICAL DIAGNOSIS

### History and Present Illness

*Pain* is the focal issue in the evaluation of the patient suspected of having an acute abdomen. The history should therefore characterize and document the pain as precisely as possible. The duration of the pain is important, but the location, mode of onset, and character of the pain help in making a diagnosis. Abdominal pain that persists for 6 hours or more with severe intensity increases the likelihood that surgical operation will be required. If the pain ebbs after a few hours, however, the probability of surgical disease decreases, but not to zero. Visceral pain caused by distention, inflammation, or ischemia usually feels dull and poorly localized in the midabdomen. Depending on the organ involved, the pain may be felt in the epigastrium, the periumbilical area, or the lower abdomen. Diseases of the kidneys or ureters produce pain in the flanks. Parietal pain, however, is sharper and better localized. Localized parietal peritonitis can produce pain confined to one of the four quadrants of the abdomen.

In an evaluation of the location of the pain, the concept of referred pain becomes important. Subdiaphragmatic disorders can produce pain referred to the shoulder.

Blood or pus beneath the left diaphragm can cause left shoulder pain. Biliary disease can cause referred pain in the right shoulder or the back. Diseases above the diaphragm such as basal pneumonia can cause pain referred to the neck or shoulder in the C4 distribution. Upper abdominal pain suggests peptic ulcer, acute cholecystitis, or pancreatitis. Conversely, ovarian cysts, diverticulitis, and ruptured tubo-ovarian abscesses produce lower abdominal pain. Small bowel obstruction usually causes midabdominal pain sometimes referred to the back.

Migratory pain shifting from one place to another can give insight into the diagnosis. For example, pain that moves from the epigastrium to the periumbilical area to the right lower quadrant suggests acute appendicitis. Distention and inflammation of the appendix produce visceral pain perceived in the periumbilical area. When the inflammation spreads and produces parietal peritonitis, the pain localizes in the right lower quadrant of the abdomen. Another example of moving or migratory pain occurs with perforated duodenal ulcer. The leakage of duodenal contents from a perforated ulcer causes intense and localized epigastric pain. However, if the leaked duodenal

content gravitates down the right paracolic gutter into the fossa iliaca. In this case, the clinical picture may resemble another disease, for example, Kocher's symptom in acute appendicitis. Late in many cases, the pain may become generalized because of diffuse peritonitis.

The initial manifestations of the acute abdomen and the evolution of the pain syndrome may give some insight into the cause of the pain. The pain can start suddenly or instantly with no prior symptoms. Sudden or explosive onset of severe abdominal pain suggests free perforation of a viscous such as the duodenum or acute intestinal ischemia from a visceral artery embolus. This type of pain onset can awaken patients from sleep or can incapacitate them during work or play. Sudden, generalized, excruciating pain suggests an intra-abdominal catastrophe that may produce shock requiring resuscitation and prompt operation. In other conditions, the pain comes on with progressively increasing intensity over 1 to 2 hours. This progressive pain represents the usual manifestation of the diseases that commonly produce the acute abdomen such as acute cholecystitis, acute pancreatitis, and proximal small bowel obstruction. Some illness begins with vague general abdominal discomfort that progress to abdominal pain over a few hours. The pain becomes more intense and subsequently localizes. This group of illnesses generally includes acute appendicitis, incarcerated hernia, distal small bowel obstruction, colon obstruction, diverticulitis, and contained or walled-off visceral perforation. The quality, severity, and periodicity of the pain may provide clues to the diagnosis. Steady, sharp pain accompanies perforated duodenal ulcer or perforated appendix.

The early pain of small bowel obstruction is vague and deep seated. This pain then assumes a crescendo-decrescendo character described as colicky pain.

However, if obstruction produces intestinal infarction, then the pain becomes dull and constant. The pain of ureteral obstruction is extremely severe and intense.

Patients with kidney stones appear restless, agitated, or hyperactive and tend to move about, in contrast to patients with peritoneal inflammation, who prefer to lie quietly and remain undisturbed. Sudden, excruciating pain in the upper abdomen or the lower chest or interscapular region suggests aortic dissection.

*Radiation of pain* or referral of pain may help in diagnosis. Radiation of pain around the right costal margin to the right shoulder and scapula suggests acute cholecystitis. Pancreatitis usually produces epigastric pain that may radiate along the costal margins to the back or straight through to the back. Kidney stones may cause pain radiating to the groin or the perineal area.

*Vomiting* may occur from the severity of the pain or because of disease in the gastrointestinal tract. Generally, patients with abdominal pain requiring surgical treatment experience the pain before vomiting occurs. Vomiting frequently precedes the pain in patients with medical conditions. Patients with appendicitis usually have pain and anorexia for a while before vomiting, and patients with gastroenteritis experience vomiting before abdominal pain. Vomiting frequently occurs in patients with acute cholecystitis, acute gastritis, acute pancreatitis, and bowel obstruction. Proximal small bowel obstruction produces more vomiting than distal small bowel obstruction.

Vomiting occurs uncommonly in patients with colon obstruction. Small bowel obstruction of longer duration can cause feculent vomiting. Obstruction distal to the ampulla of Vater causes bile-stained vomitus, whereas obstruction proximal to the ampulla causes clear vomitus. Most patients with acute abdominal pain have no desire to eat. Anorexia may precede the pain of acute appendicitis.

Bowel function, including a history of constipation, diarrhea, or a recent change in bowel habits, can be important. Watery diarrhea associated with abdominal pain suggests gastroenteritis. Immunosuppressed patients can contract cytomegalovirus (CMV) infection, salmonellosis, or cryptosporidiosis, which may produce diarrhea.

A past history of diarrhea raises the suspicion of inflammatory bowel disease, either Crohn's disease or ulcerative colitis. Failure to pass gas or bowel movements suggests mechanical intestinal obstruction. A history of jaundice, hematemesis, hematochezia, or hematuria is important in the evaluation of acute abdominal pain.

A careful menstrual history is important in women with abdominal pain. Ovulation can produce significant abdominal pain. Furthermore, abdominal pain in a woman with a missed menstrual period or irregular menstrual periods can be related to complications of an undiagnosed pregnancy or an ectopic pregnancy.

The drug history is important in managing patients with acute abdominal pain. Corticosteroids predispose to gastroduodenal ulceration and the possibility of perforation.

Corticosteroids also immunosuppress patients and obscure the manifestations of acute intra-abdominal disease. Furthermore, patients who have taken steroids for long periods require perioperative steroid supplementation. Patients who take diuretics need evaluation of their fluid and electrolyte status. Anticoagulants can cause intra-abdominal, intestinal, and mesenteric bleeding. The effects of anticoagulants must be reversed preoperatively. Cocaine can cause abdominal pain. Of course, many patients developing acute abdominal pain are taking cardiovascular drugs, hormones, tranquilizers, diuretics, and numerous other classes of agents that must be managed in the perioperative period.

Past history becomes important, especially regarding prior surgery. For example, if a patient has had an appendectomy, cholecystectomy, and so forth, it has a significant impact on the differential diagnosis of acute abdominal pain. Past history can also give clues to the diagnosis of the present illness. In addition, past history\ may reveal significant co-morbid conditions requiring careful management during the perioperative period. Systemic illnesses or cardiac or pulmonary disease must be excluded as possible causes of the abdominal pain syndrome.

**Physical Examination**

The physical examination usually provides important information that helps in the diagnosis and treatment of the cause of pain. The patient's overall appearance, ability to communicate, habitus, and signs of pain should be noted. Does the patient lie quietly in bed or actively move about? Does the patient lie on his or her side with knees and hips flexed? Does the patient appear dehydrated with dry mucous membranes? An apprehensive patient lying quietly in bed, avoiding motion, and complaining of abdominal pain probably has serious intra-abdominal disease. The physical examination continues with the evaluation of the vital signs.

Low fever often accompanies diverticulitis, appendicitis, and acute cholecystitis. High fever more often occurs in pneumonia, urinary tract infection, septic cholangitis, or gynecologic infection. Rapid heart rate and hypotension may mean advanced complicated disease with peritonitis. Peritonitis causes hypovolemia as plasma volume leaves the intravascular space. The general appearance of the patient and the vital signs determine the urgency of the diagnostic work-up and implementation of therapy.

Examination of the abdomen always begins with inspection, with particular attention to scars, hernias, masses, or abdominal wall defects. Hernias incarcerated in the groin, umbilicus, or incisions of obese patients can be difficult to detect. The examiner should observe whether the contour of the abdomen appears scaphoid, flat, or distended. Abdominal distention can mean intestinal obstruction, ileus, or fluid including ascites, blood, or bile.

*Palpation* is a crucial step in evaluating the patient with acute abdominal pain. For this examination, the patient and the examiner should be positioned comfortably to conduct gentle palpation. The examiner should assess the patient's facial expression for signs of pain or discomfort during the examination. Careful palpation for tenderness is important. This must be done gently to avoid hurting the patient and should begin in an area away from the pain site if possible. The finding and the description of tenderness are the most important steps in palpation of the abdomen of patients with acute abdominal pain. Localized tenderness over the McBurney point suggests appendicitis. Tenderness in the right upper quadrant suggests an inflamed gallbladder. Diverticulitis produces tenderness in the left lower quadrant. Tenderness throughout the abdomen may reflect diffuse peritonitis.

The detection of increased abdominal muscle tone during palpation is called *guarding.* Guarding may be voluntary, involuntary, localized, or generalized. To detect guarding, the examiner should press gently but slowly and firmly on the patient's abdomen. Using two hands works best. The detection of muscle spasm denotes guarding. If, after asking the patient to relax and breathe deeply, the patient's muscles relax, it denotes voluntary guarding. If the muscles remain rigid or tense, it indicates involuntary guarding, which means underlying peritonitis. Guarding may be localized or generalized. Generalized intense guarding produces the board-like abdomen characteristic of perforated duodenal ulcer. Careful deep palpation can detect abdominal masses. Acute cholecystitis, acute pancreatitis, abdominal aortic aneurysm, and diverticulitis can produce abdominal masses. Severe guarding can interfere with the detection of abdominal masses by palpation.

*Rebound tenderness* is also a sign of peritonitis. To detect rebound tenderness, the examiner presses deep into the patient's abdomen with flattened fingers. Sudden withdrawal of that hand may cause an increase in the abdominal pain, and this symptom indicates peritonitis. Rebound tenderness can be elicited directly over the site of the abdominal pain. Pressing and releasing the abdomen away from the site of pain can exacerbate the pain at the original site. Careful, deep palpation can detect abdominal masses. Severe guarding can interfere with the detection of abdominal masses by palpation. In acute cholecystitis, palpation in the right subcostal area during deep inspiration by the patient may elicit pain. This finding is called a positive Murphy's sign. This sign can be detected either with the patient sitting or supine.

The gallbladder may be palpated during this maneuver. Direct compression by the probe may cause pain during ultrasound examination.

*Auscultation* of the abdomen should give information about the presence or absence of bowel sounds. A quiet abdomen indicates ileus. Hyperactive bowel sounds may occur in gastroenteritis. Periods of quiet interrupted by the onset of high-pitched hyperactive bowel sounds characterize the peristaltic rushes of mechanical small bowel obstruction. Evaluation of bowel sounds requires careful auscultation for several minutes. During auscultation of the abdomen, the examiner can effectively evaluate tenderness and guarding further by palpating gently with the stethoscope. The examiner should also note the presence or absence of bruits in the abdomen.

*Percussion* is an important part of the abdominal examination. When percussion elicits tenderness, it indicates inflammation and has the same implication as rebound tenderness. Hyper-resonance or tympanic sound is heard during percussion of the abdomen means gaseous distention of the intestine or stomach. Resonance to percussion over the liver suggests free intra-abdominal gas.

Other tests or maneuvers can aid in the assessment of patients with abdominal pain. Pain during gentle tapping of a fist or deep palpation at the costovertebral angles may suggest pyelonephritis. An inflamed retrocecal appendix or a psoas abscess can produce pain or tenderness on motion of the psoas muscle. If passively extending the hip or actively flexing the hip against resistance causes pain, this is called a positive iliopsoas sign. If internal or external rotation of the flexed hip causes pain, it is referred to as a positive obturator sign.

During the bimanual pelvic examination, the physician should seek evidence of uterine or adnexal masses or tenderness. Acute salpingitis, tubo-ovarian abscess, or torsion of an ovarian cyst can cause acute abdominal pain. The speculum examination allows inspection of the cervix for discharge. Rectal examination should include tests for occult blood, and the examiner should note the presence of masses or tenderness. An inflamed pelvic appendix or a pelvic abscess can cause tenderness detected by rectal examination.

**Laboratory Testing**

Laboratory investigation of most patients with acute abdominal pain usually includes a complete blood count. Intra-abdominal inflammation can produce elevation in the white blood cell count, although this is not always true. One study demonstrated a poor correlation between the white blood cell count and the degree of intra-abdominal inflammation in patients operated on because of acute abdominal pain. If a patient with unequivocal and persistent abdominal pain has a normal or low white blood cell count, a differential count may disclose a marked left shift, which can be more significant than finding an elevation in the white blood cell count. If patients have obvious dehydration, a history of vomiting or diarrhea, or if they have been taking medications such as diuretics that may influence their serum electrolyte values, one should measure the concentrations of serum sodium, potassium, blood urea nitrogen, creatinine, glucose, chloride, and carbon dioxide. In addition, these laboratory tests enable one to detect diabetes, renal failure, or other systemic diseases. Measurements of serum amylase and lipase may help in the evaluation of upper abdominal pain by giving evidence of pancreatitis. Although elevated serum amylase accompanies pancreatitis,

other diseases such as perforated duodenal ulcer and small bowel infarction can also cause increased serum amylase concentrations. Patients with right upper quadrant abdominal pain should have measurements of serum bilirubin, alkaline phosphatase, and serum transaminase because of the possibility of obstructive jaundice or acute hepatitis. Urinalysis can detect evidence of urinary tract infection, hematuria, proteinuria, or hemoconcentration. Women of childbearing age who have acute abdominal pain or hypotension should have measurement of the serum or urine ß-human chorionic gonadotropin concentration.

**Diagnostic Imaging**

History and physical examination are the most important and useful steps in the nature of abdominal pain. However, advances in imaging of the abdomen have improved the diagnostic accuracy and the overall management of patients experiencing acute abdominal pain. After collecting the anamnesis and physical examination of the patient, the patient is subjected to additional laboratory examination, ultrasound, X-ray (including CT), and, if necessary, endoscopy. With that information, a decision to operate or not was made usually on the basis that the patient probably had some disease best treated surgically. The laparotomy was considered diagnostic as well as therapeutic. Historically, before modern imaging tests, as many as 20% of patients operated on for acute appendicitis did not have it.

Plain films still have usefulness in several circumstances. A radiograph centered on the diaphragm detects pneumoperitoneum better than other radiographic techniques. A survey radiograph of the thoracic and abdominal cavities vertically can reveal only 1 ml of air under the diaphragm. For the occasional patient who cannot stand up, a lateral decubitus radiograph of the abdomen can also detect pneumoperitoneum effectively. A cross-table lateral radiograph with the patient in the left lateral position can detect 5 to 10 mL of gas under the lateral abdominal wall. Free air in the peritoneal cavity indicates a perforation of the gastrointestinal tract. Perforated duodenal ulcers usually allow small amounts of air to escape into the peritoneal cavity. About 75% of patients with perforated duodenal ulcers have radiographically detectable pneumoperitoneum. Perforations of the stomach and the colon can cause extensive pneumoperitoneum. The amount of pneumoperitoneum can also depend on the duration of the leak from the perforation. Plain films of the abdomen can show extensive pneumoperitoneum.

If the film defines both the serous and the related mucosal walls of the bowel, it means free air is at that serous surface. In addition, free air can delineate the falciform ligament on plain abdominal films.

An extensive hydropneumoperitoneum appears as an extremely long air-fluid level on an upright film. A supine film can show a large air collection beneath the abdominal wall that does not conform to any bowel loop. Plain films show abnormal calcifications. About 10% of gallstones and 90% of kidney stones are radiopaque due to their high calcium content. Appendicoliths can calcify and appear radio-graphically in 5% of patients with appendicitis. Pancreatic calcifications characteristic of chronic pancreatitis show on plain films, and vascular calcifications can aid in the evaluation of abdominal aortic aneurysms, visceral artery aneurysms, and atherosclerosis of visceral vessels.

Supine and erect plain films of the abdomen show gastric outlet obstruction; proximal, mid, and distal small bowel obstruction; and colon obstruction. The characteristics of small bowel obstruction include multiple air-fluid levels in dilated, centrally located loops of intestine with visible valvulae conniventes and an absence or paucity of colon gas. Obstruction of the large intestine is visualized in the form of its distension with haustration. In some patients with intestinal paresis, signs of mechanical obstruction may be detected on abdominal X-rays. Paralytic ileus can produce distended bowel with multiple air-fluid levels. Plain radiographs show paralytic ileus resulting from intra-abdominal or retroperitoneal inflammation.

The radiographic findings of paralytic ileus include excessive distention and fluid with gas distributed from stomach to rectum. Plain films of the abdomen may also detect gas in the portal or mesenteric venous system, intramural gas in the gastrointestinal tract, gas in the biliary ducts or gallbladder, and gas in the urinary tract or retroperitoneal areas. When plain films show gas in the portal or mesenteric veins, it usually means advanced and serious disease. CT can show small amounts of gas in veins and also may delineate the cause of the abnormality. If the patient's history suggests renal colic, an intravenous pyelogram may confirm the diagnosis of a kidney stone.

CT scanning has provided definite improvements in diagnostic accuracy in evaluating patients with abdominal pain and also reveals anatomic and pathologic detail not possible with plain radiographs. Therefore, CT and ultrasonography now occupy the central imaging role in this situation. Although history and physical examination provide essential information in evaluating patients with the acute abdomen, modern imaging techniques, including ultrasound and CT, can lead to an anatomic diagnosis in most cases. One prospective study of 40 patients with acute abdominal pain revealed that CT significantly improved the diagnostic accuracy of clinical evaluation plus plain radiographs. Clinical examination and plain films were 50% correct, but CT scanning was 95% correct. CT scans accurately detected the specific anatomic lesion in 57.5% of cases compared with 17.5% with clinical examination and plain films. This study included no patients with appendicitis, the most common cause of the acute abdomen, because the surgeons did not refer any cases of suspected appendicitis for inclusion in the study. However, other investigators evaluated the role of CT in the diagnosis of acute appendicitis in100 consecutive patients studied prospectively. The CT interpretation had 98% sensitivity, 98% specificity, 98% positive predictive value, 98% negative predictive value, and 98% overall accuracy for diagnosing or ruling out appendicitis. According to the authors' calculations, these 100 CT scans produced a net savings of $44,731 in the care of the study patients because of improved diagnostic accuracy. CT scans can add important value to the diagnosis of acute appendicitis. However, focused specialists using excellent equipment in an environment of inquiry conducted this study, and the results may not be reproducible in all hospitals. Other researchers do not agree with this point of view (for example, in acute appendicitis).

Ultrasonography is useful for acute abdomen because it provides rapid, safe, low-cost evaluation of the liver, gallbladder, bile ducts, spleen, pancreas, appendix, kidneys, ovaries, adnexa, and uterus. Transvaginal ultrasound will help in the diagnosis of

gynecological pathology. Ultrasonography detects and characterizes the distribution of intra-abdominal fluid. Color-Doppler ultrasonography allows evaluation of the intra-abdominal and retroperitoneal blood vessels. Aortic and visceral artery aneurysms, venous thrombosis, arteriovenous fistulas, and vascular anomalies can be evaluated with ultrasound. Unfortunately, patients with acute abdominal disease frequently have excessive abdominal gas that interferes with careful and detailed sonographic evaluation of the abdominal organs, but overlying gas, bone, and Appendicitis, the most common cause of the acute surgical abdomen in North America, can be difficult to diagnose. Plain films and barium enema studies generally add little to the diagnosis. However, in patients with uncomplicated appendicitis, ultrasonography can detect appendicoliths, demonstrate a distended or thick-walled appendix, or detect periappendiceal and pericecal inflammatory changes. Ultrasound is reliable and sensitive for the detection of appendicoliths and the demonstration of an abnormally distended or thick-walled appendix. Conversely, CT detects acute appendicitis and defines the changes of complicated appendicitis. CT scans can enable the examiner to differentiate diffuse periappendiceal inflammation from an abscess. In addition, CT scans detect many of the diseases included in the differential diagnosis of acute appendicitis. CT detects blood and other fluids in the abdominal cavity. Intramural intestinal hemorrhage is readily detected by CT. CT scans accurately reveal mesenteric venous thrombosis. CT scans can delineate diverticulitis and its complications, such as abscess and even pyelophlebitis. CT is especially helpful in evaluating pancreatitis by revealing minimal edema, extensive edema, fluid collections, hemorrhage, and necrosis; in addition, it effectively evaluates the complications of pancreatitis such as abscess or pseudocyst. CT scans show the signs of advanced peritonitis. With this technique, one can also evaluate the complications of colon perforation and of small bowel disease such as intussusception. Although history and physical examination provide essential information in evaluating patients with the acute abdomen, modern imaging techniques, including ultrasound and CT, can lead to an anatomic diagnosis in the majority of cases.

## CLINICAL MANAGEMENT

### Differential Diagnosis

Information from the patient's history, physical examination, laboratory tests, and imaging studies usually permits a diagnosis, but uncertainty can still remain. Because appendicitis is a common disease, it must remain in the differential diagnosis of any patient with persistent abdominal pain, particularly right lower quadrant pain. The diagnosis of appendicitis is easy to miss, and perforation substantially increases morbidity and mortality from the disease. Delay in diagnosis is the principal reason for unfavorable outcomes in appendicitis. Appendicitis is the most common cause of the acute abdomen in childhood; however, in older patients, acute cholecystitis, bowel obstruction, cancer, and acute vascular conditions assume importance in addition to appendicitis. The differential diagnosis in young women can be difficult because they can have salpingitis, dysmenorrhea, ovarian lesions, and urinary tract infections as well as complications of pregnancy, which can confound the evaluation of abdominal pain. Of course, the medical causes of abdominal pain must be considered, but patients with medical disease generally lack specific localized tenderness and guarding.

Approximately one-third of patients present with atypical abdominal pain, making diagnosis difficult.

**Decision to Operate**

These difficulties notwithstanding, the surgeon must make a decision to operate or not. Certain indications for surgical treatment exist. For example, definite signs of peritonitis such as tenderness, guarding, and rebound tenderness support the decision to operate. Likewise, severe or increasing localized abdominal tenderness should prompt an operation.

Patients with abdominal pain and signs of intoxication should be operated on. Signs of pneumoperitoneum during X-ray examination in patients with acute abdomen, which indicates perforation of the gastrointestinal tract, are an absolute indication for surgical intervention. However, patients with signs of pneumoperitoneum without signs of an acute abdomen (for example, after colonoscopy, celiotomy) are subject to dynamic observation. If the signs of acute abdominal pathology are doubtful, it is necessary to carry out dynamic monitoring of the patient, which will help clarify the diagnosis. The patient can be discharged from the hospital if symptoms of an acute abdomen are excluded. It is also possible to appoint a repeat examination of the patient. If necessary, the patient is transferred to outpatient observation. Patients with severe concomitant heart diseases, diabetes, after a stroke or myocardial infarction, spinal cord injuries often require a more balanced follow-up examination.

A special category consists of patients in a state of drug or alcohol intoxication, cancer patients, especially those undergoing chemotherapy, and also taking analgesics, as well as other patients who need constant relief from chronic pain. Patients with concealed perforation of a gastroduodenal ulcer without signs of peritonitis, especially those hospitalized a few days after the occurrence of perforation, are subject to conservative treatment. Patients with acute cholecystitis with severe concomitant pathology without signs of gangrenous changes in the gallbladder are operated under local anesthesia by performing a cholecystostomy. Patients with acute appendicitis at the stage of dense appendicular infiltrate also require conservative treatment. In case of its abscessation, the opening and drainage of the abscess is performed by retroperitoneal access under sonographic control and local anesthesia. Tactics for acute diverticulitis are similar. The modern concept of treatment of acute pancreatitis also includes intensive therapy. Formed purulent foci are drained and cleaned under sonographic control under local anesthesia.

**Preoperative Preparation**

Before the start of the operation, the patient is given premedication, which includes the administration of a painkillers, as well as a dose of an antibiotic to prevent the development of infectious complications. In case of severe septic shock caused by peritonitis, the patient will undergo short-term preoperative preparation, which includes rehydration, correction of water-electrolyte and acid-base balance disorders.

In case of severe accompanying pathology, correction of its violations is carried out before the operation. Thus, in case of accompanying diabetes, hyperglycemia is corrected by prescribing insulin even in cases of type 2 diabetes. In patients with chronic pathology of the cardiovascular system, surgical intervention and further support is carried out under constant cardiomonitoring. In critically ill patients, the rule

of three catheters is used before surgery: nasogastric decompression (1st catheter), intravenous therapy (2nd catheter) and daily diuresis control (3rd catheter).

Optimal for a successful outcome of the operation should be its performance in conditions of hemodynamic stability. However, in the setting of septic shock caused by peritonitis, this condition can be achieved by the use of glucocorticoids or sympathomimetics.

**Operation**

After the diagnosis of acute abdominal pathology requiring surgical treatment, it is necessary to draw up an operation plan. Operative intervention is carried out under endotracheal, spinal or local anesthesia. Today, access to the abdominal cavity is carried out both open and laparoscopically, if there are no contraindications to the last method. Access during laparotomy depends on the prevalence of peritonitis: in case of local inflammation, the incision is made in the location of the inflamed organ (for example, McBurney access in acute appendicitis). In case of widespread peritonitis, a midline laparotomy is performed for a full revision of the abdominal organs, lavage and drainage of the abdominal cavity.

The emergence of laparoscopy as a diagnostic method, and a little later as a therapeutic method, made it possible to qualitatively improve the quality of life of operated patients. The improvement of surgical skills and technology of laparoscopy made it possible to expand the indications for the use of this type of surgical intervention in acute abdominal pathology and today it is considered universally recognized and indisputable.

Diagnostic and therapeutic laparoscopic techniques have an important place in the management of patients with acute abdominal pain. The diagnostic accuracy spares many patients an unnecessary laparotomy and also allows definitive laparoscopic therapy that prevents additional patients from undergoing unnecessary laparotomy. Evidence suggests that diagnostic laparoscopy reduces the cost of managing patients with acute abdominal pain. Whether diagnostic laparoscopy and therapeutic laparoscopy reduce the cost remains unclear. Most patients with acute abdominal pain should be suitable candidates for laparoscopy. Laparoscopy should be avoided in hemodynamically unstable patients and in patients with extensive gaseous distention of the abdomen. Whether pregnant women with the acute abdomen should undergo laparoscopy is a practical question. One study suggested that laparoscopy in this setting was safe and effective.

**ACUTE VISCERAL ISCHEMIA**

Although patients experiencing acute visceral ischemia account for a small percentage of the population seeking medical attention for acute abdominal pain, this topic deserves special attention because of extreme difficulty in establishing a correct and timely diagnosis and because the condition has a high mortality rate. Acute arterial disease may be either occlusive or nonocclusive, and venous disease can also produce the syndrome. Arterial occlusion may be either embolic or thrombotic.

Generally, acute superior mesenteric artery embolism causes a sudden onset of extremely severe abdominal pain. This ischemic pain persists for a long time before the development of intestinal necrosis. Because the pain results from ischemia and not from peritonitis, these patients have no abdominal tenderness, guarding, or rebound.

Therefore, abdominal pain out of proportion to the abdominal physical findings should raise a question about this diagnosis. Because ischemia stops bowel motility promptly, the abdomen may be quiet to auscultation, depending on the amount of ischemic bowel. The heart is the most likely source of a superior mesenteric artery embolus. Therefore, any patient with cardiac arrhythmias, particularly atrial fibrillation, a known mural thrombus, or a recent myocardial infarction who develops acute abdominal pain should have acute superior mesenteric artery embolismhigh in the differential diagnosis. Patients with atherosclerosis can develop thrombosis at a superior mesenteric artery stenosis. Patients with acute visceral ischemia usually have marked leukocytosis and acidosis. Because cardiovascular disease is important in the development of acute visceral ischemia, most patients with that condition are persons who are middle aged or older.

Conversely, venous thrombosis can cause visceral ischemia, and those patients can be younger. Birth control pills have been implicated in venous thrombosis in young women. Patients suspected of having acute visceral ischemia should undergo arteriography. Although duplex scanning can provide information about the visceral circulation, arteriography provides better images for planning arterial reconstruction or embolectomy. However, arteriography may not help in venous disease. CT scans or magnetic resonance imaging studies can reveal and delineate clots in visceral veins. Most patients with acute visceral ischemia should undergo laparotomy.

Some patients develop visceral ischemia because of poor perfusion resulting from decreased cardiac output. Patients usually develop nonocclusive visceral ischemia while they are in the hospital, particularly in an intensive care setting. Improving cardiac output to restore intestinal perfusion is an important step in managing this problem. Arteriography may be required for complete evaluation and allows direct infusion of vasodilators for therapy.

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**Tests**

1. A 40-year-old woman with obesity complains of pain in the right hypochondrium, which constantly recurs after mistakes in the diet (spicy, fatty food) and is bought by taking antispasmodics. The pain is paroxysmal with irradiation in the right shoulder and collarbone. Vomiting of bile occurs periodically. What is the previous diagnosis?
   A. Acute appendicitis
   B. Caecal carcinoma
   C. Hematoma of the rectus sheath
   D. Torsion of an ovarian cyst
   E. Gallstones, with biliary colic
2. A 46-year-old man developed acute pain in the right iliac region while playing tennis. After 12 hours he turned to the surgeon. During examination: subfebrile temperature, leukocytosis 13,000, disturbing nausea. During palpation, a dense painful formation in the right iliac region is determined, which is confirmed sonographically. Which of the following diagnoses most likely?
   A. Acute appendicitis
   B. Caecal carcinoma
   C. Hematoma of the rectus sheath

D. Torsion of an ovarian cyst
E. Cholecystitis

3. A 64-year-old man has been experiencing paroxysmal abdominal pain for several days, prolonged vomiting, which was accompanied by abdominal distension and absence of defecation for 5 days. Hypoperistalsis is detected by auscultation, radiologically - distended loops of small intestine and fluid levels. A few years ago, he underwent a laparotomy due to an abdominal injury. What is the diagnosis?
   A. Acute appendicitis
   B. Cecal carcinoma
   C. Hematoma of the rectus sheath
   D. Torsion of an ovarian cyst
   E. Mechanical intestinal obstruction, caused by adhesion
4. A 64-year-old man with acute intestinal obstruction underwent conservative measures: supragastric decompression, intravenous infusion therapy, and siphon enema. After 4 hours of conservative treatment, the pain in the abdomen increased, abdominal distension and gurgling noise were detected, and small intestinal contents were detected by nasogastric tube. What are the further tactics of treatment?
   A. Repeat the siphon enema.
   B. Continue infusion therapy.
   C. Urgent surgical intervention
   D. Wash the nasogastric tube.
   E. Perform paranephric blockade and repeat X-ray examination
5. A 22-year-old man develops periumbilical pain, which after a few hours moves to the right lower part of the abdomen. The abdomen in this patient is painful and tense on palpation, determined by Blumberg's symptom, temperature - 37.8°C, leukocytosis 12,500. What is it?
   A. Acute appendicitis
   B. Acute diverticulitis
   C. Strangulated obstruction
   D. Acute cholecystitis
   E. Acute pancreatitis
6. A 27-year-old man developed watery, foul-smelling diarrhea, spasmodic pain, and fever on the 8th day after appendectomy for gangrenous acute appendicitis and taking clindamycin and tobramycin for a week and leukocytosis. What complications did the patient have?
   A. Obstructive jaundice
   B. Cancer of sigmoid
   C. Strangulated obstruction
   D. Torsion of an ovarian cyst
   E. Pseudomembranous colitis
7. A 63-year-old woman developed constant moderate pain in the left lower abdomen, low-grade fever, and leukocytosis 6 hours ago. A dense painful infiltrate is palpated without peritoneal signs. Similar attacks have already been

noted three times before (she was treated with antibiotics as an inpatient). What is it?

A. Acute appendicitis
B. Cecal carcinoma
C. Strangulated obstruction
D. Acute diverticulitis
E. Torsion of an ovarian cyst

8. A 24-year-old woman suddenly developed widespread pain in the lower abdomen, loss of consciousness. On examination, the patient is pale, tachycardia and hypotension are detected. The abdomen is moderately swollen and painful, hemoglobin 70 g/l. Abdominal injury denied. She has been taking birth control pills since she was 16 years old. What is it?

A. Acute cholecystitis
B. Obstructive jaundice
C. Aortic aneurysm rupture
D. Torsion of an ovarian cyst
E. Acute pancreatitis

9. A 56-year-old man suddenly develops intense, cramp-like pain in the right lower back, radiating in right testicle, after which blood appeared in the urine. What is it?

A. Acute appendicitis
B. Ureteral colic
C. Strangulated obstruction
D. Torsion of an ovarian cyst
E. Rupture of abdominal aorta

10. A 42-year-old man with a history of ulcers for more than 6 years developed a sharp stabbing pain in the upper half of the abdomen at night. During the physical examination, there is dull tension in the abdominal muscles and peritoneal signs. X-ray – pneumoperitoneum. What is it?

A. Acute appendicitis
B. Cecal carcinoma
C. Perforated gastric ulcer.
D. Acute pancreatitis
E. Gallstones, with biliary colic

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| E | C | E | C | A | E | D | C | B | C |

**Materials for the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of organs of abdominal cavity, structure of the liver. Anatomy and features of blood circulation of the spleen. | -To represent the methods of diagnostics of diseases of abdominal cavity as a table<br>-To make the flow diagram of mechanisms pains in abdomen |

| | |
|---|---|
| – Physiology peritoneum of abdominal cavity and pelvic peritoneum<br>– Pathogenesis of abdominal disease and innervations organs of abdominal cavity.<br>– Morphological changes in the organs of abdominal cavity at disease<br>– Clinical displays and methods of diagnostics of peritonitis | |

# TOPIC 2. SYSTEMIC INFLAMMATORY RESPONSE SYNDROME IN SURGICAL PATIENTS. CRITICAL CONDITIONS IN SURGICAL PATIENTS

## 2.1. Systemic inflammatory response syndrome in surgical patients. Pathogenesis, significance in various diseases and injuries. Therapeutic tactics.

**Overview.**

The body's inflammatory response to injury and the activation of cellular processes are naturally designed to restore tissue function and eliminate harmful microorganisms. Minor injuries that last for a short time are typically followed by a recovery of function with minimal treatment. In contrast, significant damage to the body triggers a strong inflammatory response, which, if not properly and promptly managed, can result in multiple organ failure and negatively affect patient survival. Understanding how the body mobilizes and regulates the inflammatory response offers a foundation for developing interventions and treatments for surgical patients. The maturation of minimally invasive techniques for major surgery during the last decade has brought complementary perspectives to the injury response paradigm, and the immunologic benefits for these surgical approaches are undergoing validation. Furthermore, the sequencing of the human genome and available technology such as deoxyribonucleic acid (DNA) microarray analysis potentially affords surgeons additional tools to profile the genetic mechanisms governing the host response to injury.

Infections with HIV leading to acquired immune deficiency syndrome (AIDS) have been the focus of considerable academic attention during the past 20 years. This infection is an indolent process that potentially requires a full decade, even in the untreated patient, from the time of acute infection before immunosuppression becomes clinically significant. Surgical problems in the HIV patient include abnormal presentation of common illnesses but also include unique problems that are consequences of the acquired immunosuppression.

**Educational aims:**

- Interrogation and clinical inspection of patients with SIRS, sepsis.
- To determine the etiologic and pathogenic factors of SIRS, sepsis.
- To develop a plan of examination of the patients with SIRS, sepsis.
- To estimate results of laboratory data.
- To make and formulate a diagnosis of SIRS, sepsis.
- To estimate efficiency of treatment and prognosis of disease.
- The stages of AIDS, clinical picture, diagnosis, surgical aspects.

**A student must know:**

- Etiology and pathogenesis of SIRS, sepsis.
- Manifestation of SIRS, sepsis.
- Modern methods of diagnosis of SIRS, sepsis.
- Significance in different diseases and traumas
- The choice of diagnostic and curative tactics.

- Algorithm of intensive therapy implementation.
- Surgical aspects of AIDS.

**A student must be able to:**

- Collect and estimate anamnesis of the patients with SIRS, sepsis.
- Use methods of examination patients with SIRS, sepsis.
- Set the algorithm of examination patient with SIRS, sepsis.
- Estimate the results of laboratory, instrumental inspection of patients.
- Determine optimum medical tactics for a concrete patient (conservative, surgical), and in the case of SIRS, sepsis, with AIDS complicated surgical pathology.
- To prove necessity of pre-operation preparation of patient and to define the operation of choice depending on localization, age, sex.

**Terminology.**

| Term | Definition |
|---|---|
| Infection | is the pathological reaction in which fluid and circulating leukocytes accumulate in extravascular tissues in response to injury or infection, typically originating from a detectable source of microbial harm. |
| SIRS | involves highly complex processes with multiple cell types and numerous humoral mediators. SIRS is diagnosed when two or more of the following criteria are met:<br>Temperature ≥38°C or ≤36°C,<br>Heart rate ≥90 beats/min,<br>Respiratory rate ≥20 breaths/min or PaCO2 ≤32 mm Hg or mechanical ventilation,<br>White blood cell count ≥12,000/L or ≤4000/L or ≥10% band forms |
| Sepsis | refers to SIRS caused by infection, with both an identifiable source of infection and SIRS present |
| Severe sepsis | is characterized as sepsis (defined above) combined with the presence of new-onset organ failure |
| Septic shock | is a state of acute circulatory failure identified by the presence of persistent arterial hypotension (systolic blood pressure <90 mm Hg) despite adequate fluid resuscitation, without other identifiable causes (sepsis + cardiovascular collapse (requiring vasopressor support)) |
| HIV | is a blood-borne infection that is transmitted to the susceptible host after percutaneous or a mucus membrane exposure to infected blood or body fluids. |
| AIDS | is infections with HIV leading to acquired immune deficiency syndrome |

**Content:**

The systemic response to injury can generally be divided into two phases: (1) a proinflammatory phase, which involves the activation of cellular processes aimed at restoring tissue function and eliminating invading microorganisms, and (2) an anti-inflammatory or counter-regulatory phase, which is crucial for preventing excessive proinflammatory responses and restoring balance in the body. Although the terms used to describe different aspects of systemic inflammation are often used interchangeably, each term has specific criteria that distinguish it from the others.

**Systemic phase of infection: sepsis**

If local circumscription of infection is impossible either by removing the bacteria or by abscess formation, micro-organisms eventually invade the bloodstream and may reach distant organs. The presence of bacteria in the bloodstream (bacteremia) occurs transiently in healthy individuals too. In patients, bacteria often will be found on the intravascular portion of catheters. Nontoxin-producing, mostly nonmultiplying bacteria can sometimes be isolated by blood culture, but these cause no or only mild systemic symptoms. Bacteremia may, however, progress to systemic disease, especially in immunocompromised and postoperative patients. If the condition persists and is associated with multiplication of bacteria in the bloodstream, and large numbers of bacteria die as they are attacked by host defense mechanisms, then large quantities of bacterial cell-wall structures (endotoxins) are liberated and a serious state of infection termed sepsis ensues.

Sepsis is characterized not only by invasion and multiplication in the bloodstream of large numbers of bacteria but also by the potential for subsequent sudden overload of the host with endotoxins and cytokines, leading to septic shock. Key to this process is a small molecule – nitric oxide – that when excessively produced will block mitochondrial energy generation by inhibiting Krebs' cycle enzymes and cells are running out of fuel for life. Clinically we then observe sequential organ dysfunction. Sepsis is the clinical, symptomatic state resulting from the host response to bacteremia. Liberated bacterial exo- and endotoxins are deleterious to many organ functions; equally, cytokine mediators of host defenses are potentially damaging if their downregulation fails. If not treated successfully, the patient may die immediately of septic shock or later following multisystem organ failure: 1 ng endotoxin/kg body wt results in irreversible shock and death within 2 h. Clinical symptoms of sepsis include the following. Fever is usually is high, spiking, and accompanied by chills. Tachycardia accompanies or precedes the fever and is proportional to it. The total leukocyte count may not be particularly abnormal in sepsis and may even be low, due to the consumption of polymorphs. The differential count is more reliable: there is always a shift to the left. Petechial lesions may be seen in the skin or conjunctiva of patients suffering from sepsis caused by streptococci, meningococci, or pseudomonads. Anemia secondary to hemolysis may appear rapidly when sepsis is due to staphylococci, pseudomonads, coliforms, or clostridia. During the initial, hyperdynamic phase of septic shock the peripheral vasodilation is explained by a circulatory response that aims to compensate for the inability of cells to use oxygen. The last stage of septic shock is hypodynamic, due to cell death. Shock is common in sepsis caused by Gram-negative organisms, but occurs relatively less often with Gram-

positive infections. Metastatic abscesses, especially of the bone, brain, or spleen, are not unusual after a septic episode: any injured tissue is easily infected during sepsis. Diagnosis is aided by a high index of suspicion.

Thermolabile exotoxins are released by living bacteria, particularly the Gram-positive; thermostable endotoxins are released by all bacteria after death. Endotoxins are complex moieties of high molecular weight consisting of phospholipids, polysaccharides, and proteins derived from the outer cell wall, particularly of Gram-negative rods such as Escherichia coli. Clinically measurable effects of endotoxin include fever, consumptive coagulopathy, increased vagotonus, hyperglycemia followed by hypoglycemia, leucopenia or leukocytosis, increased plasma lipids, release of hepatic enzymes, thrombocytopenia, and a reduced serum iron. Low doses of endotoxin primarily affect the reticuloendothelial system. Animal studies have shown a marked reduction in the clearance of particulates such as colloidal carbon during endotoxemia. Mediators such as collagenases, pyrogenic prostaglandins, and coagulation factors are released from macrophages; after 7 days, antibodies against endotoxin are produced. Endotoxins act directly on the hypothalamic temperature-regulation center to cause fever, reinforcing the activity of pyrogenic substances released from dying neutrophils. Erythropoiesis is shifted from the bone marrow to the spleen, resulting in leucopenia followed by leukocytosis after 2 to 6 h. In small doses, endotoxins increase phagocytic activity and bacterial killing. Thrombocytopenia, accompanied by aggregation and lysis of thrombocytes, results in the release of ADP, vasoactive amines, histamine, serotonin, and platelet factor III, which in turn may lead to consumptive coagulopathy. In the extrinsic coagulation system, endotoxins cause release of a tissue factor derived from macrophages, as well as platelet factors and thromboplastins. In the intrinsic system, factor XII (Hageman factor) is activated, leading to disseminated intravascular coagulation. Endotoxin has a profound effect on metabolism. Initially it induces hyperglycemia, which is followed after several hours by hypoglycemia. Hyperlipidemia results from altered metabolism of free fatty acids, cholesterol, phospholipids, and triglycerides. Protein synthesis by the liver is stimulated; lactate dehydrogenase, transaminases, and phosphokinases are released, increasing their serum concentrations. Release of adrenocorticotrophic hormone, cortisone, and growth hormone is increased; thyrotropin and luteinizing hormone are not affected. Plasma iron and total iron-binding capacity are reduced. A vagotonic effect results in loss of thirst and appetite, stomach emptying is delayed, and diarrhea may occur.

**Pathogenesis of inflammation**

***Central Nervous System Regulation of Inflammation.*** The central nervous system plays a key role in regulating the inflammatory response through autonomic signaling, which is mainly involuntary. Traditionally, the autonomic system manages functions like heart rate, blood pressure, respiratory rate, gastrointestinal motility, and body temperature. Additionally, the autonomic nervous system regulates inflammation in a reflexive manner, similar to the patellar tendon reflex. Inflammation originating from a specific area sends signals to the hypothalamus, which then rapidly transmits anti-inflammatory messages back to the site of inflammation to reduce the release of inflammatory mediators by immune cells. Research by Tracey and colleagues has

shown that the parasympathetic nervous system, via acetylcholine, plays a role in inhibiting inflammation by reducing tissue macrophage activation. Cholinergic stimulation directly decreases the release of proinflammatory mediators like TNF-α, IL-1, IL-18, and high-mobility group protein (HMG-1) from tissue macrophages, without affecting the anti-inflammatory cytokine IL-10. This effect is supported by the discovery of acetylcholine (nicotinic) receptors on macrophages. Vagal stimulation has a variety of effects, including lowering heart rate, enhancing gut motility, dilating arterioles, and regulating inflammation. Unlike humoral anti-inflammatory mediators that circulate in the bloodstream, vagal nerve signals are targeted directly at the site of injury or infection, acting rapidly in real time. Preclinical studies suggest that impaired cholinergic activity in the vagus nerve could lead to an increased proinflammatory response in critically ill patients.

***Hormonal Response to Injury.*** The hormonal response to injury involves various chemical classes of hormones, including polypeptides (e.g., cytokines, glucagon, insulin), amino acids (e.g., epinephrine, serotonin, histamine), and fatty acids (e.g., glucocorticoids, prostaglandins, leukotrienes). Hormone receptors activate signals through three primary pathways: receptor kinases (such as insulin and insulin-like growth factor receptors), G-protein-coupled receptors (such as those for neurotransmitters and prostaglandins), and ligand-gated ion channels that regulate ion transport. These receptors often use secondary signaling pathways to amplify the initial response. Hormone signals are also mediated by intracellular receptors that bind both to the hormone and the targeted gene sequences in the DNA. These receptors can be located in the cytosol or nucleus. A key example is the glucocorticoid receptor, which is typically bound to heat shock proteins (HSP) in the cytosol. When glucocorticoid binds to the receptor, the HSP dissociates, activating the receptor-ligand complex, which then moves to the nucleus. Almost all hormones of the hypothalamic-pituitary-adrenal (HPA) axis affect the body's response to injury and stress, with some directly influencing the inflammatory response and having immediate clinical impact.

**Mediators of Inflammation**

***Cytokines*** are among the most powerful mediators of the inflammatory response. When acting locally at the site of injury or infection, they help eliminate invading microorganisms and facilitate wound healing. However, an excessive production of proinflammatory cytokines in response to injury can lead to hemodynamic instability (e.g., septic shock) or metabolic disturbances (e.g., muscle wasting). If left unchecked, these exaggerated responses can result in end-organ failure and death. Anti-inflammatory cytokines, which are part of the inflammatory cascade, counteract the excessive effects of proinflammatory cytokines. Nevertheless, inappropriate release of anti-inflammatory mediators can compromise the immune system, making the patient more vulnerable to severe infections.

***Heat Shock Proteins*** are produced in response to various stressors such as hypoxia, trauma, heavy metals, and hemorrhage. HSPs are intracellular proteins that act as protectors against the harmful effects of traumatic stress. The synthesis of HSPs is triggered by gene induction through the heat shock transcription factor.

***Reactive oxygen metabolites*** which are short-lived and highly reactive molecules, contribute to tissue injury by oxidizing unsaturated fatty acids in cell membranes.

These oxygen radicals are generated during processes involving anaerobic glucose oxidation and the reduction of oxygen into superoxide anions. The superoxide anion is further converted into other reactive species like hydrogen peroxide and hydroxyl radicals. Activated leukocytes are potent producers of reactive oxygen metabolites, which can damage cells. However, cells are generally protected from their own reactive oxygen species by antioxidants such as glutathione and catalases.

***Kallikrein-Kinin System*** involves the production of bradykinins, which are potent vasodilators. These are generated through the breakdown of kininogen by the serine protease kallikrein. Prekallikrein, the inactive form of kallikrein, is activated by factors like Hageman factor, trypsin, plasmin, and certain surfaces. Kinins increase capillary permeability and tissue edema, cause pain, inhibit gluconeogenesis, and promote bronchoconstriction. They also contribute to renal vasodilation, reducing renal perfusion pressure and triggering renin formation. This, in turn, activates sodium and water retention through the renin-angiotensin system.

***Serotonin*** (5-hydroxytryptamine, 5-HT), a neurotransmitter derived from tryptophan, is found in chromaffin cells of the intestine and in platelets. It is often secreted in excess in patients with midgut carcinoid tumors. Serotonin causes vasoconstriction, bronchoconstriction, and platelet aggregation, and acts as a myocardial chronotrope and inotrope. Although serotonin is released during injury, its exact role in the inflammatory response is not fully understood.

***Histamine***, which is derived from histidine and stored in neurons, skin, gastric mucosa, mast cells, basophils, and platelets, is released when calcium levels increase. There are two types of histamine receptors: H1 and H2. H1 receptor activation leads to bronchoconstriction, increased intestinal motility, and enhanced myocardial contractility, while H2 receptor activation inhibits histamine release. Both receptor types contribute to hypotension, blood pooling, increased capillary permeability, decreased venous return, and myocardial failure. Elevated histamine levels have been observed in conditions such as hemorrhagic shock, trauma, thermal injury, endotoxemia, and sepsis.

**Pathogens in surgical infections**

This overview of pathogens responsible for surgical infections is not a complete review, but rather it highlights key distinctions and classifications that help organize the vast body of information related to bacterial flora in surgical infections and the antibiotic susceptibility patterns of these pathogens. Pathogenic bacteria in surgical infections are generally categorized into two groups: aerobic and facultative bacteria, and anaerobic bacteria. Further classifications include gram-positive and gram-negative bacteria, as well as bacilli (rods) and cocci. The majority of surgical infections are caused by endogenous bacteria. Specific bacteria reside in specific parts of the body, and during a surgical procedure, the exposed anatomical areas are typically the source of microorganisms that cause infection. Familiarity with the body's normal microbial flora is crucial, as it helps direct prophylactic antibiotic use, initiate empirical therapy, and identify the origin of infections, particularly when blood cultures are positive. Understanding bacterial classifications is also helpful, as it may take up to 72 hours to obtain a final culture result for a specific bacterium; however, Gram staining

and biochemical tests can provide early indications regarding which group of bacteria may be responsible for an infection.

Among the ***Gram-positive cocci*** relevant to surgeons, staphylococci and streptococci are of major importance. Staphylococci are divided into coagulase-positive and coagulase-negative strains. Coagulase-positive staphylococci, notably S. aureus, are the most common pathogens in wounds and incisions that are not contaminated with endogenous bacteria. These bacteria are typically resistant to penicillin, requiring treatment with penicillinase-resistant antibiotics. Extensive use of these antibiotics has led to the emergence of methicillin-resistant Staphylococcus aureus (MRSA), which, while not more intrinsically pathogenic than other staphylococci, is more difficult to treat due to antibiotic resistance. MRSA prevalence varies by region but has been rising in the past two decades, now appearing more often in community-acquired infections. Patients from long-term care facilities, those with a history of hospitalization, antibiotic treatments, diabetes, or dialysis are at higher risk. MRSA treatment typically involves vancomycin, quinupristin/dalfopristin, or linezolid. There has also been the emergence of S. aureus strains with reduced susceptibility to vancomycin, and more recently, strains with high-level resistance. This trend suggests that the number of such strains will increase in the future.

Coagulase-negative staphylococci, once considered contaminants and harmless skin flora, can cause serious infections in the right clinical context, especially in patients who have been compromised by trauma, extensive surgery, or metabolic disease, and who have invasive vascular devices. These staphylococci are the leading cause of nosocomial bacteremia and are often associated with infections of intravascular devices. They can also cause endocarditis, prosthetic joint infections, vascular graft infections, and postsurgical mediastinitis. Most coagulase-negative staphylococci are methicillin-resistant, and while many infections linked to intravascular devices can be resolved by simply removing the device, empiric antibiotic therapy should include vancomycin, quinupristin/dalfopristin, or linezolid.

The streptococcal species include β-hemolytic streptococci (notably group A or S. pyogenes), S. pneumoniae, and other α-hemolytic streptococci. While these species were once uniformly sensitive to penicillin G and other β-lactam antibiotics, penicillin-resistant S. pneumoniae is now common in urban areas. β-hemolytic streptococci, though not frequently recovered from soft tissue wounds, can cause life-threatening infections. Other α-hemolytic streptococci or viridans streptococci, which are commonly found in mucous membranes and skin, may be present in the peritoneal cavity following upper gastrointestinal perforations, but are rarely the sole cause of significant surgical infections.

Enterococci (group D streptococci) are often recovered as part of a mixed flora in intra-abdominal infections, though they are rarely found as the sole cause of surgical infections. Enterococcal bacteremia associated with surgical infections generally carries a poor prognosis. The significance of enterococci in surgical infections has garnered attention due to their relative resistance to antibiotics. No single antibiotic effectively eradicates deep-seated infections or bacteremia caused by enterococci. The most effective treatment involves a combination of gentamicin with either ampicillin (or another advanced-generation penicillin) or vancomycin.

***Aerobic and Facultative Gram-Negative Rods.*** A wide range of gram-negative rods are involved in surgical infections, primarily from the Enterobacteriaceae family, which includes facultative anaerobic bacteria such as Escherichia, Proteus, and Klebsiella. These genera, known as "easy gram-negative rods," are common in mixed surgical infections and are generally sensitive to a broad spectrum of antibiotics, especially second-generation cephalosporins. Other genera within Enterobacteriaceae, such as Enterobacter, Morganella, Providencia, and Serratia, are also found in surgical infections but tend to exhibit greater intrinsic resistance to antibiotics, categorized as "difficult gram-negative rods."

Empiric treatment for infections caused by these more resistant organisms typically involves third-generation cephalosporins, expanded-spectrum penicillins, monobactams, carbapenems, quinolones, or aminoglycosides. In some areas, these bacteria have developed extended-spectrum β-lactamase (ESBL) enzymes that can deactivate third-generation cephalosporins, making them harder to treat. Infections caused by these organisms are more frequently associated with hospital-acquired and postoperative infections. In contrast, gram-negative rods from community-acquired infections, such as uncomplicated appendicitis or diverticulitis, are less likely to exhibit antibiotic resistance.

Obligate aerobic gram-negative rods like Pseudomonas and Acinetobacter species can also be found in surgical infections, particularly in hospital-associated pneumonias in surgical patients, but also in the peritoneal cavity or severe soft tissue infections. These organisms are often resistant to antibiotics and require treatment with specific antipseudomonal antibiotics, such as ceftazidime, cefepime, aztreonam, imipenem/cilastatin, meropenem, ciprofloxacin, acylureido-penicillin, or aminoglycosides. However, Acinetobacter species are resistant to aztreonam, and some strains exhibit resistance to even the most effective antibiotics. Empirical treatment often involves using two antibiotics until susceptibility testing results are available, as this approach ensures that at least one effective drug will remain available if resistance develops.

Stenotrophomonas maltophilia, previously known as Pseudomonas or Xanthomonas maltophilia, is uniformly resistant to imipenem and meropenem and is frequently encountered as an emerging pathogen when these carbapenems are used empirically for treating serious infections.

***Anaerobic bacteria*** are the most numerous inhabitants of the gastrointestinal tract, including the mouth. The most common anaerobic isolate in surgical infections is Bacteroides fragilis. Other common anaerobic species with significant resistance to many β-lactam antibiotics include Bacteroides thetaiotaomicron. The most effective antibiotics for these species are metronidazole, clindamycin, chloramphenicol, imipenem, meropenem, and ertapenem, as well as combinations of penicillin and a β-lactamase inhibitor (e.g., ticarcillin/clavulanate, ampicillin/sulbactam, and piperacillin/tazobactam).

Other anaerobic species commonly recovered from surgical infections, though with less significant resistance patterns, include Bacteroides melaninogenicus and many anaerobic cocci. Clostridium species are also important anaerobic pathogens in surgical infections, especially in necrotizing soft tissue infections. These gram-

positive, spore-forming rods, while capable of surviving for some time in the presence of oxygen, require an anaerobic environment to grow, invade, and produce the toxins responsible for their virulence. In infections, Clostridium species do not form spores, so gram-stained material from a soft tissue infection shows only gram-positive rods without spores. Notable species include Clostridium difficile (which causes pseudomembranous colitis) and Clostridium tetani (which causes tetanus).

The prevention of tetanus relies solely on active and passive immunization, not antibiotic treatment. Anaerobic bacteria are particularly relevant in surgical infections because they thrive in environments with low oxidation-reduction potential, conditions incompatible with the survival of healthy tissue. The presence of anaerobes in soft tissue infections or in the blood suggests that these bacteria are multiplying in necrotic tissue. The gastrointestinal tract is the primary source of anaerobic bacteria, so infections involving anaerobes typically indicate a breach in the integrity of the gastrointestinal tract. Such conditions require surgical correction, making surgical intervention essential for most anaerobic infections, excluding lung abscesses. Anaerobic bacteremia, in particular, necessitates a search for abscesses or enteric lesions that require surgical treatment.

**Diagnosis**

***History and physical examination*** The early accurate diagnosis of surgical infections is essential: delayed treatment can result in dissemination, verwhelming sepsis, and multisystem organ failure. The history and physical examination are the surgeon's most important diagnostic tools. The classic signs of tumor, rubor, calor, dolor and functio laesa are indicative of localized surgical infections. Clinical symptoms of systemic sepsis include disturbed sensorium, tachypnea, tachycardia, hypotension, fever, oliguria, and high-output heart failure. In postoperative patients, the sudden appearance of tachypnea and hypotension suggests Gram-negative sepsis. This condition has a potential mortality of 30 to 50 per cent, but early diagnosis and treatment markedly improves the chances of survival. The entire body must be examined; all dressings should be removed. Inspection and palpation of a suspicious area may reveal the first three of the classical signs of infection. Removal of the dressing around an intravenous cannula may reveal purulent drainage or thrombophlebitis. Rectal examination may show tenderness and induration as signs of a developing pelvic abscess. Auscultation of the chest may reveal the presence of pneumonia before it is evident on a chest radiograph. The patient should be examined for clues to the source of the infection, such as pain or redness in the surgical wound or at an intravenous infusion site, or purulent sputum, cough, pleuritic pain, rales, or dullness in the chest, diarrhea, dysuria, or flank pain. A foul-smelling odor may lead to the site of an anaerobic infection. Pain in the shoulder and an immobile diaphragm suggest a subphrenic abscess. A pelvic or prostatic mass on rectal examination may indicate an abscess, and headache or nuchal rigidity may indicate an infection of the central nervous system.

***Hematology, urinalysis, and radiology***Most bacterial infections produce leukocytosis and, more importantly, a shift to the left in the differential count or a relative lymphopenia. This increase in the proportion of the more immature forms of polymorphonuclear leukocytes may signal infection before an abnormal total leukocyte

count is evident. The differential count may also reveal lymphocytosis in viral infections, monocytosis in tuberculosis, eosinophilia in parasitic infections or hypersensitivity reactions (drug allergy), and toxic degranulation of leukocytes in acute bacterial infection. A low white count or a leukemoid response (a total white count of over 25 000 cells/mm ) may be seen in sepsis in general, and in pneumococcal pneumonia, liver abscess or cholangitis, infected pancreatic necrosis, necrotic bowel, or retroperitoneal phlegmon in particular. Leukopenia is a sign of overwhelming bacterial infection and carries a bad prognosis. Viral infection, typhoid perforation of the bowel, or tuberculosis may also present with leukopenia. Anemia may be associated with infection caused by bacteria, such as Clostridium perfringens, group A streptococci, or coagulase-positive staphylococci, that produce hemolytic enzymes. Routine chest films may reveal generalized or focal atelactasis, or may indicate intra-abdominal infection through signs of gastrointestinal leakage or free air identified under the diaphragm. In the investigation of patients with suspected intra-abdominal infection, flat, upright, and decubitus films may reveal a localized air–fluid level, suggesting an intra-abdominal abscess, or a spreading air-bubble pattern suggestive of infection with a gas-producing organism. Specialized radiologic procedures may be helpful in confirming the diagnosis of intra-abdominal abscess. These studies include ultrasonography and computed tomography (CT). Although a gallium scintiscan may be helpful in special circumstances, this examination is subject to appreciable error and is difficult to interpret in a patient who has had a recent operation.

***Bacteriology*** Observation of exudates and secretions such as wound drainage, urine, and sputum for odor, color, and consistency may be useful in diagnosis. Grape-like odors occur with pseudomonal infections, urea-like odors with Proteus infections, and feculent odors with anaerobic organisms such as Bacteroides, fusobacteria, clostridia, and peptostreptococci. A Gram stain offers the earliest clue to the cause of an infection, particularly when a specific monobacterial infection is suspected. Since surgical infections are mostly due to multiple infecting organisms that are obligate or facultative anaerobes, the Gram stain usually shows a variety of pathogenic bacteria. Note should be taken of the numbers of polymorphonuclear leukocytes on the slide (few, many, loaded) and whether organisms can be seen inside them. Acid-fast and fungal stains can be used if such infections are likely.

***Technique of obtaining the specimen*** Purulent material from the deepest aspect of the wound should be aspirated into a syringe and any air evacuated. Pus is the best medium in which to preserve bacteria for transport to the laboratory. The capped syringe is sent for aerobic and anaerobic culture, and for assay of antibiotic sensitivity. Alternatively, a moist swab can be used to obtain bacteria from a site of suspected infection. Ideally, anaerobic specimens should be transported immediately in a CO - filled tube and plated within 1 h of 2 sampling; fastidious organisms may otherwise die, resulting in a false-negative culture result. If the specimen is held overnight, it should be placed in an anaerobic sterile vial or tube; under no circumstances should an anaerobic specimen be refrigerated. Generally speaking, E. coli (aerobe) and Bacteroides fragilis (anaerobe) are the usual causes of wound infection following gastrointestinal or gynecologic operations, while streptococci, staphylococci, and peptostreptococci are the usual causative organisms when intra-abdominal viscera have

not been resected or opened. Blood cultures are helpful in guiding the specific antibiotic therapy of serious infections if the empirically started, initial antibiotics fail. Following careful disinfection of the venepuncture site with an iodophor preparation, blood samples should be obtained for aerobic and anaerobic culture. Blood should not ordinarily be drawn for culture through an existing intravenous needle or catheter. It is important to obtain a number of blood cultures from different sites and at different times. Once the patient chills and a fever spike is observed, most bacteria have already been killed by host defense mechanisms and blood cultures will be negative. It is possible, however, to predict the time of the next bacteremic episode, because fever spikes occur intermittently. Drawing four blood samples at hourly intervals before the next peak will increase the likelihood of a positive culture. If the patient is receiving treatment with antimicrobial drugs, a drug-removing device is helpful in obviating antimicrobial action during culture.

Sensitivity tests need to be interpreted appropriately and with caution since they are not always reproducible and are an oversimplification of the complex foundations upon which antimicrobial chemotherapy is based. Disc diffusion tests are highly sensitive to small technical and environmental changes. Their results may not correlate well with the actual minimal inhibitory or bactericidal concentration of an antibiotic, or with the concentration of antibiotic achieved at the site of infection with the chosen dosage. While important for epidemiologic purposes, routine disc sensitivity tests are generally of little value in guiding an individual patient's antibiotic therapy. The minimal inhibitory concentration (MIC), or the minimal bactericidal or fungicidal concentrations, are more useful clinically, because antimicrobial dosing can be adjusted to achieve and sustain antibiotic concentrations at the focus that are three to four times in excess of the concentration required to kill bacteria in the test-tube.

**Therapy of surgical infections**

All wounds, whether made at the operating table or resulting from trauma, expose normally sterile tissue and provide an environment for bacterial growth. Infections can be minimized if wound management follows these principles.

- Tissue should be handled gently, and operative trauma kept at a minimum.
- Further contamination should be minimized by use of aseptic techniques.
- Devitalized tissue, debris, and traumatic foreign bodies should be removed.
- Complete hemostasis should be achieved.
- Blood supply is essential for healing and should not be impaired.
- Formation of dead space should be avoided during closure.
- The wound should be closed by layer-to-layer approximation without tension.
- Operative time should be kept to a minimum to reduce the numbers of bacteria entering the wound.
- The wound may be irrigated with liberal amounts of sterile saline/Ringer's lactate solution prior to closure.

Basic understanding of how the body defends itself against infection is essential to a rational application of surgical and other therapeutic principles to the control of infection.

**General principles of therapy**

The goal of antibiotic therapy is to achieve concentrations at the site of infection that exceed the minimum inhibitory concentration (MIC) for the pathogens causing the infection. In cases of mild infections, such as those treated on an outpatient basis, oral antibiotics can often be effective if appropriate choices are available. However, in severe surgical infections, the systemic response to infection may impair gastrointestinal absorption of antibiotics, making oral therapy unreliable. Additionally, in intra-abdominal infections, gastrointestinal function is often compromised. Therefore, initial antibiotic therapy for surgical infections is typically started intravenously to ensure reliable drug delivery.

Each patient with a serious infection must be evaluated daily (or more frequently) to assess their response to treatment. If no clear improvement is observed within 2 to 3 days, it is common to reconsider the treatment strategy. The key question to ask is: Why is the patient failing to improve? Possible reasons include:

- The initial surgical procedure was inadequate.
- The initial procedure was adequate, but a complication has developed.
- A superinfection has occurred at a new site.
- The drug dosage is insufficient.
- A different or additional drug is needed.

Unless the initial choice of antibiotics was inappropriate (e.g., failure to provide coverage for anaerobes in intra-abdominal infections), the choice of antibiotics is generally not the main cause of treatment failure. It is essential to address the underlying reason for lack of improvement before switching antibiotics.

**Duration of Antibiotic Therapy**

Once the patient shows signs of clinical improvement, the decision to stop antibiotics must be made. While there is no universally recommended duration for antibiotic treatment in surgical infections, the general approach is to support local host defenses until they can control the infection. In cases where an abscess has been drained, antibiotics help prevent the spread of infection in the newly opened tissue planes. Typically, after 3 to 5 days of antibiotic therapy, the local inflammatory response (e.g., new capillary formation and infiltration by immune cells) is sufficient to control the infection.

For deep-seated or poorly localized infections, a longer course of antibiotics may be necessary. A general guideline is to continue antibiotics until the patient has shown clinical improvement, which may include:

- Improved mental status
- Return of normal bowel function
- Resolution of tachycardia
- Spontaneous diuresis

Additionally, a normal body temperature for 48 hours or more is often considered a sign of improvement.

Although shorter courses of antibiotics may sometimes be sufficient, there is no definitive data to support a specific ideal duration. With the advent of potent systemic antibiotics that can be administered orally, studies have shown that patients with intra-

abdominal or other severe infections may be treated initially with intravenous antibiotics and then switched to oral antibiotics to complete the course. This approach can reduce overall treatment costs but also carries the risk of unnecessarily prolonging the duration of antibiotic therapy.

In some cases, physicians may be tempted to discharge patients with oral antibiotics, simply because it is easier, when previously they would have been sent home without antibiotics. However, this temptation should be avoided to ensure appropriate management of the infection.

**White Blood Cell Count and Antibiotic Therapy**

The white blood cell count (WBC) may not return to normal after stopping antibiotics, but it can provide important information about the patient's condition:

- Normal WBC: If the WBC normalizes, the likelihood of further infections is low.
- Elevated WBC: If the WBC is elevated, new or ongoing infections may be present. However, continuing antibiotics usually does not prevent new infections. The best approach is to stop the current antibiotics and closely monitor the patient for further developments.

When selecting an antibiotic for empiric treatment, the following guidelines should be followed:

- Coverage of presumed microorganisms: Start with broad-spectrum antibiotics that can later be narrowed down based on the specific microorganism isolated. Anaerobic antibiotics should be avoided unless necessary, as these bacteria play an essential role in maintaining the gastrointestinal microenvironment.
- Antibiotic penetration to the infection site: The antibiotic chosen should be able to reach the infection site. For urinary tract infections (UTIs) and cholangitis, antibiotics with high renal and biliary concentrations, respectively, should be selected. Skin, lungs, and central nervous system tissue concentrations should be considered for infections in these areas.
- Toxicity: Toxicity, especially in critically ill patients, should be considered. These patients may have unpredictable bioavailability and therapeutic/toxic level ranges. When using an antibiotic with significant toxic side effects, close monitoring of blood levels and organ function is essential.
- Aggressive dosing: Whenever an antibiotic regimen is initiated, it should be dosed aggressively. The volume of redistribution in these patients is unpredictable, especially as they often receive aggressive fluid resuscitation and support.
- Time limit for antibiotic therapy: Set a clear time limit for how long antibiotics will be administered.

A superinfection are new infections that develop during antibiotic treatment for the original infection. Antibiotics exert selective pressure on the patient's endogenous flora and on exogenous bacteria colonizing vulnerable sites. Resistant bacteria that remain can then become pathogens in superinfections. Respiratory tract infections are common superinfections during intra-abdominal infections, with the risk of pneumonia increasing with the severity of the abdominal infection.

In hospitalized patients, superinfections occur in 2% to 10% of those receiving antibiotics, depending on underlying risk factors. The best preventive measure is to limit the dose and duration of antibiotics to what is absolutely necessary and to remain vigilant about the possibility of superinfections.

The use of broad-spectrum antibiotics in recent decades has led to an increasing incidence of fungal superinfections. Antibiotic-associated colitis, caused by "C. difficile", is a significant concern in hospitalized patients. It can range from mild and self-limited to rapidly progressive, leading to death. The key to managing C. difficile colitis is early recognition. Diagnosis is best confirmed by detecting C. difficile toxin in stool. In severe cases, endoscopy can reveal characteristic mucosal changes. Treatment includes supportive care with fluids and electrolytes, discontinuation of the offending antibiotic (if possible), and oral metronidazole. Vancomycin should be reserved for metronidazole-resistant cases. In rare instances, emergency colectomy may be needed when the colitis is unresponsive to medical treatment.

Antibiotic resistance is an escalating issue, especially in ICU patients. It leads to prolonged hospital stays, increased healthcare costs, and, most importantly, higher morbidity and mortality from infections that are not adequately treated. Resistance can be classified into two types:

1. Intrinsic resistance: When a species is inherently resistant to a particular antibiotic (e.g., gram-negative bacteria to vancomycin).

2. Acquired resistance: When the genetic composition of a bacterium changes, either through intrinsic changes or by acquiring resistance genes from other bacteria. This can involve:

- Decreased intracellular antibiotic concentration due to decreased influx or increased efflux (e.g., Pseudomonas/Enterobacteriaceae to β-lactams).
- Neutralization by inactivating enzymes (most common mechanism, especially for β-lactam antibiotics).
- Alteration of the antibiotic's target site, affecting all antibiotics (e.g., pneumococcus to penicillin or MRSA to all β-lactam antibiotics).
- Complete elimination of the target by creating new metabolic pathways (e.g., VRE).

Antibiotic resistance is primarily mediated by plasmids, which carry genes for resistance. These plasmids can be exchanged between bacteria, leading to horizontal gene transfer. Resistance mechanisms can increase the ability of bacteria to survive even in the presence of antibiotics.

Risk factors for antibiotic resistance include:

- Antibiotic use
- Prolonged hospital stays
- Broad-spectrum antibiotics
- Use of invasive devices (e.g., endotracheal tubes, central lines, Foley catheters)
- Outbreaks of resistant bacteria

ICU patients are at the highest risk of antibiotic resistance, which correlates with higher mortality rates. Preventive strategies involve guidelines for antibiotic use,

narrow-spectrum antibiotics, and non-antibiotic strategies like infection control practices and hand hygiene. The battle against antibiotic resistance requires a multidisciplinary approach, including the development of new antibiotics and stringent infection control practices.

**Empiric Antimicrobial Therapy in Severe Sepsis and Septic Shock**

Numerous studies highlight the significance of empiric antimicrobial therapy in patients with severe sepsis syndrome, especially those who develop bacteremia. This therapy, combined with fluid resuscitation, metabolic support, and control of infection at the site of origin, plays a critical role in preventing secondary bacteremic events. The use of institutional and unit-specific sensitivity patterns, along with knowledge of likely pathogens, is essential for selecting the right antibiotic. Retrospective studies have shown that appropriate antimicrobial therapy is linked to a two- to three-fold reduction in mortality rates.

In recent years, several new treatments for patients with severe sepsis or septic shock have been demonstrated to offer substantial benefits. One key area of focus has been the use of corticosteroids in patients with septic shock. Corticosteroid therapy was initially investigated in the late 1980s and early 1990s but failed to show benefit. However, recent research has renewed interest in corticosteroids for septic shock, particularly following the discovery that many patients in this condition experience adrenal insufficiency.

Randomized controlled trials have confirmed that replacement doses of corticosteroids are beneficial in patients with severe shock. Currently, the recommended approach is to administer low-dose hydrocortisone (100 mg every 8 hours) to patients with septic shock after conducting a corticotropin stimulation test. The test involves measuring the baseline cortisol level, followed by administering 250 μg of corticotropin intravenously, and assessing the cortisol level one hour later. Adrenal insufficiency is diagnosed if the baseline cortisol level is less than 30 μg/dL or if the cortisol increase after stimulation is less than 9 μg/dL. If normal adrenal function is confirmed, low-dose steroid therapy should be discontinued.

**Nutrition in the surgical patient**

The goal of nutritional support in surgical patients is to prevent or reverse the catabolic effects of disease or injury. Various biological parameters are used to assess the efficacy of nutritional regimens, but the ultimate validation comes from clinical outcomes and the restoration of function. Ensuring optimal nutritional support is critical for improving recovery, particularly for patients who have undergone surgery or are experiencing significant stress due to illness or injury

***Rationale for Parenteral Nutrition*** The principal indications for parenteral nutrition are found in seriously ill patients suffering from malnutrition, sepsis, or surgical or accidental trauma, when use of the gastrointestinal tract for feedings is not possible. In some instances, intravenous nutrition may be used to supplement inadequate oral intake. The safe and successful use of parenteral nutrition requires proper selection of patients with specific nutritional needs, experience with the technique, and an awareness of the associated complications. As with enteral nutrition, the fundamental goals are to provide sufficient calories and nitrogen substrate to promote tissue repair and to maintain the integrity or growth of lean tissue mass. Listed

below are situations in which parenteral nutrition has been used in an effort to achieve these goals:

- Newborn infants with catastrophic gastrointestinal anomalies, such as tracheoesophageal fistula, gastroschisis, omphalocele, or massive intestinal atresia.
- Infants who fail to thrive due to gastrointestinal insufficiency associated with short bowel syndrome, malabsorption, enzyme deficiency, meconium ileus, or idiopathic diarrhea.
- Adult patients with short bowel syndrome secondary to massive small bowel resection (<100 cm without colon or ileocecal valve, or <50 cm with intact ileocecal valve and colon).
- Enteroenteric, enterocolic, enterovesical, or high-output enterocutaneous fistulas (>500 mL/d).
- Surgical patients who experience prolonged paralytic ileus following major surgeries (lasting more than 7 to 10 days), multiple injuries, blunt or open abdominal trauma, or those with reflex ileus due to various medical diseases may require nutritional support.
- Individuals with normal bowel length but experiencing malabsorption due to conditions like sprue, hypoproteinemia, enzyme or pancreatic insufficiency, regional enteritis, or ulcerative colitis.
- Adult patients with functional gastrointestinal disorders, such as esophageal dyskinesia following a cerebrovascular accident, idiopathic diarrhea, psychogenic vomiting, or anorexia nervosa.
- Patients with granulomatous colitis, ulcerative colitis, or tuberculous enteritis, where major portions of the absorptive mucosa are diseased.
- Patients with malignancies, with or without cachexia, where malnutrition could hinder effective treatment options.
- Those who have had unsuccessful attempts to provide adequate nutrition via enteral tube feedings or have high residuals.
- Critically ill patients who are hypermetabolic for more than 5 days or in cases where enteral nutrition is not feasible.

Certain conditions make parenteral nutrition inappropriate. These include:

- Lack of a specific goal for patient management, especially in cases where life extension is futile.
- Periods of hemodynamic instability or severe metabolic imbalances, such as severe hyperglycemia, azotemia, encephalopathy, hyperosmolality, and electrolyte disturbances, which need to be corrected before attempting hypertonic intravenous feeding.
- Patients with functional gastrointestinal tracts where feeding is feasible, as this is generally the best route to provide nutrition.
- Patients with good nutritional status.
- Infants with less than 8 cm of small bowel, as these infants rarely adapt sufficiently to parenteral nutrition.
- Irreversibly decerebrate or dehumanized patients.

Rationale for Enteral Nutrition Enteral nutrition is generally preferred over parenteral nutrition due to its lower cost and fewer associated risks. Laboratory studies have shown that direct nutrient contact in the intestinal lumen helps reduce intestinal mucosal atrophy compared to parenteral or no nutritional support. Studies comparing postoperative enteral and parenteral nutrition in patients undergoing gastrointestinal surgery have shown fewer infections and better acute-phase protein production with enteral feeding. However, randomized studies involving patients with adequate nutritional status (albumin ≥ 4 g/dL) undergoing gastrointestinal surgery found no significant differences in outcomes or complications when enteral nutrition was compared to maintenance intravenous fluids in the initial postoperative days.

Meta-analysis of critically ill patients shows a 44% reduction in infectious complications in those receiving enteral nutrition compared to those on parenteral nutrition. Most studies on severe abdominal and thoracic trauma demonstrate that early enteral nutrition significantly reduces infectious complications compared to no feeding or parenteral nutrition. However, in studies for closed-head injury patients, no significant differences were found between early jejunal feeding and other nutritional support methods.

**Recommendations for Enteral Nutrition**

For surgical patients with moderate malnutrition (albumin 2.9 to 3.5 g/dL), evidence for early enteral nutrition is limited. In these cases, enteral nutrition should be considered based on the patient's measured energy expenditure and any complications that might affect recovery, such as anastomotic leaks, sepsis, or failure to wean from the ventilator. Enteral nutrition is particularly beneficial for patients with permanent neurological impairments, oropharyngeal dysfunction, short bowel syndrome, or those undergoing bone marrow transplantation.

Collectively, the evidence supports the use of early enteral nutrition after major trauma or surgery, especially for patients with anticipated prolonged recovery. Healthy patients without malnutrition undergoing uncomplicated surgery can tolerate 10 days of partial starvation (maintenance intravenous fluids only) without significant protein catabolism. Early intervention is more crucial for patients with poor preoperative nutritional status.

**Initiating Enteral Nutrition**

Enteral nutrition should be initiated as soon as the patient has been adequately resuscitated, which can be determined by the presence of adequate urine output. Bowel sounds and the passage of flatus or stool are not absolute requirements for starting enteral feeding. However, in cases of gastroparesis, feedings should be administered distal to the pylorus. If gastric residuals exceed 200 mL over a 4- to 6-hour period, or if there is abdominal distention, feeding should be paused, and the infusion rate adjusted. Gastric decompression combined with distal small bowel feeding may be appropriate for certain patients, such as those with closed-head injury and gastroparesis.

There is no evidence to support withholding enteral feeding in patients after bowel resection or those with low-output enterocutaneous fistulas (less than 500 mL/day), although low-residue formulas may be preferred. Enteral feeding should also be provided to patients with short-bowel syndrome or clinical malabsorption, but caloric

needs, essential minerals, and vitamins should be supplemented with parenteral nutrition.

**AIDS**

HIV is a blood-borne infection that is transmitted to the susceptible host after percutaneous or a mucus membrane exposure to infected blood or body fluids. The virus attaches to specific receptors on the host CD4 lymphocytes. The virus is internalized with release of the viral RNA. The unique enzyme, reverse transcriptase, of the virus then results in the synthesis of complementary copies of DNA to the RNA template of the virus (cDNA). This cDNA then migrates into the nucleus of the infected cell, is incorporated into the chromosomal configuration of the host cell, and then initiates the synthesis of new viral particles. The viral burden within the infected cell reaches a critical level with lysis of the infected cell and release of viral particles to infect other cells. The result of this process over time is the systematic depletion of CD4 depressor cells with dominance of the CD8 cells and subsequent immunosuppression of the host. The natural history of HIV infection passes through ***four phases.*** First there is the acute viral infection, which includes fever, malaise, pharyngitis, and other symptoms that would be nonspecific features of many viral infections. Second, there is a sustained period of asymptomatic disease. It is during this asymptomatic disease period that active viral replication is occurring in a slow but progressive fashion, which slowly progresses to a state with significant reduction in CD4 cell counts. This indolent second phase is highly variable in different patients and for many patients evolves over a decade or longer. A third phase, which was formerly referred to as AIDS-related complex, represents the first evidence of symptomatic AIDS. The patients present with evidence of regional adenopathy. During this early symptomatic period, the viral load in the patient is increasing and the CD4 count is progressively declining. Clinical AIDS is considered to exist when the patient has an indicator condition or has a CD4 count lower than 200. The indicator conditions may present as problems requiring surgical care, or conventional illnesses may present with an obscure presentation because of the patient's immunosuppressed state, or conventional illnesses may be mistaken for nonsurgical illnesses associated with the primary HIV infection.

***Acute Abdomen.*** The AIDS patient has an increased frequency of the clinical acute abdominal pain syndrome than does the age-matched non-AIDS population. AIDS patients undergo abdominal exploration for a host of different reasons. It is likely that AIDS patients actually have an increased rate of emergency abdominal procedures because they have the anticipated rates of operation for commonly seen indications (e.g., appendicitis) but have indications in addition to those that are specific for this disease. An increased probability of abdominal operation but also increased nonsurgical causes for abdominal pain means that a discriminating evaluation of these patients is always necessary. Acute appendicitis in the AIDS patient occurs due to the conventional occlusion of the appendiceal orifice by a fecalith but also due to occlusion of the orifice by Kaposi's sarcoma lesions and acute CMV infections. Accumulated appendicitis cases in aged patients indicate that 30% are caused by complications of AIDS-related conditions. Clinical presentation for the AIDS patient with appendicitis is with characteristic right lower pain but is commonly associated with normal WBC

counts in most patients. Most have fever, but fever and nonspecific abdominal pain alone are common findings among AIDS patients without surgical illness. Although there is no clear definition in the published literature, there appears to be an increased rate of perforation, gangrenous appendicitis, and initial appendiceal abscess among AIDS patients. Delay in patient presentation because of frequent abdominal pain and fever, and delay by the physician because of the numerous nonsurgical causes of abdominal pain, may account for this apparent observation.

*Perforation of the gastrointestinal tract* not related to appendicitis is certainly increased in the AIDS patient. The mean age of the clinical onset of AIDS is in the late 30s. Other than appendicitis, this age group infrequently has a perforated viscus. AIDS patients have perforation of the gastroduodenum, small bowel, and colon due to CMV infection in particular. The terminal ileum and colon are most common sites for CMV perforations. The diagnosis of CMV perforations is confirmed by identification of the intranuclear inclusion bodies on biopsy specimens from sites of perforation. Appropriate surgical management of the site of perforation requires suture plication of gastroduodenal perforations, resection and anastomosis of small bowel perforations, and colostomy for colonic perforations. Acute antiviral chemotherapy is initiated. CMV perforation is an indicator of advanced HIV disease and carries a grave prognosis owing to death from peritonitis or other AIDS-related complications. Kaposi's sarcoma, gastrointestinal lymphoma, and severe ileocolitis from Mycobacterium avium intracellulare are additional causes of AIDS-related perforations of the gastrointestinal tract. Biopsies of the site of perforation at operation are necessary to establish causation. Management of the perforated site is the same as for any other perforation due to infectious causes.

*Gastrointestinal obstruction* is seen secondary to AIDS-related disease. Causes include gastric outlet obstruction secondary to lymphoma, small bowel obstruction due to mycobacterial disease, intussusceptions secondary to Kaposi's sarcoma, and an Ogilvie-like syndrome progressing to toxic megacolon due to CMV infection. The diagnosis of the AIDS-related events needs to be differentiated for more conventional causes of obstruction. In the usual age group for AIDS patients, particularly when prior abdominal operation and the risk of adhesions are not present, most intestinal obstruction events are AIDS related.

*Gastrointestinal bleeding* is similarly seen by the same array of disease processes that are responsible for perforation and obstruction. When bleeding arises from the gastroduodenum or colon, endoscopy procedures will assist diagnosis. Operation is required only when medical measures to control hemorrhage have failed.

*Hepatobiliary disease* is common in the HIV-infected patient. Chronic hepatitis B and C infections share common routes of transmission with HIV disease. Persistent elevation of hepatic enzymes from chronic hepatitis is common with cirrhosis as a frequent result. Once clinical AIDS has evolved, infection of the liver parenchyma from Candida albicans and M. avium intracellulare result in small hepatic abscesses, which may require liver biopsy for diagnosis. Although infections with Entamoeba histolytica among the male homosexual population with AIDS are common, amebic abscess is much less common.

A particularly interesting but infrequent infectious problem is AIDS-associated *cholangiopathy.* This appears to be the consequence of infection of the actual bile ducts themselves with opportunistic pathogens including Cryptosporidium species, CMV, and Microsporidia. Inflammatory changes secondary to invasion of the ducts result in a sclerosis-like picture. The patients have new-onset right upper quadrant pain, fever, alkaline phosphatase elevations, but rarely jaundice, at the time of initial presentation. Jaundice becomes more of a feature of the disease as the process advances over time. Diagnosis is suggested by ultrasound demonstration of thickened ducts. Endoscopic retrograde cholangiopancreatography is used to culture the bile or obtain biopsies of the bile ducts. Specific antimicrobial chemotherapy is used for treatment. Surgical care for these patients is limited. An occasional patient may develop acute cholecystitis secondary to cystic duct occlusion. Radionuclide scans are used for diagnosis, and cholecystectomy may be necessary in the patient with acute cholecystitis.

*Splenomegaly* is a common finding among AIDS patients but may be the result of multiple causes. Patients may have portal hypertension from severe liver disease or portal fibrosis. Parenchymal infection of the spleen may be secondary to CMV, Microbacterium, Pneumocystis carinii, and other pathogens. Splenic enlargement may be secondary to lymphoma or Kaposi's sarcoma. The patients commonly have left upper quadrant pain and the spleen is palpable and quite tender on physical examination. Splenectomy may infrequently be necessary secondary to spontaneous rupture or to rupture from incidental trauma.

*Vascular infections* are reported among AIDS patients. Some infected pseudoaneurysms are seen among the intravenous drug abuse population with common bacteria (e.g., S. aureus). These infections among the AIDS population are difficult to eradicate. Perhaps more interesting and somewhat unique to the AIDS patient is Salmonella arteritis. AIDS patients have a high incidence of Salmonella infection. Apparently Salmonella has a particular affinity for atherosclerotic plaque. Adherence of the microbe to an atheroma of the distal aorta or iliac arteries can result in invasive infection, pseudoaneurysm formation, and potential rupture. Surgical management prior to rupture is desired. Reconstruction of these patients following resection proceeds along guidelines for management of any mycotic aneurysm infection.

*Neoplasms.* B-cell lymphoma occurs commonly among AIDS patients. These malignancies are commonly undifferentiated and aggressive. Operative intervention for the lymphoma patient is for the purpose of diagnosis of the disease (e.g., needle biopsy). More commonly surgical intervention is for the management of complications secondary to bleeding, obstruction, or perforation of the gastrointestinal tract. Primary management of the lymphoma disease is medical. Kaposi's sarcoma is a neoplasm of the skin that was uncommon until the AIDS epidemic. Kaposi's sarcoma is the result of the patient having chronic infection with human herpesvirus-8, but clinical disease occurs only when the patient's clinical immunosuppression reaches an advanced stage. Kaposi's sarcoma in the AIDS patients occurs at numerous different sites, including skin, gastrointestinal tract, lung, liver, and even the heart. Surgical involvement is primarily for diagnosis and the management of complications, particularly in the gastrointestinal tract. As noted earlier, perforation, bleeding, and small bowel

intussusceptions are noted gastrointestinal complications from Kaposi's sarcoma. Radiation and chemotherapy are the primary treatment modalities for this neoplasm.

*Anorectal Disease.* The immunosuppressed AIDS patient is at increased risk for human papillomavirus infection. Large condylomata acuminata are the result. Very large condylomata commonly need to be surgically reduced to be followed by local topical therapy. Squamous cell carcinoma of the anus occurs with increased frequency presumably due to the role of papillomavirus in causing this disease.

*Occupational Risk of Infection.* A major surgical concern about the HIV-infected patient was the potential of infection being occupationally acquired during the course of providing surgical care for these patients. Surgical exposure to patient blood during the performance of operative procedures has been well documented. With the knowledge that HIV is a blood-borne pathogen and that other blood-borne pathogens (e.g., hepatitis B) have been documented to be transmitted in the operating room, many surgeons have been concerned about this risk.

At this time it can be said that the risk of occupational transmission of HIV disease is low, but it is not zero. As of the last available Centers for Disease Control and Prevention report, 57 documented cases of occupational transmission of HIV have occurred and 138 cases of probable transmission among health care workers have been identified. No documented cases have been seen in surgeons. Most occupational infections have come from major percutaneous injuries from hollow needles. Solid-needle injuries have not been documented to occur in the United States. Current rates of transmission from hollow needles are about 0.2% to 0.3%. Surgeons should feel comfortable in providing care for HIV-infected patients but should use appropriate and standardized safeguards to prevent blood exposure in the care of all patients.

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**Tests**

1. Which of the following surgical interventions is least likely to provide acceptable prolongation of life for patients with AIDS?
   A. Splenectomy for AIDS-related idiopathic thrombocytopenic purpura
   B. Colonic resection for perforation secondary secondary to cytomegalovirus infection
   C. Cholecystectomy for acalculous cholecystitis
   D. Tracheostomy for ventilator dependent patients with respiratory failure
   E. Gastric resection for a bleeding gastric lymphoma or Kaposi’s sarcoma
2. Human immunodeficiency virus (HIV) has been isolated from many body fluids. Which of the following is a major source of transmission?

A. Tears
B. Sweat
C. Semen
D. Urine
E. Breast milk

3. Signs and symptoms associated with early sepsis include
   A. Respiratory acidosis
   B. Decreased cardiac output
   C. Hypoglycemia
   D. Increased arteriovenous oxygen difference
   E. Cutaneous vasodilation
4. A 42-year-old man has a calculated resting energy expenditure of 1800 kcal/day (basal energy expenditure plus 10%). Match the following clinical situations with the appropriate daily energy requirement. In case of sepsis energy expenditure is
   A. 1600
   B. 2300
   C. 2800
   D. 3600
   E. 4500
5. Major alterations in pulmonary function associated with adult respiratory distress syndrome (ARDS) include
   A. Hypoxemia
   B. Increased pulmonary compliance
   C. Increased resting lung volume
   D. Increased functional residual capacity
   E. Decreased dead space ventilation
6. Which statement regarding transmission of viral illness through homologous blood transfusion is true?
   A. The most common viral agent transmitted via blood transfusion in the United States is human immune deficiency virus (HIV)
   B. Blood is routinely tested for cytomegalovirus (CMV) because CMV infection is often fatal
   C. The most frequent infectious complication of blood transfusion continues to be viral meningitis
   D. Up to 10% of those who develop post transfusion hepatitis will develop cirrhosis or hepatoma or both
   E. The etiologic agent in post transfusion hepatitis remains undiscovered
7. Central venous pressure (CVP) may be decreased by
   A. Pulmonary embolism
   B. Hypervolemia
   C. Positive-pressure ventilation
   D. Pneumothorax
   E. Gram-negative sepsis
8. The appropriate antibiotic to prescribe while awaiting specific culture verification is

A. Penicillin
B. Erythromycin
C. Tetracycline
D. Azathioprine
E. Cloxacillin

9. Tissue injury or infection results in the release of tumor necrosis factor (TNF) by which of the following cells?
   A. Fibroblasts
   B. Damaged vascular endothelial cells
   C. Monocytes/macrophages
   D. Activated T lymphocytes
   E. Activated killer lymphocytes
10. Which statement about transmission of HIV in the health care setting is true?
   A. A freshly prepared solution of dilute chlorine bleach will not adequately decontaminate clothing
   B. All needles should be capped immediately after use
   C. Cuts and other open skin wounds are believed to act as portals of entry for HIV
   D. Double gloving reduces the risk of intraoperative needle sticks
   E. The risk of seroconversion following a needle stick with a contaminated needle is greater for HIV than for hepatitis B

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| D | C | E | C | A | D | E | A | C | C |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Inflammation<br>– Pathophysiology of inflammation<br>– Stages of AIDS. | -To represent the methods of HIV diagnosis<br>-To make the flow diagram of mechanisms of sepsis. |
| Study:<br>– Stages of sepsis.<br>– Methods of diagnosis.<br>– Antibiotic resistance | -To conduct differential diagnosis different tapes of SIRS |

## 2.2. Critical conditions in surgical patients. Acute respiratory distress syndrome, abdominal cavity syndrome, collapse. Shock in surgical patients. Causes, diagnosis, treatment tactics.

**Overview.**

Shock remains one of the surgeon's most formidable foes. Sixty years after publication of Alfred Blalock's classic textbook, Principles of Surgical Care: Shock and Other Problems, the diagnosis of shock and the management of shock resuscitation continue to challenge the clinician and investigator. The objective of this guide is to

provide the reader with practical methods for recognizing and resuscitating patients who are in shock.

**Educational aims:**

- Interrogation and clinical inspection of patients with shock.
- To determine the etiologic and pathogenic factors of different forms of shock, abdominal compartment syndrome and acute respiratory distress syndrome.
- To find out the clinical features of the intraabdominal hypertension.
- To develop a plan of examination and management of the patients with shock.
- To estimate results of physical examination, laboratory tests, ECG, ultrasonography and X-ray examination of patients in critical conditions.
- To make a differential diagnosis between different forms of shock.
- To determine the criteria for the diagnosis of acute respiratory distress syndrome.
- To compare the different approaches to the treatment of shock.

**A student must know:**

- Classification of shock.
- Pathogenesis of different kinds of shock.
- Risks of resuscitation of the patient with shock
- The algorithms of management in case of sudden circulatory collapse of a patient in the operating room, immediate, intermediate and late postoperative period.
- Factors influencing intraabdominal pressure
- Mechanisms of the development of the intraabdominal hypertension and ACS.
- Different causes of circulatory collapse.
- Pathogenesis of ARDS.
- The optimal ventilator strategy for ARDS
- Predisposing conditions to ACS
- Grading of ACS

**A student must be able to:**

- Prescribe the investigations to the patients in critical conditions.
- Define the rational quantity of laboratory and instrumental research methods.
- Be able to examine patients with shock, ARDS and ACS.
- Define the indications to resuscitation of the patients with shock.
- To prescribe the treatment to the patients with ARDS.
- To recognize the clinical signs of ACS.
- To differentiate the types of ACS and prescribe appropriate treatment.

**Terminology**

| Term | Definition |
|---|---|
| ARDS | Acute Respiratory Distress Syndrome |
| PAWP | Pulmonary artery wedge pressure |

| | |
|---|---|
| $PaO_2$: $FiO_2$ | An index of arterial oxygenation efficiency that corresponds to ratio of partial pressure of arterial $O_2$ to the fraction of inspired $O_2$ |
| Circulatory collapse | is defined as a general or specific failure of the circulation, either cardiac or peripheral in nature. |
| Abdominal Compartment Syndrome | is the end result of a progressive, unchecked increase in intra-abdominal pressure from a myriad of disorders that eventually lead to multiple organ dysfunction. |

**Content:**

**Shock**

Shock is a condition where the body's homeostasis is disrupted, leading to inadequate oxygen delivery to the mitochondria of cells. This is a universal physiological threat to patients in shock. Without sufficient oxygen, aerobic metabolism cannot be maintained at the required rate to sustain cell function. As a result, cells are unable to recover from the interruption of aerobic metabolism, and ultimately, cell death occurs, leading to organ failure.

There are numerous mechanisms that can cause shock, and surgeons often focus on treating the underlying issues that affect cardiovascular function. This typically involves addressing impaired cardiac contractility, a decrease in systemic vascular resistance, or reduced intravascular volume. However, while therapies aimed at improving whole-organ function are crucial, it is important to remember that patient survival is ultimately determined by events at the cellular level.

In cases of profound hemorrhage, shock can become rapidly lethal. Similarly, even a modest reduction in oxygen delivery over a sustained period can lead to irreversible intracellular dysfunction, which can be just as fatal.

Descriptions of the shock syndrome aggregate pathogenesis, manifestation, and physiologic responses in ways that facilitate clinical recognition but conveniently obscure causal relationships. Despite decades of research, the causes of shock (or, more important, the causes of irreversibility and the sequelae of shock) remain enigmatic. The problem with current descriptions, many of which focus on a mismatch between metabolic supply and demand and its consequences, is that they do not capture either the self-sustaining nature of shock or the importance of timeliness in recognition and management. Whereas most authors cite these latter characteristics as “features” of shock and of shock therapy, the self-sustaining characteristic and the effectiveness of early intervention may well be fundamental to the physiologic derangement and rescue, respectively. Because “shock” is commonly described in terms of metabolic shortfall (metabolic demand exceeding supply of essential nutrients), “not shock” or the basal physiologic state must also be examined through the lens of energetics. All life forms, from the prokaryotes to complex mammals, share three imperatives: to extract energy from the environment to hold entropy at bay; to adapt to (the variable) external environment to maintain constant the internal environment; and to replicate. We focus first on energy extraction. Organisms and their constituent cells are thermodynamically open systems. Ingested carbohydrate, fat, and protein are biochemically degraded into primitive units. The currency of biochemical energy, high-energy phosphates

(including adenosine triphosphate, guanosine triphosphate, creatine phosphate, and others), can be generated directly from the primitives or, alternatively, the primitives can be stored. Most humans have several months' worth of stored fat and a day's worth of stored carbohydrate (hepatic and muscle glycogen). There is no storage form of protein: all known proteins are structural or catalytic. The primitives—glucose and fatty acids—generate high-energy phosphates through two biochemical pathways, one that requires molecular oxygen (oxidative phosphorylation) and one that does not (anaerobic glycolysis). The yield of high-energy phosphates through the oxidative pathway is sufficient to sustain life, whereas the yield through the anaerobic pathway is not. Proof of this distinction is clinical: carbon monoxide poisoning (which prevents oxygen transport on hemoglobin) and cyanide poisoning (which uncouples oxidative phosphorylation) are lethal. These represent two unusual causes of shock that the surgeon occasionally encounters. There is no storage form of oxygen. Arterial hemoglobin is normally 95% to 98% saturated with oxygen, whereas mixed venous blood is normally 70% to 75% saturated, suggesting that approximately one fourth of the available oxygen is removed during each circuit through vital organs and tissues. Human blood volume normally circulates approximately once each minute. These facts suggest that even if every oxygen molecule could be unloaded from hemoglobin to cells, unreplenished oxygen delivery will be exhausted in approximately 4 minutes. This is important for three reasons. It points to oxygen as the critical nutrient; it points to the importance of efficient resuscitation; and it points to restoration of oxygen delivery as the imperative in resuscitation from shock. If resuscitation is untimely or incomplete, the consequences are predictable and often lethal. Cells initially switch from oxidative phosphorylation to the more anaerobic metabolic pathways. End products of anaerobic metabolism, notably lactic acid, accumulate. More important, the electrochemical gradients across cytoplasmic and subcellular membranes that are normally maintained by a constant supply of high-energy phosphates start failing. As gradients fail, water and salt on either side equilibrate, disrupting the three-dimensional organization of proteins. Disrupted proteins cannot be repaired because the repair mechanisms require high-energy phosphates. Disrupted proteins cannot be recycled because the recycling mechanisms require high-energy phosphates. Beyond a salvage threshold of failed gradients and disrupted proteins, the affected cell becomes necrotic. Unfortunately, clean-up of necrotic tissue also requires energy. The result is a collective, accelerating spiral of deteriorating function of cells, tissues, and organs. Decades of research offer no better therapy than the prompt restoration of oxygen delivery. The adequacy of oxygen delivery is properly local, but oxygen delivery itself can be estimated from global measures. Thus, oxygen delivery is the geometric product of arterial oxygen saturation, hemoglobin concentration, stroke volume, and heart rate. The product of stroke volume and heart rate is cardiac output, or, equivalently, the amount of venous blood returning to the heart. The focus of shock resuscitation is the optimization of these parameters, and it is therefore worthwhile restating the definition of oxygen delivery in the form of a relation:

$$DO_2 = S_aO_2 \times [Hgb] \times \text{heart rate} \times \text{stroke volume}$$

However, optimization of these four parameters is only the second most important clinical imperative. The most important task in shock intervention is early recognition

of the shock syndrome. The syndrome is composed not only of the metabolic derangements directly attributable to inadequate perfusion but also of the reflex responses teleologically aimed at mitigating the inadequate perfusion. *The reflex responses are clinically appreciable far earlier than the derangements themselves.* These reflex responses are mediated by the neuroendocrine system, which secretes a series of hormones to sustain delivery of nutrients to cells and promote diffusion and transport of nutrients into cells. Several classes of hormones are released during the initial response to shock, the catecholamines, the renin-angiotensin-aldosterone axis, as well as antidiuretic hormone. The catecholamines, epinephrine and norepinephrine, are full agonists for both a-adrenergic (vasoconstrictor) and b 1-adrenergic (increased heart rate, increased heart contractility, increased heart conduction velocity) receptors. (Each is a partial agonist for b 2-adrenergic receptors, which mediate vasodilatation). The catecholamines cause three early events. First, the heart rate accelerates, second, peripheral arterial beds and splanchnic beds empty into the systemic circulation, and third, potassium is shifted to intracellular compartments. These events are appreciated as tachycardia; as delayed capillary refill and a slight rise in diastolic blood pressure; and as mild hypokalemia. Catecholamine secretion is prominent in all forms of shock and the effects of catecholamines are nearly always the first physical signs of shock. The observed response to catecholamines is less effective when the specific cause of shock renders target cells refractory to catecholamines, namely, septic shock. The response is also less effective when catecholamine responsiveness has been altered with drugs such as b-adrenergic blockers. Renin is released from the kidneys in response to hypovolemia, and the release is potentiated by epinephrine. The release catalyzes the conversion of angiotensinogen to the angiotensins. The sudden rise in circulating angiotensins contributes substantially to overall splanchnic vasoconstriction. Such constriction can mobilize up to 30% of the total blood volume, compensating for but also masking the loss of blood from the systemic circulation. The combination of catecholamines, renin, and antidiuretic hormone released early in response to shock causes the kidneys to retain water and sodium and decreases splanchnic perfusion. Urine output is therefore modulated relatively early in the response to shock. Other hormones secreted somewhat later in response to shock include glucagon, cortisol, and growth hormone. Collectively, they alter physiology to create a state similar to diabetes, including mild hyperglycemia and insulin resistance. Both muscle protein and fat stores are mobilized during recovery from shock to augment plasma glucose through gluconeogenesis. Except to correct demonstrable deficiencies, administration of these hormones has not been shown to improve outcome from shock.

**THE PATHWAYS TO SHOCK**

Once shock is recognized, the surgeon must simultaneously identify and reverse the underlying cause while performing resuscitation. The former is more difficult than the latter. It is helpful to remember that *there are three fundamental pathways to shock.* These pathways reflect problems with the “three P's”:

- the perfusate (intravascular volume);
- the pump [problems with the heart or getting blood into the heart (obstruction)];

– the pipes (distributive problems that allow blood to pool into the periphery and to pass by starving tissues without unloading nutrients).

This classification is simple enough to commit to memory and can guide decision making for the first several minutes of resuscitation.

The surgeon most commonly encounters shock through the *perfusate pathway*. The associated clinical syndromes are hypovolemic shock due to dehydration and hemorrhagic shock due to acute loss of blood volume. Mild perfusate loss is common and does not cause clinical symptoms. For example, voluntary blood donation corresponds to acute loss of approximately 10% of the circulating blood volume. The skin and skeletal muscle vasculature experience a slight decrease in perfusion. However, such a small acute loss is well tolerated because the intravascular volume can be quickly recruited from interstitial and intracellular reserves. Beyond 10% loss, however, the neuroendocrine response to shock becomes clinically apparent. The adrenal medulla increases its blood flow, ensuring both adequate oxygen delivery to its own tissues as well as swift delivery of catecholamines into the systemic circulation. As occupancy of the peripheral adrenergic receptors increases, heart rate and diastolic blood pressure rise, even while blood is squeezed out of the splanchnic bed. This compensatory redistribution fails at approximately 30% volume loss, a failure clinically manifested as the onset of systolic and diastolic hypotension. The decrease in urine flow in the early stages of volume loss is not due to early failure of renal blood flow, but rather to (a) a fall in glomerular filtration rate, (b) the sympathetically induced increases in resorption of sodium from the proximal tubules, and (c) the effects of antidiuretic hormone on retention of free water and that of aldosterone on distal tubular sodium resorption. Once hypotension occurs, further blood flow redistribution occurs in favor of the brain, but at the expense of the heart and the kidneys. A 40% to 50% volume loss exhausts all compensatory mechanisms. The need to restore perfusion and eliminate the cause of shock is evident.

*The pump pathway* to shock has two important entrances: primary pump failure and inability of the pump to accept the perfusate. The latter is commonly termed *obstructive shock* and is considered separately. The causes of pump failure, or cardiogenic shock, are familiar: acute failure of the cardiac muscle or a cardiac valve, and acute dysrhythmias. Specific diagnoses include myocardial infarction, rupture of a papillary muscle, and fracture of the chordae tendineae (the latter processes, thankfully rare, lead to acute regurgitation and failure of the left heart). *The obstructive pathway* to shock, the inability of the pump to accept the perfusate, is frequently traversed by injured patients. The specific diagnoses causing obstruction in the acutely injured are tension pneumothorax and pericardial tamponade. These diagnoses share a pathophysiologic process that transmits pressure to the external wall of the atria, thereby preventing blood flow into the cardiac chambers. Decompression (of the pleural space or of the pericardial space) is lifesaving. Acute embolism of a blood clot from the systemic veins into the heart (pulmonary embolism) is a common cause of obstructive shock among surgical patients. Therapy is focused on relief of the intraluminal obstruction. A less common but deadly cause of obstructive shock is air embolism consequent to inadequately filled systemic veins brought into contact with the atmosphere, either during surgery or by a central venous catheter. A large air

embolus obstructs the right ventricular outflow tract, whereas slow entrainment of air causes distal pulmonary arteries to become obstructed with acute right ventricular dysfunction. Therapy of a large right ventricular air lock requires relief of the obstruction through positioning (placing the patient in the right side down, head down position to try to move the embolus to the apex of the right ventricle), aspirating the right heart through a preexisting central venous catheter, cardiac massage, or direct puncture of the right heart to aspirate the air. These maneuvers are usually performed in the sequence listed until one is successful.

Discrimination between "perfusate" problems and "pump" problems is critical because the therapies are distinct. Unfortunately, the neuroendocrine response to pump shock is clinically indistinguishable to the response to perfusate shock: the skin is poorly perfused, moist, and cool; the pulse is weak; the heartbeat and respiratory rates are rapid; and the urine flow is reduced. Bedside discrimination between pump and perfusate shock is based directly on mechanism: in pump shock, the capacitance (venous) vascular beds are full because the pump cannot or will not accept inflow. Thus, in pump shock, the neck veins are distended, the patient has an elevated central venous pressure, and abnormal heart sounds may be present. In perfusate shock, the neck veins are collapsed and the central venous pressure is low. This difference cannot be overemphasized: given a patient with the classic presentation of shock (ashen facies, diaphoresis, tachycardia, tachypnea, and hypotension), attention should be immediately directed at the neck veins to discriminate pump from perfusate pathways.

*"Pipe" shock* (formally, distributive shock and neurogenic shock) follows failure of mechanisms that regulate tissue-specific resistance and capacitance. There are two routes to this form of shock. The first is through interruption of the sympathetic nervous system, the consequence of either spinal cord injury or neuraxial instillation of local anesthetic agents (spinal or epidural anesthesia). The second route is through attenuation of the sympathetic effects in the periphery, most commonly in the context of sepsis. Unlike the patient with perfusate or pump shock, patients with pipe shock fail to vasoconstrict in the periphery and therefore usually have warm skin. Tachycardia may be absent and bradycardia is often observed in spinal shock, particularly when the level of the spinal cord injury is at or above T-4. Importantly, the distributive cause of the shock also underlies the early failure of redistributive compensatory mechanisms. Neck veins are typically flat and the central venous pressure remains low.

## TYPES OF SHOCK

### Hypovolemic Shock

The most common shock state encountered by the surgeon is hypovolemic shock. Acute hypovolemia causes a parallel left shift of the venous return curve. The intersection with the normal cardiac function curve also shifts down and to the left. The neuroendocrine response, by releasing catecholamines into the circulation, rotates the cardiac function curve up and to the left, increasing cardiac flow, but only marginally. Clinically, tachycardia, tachypnea, and oliguria are reliable guides to the depth of the hypovolemia.

**Cardiogenic Shock**

Cardiogenic shock causes a pivot, rotating the cardiac function curve down and to the right. The equilibrium intersection between the venous return curve and the depressed cardiac function curve causes the low cardiac output. The physiologic compensatory response is to increase P ms, thus shifting the venous return curve up and to the right in parallel to the original venous return curve. This is a good time to consider and compare the vascular effects of dopamine (which has a predominantly vasoconstrictor effect in high doses) with the effects of dobutamine (which has a more vasodilatory profile). The two drugs have similar inotropic effects, so that administration of either drug partially restores cardiac function, pivoting the cardiac function curve up and to the left. The increase in afterload associated with dopamine may attenuate the left pivot. The effects on the venous return curve are quite different, however. Dopamine further increases P ms, shifting the venous return curve to the right in parallel with the other venous return curves. Dobutamine functions quite differently, keeping P ms roughly constant and pivoting the curve up and to the right as vascular resistance falls. These effects help to explain why dobutamine is usually preferred over dopamine in cardiogenic shock.

**Septic Shock**

Septic shock is the most common form of distributive shock encountered by the surgeon. Absent medical intervention, the venodilatation of sepsis causes not only a decrease in venous resistance but a fall in P ms. Volume resuscitation restores P ms to its normal value, but now with a markedly decreased venous resistance. The competing cardiac effects of sepsis are readily modeled with appropriate shifts in the cardiac function curves: whereas afterload reduction tends to increase cardiac performance, direct myocardial depression overwhelms the advantage of this afterload reduction in late, uncompensated sepsis, or in sepsis with preexisting cardiac disease. Bone and colleagues convened a consensus conference to define criteria for categorization of sepsis-related inflammatory response. The results of their deliberations were published in 1992, and these definitions have been widely used in subsequent studies to categorize inflammatory responses to infection. These authors defined four categories of clinical disease that represented successive levels of escalating severity of inflammatory response. The core concept of Bone and colleagues was that as the burden of bacterial toxins increases and the extent of endogenous inflammatory response intensifies, the clinical manifestations of the severity of illness become exaggerated and the risk of death increases. According to this classification severe sepsis is when patients are not responsive to intravenous fluid infusion for resuscitation or require inotropic or vasopressor agents to maintain systolic blood pressure.

**Obstructive Shock**

Obstruction to venous return is a surgical emergency. The two common causes encountered by general surgeons are pericardial tamponade and tension pneumothorax; obstetricians encounter a similar physiologic effect when the gravid uterus presses on the inferior vena cava. All abdominal surgeons occasionally cause transient obstructive shock by pressing on the inferior vena cava during surgery. Pulmonary embolism and air embolism are the other two major causes of obstructive shock. The venous return curve is markedly distorted because the pleural pressure exceeds the right atrial

pressure. Venous return no longer depends on the arithmetic difference between P ms and right atrial pressure, but on the difference between P ms and (the very positive) pleural pressure. The cardiac function curve is also adversely affected by two mechanisms. The rightward shift occurs because the transmural filling pressure is zero when the right atrial pressure falls to the (now positive) value of the pleural pressure. The downward pivot of the cardiac function curve is caused by a reflex increase in pulmonary vascular resistance. Although there is an endogenous catecholamine surge, it is apparent from the analysis that neither a volume load nor administration of exogenous catecholamines will have a significant effect on circulation. The only effective therapy is immediately to reduce pleural pressure by relieving the tension pneumothorax. Pericardial tamponade provides a nearly identical analysis, except that the limitation on transmural pressure is not pleural pressure but pericardial pressure.

**Neurogenic Shock**

Surgeons encounter neurogenic shock in two arenas: the trauma resuscitation bay and the operating room. Traumatic spinal injury occurs when the cord is severed at a level within or above the sympathetic chain, whereas neurogenic shock encountered in the operating room is the consequence of a neuraxial anesthetic that has extended beyond its intended effect. Bearing in mind that the heart also receives sympathetic input, there is an important functional distinction between an injury above T-4 and one below T-4. The former depresses cardiac function in addition to affecting venous return, whereas the latter leaves cardiac performance unaffected. When cardiac performance is unaffected, limited volume resuscitation and treatment with a pure a-agonist such as phenylephrine is sufficient therapy. However, if the cardiac sympathetic innervation is compromised, vagal parasympathetic innervation may predominate and administration of phenylephrine may aggravate reflex bradycardia. To preclude this undesirable effect of therapy, a mixed inotrope and chronotrope such as dopamine or norepinephrine is used. In extreme cases, temporary cardiac pacing may be lifesaving. Volume restoration is also required.

**Shock recognition and resuscitation: practical aspects**

Resuscitation from shock must begin immediately on recognition. Restoration of oxygen delivery is the imperative. The simple ABC approach is effective: establish and maintain an airway, ensure breathing with 100% oxygen, and restore the circulation. Supplemental oxygen must be administered by nonrebreather face mask, Ambu-bag, or tracheal intubation. Chronic obstructive airway disease is not a contraindication to the administration of oxygen. Ventilation should be confirmed by auscultation (axillae and stomach) and by demonstration of end-tidal carbon dioxide if the trachea has been intubated. The neck veins should be inspected to discriminate pump shock from perfusate shock. If the neck veins are distended, the axillae should be reauscultated to exclude a tension pneumothorax and consideration should be given to the possibility of pericardial tamponade. The heart should be auscultated to determine whether heart sounds are audible and, if so, whether abnormal heart sounds such as pathologic murmurs and third and fourth heart sounds are present. A pulse should be sought. The absence of central pulses (femoral, carotid) mandates cardiac life support including cardiopulmonary resuscitation, determination of rhythm, and cardioversion-defibrillation. More commonly, a faint pulse is palpable. The carotid pulse ordinarily

is present in adults with systolic blood pressures greater than 60 mm Hg. A short, wide-bore intravenous catheter should be inserted into a peripheral vein and an aliquot of blood taken for analysis. Given the frequency of hypovolemic blood loss in the surgical population, one of the most important steps is an immediate crossmatching of blood. A hemoglobin determination is also desirable. The venous catheter then serves as the conduit for rapid infusion of a balanced salt solution such as lactated Ringer's solution; 20 mL/kg should be administered as rapidly as practicable (within 5 minutes). The fluid bolus serves to increase preload, diminish venous resistance, and possibly decrease arterial afterload, all of which augment cardiac performance. Stroke volume improves by this mechanism even in patients who have sustained an acute myocardial infarction or have pericardial tamponade, as seen in the Guyton diagrams. Fluid bolus should therefore be withheld only when there is incontrovertible evidence that the cause is cardiogenic and associated with frothy pink pulmonary edema. The fluid bolus can be repeated immediately if the shock is not immediately responsive. While the fluid is being administered, electrophysiologic monitoring should begin. An electrocardiogram (ECG) should be obtained and the rhythm monitored continuously. The systemic blood pressure and heart rate should be determined at regular, frequent (e.g., every 2 to 5 minutes) intervals and recorded on a purpose-specific form. A pulse oximeter should be applied to determine the oxygen saturation of capillary blood. The signal may be difficult to obtain because of intense vasoconstriction and application of the probe to the earlobe or nose may be helpful. The stomach should be decompressed to prevent the complication of aspiration. None of the aforementioned methods is especially effective in determining the quality of organ perfusion or the adequacy of shock reversal. The best first proxy for the adequacy of organ perfusion appears to be urine output, which should be measured every 30 minutes by an indwelling bladder (Foley) catheter. The best first proxy for the adequacy of shock reversal is the pH on arterial blood gas analysis. During shock, the pH falls into the acid range as a consequence of anaerobic metabolism with obligatory accumulation of lactic acid. Once resuscitation is adequate, the lactate should be metabolized and the anion gap acidosis should normalize. Persistent anion gap acidosis suggests inadequate resuscitation or frankly nonviable tissue. A non-anion gap acidosis is less worrisome and may follow resuscitation with normal saline. To avoid confusion, Ringer's solution is often used as the balanced salt solution. To reiterate, heart rate and urine output are often the best indicators of the current depth of shock but do not indicate adequacy of resuscitation. Because shock is operationally defined as metabolic shortfall, the adequacy of resuscitation is reflected in tissue perfusion. The most immediately available measures of this perfusion include arterial blood gas analysis for pH and the by-product of anaerobic metabolism, lactate. Neither of these proxies directly assesses oxygen delivery to tissues most vulnerable to nutrient deprivation, such as neural tissue. However, owing to the sensitivity of the splanchnic bed to even mild shock states, perfusion of abdominal viscera can be used as an intermediate surrogate. The mucosal lumen of the gastrointestinal (GI) tract is accessible though the mouth and the rectum, and there appears to be a tight correlation between the adequacy of mucosal perfusion and outcome. The adequacy of mucosal perfusion can be assessed by tonometry, a technique that indirectly measures the accumulated acids in mucosal cells. However, it

is disputed whether titration of care to a predetermined tonometric value improves outcome. Given the metabolic imperatives, it is important frequently to reassess the determinants of oxygen delivery. Oxygen saturation should be maintained above 95%. After resuscitation from shock, metabolic demands can be substantial. The ideal hemoglobin concentration in unstable patients is unknown. Although stable patients can often tolerate hemoglobin levels as low as 7 g/dL, the unstable patient may be better served by targeting a slightly higher level until the shock is fully reversed. The heart rate should be maintained in a physiologic range, usually between 80 to 120 beats/min. The upper constraint on heart rate is lifted for children, who regulate cardiac output almost exclusively by heart rate. The stroke volume should be optimized by augmenting preload. If (and usually, only if) there is uncertainty about the magnitude of the cardiac output, it should be measured. This can be done by the thermodilution technique [after insertion of a pulmonary artery catheter (PAC)], by the less invasive dye clearance technique, or by aortic flow assessment with an esophageal ultrasound transducer. Resuscitation from shock states ordinarily requires a minimum indexed cardiac output (cardiac index) of 2.5 L/min/m2 of body surface area, and often considerably more, to deliver sufficient oxygen. Although the definition of "sufficient" oxygen delivery is still contested, all agree that a subnormal oxygen delivery is never adequate. Thus "sufficient" is at least "normal" oxygen delivery, which is approximately 500 mL/min/$m^2$. Because fully saturated oxyhemoglobin at a normal hemoglobin concentration of 15 g carries approximately 20 mL oxygen per 100 mL blood, the minimum cardiac index of 2.5 L/min/m2 is adequate only if there is a normal hemoglobin concentration that is fully saturated with oxygen. Decreases in either hemoglobin concentration or oxygen saturation require compensatory proportionate increments in cardiac output to maintain oxygen delivery at a minimum value of 500 mL/min/$m^2$.

**Risks of resuscitation**

Perhaps the most vexing aspect of shock is that resuscitation is not synonymous with reversal, much less a guarantee of recovery. Despite timely intervention and aggressive management, a significant number of patients with shock sustain secondary injuries attributable to reperfusion and inflammation associated with resuscitation. The biology of reperfusion and inflammation is complex and incompletely understood.

**Pulmonary artery catheter in shock**

The indications for use of the PAC (Swan-Ganz catheter) are in flux. This device is nontherapeutic and performance depends heavily on the skill of the operator and the expertise of the interpreter. Conclusions regarding its value need to be considered in the context of its use. With this in mind, the PAC appears to be overused. Acknowledging the paucity of data from randomized, prospective studies, it is suggested that surgeons managing myocardial infarction with progressive cardiogenic shock are justified in prompt placement of a PAC. The value of the PAC in other forms of shock is indeterminate, but in any case, its use should follow routine management with fluids and pressors guided by central venous and systemic arterial pressure monitoring. Failure to respond to routine management or uncertainty concerning the response are adequate reasons to use a PAC with its attendant risks.

**Problem of secondary sepsis**

Once a patient has been resuscitated and moved to the intensive care unit (ICU), the risks of sepsis are increased. The ICU has an indigenous microflora of virulent organisms available to attack immunocompromised, resuscitated patients whose defenses are further weakened by invasive devices. Prevention of secondary sepsis remains an important but very elusive goal for surgical critical care. The nosocomial component of ICU sepsis is widely acknowledged but poorly controlled. Transmission of disease from patient to provider to patient has been documented in many studies, yet compliance with hand washing and barrier (isolation) directives is poor. The indigenous flora is different from the endogenous flora, most likely as a consequence of widespread (ab) use of potent antimicrobials. As in the initial shock, recognition that the resuscitated patient is becoming secondarily septic is difficult. Once the diagnosis is suspected, selection of appropriate antibiotic therapy is relatively simple.

**Shock in the Operating Room**

Although anesthetics and operations have become progressively safer, most surgeons are eventually confronted with sudden circulatory collapse of a patient in the operating room. Such situations can be salvaged if the surgeon and anesthesiologist work rapidly to analyze and correct the problem. Should the anesthesiologist announce that the patient is *in extremis,* the most important next step is to determine whether there is ventilation and circulation. Presence of carbon dioxide in the end-tidal gas confirms that both are present. Conversely, absence of end-tidal carbon dioxide means that either ventilation or circulation, or both, has failed. Such failure requires immediate confirmation that the endotracheal tube is in the airway, immediate ventilation, and initiation of cardiac compression while the underlying cause of the arrest is sought. Open cardiac massage is more effective than closed massage, and there should be no hesitation in performing a sternotomy or thoracotomy if closed massage is not immediately effective. The cardiac rhythm should be inspected on the monitor, and the anesthesiologist asked about any changes in morphology (suggestive of myocardial ischemia or infarction) before the collapse. If a life-threatening arrhythmia is noted, it should be treated using advanced cardiac life support guidelines. If ventilation and circulation are present but there is circulatory collapse in the context of a reasonably normal cardiac rhythm, the next step is to look at the operative field while asking the anesthesiologist about the airway pressures. The surgeon must look for excessive bleeding and at the shape of the diaphragms. If significant bleeding is observed, isolation and control become the next priority. The reason to inspect the diaphragms while asking about airway pressures is that pneumothoraces are not only common, but quickly become tension pneumothoraces under positive-pressure ventilation. The diaphragm on the affected side billows into the abdomen and remains relatively distended throughout the ventilatory cycle. The airway pressures are higher than previously observed. If such a billowing diaphragm is observed, it should be immediately incised (1 to 2 cm) to convert the tension pneumothorax into an open pneumothorax. While the surgeon is inspecting the diaphragms, the anesthesiologist should be listening for breath sounds and heart sounds. The reason for listening to the heart sounds is to exclude a rarer cause of obstructive shock, air embolism, a cause that should be suspected in any patient who either has a central venous catheter in place or

who has had a large vein open in the operative field. Diagnosis is based entirely on suspicion, but the central venous catheters should be inspected and the heart should be auscultated for a continuous murmur. If air embolism is thought likely, an attempt should be made to aspirate air back through the central catheter while the patient is placed in Trendelenburg position. Management of this complication in the operating room is typically operative, aspirating the right ventricular outflow tract by direct puncture if cardiac massage proves insufficient immediately to break up the air lock. If breath sounds and heart sounds are normal and bleeding is not a problem, it should be ascertained whether there was a drop in end-tidal carbon dioxide just before the circulatory collapse. When such a drop has occurred, it suggests acute pulmonary embolism. Refractory shock caused by acute pulmonary embolism can occasionally be reversed by direct cardiac massage (breaking up the large embolus into smaller pieces) or, if appropriate personnel and equipment are immediately available, surgical retrieval of the clot. Finally, the possibilities of anaphylaxis to a recently administered drug and of a major transfusion reaction need to be considered. In the operating room and ICU, a view of the heart and aorta in real time can provide helpful information about cardiac performance. Personnel skilled in transesophageal echocardiography who can avail themselves of the necessary equipment can rapidly obtain information about cardiac performance, exclude pericardial tamponade, and make inferences about whether the venous system is sufficiently filled within a minute or two. Resuscitation should not be interrupted while the views are being obtained.

**Shock in the Immediate (0- to 4-Hour) Postoperative Period**

Shock in the immediate postoperative period is attributed to bleeding until proven otherwise. Plans should be made to return the patient to the operating room while an alternative cause is sought. Alternative causes are common and include acute myocardial dysfunction and delayed presentation of a pneumothorax after positive-pressure ventilation. The value of an immediate ECG and chest radiograph cannot be overemphasized. More often than not, bleeding is either an obvious cause of the shock state or is suggested by a lower-than-expected hematocrit. Although exploration of the surgical site is mandatory, the cause of the bleeding is not always surgical, and appropriate coagulation studies should be ordered along with blood products as soon as immediate postoperative shock is recognized.

**Shock in the Intermediate (4- to 24-Hour) Postoperative Period**

As anesthetics and pain medications wear off, patients often experience significant pain and respond with a catecholamine surge. The associated increase in heart rate can cause or mask an evolving myocardial infarction in patients at cardiovascular risk. Surgical site pain can extinguish anginal pain, and an ECG along with chemical tests for myocardial damage should be obtained promptly. Bleeding should be no lower than number two on the differential diagnosis of shock, and resuscitation should proceed even while plans are made to return the patient to the operating room. During this interval, serious surgical site infections can cause shock. These site infections, typically streptococcal, cause a brawny cellulitis (sometimes associated with brown edema fluid) that masks a necrotizing myofascial infection. For this reason, shock appearing during the intermediate postoperative period mandates at least an inspection of the wound. If cellulitis is present, the wound should be promptly

explored in the operating room, where radical debridement is undertaken. Aggressive antibiotic therapy, an adjunct to surgical debridement (not a substitute), may be lifesaving.

**Shock in the Late (>24 Hours) Postoperative Period**

There are four common causes of unexplained shock in the late postoperative period. Sepsis is by far the most common, including surgical site infections, bloodstream (catheter-associated) infections, urinary tract infections, and pneumonias. Myocardial infarction is also common and can occur without significant pain during the first few postoperative days. Pulmonary embolism in the setting of occult deep venous thrombosis tends to occur somewhat later because the operation and consequent immobility are usually the cause of the deep venous thrombosis, and pulmonary embolism must follow its formation. Shock and unexplained hypoxemia should suggest pulmonary embolism. Finally, occult GI bleeding causes painless hypovolemic shock that is unexplained until the oral or rectal passage of blood. Above all, the surgeon should be aware of time. Regardless of the cause of the shock, prompt recognition of the shock state, correction of the underlying problem, and immediate resuscitation appear to be the best guarantors of a favorable outcome.

**Acute Lung Injury/Acute Respiratory Distress Syndrome**

Acute lung injury and ARDS are clinical syndromes of pulmonary dysfunction that may result from any number of infectious, inflammatory, or tissue injury or cellular shock conditions.

*Criteria for the diagnosis of ARDS include:*

- acute onset,
- bilateral pulmonary infiltrates on chest radiography,
- the absence of cardiogenic pulmonary edema (i.e., pulmonary artery wedge pressure < 18 mm Hg), and
- hypoxemia (PaO2:FIO2 < 200).

On the same continuum*, acute lung injury* is a milder form, with PaO2:FIO2 = 201–300.

The mortality of ARDS approaches 40% to 50%, with most deaths attributed to MOF.

The pathogenesis of ARDS involves three stages. *The first stage*, coinciding with the acute onset of respiratory failure, is known as the exudative phase. Disruption of the alveolar epithelium results in the influx of protein-rich edema fluid and a leukocytic infiltrate. Destruction of type II pneumocytes disrupts normal alveolar fluid transport and surfactant production, leading to alveolar flooding and collapse. Macrophages release proinflammatory cytokines that attract and activate neutrophils, provoking tissue injury. Some patients have an uncomplicated course with resolution of the process, but others progress to the *fibroproliferative phase*. Mesenchymal cells fill the alveolar space and initiate fibrosis, with collagen and fibronectin accumulating in the lung. In the *resolution phase*, alveolar edema is resolved as type II pneumocytes repopulate the epithelium; protein is cleared; and there is gradual remodeling of granulation tissue and fibrosis.

**The treatment of ARDS** is primarily supportive.

- The underlying cause should be identified and treated.
- Nutritional support should be provided.
- Appropriate prophylactic measures against venous thromboembolism and stress gastritis.

*Adequate oxygenation and ventilation* must be provided; this generally requires intubation and mechanical ventilation. A number of novel adjunctive therapies have been studied in ARDS. Preliminary clinical studies suggest that *fluid management aimed at lowering filling pressures* may decrease pulmonary edema; whether this improves outcome remains to be seen. *Surfactant-replacement therapy* has been successful in neonates but not yet proven beneficial in adults with ARDS. Despite encouraging results in observational studies, *nitric oxide* has not proven beneficial in PRCTs; the same goes for other vasodilators. *Corticosteroids* were never found to be beneficial when administered early in ARDS. However, as the pathophysiology became better understood, the therapy was applied to the fibroproliferative phase. Encouraging results were reported in observational studies as well as in a small PRCT. Corticosteroids warrant consideration as salvage therapy for severe ARDS that is not resolving but must be used with caution because they predispose patients to the risk of infection.

*The optimal ventilatory strategy* for ARDS patients remains elusive. A number of methods have been employed, including extracorporeal membrane oxygenation (ECMO); extracorporeal carbon dioxide removal; high-frequency jet ventilation; high-frequency oscillatory ventilation; liquid ventilation; permissive hypercapnia; and inverse-ratio ventilation. None of these has been associated with a mortality reduction. Prone positioning has been proposed as a means to improve oxygenation by increasing end-expiratory lung volume, improving ventilation and perfusion matching, and changing chest wall mechanics. In a multicenter PRCT, prone positioning improved oxygenation but not survival. Although this intervention may be useful in treating severe hypoxemia for short periods, care must be exercised to minimize complications such as pressure ulceration, accidental extubation, and loss of vascular catheters and feeding/drainage tubes. Low tidal volume (VT) ventilation has been the focus of a number of PRCTs. The National Institutes of Health ARDS Network study group performed a multicenter PRCT in which patients were randomized to a VT of 12 mL/kg vs 6 mL/kg, with plateau pressures maintained at less than 50 versus less than 30 cm $H_2O$, respectively. After enrolling 861 patients, the trial was stopped because in-hospital mortality was reduced from 40% to 31% in the low VT group. The results of this study were discrepant from earlier, smaller trials. Whether the benefit was attributable solely to lower VT is unclear; nevertheless, this approach has gained widespread support. Positive end-expiratory pressure (PEEP) can improve oxygenation by recruiting collapsed alveoli and reducing functional residual capacity. "Conventional" ventilation generally calls for the minimal PEEP necessary to provide acceptable oxygenation. However, in the setting of ARDS, there may be benefit to increasing PEEP to improve oxygenation as well as to protect the lung by preventing repetitive recruitment/de-recruitment of alveoli, reducing cyclic reopening and stretch during mechanical breaths. The optimal level of PEEP may be determined by incrementally increasing PEEP to maximize the $PaO_2$:$FIO_2$ ratio; however, some argue

that this ignores lung mechanics. A lung pressure-volume curve may be generated for a given patient, and the lower inflection point (PFLEX)—the point at which the slope increases in steepness, representing a pressure at which the majority of alveolar units are open—identified. Alternatively, the PEEP may be titrated to maximal compliance, which may be easier to measure at the bedside. A “lung-protective” strategy employed in a PRCT included a VT less than 6 mL/kg, PEEP above PFLEX, driving pressures less than 20 cm $H_2O$ above the PEEP level, pressure-limited ventilation, and permissive hypercapnia. Compared with conventional ventilation, there was improved 28-day survival, less barotrauma, and a higher rate of weaning from mechanical ventilation. This trial was small and had a higher than expected mortality in the conventional ventilation group, but it has stimulated further study into the use of higher PEEP levels.

A **circulatory collapse** is defined as a general or specific failure of the circulation, either cardiac or peripheral in nature. A common cause of this could be shock or trauma from injury or surgery. A "general failure" is one that occurs across a wide range of locations in the body, such as systemic shock after the loss of a large amount of blood collapsing all the circulatory systems in the legs. A specific failure can be traced to a particular point, such as a clot.

**Intra-Abdominal Hypertension (IAH) and Abdominal Compartment Syndrome (ACS)**

As John Hunt stated abdominal Compartment Syndrome is “the end result of a progressive, unchecked increase in intra-abdominal pressure from a myriad of disorders that eventually leads to multiple organ dysfunction.”

Korn and associate first used the term ACS in 1980s. It is only in the past decade, that the pathophysiological repercussions of the increased intraabdominal pressure (IAP) and ACS have been recognised in a wide spectrum of surgical patients and treated aggressively. Emerson first noted the cardiovascular morbidity and mortality associated with elevated intra-abdominal pressure in 1911. However, the recognition of abdomen as a compartment and the concept of intraabdominal hypertension (IAH) resulting in ACS have only recently received attention.

The abdomen is a closed cavity. The pressure values inside follow the hydrostatic laws. The IAP is the steady state pressure within the abdominal cavity and changes with respiration.

*Factors influencing IAP:*

- The movements of diaphragm
- The costal arch shifts
- The contractions of the abdominal wall
- The volume of the intestines, which may be empty or over distended
- The presence of any additional content in the abdominal cavity
- *Existing points of view concerning normal values of IAP:*
- Normally, mean intra-abdominal pressures are zero or less (A.A. Meyer).
- The normal values of IAP are 0-5 mm Hg (M. Malbrain, 2002).
- Mean intraabdominal pressure is 6.5 mm Hg (range 0.2-16.2 mm Hg) (N.C. Sanchez et al., 2001).

Traditionally, ACS was considered a traumatic surgical disease. Nowadays it is proved that ACS is a problem in many critically ill patients who have suffered no trauma, especially those suffering systemic inflammatory response syndromes (SIRS). In untreated cases mortality is about 100%.

*Causes of Intra-abdominal Pressure (IAP) Elevation*

- Retroperitoneal: pancreatitis, retroperitoneal or pelvic bleeding, contained AAA rupture, aortic surgery, abscess, visceral edema
- Intraperitoneal: intraperitoneal bleeding, acute gastric dilatation, bowel obstruction, ileus, mesenteric venous obstruction, pneumoperitoneum, abdominal packing, abscess, visceral edema secondary to resuscitation (SIRS)
- Abdominal Wall: burn eschar, repair of gastroschisis or omphalocele, reduction of large hernias, pneumatic anti-shock garments, lap closure under tension, abdominal binders
- Chronic: central obesity, ascites, large abdominal tumors, peritoneal dialysis, pregnancy.

*The following predisposing conditions are well recognised:*

- hypothermia
- acidosis
- polytransfusion
- dilutional coagulopathy or disseminated intravascular coagulation
- sepsis with capillary leakage
- vasculopathy, or liver dysfunction
- The combination of acidosis, hypothermia and coagulopathy has been proposed as a triad, leading to ACS.

*Grading of ACS (J.M. Burch and colleagues, 1996)*

- Grade I = 10 to 15 mm of Hg
- Grade II = 15 to 25 mm of Hg
- Grade III = 25 to 35 mm of Hg
- Grade IV >35 mm of Hg

*Morris definition of ACS:*

- A pathologic state caused by an acute increase in IAP above 20-25 mm Hg (or 27.2-34 $cmH_2O$)
- End-organ dysfunction or serious wound complications and
- Improvement by abdominal decompression

*Types of ACS*

- PRIMARY ACS is essentially organ dysfunction and IAH in the presence of direct injury to the abdominal contents. The examples are trauma, peritonitis, ileus, and haemorrhage etc.
- SECONDARY ACS consists of elevated pressure and organ dysfunction caused by third space oedema and resuscitation. The examples are resuscitation of haemorrhagic shock patients, burns etc.

- RECURRENT ACS in which the patient has recovered from the ACS once but because of secondary insults the cycle begins again. This verity is associated with very high mortality rate.

*Physiologic sequelae of high intraabdominal pressure*

1. Cardiovascular:

- Increased intra-abdominal pressures causes:
- Decrease in the preload
- Increase in the afterload due to an elevation in systemic vascular resistance (SVR)
- Impairment of cardiac contractility

*End result:* Dramatic reduction in venous return to the heart (preload). Right coronary artery blood flow drop with resultant RV subendocardial ischemia and worsening cardiac dysfunction. Elevated SVR leads to reduced blood flow to organs already suffering from ischemia and venous engorgement. They are now more ischemic and the capillary leak worsens, further exacerbating the syndrome.

Elevated intra-thoracic pressure directly impacts traditional pressure-based cardiac filling measurements such as CVP and PAOP (wedge). These pressure measurements are erroneously elevated and do not reflect actual fluid resuscitation end-points. Failure to understand this, and reliance on pressure-based cardiac indices will lead to inadequate fluid resuscitation, persistent global organ ischemia and higher instances of MOF and death. Volumetric indices such as RVEDVI and GEDVI accurately reflect fluid volume status in the face of elevated IAP and ITP. Focusing volume resuscitation end points on a volume-based index will result in improved cardiac function and reduced organ failure.

2. Pulmonary:

Increased intra-abdominal pressures causes:

- Elevation of the diaphragms with reduction in lung volumes

*The result:*

- Elevated intrathoracic pressure (which further reduces venous return to heart, exacerbating cardiac problems)
- Increased peak pressures
- Reduced tidal volumes
- Barotrauma, atelectasis, hypoxia, hypercarbia

3. Gastrointestinal:

Increased intra-abdominal pressures causes:

- Compression of mesenteric arteries
- Congestion of mesenteric veins and capillaries
- Reduced cardiac output to the gut

*The result:*

- Decreased gut perfusion, increased gut edema and leak
- Ischemia, necrosis, cytokine release
- Bacterial translocation
- Development and perpetuation of SIRS
- Further increases in intra-abdominal pressure

4. Renal:

Elevated intra-abdominal pressure causes:

- Compression of renal veins and arteries
- Reduced cardiac output to kidneys

*The Result:*

- Decreased renal artery and vein flow
- Renal congestion and edema
- Decreased glomerular filtration rate (GFR)
- Acute tubular necrosis (ATN)
- Renal failure, oliguria/anuria

5. Neuro:

Elevated intra-abdominal pressure causes:

- Increases in intrathoracic pressure
- Increases in superior vena cava (SVC) pressure with reduction in drainage of SVC into the thorax

*The Result:*

- Increased central venous pressure and IJ pressure
- Increased intracranial pressure
- Decreased cerebral perfusion pressure
- Cerebral edema, brain anoxia, brain injury

6. Miscellaneous

Elevated intra-abdominal pressure causes:

- Reduces perfusion of surgical and
- traumatic wounds
- Reduced blood flow to critical organs and tissues

*The Result:*

- Poor wound healing and dehiscence
- Coagulopathy
- Immunosuppression

**Classic signs of ACS (It normally develops 12 to 24 hours after the first operation) are:**

- decreased $PO_2$
- very highly elevated $PCO_2$
- high peak inspiratory pressure
- lack of urinary output
- a massively distended abdomen.

A better way of diagnosing this condition, however, is through continuous intra-abdominal pressure monitoring in the intensive care unit (ICU) in all critically ill patients at high risk for these complications.

*The principle ways of assessing pressure are:*

- inferior vena cava catheter
- urinary bladder catheter
- peritoneal catheter

- vaginal catheter
- gastric balloon

Intraabdominal pressure is most commonly measured indirectly by monitoring bladder pressures. Bladder pressure monitoring through the Foley catheter is comparable to direct intraperitoneal pressure measurements, but is non-invasive, more reliable and reproducible than clinical judgment, allows early detection of intra-abdominal hypertension, allowing intervention before ACS develops.

*IAH/ACS Management*

- Fluids – adequate fluid resuscitation
  - normovolemic in patients with Grade I IAH
  - hypervolemic in patients with Grade II-IV
- Abdominal perfusion pressure-optimize fluids first then add vasopressors. Shoot for a on perfusion pressure > 60 mm Hg
- Paralytics
- Cathartics / enema to clear bowel?
- Paracentesis
  - need significant free fluid on US
  - can place temporary catheter
- Decompressive laparotomy – in patients with Grade III–IV is obligatory, in some patients with Grade II also can be recommended,
  - can be performed bedside for unstable patients
  - delay in abdominal decompression may lead to intestinal ischemia

*Post-laparotomy ACS* – Same problem the same is the treatment

- Be aware that ACS can recur following a decompression laparotomy
- Score or replace dressing to treat recurrence

***Indications for IAP monitoring:***

- Sepsis/SIRS/Ischemia-reperfusion
  - Sepsis and resuscitation with > 6 l crystalloid/colloid or > 4 units of blood in 8 hours
  - Pancreatitis
  - Peritonitis
  - Ileus/bowel obstruction
  - Mesenteric ischemia/necrosis
- Visceral compression/reduction
  - Large ascites/peritoneal dialysis
  - Retroperitoneal/abdominal tumor
  - Laparotomy closed under tension
  - Gastroschisis/omphalocele
- Surgical
  - Intra-operative fluid balance > 6 l
  - Abdominal aortic aneurysm repair
- Trauma
  - Shock requiring resuscitation (ischemia-reperfusion)
  - Damage control laparotomy

- Multiple trauma with or without abdominal trauma requiring resuscitation with > 6 l crystalloid/colloid or > 4 units blood in 8 hours
- Major burns (> 25%)

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**Tests**

11. Signs and symptoms associated with early sepsis include
    A. Respiratory acidosis
    B. Decreased cardiac output
    C. Hypoglycemia
    D. Increased arteriovenous oxygen difference
    E. Cutaneous vasodilation
12. Major alterations in pulmonary function associated with adult respiratory distress syndrome (ARDS) include
    A. Hypoxemia
    B. Increased pulmonary compliance
    C. Increased resting lung volume
    D. Increased functional residual capacity
    E. Decreased dead space ventilation
13. A conscious person in shock should be given:
    A. coffee or tea.
    B. orange juice.
    C. clear liquids.
    D. nothing by mouth
    E. alcohol
14. The response to shock includes which of the following metabolic effects?
    A. Increase in sodium and water excretion
    B. Increase in renal perfusion
    C. Decrease in cortisol levels
    D. Hyperkalemia
    E. Hypoglycemia
15. Animal and clinical studies have shown that administration of lactated Ringer's solution to patients with hypovolemic shock may
    A. Increase serum lactate concentration
    B. Impair liver function
    C. Improve hemodynamics by alleviating the deficit in the interstitial fluid compartment
    D. Increase metabolic acidosis
    E. Increase the need for blood transfusion
16. Which of the following signs and symptoms suggest possible hypovolemic shock?
    A. Weak, rapid pulse; cold, clammy skin; pallor; shallow breathing
    B. Slow, strong pulse; dizziness; cold perspiration; nausea
    C. Blank expression; cold extremities; regular breathing
    D. Blank expression; chills; unconsciousness; dry skin
17. 7. Which of the following statements regarding stress ulceration is true?

A. It is true ulceration, extending into and through the muscularis mucosa
B. It classically involves the antrum
C. Increased secretion of gastric acid has been shown to play a causative role
D. It frequently involves multiple sites
E. It is seen following shock or sepsis, but for some unknown reason does not occur following major surgery, trauma, or burns

18. The principle ways of assessing intraabdominal pressure are the following EXCEPT:
A. superior vena cava catheter
B. urinary bladder catheter
C. peritoneal catheter
D. vaginal catheter
E. gastric balloon

19. Predisposing factors for the development of intraabdominal hypertension include the following EXCEPT:
A. hypothermia
B. acidosis
C. polytransfusion
D. dilutional coagulopathy or disseminated intravascular coagulation
E. spinal cord injury

20. The clinical signs of ACS don't include:
A. decreased $PO_2$
B. very highly elevated $PCO_2$
C. high peak expiratory pressure
D. lack of urinary output
E. a massively distended abdomen.

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| E | A | D | D | C | A | D | A | E | C |

**Materials for self-education**

| Main tasks | Notes(instructions) |
|---|---|
| Repeat:<br>– Anatomy of the vascular system<br>– Physiology of blood circulation<br>– Physiology of breathing | 1. Make a scheme of blood circulation, formula of an average blood pressure calculation<br>2. Formula of peripheral vascular resistance assessment |
| Study:<br>– Surgical approaches to the heart to perform direct heart massage<br>– BSI technique<br>– Methods of evaluation of abdominal compliance | 1. Make the algorithm of actions in case of circulatory collapse in operative room<br>2. Make the algorithms of actions in cases of postoperative shock |

| | |
|---|---|
| – The optimal ventilatory strategy for ARDS patients | |

## TOPIC 3. DYSPHAGIA SYNDROME, VOMITING AND DEFECATION DISORDERS. INTESTINAL OBSTRUCTION SYNDROME. ETIOLOGY. PATHOGENESIS

### 3.1. Dysphagia syndrome, vomiting and defecation disorders. Colitic syndrome. Features of colitis syndrome depending on diseases of the colon. Modern methods of diagnosis of colon diseases. Their importance in the diagnosis and differential diagnosis of abdominal organs.

**Overview.**

Dysphagia is the cause of 3-4% of visits to general practitioners and 10% of visits to a gastroenterologist. In 25% of patients, it has a functional basis, and in other cases - an organic nature. Every 10th case of dysphagia is due to conditions that require surgical intervention. Dysphagia is a frequent (up to 80%) symptom of esophageal damage and one of the few direct, alarming symptoms of oncology-gastroenterology. In this regard, all patients with dysphagia should be carefully examined in order to establish the specific cause of its development. Vomiting is a symptom of many diseases, so if it is often repeated, it requires a thorough examination and treatment according to the cause. Violation of the act of defecation (diarrhea, constipation) is an important manifestation of many diseases of the digestive tract, which requires careful differential diagnosis.

**Educational aims:**

- Conduct a survey and physical examination of patients with pathological syndromes.
- Develop a plan for examining patients with pathological syndromes.
- Based on the analysis of laboratory and instrumental examination data, conduct differential diagnostics, substantiate and formulate a diagnosis.
- Prescribe treatment for diseases of the abdominal organs that have pathological syndromes.
- Demonstrate mastery of the moral and deontological principles of a medical worker and the principles of professional subordination.

**A student must know:**

- Definition of dysphagia, vomiting and disorders of the act of defecation.
- The main causes of dysphagia, vomiting, disorders of the act of defecation and the features of these syndromes depending on the disease.
- Features of the pathophysiology of the indicated pathological syndromes.
- Modern methods of examination of patients with the indicated pathological syndromes.
- Algorithm of diagnosis and differential diagnosis of diseases or pathological conditions accompanied by the indicated syndromes.
- Methodology of differential selection of treatment methods for diseases manifested by the indicated pathological syndromes.

**A student must be able to:**

- Clinically determine the probable causes of the presence of the named pathological syndromes in a patient.
- Determine the algorithm for diagnosis and differential diagnosis of diseases manifested by the indicated pathological syndromes.
- Correctly evaluate the results of the conducted clinical, laboratory and instrumental examination in patients with pathological syndromes.
- Formulate a preliminary, clinical and final diagnosis of diseases manifested by the indicated pathological syndromes.
- Differentiatedly determine the treatment tactics for diseases manifested by the indicated pathological syndromes.

**Terminology.**

| Term | Definition |
|---|---|
| Dysphagia | Disorder (difficulty) of the act of swallowing, as well as the transportation of solid and / or liquid food from the oral cavity to the stomach. Patients perceive this condition as a feeling of "stuck" food when passing through the oral cavity, pharynx or esophagus. |
| Vomiting | A complex reflex act (involuntary impulse emissions of stomach contents through the mouth), associated with a violation of the vomiting center located in the medulla oblongata. |
| Defecation disorders: | |
| Diarrhea | Accelerated or single bowel movements with the release of liquefied feces. |
| Functional constipation | Chronic delay in emptying the colon for more than 48 hours, observed for at least 3 months and accompanied by at least one of the following signs: a feeling of incomplete emptying, a small amount (less than 100 g) and a thick consistency of feces, straining for at least a quarter of the time of defecation. |
| Chronic proctogenic stasis | Functional, and over time, organic damage to the colon, which is based on a violation of the plastic and kinetic properties of the colon and rectum due to congenital or acquired disorders of vascular and nervous trophism of the intestinal wall, the symptoms of which are persistent constipation, accompanied by pain and bloating, and in severe cases, the phenomena of mechanical obstruction of the colon and intoxication. |
| Obstructive defecation | Violation of the evacuation of intestinal contents associated with changes in the posterior pelvic floor, which arose against the background of pelvic distension syndrome (prolapse or prolapse of the pelvic organs due to weakness of the pelvic floor), the inherent |

| | |
|---|---|
| | manifestations of which may be rectocele, internal intussusception and prolapse of the rectum, enterocele and sigmoidcele in combination with discoordination and/or atrophy of the pelvic floor muscles. |

**Content:**

The causes of dysphagia are numerous and diverse. To achieve the main goals, namely, to timely establish a diagnosis and prescribe adequate treatment, knowledge of the semiotics of swallowing disorders is necessary. First of all, the localization of swallowing disorders should be clarified. In this regard, oropharyngeal and esophageal dysphagia are distinguished.

**Classification**

By type:

- Oropharyngeal (preesophageal), upper, middle, lower.
- Esophageal (esophageal), upper, middle, lower.

By pathogenesis:

- Mechanical (peripheral-organic).
- Motor (neurogenic).

By the degree of severity of esophageal obstruction:

- 1st degree - characterized by periodic difficulty swallowing solid food, pain with the movement of the food lump;
- 2nd degree - only semi-liquid food is allowed to move;
- 3rd degree - only liquid food is allowed to move;
- 4th degree - even swallowing saliva and water is impossible

The causes of the main types of dysphagia in various diseases are presented in the table.

| Oropharyngeal dysphagia (preesophageal) | | Esophageal dysphagia (esophageal) | |
|---|---|---|---|
| Causes | Diseases | Causes | Diseases |
| Neuromuscular lesions (paralysis of the pharyngeal muscles) | Myasthenia. Rabies. Botulism. Encephalitis. Diphtheria. Bulbar paralysis. Pseudobulbar paralysis in cerebral atherosclerosis. Parkinson's disease. Poliomyelitis. | Anatomical anomalies | Zenker's diverticulum. Cervical osteophyte. Hiatal hernia |

| | | | |
|---|---|---|---|
| Myopathies, metabolic and endocrine diseases | Myxedema (hypothyroidism). Toxic goiter. Diabetes mellitus. Amyloidosis. Dermatomyositis. | Disorders of esophageal tone and motility | Achalasia. Idiospastic dilatation of the esophagus. Diffuse spasm of the esophagus. Scleroderma. Diverticulosis (traction, etc.). Gastroesophageal reflux disease (GERD). Systemic diseases. Senile esophagus. |
| Inflammatory diseases | Angina. Infectious pharyngitis. Peritonsillar abscess. Acute and subacute thyroiditis. | Mechanical narrowing | Tumors: squamous cell carcinoma, adenocarcinoma, gastric fundic carcinoma, benign tumors. Peptic strictures. Vascular lesions (varicose veins) |
| Tumors. Damage caused by physical factors | Bulky mass in the oropharynx. Radiation injury. Aphthous ulcers. Candidiasis. Xerostomia (dry mouth). Pharyngeal diverticulum. | Others | Sideropenia. Infectious esophagitis. Esophagitis of drug origin. Burns with alkalis and acids. Radiation esophagitis. Radiation strictures. Postoperative conditions: after trunk vagotomy or surgical intervention on the esophagus. Crohn's disease. Sarcoidosis. External compression (lymph nodes, aortic aneurysm, left ventricular enlargement, retrosternal goiter). |

Patients with oropharyngeal dysphagia complain of food accumulation in the mouth, inability to swallow, or difficulty swallowing that lasts no more than 1 second after swallowing. In addition, such patients have aspiration before, during, or after swallowing. Along with this, aspiration during swallowing may cause coughing or wheezing. Nasopharyngeal regurgitation, nasal congestion, ptosis, photophobia, and visual impairment are also possible, as well as weakness, usually increasing by the end of the day. The most common causes of oropharyngeal dysphagia are aphthae, candidiasis, and cerebrovascular disorders. Less commonly, it occurs against the background of Parkinson's disease, globus histericus, pseudobulbar palsy, myasthenia gravis, Sjögren's disease, poliomyelitis, botulism, and syringobulbia.

In esophageal dysphagia, the feeling of difficulty in swallowing is localized retrosternal, or in the area of the xiphoid process, and occurs after several consecutive swallows. In the presence of esophageal dysphagia, patients do not always and not always accurately indicate the location of the lesion that interferes with the passage of food. Thus, only 60-70% of them can correctly indicate the level of food retention in the esophagus. The rest mistakenly believe that the lesion is located more proximal to the true location of the obstacle. Determining the time elapsed from the moment of swallowing to the onset of dysphagia can serve as a more objective and simple initial assessment of the level of damage. Dysphagia of the cervical esophagus manifests itself immediately after swallowing - after 1-1.5 seconds, dysphagia of the middle third of the esophagus - after 4-5 seconds, distal dysphagia - after 6-8 seconds.

Clarification of the localization of dysphagia has diagnostic value when patients describe it as compression in the chest, more often behind the sternum, which usually corresponds to the level of esophageal obstruction. The feeling of dysphagia in the neck has practically no differential diagnostic value, since this localization can be a manifestation of damage to the pharynx, almost all parts of the esophagus (including the cardiac part), and is also possible with globus hystericus.

By duration, intermittent (paroxysmal) and persistent (permanent) dysphagia are distinguished. The first, as a rule, is caused by hypermotor dyskinesia of the esophagus. It should be remembered that such dyskinesia often accompanies the course of such diseases as hiatal hernias, esophagitis of various genesis, and esophageal tumors. Permanent dysphagia is observed in most cases in patients with organic pathology and is manifested by difficulty in passing mainly solid food.

The extreme degree of severity of dysphagia is aphagia, in which there is a complete closure of the alimentary canal, which requires urgent instrumental or surgical intervention. The differential diagnostic algorithm requires an analysis not only of the patient's complaints, but also a detailed analysis of the anamnesis. Long-standing heartburn, previous dysphagia, may indicate peptic stricture. Short-term intermittent dysphagia may be due to an inflammatory process. Its combination with painful swallowing (odynophagia) suggests the presence of esophagitis (possibly candidal or herpetic, which occur in cancer patients or patients receiving immunosuppressive therapy). Recurrent odynophagia may occur against the background of diffuse spasm of the esophagus. The type of dysphagia also depends on the consistency of food, which causes discomfort when swallowing. Difficulties that arise when swallowing only solid food indicate organic dysphagia. A stuck lump can be pushed through the narrowed area of the esophagus by drinking some liquid. Liquid food is worse when motor function is impaired.

Vomiting is a symptom of many diseases of internal organs and conditions: diseases of the digestive system, organic diseases of the brain or its membranes (cerebral hemorrhage, vascular thrombosis, tumors), kidney diseases, toxicosis of pregnant women, eye diseases, vestibular apparatus, diabetes mellitus, myocardial infarction, infections, intoxications, febrile states and other diseases. Vomiting can occur as a result of hypersensitivity to drugs, when taking incompatible drugs, overdose of medications.

**Classification**

By pathogenesis:

- Vomiting, caused by irritation of receptors of various organs - the root of the tongue, soft palate, gastric mucosa, gallbladder, organ of balance, etc., which is transmitted to the vomiting center of the medulla oblongata.
- Vomiting, caused by direct irritation of the vomiting center in CNS lesions (various tumors, trauma, meningitis), with increased intracranial pressure, various intoxications (overdose, toxicosis of pregnancy, uremia).
- Vomiting, as a protective physiological act (if the stomach is full or harmful substances have entered it).
- Psychogenic vomiting - with strong excitement, negative emotions.

**The main etiological factors of vomiting are presented in the table.**

| Pathophysiological mechanism | Groups of diseases, conditions | Diseases, conditions |
|---|---|---|
| Vomiting of nervous (central) origin | Diseases and injuries of the central nervous system | Vestibular disorders, psycho-emotional disorders, stress, psychology, depression, history. |
| | Taking medications. | Drugs (most often opiates), digitalis preparations, aminophylline, chemotherapeutic agents. |
| Hematogenous toxic vomiting | Infectious diseases or the effects of toxins | Epidemic diseases. Viral hepatitis, food poisoning, viruses (enteroviruses). |
| | Metabolic disorders | Liver failure. Kidney failure. Ketoacidosis, Addison's disease. |
| | Pregnancy | Toxicosis of pregnant women |
| Vomiting of peripheral origin (reflex), including "gastric" | Diseases of the abdominal organs | Acute inflammatory diseases of the stomach. Gastrointestinal tract obstruction. Peritonitis, Acute pancreatitis, acute cholecystitis. Chemically poor-quality food. |
| | Diseases of other systems and organs | Acute myocardial infarction (most often affecting the posterior wall of the left ventricle), congestive heart failure |

In most cases, vomiting is preceded by nausea, increased salivation, rapid, deep breathing. The diaphragm descends sequentially, the glottis closes, the pyloric section contracts sharply, the body of the stomach and the lower esophageal sphincter (the area of the esophageal-gastric junction) relax, and antiperistalsis occurs. The contraction of the diaphragm and abdominal muscles is accompanied by an increase in intra-abdominal and intragastric pressure, which leads to the rapid ejection of stomach contents through the esophagus and mouth to the outside. Vomiting is usually

accompanied by pallor of the skin, increased sweating, severe weakness, tachycardia, and a decrease in blood pressure. In diseases of the digestive system, vomiting is usually preceded by nausea, sometimes abdominal pain. Nausea, an unpleasant sensation in the epigastric region, is often accompanied by a feeling of nausea, salivation, pallor of the skin, sweating, dizziness. Nausea occurs as a result of irritation of the vagus and intestinal nerves, which are transmitted to the vomiting center with subsequent efferent pathological impulses. Accompanying many diseases of the digestive system, nausea is not a specific symptom. However, it has been noted that vomiting without prior nausea is more often of central origin. If vomiting is preceded by a headache, especially of the hemicrania type, one should think about migraines. Morning vomiting, before breakfast, is most often caused by the toxic effect of exogenous (alcohol) or endogenous (uremia) factors, possibly toxicosis of pregnant women. Esophageal vomiting, as a rule, is not preceded by nausea. Vomiting occurs when the patient assumes a horizontal position or is accompanied by retention and accumulation of food in the esophagus - esophageal stenosis of various genesis (tumor, post-burn or peptic stricture), achalasia of the cardia, diverticulum, esophageal dyskinesia and insufficiency of the lower esophageal sphincter (cardium). Early and late esophageal vomiting are distinguished. Early esophageal vomiting occurs during eating, often with the first sips of food, is accompanied by pain behind the sternum, dysphagia. It can be observed both with organic lesions (cancer, ulcer, stricture) and with functional disorders of the esophagus. With organic lesions of the esophagus, attacks of dysphagia, pain and vomiting directly depend on the density of the swallowed food lump: the denser the food, the more sharply these symptoms manifest. In functional disorders of the esophagus, such a dependence is not observed, often more solid food does not cause any complications, and liquid food leads to vomiting. Late esophageal vomiting occurs 3-4 hours after eating and indicates a significant expansion of the esophagus. Usually this is a sign of achalasia cardia. Late vomiting may be a consequence of a large esophageal diverticulum, but the volume of vomited masses is much smaller than in achalasia cardia. In esophageal vomiting, the contents consist of undigested food masses, saliva and mucus without an admixture of gastric juice. In peptic esophagitis (reflux esophagitis), vomiting can occur both during a meal and after some time, sometimes at night in the horizontal position of the patient, with a sharp tilt of the torso forward, with increased intra-abdominal and intra-gastric pressure. Vomiting consists of undigested food residues with a large admixture of acidic or bitter-tasting liquid (gastric juice, bile). Vomiting at night, due to the ingress of vomit into the respiratory tract, can cause a severe painful cough.

In diseases of the stomach and duodenum, vomiting usually occurs after eating, and this period of time is quite constant. In duodenal ulcer, vomiting usually occurs 2-4 hours after eating or at night against the background of severe pain in the upper abdomen, preceded by severe nausea. The pain after vomiting in these patients weakens or completely subsides, so patients often deliberately provoke vomiting in order to get relief. When bleeding from a stomach ulcer, less often from a duodenal ulcer, vomiting of unchanged or slightly altered blood (hematemesis) or "coffee grounds" (due to the formation of hydrochloric acid hematin of brown color) is characteristic. Stenosis of the gastric outlet of organic origin (cancer, post-ulcer cicatricial deformation) is often

accompanied by profuse vomiting with an admixture of food residues eaten the day before or a few days ago, which have putrefactive properties. With pylorospasm, caused more often by functional disorders (reflex effects in peptic ulcer disease, diseases of the biliary tract and gallbladder, as well as neuroses) and less often by other causes (lead intoxication, insufficiency of the parathyroid glands), a tendency to frequent vomiting is often observed. However, unlike organic stenosis, vomiting with pylorospasm is less quantitative, contains a small amount of gastric contents with an admixture of recently eaten food, its frequency depends on the severity of the underlying disease and the emotional lability of the patient.

In acute gastritis, vomiting is frequent, accompanied by sharp, sometimes burning pain in the upper abdomen, occurs during or immediately after eating, bringing temporary relief. For chronic gastritis, vomiting is not characteristic, except for one form - chronic gastritis with normal or increased secretory function. Vomiting more often occurs in the morning on an empty stomach, sometimes without prior pain and nausea. Intestinal obstruction is often accompanied by vomiting. Vomiting is preceded or accompanied by severe abdominal pain and nausea. Vomiting masses with high obstruction of the small intestine consist mainly of gastric contents and a large amount of bile. With obstruction of the middle and distal parts of the intestine, a brown tint with a fecal odor appears in the vomit. True "fecal vomiting" usually indicates the presence of a fistula between the stomach and the transverse colon or indicates a critical condition of the patient with long-standing (decompensated) intestinal obstruction.

In acute appendicitis, vomiting usually occurs in the presence of abdominal pain, which after only a few hours is concentrated in the right iliac region and becomes constant. The pain attack is accompanied by a moderate increase in body temperature without chills and a gradual increase in peritoneal phenomena in the right iliac region.

Thrombosis of mesenteric vessels is characterized by the sudden onset of vomiting, often with an admixture of blood; vomiting is usually preceded by sharp abdominal pain and collapse.

In peritonitis, vomiting often occurs, which disappears and reappears with the expansion of the pathological process zone. In the toxic stage, peritonitis is manifested by recurrent vomiting, abdominal pain, and symptoms of peritoneal irritation.

Diseases of the liver, biliary tract and pancreas are characterized by repeated vomiting of bile, pain in the right hypochondrium, transient jaundice, which develop after eating fatty foods. In acute pancreatitis, vomiting usually occurs simultaneously with an attack of severe pain in the upper abdomen. Vomiting with blood is often present. In biliary (hepatic) colic, which develops with cholelithiasis, acute and chronic cholecystitis, stenosis of the major duodenal papilla, stricture and dyskinesia of the biliary tract, vomiting accompanies a pain attack along with other symptoms (nausea, flatulence, fever).

The syndrome of defecation disorder can be presented in two variants: diarrhea, functional constipation, chronic colo-proctogenic stasis, obstructive defecation. It should be noted that in the presence of such variants of the act of defecation disorder, it is necessary, first of all, to determine whether these disorders are clinical signs of oncological disease (colon cancer with its obstructive or enterocolitic clinical forms) or other organic pathology (inflammatory or cicatricial stricture) of the colon.

To understand these disorders, it is necessary to know the definition of the concept of defecation. Defecation is a physiological act of freeing the intestines from fecal masses. Defecation is regulated by the central nervous system and occurs as a result of irritation of the sensitive nerve endings of the rectal mucosa. Excitation from the nerve endings is transmitted to the spinal cord, where the defecation center is located at the lumbar level. From this center, the excitation spreads to the cerebral cortex, causing a specific sensation and urge to defecate.

But even in the presence of an urge to defecate, fecal masses are held in the rectum due to the constant tonic tension of the two anal sphincters - external and internal. The act of defecation occurs reflexively. Irritation of the receptors of the wall of the rectal ampulla causes involuntary relaxation of its internal sphincter, peristaltic contractions of the muscles of the colon and rectum walls. Then there is a voluntary relaxation of the external sphincter of the rectum. Emptying of the rectum is facilitated by holding the breath, tension of the abdominal press, diaphragm and pelvic floor muscles.

When the spinal cord is damaged (inflammatory processes, injuries, tumors) above the center of defecation, the influences coming from the higher structures of the brain, especially the cerebral cortex, are interrupted, and defecation becomes involuntary. Involuntary defecation can occur with strong emotional influences (for example, with fear). Some diseases of the gastrointestinal tract (dysentery, tumors, ulcers) can cause a violation of the act of defecation, which manifests itself in the form of diarrhea or constipation.

**Classification**

- Diarrhea.
- Functional constipation.
- Chronic colo-proctogenic stasis.

By form:

- Cologenic
- Proctogenic
- Mixed form

By degree of compensation:

- Compensated
- Subcompensated
- Decompensated
- Obstructive defecation.

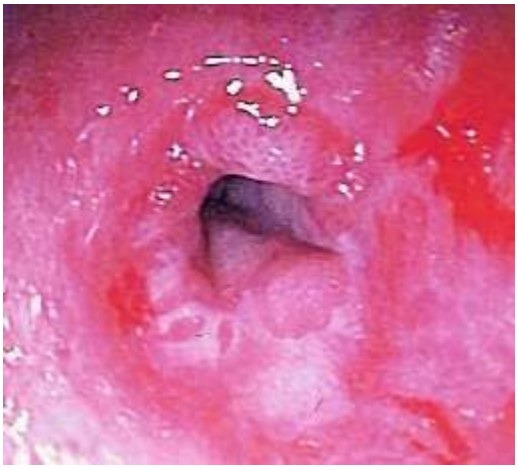
Most often, diarrhea, as one of the clinical manifestations, is found in such diseases of the colon as: chronic inflammatory diseases (ulcerative colitis, Crohn's disease, pseudomembranous colitis), familial adenomatous polyposis. The presence of pathological impurities in the feces (blood, mucus, pus) is characteristic. It should be noted that in the enterocolitic clinical form of colon cancer, alternating diarrhea and constipation is characteristic. The features of obstructive defecation are the presence of the phenomenon of obstruction of the colon opening with impaired evacuation of intestinal contents due to rectocele, internal intussusception and prolapse of the rectum, enterocele and sigmoidcele in combination with discoordination and/or atrophy of the pelvic floor muscles. In the absence of an organic cause, delayed movement and

evacuation of the colonic contents (constipation) can most likely be explained by the presence of chronic colo-proctogenic stasis in various degrees of compensation.

**Diagnostic methods**

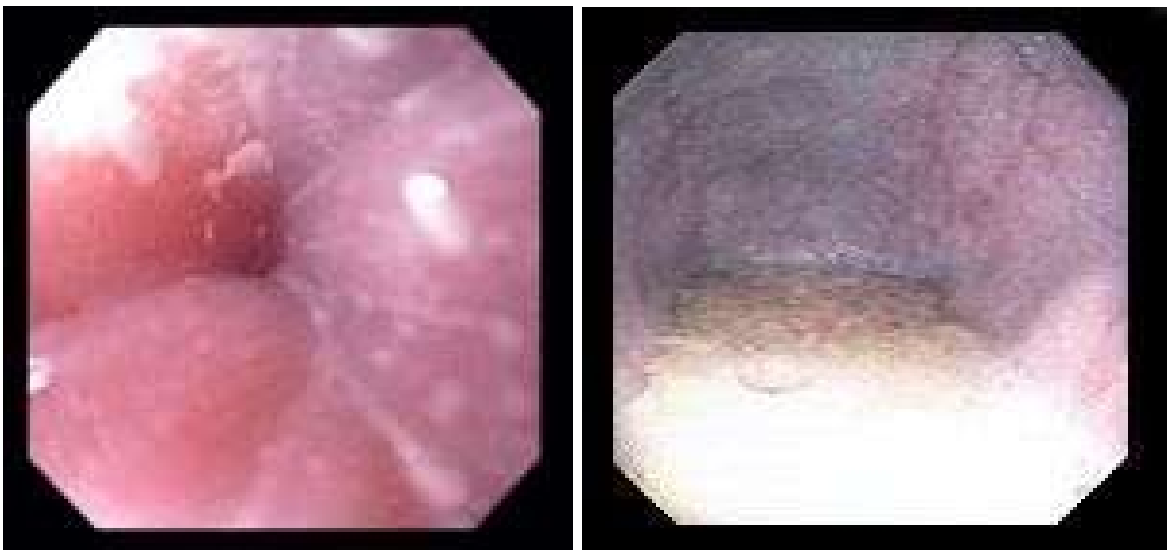
Dysphagia:

- Collection and analysis of anamnesis.
- Review of previous medical data and diagnoses, assessment of neurological status and the need for care.
- Physical examination.
- Laboratory tests of blood and urine.
- Instrumental methods of examination - esophagogastroscopy with endosonography, biopsy and histopathological examination, esophageal radiography, esophageal tonometry, esophageal and gastric pH-metry, esophageal scintigraphy, contrast-enhanced computed tomography.
- When excluding esophageal dysphagia (peripheral-organic), psycho-neurological examinations (including MRI of the brain and spine) and consultation with relevant specialists are necessary for the diagnosis of preesophageal (neurogenic) dysphagia.



**Gastroesophageal reflux disease complicated by peptic stricture of the esophagus in the region of the lower esophageal sphincter**

**Stage III achalasia of the esophagus. Esophagogastrography. Suprastenotic dilatation of the esophagus is determined, and a "mouse tail" sign is detected radiographically in the area of esophageal narrowing in the projection of the lower esophageal sphincter.**



**1** **2**

**Esophageal achalasia of stages III (1) and IV (2). Esophagoscopy. Narrowing of the cardiac part of the esophagus in the projection of the lower esophageal sphincter (1), significant suprastenotic dilatation of the esophagus with accumulation of food and fluid in it (endoscopic sign of cardiac stenosis).**

Vomiting:

- Collection and analysis of anamnesis.
- Familiarization with previous medical data and diagnoses, assessment of neurological status and the need for care.
- Physical examination.
- Laboratory tests of blood and urine.
- Instrumental methods of examination - esophagogastroduodenoscopy with endosonography, biopsy and pathohistological examination, balloon enteroscopy, radiography of the esophagus, stomach and duodenum, according to indications - determination of the features of the transit of the

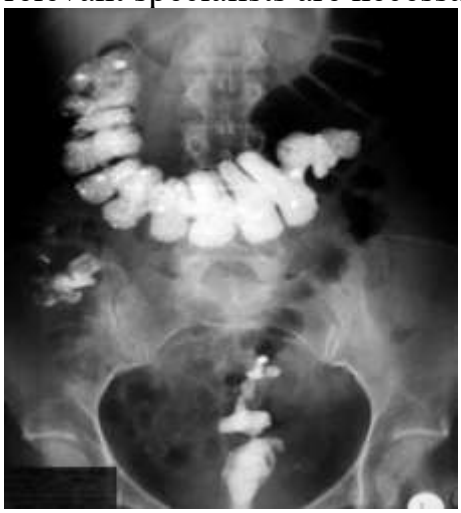
contrast medium through the intestinal canal, computed tomography with contrast enhancement.

- If an organic cause is excluded (the presence of an organic obstacle to the advancement of contents through the digestive canal), psycho-neurological examinations (including MRI of the brain, spine), consultation of relevant specialists are necessary.

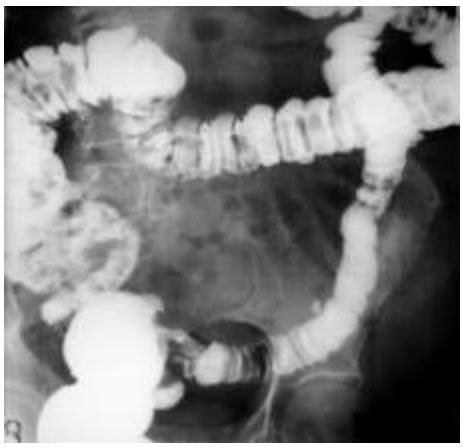
Peptic ulcer of the duodenum complicated by subcompensated cicatricial stenosis of the source. X-ray of the stomach, delay of contrast medium (barium sulfate) in the stomach for up to 14 hours.

Disorders of the act of defecation

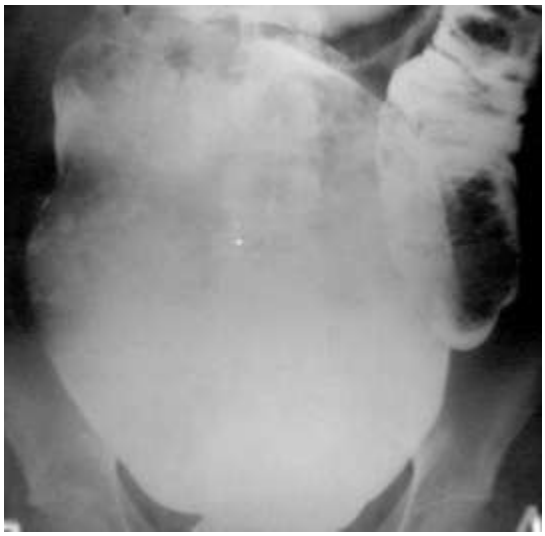
- Collection and analysis of anamnesis.
- Familiarization with previous medical data and diagnoses, assessment of neurological status and the need for care.
- Physical examination.
- Laboratory tests of blood and urine.
- Instrumental methods of examination - esophagogastroduodenoscopy, proctological examination, colonoscopy, survey radiography of the abdominal cavity, irrigography (in two phases), determination of the features of the transit of the contrast medium through the intestinal canal, functional studies of the rectum (including proctography, MRI-defecography and computer pneumokinesometry), computer tomography with contrast enhancement.
- When excluding an organic cause (the presence of an organic obstacle to the movement of contents through the colon), chronic colonic, proctogenic stasis, psycho-neurological examinations (including MRI of the brain, spine), and consultation with relevant specialists are necessary.



**Chronic decompensated colonic stasis. Colonic transit study of contrast medium (barium sulfate), colonic stasis up to 120 hours, grade II colonoptosis, clinical colon emptying has not yet occurred.**



**Endophytic cancer of the distal sigmoid colon, stage III, complicated by chronic subcompensated obturation obstruction of the colon.**



**Chronic decompensated proctogenic stasis, inert rectum.**

Proctogram. Significant dilation of the ampullary part of the rectum is noted in the absence of a natural urge to defecate. The conditional urge to defecate is abdominal pain that occurs when 870 ml of contrast medium (barium sulfate) is injected transanally into the rectum.

**Treatment**

Not pathological syndromes (dysphagia, vomiting and impaired defecation) is treated, but diseases, the clinical signs of which are the indicated syndromes. The differentiated choice of treatment method depends on the nature of the disease, the features of its course and the presence of complications.

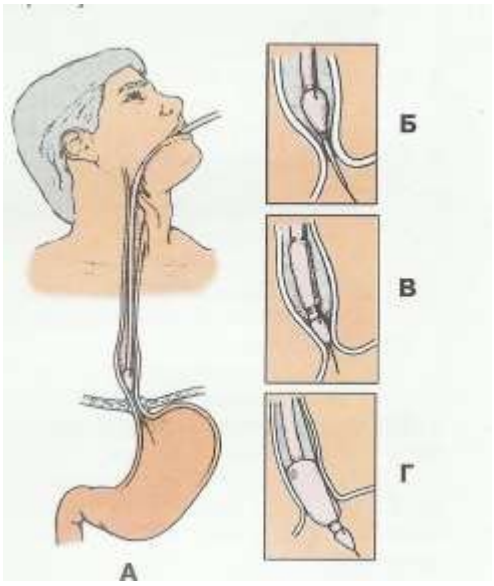
Thus, if the cause of dysphagia is a cancer of the esophagus, planned surgical treatment is indicated; cicatricial stricture of the esophagus is initially treated conservatively by conducting therapeutic gurgling, if they are ineffective, planned surgical intervention is performed; with achalasia of the esophagus in stages I and II, conservative gurgling is performed, including using cardiodilation, and in stages III and IV, planned surgical treatment is indicated.

If the cause of vomiting is a chronic complication of peptic ulcer disease in the form of subcompensated cicatricial stenosis of the pyloric canal or duodenum, planned surgical treatment is indicated. In case of acute complications of peptic ulcer disease in the form of acute bleeding from the ulcer, the presence of hematemesis,

endoscopically detected type I bleeding from the ulcer, urgent surgical treatment is indicated. In the presence of vomiting in acute surgical diseases of the abdominal cavity organs (acute appendicitis, acute cholecystitis, acute pancreatitis, etc.), conservative or surgical treatment of the specified diseases is indicated.

In the presence of diarrhea, as a clinical sign of chronic severe total ulcerative colitis of the II degree of activity, intermittent course, conservative treatment is indicated, and in the presence of irreversible morphological changes in the colon wall, refractoriness to conservative therapy, and especially in the event of acute or chronic complications of the disease, surgical treatment is already indicated. In the presence of familial adenomatous polyposis, only surgical treatment is indicated.

In the presence of a syndrome of defecation disorder, the cause of which is obstructive defecation with impaired evacuation of intestinal contents due to rectocele, internal intussusception and rectal prolapse, enterocele and sigmoidcele in combination with discoordination and/or atrophy of the pelvic floor muscles, or chronic decompensated colon or proctogenic stasis, only surgical treatment is indicated. At the same time, in the presence of chronic compensated or subcompensated colon or proctogenic stasis, conservative treatment is justified. Of course, if the cause of the defecation disorder is a cancerous tumor of the colon, only surgical treatment is indicated (in this case, the defecation disorder should be treated as chronic obstructive obstruction of the colon).




**Esophageal achalasia stage I, II. Balloon cardiodilation**

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**Tests**

21. What are the main causes of esophageal dysphagia?
    A. Acute esophagitis
    B. Achalasia of the esophagus
    C. Esophageal cancer
    D. Foreign body of the esophagus
    E. Scarring stricture of the esophagus

    Explanation: The main causes of dysphagia are achalasia of the esophagus (B), cancer of the esophagus (C) and scarring stricture of the esophagus (E), which is explained by the occurrence of an organic obstacle in the esophagus with impaired food movement in the form of a tumor or scar or functional (stage I-II achalasia) narrowing of the esophagus.
22. What are the main causes of the combination of dysphagia with odynophagia (pain behind the sternum during swallowing)?
    A. Foreign body in the esophagus
    B. Chemical burn of the esophagus
    C. Acute esophagitis
    D. Esophageal cancer
    E. Scarring stricture of the esophagus

    Explanation: The main causes of the combination of dysphagia and odynophagia are chemical burn of the esophagus (B) and acute esophagitis (C), which is explained by the presence of an inflammatory process in the wall of the esophagus (pain as a sign of inflammation) and its irritation during swallowing.
23. What are the main modern diagnostic methods that allow determining the cause of dysphagia?
    A. Esophagogastroduodenoscopy
    B. Esophageal radiography
    C. Ph-monitoring of the esophagus
    D. Computed tomography of the chest cavity
    E. Magnetic resonance imaging of the chest cavity

    Explanation: The main diagnostic methods that allow determining the cause of dysphagia are esophagogastroduodenoscopy (A) and esophageal radiography (B), which is explained by the modern capabilities of endoscopic technology (including endosonoscopy, high-magnification endoscopy, endoscopic microscopy), as well as radiological determination of the features of the movement of contents through the esophagus.
24. Vomiting, as a complex reflex act, is caused by irritation of which receptors?

A. Root of the tongue
B. Mucous membrane of the stomach
C. Gallbladder
D. Skin
E. Pain

Explanation: Vomiting is caused by irritation of the receptors of the root of the tongue (A), mucous membrane of the stomach (B) and gallbladder (C), which is transmitted to a special vomiting center in the medulla oblongata, which coordinates the coordinated contraction of the muscles of the stomach, as well as the movements of the respiratory muscles and abdominal press.

25. Vomiting caused by direct irritation of a special vomiting center is possible in:
A. Damage to the central nervous system
B. Increased intracranial pressure
C. Various intoxications
D. Uncomplicated diverticular disease of the colon
E. Penetrating duodenal ulcer

Explanation: Vomiting caused by direct irritation of a special vomiting center is possible in damage to the central nervous system (tumors, trauma, meningitis) (A), increased intracranial pressure (B) and various intoxications (overdose, toxemia of pregnancy, uremia) (C).

26. In which surgical diseases does vomiting occur as a clinical symptom?
A. Acute cholecystitis
B. Acute pancreatitis
C. Acute small bowel obstruction
D. Uncomplicated inguinal hernia
E. Chronic hemorrhoids

Explanation: Vomiting as a symptom of a surgical disease occurs in acute cholecystitis (A), acute pancreatitis (B) and acute small bowel obstruction (C), is reflex in nature and is regulated by a special vomiting center in the medulla oblongata.

27. In the presence of hematemesis (vomiting of unchanged or slightly changed blood), which disease should be thought of first?
A. Ulcerative bleeding
B. Mallory-Weiss syndrome
C. Chronic atrophic gastritis
D. Bleeding from esophageal varices
E. Cicatricial stenosis of the pyloric canal

Explanation: In the presence of hematemesis, one should first consider ulcerative bleeding (A), Mallory-Weiss syndrome (B) and bleeding from esophageal varices, which is explained by the presence of a morphological defect (rupture or ulcer) in the mucosa-submucosal sheath of the esophagus, stomach or initial part of the duodenum.

28. What is the normal frequency of defecation in the world today?
A. Once a day
B. From one to three times a day
C. From 1-3 times a day to 2-3 times a week
D. From two to three times a week

E. From two to three times a day

Explanation: The normal frequency of defecation accepted in the world is from 1-3 times a day to 2-3 times a week. Only 30% of people defecate 2-3 times a week, and up to 90% of them are women, this is the so-called slow-transient type of evacuation, in the intervals between such more "loose" defecations people feel normal. If between "loose" defecations a person feels pain in the abdomen, its bloating, deterioration of the general condition, a thorough diagnosis of the cause of the delay in evacuation is necessary.

29. The main causes of defecation disorders are:

A. Obstructive defecation syndrome

B. Chronic colo-proctogenic stasis

C. Ulcerative colitis

D. Colon cancer

E. Diverticular disease of the colon

Explanation: The main causes of defecation disorders are obstructive defecation syndrome (A), chronic colo-proctogenic stasis (B) and colon cancer (D), which is explained either by the presence of pelvic dissection syndrome (prolapse or prolapse of the pelvic organs due to weakness of the pelvic floor), or by a violation of the plastic and kinetic properties of the colon and rectum, due to congenital or acquired disorders of the vascular and nervous trophism of the intestinal wall, or in the presence of a mechanical obstacle to the advancement of the contents in the form of a tumor.

30. What are the main special research methods that should be used for differential diagnosis of the causes of defecation disorders?

A. Irrigography

B. Colonoscopy

C. Study of the transit of contrast medium through the digestive tract

D. Functional studies of the rectum

E. Proctological examination

Explanation: The main special methods of differential diagnosis of disorders of the act of defecation (constipation or chronic obstructive obstruction of the colon) are irrigography (A), colonoscopy (B) and proctological examination (E). This is explained by the diagnostic capabilities of these research methods in determining the organic or functional cause of the disorder of the act of defecation.

## 3.2. Intestinal obstruction syndrome. Etiology. Pathogenesis. Clinic. Diagnosis. Differential diagnosis. Treatment tactics.

**Overview.**

Acute intestinal obstruction (AC) remains one of the most pressing problems of modern emergency surgery. Among acute surgical diseases of the abdominal cavity, it occurs with a frequency of 4.2 - 9.4%. Every year, about 4% of laparotomies are performed in the world due to acute intestinal obstruction. The incidence of acute intestinal obstruction in Ukraine averaged 25 cases per 100,000 population. Mortality rates remain high and differ significantly depending on the etiology of acute intestinal obstruction. Lethality in non-neoplastic forms of acute intestinal obstruction is 4-32%, while in tumor genesis it reaches 22-47%.

The leading factors that determine the high percentage of mortality in acute intestinal obstruction are late hospitalization, a high frequency of diagnostic mistakes, the age of the patient and the presence of concomitant pathology, unjustified prolongation of the terms of conservative therapy, intraoperative and postoperative complications

Intestinal obstruction may be defined as failure of propulsion of intestinal contents aborally. The condition occurs in many forms in both the small and large intestine, due to either mechanical obstruction or a motility problem caused by neuromuscular failure or ischemia. Neuromuscular failure is frequently associated with inflammation in the peritoneal cavity or in the retroperitoneum. This type of intestinal obstruction, where the intestinal lumen is not compromised, is also known as adynamic ileus. Pain originating from distension of the intestine, as occurs in bowel obstruction.

**Educational aims:**

- Interrogation and clinical inspection of patients with bowel obstruction.
- To determine the etiologic and pathogenic factors of bowel obstruction.
- To find out the types of bowel obstruction, the clinical features, different variants of manifestation and complications.
- To develop a plan of examination of the patients with bowel obstruction.
- To estimate laboratory data, results of X-ray, ultrasound examination, computed tomography (CT), magnetic resonance imaging (MRI) of the intestines.
- To make a differential diagnosis, form a diagnosis of intestinal obstruction by reasoning.
- To prescribe the treatment for patients with the bowel obstruction.
- To justify indications for surgical treatment of patients with the bowel obstruction.
- To cure of the patients with bowel obstruction after operations.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomy -physiological information about of the intestines.
- Classifications of bowel obstruction.
- Mechanisms of development of bowel obstruction.
- Clinical picture of bowel obstruction.
- Methods of diagnostics of bowel obstruction.
- Principles of treatment of bowel obstruction.
- Features of surgical interventions for the different types of bowel obstruction.

**A student must be able to:**

- Collect and estimate the complaints of patient with the bowel obstruction, information of anamnesis, to conduct physical research and correctly interpret the results obtained.

- Define the rational volume of laboratory and instrumental methods of research. Correctly interpret the results of clinical analyses, X-ray, Ultrasound diagnostics, computed tomography, magnetic resonance imaging.
- Define indications for operation and other methods of treatment of patients.
- Perform pre-operative preparations of patients. Conduct post-operative care

**Terminology.**

| Term | Definition |
|---|---|
| Mechanical obstruction | means that luminal contents cannot pass through the gut tube because the lumen is blocked. |
| Functional obstruction | Luminal contents fail to pass because of disturbances in gut motility that prevents coordinated peristalsis from one region of the gut to the next. |
| Strangulated obstruction | Blood flow to the obstructed segment is compromised, and tissue necrosis and gangrene are imminent. Strangulation usually implies that the obstruction is complete, but some forms of partial obstruction can also be complicated by strangulation. |
| The Vala's sign | Is the elastic sausage-shaped deformity of the bowel. |
| Sklarov's sign | Is the sound of intestinal splash. |
| Kywul's sign | Is the sound on percussion above the exaggerated bowel. |
| Spasokukotsky's sign | Is "sound of falling drop". |
| Hochenegg's sign | incompletely closed anus in combination with balloon expansion of ampulla of rectum |
| Inflammatory bowel disease (IBD) | is a term generally used to denote two diseases of unknown etiology with similar general characteristics: ulcerative colitis and Crohn's disease |

**Content:**

**Etiology**

| | Small Intestine | Colon |
|---|---|---|
| **Luminal** | Gallstone ileus<br>Foreign body<br>Worms | Fecal impaction<br>Foreign body |
| **Bowel wall lesions** | Tumor<br>Strictures<br>Intussusceptions<br>Radiation enteritis | Tumor<br>Strictures<br>Diverticular disease<br>Intussusceptions |
| **Extrinsic compression** | Adhesions<br>Hernias<br>Volvulus<br>Extrinsic tumor<br>Extrinsic inflammation | Volvulus<br>Extrinsic tumor<br>Hernias |

**Classification** ***(by D.P.Chuhrienko, 1958)***

**Acute intestinal obstruction is divided:**

**I.** ***According to morphofunctional signs.***

**1. Dynamic intestinal obstruction:**

- paralytic;
- spastic;
- Inadequate blood supply of intestines(embolism, thromboembolism).

**2. Mechanical intestinal obstruction:**

- strangulation (volvulus, node formation)
- obturation (closing of bowel lumen in side or compression from outside)
- mixed (invagination, adhesive [intestinal] obstruction).

**II.** ***According to clinical features.***

- Acute.
- Chronic.

**III.** ***According to the level of obstruction.***

- Small - bowel obstruction.
- Large -bowel obstruction.

**IV.** ***According to the passing of intestinal contents.***

- Complete.
- Partial.

**V.** ***According to the origin.***

- Innate.
- Acquired.

**VI.** ***According to development of pathological process.***

- Stage of acute violation of intestinal motility.
- Stage of hemodynamic disorders of bowel wall and its mesentery.
- Stage of peritonitis.

**Pathophysiology**

In acute obstruction, there is an accumulation of intestinal contents (liquid and gas) before of the site of obstruction. Gradually, this leads to stretching of the intestine and an increase intraluminal and intramural pressure. When the intramural pressure becomes critical, a violation occurs intramural perfusion. This contributes to the development of intestinal ischemia and further development of wall necrosis.

When a part of the intestinal lumen is blocked, some volume of intestinal contents passes through. At the same time, pathophysiological changes develop not as quickly as with complete blockage. Ischemic changes develop most quickly during strangulation (for example, when volvulus), when the mesentery and vessels are compressed

**Clinical picture.** Symptoms of intestinal obstruction include abdominal pain, nausea and vomiting, as well as constipation and bloating. Present of gases and stool with obstruction may be in partial obstruction. Strangulated intestinal obstruction. Blood supply disturbance and ischemia (compression of mesenteric vessels) is the characteristic feature of this form of intestinal obstruction. It determines the pathomorphologic changes and clinical signs of disease. Acute pain syndrome and

ischemic disorders in the wall of bowel cause necrotic changes in area of bowel affected by the disease. It is accompanied by the progressive worsening of the patient condition and lead to endotoxicosis.

Strangulation obstruction of the small or large intestine is accompanied by symptoms and signs that suggest peritonitis. Large fluid shifts and systemic toxicity are imminent or have already occurred. These signs include abdominal tenderness or involuntary guarding localized to the area of the strangulated loop of bowel, decreased urine output, fever, and tachycardia. Obturation intestinal obstruction, unlike strangulated, does not progress quickly.

**HIGH SMALL BOWEL OBSTRUCTION**. The presenting symptoms of high small bowel obstruction are colicky upper abdominal pain and profuse, bilious vomiting. The onset of vomiting is close to the onset of pain, and the vomitus is nonfeculent. On physical examination, vital signs are normal except late in the course, when dehydration is present. Distension, if present, is not prominent. Abdominal tenderness is absent, and bowel sounds are hyperactive, the crescendos of which coincide with attacks of colicky pain.

**LOW SMALL BOWEL OBSTRUCTION**. Colicky, midabdominal pain, vomiting, and abdominal distension are the presenting symptoms of low small bowel obstruction. The interval between onset of pain and vomiting lengthens as the site of obstruction is more distal. Vomiting may be feculent. No gas or feces will have been passed through the rectum for variable periods of time. Abdominal tenderness is minimal or absent, and rectal examination is normal.

**LARGE BOWEL OBSTRUCTION.** Colon cancer and diverticulitis account for nearly 90% of cases of large intestinal obstruction, with colon cancer alone responsible for 65% to 70% of these. The most frequent site of obstruction from either disease is the sigmoid. Other causes of colon obstruction include inflammatory bowel disease, postanastomotic strictures, benign tumors, and fecal impaction. Symptoms and signs include those of mechanical obstruction as well as those of underlying disease producing the obstruction. The pain is crampy and suprapubic. Vomiting is a late symptom, and the vomitus is typically feculent. Constipation and obstipation are constant features. Abdominal distension can be prominent, especially in sigmoid volvulus. Bowel sounds are hyperactive and high pitched. Superimposed on these symptoms and signs are those of the underlying disease producing the obstruction. Patients with colon cancer may complain of a change in bowel habits and rectal bleeding. Patients with diverticulitis may have a history of alternating diarrhea and constipation and usually have signs of the inflammatory process: fever, tachycardia, and tenderness or mass in the left lower quadrant

**SIGMOID VOLVULUS.** Three conditions promote sigmoid volvulus: a redundant sigmoid, long sigmoid mesentery with a narrow base, and fecal loading due to chronic constipation. The condition tends to occur in the elderly, in those who are bedridden, and in those receiving psychotropic medication for a psychiatric disorder. The bowel twists counterclockwise about its long mesentery. A complete twist of 360° leads to occlusion of not only the bowel lumen but also of the vascular pedicle in the mesentery. If the obstruction is not reversed promptly, sigmoid gangrene and perforation ensue. Usually, abdominal distension is very prominent, and colicky

suprapubic pain develops. Pain is usually not an important symptom, but abdominal distension can be massive and lead to cecal perforation. Cecal Volvulus Cecal volvulus is much less common than sigmoid volvulus, occurring with half the frequency. A predisposing condition is incomplete embryologic fixation of the cecum, which results in hypermobility of the organ. Distension and colicky pain develop in the midabdomen. The patient often has a previous medical history of similar but milder attacks.

**Diagnostics.** Diagnostic procedures should be aimed at: separating mechanical from dynamic obstruction, identifying the causes of obstruction; distinguishing complete from partial obstruction, distinguishing obturation from strangulation obstruction. History is also important - previous abdominal surgery (presence of adhesions), hereditary abdominal oncology, inflammatory bowel disease, which may contribute to the etiology of obstruction.

During the examination, it is necessary to conduct a thorough diagnosis of inguinal and femoral hernias (especially in obese patients). It is necessary to examine the stool for the presence of blood.

Patients with palpation note both a soft abdomen and resistance of the anterior abdominal wall, and percussion— high tympanitis. Auscultatively, at the beginning of the disease, there are increased peristaltic noises, then — a gradual fading of peristalsis (positive Mondor symptom, "noise of the beginning, silence of the end").

There are other symptoms pathognomonic for intestinal obstruction. Val's symptom — a limited elastic sausage-like formation. Sklyarov's symptom — the sound of intestinal slapping. Kivul's symptom — a metallic sound over a distended intestine. Schlange's symptom — intestinal peristalsis that occurs after palpation of the abdomen. Spasokukotsky's symptom — "the sound of a falling drop". Grekov's symptom (Obukhov Hospital) — gaping of the anus combined with a balloon-like expansion of the rectal ampulla.

During a X-ray scopy or radiography of the abdominal cavity, fluid and gas levels are noted in the loops of the intestines - Kloiber's cups.

Computed tomography (CT) of intestinal obstruction reveals an area of dilated bowel before obstruction and colapse of the bowel after obstruction, and free fluid in the abdominal cavity. The bowel wall appears thickened, air in the bowel wall and poor contrast uptake in the wall suggests ischemic damage. CT can identify the cause of the obstruction and also allows for assessment of the condition of other abdominal organs.

Ultrasound in intestinal obstruction provides the same data as CT, but allows you to detect pendulum-like movements of intestinal contents.

Examination of the intestines with contrast, which can be administered antegradely or retrogradely. The contrast is swallowed or introduced through a tube into the stomach. Several radiographs are taken as the contrast moves caudally through the intestines. With a barium enema, 200-250 ml of barium solution is administered, then several radiographs are taken to monitor how the intestine fills and empties. This reveals strictures and filling defects.

In obstruction, laboratory studies do not play a direct role in diagnosis, but are helpful in evaluating complications such as dehydration, strangulation, and sepsis.

Other abdominal conditions, such as appendicitis, diverticulitis, perforated peptic ulcer, cholecystitis, or choledocholithiasis, can usually be distinguished from bowel

obstruction by clinical examination and basic laboratory data. Bowel obstruction can complicate any of these abdominal conditions. Thus, the presence of another abdominal process does not exclude the complication of bowel obstruction, and the symptoms of bowel obstruction do not exclude other conditions.

**Therapy**

During the first 1.5-2 hours after the patient's hospitalization in the hospital, a comprehensive conservative therapy is carried out, which has a differential diagnostic value and by its nature can be a preoperative preparation.

It is aimed at preventing complications associated with pain shock, correcting homeostasis and at the same time represents an attempt to eliminate intestinal obstruction by non-operative methods.

1. Measures aimed at combating abdominal pain shock include the administration of antispasmodics (baralgin, spazmoverin, no-shpa), paranephral novocaine blockade, in severe cases, neuroleptanalgesia (droperidol, fentanyl). In patients with severe pain syndrome and spastic intestinal obstruction, a positive effect can also be achieved with the help of epidural anesthesia.

2. Elimination of hypovolemia with correction of electrolyte, carbohydrate and protein metabolism is achieved by the introduction of salt blood substitutes, 5-10% glucose solution, gelatin, albumin and blood plasma. There are several methods suitable for use in emergency surgery to calculate the amount of fluid required to level hypovolemia.

3. Correction of hemodynamic parameters, microcirculation and detoxification therapy is carried out using intravenous infusion.

4. Decompression of the gastrointestinal tract is carried out using a nasogastric tube, gastric lavage, and also performing a siphon enema. It is possible to eliminate intestinal obstruction with such conservative means in 20-30% of patients with mechanical intestinal obstruction.

Patients with dynamic (paralytic) intestinal obstruction should be stimulated with intestinal peristalsis. It is performed necessarily after infusion therapy and correction of hypovolemia. C

**Surgical treatment** of intestinal obstruction should include the following points that must be performed during surgery:

After laparotomy, an abdominal inspection is performed to determine the cause of intestinal obstruction and the viability of the intestines. The absence of pathological processes after the inspection requires an inspection of the places of formation and pinching of internal hernias: internal inguinal and femoral rings, obturator foramina, pockets of the ligament of Treitz, the foramen magnum, the diaphragm and the paraesophageal foramen. To determine the level of obstruction, a visual assessment of the transverse size of various sections of the intestine is used. Above the level of obstruction (obstruction), intestinal loops are always full of contents, and below they collapse.

Elimination of the causes of non-conduction (cutting the adhesion that compresses the intestine, straightening the volvulus and knotting of the loops, disinvagination, removal of obstructing tumors, etc.). If the cause of obstruction is a cancerous tumor, various surgical options can be used. In the presence of a tumor of the right half of the

colon (caecum, ascending colon, hepatic angle) without signs of peritonitis, a right-sided hemicolectomy with the imposition of a primary anastomosis is performed.

In case of peritonitis, a serious condition of the patient, it is necessary to impose an ileostomy, lavage, sanitation and drainage of the abdominal cavity. Patients with inoperable tumors (the presence of metastases) who do not have peritonitis are imposed with an ileotransverse anastomosis.

A slightly different approach to choosing the method of surgery in the presence of tumors of the left half of the colon and rectum.

In most cases, with cancer of the left half of the colon and rectal cancer with obstructive intestinal obstruction, it is necessary to perform a Hartmann operation, which consists in resection of the affected segment of the intestine, suturing its distal end and removing the proximal end of the intestine in the form of a single-barrel colostomy. After such an operation, there is a possibility of restoring the patency of the colon. An important principle of eliminating intestinal obstruction should be the choice of a gentle, but sufficiently radical method of eliminating the mechanical obstruction of the intestine. The scope of this intervention can be different - from extensive resection of the intestine to the imposition of an unloading intestinal fistula or bypass interintestinal anastomosis. In case of intestinal non-viability, which can be determined visually by changing the color, peristalsis, pulsation of the mesenteric arteries, Dopplerography, spectroscopy, resection of the non-viable area is performed from 30-40 cm of the afferent and 15-20 cm of the afferent section with the imposition of a "side-to-side" or "end-to-end" anastomosis.

Sanitation and drainage of the abdominal cavity are performed by generally accepted methods using washing with antiseptic solutions, electric suction devices and wipes. The abdominal cavity should be drained from four places: in both iliac regions and both hypochondria, preferably with paired synthetic drainage tubes.

**Colonic Pseudo obstruction** Etiologic factors Acute pseudo obstruction of the colon, also known as Ogilvie's syndrome, is an often painless paralytic ileus of the large bowel characterized by rapidly progressive abdominal distention. Plain radiographs of the abdomen may reveal air in the small bowel and distention of discrete segments of the colon (cecum or transverse colon) or of the entire abdominal colon. Although the distention of the colon is not caused by mechanical obstruction, the wall of the bowel, particularly that of the cecum, can become sufficiently distended so that its blood supply is compromised. Gangrene, perforation, peritonitis, and shock can follow. Major risk factors for the development of Ogilvie's syndrome include severe blunt trauma, orthopedic trauma or procedures, acute cardiac events or coronary bypass surgery, acute neurologic events or neurosurgical procedures, and acute metabolic derangements. Only 5% of cases develop in the absence of another conditions. Diagnosis. The diagnosis is usually apparent from plain films. In doubtful cases, and when bowel necrosis is not a significant worry, a gentle Hypaque contrast enema can establish the nonmechanical nature of the dilatation. Colonoscopy can be both therapeutic and diagnostic. Features suggesting the complication of bowel ischemia include localized tenderness, leukocytosis, metabolic acidosis, evidence of sepsis, and a rapidly deteriorating clinical course. Management Initial management includes resuscitation and correction of underlying metabolic or electrolyte imbalances. A

nasogastric tube is helpful if the patient is vomiting and can prevent swallowed air from passing distally. When bowel ischemia is suspected, surgery is indicated. If bowel necrosis is found, the affected segment is resected and an ileostomy or colostomy established. If the bowel is viable, a cecostomy is placed to vent the colon and prevent distention. If distention is painless and the patient shows no signs of toxicity or bowel ischemia, expectant management is successful in approximately 50% of cases. If the distention worsens so that the cecal diameter increases beyond 10 to 12 cm, or if it persists for more than 48 hours, colonoscopy is recommended. Endoscopic decompression is successful in 60% to 90% of cases but colonic distention can recur in up to 40%. Rectal tubes are ineffective in managing distention of the proximal colon. Such tubes can be useful in promoting passage of air and feces after colonoscopy, but should not be used as temporizing measures to avoid colonoscopic decompression. In anecdotal reports, prokinetic agents such as cisapride and erythromycin. Ogilvie's syndrome with success. Successful resolution of pseudo obstruction can be treated with sympatholytic agents or spinal sympathetic block. The efficacies of these modalities have not been systematically evaluated. In the most recent studies, the sympatholytic agent, neostigmine, has been advocated if a 24-hour interval of conservative measures (nasogastric suction, intravenous fluids, nothing by mouth) has failed to improve symptoms. Serious cardiovascular complications can occur and patients require telemetry. In addition, underlying factors (sepsis, electrolyte abnormalities, and ileus-promoting medications) should be addressed to obtain the earliest and maximum benefit.

Special forms of obstructive intestinal obstruction include **obstruction caused by gallstones**. The latter enter the small intestine as a result of pressure in the walls of the gallbladder and the intestine adjacent to it. It should be borne in mind that intestinal obstruction can be caused by a concrement with a diameter significantly smaller than the lumen of the intestine. The mechanism of such a phenomenon is associated with the irritating effect of bile acids on the intestinal wall. The latter responds to this with a spasm, as a result of which the stone is tightly wedged into its lumen. The development of intestinal obstruction of cholelithiasis is always preceded by an attack of colic and the clinic of acute cholecystitis. It is characteristic that in the process of the development of the disease, the pain caused by acute cholecystitis subsides, after which a new pain appears, characteristic of another pathology - intestinal obstruction.

**Hemodynamic intestinal obstruction (MESENTERIC ISCHEMIA)** develops on the basis of embolism or thrombosis of mesenteric arteries and vein thrombosis, there may be mixed forms. Embolism of mesenteric arteries most often occurs in patients with heart disease (mitral and aortic defects, myocardial infarction, warty endocarditis) and is manifested by lesions, mainly, of the superior mesenteric artery. This is primarily facilitated by its wide lumen and its departure from the aorta at an angle of 45 °. The onset of the disease is usually acute, with nausea, sometimes vomiting. In the first hours, a picture of acute abdominal ischemic syndrome appears, which is often accompanied by shock (rapid pulse, decreased arterial and pulse pressure, cold sweat, cyanosis of the mucous membranes and acrocyanosis). Patients become irritable, restless, take a forced knee-elbow position or lie on their side with bent legs.

On examination, the abdomen remains symmetrical, its anterior wall is soft, increased peristalsis is heard from the first minutes for 1-2 hours (hypoxic stimulation of peristalsis), later it gradually fades away ("grave silence"). Over time, the symptoms of intoxication and peritonitis rapidly increase. At the beginning of the disease, patients have gas and stool retention, later diarrhea with blood in the stool. When the latter is difficult to establish macroscopically, it is necessary to examine the intestinal lavage fluid.

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**Tests**

1. The most informative investigation in acute bowel obstruction is
   A. X-ray of abdomen
   B. Ultrasound
   C. Colonoscopy
   D. Anoscopy
   E. Barium enema
2. All of statements about Cloiberg's cup are correct EXEPT
   A. Sign of acute bowel obstruction
   B. Has a form of the inverted bowl
   C. Can be observed after an enema
   D. Is formed by the level of liquid and air
   E. Cloiberg's cup is free air in an abdomen
3. Cloiberg's cup in small bowel obstruction is
   A. Wide and tall
   B. Narrow and low
   C. Wide and low
   D. Narrow and tall
   E. Is located on each side of abdomen
4. Cloiberg's cup in large bowel obstruction is
   A. Wide and tall
   B. Narrow and low
   C. Wide and low
   D. Narrow and tall
   E. Narrow or wide and tall
5. Which of the following would be expected to stimulate intestinal motility?
   A. Fear
   B. Gastrin
   C. Secretin
   D. Acetylcholine
   E. Prolactin
6. For a symptomatic partial duodenal obstruction secondary to an annular pancreas, the operative treatment of choice is

A. A Whipple procedure
B. Gastrojejunostomy
C. Vagotomy and gastrojejunostomy
D. Partial resection of the annular pancreas
E. Duodenojejunostomy

7. Operative planning and preoperative counseling for a patient with a rectal carcinoma can be best provided if the patient is staged before surgery by
A. Rigid proctoscopy
B. Barium enema
C. MRI of the pelvis
D. CT scanning of the pelvis
E. Rectal endosonography
8. A 66-year-old woman was hospitalized for pancreatic adenocarcinoma. She developed constipation and bloating. Which of the following tactics is more appropriate?
A. Cecostomy or colostomy
B. Correction of metabolic disorders, Withdrawal of narcotics and anticholinergics.
C. Digital removal of fecal stones from the rectum
D. Examination of the intestine using a colonoscope andendoscopic treatment
E. Elimination of volvulus with subsequent resection and colopexy
9. Criteria suggesting viability of intestine are all EXEPT:
A. Normal color
B. Temperature of intestine
C. Peristalsis
D. Marginal arterial pulsations
E. Absent of veins obstructed by clots.
10. All statements about intestinal obstruction are true EXCEPT
A. The etiology of the obstruction determines the surgical procedure
B. Adhesions can be lysed
C. Tumors should be resected
D. The jejunum is more commonly affected in middle age than the ileum
E. Nonviable bowel should be removed.

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| A | E | C | D | D | E | E | D | E | D |

**Materials for independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Surgical anatomy of the intestines<br>– Physiology of intestines<br>– Pathogenesis of development of complications of bowel obstruction. | -To represent the methods of diagnostics of acute bowel obstruction as a table.<br>-To make the flow diagram of mechanisms of damage of intestines in acute bowel obstruction. |

| | |
|---|---|
| – Morphological changes in the intestines in bowel obstruction.<br>– Clinical manifestations and diagnostic methods in acute bowel obstruction. | - Make the scheme of T-cells response in normal mucosa, pro-inflammatory and anti-inflammatory mediators |
| Study:<br>– Types of acute bowel obstruction.<br>– Types of accesses to the intestines.<br>– Tactics at arterio-mesenteric duodenal obstruction<br>– Tactics in chronic bowel obstruction<br>– Endoscopic signs of IBD, diverticulosis, colorectal polyps<br>– Diagnostic value of barium enema, US, and spiral CT in verifying the colorectal diseases. | -To conduct differential diagnosis between acute diseases of abdominal organs and acute bowel obstruction<br>-To conduct differential diagnosis of pathologies of the intestines<br>-Write the main endoscopic symptoms of the mentioned diseases.<br>- List radiological, US, and CT-scan symptoms of the mentioned colorectal diseases. |

## TOPIC 4. ACUTE ABDOMINAL SYNDROME. SYNDROME OF LOCAL AND WIDESPREAD PURULENT AND INFLAMMATORY PERITONEAL PROCESS

### 4.1. Acute abdominal syndrome. Diagnosis, differential diagnosis and treatment tactics. Differential diagnosis of acute diseases of the abdominal cavity.

**Overview**

Acute disease within the abdomen is common and many patients with abdominal symptoms present every day to doctors working in the community. Within a Western population of half-a-million people, between 5 and 10 patients are admitted to a surgical ward each day with acute abdominal pain. One or two more will complain of acute abdominal symptoms after an accident. By definition the illness starts suddenly and most patients present to a hospital within 7 or 10 days of the onset of symptoms. In the majority of patients, symptoms arise from disease within the abdominal cavity itself, but occasionally they originate elsewhere in the body. The range of disease extends from the relatively trivial to the immediately life threatening, and attempts to reach a diagnosis must sometimes be curtailed in the interests of immediate treatment. More commonly there is time to take a history, to examine the patient, and to organize the investigations that will be helpful in establishing a diagnosis and planning treatment. Accurate recording of the relevant facts is vital, and a clear understanding of the anatomy and pathophysiology of intra-abdominal disease is necessary for both diagnosis and treatment. These patients are therefore ideal for training junior members of a surgical team. Some patients require early surgery. This in itself varies from a simple, straightforward procedure to a highly complex operation that stretches the ability and skill of even the most experienced surgeon. The immediate feedback that an emergency operation provides on the accuracy and the adequacy of the preoperative assessment and preparation is another reason why the patient with an acute abdomen is an important part of surgical training.

**Educational aims**

- To collect the anamnesis and to spend clinical inspection sick with acute abdomen.
- To know an etiology and pathogenesis of acute abdomen.
- To know a clinical picture of acute abdomen.
- The complication nobility of acute abdomen.
- To make the plan of inspection sick of acute abdomen.
- To carry out the analysis of data the received at laboratory and tool methods of inspection sick with acute abdomen.
- To define indications to surgical treatment acute abdomen or to appoint conservative treatment.
- To spend care sick with acute abdomen.
- To estimate work capacity sick with acute abdomen.

**A student must know:**

- Surgical anatomy of the organs of abdominal cavity.

- Pathophysiology and classification of acute abdomen.
- Uncomplicated and complicated of acute abdomen.
- Methods of examination of patients with the acute abdomen.
- Symptoms and signs of the acute abdomen.
- Clinical examination and tests.
- Comparison of diagnostic methods.
- Conservative treatment and operative treatment.
- Indications and contraindications to the surgical interference and choice of the method of the operation of acute abdomen.

**A student must be able to:**

- Take anamnesis carefully.
- Make diagnosis correctly.
- Order additional examination.
- To establish pathogenetic factors of occurrence of acute abdomen.
- To diagnose complications of acute abdomen.
- To make the plan of inspection sick with acute abdomen.
- To estimate data of tool methods of inspection at patients with acute abdomen (ultrasonic research, computer tomography, diagnostic laparoscopy).
- To carry out differential diagnostics acute abdomen and pseudoacute abdomen.
- To establish indications for surgical treatment and conservative treatment.
- To estimate efficiency of treatment and patients and work capacity of patients.

**Terminology.**

| Term | Definition |
|---|---|
| Acute abdomen | Designates symptoms and signs of intra-abdominal disease usually treated best by surgical operation |
| Peritonism | implying thereby irritation of the peritoneum without inflammation |
| Septic shock | circulation bacterial endotoxins give rise to widespread peripheral vasodilatation and increased capillary permeability with fluid leakage into the tissue |
| Signs of peritonitis | are tenderness, guarding, rigidity and rebound tenderness |
| Pseudo acute abdomen | is abdominal symptoms causes pathologic processes outsides the abdominal cavity |

**Content:**

**Anatomy**

A good knowledge of normal and abnormal abdominal anatomy, and particularly surface anatomy, is essential. Variations within and between individuals are obvious, but normal anatomy also changes with age, posture, respiration, disease, and previous surgery. Nevertheless, with experience most surgeons carry a remarkably accurate mental picture of the expected internal position of any particular organ in any particular patient. The embryological development of the abdomen is relevant in two respects.

The intestine and all its associated organs such as the liver and the pancreas develop initially as midline structures. Thus, visceral pain is usually felt along the midline of the abdomen. The gut also has a segmental origin so that the division into foregut, midget, and hindgut exactly correlates with the vascular supply, and, correspondingly, pain is felt in the epigastria, the umbilical area, and the hypogastria. Certain congenital abnormalities can predispose to acute abdominal complications. In contrast to the visceral peritoneum, the parietal peritoneum is innervated by somatic nerves. Pain is therefore accurately localized to the site of irritation of the abdominal wall and is accompanied by a reflex contraction of its muscles. This applies both to the anterior and the posterior abdominal walls. Poses spasm from acute appendicitis and a scoliosis concave to the side of intra-abdominal inflammation are two good examples. Inflammation confined to the pelvis may not, however, be accompanied by spasm of anterior abdominal muscles and this may cause clinical confusion. This is because the somatic nerves that supply the organs in the pelvis do not supply the muscles of the anterior abdominal wall. When describing the findings of abdominal examination the surface is best divided into six areas by a transverse line going through the umbilicus and longitudinal lines running through the tip of the ninth rib on each side. Thus there are epigastria and hypo gastric areas in the middle, and an iliac fosse and hypochondria laterally. It is often also useful to describe the periumbilical area. How-ever, it is important to realize that none of these divisions has a true anatomical basis.

**Physiology and pathology**

Normal physiology is rapidly disrupted by the onset of acute intra-abdominal disease. Many patients vomit, and gastrointestinal secretion, absorption and motility all change in the presence of obstruction, luminal infection, or peritonitis. Urine is reduced in volume and altered in content, usually secondary to redistribution of fluid in the body compartments but sometimes because of a direct toxic effect on the kidneys. The mediation of abdominal pain is not well understood. It is perfectly possible to handle the intra-abdominal organs and even divide the bowel of a conscious patient without causing any pain. However, distension or stretching of the bowel wall is accompanied by reflex contraction of the smooth muscle in the wall, which is immediately painful. This may be due to transient ischemia of the muscle. The pain fibers run with the splanchnic sympathetic nerves to the spinal column, where they are distributed segment ally. The pain is localized to the abdominal cavity but not to the precise segment of bowel that is being stretched. Other pathways within the spinal column are also stimulated, and vomiting, which is a common accompaniment of severe pain, can also be centrally mediated.

The gastrointestinal tract is a significant source of a wide variety of hormones. These change in response to acute disturbances of function but whether this is a primary or a secondary effect is not yet clear. Inflammation is the most common cause of acute pathology within the abdomen, followed by obstruction, hemorrhage, trauma, and ischemia. Bacteria, viruses, fungi, parasites, and chemicals can all cause inflammation: bacteria from the bowel, such as *Escherichia coli, Streptococcus facials*, and various anaerobes, are by far the most important. Other bacteria that cause acute abdominal pain are *Salmonella* and *Shigella* spp., *Yersinia*, and *Campylobacter*. Acute inflammation normally develops into clinical significance over hours rather than

minutes or days, and progression either to suppuration or resolution also takes time. Perforation and ischemia develop in minutes and cause very acute symptoms. Resolution, whatever the underlying pathology, always takes longer than development. Neoplasia, neurogenic, and metabolic disorders occur less commonly but they are all well-recognized causes of acute abdominal pain. Some of these pathological processes are closely interlinked. There are a number of causes of intestinal obstruction, of which neoplasia is one. Peritonitis from perforation of the bowel into the potential peritoneal space usually arises from local ischemia, but this may in turn be caused by inflammation or obstruction that has progressed to strangulation. The clinical presentation and the physiological consequences of obstruction or peritonitis may be similar whatever the cause, but a careful history and examination should enable the underlying pathology to be discerned.

**Clinical diagnosis**

Most patients with an acute abdomen can be managed using simple clinical skills. An accurate history and a thorough examination are often sufficient to make a diagnosis and recommend treatment; modern investigations can help and may reassure the anesthetist that the patient is fit for an operation. The primary objective when the patient and the doctor first meet is, therefore, to elicit the symptoms and the signs necessary to make a rapid and accurate diagnosis. It is sometimes obvious that the patient is in severe pain or seriously ill. The necessary immediate treatment must then take precedence over making a diagnosis.

Unfortunately, even the most experienced clinicians only make a correct clinical diagnosis of acute abdominal pain on four occasions out of five; younger doctors and those who practice in the community are only right half the time. Many attempts have been made to improve on these results and one method that has attracted much attention is computer-assisted diagnosis. By a curious coincidence this has simply taught us once again that taking an accurate history and examining the patient carefully are still the most important factors in making a correct diagnosis.

**History**

Many patients will make their own diagnosis as one listens to their story: the art of taking a history is to induce every patient to do so. Doctor and patient have not usually met before, and the style and the approach of the doctor really do matter. A relaxed, confident manner and a smile always help, and you must make it absolutely plain to the patient that they have your complete attention and that you have plenty of time to listen, even if this is not so. You should discourage interruptions by other members of staff or requests to answer the telephone. Patients like to be treated as individuals. Go and sit by their bedside knowing their name and introduce yourself clearly with your own name. Some patients will immediately start to describe their symptoms and must be left to continue. Others look for a cue from the doctor. Simple, non-specific questions such as ‘what has happened?' or ‘why have you come to hospital?' are best. Some will then give their history spontaneously; others reply in only a few words and then need prompting again. It is occasionally better initially to engage the patient in conversation about something entirely unrelated, such as their job or their family, and then when they are relaxed lead the discussion back to the acute problem. This is particularly useful with very anxious patients. The most difficult patient is the

one who is garrulous about everything but the reason they have come for help. Often there is nothing for it but to stop the flow of words deliberately and redirect the patient to the current problem. It is difficult to do this without appearing rude or disinterested: beware of the temptation to assume that there is little wrong with these patients. They are sometimes simply frightened.

Most patients come to the end of their story spontaneously, and sometimes they have told you everything you need to know in perfect order. Never intervene to clarify a point of detail but do stop the `patient glen the information they offer becomes irrelevant: it is important not to overload the brain with too many facts. When the patient has finished there will usually be some points that need amplifying or some further information that is essential. This is best obtained by asking direct, but not leading, questions. It is very easy indeed to suggest the answer you want either by the words you use, your facial expression, or the manner in which you speak or behave. If you do this the answers will be unreliable. Asking questions is also an art that requires tact and skill. Short, specific questions are best, and they must be phrased clearly without using jargon and in language the patient understands. Some patients, like most politicians, do not answer the question they are asked. You should insist, politely, on a specific answer if one is possible. No two doctors ever obtain exactly identical histories: a young surgeon may be amazed to hear a patient give a totally contradictory reply to an apparently identical question from a senior colleague. It is also surprising how often it is the very last thing the patient says that clinches the diagnosis. Not everyone can give a history themselves. Most children are shy or frightened, although others, even the very young, sometimes tell a perfect story. The confused and the mentally handicapped are often unreliable as regards facts, while the memory of an elderly patient who is ill is often faulty. A relative or a friend must then relate the history, but the clinician should remember that his or her personality then intervenes. This is a particular problem if the patient is foreign and the history has to be taken through an interpreter.

Complete attention to the patient and absolute concentration on everything he or she says and how it is said is essential. Observation of the patient is slightly different from inspection during the examination. It encompasses demeanor as well as an assessment of personality, mood, and reaction to the illness. Movement, particularly expressive movement of the hands, is always useful. Patients with peritonitis lie quite still and look ill, patients with colic really do roll around, and patients with cholelithiasis often describe the pain radiating round into the flanks with their hands, for example. Obvious and significant physical signs such as gross abdominal distension with audible borborygmi, jaundice, or the smell of Melina should not be ignored: they all point to a specific pathology that may be confirmed by specific questions. Allowing the patient to talk freely does not prevent recording the facts in a systematic fashion. In most hospitals this has to be done freehand but there are advantages in specially designed forms. The information is recorded systematically, and omissions are obvious and can be corrected at once. Such forms also require the clinician to be specific about the features of certain symptoms.

**Pain**

Most patients admitted with an acute abdominal problem complain of abdominal pain. Cope, in his classic book, observed that acute pain lasting for more than 6 h in a previously fit patient usually has a surgical cause. It is also a most important symptom: detailed enquiry about the nature of the pain will often indicate the correct diagnosis.

***Site***

The first thing to establish is the precise site of the pain that the patient has now. Some patients are extraordinarily obtuse about this, partly because they have difficulty in answering and partly because they often do not understand why you want to know. It is best to ask the patient to point with one finger to where the pain is worst and to record this site in the notes. Those who wave a hand vaguely everywhere probably do not have too much wrong with them. Pain often moves during the course of an illness and it is then worthwhile asking where the pain was situated at the beginning.

***Radiation***

Radiation of the pain to other parts of the body is often diagnostic. Radiations of the pain to the testicle in urethras colic, to the shoulders in acute cholecystitis, and to the knee with an obstructed obturator hernia are specific and typical examples. Sometimes patients volunteer that a pain radiates elsewhere but more commonly it is necessary to ask directly.

***Onset***

Some patients can say exactly when the pain started. They may be able to give a time or say what they were doing. This always suggests a significant cause and an acute pathological process, such as perforation or strangulation. Pain that wakes the patient up at night is also significant, although it is not often possible to describe the acuteness of onset. Sometimes pain is not the first symptom the patient noticed and this may suggest a medical cause, as with the vomiting from gastroenteritis or the marked anorexia of hepatitis. The duration of the illness gives some idea how far any pathology may have progressed and this can be correlated with the findings on clinical examination. Some patients relate the onset of their pain to an injury. Apparently mild trauma is occasionally followed by serious intra-abdominal injury; on the other hand it is more common for patients, after the onset of the symptoms, to try and relate them to an injury. This can be dangerously misleading, as with acute testicular torsion for example.

***Frequency***

There are two aspects to frequency. Alterations in the pain since this episode started are useful pointers to the immediate diagnosis, whereas pains that have come and gone in a similar way in the previous weeks or months suggest a longer term and more chronic disease process. Variations in intensity in the short term can be classified into two types. Either the pain is constant or it comes and goes. If it comes and goes with some degree of regularity, it is colic. Constant pain is associated with inflammatory conditions and colicky pain with distension of smooth muscle, as described below.

***Aggravation and alleviation***

Any movement makes the pain of peritonitis worse, while lying still makes it better. Acute exacerbation of the pain on walking, breathing, coughing, or going over

a bump in the road on the ride in to the hospital is equivalent to rebound tenderness on examination. Pain in the shoulder on lying down comes from diaphragmatic stimulation by an irritant fluid. The fluid is often blood from an intra-abdominal injury or an ectopic pregnancy. Analgesics usually make the pain better; this can be deceiving. Sometimes vomiting temporarily relieves the pain of obstruction.

***Severity and type***

Pain is a very subjective symptom and people's reaction to it varies widely. Accompanying signs such as sweating and tachycardia give the observer some idea of severity, but this only establishes that there is something wrong with the patient, which is often perfectly obvious anyway. Most patients find it very difficult to describe the nature of their pain and require prompting. No particular diagnoses are suggested by such descriptions as boring, dragging, sharp or dull, and they are best avoided.

**Nausea and vomiting**

These are two quite separate symptoms and both are useful in diagnosis. Nausea may precede vomiting but it need not do so and neither does vomiting always follow nausea. Nausea by itself is a less specific symptom, although it is a common accompaniment of gallstone disease. Anorexia is a separate and somewhat non-specific symptom since most people, and particularly children, lose their appetite when they are unwell. Pain normally precedes vomiting in surgical disease of the abdomen whereas the reverse is often the case in medical conditions.

Vomiting is a classic symptom of intestinal obstruction and it usually accompanies colic. Vomiting often occurs after a bout of pain in obstruction and the shorter the interval between the two the higher the obstruction. The vomit itself is initially green in color but turns yellow and then frankly fecal as the obstruction persists. Retching without vomiting suggests acute torsion of an intra-abdominal structure. Vomiting does not often accompany perforation of a peptic ulcer or intra-abdominal hemorrhage, and it is a late event in distal obstruction of the large bowel if it occurs at all. Nausea and anorexia are more common than vomiting in appendicitis.

**Bowel function**

Diarrhea and constipation are two potentially confusing symptoms because they mean different things to different people. It is important first to establish the patient's normal bowel habit and the normal consistency of the stool, and then to decide if there have been any recent changes. Diarrhea to some people simply means frequent defecation of normal fecal material, whereas repeated loose watery stools are of greater interest to the surgeon. When true diarrhea is present it is important to establish whether other members of the household are afflicted. The presence of blood, slime, or the black, tarry stools of Melina are all of obvious diagnostic value. If intestinal obstruction is suspected, then failure to pass wind as well as stool is important.

**Gynecological symptoms**

Symptoms arising from the uterus, fallopian tubes, and ovaries are a common reason for admission to hospital with acute abdominal pain. Furthermore, the ‘negative laparotomy' rate is highest in young women. Questions about normal and abnormal menstrual function, vaginal discharge, and the risk of pregnancy are therefore essential. Tact and sensitivity are required, but the answers really do matter: a ruptured ectopic pregnancy is a potentially lethal condition.

**Urinary symptoms**

Alterations in the pattern of maturation suggest disease of the urinary tract. Frequency is linked with inflammation, while anuria is most commonly caused by acute retention in elderly men. Pain on passing urine must be separated into two classes. Abdominal pain exacerbated by maturation suggests irritation of the peritoneal surface of the bladder, while stinging pain in the urethra on urination is characteristic of infection. Patients should also be asked about the color of the urine and the presence of blood or pus. Dysuria is a symptom that means different things to different doctors, and the term should not be used without specifying what is meant.

**Past history**

Any previous medical problem may be relevant to the cause of an acute admission for abdominal pain and it will certainly be relevant to the management. Chronic indigestion can be a useful pointer to a possible cause of peritonitis. A past history of abdominal surgery is important because adhesions have now overtaken hernias as the most common cause of intestinal obstruction. Patients often report previous episodes of abdominal pain and it is useful to establish whether this episode is identical. If it is, then chronic surgical diseases that flare up intermittently must be considered. Recurrent acute pancreatitis would be a good example.

**Drugs**

Many people take therapeutic drugs. Most patients, when asked, think only of those prescribed by the doctor but in many countries in the world, including the United Kingdom, it is possible to buy drugs without a doctor's prescription and these may be relevant too. Diuretics and sympathomimetic drugs may be implicated in the onset of acute retention; digoxin overdose classically causes vomiting followed by abdominal pain, and many drugs cause cholestatic jaundice. Not all patients know what drugs they are taking and pills may be transferred from bottle to bottle so that the labels are unreliable. Ultimately, a direct enquiry to the doctor or the pharmacist who wrote or supplied the prescription may be necessary.

**Examination**

No experienced doctor completely separates examination from taking the history. Observation begins the moment the doctor meets the patient and does not end until they part company. Most clinicians rapidly assimilate, almost unconsciously, many features of a new patient, and not all of them can easily be described in words. Attitude, alertness, mood, agitation, sweating, respiration, movement, the eyes, the color, the facial expression, the pulse, the handshake, and many other factors are all put together to give an instant impression of the severity of the illness and sometimes the diagnosis. The restlessness of a patient with colic is in marked contrast to the immobility of peritonitis. The gaunt patient with sunken eyes, a weak, thready pulse, and little respiratory or abdominal movement looks the same today as did patients' two-and-a-half thousand years ago when Hippocrates first described the fancies of severe peritonitis. First impressions can, of course, be false and they are not a substitute for a systematic examination. Some would say that examination does not add much to a well taken history but more evidence to help unravel a diagnosis is usually welcome. As with the history, examination of the whole patient is relevant in the overall

management, although here we are concerned with the signs that are important in the diagnosis of the acute abdomen.

**Vital signs**

Pulse rate, respiratory rate, temperature, and blood pressure are all essential observations. The initial values on admission may be misleading because of the hustle and bustle of the journey to hospital, but subsequent measurements are important in any patient whose condition is observed following admission. The charts may give a general clue as to the diagnosis. An increase in respiratory rate suggests pulmonary pathology rather than abdominal disease. An isolated rise in temperature certainly indicates disease but it does not specify where, nor does it necessarily signify infection. The height and the course of a fever in an adult may point to a diagnosis; in children, fever is an unreliable guide as it is notoriously labile. Consistent changes in these four vital signs over time are useful indicators of progressive pathology. A persistent rise in the pulse with an accompanying fall in the blood pressure is sure evidence that a peptic ulcer is still bleeding; increasing fever means that an empyema of the gallbladder needs draining. Changes in pulse and blood pressure following abdominal trauma are useful, although they usually indicate the need for active treatment rather than specifying the underlying diagnosis.

**General features**

There are many signs found elsewhere in the examination that indicate disease within the abdomen. General features of the patient, such as anemia, jaundice, and facial flushing, all have a direct relevance to abdominal diagnosis. The pallor of fear must not be confused with the pallor of anemia, and cyanosis often accompanies an acute intra-abdominal catastrophe. In children, acute inflammation of the upper respiratory tract can present with abdominal pain and examination is not complete until the tonsils and the ear-drums have been inspected. Here, however, we are primarily concerned with the abdominal signs.

**Examination of the abdomen**

Physical examination of the abdomen follows the time-honored sequence of inspection, palpation, percussion, and auscultation. Many signs can be seen and few patients, even young children, object to simple observation. Palpation can be painful and it is certainly unusual. Explaining what you are doing helps a patient to relax and so does distraction with conversation. Sometimes palpation with a stethoscope is useful. Percussion and auscultation are less useful in the abdomen than in the chest. Different doctors obtain different histories and variations in the interpretation of physical signs are even more marked. Natural variation is compounded by the lack of universal agreement on the definition of some physical signs. Despite this the basic findings should be recorded in the notes. Eponymous signs are best avoided. In practice they are rarely absolutely path gnomonic of one condition.

***Inspection***

Inspection of the abdomen is a subtle art. First and foremost both the patient and the examiner should be comfortable. The patient must lie as flat and as straight as possible with the head on a single pillow. Daylight and warmth are desirable and adequate exposure of the abdomen essential, although it is kind to keep the genitalia covered until they are actually examined. Time should then be spent simply looking

but looking in an intelligent and thoughtful way. Most important physical signs can often be seen. The history will have given some clues as to possible diagnoses, and there will be specific signs to look for while remembering that negative findings are equally important. Previous abdominal operations will have been noted in the history and the scars can be examined. Their only importance now is that there may be an incisional hernia or underlying intraperitoneal adhesions. Obvious discoloration is always important. Bruising from a seat-belt injury or the blue-grey discoloration in the flanks or around the umbilicus from hemorrhagic pancreatitis are both good examples.

***Shape***

The first thing to decide is whether the shape, symmetry, and contour of the abdominal wall are normal. Generalized distension is usually obvious except in obese patients, when it can be very difficult to decide if the abdomen is simply fat. The most common cause of generalized distension is a fetus. Excess fluid and air in the gut and ascites are the common pathological causes of distension; this is usually symmetrical. Asymmetrical distension is best judged from the end of the bed and is caused either by a mass within the abdominal cavity or a lump in the abdominal wall. The two can be differentiated because the latter must always move with the abdominal wall whereas intraperitoneal lumps do not necessarily do so.

***Movement***

The abdominal wall normally moves with respiration. With the patient laying on his or her back the abdominal wall rises up on inspiration as the diaphragm descends and falls back on expiration. If this respiratory movement hurts, then the patient will try to reduce or eliminate any movement by keeping the abdominal wall over the painful area still. This can often be seen and the effect can be enhanced by asking the patient to take a deep breath. Another common technique, but one that is less useful in the author's experience, is to ask the patient to blow their tummy out and to suck it in. Patients with peritonitis find this painful, as they do when asked to cough. Sometimes, in thin patients, it is possible to see the muscles of the abdominal wall contract spontaneously in response to the painful stimulus. This is visible guarding. Sometimes movement within the abdominal cavity can be seen on the surface. Aortic pulsation and fetal movements are both normal, and so, occasionally, in elderly individuals or those with gastroenteritis, is visible peristalsis. It is, however, a classic sign of intestinal obstruction. Distended loops of bowel can be seen through the abdominal wall and peristaltic contractions can often also be seen. These contractions are sometimes accompanied by borborygmi audible with or without stethoscope. Patience is needed, and sometimes peristalsis can be stimulated by palpation of the abdomen.

***Palpation***

Palpation of the abdomen requires warm hands, short fingernails, and care. By convention the doctor sits on the patient's right with the right hand flat on the abdomen in a comfortable position. Students, however, should learn to be ambidextrous because sometimes only the left side of the patient is accessible and some organs, such as the gallbladder, are occasionally easier to feel from the left. Superficial palpation should consist of gentle movements of the whole hand. Deep palpation is achieved by gentle pressure and by flexion of the metacarpophalangeal joints whilst keeping the fingers extended. It is best to begin by asking the patient where the abdomen hurts and then to

start palpating in the opposite corner. Work towards the painful area but do take care. Once hurt, few patients will relax. The signs then become difficult to interpret and are sometimes actually misleading. The abnormalities of importance in the acute abdomen separate into three groups. There are the signs associated with peritonitis, those that accompany a mass or enlargement of one of the solid organs, and finally those that differentiate the causes of abdominal distension.

***Signs of peritonitis*** The four signs of peritonitis are tenderness, guarding, rigidity, and rebound tenderness. Eliciting these signs is painful and it is better to see than to hear the pain. A flicker of the eyelids or a facial grimace is quite sufficient to establish the presence of pain, although guarding and rigidity are usually felt.

***Tenderness*** This is present when any palpation of the abdominal wall causes pain. It is either present or absent, although it is also possible to establish the extent of the tenderness over the abdominal wall. It is not easy to assess severity because patients vary so much in their reaction to pain. It is useful to establish where in an individual patient the pain is worst. Pain arising from the parietal peritoneum is accurately localized and patients can often point to the site of most intense pain. The examiner can also ask the patient to compare the intensity of pain by direct pressure in the four quadrants of the abdomen.

***Guarding*** There are different opinions about the physical signs of guarding and rigidity, so the examiner must be specific about what s/he actually means. In the author's opinion, guarding is present when there is reflex contraction of the muscles of the abdominal wall when the examining hand palpates it and thus causes slight pain. This may be seen but is more commonly felt.

***Rigidity*** Again there is no generally accepted definition of this sign, but the most useful description is of an involuntary increase in the resting tone of the muscles of the abdominal wall. It may be localized or generalized. It is felt as an increased resistance of the abdominal wall to palpation. The intensity varies from minor increases in tension right up to the typical generalized, board-like rigidity classically associated with perforation of a peptic ulcer.

***Rebound tenderness*** This is the most important physical sign of the four. It can be a difficult sign to elicit but when present it establishes the presence of peritonitis. It occurs when inflamed visceral peritoneum moves across and irritates the parietal peritoneum, and is best detected by percussion. This produces small movements of the underlying tissues, causes least pain, and can even localize the sign to specific areas within the abdomen. The classical method of detecting rebound tenderness by gross depression of the abdominal wall with the hand and then sudden release (hence the term ‘release tenderness') is both crude and unkind, and while sometimes useful should generally be abandoned. Rebound tenderness is also a symptom. Movement such as walking or the jolting of a vehicle may exacerbate the abdominal pain and it always worth enquiring about this whilst taking the history.

***Abdominal swellings*** It is essential to establish the size of all the solid intra-abdominal organs during palpation and equally important to identify any abnormal

masses. When the liver, spleen, and kidneys are enlarged there are certain specific signs that must be sought. When an abnormal mass is felt either within or separately from the solid organs, then all the usual rules relating to the examination of lumps

apply, although it may be impossible to assess swellings that lie deep within the abdominal cavity. Particular attention should be paid to the anatomical origin of the lump. Here mobility, and movement with respiration and pulsation, is useful. It is always helpful to establish that a swelling is cystic. Sometimes tenderness and the other signs of peritonitis coexist with an abdominal swelling.

***Abdominal distension*** Abnormal abdominal distension may be caused by an abdominal swelling but flatus, fluid, and feces are more common. Pregnancy is generally obvious and feces are easily discovered on rectal examination. Excess gas or fluid within the abdominal cavity is easy to demonstrate, but establishing the

presence of free intraperitoneal air or ascites can be difficult.

***Groins and genitalia*** No abdominal examination is complete without examination of the groins and the genitalia, particularly in men. Hernias are common but not always obvious. A small femoral hernia in a large woman is easily missed. If the hernia is the cause of an obstruction it will also be tense, tender, and irreducible, but it may not be very large. Scrotal abnormalities such as testicular torsion and epididymo-orchitis can present with abdominal pain, but there are always abnormal scrotal signs on examination.

***Rectal and vaginal examination*** No patient likes a rectal or a vaginal examination but they are essential. Again, the examination needs to be conducted with thought.

Consider all the anatomical structures in the pelvis, including the prostate and the cervix, and look at the glove for blood or pus when the examination is finished. Rectal tenderness on the patient's right side may be the only sign of pelvic appendicitis. A swelling in a fallopian tube on vaginal examination may be the only sign of an ectopic pregnancy.

***Percussion***

Percussion of the abdomen has three specific uses. First, it is the best method of eliciting rebound tenderness. Second, it is the most sensitive method for detecting enlargement of the bladder. Third, shifting dullness determines the presence or absence of ascites. It has a subsidiary role in confirming the size of the spleen and liver, and may sometimes be useful in outlining an intra-abdominal mass.

***Auscultation***

Auscultation of the abdomen is not very helpful but the presence or absence of bowel sounds is a useful physical sign. Qualitative observations are less reliable. Nevertheless, an increase in the magnitude and the frequency of bowel sounds accompanies mechanical intestinal obstruction whilst a succession splash, which can sometimes be heard without a stethoscope, is a sure sign of obstruction. Bowel sounds that definitely disappear during observation of a patient with abdominal pain and tenderness indicate the onset of peritonitis and the need for a laparotomy.

**Investigations**

Although investigations are more or less routinely requested in most patients with acute abdominal pain, very few of the tests are actually valuable in making a diagnosis. In a few patients no investigations are necessary because the diagnosis is clinically obvious. In the majority the cause of the pain is initially uncertain and it is hoped that tests will help. Older and more experienced surgeons maintain that it is preferable to wait and see in these circumstances. They argue that significant disease is usually

progressive and when the patient is re-examined after an interval the physical signs are more marked and the diagnosis easier. Younger surgeons think that the delay gives time for complications to develop, with a consequent increase in postoperative complications that diagnostic investigations might avoid. However, their enthusiasm for investigation can also delay a necessary operation if the tests take too long to perform. In a few patients an accurate diagnosis that is essential for correct treatment can only be made with the help of special investigations. We are most concerned here with tests that can help in the diagnosis and the treatment of the patient within the first 24 h of admission. After that the number of tests that can sometimes be useful is vast and they are considered in the individual subject chapters. Analysis of venous blood and various radiographs are the most popular immediate investigations, with the addition of ultrasonographic examination and computed tomography. They can be divided into two groups. Those tests that help in diagnosis and those that help in management.

**Tests useful in diagnosis**

***Testing the urine***

Simple clinical inspection of the urine should still be regarded as an essential part of examination of the abdomen. Urine containing tiny amounts of blood looks smoky. Sugar and ketones can both be smelt. Infected urine may be cloudy or blood-stained, smells unpleasant, and contains nitrites and white blood cells. Confirmation of all these findings using biochemical sticks is convenient and easy. When an infection of the urinary tract is suspected, then a carefully collected urine specimen should also be sent immediately to the laboratory for analysis. It is not easy for any patient to provide a true mid-stream urine specimen, and they must be both helped and supervised. Even then, contamination can be a problem and there are occasions, particularly in women and children, when a catheter specimen should be collected. Urethral catheterization is usually appropriate, but suprapubic puncture of the bladder provides the least contaminated specimen and carries the least risk of introducing an infection. Even though the culture result will not be available for a few days the sample must be sent immediately otherwise the opportunity to identify the organism responsible may be lost, as most patients with a urinary-tract infection presenting with acute abdominal pain will need immediate treatment with antibiotics.

***Blood tests***

*White blood-cell count* Many significant cause of acute abdominal pain are associated with some degree of inflammation. As a consequence, an increase both in the absolute numbers of white cells and in the proportion of neutrophils might be expected. The reverse observation is also true: an increase in the white-cell count indicates the existence of inflammation. It is always necessary to interpret the result in the clinical context, for the inflammation may not necessarily be within the abdomen. A value within the normal range does not exclude intra-abdominal inflammation. This very simple way of looking at the white-cell count is not the most useful. It is more helpful to interpret the result in a statistical sense. In other words, the probability of a patient with a normal white-cell count having acute appendicitis, for example, is low, whilst the chances with a raised count are higher. The same observations may be made about an excess of neutrophils in the differential white-cell count. Indeed the results of

all such tests used to establish a diagnosis should ideally be analyzed in this way. In practice a normal white-cell count is often used to reassure the surgeon who wants to wait and see, while an increased count supports a decision to operate. The surgeon should realize, however, that the test is then being used to help in a management decision and not to make a diagnosis.

*Serum amylase* Acute pancreatitis usually presents with the symptoms and signs of peritonitis, and normally patients with peritonitis warrant an immediate laparotomy. Surgery is, however, best avoided in patients with acute pancreatitis. The rise in serum amylase that usually accompanies pancreatitis allows the correct diagnosis to be made and a laparotomy is thus averted. Because the result is so important for both diagnosis and treatment it is essential to appreciate the limitations of the test. Other intra-abdominal catastrophes, such as a perforated peptic ulcer, a ruptured aortic aneurysm, or dead gut, can cause a modest rise in the serum amylase, while if the blood sample is taken too long after the onset of the pancreatitis the enzyme's concentration may have reverted to normal and so give a false-negative result. Again, a statistical approach can be adopted. A low serum amylase carries a low chance that the patient has acute pancreatitis whilst a high concentration implies a high chance (but not a certainty) that pancreatitis is indeed the diagnosis.

**Radiological investigations**

***Plain abdominal radiographs*** Controversy surrounds the use of plain abdominal radiology. Sometimes the films confirm the clinical diagnosis, add further detail, and modify the management of an individual patient. At other times the films are simply misleading, although occasionally they suggest a diagnosis that the clinician has not considered. One thing is certain. Not every patient with acute abdominal pain needs an abdominal radiograph. When one is requested the doctor should be clear what information s/he hopes to gain and s/he must have the skills to interpret the films if no radiologist is available. Traditionally two films are taken, one with the patient lying supine and the other with the patient sitting or standing erect. Modern protagonists of a single supine film point out that little additional information is derived from the erect film and add that not every patient with acute abdominal pain can safely or comfortably sit or stand. Some radiologists prefer, as an alternative to the erect film, to lay the patient on their right side and then take a lateral radiograph (the lateral decubitus view). In the author's opinion an erect view does, on occasion, add useful information whereas the lateral decubitus view usually does not. It provides only a limited view of the abdominal cavity and free intraperitoneal gas is better seen on a chest radiograph.

Abdominal films are more use in some circumstances than in others. None of the radiological signs of acute appendicitis is truly helpful, but radiological examination should be performed in patients with suspected intestinal obstruction and those who have suffered abdominal trauma. Stones in the kidney, the ureter, or the gallbladder are sometimes confirmed on a plain film, and calcification of the wall of an abdominal aortic aneurysm may be the only clue to its presence. Radiology of the abdomen is more useful in older patients, who tend to have more significant pathology and thus more abnormalities on such films. It is important to remember that the presence of abnormalities on any abdominal radiograph is valuable but their absence is meaningless.

***Chest radiography*** A good-quality, erect chest radiograph is the best film with which to confirm the presence of free intraperitoneal air. This can be seen as a black crescent, sometimes with an air/fluid level, underneath one or both diaphragms. Proximal perforations of the bowel tend to lead to larger amounts of free air than distal ones; if the perforation has occurred some time before presentation, as can happen in patients with diverticular disease, the margin of the pneumoperitoneum on the radiograph is often rather hazy and irregular. There may also be a small pleural reaction above the diaphragm. In very old and very young patients, pneumonia and pleurisy present with referred abdominal pain. Fractures of lower ribs may indicate a ruptured spleen or lacerated liver.

***Intravenous urography*** Renal colic is usually an easy clinical diagnosis to make because of the characteristic distribution of the pain. An emergency ultrasonographic examination may show a hydronephrosis on the affected side and will occasionally identify a calculus. An urgent intravenous urogram adds additional information and will indicate the site of an obstruction, and outline the degree of dilatation of the urinary tract and the size of an offending stone.

Occasionally, emergency urography is useful. Delayed excretion of contrast on the side where the patient complains of pain confirms the diagnosis. A normal urogram effectively excludes the diagnosis, provided the examination is done within a short time of the last episode of pain. Other causes of the abdominal pain can then be considered. Intravenous urography is also useful in trauma to the urinary tract. Most such patients have haematuria. The degree and the site of any damage may be displayed and the presence of a normally functioning kidney on the unaffected side can be confirmed.

***Ultrasound examination***

Ultrasound is widely used in the diagnosis of acute abdominal pain. Its place in elective diagnosis of conditions affecting the upper abdomen, the pelvis, and the retro peritoneum is already established; it is also useful in the emergency patient. Gallstones and an aortic aneurysm are easy to see, as are the edematous gallbladder wall and a tear in the aneurysm. Transabdominal and vaginal ultrasound are both useful in the pelvis to identify swellings of the uterus, ovaries or fallopian tubes. The ultrasound probe can also be used, like the examining hand, to identify the specific structure that hurts.

Ultrasound is less useful in examining the bowel because of the presence of gas. However, the inflamed appendix often lies behind the caecum and contains little air. Certainly the ultrasound probe can localize the tenderness to this specific area and sometimes it can also demonstrate an edematous tubular structure at the site where a retrocaecal appendix should lie.

Doppler ultrasound, which demonstrates flow in vessels, can help decide the cause of acute testicular pain. The hyperemia of epididymo-orchitis is in marked contrast to the ischemia of torsion. Following trauma, ultrasound can demonstrate the presence of free intraperitoneal fluid and look for damage to the liver, spleen, kidneys and pancreas. It cannot identify blood clot very well and it is of no practical use in looking for injury to the gut. Ultrasound is more readily available and can be brought to the patient's bedside but computed tomography is more accurate.

***Computed tomography (CT)***

CT has become an essential tool in the diagnosis and the treatment of the acute abdomen. Swelling of the pancreas and per pancreatic edema will confirm a diagnosis of acute pancreatitis. Lack of perfusion of parts of the pancreas on a contrast study implies potential pancreatic necrosis and identifies a patient with a high risk of complications. CT with contrast is the best method of confirming a diagnosis of acute diverticulitis. It can also identify a per colic abscess, which may best be treated by CT-guided drainage. CT is of occasional use in the diagnosis of an abdominal aortic aneurysm. Many patients with abdominal trauma require an immediate laparotomy. Other patients are less acutely injured and CT is helpful in identifying the nature and the extent of any intra-abdominal injury.

**Tests useful in management**

Many of the tests that are useful in diagnosis also have a role in management. A progressive reduction in the white-cell count or an improvement in the radiological signs of obstruction after treatment both indicates resolution of the pathology. A large number of other tests also help in the treatment of a patient, many of which are undertaken soon after the patient is admitted to hospital. Some of them also play a part in diagnosis as well.

***Blood tests***

*Hemoglobin concentration and packed cell volume* The clinical diagnosis of anemia is not always reliable, and in any patient who may possibly have an anesthetic it is clearly important to know the oxygen-carrying capacity of the blood. The initial hemoglobin value does not indicate the volume of blood lost in patients with overt evidence of acute hemorrhage, but sequential measurements can give a rough guide, provided any blood transfused is taken into account. Occasionally, the discovery of an unexpectedly low hemoglobin can help in diagnosis: carcinoma of the caecum as a cause for intestinal obstruction with anemia is a classic example. Packed cell volume accurately reflects the severity of fluid loss in a dehydrated patient and it is a good guide to the adequacy of rehydration.

*Creatinine and electrolytes* Most patients with significant intra-abdominal pathology should have their creatinine and electrolyte concentrations measured on admission. The initial values must be interpreted in the clinical context, particularly if the patient is dehydrated; in most circumstances it is the serum potassium that is the most important because of its role in cardiac function. Serial values are vital for proper postoperative fluid management.

*Liver function tests* Most patients with an acute abdomen due to liver and biliary disease are jaundiced. The depth of the jaundice reflects the severity of the pathology, and it is rare to need to measure the liver function acutely. It is, however, essential to obtain a blood sample on admission for later analysis because subsequent biochemical deterioration, which may not be clinically obvious, will demand further action. This particularly applies to elderly patients in whom the signs and symptoms of biliary disease are often obscure. The diagnosis is sometimes not even considered until abnormal results of liver function tests are discovered.

*Calcium concentration* This is only of immediate value in patients with acute pancreatitis. Depleted values are an indirect guide to the diagnosis and are used in some

severity-scoring systems. When low calcium threatens to induce tetany, intravenous calcium supplements will be needed. Calcium is always measured in patients with renal colic, in whom evidence of hyperparathyroidism is sought, but hypercalcaemia is rarely found.

*Blood gas analysis.* An arterial blood sample should be analyzed in a patient who is severely ill with an acute abdomen from whatever cause. Many such patients are covertly hypoxic, and the result of blood gas analysis may indicate the need for immediate ventilatory support. More commonly, patients will need ventilation after an emergency operation; preoperative values are then a useful measure of the patient's progress. Blood gas analysis is also a component of many scoring systems to assess the severity of acute pancreatitis.

**Radiology**

If a chest radiograph is not necessary for diagnosis it is unlikely that it will be needed in the management of the patient. Nevertheless, there are times when, although a clinical diagnosis can be made, a chest radiograph should be obtained simply to provide a baseline. It is often useful to know that postoperative changes in a number of investigations, particularly the chest radiograph, were not present before surgery.

***Contrast radiology*** Conventional contrast radiology is rarely needed as an emergency, although an urgent barium or air enema is important in a child with suspected intussusceptions. In adults with obstruction of the large bowel a limited barium enema examination is sometimes useful to establish the presence of a mechanical rather than pseudo-obstruction. In patients with obstruction of the small bowel where the cause is obscure or resolution is not occurring as fast as expected, then a small-bowel enema is always helpful.

**Electrocardiography**

Anyone over the age of 40 years, who presents with acute abdominal pain, particularly if the diagnosis is not straightforward, should have an electrocardiograph. Very occasionally a myocardial infarct will present with abdominal pain and recovery is unlikely to be helped by an unnecessary laparotomy.

**Endoscopy and arteriography**

Emergency gastro copy and colonoscopy, occasionally performed on the operating table, are helpful in patients who present with acute gastrointestinal hemorrhage. Precise localization of the bleeding point is essential for effective treatment. Mesenteric angiography may be needed as well. In both instances, treatment as well as diagnosis may be possible. Endoscopy has no part to play in the diagnosis of a perforated peptic ulcer or diverticulitis. It may make matters worse by blowing air into the peritoneal cavity through the perforation.

**Peritoneal lavage**

This is a useful investigation in patients with abdominal trauma, particularly if they are unconscious or are otherwise unable to cooperate in an abdominal examination. The presence of significant amounts of blood or intestinal contents in the wash-out fluid is a clear indication for an urgent laparotomy. Excess neutrophils in fluid aspirated from, or washed out of, the peritoneum is a reliable indicator of the presence of peritonitis. The test is rarely used because it gives no indication of the site or the cause of the inflammation.

**Laparoscopy**

Laparoscopy is invaluable in the diagnosis and the treatment of patients with acute abdominal pain, although it does involve a general anesthetic. All the organs responsible for the common causes of acute abdominal pain, such as the appendix, the gallbladder, the fallopian tubes, the ovaries, most of the small bowel, the stomach and the sigmoid colon, are easily seen through the laparoscope. In many cases it will be appropriate to treat the problem as well. The technique of laparoscopic appendictomy is now well established. In any event, it is usually possible to use a better-placed and often smaller incision once the diagnosis is established even if a laparotomy is needed. Laparoscopy has a limited role in the victim of abdominal trauma. If blood is found, then the endoscopist must look for the source and decide if the severity is sufficient to justify a laparotomy. If intestinal contents are found, a laparotomy is obligatory.

**Making a diagnosis**

No one really understands how a doctor makes a diagnosis, although the process has been analyzed many times. In theory it is simply a matter of collecting all the relevant facts and analyzing them correctly. The contrast between the junior clinician who takes time and trouble over the patient and yet makes the wrong diagnosis half the time and the senior colleague who asks a few questions, performs a limited examination, and is right eight times out of ten shows that this is not the whole story. Very few patients present with all the symptoms and signs of their disease and only experience can teach the clinician which few questions to ask, how to ask them, and how to interpret the answers correctly in the context of the individual patient. The last skill is particularly important when some of the facts conflict. Experience and constant practice are certainly essential for maximum accuracy. In actual clinical practice, several methods are used to make a diagnosis. Some involve purely practical considerations whilst others look at the same data in different ways. Most clinicians use all the methods at one time or another, often together, and usually without giving the matter a second thought.

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**Tests**

1. Which of the following statements regarding the etiology of obstructive jaundice is true?
   A. A markedly elevated SGOT and SGPT are usually associated with obstructive jaundice

B. When extra hepatic biliary obstruction is suspected, the first test should be endoscopic ultrasonography (EUS)
C. A Klatskin tumor will result in extra hepatic ductal dilation only
D. A liver-spleen scan will add significantly to the diagnostic workup for obstructive jaundice
E. Carcinoma of the head of the pancreas can cause deep epigastria or back pain in as many as 80% of patients

2. A 15-year-old patient was admitted to the emergency department with complaints of nausea, pain in the right lower quadrant of the abdomen, and progressive anorexia (symptoms began 12 hours ago). Examination revealed: rectal temperature 38.8°C, tenderness at the McBurney point, and tension of the anterior abdominal wall in the right lower quadrant. Intraoperatively: appendix and cecum without pathology, expressed by edema of the terminal ileum with fibrinous purulent exudate. The correct procedure is to
A. Close the abdomen after culturing the exudates
B. Perform a standard appendectomy
C. Resects the involved terminal ileum
D. Perform the ileocolic resection
E. Perform an ileocolostomy to bypass the involved terminal ileum
3. A 55-year-old obese patient complains of weakness, sweating, tachycardia, dizziness, and headache that occur after fasting for several hours. Eating provides relief. Which disease is characterized by these symptoms?
A. Diabetes mellitus
B. Insulinoma
C. Zollinger-Ellison syndrome
D. Carcinoid syndrome
E. Multiple endocrine neoplasia
4. An 80-year-old patient was hospitalized with complaints of nausea, abdominal pain, bloating, and diarrhea. Transanal contrast study revealed an “apple” configuration in the sigmoid rectum. What treatment does this patient require?
A. Colonoscopic decompression
B. Saline enemas and digital disimpaction of fecal matter from the rectum
C. Colon resection
D. Oral administration of metronidazole and checking a *Clostridium difficile* titer
E. Evaluation of an electrocardiogram and obtaining an angiogram to evaluate for colonic mesenteric ischemia
5. Which of the following statements regarding appendicitis during pregnancy is correct?
A. Appendicitis is the most prevalent extra uterine indication for celiotomy during pregnancy
B. Appendicitis occurs more commonly in pregnant women than in nonpregnant women of comparable age
C. Suspected appendicitis in a pregnant woman should be managed with a period of observation of due to the risks of laparotomy to the fetus

D. Noncomplicated appendicitis results in a 20% fetal mortality and premature labor rate
E. The severity of appendicitis correlates with increased gestational age of the fetus

6. A 32-year-old female patient was hospitalized with complaints of pain in the right lower quadrant, lasting 24 hours. The day before, she had undergone surgery for acute appendicitis and histologically revealed a carcinoid tumor (1.2 cm) in the apex of the appendix. Which of the following statements are correct?
   A. The patient should be advised to undergo ileocolectomy
   B. The most common location of carcinoids is in the appendix
   C. The carcinoid syndrome occurs in more than half the patients with carcinoid tumors
   D. The tumor is an apudoma
   E. Carcinoid syndrome is seen only when the tumor is drained by the portal venous system
7. Which of the following statements regarding direct inguinal hernias is true?
   A. They are the most common inguinal hernias in women
   B. They protrude medially to the inferior epigastria vessels
   C. They should be opened and ligated at the internal ring
   D. They commonly protrude into the scrotal sac in men
   E. They incarcerate more commonly than indirect hernias
8. An 88-year-old patient was admitted to the hospital with a diagnosis of acute cholecystitis. He has a history of end-stage renal disease, ischemic heart disease, and lung cancer metastases to the brain. What is the best treatment strategy for this patient?
   A. Tube cholecystostomy
   B. Open cholecystectomy
   C. Laparoscopic cholecystectomy
   D. Intravenous antibiotics followed by elective cholecystectomy
   E. Lithotripsy followed by long-term bile acid therapy
9. Which statement concerning cholangitis is correct?
   A. The most common infecting organism is *Staphylococcus aureus*
   B. The diagnosis is suggested by the Charcot triad
   C. The disease occurs primarily in young, immunocompromised patients
   D. Cholecystostomy is the procedure of choice in affected patients
   E. Surgery is indicated once the diagnosis of cholangitis is made
10. A 28-year-old previously healthy woman arrives in the emergency room complaining of 24 h of anorexia and nausea and lower abdominal pain that is more intense in the right lower quadrant than elsewhere. On examination she has peritoneal signs of the right lower quadrant and a rectal temperature of 38C. At exploration through incision of the right lower quadrant, she is found to have a small, contained perforation of a cecal diverticulum. Which of the following statements regarding this situation is true?
    A. Cecal diverticula are acquired disorders
    B. Cecal diverticula are usually multiple

C. Cecal diverticula are mucosal herniations through the muscularis propria
D. Diverticulectomy, closure of the cecal defect, and appendectomy may be indicated
E. An ileocolectomy is indicated even with well-localized inflammation

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| B | B | B | C | A | D | B | A | D | A |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of organs of abdominal cavity, sectoral structure of liver. Anatomy and features of blood circulation of the spleen.<br>– Physiology peritoneum of abdominal cavity and pelvic peritoneum<br>– Pathogenesis of abdominal disease<br>– Morphological changes in the organs of abdominal cavity at disease<br>– Clinical displays and methods of diagnostics of peritonitis | -To represent the methods of diagnostics of diseases of abdominal cavity as a table<br>-To make the flow diagram of mechanisms of damage of abdomen |

## 4.2. Syndrome of local and widespread purulent and inflammatory peritoneal process. Etiology, pathogenesis, clinic, diagnosis, treatment tactics.

**Overview.**

Intra-abdominal bacterial infection usually presents as one of three syndromes:

- spontaneous bacterial peritonitis, which typically occurs in patients with preexisting ascites;
- secondary peritonitis resulting from an identifiable preceding cause such as a perforated organ;
- intra-abdominal abscess, commonly a postoperative complication.

Polymicrobial infection with both Gram-negative aerobic enteric bacilli (e.g., *Escherichia coli*) and anaerobes (e.g., *Bacteroides fragilis*) is regularly present except in spontaneous bacterial peritonitis, which is most often caused by a single isolate such as *E. coli* or *Streptococcus pneumoniae.* Although the diagnosis of secondary peritonitis or intra-abdominal abscess is usually apparent clinically, occasionally paracentesis or laparotomy may be initially required to confirm the diagnosis. The diagnosis of intra-abdominal abscess has been facilitated by ultrasonography, computed tomography, and radionuclide scanning. Percutaneous guided-needle aspiration can confirm the diagnosis and simultaneously provide successful drainage in selected cases. Treatment consists of prompt and adequate drainage combined with effective antimicrobial therapy.

**Educational aims:**

- Interrogation and clinical inspection of patients with the peritonitis.
- To determine the etiologic and pathogenic factors of different forms of peritonitis.
- To find out the clinical features of the intra-abdominal sepsis.
- To develop a plan of examination of the patients with the abdominal infection.
- To estimate results of physical examination, laboratory tests, ultrasonography and X-ray examination in patients with abdominal sepsis.
- To make a differential diagnosis between peritonitis and other forms of acute abdomen.
- To determine the indications for treatment of patients with the peritonitis
- To compare the different surgical approaches to the treatment of diffuse and localized peritonitis.
- To provide the postoperative care after surgery.

**A student must know:**

- Anatomy of the abdominal cavity.
- Classification of peritonitis.
- Mechanisms of the development of the intra-abdominal infection and inflammation of the peritoneum.
- Clinical presentations of different form of peritonitis.
- Methods of diagnostics of abdominal infection.
- Differential diagnosis of the peritonitis with other urgent abdominal conditions.
- Surgical approaches to the management of the peritonitis.

**A student must be able to:**

- Collect and estimate the complaints of patient with abdominal infection, gather anamnesis, conduct physical research and interpret the received results.
- Define the rational quantity of laboratory and instrumental research methods.
- Be able to examine patients with peritonitis.
- Define the indications to surgical interventions and choose the appropriate surgical method.
- To prescribe preoperative preparation depending on patient's state.

**Terminology**

| Term | Definition |
|---|---|
| Systemic inflammatory response syndrome | Is an inflammatory state affecting the whole body, frequently in response of the immune system to infection, but not necessarily so; it is related to sepsis, a condition in which individuals both meet criteria for SIRS and have a known or highly suspected infection. |
| Primary peritonitis | is spontaneous bacterial peritonitis (SBP) due to chronic liver disease and ascites |

| | |
|---|---|
| Secondary peritonitis | is caused by perforation or necrosis (transmural infection) of a hollow visceral organ with bacterial inoculation of the peritoneal cavity |
| Tertiary peritonitis | represents the persistence or recurrence of peritoneal infection following apparently adequate therapy of SBP or SP, without pathology of the abdominal organs |
| Chemical (sterile) peritonitis | caused by irritant substances |

**Content**

***History***

Acute peritonitis is a serious disease with a high risk of death. In 1926, Kirschner reported that the introduction of surgical treatment in the period from 1890 to 1924 led to a decrease in the mortality rate from intra-abdominal infections from 90% to 40%.

Today, the treatment of this pathology is preferred to a multimodal approach, aimed at correcting the underlying cause and prescribing systemic antibiotics and supportive therapy to prevent or limit secondary complications due to organ system failure.

Intra-abdominal infections are classified as primary (i.e. spontaneous), secondary (i.e. associated with a pathological process in a visceral organ), or tertiary (i.e. persistent or recurrent infections after adequate initial therapy) and are divided into generalized (peritonitis) and localized (intra-abdominal abscess).

***Frequency***

Peritonitis (peritonitis) – acute or chronic inflammation of the parietal and visceral peritoneum, which occurs as a result of the action of microorganisms or physical and chemical factors on it and is manifested by both local changes and general disorders of the functional state of various organs and systems of the body.

Peritonitis still remains the cause of lethal consequences in surgical patients. Mortality from peritonitis has not only not decreased, but has a tendency to increase and fluctuates within 20-50%. Mortality depends primarily on the cause that caused peritonitis. Thus, the lowest mortality in appendicular peritonitis is 0.53-11.5%, and in peritonitis caused by other inflammatory diseases of the abdominal organs it reaches 30%. In addition, mortality depends on the prevalence of peritonitis, being 0-1.5% for local peritonitis and 36.6 to 55% for general peritonitis. In postoperative peritonitis, mortality is 75-90% or more.

The problem of peritonitis treatment is far from being solved. This is explained primarily by the untimely hospitalization of patients with acute surgical diseases. There is a direct dependence of mortality on the timing of hospitalization of patients. Among patients delivered in the first 12 hours from the onset of the disease, mortality is 11%, and among patients delivered at a later date – 45.9%.

The difficulties of treating acute general peritonitis are determined by its polyetiology, the complexity of pathogenesis, as well as insufficient attention to the features of pathogenetic correction of homeostasis disorders.

It follows that the treatment of patients with general peritonitis is a major organizational and medical problem.

***List of basic terms, parameters, and characteristics that a student must learn in preparation for the class***

Peritoneum - a thin, transparent connective tissue membrane that lines the walls of the abdominal cavity (lamina parietalis peritoneum) and covers the surface of the organs located in the abdominal cavity (lamina visceralis).

Peritonitis - acute or chronic inflammation of the parietal and visceral peritoneum, which occurs as a result of the action of microorganisms or physical and chemical factors on it and is manifested by both local changes and general disorders of the functional state of various organs and systems of the body.

Ascites-peritonitis (spontaneous bacterial peritonitis) - the most severe complication of ascites. In this case, there are no other sources of intra-abdominal infection in the body, and the ascitic fluid contains microbes, or neutrophils in the amount of more than 250 cells per 1 ml. Most often, cirrhosis of the liver is complicated, much less often nephrotic syndrome, systemic lupus erythematosus, sometimes cardiovascular failure.

Peritoneal dialysis - constant washing of the abdominal cavity with a special, so-called dialysis solution.

Guided laparostomy - a surgical treatment method used for peritonitis. In this case, the abdominal cavity is not sutured, but closed with the help of special adhesive strips, locks and other devices. In the postoperative period, the locks or adhesive strips are “opened” and the abdominal cavity is washed until it is completely cleansed. The abdominal cavity is closed only after the peritonitis is eliminated.

Scheduled relaparotomy - a surgical treatment method used for peritonitis. The condition of the peritoneum and abdominal organs is assessed, and a second intervention is scheduled depending on the severity of the injury. The scheduled relaparotomy and sanitation are repeated several times until the condition of the peritoneum and organs improves significantly and pus disappears from the abdominal cavity.

Functions of cytokines:

- participation in the inflammatory reaction,
- regulation of growth and differentiation of individual cells,
- influence on tumor growth,
- participation in the regeneration of damaged cells,
- provision of immune protection.

Classifications of peritonitis:

- by etiology;
- by origin;
- by causes of occurrence;
- by the spread of the process;
- by clinical course;
- by the nature of the exudate;
- by stages of development;
- by degree of severity.

Methods for determining the severity of the patient's condition and the prognosis of the disease in peritonitis - determination of the Mannheim peritoneal index, the ARASNE II system, the ALTONA peritoneal index, the IAPI system.

***Etiology, pathogenesis, classification of acute peritonitis. Clinic. Treatment tactics for acute peritonitis***

Definition - peritonitis is an acute or chronic inflammation of the parietal and visceral peritoneum, which occurs as a result of the action of microorganisms or physical and chemical factors on it and is manifested by both local changes and general disorders of the functional state of various organs and systems of the body.

Anatomical and physiological features of the peritoneum - the peritoneum is a thin connective tissue, transparent membrane that lines the walls of the abdominal cavity (lamina parietalis peritoneum) and covers the surface of the organs located in the abdominal cavity (lamina visceralis). The area of the peritoneum is equal to the area of the skin and reaches 2-3 m2. The histological structure of the peritoneum is complex. It is formed by six layers that have different anatomical structures.

***Histological structure of the peritoneum***

- Mesothelium - represented by cells of mesodermal origin, called mesotheliocytes. They are characterized by fibrinolytic activity. When these cells are irritated, a fibrin film is formed, which protects the layers of the peritoneum located underneath it.
- The border or basement membrane, the surface part of which is homogeneous, and in the deep part there are delicate plexuses of reticular fibers.
- The superficial wavy collagen layer, represented by thin, compact collagen fibers, which are located along the intestine.
- The superficial diffuse elastic network, the fibers of which are densely located.
- The deep elongated elastic network, the fibers of which are thick, connected by thin connections.
- The deep lattice collagen-elastic layer.

In different parts of the abdominal cavity, the peritoneum has a different number of layers. Thus, the peritoneum of the small intestine and abdominal wall consists of 6 layers, the cap - 4, the diaphragmatic surface - 3. This should be remembered and taken into account when treating patients with peritonitis, because the intensity of absorption and secretion of fluid depends on the number of layers of the peritoneum. The fewer layers of the peritoneum, the more intense the absorption. Blood and lymphatic vessels are located only within the sixth layer and only in it do the processes of secretion and absorption occur.

***Physiological functions of the peritoneum***

- Absorption or resorption function. The peritoneum absorbs 3-6 liters per hour, up to 70 liters per day of tissue fluid. The intensity of absorption by the peritoneum is confirmed by experiments conducted by Knutsen. Cyanides, when administered intraperitoneally, kill experimental animals as quickly as when administered intravenously. Similar results were obtained with the administration of narcotic drugs.

- Excretory or transudative function. As much fluid is secreted per hour and day as is absorbed. The peritoneum of the duodenum and small intestine has the greatest excretory property.
- Protective or barrier function. The exudate of the abdominal cavity has bactericidal and bacteriostatic properties. Numerous experiments have proven the resistance of the peritoneum to infection. This was confirmed by Netzel's experiments with anthrax. During subcutaneous administration of 60 anthrax bacilli, the experimental animal dies on the 5-6th day after administration, and when 1000 bacilli are administered into the abdominal cavity, the disease does not occur.
- Plastic function. The peritoneum is able to respond to irritation by secreting fibrin and forming adhesions, thereby limiting the inflammatory process, and during operations on hollow organs, fibrin seals the sutures.

***Etiopathogenesis***

Microbes that lead to the development of acute peritonitis are divided into:

- Gram-negative aerobes - Escherichia coli, Pseudomonas aeruginosa, Enterobacteriaceae, Citrobacteria.
- Gram-positive aerobes - Staphylococcus, Streptococcus.
- Gram-negative anaerobes - Bacteroides, Fusobacteria, Violonela.
- Gram-positive anaerobes - Clostridia, Lactobacteria, Pentococci, Pentostreptococcus, Zubacterium.

The pathogenesis of acute disseminated peritonitis is very complex. In response to inflammation, microbial intoxication and the appearance of a large number of toxins, biologically active substances are activated. Under their action, intrareceptors are impressed, capillary permeability is disturbed, stasis develops in them and disorders of water-electrolyte balance, acid-base state occur, and the metabolism of proteins, carbohydrates and fats is disturbed. This leads to the development of tissue hypoxia, acidosis, increased blood clotting, increased capillary toxicosis, blood stasis - disseminated intravascular coagulation syndrome develops.

Damage to interoreceptors leads to a decrease in tone and paresis of the intestines with sequestration of a large amount of fluid, salts, trace elements, proteins, carbohydrates, fats and blood cells both in the lumen of the intestine and in the abdominal cavity. Due to this, ischemic changes develop in the intestinal wall, making it permeable to microbes that enter the abdominal cavity.

Cytokines are low-molecular protein mediators produced by various cells /endothelium, leukocytes, fibroblasts, etc./. The biological activity of cytokines is manifested through very specific receptors located on cells in the area of their formation, they are less active than hormones and act on the cells in which they were formed or on nearby cells, that is, their action is very specific and only interleukin and tumor necrosis factor act on all cells, thus exhibiting a general effect.

The main functions of cytokines are: 1/participation in the inflammatory reaction, 2/regulation of growth and differentiation of individual cells, 3/influence on tumor growth, 4/participation in the regeneration of damaged cells, 5/ensuring immune protection.

In acute peritonitis, cytokines are produced much more, or unusual pathways of their formation appear and the autoregulation reaction is modified and becomes unregulated. What occurs is not just an increase in energy production, but self-combustion of the body's tissues, not an inflammatory reaction, but capillary fluid loss with interstitial edema, not stimulation of regeneration, but tissue destruction.

If we consider that the endothelium, blood cells and tissue macrophages are everywhere, and they are the nodal points of cytokine production in the processes), it becomes clear that an increase in the content of cytokines leads to a disruption of the functions of all organs and systems of the body, and not only those that become the causes of the development of a critical condition.

In diseases of the abdominal cavity, a particularly pronounced mediational aggression occurs due to the appearance of endotoxin of gram-negative microbes in the blood.

From the above, we can conclude that in critical conditions and acute peritonitis in particular, failure of various organs and systems of the body may occur.

***Classification***

By etiology:

- infectious – non-specific or specific. Specific peritonitis is a consequence of microbes that are not related to the gastrointestinal tract /gonococci, pneumococci, tubercle bacillus/;
- non-infectious /chemical substances, organ secretions - bile, gastric juice, urine, blood.

By origin:

- primary – due to the entry of microbes into the abdominal cavity by hematogenous or lymphogenous routes, as well as due to transudation of microbes from other organs;
- secondary due to the entry of microbes into the abdominal cavity due to acute surgical diseases of the abdominal organs or damage to the abdominal organs during abdominal trauma;
- tertiary – purulent forms of peritonitis that arise and proceed without a pronounced clinical picture against the background of prolonged treatment of weakened patients with secondary intoxication and often impaired immunogenesis of various nature. Previously, such peritonitis was called «sluggish».

By causes of occurrence:

- traumatic,
- inflammatory /when the process moves from a diseased organ without perforation/,
- perforation,
- postoperative,
- adjacent /when the organs of the thoracic cavity are diseased,
- lymphogenic.

By process distribution:

- local /delimited, not delimited/, when the inflammatory process is localized in 1-2 areas of the abdominal cavity;
- widespread or diffuse, the inflammatory process spreads to 3-5 areas, without delimitation of the process;
- general or total - the inflammatory process captures more than 5 areas, or the entire peritoneum, involving all depressions, bays, and twists.

By clinical course:

- acute,
- chronic.

By the nature of the exudate:

- serous,
- serous-fibrinous,
- fibrinous-purulent,
- purulent,
- bilious,
- hemorrhagic,
- fecal,
- chemical.

By stages of development:

- reactive - the first 24 hours from the onset of the disease;
- toxic - from 24 hours to 72 hours after the onset of the disease when - this is the flowering of peritonitis;
- terminal - after 72 hours from the onset of the disease, a violation of the activity of vital and body systems develops.

By severity:

- I degree - mild when signs of endotoxicosis are not pronounced;
- II degree - moderate severity (to eliminate the signs of endotoxicosis, it is enough to eliminate the cause of peritonitis by surgery and conduct intensive therapy);
- IIIa degree - severe, the phenomena of endotoxicosis after eliminating the cause of peritonitis can be eliminated only by using extracorporeal detoxification methods (hemosorption, lymphosorption, plasmaphoresis);
- IIIb degree - very severe in which it is necessary to carry out repeated sanitation of the abdominal cavity;
- IV degree - terminal when multiple organ failure is manifested, incompatible with life.

***Clinic***

In clinical practice, subjective and objective signs of acute disseminated peritonitis are distinguished.

Subjective signs include: a/ abdominal pain, b/ nausea, c/ vomiting, d/ intestinal dysfunction, which patients pay attention to /gas retention, bloating, delayed bowel movements/.

Objective symptoms:

General:

- patient behavior;
- appearance,
- cardiovascular status, respiratory disorders;
- morphological and biochemical changes in blood parameters, changes, etc.

Local (associated with changes in the abdominal cavity and anterior abdominal wall):

- appearance of the abdominal wall, its participation in the act of breathing;
- findings of palpation, percussion and auscultation;
- findings of rectal and vaginal examination;
- findings of X-ray and ultrasound examination of the abdominal organs, laparocentesis and laparoscopy of the abdominal cavity.

The disease in its course goes through three stages:

- The first – reactive.
- The second – toxic action.
- The third stage – terminal.

To assess the severity of the patient's condition and the prognosis of the disease, various methods of its determination have been proposed. The most widespread in the clinic are the determination of the Mannheim peritoneal index, the ARASNE II system, the ALTONA peritoneal index, and the IAPI system.

***Diagnostics***

The diagnosis of peritonitis is based on the patient's complaints, history, results of the patient's examination, objective examination of the abdomen, changes in the cardiovascular and pulmonary systems, changes in the general blood test.

To confirm the diagnosis, especially in case of doubt, additional methods of examination of the abdominal cavity are performed - survey fluoroscopy and radiography, computed tomography, radioisotope methods, ultrasound examination, laparocentesis, laparoscopy.

***Differential diagnosis***

Differential diagnosis of peritonitis should be carried out primarily with diseases that resemble acute peritonitis in clinical course, but do not require surgical treatment (so-called “false” acute peritonitis). These include diseases of the lungs and pleura (lower lobe pneumonia, basal pleurisy), diseases of the cardiovascular system (myocardial infarction, abdominal rheumatic syndrome), gynecological diseases (adnexitis), urological diseases (renal colic) and toxic infections.

| | Abdominal syndrome (peritonitis) | Thoraco-abdominal syndrome | |
|---|---|---|---|
| | | Pleuropulmonary | Cardiac |
| *Onset of the disease* | Gradual in inflammatory processes, sudden in perforation, trauma | Gradual | Sudden or gradual |
| *Anamnesis* | The disease begins with abdominal pain, | Often a cold factor, fever before the onset of the disease | There is usually no history of heart disease or fever |

| | | | |
|---|---|---|---|
| | without prior fever and increased | | |
| *Patient behavior in bed* | Lies calmly, often in a forced position, on the side with legs bent | Calm, freely turning over in bed, semi-sitting position | Anxious, in bed, semi-sitting position |
| *Abdominal pain and pain when coughing* | The pain appears suddenly, constantly, is accompanied by vomiting, and worsens when coughing. | Pain in the upper parts appears gradually, is constant, not localized, and is significantly worse with deep breathing | The pain appears gradually, is not localized, and worsens with physical exertion. |
| *Face* | Pale, cold sweat on forehead, facial features pointed, vision blurred | Hyperactive, often cyanotic lips | Suffering, slight cyanosis of the skin of the face and extremities |
| *Pulse* | Frequent, weak, ahead of temperature | Full, slightly accelerated, coincides with the temperature | Weak, often arrhythmic |
| *Breath* | Superficial, frequent | Difficult, frequent, cough | Not broken |
| *Tongue, lips* | Dry, crusty, lips are bluish | Wet, herpes is not uncommon on the lips, slight cyanosis | Wet, humid |
| *Abdominal palpation* | Painful, pain intensifies with deep palpation | Painful, aggravated by superficial palpation | Painful, pain does not increase with deep palpation |
| *Muscle protection* | Severe, especially in the area of the source of peritonitis | Not sharply expressed, decreases with willpower | Not pronounced or absent |
| *Shchetkin-Blumberg symptom* | Positive | Negative | Negative |
| *Intestinal peristalsis* | Slowed, progressively decreases, then disappears | Not broken | Not broken |
| *Temperature* | Slightly elevated | Significantly increased | Increases by 2-3 days |
| *Leukocytosis* | High, shift of leukocyte formula to the left | High, lymphopenia | Moderate |

| | | | |
|---|---|---|---|
| *Chest X-ray* | No pathology | Signs of inflammation of the lungs, pleura | Indistinct heart pulsation, small amplitudes |
| *Electrocardio-graphy* | No changes | No changes | Changes are present |
| *Increasing peritoneal symptoms* | Progressively increasing | Progressively decreasing | Progressively decreasing |

***Treatment tactics***

Preoperative preparation:

- Gastric decompression.
- Correction of water-electrolyte metabolism.
- Protein-energy metabolism.
- Detoxification therapy.
- Correction of multiple organ failure.

Surgical intervention:

- Elimination of the source of peritonitis (appendectomy, cholecystectomy, removal of a perforated ulcer, bowel resection. Preference is given to the imposition of primary anastomoses (small-small intestine, small-colon), left-sided hemicolectomy is recommended to be performed of the obstructive type.
- Lavage of the abdominal cavity.
- Nasointestinal intubation to eliminate intestinal paresis (if indicated).
- Drainage of the abdominal cavity.
- Drainage for the purpose of flow dialysis is recommended to be performed only in the presence of local peritonitis.

The positive result of the treatment of widespread peritonitis depends on optimal surgical tactics by 80% and only on antibacterial and intensive therapy by 20%.

Management of patients in the postoperative period.

- Antibacterial therapy.
- Detoxification therapy.
- Correction of hypovolemia, water-electrolyte balance, protein deficiency, blood coagulation system, multiple organ failure.

***Abdominal abscesses***

Abscess of douglas space - it is best diagnosed by rectal or vaginal examination.

Characteristic features:

- the presence of a painful infiltrate,
- overhang of the anterior wall of the rectum, and sometimes fluctuation,
- the rectal mucosa is swollen, wrinkles are straightened.

A pelvic puncture through the rectum or posterior fornix of the vagina helps to clarify the diagnosis.

Conservative therapy:

- the appointment of massive doses of antibiotics,
- warm enemas,

– physiotherapy procedures.

Surgical treatment:

– opening and drainage of the abscess through the rectum or posterior fornix of the vagina.

Laparoscopic drainage.

***Subdiaphragmatic abscess.***

– There are right-sided (anterior, posterior, lateral, medial) and left-sided subdiaphragmatic abscesses.
– Diagnosis is very difficult.
– The most persistent symptoms: pain in the lower chest, in the hypochondrium and epigastrium; hiccups, hectic body temperature, the patient is half-sitting or lying on the sore side.
– The abdomen is swollen, peristalsis is weakened. Soreness and stiffness of the muscles in the right hypochondrium. The chest lags behind in the act of breathing, with percussion, blunting is determined, due to the high standing of the diaphragm and fluid in the pleural cavity. Blunting sometimes reaches 2-3 ribs. On auscultation - weakened breathing, pleural friction noise, sometimes there is a displacement of the mediastinum.
– Plain radiography of the lungs and domes of the diaphragm.
– Ultrasound diagnostics.
– Computed tomography.

Treatment:

– Transcutaneous puncture of the abscess with the establishment of a «pig's tail».

Operative accesses:

– abdominal extraperitoneal;
– abdominal transperitoneal;
– transpleural (according to Troyanov);
– extrapleural.

***Interintestinal abscess.***

– They arise between the individual loops of the small and large intestines. The walls of such abscesses are the parietal peritoneum, intestinal loops, their mesentery and a large cap.
– Clinical manifestations of an abscess, as a rule, develop 14-21 days from the onset of the development of the underlying pathology. A limited painful infiltrate in the abdominal cavity is palpable. With superficial abscesses, limited protrusion is observed, and with large suppurations, a symptom of fluctuation. The muscular protection of the anterior abdominal wall is usually absent. Symptoms of peritoneal irritation are not always detected. Percussion in the abscess zone is determined by bluntness. Often, periodic attacks of acute intestinal obstruction are added to the listed signs.
– To clarify or confirm the diagnosis, ultrasound examination of the abdominal organs is used.

- Treatment begins with intensive antibiotic therapy (at least two broad-spectrum antibiotics).
- Transcutaneous puncture of the abscess with the installation of a «pig's tail».

In the absence of effect and suspicion of interintestinal accumulation of pus, patients are shown surgical treatment - opening, sanitation and drainage of the abscess.

***Postoperative peritonitis.***

Main reasons:

- insufficiency of sutures of the duodenal stump after gastric resection;
- insufficiency of sutures of the anastomosis of the gastrointestinal tract, biliary tract, etc.;
- postoperative traumatic pancreatitis;
- penetration of infection through the intestinal wall with intestinal obstruction or necrosis of their wall;
- infection of the abdominal cavity from the outside and from the inside;
- postoperative intra-abdominal bleeding;
- infection of the abdominal cavity with suppuration of the postoperative wound of the anterior abdominal wall.

***Tuberculous peritonitis.***

- Serous (exudative) peritonitis, which is characterized by rashes on the peritoneum and exudation of a significant amount of serous effusion.
- Clumpy (dry) peritonitis, which is characterized by the formation of a significant number of infections between the loops of the intestines, peritoneum and cap.
- Caseous (nodular-tumor-like) peritonitis, which is characterized by the formation of intense adhesions with large tumor-like formations, caseous tissue decay and purulent contents.
- Mixed tuberculous peritonitis.

***Laboratory diagnostics***

- Complete blood count - leukocytosis, shift of the formula to the left, toxic granularity of leukocytes, increased erythrocyte sedimentation rate.
- General urinalysis - proteinuria, leukocyturia.
- Biochemical blood test - hypoproteinemia, high creatinine, urea, ALT, AST.

Additional laboratory methods of research:

- immunogram (including determination of IL 1, 2, 6, 8, 10, TNF-a);
- procalcitonin concentration (determination of procalcitonin concentration in blood plasma can be a simple and reliable way to assess the severity of bacterial infection);
- malonic dialdehyde;
- ceruloplasmin, a 1-antitrypsin, polymorphonuclear elastase, phospholipase A2 type I;
- amylase, blood lipase.

***Instrumental diagnostics***

- examination radiography of the abdominal cavity;

- radiography of the chest cavity;
- ultrasound of the abdominal cavity:
- antibioticogram;
- blood culture for sterility;
- biochemical, bacteriological analysis of the abdominal exudate (if laparocentesis was performed);
- CT of the abdominal cavity, when there is a suspicion of an abdominal abscess.

Additional instrumental methods of research:

- Esophagogastroduodenoscopy;
- Magnetic resonance imaging.

***Preoperative preparation***

- Gastric decompression.
- Correction of water-electrolyte metabolism.
- Protein-energy metabolism.
- Detoxification therapy.
- Correction of multiple organ failure.

***Surgical intervention***

- Elimination of the source of peritonitis (appendectomy, cholecystectomy, removal of a perforated ulcer, bowel resection. Preference is given to the imposition of primary anastomoses (small-small intestine, small-colon), left-sided hemicolectomy is recommended to be performed of the obstructive type.
- Lavage of the abdominal cavity.
- Nasointestinal intubation to eliminate intestinal paresis (if indicated).
- Drainage of the abdominal cavity.
- Drainage for the purpose of flow dialysis is recommended to be performed only in the presence of local peritonitis.

***Options for completing surgical intervention***

- Closed - Passive, active drainage. Peritoneal dialysis. Laparotomy in the mode «as needed» in the event of intra-abdominal complications.
- Semi-open - Staged revision, sanitation according to the program. Temporary closure of the laparotomic wound.
- Open - Laparostomy, omentobursostomy, lumbostomy.
- Staged surgical treatment (programmed sanitation relaparotomies method).
- Laparoscopic - The method allows you to control the dynamics of the inflammatory process, perform repeated sanitation manipulations, prevent the performance of unjustified relaparotomy.
- Recently, preference has been given to the method of bacterotherapy, which provides active aspiration of purulent effusion, inflammatory exudate from the abdominal cavity.

***Management of patients in the postoperative period***

- Antibacterial therapy. Preference is given to monoantibiotic therapy:

- cyclosporines of the latest fourth generation (cefoxitin, cefmetazole,) + metronidazole preparations;
- fluoroquinolones (ciprofloxacin, levofloxacin, gatifloxacin);
- carbapenem derivatives (thienam, meronem, carbopenem, invanz).

– Detoxification therapy.
– Correction of hypovolemia, water-electrolyte balance, protein deficiency, blood coagulation system, multiple organ failure.

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2. SCHWARTZ'S PRINCIPLES OF SURGERY 2-volume set 11th edition 11th Edition/ F. Brunicardi (Author), Dana Andersen (Author), Timothy Billiar (Author), David Dunn (Author), John Hunter (Author), Lillian Kao (Author), Jeffrey Matthews (Author), Raphael Pollock (Author) - McGraw Hill / Medical, 2019. - 2448
3. Kirk's Basic Surgical Techniques, 7th Edition/ By Fiona Myint, FRCS - 2019. - 272

**Tests**

1. All of the following statements concerning intraabdominal abscesses are correct EXCEPT:
   A. A common cause is perforation of hollow viscus
   B. Treatment usually includes surgical exploration with drainage of the abscess
   C. A high index of suspicious is essential for the diagnosis because there may be no physical signs of infection
   D. Ultrasonography, CT scan, or MRI can be used to direct surgical drainage
   E. These infections are usually due to staphylococcal organism
2. Postoperatively, the patient requires an indwelling bladder catheter for 5 days to treat urinary retention. He does well until the tenth postoperative day, at which point he develops a fever of 39 $C^0$, right lower quadrant pain, and an ileus. The midline wound is not inflamed. The most likely development is:
   A. Blind loop syndrome
   B. Pyelonephritis
   C. Recurrent Crohn’s disease
   D. Intraabdominal abscess
   E. Pseudomembranous enterocolitis
3. Choose the incorrect statement
   A. Peritonitis causes a reduction in the intra-abdominal fibrinolytic activity
   B. Large number of bacteria with fibrin matrix
   C. Fibrinolytics improve the outcome
   D. Exudate formation is an important part of defense
   E. Thanks to the fibrin matrix, bacteria are protected
4. Following factors lead to persistence of infections and abscess formation EXCEPT:
   A. Capsule formation
   B. Facultative anaerobic growth
   C. Adhesion capabilities
   D. Succinic acid production
   E. Increase in the intraabdominal fibrinolytic activity
5. Predisposing factors for the development of abdominal candidiasis include the following EXCEPT:
   A. Prolonged use of broad-spectrum antibiotics
   B. Gastric acid suppressive therapy
   C. Central venous catheters and intravenous hyperalimentation
   D. Laparostomy

E. Steroids and other forms of immunosuppression

6. The role of which cytokine has been proven in the development of systemic inflammatory response syndrome and multiple organ failure:
   A. TNF-α
   B. IL-10
   C. IL-2
   D. CRP
   E. leptin
7. The clinical signs of peritonitis do not include:
   A. Abdominal pain
   B. Anorexia and nausea
   C. Jugular veins distension
   D. Increased abdominal wall rigidity
   E. Tenderness to palpation of the abdomen
8. The general principles guiding the treatment of intra-abdominal infections include the following EXCEPT:
   A. To control the infectious source
   B. To eliminate bacteria and toxins
   C. To maintain organ system function
   D. To control the inflammatory process
   E. To prescribe corticosteroids
9. While treating SBP should be avoided:
   A. Aminoglycosides
   B. Norfloxacin
   C. Quinolone
   D. Cephalosporins
   E. Metronidazole
10. Methods for preventing hernia development and external contamination of abdominal contents during temporary closure of the abdominal cavity:
    A. Self-adhesive membrane dressings
    B. Mesh
    C. Velcro-like closure devices
    D. Adhesive tape
    E. Vacuum-assisted closure (VAC) devices

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| E | D | C | D | D | E | C | E | A | E |

**Materials for self-education**

| Main tasks | Notes(instructions) |
|---|---|
| Repeat:<br>– Anatomy of the abdominal cavity<br>– Physiology of the peritoneum<br>– Types of virulent microflora | 1. Make a scheme of the abdominal cavity compartments<br>2. Make a scheme of transperitoneal filtration |

| | |
|---|---|
| Study:<br>– Methods of the assessment of abdominal fluid<br>– Sensitivity of instrumental diagnostic methods that can be utilized for the assessment patient with peritonitis.<br>– Causes of the intraabdominal hypertension and pathophysiology of the abdominal compartment syndrome<br>– Causes of intraabdominal abscess formation<br>– Methods of temporary abdominal closure | 1. Make the algorithm of abdominal exudates evaluation.<br>2. Make a scheme of the measuring of the intraabdominal pressure via the urinary bladder pressure.<br>3. Make a scheme of vacuum-assisted closure. |

## TOPIC 5. MECHANICAL JAUNDICE SYNDROME IN TUMOR AND NON-TUMOR LESIONS OF THE BILIARY TRACT

**Etiology. pathogenesis. clinic. differential diagnostic tactics. modern approaches to treatment. Liver failure in surgical diseases, methods of prevention and treatment.**

**Overview.**

**Jaundice** is a clinical symptom of acute infectious and chronic diseases of liver, bile duct obturation sign and pathology of erythrocytes. Jaundice with any etiology accompanied by pronounced intoxication, dysfunction of liver, kidneys and other systems, is a real threat to the life of the patient. The main cause of obstructive jaundice is choledocholithiasis (70%). In a number of cases of jaundice after the elimination of the changes occurring in the liver parenchyma, leading to the chronic organ disease (cirrhosis). Obstructive jaundice requires surgical treatment.

**Calculous biliary disease** - disease that is characterized by the formation of stones in the biliary system. In Europe, the prevalence of cholelithiasis is 10-12%.

**Postcholecystectomical syndrome** - covers various types of diseases that differ as a cause and clinical manifestations. In development postcholecystectomical syndrome leading role is forgotten during cholecystectomy (35-40%) in common bile calculus duct.

**Educational aims:**

- To know anatomy and physiology liver and biliary tract.
- To collect the anamnesis data and evaluate clinical inspection of mechanical jaundice, calculous biliary disease and post-cholecystectomy syndrome (PCES).
- To know the etiology and pathogenesis of mechanical jaundice, calculous biliary disease and PCES**.**
- To know clinical pictures of mechanical jaundice, calculous biliary disease and PCES**.**
- To determine mobility complications of mechanical jaundice, calculous biliary disease and PCES**.**
- To make the plan for inspection of mechanical jaundice, calculous biliary disease and PCES**.**
- To carry out the received data at laboratory analysis and instrumental methods of inspection in mechanical jaundice, calculous biliary disease and PCES**.**
- To define indications for surgical treatment of mechanical jaundice, calculous biliary disease and PCES or to appoint conservative treatment.
- To treat mechanical jaundice, calculous biliary disease and PCES**.**
- To estimate work capacity in of mechanical jaundice, calculous biliary disease and PCES**.**

**A student must know:**

- Anatomo-physiological features of liver and extrahepatic bile duct, most frequent anomalies of development, gallstone formation.

- Etiology and pathogenesis of mechanical jaundice, calculous biliary disease and PCES**.**
- Methods of inspection of the functional state of liver and extrahepatic bile duct.
- Clinical examination and tests.
- Ultrasonography and Endoscopic Retrograde Cholangiography and Endoscopic Ultrasound.
- Clinical symptomatic of mechanical jaundice, calculous biliary disease and PCES**.**
- Differential diagnostic of mechanical jaundice, calculous biliary disease and PCES**.**
- Contra-indication for surgical and conservative methods of treatment.
- Methods of surgical treatment in different forms of mechanical jaundice, calculous biliary disease and PCES**.**

**A student should be able to:**

- To collect anamnesis data and complaints of patient with of mechanical jaundice, calculous biliary disease and PCES, to underline importance of them.
- To examine a patient to define the volume of the special, instrumental, functional and biochemical researches.
- To estimate the received information.
- To define tactic and volume of further treatment.
- To carry out differential diagnostics of mechanical jaundice, calculous biliary disease and PCES**.**
- To make the plan of inspection of mechanical jaundice, calculous biliary disease and PCES.
- To establish indications for surgical treatment and conservative treatment.
- To prepare patients for the operation, to assist during the time of operation, to give indications for a patient in post-operation period.
- To predict possible complications during the treatment of patients with of mechanical jaundice, calculous biliary disease and PCES, to give suggestions in relation to prevent these complications.

**Terminology**

| | |
|---|---|
| Jaundice | is a pathologic process which characterized by yellow discolouration of skin owing to excess bile acids in the blood |
| Obstructive jaundice | Is the type of jaundice which is seen as a result of violation of patency of biliary tract due to obliteration by internal or external compression, or cicatrical narrowing. |
| Calculous biliary disease | is the formation of stones into gallbladder, hepatic and extrahepatic bile duct in consequence of violation of function of gallbladder and metabolism of bile acids |

| | |
|---|---|
| Choledocholithasis | is the complication of gallstone disease and migrated stones through the cystic duct into the common bile duct |
| Postcholecystectomy syndrome | is abdominal pain or other symptoms originally attributed to the gallbladder, may persist or recur months or years following cholecystectomy |

**Content**

**ANATOMY**

Extrahepatic Biliary Tract

Intrahepatic ducts from II, III and IV segments of the liver form the left hepatic duct, which has an extrahepatic length of 2 cm or more. The posterior right VI and VII segments and the anterior right V and VIII segments form the right hepatic duct, which has a short extrahepatic length. Merging together in a bifurcation, the right and left hepatic ducts form the common hepatic duct, which is located anterior to the portal vein in the thickness of the hepatoduodenal ligament. The common hepatic duct, connecting with the cystic duct, forms the common bile duct, which goes towards the duodenum. The length of the common bile duct varies within 5-9 cm, and depends on the level of confluence of the cystic duct into it. It is generally accepted to divide the common bile duct into 4 parts: supraduodenal (0.3-3.5 cm), retroduodenal (up to 1.8 cm), pancreatic (about 3.0 cm), and intramural.

**BILIARY PHYSIOLOGY**

During its passage through the bile ductules and hepatic duct, canalicular bile is modified by the absorption and secretion of electrolytes and water. The gastrointestinal hormone (secretin) increases bile flow primarily by increasing the active secretion of chloride-rich fluid by the bile ducts and ductules. Bile duct secretion is also stimulated by other hormones such as cholecystokinin (CCK) and gastrin. The bile ducts, along with the gallbladder and sphincter of Oddi, work together to regulate the secretion of bile into the duodenum.

**Gallbladder**

The capacity of the human gallbladder is about 40-50 ml. The gallbladder stores hepatic bile and concentrates it between meals. The gallbladder mucosa absorbs water and electrolytes from the bile, which significantly changes its composition.

Active NaCl transport by the gallbladder epithelium is the driving force for the concentration of bile. Water is passively absorbed in response to the osmotic force generated by solute absorption. The concentration of bile may affect the solubility of two important components of gallstones: calcium and cholesterol. Although the gallbladder mucosa does absorb calcium, this process is not nearly as efficient as for sodium or water, leading to greater relative increase in calcium concentration. As the gallbladder bile becomes concentrated, several changes occur in the capacity of bile to solubilize cholesterol. The solubility in the micellar fraction is increased, but the stability of phospholipid-cholesterol vesicles is greatly decreased. Because cholesterol crystal precipitation occurs preferentially by vesicular rather than micellar mechanisms, the net effect of concentrating bile is an increased tendency to nucleate cholesterol (see Gallstone Pathogenesis).

The epithelial cells of the gallbladder mucosa secrete glycoproteins and hydrogen ions. Mucus glycoprotein secretion occurs primarily from the glands of the gallbladder neck and cystic duct. The resulting mucin gel is thought to form an important part of the impermeable layer (diffusion-resistant barrier) that separates the gallbladder cell membrane from the bile lumen. This mucus barrier may be very important in protecting the gallbladder epithelium from the strong detergent effect of the highly concentrated bile salts contained in the gallbladder.

Hydrogen ions lower the pH of bile in the gallbladder, which helps dissolve calcium, preventing it from being precipitated as salts. The normal process of gallbladder acidification lowers the pH entering hepatic bile from 7.5–7.8 to 7.1–7.3.

**OBSTRUCTIVE JUANDICE**

Obstructive jaundice is the type of jaundice which is seen as a result of violation of patency of biliary tract due to obliteration by internal or external compression, or cicatrical narrowing.

**Etiology and pathogenesis**

Obstruction of the external biliary tract occurs as a result of the inflammatory process of the hepaticocholedochal tract, choledocholithiasis, lymphadenopathy of the paracholedochal lymph nodes, acute pancreatitis, stenosing papillitis, intraoperative damage to the duct wall, as well as cancer of the head of the pancreas and the major papilla of the duodenum.

Complete obstruction of the extrahepatic bile ducts leads to an increase in pressure above 300 mm of water column and cessation of bile secretion in the bile capillaries. Hepatocyte apoptosis occurs and bile enters the lymphatic vessels and veins of the liver, and from there into the blood, causing jaundice syndrome.

**Classification *(by O.O. Shalimov, 1993)***

Obstructive jaundices are divided into:

*I. By the level of obstruction:*

- Obstruction of distal parts of common bile duct;
- Obstruction of supraduodenal part of common bile duct;
- Obstruction of initial part of general hepatic duct and bifurcation of hepatic ducts.

*II. According to the etiologic factor:*

- Characterized obstruction by calculus, foreign bodies, blood during hemobilia, parasites, iatrogenic influence during operation;
- Obstruction due to diseases of the wall of biliary tract - innate anomalies (hypoplasia, cysts and atresia), inflammatory diseases (obstructing papillitis and cholangitis), scar strictures (posttraumatic and inflammatory), biliary tract tumors;
- Obstruction caused by diseases of other organs lying near the biliary tract, which drags them into the process (tubular stenosis of common bile duct of pancreatic origin, ulcer disease of duodenum, paracholedocheal lymphadenitis, adhesions).

*III. By duration of the disease:*

- Acute, lasting 10 days;

- Prolonged, lasting from 10 to 30 days;
- Chronic, lasting more than 30 days.

**Clinical management**

Pain syndrome preceding the appearance of jaundice is characteristic of the benign genesis of the latter. However, painless appearance of jaundice is usually observed in patients with cancer of the head of the pancreas or the major papilla of the duodenum.

The intensity of jaundice depends on the amount of bile that enters the intestine. The skin acquires a yellowish-green color, and in tumors - a characteristic earthy shade. With very long-term mechanical jaundice, the skin acquires a blackish-bronze color. Along with the appearance of jaundice, itching of the skin occurs, associated with the action of bile acids. On external examination, yellowness of the sclera, mucous membranes and skin is observed, the urine acquires a dark “beer” color, and the feces become acholic “white color”. An increase in body temperature indicates the development of cholangitis

During objective examination, in the presence of jaundice, in patients with palpation in the right hypochondrium of an enlarged painless gallbladder (Courvoisier's symptom), which indicates cancer of the head of the pancreas or distal parts of the common bile duct.

**Diagnostic algorithm.**

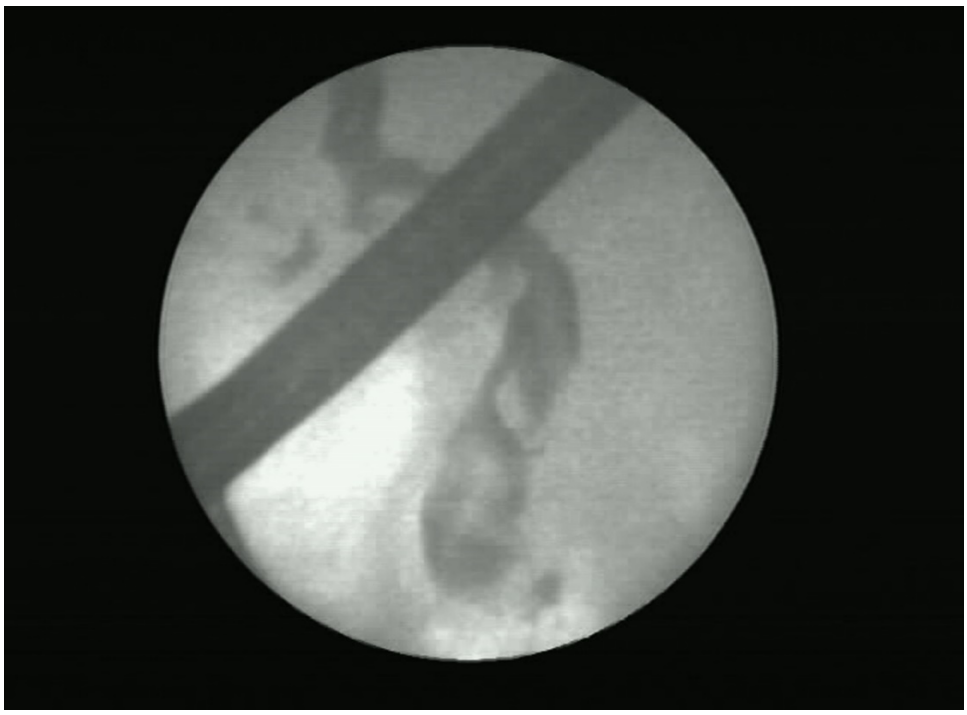
Laboratory diagnostics. Mechanical jaundice is characterized by the development of cholestatic syndrome, which is accompanied by an increase in total bilirubin, mainly due to the direct fraction, the appearance of bile acids in the urine, the absence of urobilin in the urine and stercobilin in the feces, high alkaline phosphatase activity. The thymol test is negative, and the activity of transaminases is slightly increased.

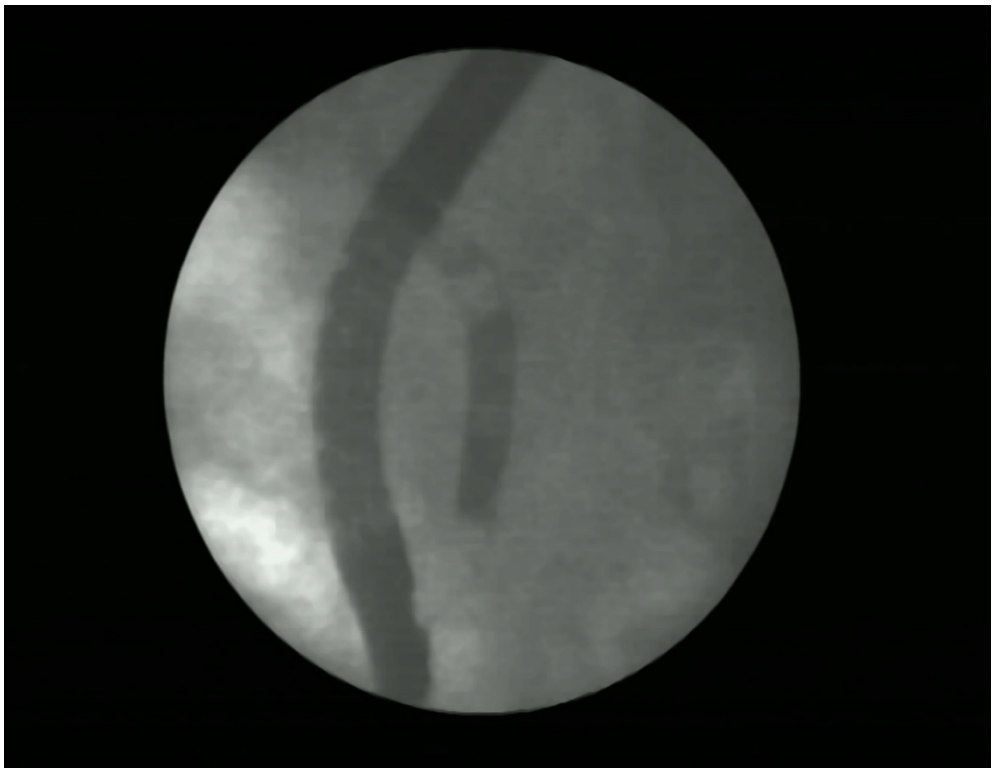
With the increase in hyperbilirubinemia, changes also occur in the general blood test. They depend on the degree of intoxication or the development of cholemic bleeding.

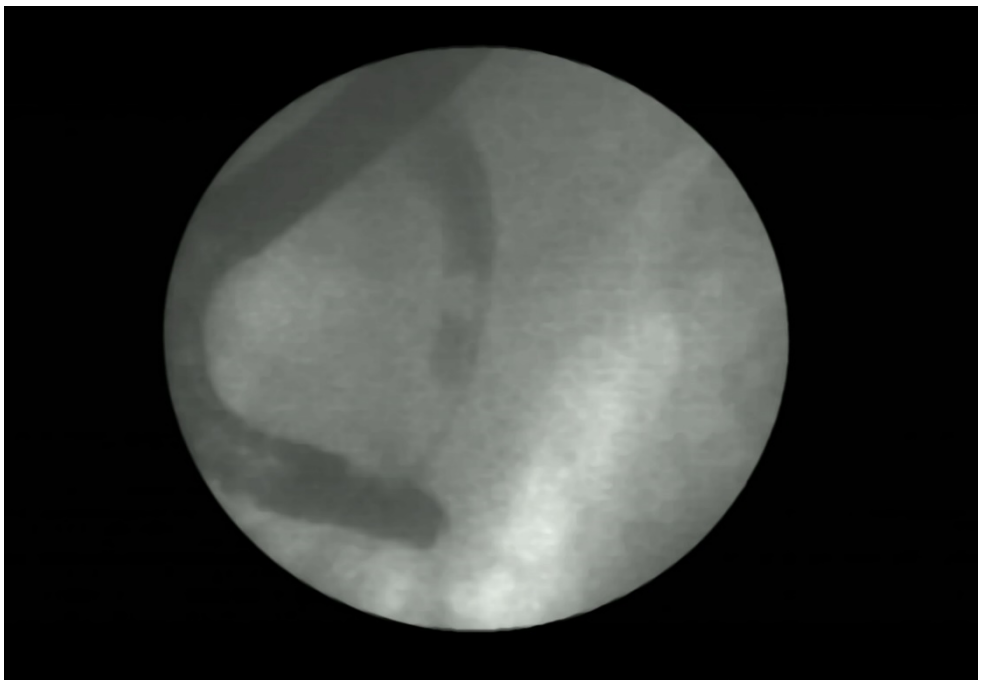
Ultrasound examination allows you to determine the size of the liver, gallbladder, the state of the intra- and extrahepatic bile ducts, the degree of their expansion or narrowing, the presence or absence of stones, the presence of neoplasms in the liver parenchyma, the head of the pancreas.

Duodenoscopy allows endoscopic examination of the stomach, duodenum, biopsy, and collection of bile and pancreatic juice for examination.

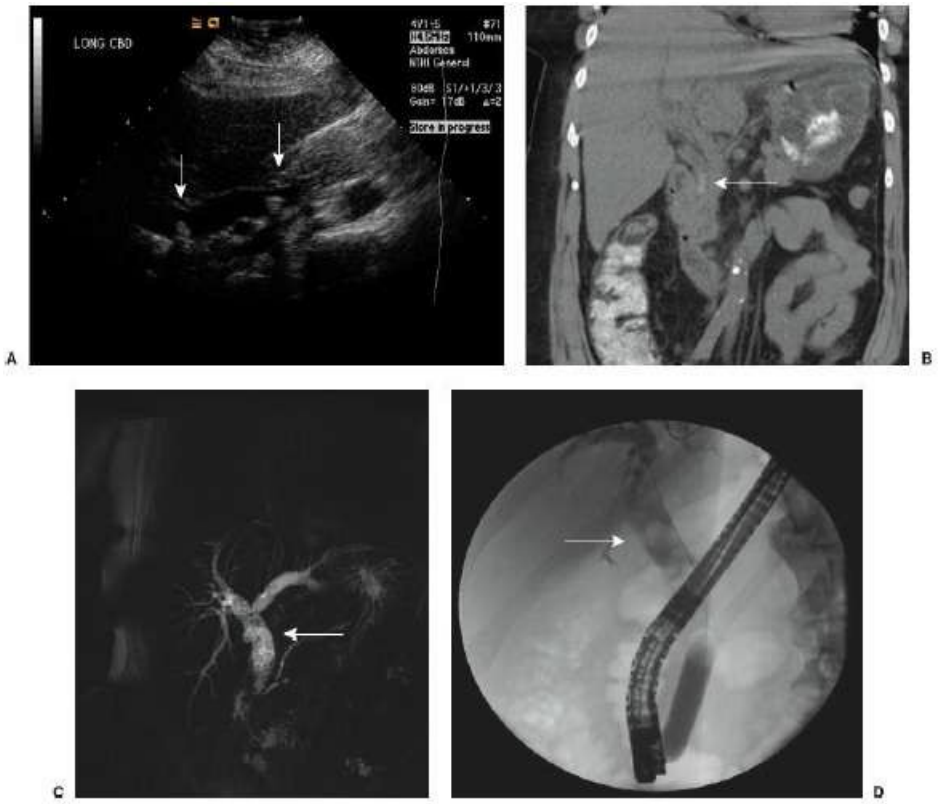
Endoscopic retrograde cholangiopancreatography - localization and nature of the obstruction, its extent. In the presence of stones, endoscopic papillosphincterotomy and lithoextraction may be performed.







**Endoscopic retrograde cholangiopancreatography - Stones in the lumen of the common bile duct.**




**A. Ultrasound examination - Stones in the lumen of the gallbladder. B. Computed tomography - The arrow indicates a stone in the common bile duct. C. Magnetic resonance cholangiopancreatography - The arrow indicates a stone in the common bile duct, dilation of the common bile duct. D. Endoscopic retrograde cholangiopancreatography - Stones in the lumen of the common bile duct.**

Percutaneous transhepatic cholangiography or laparoscopic cholangiography - is not inferior in informativeness to endoscopic retrograde cholangiopancreatography, but has a greater number of complications.

Computed tomography allows to identify the cause of mechanical jaundice and the extent of the tumor process, the presence of distant metastases.

Magnetic resonance cholangiopancreatography - a non-invasive method that allows to identify the cause of mechanical jaundice and the condition of the intra- and extrahepatic bile ducts.

Patients with mechanical jaundice should be hospitalized in a surgical hospital for examination and preoperative preparation for 5-6 days. Jaundice causes pronounced disorders of protein, carbohydrate, fat metabolism, vitamin metabolism, and the blood coagulation system is disrupted. In the preoperative period, during the examination of the patient, it is necessary to carry out a set of measures aimed at normalizing homeostasis, treating cholangitis, hemorrhagic disorders. Such preoperative preparation allows performing surgical intervention in safer conditions and reducing the frequency of postoperative complications and mortality.

Preoperative preparation should be aimed at:

- Correction of hemostasis and hemocoagulation disorders (e-aminocaproic acid, vicasol, calcium chloride, single-group fresh frozen plasma, inhibitors of proteolytic enzymes);
- Improvement of microcirculation in the liver (glucose solution with insulin, rheopoliglyukin, refortan, hepatoprotectors);
- Detoxification (lactosorbitol, enterosorbents: enterosgel);
- Use of antioxidants (high doses of vitamins C, B, E, mildronate).

Surgical treatment of obstructive jaundice depends on the cause of its occurrence.

At the current stage, the following surgical tactics are used:

- Simultaneous decompression of the biliary tract and elimination of the cause of mechanical jaundice
- Stage 1 - decompression of the biliary tract, stage 2 - elimination of the cause of obstructive jaundice
- Only decompression of the biliary tract

The main types of decompression are:

- endoscopic papillosphincterotomy;
- nasobiliary drainage of the hepatic choledochus;
- percutaneous transhepatic cholangiostomy;
- microcholecystostomy;
- endoscopic cholecystostomy;
- endoscopic transpapillary stenting of the common bile duct.

Elimination of the cause:

- endoscopic papillosphincterotomy and lithoextraction of stones through the papillotomy incision;
- laparoscopic cholecystectomy (LCE);
- laparotomic (traditional) cholecystectomy;
- choledocholithotomy.

Indications for choledochotomy are:

- mechanical jaundice at the time of surgery;

- stones in the common bile duct found by palpation or on intraoperative cholangiogram;
- the diameter of the common bile duct is more than 1.5 cm.

Choledochotomy is performed in the supraduodenal part of the hepaticocholedochus, by dissection of its anterior wall for 1-1.5 cm.

Endoscopic retrograde cholangiopancreatography in combination with endoscopic papillosphincterotomy and lithoextraction is the “gold standard” in the treatment of patients with mechanical jaundice of benign genesis.

If it is impossible to use modern minimally invasive surgical interventions, traditional ones are used, which include - laparotomy, cholecystectomy, choledochotomy, lithoextraction with subsequent revision of the common bile duct, its sanitation and external drainage by one of the known methods (Holstead-Pikovsky, Vishnevsky, Doliotti). When concrements are wedged into the lumen of the major duodenal papilla, the latter is removed by duodenotomy and dissection of the latter with subsequent papillosphincteroplasty.

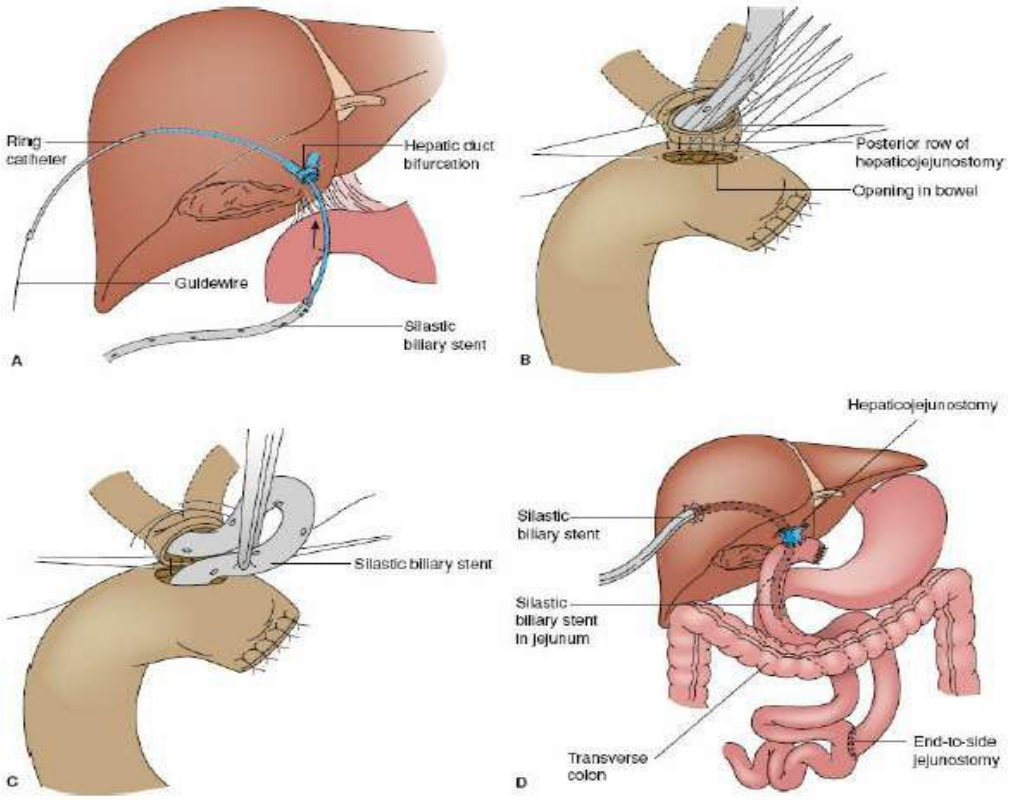
In case of mechanical jaundice of malignant genesis, radical and palliative operations are performed.

I. Radical operations:

- Resection of extrahepatic bile ducts with restoration of bile passage.
- Pancreatoduodenal resection (one-stage for moderate jaundice and two-stage – for severe jaundice 30-35 days after restoration of bile passage by endobiliary stenting or nasobiliary drainage of the hepaticocholedochus).

II. Palliative operations:

- Biliodigestive anastomoses (hepaticojejunostomy, cholecystojejuno-, choledochojejunoanastomosis).
- External drainage of the bile ducts.




**Scheme of performing hepaticojejunostomy.**

**Acute hepatic insufficiency in case of surgical diseases**

**Introduction**

Postoperative liver dysfunction is a common problem. Although the incidence after elective abdominal surgery is less than 1 per cent, much higher rates occur after major surgery, multiple trauma, and prolonged intervention. The majorities of cases is mild, transient, and resolve spontaneously, but occasionally the liver injury may be severe and result in fulminant liver failure and/or chronic liver disease. There are many etiological factors, and in any one patient the pathogenesis is often multifactorial.

**The surgical patient with normal preoperative liver function**

**Introduction**

Postoperative hepatic dysfunction in surgical patients with normal liver function can be classified into three groups: those due to (1) overproduction of bilirubin; (2) hepatocellular dysfunction; and (3) extrahepatic biliary obstruction.

**Overproduction of bilirubin**

In a healthy individual the liver conjugates up to 500 μmol of bilirubin per day as a result of the breakdown of red blood cells. The liver is capable of handling several times this quantity without the occurrence of hyperbilirubinaemia and only if haemolysis is severe or occurs in conjunction with hepatocellular insufficiency does jaundice develops. Unconjugated bilirubin comprising 90 per cent of the total is suggestive of haemolysis. When the level of unconjugated bilirubin is excessively high there appears to be a concomitant rise in the conjugated fraction. The cause of significant haemolysis may be haemolytic anaemia, blood transfusion, resorption of hematomas, sepsis, or open-heart surgery.

**Haemolytic anemia**

Congenital and acquired haemolytic anemia can be associated with postoperative jaundice. In surgical patients with sickle-cell disease there are increased risks as acute haemolysis and severe pain can be precipitated by infection, dehydration, acidosis, and hypoxia. In the postoperative period the earliest signs of infection must be treated promptly, especially as these patients have splenic hypofunction and are susceptible to bacterial infection. Patients from the African continent, parts of Asia, the Arabian Peninsula, and southern Europe should be screened for sickle-cell disease. Patients with hereditary spherocytosis may also experience a haemolytic crisis following infection, and in the postoperative period this can cause an unconjugated hyperbilirubinaemia. The diagnosis is made on the basis of a family history or the presence of an elevated average hemoglobin concentration in the body, with more than 1-2 % spherocytes in the blood count.

Surgery, infection, acidosis, and many drugs, including antibiotics and analgesics may precipitate haemolysis in patients with glucose 6 –phosphate dehydrogenase deficiency. At least 10 million people worldwide have this red cell enzyme deficiency, and thus patients from the Mediterranean, South-East Asia, the

Middle East and West Africa should be screened preoperatively. The cresyl blue decoloration test or the methaemoglobin reduction test can be used in screening, and the diagnosis made by enzyme assay. Pyruvate kinase deficiency is another red cell enzymopathy in which infection can precipitate haemolysis. Patients should be aware of their diagnosis but macrocytosis and an abnormal enzyme assay will confirm the diagnosis. Causes of non-immune acquired haemolytic anaemia include disseminated intravascular coagulation; vasculitis; pneumococcal, meningococcal, and Gram-negative sepsis; *Clostridium perfringens* (was *C. welchii)* infection; burns; drowning; and some drugs. These are covered in other parts of this section.

**Blood transfusion**

Immediate and delayed haemolytic reactions may occur following blood transfusion. Within 24 h of the transfusion of one unit of stored blood at least 10 per cent undergoes haemolysis. Transfusion of two units of blood should not result in an increase in the serum bilirubin. If transfusion is rapid, massive, or occurs in a patient with impaired liver function, the capacity of the liver to conjugate bilirubin may be exceeded. Jaundice in this situation occurs 10 to 12 h after transfusion. Incompatibility of transfused blood may result in a severe immediate haemolytic reaction, which may occur if there are antibodies to the donated blood in the recipient's plasma. Jaundice appears at 12 h after commencing transfusion, peaks at between 24 and 36 h, and lasts for a total of 4 or 5 days. Delayed haemolytic transfusion reactions are seen between 3 days and 3 weeks post-transfusion, with the peak reaction being at around 7 to 10 days. They are due to a secondary immune response, and in the majority of cases there has been sensitization to red cell antigens through past transfusion or pregnancy. This response is often to Rhesus and Kidd antigens, and is seen clinically as extravascular haemolysis with fever, jaundice, and anaemia. A serum sample should be screened for antibodies and future transfusions proceeded by careful compatibility testing.

**Resorption of hematomas**

Large hematomas, crush injury, and bleeding from major vessels result in large pools of extravascular blood which, when reabsorbed, can result in an unconjugated hyperbilirubinaemia. As these patients often have hepatocellular dysfunction due to hypotension, hypoxia, and major surgery, as well as renal impairment, the severity and duration of jaundice may be marked. In a similar way, massive pulmonary infarction can cause hyperbilirubinaemia.

**Sepsis**

A massive haemolysis can occur in association with *Clostridium perfringens* (was *C. welchii)* infection 24 to 72 h after gastric, biliary tract, or colonic surgery. The typical clinical picture is of a restless hypotensive patient, an acute rise in serum bilirubin, and crepitus around the wound site. Several causes of liver dysfunction probably occur simultaneously in these patients as the conjugated bilirubin level can be greater than the unconjugated. As these cases can be fatal, prompt treatment with massive doses of penicillin and hyperbaric oxygen are imperative. Meningococcal, pneumococcal, and Gram-negative sepsis can cause haemolysis through disseminated intravascular coagulation and secondary microangiopathic haemolysis. As sepsis can also cause intrahepatic cholestasis, a combination of the two factors may cause marked jaundice.

**Open heart surgery**

Early and late rises in bilirubin are seen after open-heart surgery. Early onset jaundice may be seen in up to 23 per cent of such patients and the main contributing factors are hypoxia, severity of right-heart failure preoperatively, and number of units of blood transfused. Although it has been suggested that cardiopulmonary bypass and prosthetic valves cause haemolysis, these are probably not significant contributors to the increased bilirubin. Late jaundice due to an autoimmune haemolytic anaemia has been reported where anaemia and jaundice, exacerbated by repeat transfusion, occur a few weeks after surgery. The presence of antiglobulin antibodies confirms the diagnosis; steroids are the treatment of choice.

**Gilbert's syndrome**

Gilbert's syndrome is a benign familial mild unconjugated hyperbilirubinemia that affects 2 to 5% of the population. Men are more commonly affected than women and presents in the second or third decade. It is usually diagnosed incidentally during a routine physical examination or during laboratory testing. Serum bilirubin levels do not exceed 100 μmol/L and are usually less than 50 μmol/L. Jaundice is usually mild but worsens with fasting and may therefore occur postoperatively.

**Circulatory failure**

**Circulatory failure/ surgical shock**

Circulatory failure contributes to hepatic dysfunction in many surgical situations, although it is rarely the sole cause of the liver abnormality. Major trauma, burns, sepsis, massive blood loss, and surgery can be precipitants of “shock”, and these factors often occur together. In particular, gastrointestinal blood loss and septicaemia increase the risk of liver dysfunction when associated with hypotension. Cholestasis is the most common pattern of injury following hypotension, and this is a benign complication with a good prognosis. Prolonged hypotension, which is often associated with

increased right atrial pressure, results in ischaemic hepatitis, for example in open-heart surgery. There is an initial striking elevation of serum aminotransferases up to 200 times the normal level, a marked decrease in prothrombin time, and a typically delayed bilirubin rise. These dramatic changes are seen within hours of surgery, and where no severe liver damage has occurred they revert rapidly to normal with restoration of liver blood flow and oxygenation. However, massive centrilobular hepatic necrosis can occur, and the ischaemic hepatitis can progress to fulminant hepatic failure, which has a high mortality rate. The clinical manifestation of hypoxic liver cell necrosis inevitably postdates the hypoxic event, and other causes, especially a viral hepatitis, must be considered.

Massive haemorrhage in combination with massive transfusion (for example, more than 20 units of blood) puts the liver particularly at risk of damage, should the patient survive. Patients with major trauma are particularly at risk of this form of liver damage as well as that due to direct liver injury. In one study, 2 per cent of patients with major trauma and shock developed significant jaundice. Patients with major burns form another group in which circulatory failure is an important factor in the etiology of the associated hepatic dysfunction. Haemolysis often adds to the bilirubin load on the liver.

**Hepatic artery ligation**

The normal liver usually tolerates hepatic artery ligation without significant sequel unless the flow of portal-vein blood is inadequate because of vascular stricture and sepsis. Minimal derangements of bilirubin and alkaline phosphatase levels occur, and moderate increases in the aminotransferase levels in the first week may be the only consequence. Hepatic arterial collateral vessels develop very rapidly and this, in combination with the portal circulation, reduces the ischaemic insult. Extensive mobilization of the liver can involve division of the ligamentum and triangular ligament and if this precedes hepatic artery ligation, massive liver necrosis may result. If infarction occurs, the amounts of bilirubin and aminotransferases rise rapidly to high levels.

**Post-transfusion hepatitis**

The incidence of posttransfusion hepatitis has declined dramatically over the past 50 years with the development of antibody tests to screen donated blood for the presence of these antibodies. In countries where voluntary blood donations are used and screening for hepatitis B surface antigen and hepatitis C antibodies is routine, clinical posttransfusion hepatitis has been virtually eliminated. Although hepatitis G virus is certainly a transfusion-transmitted agent, there is currently no evidence to suggest a causal relationship between HGV infection and hepatitis. Therefore, screening for HGV is not routinely performed.

**Drugs**

Many drugs used in the pre- and postoperative period have been associated with liver dysfunction. Almost every naturally occurring liver disease that affects humans can be mimicked by the toxic effects of drugs on the liver and this occurs through a wide range of mechanisms. Drugs can affect bilirubin metabolism at any stage causing hyperbilirubinaemia. The drug or its metabolite can be hepatotoxic or can precipitate a hypersensitivity reaction. Hepatocellular dysfunction may be due to cellular necrosis

or intrahepatic cholestasis. Factors that increase the risk of drug-induced hepatic injury include pre-existing liver disease, increasing age, female sex, concurrent therapy, and genetic polymorphism. Early symptoms of drug-induced liver injury are non-specific and include loss of appetite, lassitude, and occasionally right upper quadrant discomfort. There may be few clinical signs however, even in a patient who has biochemical and histological evidence of considerable hepatobiliary damage. Hypersensitivity reactions may be associated with a fever, rash, or eosinophilia. Jaundice in drug-induced liver injury carries a poor prognosis with a fatal outcome of approximately 10 per cent.

The list of potentially hepatotoxic agents is large and ever increasing. The general anaesthetic drugs are discussed separately. A hepatitic serum biochemical pattern must lead to exclusion of a viral etiology, and the differentiation of intrahepatic and extrahepatic cholestasis is important and should be elucidated with ultrasound scanning. Liver biopsy will only rarely give a diagnosis. Diagnostic challenge with a suspected drug is not recommended as a severe or even fatal reaction can occur.

**General anaesthetic drugs**

Halothane is amongst the most important of the idiosyncratic hepatotoxins. It was first introduced in 1956 and within 4 years there had been several reports of postoperative liver necrosis. The National Halothane Study reported the incidence of massive hepatic necrosis to be 1 in 35 000 halothane anaesthetics. Two subsequent studies suggested that the incidence was even higher at 1 in 6000 and 1 in 20 000 uses. After acetaminophen, halothane is the second commonest drug cause of fulminant hepatic failure. Nevertheless, it is still a commonly used general anaesthetic agent with many favorable properties and few adverse effects. Two types of halothane-induced liver injury appear to exist. Ten to thirty per cent of patients exposed to halothane develop asymptomatic elevations of aminotransferase levels with no clinical features of liver disease. This condition is benign and self-limiting. Its relationship to the rare, severe syndrome of halothane hepatitis is unclear. The latter condition may represent the severe end of a spectrum of liver injury associated with halothane exposure or, more likely, is a separate idiosyncratic reaction. Multiple exposures are the single most important risk factor for halothane hepatitis. Eighty per cent of patients developing the condition have received halothane more than once, usually in the preceding 28 days. Women are more commonly affected, as is the case with many other types of idiosyncratic hepatic drug reactions. Obesity is also a significant risk factor, possibly due to increasing body stores of halothane or because of higher hepatic activity of P450 2E1, an enzyme which catalyses the metabolism of halothane to reactive metabolites. Concomitant drug therapy with microsomal enzyme inducing agents may also predispose to halothane hepatitis, and there is evidence of a genetic predisposition to developing the condition.

Fever is usually the initial symptom of halothane hepatitis and this occurs 7 to 14 days after a first exposure to the drug but earlier after multiple exposures. Symptoms of hepatitis occur 2 to 5 days later with anorexia, malaise, nausea, vomiting, and right upper quadrant pain. In most cases, dark urine, pale stools, and jaundice follow, although icteric cases of halothane hepatitis also occur. Liver biochemistry is typical for acute hepatocellular necrosis with grossly elevated aminotransferase levels (e.g.

alanine aminotransferase raised to 10 times the normal level) and elevated serum bilirubin levels, whereas the alkaline phosphatase level is often less than twice normal. Between 10 and 40 per cent of patients develop eosinophilia. The main histological feature is centrilobular necrosis, varying from a multifocal spotty picture to confluent massive necrosis. Ballooning degeneration of hepatocytes, inflammatory infiltrate, stromal fibrosis, fatty change, and occasionally granulomatous aggregates are also seen. Distinction from viral hepatitis may be difficult. A number of factors have been postulated in the pathogenesis of halothane hepatotoxicity which include toxic products of metabolism, hypersensitivity, genetic predisposition, regional hepatic hypoxia, and altered calcium homeostasis.

Management, as for all types of drug-induced acute hepatitis, is supportive. Patients with fulminant hepatic failure are best cared for in specialist centers and ideally in proximity to a transplant unit. Patients who have had an adverse hepatic reaction to halothane should be warned about the dangers of future exposure to the drug and advised to wear a Medic Alert bracelet. Up to 90 per cent of cases of halothane hepatitis could be prevented by taking an appropriate history before administering anaesthesia and by adhering to safety guidelines. Enflurane hepatitis has been described, but on closer examination in many cases the alternative causes of liver injury had not been adequately excluded. True cases are extremely rare. The difference in hepatotoxicity between halothane and the other haloalkane anaesthetics is directly related to their potential to undergo P450-mediated metabolism. Around 30 per cent of halothane is metabolized, whereas the figures for enflurane and isoflurane are 2 per cent and less than 1 per cent, respectively. When enflurane hepatitis does occur, it is similar to halothane hepatitis in clinical presentation and histological features, and the two conditions probably share the same pathogenesis. Case reports of hepatotoxicity associated with isoflurane are extremely rare and so far sevoflurane and desflurane do not appear to have any adverse effects on the liver.

**Total parenteral nutrition (TPN)**

Since its advent in the 1960s, parenteral nutrition has become safer, more reliable, and progressively more efficient. However, complications still occur and hepatobiliary abnormalities are second only to catheter sepsis in requiring cessation of parenteral feeding. A number of different patterns of liver dysfunction occur.

**Short-term TPN**

Hepatic steatosis (fatty change) is the earliest and most benign hepatic lesion. It occurs within the first 14 days of TPN administration and is often, but not necessarily, paralleled by a rise in the serum aminotransferase levels. Patients receiving fat-free TPN are much more likely to develop steatosis, and standard TPN regimens now supply a proportion of calories as a lipid emulsion to minimize this problem. The initial change on histology is periportal fat infiltration but this may progress to pan- or centrilobular infiltration. A number of factors influence the accumulation of fat within the liver. Hepatic lipid metabolism is influenced by the balance between insulin and glucagon. High concentration glucose infusions induce high insulin levels and suppress glucagon production. Glycogenesis is therefore favored over lipolysis. Lipid may also accumulate because of increased delivery from peripheral fat stores and from defective production of lipoproteins that transport triglycerides from the liver. Other proposed

causes of a fatty liver include excess activity of endotoxins or tumor necrosis factor, glutamine deficiency, a toxic effect of tryptophan metabolites, choline and carnitine deficiency, and increased bacterial translocation from an atrophic gut.

**Long-term TPN**

Chronic progressive liver disease is rare but well described in patients receiving long-term TPN. One study found 3 of 60 patients on home TPN developed clinically severe liver disease. Of these, one died from hepatic encephalopathy and hepatorenal syndrome after 11 years of TPN and another patient died postoperatively following a cholecystectomy and duct exploration. Patients requiring parenteral nutrition are likely to have multiple other risk factors for hepatic dysfunction such as hepatotoxic drugs, multiple transfusions, and repeated surgery. It can therefore be difficult to isolate TPN as the cause. The histological picture may be similar to that seen in patients on shorter-term TPN, but beyond 6-months therapy a cholestatic picture is the common finding. Cholestasis, hepatocyte necrosis, an alcoholic hepatitis-like picture, steatonecrosis, and early cirrhosis may all occur. The pathogenesis is likely to multifactorial, involving any or all of the mechanisms discussed under short-term nutrition and complicated by the underlying disease process. Patients with short bowel syndrome have the worst prognosis. Cholelithiasis becomes progressively more common with increasing length of parenteral feeding. Biliary sludging has been found in 100 per cent of patients treated with more than 6 weeks of TPN and 23 per cent of 109 patients developed clinical cholecystitis during TPN treatment. Gallbladder stasis is the most likely cause of gallstone disease in patients on TPN. Gallbladder contractions are reduced by approximately two-thirds during exclusive parenteral nutrition.

**Management of TPN-induced hepatobiliary disease**

Once TPN has been identified as the most likely cause of deranged liver function, the optimal management is to restart enteral nutrition where possible. Liver function tests will return to normal in most patients within 1 month of cessation of TPN. If however continued parenteral nutrition is unavoidable and liver abnormalities persist and worsen, a number of therapeutic measures can be attempted. First, a change in the composition of the TPN may be helpful. Lipid emulsions should be administered as approximately one-third of total calories. Patients should not receive more than 3 g/kg per day of the lipid preparation, however, as this may predispose to hepatic fat accumulation. Second, changing the timing of TPN administration (‘cycling') may improve liver function. TPN solution is given for 8 to 12 h every 24 h rather than as a continuous infusion. This approach reduces the time during which the serum glucose concentrations are high, thereby avoiding persistently high insulin levels which may stimulate hepatic lipogenesis. An improvement in liver function tests will take 2 to 3 weeks to manifest after changing from continuous infusion to cycling. If neither of these two approaches is effective, the total caloric intake will need to be reduced to prevent progressive liver disease.

A number of other therapeutic options are currently under investigation. Metronidazole appears to prevent the development of intrahepatic cholestasis in some adult patients on TPN. The proposed mechanism for this action is the prevention of the intestinal overgrowth of anaerobic bacteria allowing the bacterial 7à-dehydroxylation of chenodeoxycholic acid to the potentially hepatotoxic lithocholic acid.

Ursodeoxycholic acid has been shown to improve the cholestatic liver function tests of patients with intestinal failure treated with home TPN. The most effective approach to the problem of TPN-induced biliary disease has yet to be established. Cholecystokinin, chosen for its prokinetic effect on the gallbladder, has been used in one small study, where it seemed to prevent the formation of biliary sludge in patients treated with TPN. There is no evidence available yet, however, that it has any useful effect on patients with established biliary disease. Ursodeoxycholic acid and chenodeoxycholic acid have been shown to prevent gallstone formation in animal studies of TPN-induced biliary disease.

**Fasting**

Mild hyperbilirubinaemia can be precipitated by fasting and is due mainly to an unconjugated bilirubin rise. The majority of patients showing this effect are probably those with Gilbert's syndrome. Fatty change is also seen, particularly in acute weight loss or starvation. This is related to the increase in serum fatty acids and increased fatty acid turnover precipitated by decreased availability of glucose, a rise in glucagon levels, and increased sympathetic nervous activity. Obese subjects who lose weight rapidly may show a transient elevation of serum liver enzymes.

**Obesity**

Fatty change in the liver is seen in up to 50 per cent of subjects who are obese, with occasional periportal inflammation and fibrosis. Fifty per cent of patients who are obese can be shown to be glucose intolerant, and this and excess dietary fat and carbohydrate in relation to protein intake may be involved in the etiology of steatosis. The fatty infiltrations are perivenular and diffuse. Liver function tests may be abnormal and reflect more severe histological change. The changes are, in general, benign and non-progressive, and can be reversed by weight loss.

**Diabetes mellitus**

Patients with diabetes also show fatty change in the liver; the majority of patients being non-dependent on insulin and also overweight. Steatosis is very rare in patients with juvenile-onset insulin-dependent diabetes. Symptoms are rare; an enlarged, slightly tender liver may be found on examination, and liver function tests may be slightly deranged in about 20 per cent of patients with diabetes, but do not correlate with histology. The fatty changes are centrilobular and diffuse. Weight loss and good diabetic control will resolve these abnormalities. Steatonecrosis may also occur and this is seen in the non-insulin-dependent group. Patients with diabetes are twice as likely to develop cirrhosis of the liver. This suggestion is unproved and may originate in the number of patients with cirrhosis who are glucose intolerant and have wrongly been classified as having primary diabetes. Emergency biliary surgery in patients with diabetes has a higher than expected mortality. This is due in part to the disruption of glucose control caused by surgery, the increased risk of infection due to leukocyte dysfunction, and poorer wound healing.

**Sepsis**

Hepatocellular dysfunction occurs early in sepsis despite a hyperdynamic circulation and increased hepatic perfusion. This effect is mediated via Kupffer cell- or macrophage-derived proinflammatory cytokines such as tumour necrosis factor and interleukin 2. Sepsis can produce a deep jaundice, which may be cholestatic and occurs

2 to 4 days after the onset of bacteraemia. Pneumonia, Gram-negative bacteraemia, intra-abdominal abscess, and pyelonephritis can all cause a raised bilirubin. Gram-negative infection in infants frequently causes cholestasis. As in most cases of hepatic dysfunction discussed here, sepsis may be only one element in a multifactorial etiology. Biochemically and histologically, the changes are very similar to those observed with circulatory failure, with a moderate rise in conjugated bilirubin, aminotransferases, and alkaline phosphatase levels. However, an increase in the unconjugated bilirubin level also occurs, giving a rise in total bilirubin out of keeping with the increase in liver enzymes. Hepatic histological changes include biliary stasis, fatty change, and periportal inflammation. Extrahepatic biliary obstruction must be excluded. Pneumococcal, meningococcal, and Gram-negative sepsis may cause haemolysis by disseminated intravascular coagulation or a secondary microangiopathic haemolysis, and in these conditions the rise in unconjugated bilirubin will be prominent.

**Benign postoperative intrahepatic cholestasis**

"Benign postoperative intrahepatic cholestasis" is unlikely to be a specific entity. It occurs in situations where blood loss is a prominent problem and is probably due to a combination of hypotension and multiple blood transfusions. Caroli in 1950 was the first to describe the occurrence of postoperative cholestatic jaundice. Benign postoperative intrahepatic cholestasis has been included in all lists of causes of postoperative jaundice since about this time. The etiology of postoperative cholestasis is discussed within this section, and the majority of cases given this label in the past now have a definable cause.

**Extrahepatic obstruction**

**Bile duct injury**

Bile duct injury may occur after cholecystectomy, exploration of the common bile duct, or any upper abdominal surgery. If not recognized during surgery, jaundice, biliary fistula, or biliary peritonitis will occur in the early postoperative period. Endoscopic retrograde cholangiopancreatography allows the diagnosis and treatment of postoperative biliary tract injuries.

**Common bile duct stones**

Stones remaining in the common bile duct after cholecystectomy or exploration of the common bile duct are rare. In most cases, endoscopic retrograde cholangiopancreatography can both diagnose and treat this problem with sphincterotomy. Repeat surgery is necessary if endoscopic retrograde cholangiopancreatography is inconclusive or unavailable. Some practitioners advocate visualization and, if necessary, cleaning of the bile duct with endoscopic retrograde cholangiopancreatography before cholecystectomy. Occasionally, blood can collect in the common bile duct and cause obstruction.

**Postoperative pancreatitis**

Acute postoperative pancreatitis is uncommon and the cause is unknown. Thirty per cent of patients may be jaundiced, and oedema of the head of the pancreas is thought to result in some degree of obstruction and a low-grade hyperbilirubinaemia.

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**Tests**

1. 60 years old patient, male, has jaundice for over 3 weeks, began without pain and pain is getting more intensive. Abdomen palpation is soft. Symptom Courvoisier's is positive. Ultrasound marked biliary tract and gallbladder enlargement choledoch. What is the origin of these changes?
   A. Hepatitis.
   B. Calculous biliary disease.
   C. Chronic pancreatitis.
   D. Cancer head of the pancreas.
   E. Cancer of the liver.
2. A 68-year-old woman complains of yellow skin and sclera, gray feces, and dark urine. Body temperature rose to 37.4°. A day after the pain in the right hypochondrium, jaundice appeared. The abdomen is soft, painful in the right hypochondrium, the liver is not enlarged. Murphy's sign is positive. The gallbladder is enlarged. What is the most likely diagnosis?
   A. Acute cholecystitis, obstructive jaundice.
   B. Cholestatic hepatitis.
   C. Residual choledocholithiasis.
   D. Cholecystolythiasis.
   E. Cancer head of the pancreas.
3. Female 53-year old was operated with cholecystectomy for acute calculous cholecystitis one month ago. The operation finished outer drainage of choledoch. Cholangiography was not made. Drainage removed on 7 days after the operation. Three days ago, the temperature increased to 38.8, had chills and sweating. Scleras are yellow, stool is grey color. Abdominal palpation reveals pain in the right subcostal area. Formulate a preliminary diagnosis?
   A. Residual choledocholithiasis, cholangitis, obstructive jaundice.
   B. Postcholecystectomy syndrome.
   C. Abscess of the liver.
   D. Stenosis papillitis.
   E. Stricture of the choledoch.
4. Indications to choledochotomy:
   A. Empyema of the gallbladder.
   B. Cholangitis, obstructive jaundice, stones in the duct.
   C. Acute pancreatitis.
   D. Hepatitis.
   E. Perforated ulcer.
5. 43 years old patient complains of having night pain in the right subcostal area, general weakness, bloating, and yellowness of the scleras. ALT 1.9., AST 2.4., bilirubin 88 μmol/L (direct 40, indirect 48). What is your recommendation for treatment?
   A. Bile stimulated.

B. Antispasmodic.
C. Vitamins.
D. $H_2$-blockers.
E. Ice on the abdomen wall.

6. Child 9-years old complaints of abdominal pain, more from the right, subfebrile temperature, vomiting that does not bring relief. In the blood, clotting speed of erythrocytes was found. He has been already ill for three years. What disease caused this clinical picture?
   A. Chronic cholecystitis
   B. Chronic enterocolitis
   C. Ulcerative colitis
   D. Dyskinesia of the bill duct.
   E. Acute intestinal infection
7. A 53-year-old woman came to the surgical department with complaints of pain in the right hypochondrium, radiating to the right shoulder, nausea, vomiting, increased body temperature to 37.8 C. The abdomen is distended on palpation, tenderness in the right hypochondrium. Positive symptoms of Ortner and Musi-Georgievsky. In the general blood test - leukocytosis 12.6·109/l. Formulate a preliminary diagnosis:
   A. Tumor of the liver
   B. Acute pancreatitis.
   C. Acute gastroduodenitis.
   D. Calculous biliary disease.
   E. Perforated ulcer.
8. Which of these operations used for postcholecystectomy syndrome and stenosis of large duodenal papilla?
   A. Choledochojejunoanastomosis.
   B. Hepaticotomy.
   C. Resection of the major duodenal nipple.
   D. Transduodenal choledochoduodenostomy.
   E. Endoscopic papillosphincterotomy.
9. What type of operation is used for removing stones in ampoule of the major duodenal papilla?
   A. Transduodenal papillosphincterotomy.
   B. Cholecystectomy.
   C. Choledochotomy.
   D. Segmental resection of choledochal.
   E. External drainage of choledochal.
10. What operation results in syndrome of “blend bag”?
    A. Choledochoduodenostomy.
    B. Cholecystectomy.
    C. External drainage of choledochal.
    D. Choledochotomy.
    E. Papillosphincterotomy.

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| D | A | A | B | A | A | D | E | A | A |

**Materials for self-education**

| Main tasks | Notes(instructions) |
|---|---|
| <u>To repeat:</u><br>– Anatomy of a gall bladder and hepatoduodenal ligament.<br>– Pathophysiology of liver and pathophysiology extrahepatic biliary tract.<br>– Pathogenesis of calculous biliary disease and obstructive jaundice and complications. | – Represent methods of chronic calculous cholecystitis diagnostics using scheme.<br>– Make up a block diagram of symptoms of. calculous biliary disease and obstructive jaundice and complications. |
| <u>To study:</u><br>– Age-specific features of calculous biliary disease and obstructive jaundice and complications.<br>– Tactic at complications of obstructive jaundice. | – To do a differential diagnostic of abdominal painful syndrome.<br>– To do a differential diagnostic of types of jaundice. |

# TOPIC 6. BLEEDING SYNDROME FROM THE DIGESTIVE TRACT. SURGICAL ASPECTS OF GASTRIC ULCER AND DUODENAL ULCER

## 6.1. Bleeding syndrome from the digestive tract. Causes of occurrence, diagnosis and differential diagnosis, treatment tactics.

**Overview.**

Hemorrhage from the gastrointestinal tract is a common and serious clinical problem. In the United States, 1% to 2% of acute hospital admissions are for patients requiring evaluation and treatment of gastrointestinal hemorrhage. With an incidence of 170 per 100,000 adults per year, gastrointestinal hemorrhage is a leading diagnosis in patients admitted to intensive care units (ICUs). Although the overall mortality rate for these patients ranges from 5% to 12%, the mortality rate in patients with persistent or recurring hemorrhage approaches 40%. Mortality is linked not only to the degree of hemorrhage but also, more importantly, to the coexisting medical conditions in the patient with hemorrhage. Up to 85% of bleeding episodes cease spontaneously, allowing a less urgent approach to identify the source of bleeding and to provide definitive therapy; however, 15% of patients present with major, ongoing bleeding that require aggressive emergency diagnosis and management to allow successful clinical outcomes. These high-risk patients are most likely to require surgical intervention and to have poor outcomes.

Hemorrhage can arise in any area of the gastrointestinal tract: the esophagus, stomach and duodenum, small bowel, and colon as well as organs that empty secretions into the gastrointestinal tract, such as the liver through the biliary system and the pancreas through the pancreatic duct. Although the spectrum of conditions giving rise to acute hemorrhage varies, more than 85% of major bleeding episodes can be linked to one of four diagnoses: peptic ulcer disease, variceal hemorrhage, colonic diverticulosis, or angiodysplasia. Other sources of hemorrhage are distinctly less common. Gastrointestinal hemorrhage spans the socioeconomic strata and is equally common in urban and rural environments. Only advancing age appears to be a risk factor for hemorrhage that applies across the full spectrum of bleeding conditions of the intestinal tract. Up to half of patients with acute gastrointestinal hemorrhage are older than 60 years of age.

Numerous advances in medical technology during the 1990s, particularly the improved availability and application of diagnostic and therapeutic endoscopy, have been instrumental in the evaluation and successful treatment of patients with major bleeding. Although surgery is required for control of hemorrhage in only 5% to 10% of patients hospitalized with gastrointestinal hemorrhage, it remains an essential emergency intervention for those patients with severe or recurrent hemorrhage from both the upper and lower gastrointestinal tract. Successful collaboration between the surgeon and the gastroenterologist is essential for optimal management of these complicated patients. In all patients, regardless of bleeding source, successful initial management requires that the treating physician be mindful of the potential severity of gastrointestinal hemorrhage. Appropriate resuscitation to restore volume and red blood cell deficits is critical in patients with major hemorrhage. This resuscitation phase must

be followed by rapid diagnosis of the source of bleeding. Subsequently, establishment of appropriate specific therapies may be done to achieve successful management.

**Educational aims:**

- Interrogation and clinical inspection of patients with GI bleeding.
- To determine the etiologic and pathogenic factors of GI bleeding.
- To know the complications of peptic ulcer.
- To develop a plan of examination of the patients with GI bleeding.
- To estimate results of palpation, percussion of abdomen, X-ray, endoscopy, laboratory data.
- To make a differential diagnosis, substantiate and formulate a diagnosis of GI bleeding.
- To determine the indications for treatment of patients with GI bleeding.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomo-physiological information about a stomach and duodenum, intestine and colon.
- Etiology and pathogenesis of GI bleeding.
- Manifestations of GI bleeding.
- Modern methods of instrumental diagnostics of GI bleeding.
- Differential diagnostics of GI bleeding with other internal bleeding.
- Basic principles of conservative treatment of GI bleeding.
- Modern methods of surgical treatment of GI bleeding.
- Question of prophylaxis of postoperative complications, rehabilitation and health centre system of patients.

**A student must be able to:**

- Collect and estimate anamnesis of the patients with GI bleeding.
- Use methods of examination patients with GI bleeding.
- Set the algorithm of examination patient.
- Estimate the results of laboratory, instrumental inspection of patients.
- Determine optimum medical tactics for a specific patient (conservative, surgical), and in the case of GI bleeding
- To prove necessity of pre-operation preparation of patient and to define the operation of choice depending on localization, age, sex.

**Terminology.**

| Term | Definition |
|---|---|
| Bleeding gastroduodenal ulcers | Are due to bleeding in the gastrointestinal tract cavity as a result of increasing necrosis in the ulcer area with blood vessels, with the subsequent damage of their walls. |
| Mallory-Weiss syndrome | Upper gastrointestinal bleeding secondary to longitudinal mucosal lacerations at the gastroesophageal junction or gastric cardia. |

| | |
|---|---|
| Dieulafoy's lesion (*exulceratio simple)* | is a medical condition characterized by a large tortuous arteriole in the stomach wall that erodes and bleeds |
| Hematemesis | vomiting of blood or bloody gastric contents (“coffee grounds”) |
| Melena | passage of dark tarry or maroon stool |
| Hematochezia | passage of bright red blood from the rectum |

**Content:**

Upper gastrointestinal hemorrhage is present in 85% of patients with acute gastrointestinal bleeding; lower gastrointestinal bleeding occurs in 10% to 15% of patients, with the small bowel as the source in only 1% to 5% of patients. Hemorrhage from small bowel sources may be difficult to diagnose and is frequently referred to as hemorrhage of obscure origin. The introduction of video capsule endoscopy 3 years ago has enhanced the diagnosis and treatment of the uncommon small bowel lesion.

**Initial evaluation and treatment of patients with acute gastrointestinal hemorrhage**

Initial management of a patient with acute gastrointestinal hemorrhage has four primary goals: (1) comprehensive patient assessment, with attention to hemodynamic status and identification of significant medical co morbidities; (2) institution of appropriate resuscitation and monitoring; (3) identification of the major source of gastrointestinal bleeding; and (4) institution of specific therapeutic interventions to stop or control the bleeding. When the level of severity of the bleeding is clarified and initial assessment and resuscitation are complete, the patient may be triaged to the appropriate level unit of care.

***Goal 1: Initial Patient Assessment -*** Most patients with acute gastrointestinal hemorrhage present for initial assessment in the emergency department. One quarter of patients develops gastrointestinal hemorrhage during hospitalization for a concurrent illness; this group is particularly high risk for subsequent mortality. Initial assessment in either case calls for a focused history and physical examination, with attention to risk factors for gastrointestinal hemorrhage and laboratory evaluation.

***History*** Except for patients in hemorrhagic shock, revealing information can be obtained from the patient’s history. The essential elements to be ascertained are the characteristics of the bleeding; the onset and duration of bleeding (hours or days antecedent); the associated symptoms; the use of concurrent medications; and previous significant medical conditions, particularly liver diseases.

***Characteristics of Bleeding*** Acute gastrointestinal hemorrhage can present with hematemesis (vomiting of blood or bloody gastric contents), melena (passage of dark tarry or maroon stool), or hematochezia (passage of bright red blood from the rectum). On initial evaluation of a patient with acute gastrointestinal hemorrhage, it is important to determine whether the patient has experienced hematemesis, melena, or hematochezia. Gastrointestinal bleeding that is slow or intermittent is usually not evident to the patient; hence, the term occult is associated with this pattern of blood loss. Such patients present to primary care venues with secondary signs of slow blood loss, such as anemia or fatigue. Hematemesis is diagnostic of upper gastrointestinal bleeding, which is, bleeding from the esophagus, stomach, or duodenum. Rarely,

hematemesis may result from brisk hemorrhage from the nasal passages or pharynx when the patient swallows large volumes of blood. Melena can be indicative of either upper or lower gastrointestinal hemorrhage. Dark, tarry stools are most commonly a sign of an upper gastrointestinal source in which the blood has traversed the small bowel and colon. Gastric acid degrades hemoglobin to hematin, and the actions of digestive enzymes and luminal bacteria further contribute to the appearance of melena. Melena may also represent bleeding from lesions in the small bowel or right colon. Hematochezia is the characteristic sign of colonic hemorrhage and reflects rapid elimination of blood from the bowel. Ten percent of patients with very rapid upper gastrointestinal hemorrhage may also have a history of hematochezia and syncope. It is essential to determine the onset of bleeding and the frequency of episodes of hematemesis, melena, or hematochezia and to make a rough estimate of the volume of blood loss.

***Associated Symptoms*** Inquiry regarding associated symptoms is also of important value. A history of orthostatic dizziness or syncope indicates rapid and profound blood loss. Antecedent dyspepsia is suggestive of peptic ulcer disease; crampy abdominal pain is more consistent with upper gastrointestinal bleeding, whereas hematochezia is usually painless. Antecedent vomiting may suggest Mallory-Weiss tears; weight loss raises the possibility of malignancy.

***Medications*** The risk for gastrointestinal ulceration and hemorrhage is elevated in patients taking salicylates or nonsteroidal anti-inflammatory drugs (NSAIDs). Use of these medications is linked not only to gastritis and gastric and duodenal ulcers but also to much less commonly seen ulcerated lesions of the colon and small bowel. Further, salicylates and NSAIDs impair platelet function and may contribute to poor coagulation in patients who develop the complication of hemorrhage. Use of other medications that predisposes the patients to hemorrhage, such as warfarin and low-molecular-weight heparin, should be elicited.

***Past Medical History*** The past medical history should identify previous episodes of gastrointestinal bleeding or past history of conditions associated with acute hemorrhage.

***Physical Examination*** The major initial objective of the physical examination is to determine the degree of blood loss and volume depletion. Patients in shock with hypotension (systolic blood pressure <90 mm Hg in the supine position), tachycardia, and cold extremities can be assumed to have a deficit of at least 40% of blood volume. Patients with less severe but substantial blood loss of 20% to 40% show hypotension in the upright position. Orthostatic vital signs should be checked in all patients not in shock by allowing the patient to sit up with the legs dangling for a period of 5 minutes. An elevation in pulse of more than 20 beats/min or a fall in blood pressure of more than 10 mm Hg is a positive sign, indicative of at least a 20% blood volume loss. Signs of peripheral hypo perfusion, such as clammy, cool, pale extremities, also reflect a volume loss of at least 20%. These signs are less reliable in elderly patients, who may show exaggerated postural changes or dull changes in heart rate, or are more likely to be using β-blocker medication. All patients showing a volume deficit of greater than 20% of blood volume require prompt and aggressive resuscitation. The physical examination generally offers few specific signs relative to the source of gastrointestinal

hemorrhage. The oropharynx and nose should be examined to exclude the rare unrecognized nasopharyngeal source of bleeding. Although epigastric tenderness may be elicited in patients with peptic ulcer conditions, this is not a reliable sign. A rectal examination, noting the quality of the stool (i.e., brown, melena, or hematochezia) should also be completed.

***Initial Laboratory Assessment*** All patients with gastrointestinal hemorrhage should have basic laboratory testing, including hemoglobin and hematocrit, coagulation profile, liver function tests, serum electrolytes, and renal function. The initial hematocrit may not reflect the actual degree of hemorrhage because intravascular volume repletion from extracellular fluids may not have occurred. The finding of initial hemoglobin of less than 10 g/100 mL is associated with an increased risk for morbidity and mortality.

***Goal 2: Resuscitation*** Based on the estimated volume deficit, rapid restoration of intravascular volume is indicated. All patients with gastrointestinal hemorrhage should have two large-bore intravenous lines for administration of lactated Ringer's solution. Patients in shock should receive prompt transfusion of packed red blood cells if immediate response to electrolyte solutions is not evident. Patients with major hemorrhage, elderly patients, and patients with significant co morbidities (including cardiac, pulmonary, hepatic, or renal insufficiency) should be monitored with central venous or pulmonary artery catheters. Urine output should be monitored with a Foley catheter. Ongoing hemorrhage requires continuous resuscitation with saline and red blood cell transfusion. Coagulation defects should be corrected with component therapy or fresh frozen plasma and platelets. Hemodynamically unstable patients should have endotracheal intubation performed to protect the airways.

***Goal 3: Identification of Source of Bleeding*** Successful management of a patient with acute gastrointestinal hemorrhage requires knowledge of the site of bleeding. The specific aspects of diagnostic testing are considered in the detailed sections that follow. The general considerations are reviewed here. Patients with hematemesis, melena, or hematochezia require emergency upper endoscopy by an endoscopist capable of therapeutic intervention. Prior to this examination, a large-caliber orogastric tube should be placed to lavage the gastric lumen to enhance visual examination. Airways protection may require endotracheal intubation. Active volume resuscitation must continue during the examination. If patients are hemodynamically stable and show no signs of ongoing hemorrhage, endoscopic examination may be deferred to an urgent status (within 12 hours) provided the patient can be carefully observed in the meantime. Patients presenting with melena and hematochezia without a history of hematemesis should have a nasogastric tube inserted to examine the gastric contents. Findings of blood-tinged secretions, "coffee grounds," or guaiac-positive fluid should prompt upper endoscopy. Patients with melena and hematochezia with hemodynamic instability should have initial emergency upper endoscopy. Bleeding peptic lesions in the duodenum can elicit pyloric spasm precluding reflux of sufficient amounts of blood into the gastric lake to cause hematemesis. Endoscopy is essential to examine the duodenum in these patients. Even in stable patients, this examination should be performed within 24 hours of the bleeding episode to optimize outcome. Hemodynamically stable patients with hematochezia and patients with melena with a

negative upper gastrointestinal examination may be presumed to have acute lower gastrointestinal hemorrhage. For these patients, the choice of initial diagnostic test remains controversial. Mesenteric arteriography, colonoscopy, and labeled red blood cell scintigraphy are potentially valuable based on the clinical presentation. Diagnostic approaches are considered later in the section on lower gastrointestinal hemorrhage.

***Goal 4: Institution of Specific Therapy*** After resuscitation and identification of the source of bleeding, specific therapy can be instituted. For the 15% of patients with ongoing gastrointestinal hemorrhage and hemodynamic instability, the time interval until this intervention should be less than 2 hours, and all measures to provide ongoing support to avoid shock should be employed during the interval. Fortunately, bleeding stops spontaneously in most patients, allowing a more deliberate evaluation. After the source of bleeding has been identified, specific intervention can be provided.

## ACUTE UPPER GASTROINTESTINAL HEMORRHAGE

Upper gastrointestinal bleeding is defined as bleeding from a source proximal to the ligament of Treitz. Acute upper gastrointestinal hemorrhage is a common and potentially deadly condition accounting for approximately 85% of hospital admissions for gastrointestinal bleeding. Despite the availability of effective antiulcer medications and an improved understanding of the pathogenesis of ulcer disease, gastroduodenal ulcer disease remains the most common cause, responsible for half of bleeding episodes. In urban environments, hemorrhage from esophageal and gastric varices secondary to portal hypertension of alcoholic cirrhosis constitutes the next most frequent source, identified in 10% to 20% of patients. Acute mucosal lesions, broadly characterized as gastritis or duodenitis, are observed in 15% to 30% of patients with hemorrhage in both urban and nonurban settings. Other causes have remained relatively stable in frequency since the early 1970s, including Mallory-Weiss mucosal tears at the gastroesophageal junction (8% to 10%), esophagitis (3% to 5%), malignancy (3%), Dieulafoy's lesion (1% to 3%), and more recently, "watermelon" stomach (1% to 2%). A differential diagnosis for acute upper gastrointestinal hemorrhage is shown in Table 1.

**Table 1. A differential diagnosis for acute upper gastrointestinal hemorrhage**

| |
|---|
| Peptic disorders |
| Duodenal ulcer |
| Gastric ulcer |
| Reflux esophagitis |
| Gastritis |
| Duodenitis |
| Nonsteroidal anti-inflammatory drug–associated disorders |
| Acute gastric mucosal lesions |
| Portal hypertension–related causes |
| Esophageal varices |
| Gastric varices |
| Portal hypertensive gastropathy |
| "Watermelon" stomach |
| Mallory-Weiss tear |
| Neoplasms of the esophagus, stomach, or duodenum |
| Esophagitis due to infection |
| Dieulafoy's lesion |
| Aortoduodenal fistula |
| Angiodysplasias |
| Crohn's disease |
| Hemobilia |
| Hemorrhage from a pancreatic source |

**Clinical Presentations** Hematemesis and melena are the most frequent clinical findings in significant upper gastrointestinal bleeding. However, massive bleeding from an upper source may be associated with hematochezia. Even in instances where a lower gastrointestinal bleeding source is suspected, the passage of a nasogastric tube is required to interrogate for the presence of blood in the stomach. Although all sources of gastrointestinal bleeding have high associated morbidities, upper gastrointestinal bleeding has the highest risk for life-threatening hemorrhage.

**BLEEDING PEPTIC ULCER**

Peptic gastric and duodenal ulcers are the most common cause of acute hemorrhage in the upper gastrointestinal tract, each accounting for about 25% of cases. Bleeding peptic ulcers account for about half of the clinically significant cases of upper GI bleeding at most medical centers. About 5% of patients with peptic ulcer disease have hemorrhage as the initial manifestation of the condition, and up to 20% of patients with peptic ulcers develop bleeding at least once. Hemorrhage remains the most lethal form of complicated ulcer disease; 80% of ulcer deaths in the elderly occur as a consequence of an episode of acute hemorrhage.

The actual appearance of the ulcer at endoscopy is the most important predictor of rebleeding. Ulcers generally have one of five appearances: a clean ulcer base; a flat, pigmented spot, which may be purple, brown, or black, on the ulcer surface; an adherent clot; a visible vessel, which appears as a smooth surfaced or tubular protuberance on the smooth ulcer surface; or active bleeding with either spurting blood, continuous oozing, or oozing around an adherent clot. Descriptive identification of the ulcer characteristics has also been reported as the Forrest classification system, where FI ulcers show active bleeding, FIIa represents an ulcer with a visible vessel or pigmented protuberance, FIIb represents an ulcer with an adherent clot, FIIc represents an ulcer with a pigmented spot, and FIII shows a clean ulcer base without stigmata of bleeding. Rebleeding rates increase with ulcer size; ulcers greater than 2 cm in diameter are high risk. As discussed later, endoscopic therapy is appropriate for ulcers with stigmata of bleeding. In contrast, active bleeding which is not controlled with endoscopic measures mandates immediate surgical intervention. The transendoscopic Doppler device has been evaluated to assess blood flow beneath the ulcer surface. A positive Doppler study indicating a blood vessel beneath the ulcer was a strong predictor of rebleeding, although the value of this method of evaluation to predict rebleeding is yet to be demonstrated in a large cohort.

**Therapeutic Interventions**

***Medical Management.*** Therapy is based on clinical presentation and endoscopic findings. A patient with minimal bleeding and a clean ulcer base on endoscopy is at very low risk for recurrent hemorrhage. Young patients may be discharged with specific antiulcer therapy: an antisecretory agent (proton-pump inhibitor), cessation of NSAIDs if applicable, and H. pylori eradication with antibiotics if H. pylori positive. Older patients with this clinical presentation should be admitted for a brief period of in-hospital observation before discharge on a similar regimen. Follow-up endoscopy is indicated at 6 weeks for patients with gastric ulcer to ensure healing and to exclude malignancy but not for patients with duodenal ulcer. Patients with more significant hemorrhage and findings on endoscopy with stigmata for lesions at increased risk for rebleeding should be admitted to the hospital. Patients with clinical risk factors for adverse outcome should be admitted to the ICU.

***Endoscopic Therapy.*** Endoscopic therapy can be used to arrest active ulcer bleeding and to prevent rebleeding in patients with ulcers at high risk for rebleeding (FI, FIIa, and FIIb ulcers). Several endoscopic devices can deliver the thermal energy required to achieve coagulation. Transendoscopic bipolar electrocoagulation and heater probe therapy can decrease rebleeding rates and the need for surgical intervention by up to 50%. In skilled hands, light amplification by stimulated emission of radiation (LASER) coagulation offers similar results, although the risk for perforation is higher. Injection therapy is an equally effective nonthermal method to secure hemostasis. Available sclerosing or vasoconstriction agents include absolute alcohol, epinephrine, fibrin glue, and polidocanol. The choice of method and agent is according to the preference of the endoscopist and equipment availability.

Endoscopic therapy fails in about 20% of patients, manifest as either failure to control hemorrhage on initial presentation or as early recurrent hemorrhage. In the recent past, rebleeding patients were treated surgically.

***Surgical Therapy.*** Surgery is ultimately required in roughly 10% of patients with bleeding ulcer. Surgery is indicated for patients with active hemorrhage not responsive to endoscopic measures, significant recurrent hemorrhage after endoscopic treatment, an ongoing transfusion requirement, or transfusion requirements exceeding 6 units of packed red blood cells in a 24-hour interval. Now, the decision for surgery is balanced by endoscopic expertise, patient characteristics, and transfusion requirements.

***Choice of Operation.*** The goal of surgical intervention in bleeding peptic ulcer is to control hemorrhage. This may be achieved by duodenoplasty added PPI or, in the case of gastric ulcer, with partial gastric resection.

**Bleeding Duodenal Ulcer**

Operative intervention for bleeding duodenal ulcer requires direct exposure of the ulcer in the duodenum by way of duodenoplasty. Provided the patient is stable and free of life threatening, preoperative co morbid conditions, a definitive antisecretory procedure is indicated. Postoperative antisecretory and eradicated therapy is used. In case of negative compliance, the parietal cell vagotomy with duodenoplasty has been advocated by some surgeons. Duodenoplasty is successful in acutely controlling hemorrhage in 96% of patients; duodenoplasty with PCV is successful in acutely controlling hemorrhage in 90% of patients. Up to 10% of patients may develop early rebleeding. Repeat surgical intervention is rarely required for these patients, however, because bleeding frequently ceases with supportive measures. Reported operative mortality rates range from less than 1% to 50% based on the patient's co morbid conditions.

**Bleeding Gastric Ulcer**

Again, the primary goal of surgical intervention for bleeding gastric ulcer disease is to stop hemorrhage. Unlike duodenal ulcer, there is a chance that a gastric ulcer may be malignant; up to 1% of gastric ulcers prove to be a gastric adenocarcinoma or lymphoma. Additionally, rebleeding rates for gastric ulcer treated with simple ligation approach 30%. Ideally, therefore, the surgical procedure should include ulcer excision (organ-saving (economy) resection).

**BLEEDING CAUSED BY PORTAL HYPERTENSION**

For completeness we briefly discuss bleeding related to portal hypertension, a frequent cause of upper gastrointestinal bleeding. Bleeding from esophagogastric varices is responsible for one third of all deaths in patients with cirrhosis and portal hypertension. As many as 90% of cirrhotic patients develop esophageal varices, and 25% to 30% of these develop hemorrhage.

**Treatment of Acute Variceal Hemorrhage** Initial management calls for prompt resuscitation with particular attention to correction of volume deficit, coagulopathy, and airways management. Treatment in an ICU is imperative. Endoscopy is necessary both to confirm the source of bleeding and to allow endoscopic therapy. Both variceal sclerotherapy and rubber band ligation are effective endoscopic measures. Complications of sclerotherapy include esophageal ulceration, bleeding perforation, mediastinitis, pleural effusion, and pulmonary edema. Late stricture has also been observed. Gastric varices are not effectively treated by sclerotherapy. In good-risk patients with bleeding gastric varices, prompt surgical decompression should be considered. Concomitant treatment with vasoactive drugs is indicated. The

somatostatin given by continuous intravenous infusion, offers the best efficacy and safety profile. Vasopressin alone and vasopressin plus nitroglycerin by continuous systemic intravenous infusion are also of benefit in decreasing splanchnic blood flow and decreasing variceal bleeding.

## ACUTE GASTRIC MUCOSAL LESIONS

Acute gastric mucosal lesions (AGMLs) include a broad category of acute erosive mucosal conditions that develop in critically ill patients. Also known as stress gastritis, acute mucosal ischemia, erosive gastritis, or stress ulceration, these conditions share a common epidemiology and clinical presentation. Lesions resembling AGMLs are also observed in patients on chronic NSAID therapy.

**Treatment:** Emergency evaluation with upper endoscopy, a procedure that should be completed in the ICU, is indicated. Careful attention to prevent aspiration and hypovolemia during the procedure is mandatory. If a solitary site of bleeding is identified, endoscopic therapy, such as thermal or bipolar electrocoagulation, fibrin glue application, or injection therapy, is appropriate. Frequently, however, bleeding is too diffuse to allow endoscopic therapy. In this case, aggressive medical management with transfusion and component therapy to correct coagulation defects and anemia is indicated. The role of angiography in stress ulceration is limited to diffuse, unremitting hemorrhage. Selective celiac catheterization may allow identification of the bleeding arteries of origin. Embolization with coils or collagen gel or selective intra-arterial infusion of vasopressin may arrest bleeding in up to 80% of patients provided that selective bleeding vessels can be identified. Surgery is rarely used to treat AGML. Only those patients who have failed aggressive medical management and endoscopic therapy and who have treatable critical illness are candidates for surgery. Multiple sites are usually found, leaving only subtotal or near-total gastrectomy, with Roux-en-Y gastrojejunostomy as the only viable option. Regardless of surgical procedure, the postoperative mortality rate is high, in excess of 50%. Death is usually from multisystem organ failure.

## MALLORY-WEISS TEARS

About 10% of cases of upper gastrointestinal hemorrhage are caused by Mallory-Weiss tears. The lesion is characterized by a tear in the proximal gastric mucosa near the esophagogastric junction. Up to 90% of these lesions stop bleeding spontaneously without specific intervention. Patients with cirrhosis and portal hypertension with coagulopathy are at greatest risk for mortality, which overall averages 3%. Initial assessment and treatment should include prompt history and physical examination, resuscitation, and endoscopic evaluation. Endoscopic therapy by either injection or thermal energy is efficient in patients with active bleeding. Transfusion of packed red blood cells is required in 40% to 70% of patients. Patients with active bleeding at initial endoscopy and those with coagulation disorders are at greatest risk for rebleeding; roughly 30% bleed again within the first 24 hours. Medical therapy includes acid reduction with antisecretory agents. Surgery is rarely required for control of hemorrhage. If bleeding fails to stop after endoscopic therapy, laparotomy for oversewing of the mucosal tear through a high gastrotomy is appropriate. An acid-reducing procedure is not required.

## UNUSUAL CAUSES OF ACUTE UPPER GASTROINTESTINAL HEMORRHAGE

***Esophageal Sources*** The esophagus is the source of major hemorrhage in fewer than 3% of patients admitted for evaluation of acute upper gastrointestinal hemorrhage. The most common causes are infectious esophagitis, gastroesophageal reflux disease (GERD), Barrett's esophagus, malignancy (including adenocarcinomas and squamous carcinomas), medication-induced erosions, Crohn's disease, and radiation. Patients with human immunodeficiency virus (HIV) infection and other immunocompromised patients are at particular risk for infectious esophagitis, including erosive esophagitis caused by Candida albicans, other fungi, herpes simplex virus, cytomegalovirus, and mycobacterial infection. Bleeding may be massive, although episodes of less severe hemorrhage are typical. Therapy is targeted to the cause of bleeding. If active hemorrhage is identified, endoscopic electrocoagulation or heater probe therapy is usually effective in stopping hemorrhage, at least temporarily while definitive management is planned. Specific therapy is targeted to the etiology and includes appropriate antibiotics for infectious causes, proton-pump inhibitors for reflux-associated conditions, and definitive multimodality cancer therapies for malignant tumors. Emergency surgery to control hemorrhage is rarely required. Treatment with definitive surgical management, resection for tumors, or antireflux therapy for patients with reflux-induced esophagitis is dictated by the specific cause of the hemorrhage.

***Dieulafoy's Lesions*** arise as a result of malformations (aneurysms) of the vessels of the submucosal layer and are quite rare. During endoscopic examination, the source of bleeding is not revealed, only a stream of blood from under the mucous membrane is recorded. If a lesion is definitively identified, the site should be marked endoscopically with India ink injection to allow precise surgical resection. Recurrent hemorrhage is common; often, several episodes occur before accurate diagnosis. Efforts at endoscopic ablation with sclerotherapy and electrocoagulation have not proved effective in the few reported series. Rather, appropriate definitive management calls for wedge resection of the gastric wall. Precise endoscopic localization of the lesion allows this limited resection in lieu of more extended blind gastric resection. Because the condition is not associated with peptic mucosal injury, vagotomy is not indicated.

***Aortoenteric Fistula***

***Aorto-enteric fistula*** occurs as a result of inflammation between the aorta and the digestive organ. It occurs primarily - actually between the aorta and the intestine, or secondary - between the part of the synthetic or autograft replaced as a result of the operation. Fistula occurs with any part of the intestine (more often with the duodenum or jejunum). Emergency upper endoscopy is mandatory for all patients with suspected aortoenteric fistula. If endoscopy is negative, computed tomography (CT) to look for evidence of inflammation at the aortic anastomosis is indicated. Others advocate emergency angiography, including lateral views, to identify the small mycotic aneurysm that is frequently present. Angiography should be pursued in all patients with negative CT scans. In patients with exsanguinating hemorrhage, emergency laparotomy with control of the proximal aorta is indicated. Effective surgical

management calls for removal of the aortic graft and extra-anatomic vascular bypass to restore distal aortic flow.

**ACUTE LOWER GASTROINTESTINAL HEMORRHAGE**

Acute lower gastrointestinal bleeding is hemorrhage arising distal to the ligament of Treitz. The colon is the source of hemorrhage in more than 95% to 97% of cases, with the remaining 3% to 5% arising in small bowel sites. Lower gastrointestinal bleeding accounts for about 15% of major episodes of gastrointestinal hemorrhage and hence is much less common than upper gastrointestinal bleeding. The incidence of lower gastrointestinal bleeding increases with age, reflecting the parallel increase in acquired lesions responsible for colonic bleeding: diverticulosis and angiodysplasias. The differential diagnosis of acute lower gastrointestinal hemorrhage is shown in Table 2.

**Clinical Presentation** The hallmark of acute lower gastrointestinal hemorrhage is hematochezia; passage of bloody stool, blood, or blood clots per rectum. If bleeding is slower and of lesser volume, melena may also be a presenting sign, although this is more characteristic of an upper gastrointestinal source.

**Table 2. Differential Diagnosis of Lower Gastrointestinal Hemorrhage**

| |
|---|
| Diverticulosis |
| Angiodysplasia |
| Neoplasm |
| Polyps |
| Meckel's diverticulum |
| Inflammatory disorders |
| Ulcerative colitis |
| Crohn's disease |
| Infectious colitis |
| Radiation-induced disorders |
| Nonsteroidal anti-inflammatory drug–associated disorder |
| Hemorrhoids |
| Varices |
| Dieulafoy's lesions |
| Osler-Weber-Rendu syndrome |
| Aortoenteric fistula |
| Vasculitis |
| Mesenteric ischemia |

Similarly, up to 15% of patients with massive hemorrhage from an upper gastrointestinal source may present with hematochezia, which is indicative of at least 1000 mL of hemorrhage over a short interval from an upper gastrointestinal source. Roughly half of patients present with both a decrease in hemoglobin and hematocrit and hemodynamic instability; 30% have orthostatic changes, 10% syncope, and 19% shock. Although lower gastrointestinal hemorrhage represents a genuine emergency, it is generally less life-threatening than upper gastrointestinal hemorrhage. Patients are

less likely to present in shock, more likely to cease bleeding spontaneously, and usually have a lower transfusion requirement.

**Colonic diverticulosis** represents the most common source of lower gastrointestinal hemorrhage, responsible for 40% to 55% of cases of hemorrhage in most series. Colonic diverticula are common acquired lesions of the abdominal colon. Although 40% of patients in the 5th decade of life have diverticula, this incidence rises to 80% by the 9th decade. Diverticulosis is complicated by bleeding in 5% of cases.

As a rule, the rupture develops in the intramural branches of the marginal artery. Tears are localized on the dome or opposite the mesenteric edge of the diverticulum. Despite the greater frequency of diverticula of the left half of the colon, diverticula of the ascending colon bleed heavily. As a rule, bleeding stops on its own.

**Angiodysplasias** are responsible for 3% to 20% of cases of acute lower intestinal bleeding. Angiodysplasias, also referred to as arteriovenous malformations, are small ectatic blood vessels in the submucosa of the gastrointestinal tract. The overlying mucosa is often thin, and superficial erosion at the site of an angiodysplasia has been observed on histologic examination of surgical or autopsy specimens. Angiodysplasias are identified in 1% to 2% of autopsy evaluations and increase in frequency with the age of the patient. Angiodysplasias may occur throughout the gastrointestinal tract and represent the most common cause of hemorrhage from the small bowel in patients older than 50 years of age. Angiodysplasias are evident on colonoscopy as red, flat lesions about 2 to 10 mm in diameter. Lesions may appear stellate, oval, sharp, or indistinct. Colonoscopy is the most sensitive method to identify angiodysplasias, although angiography is also able to identify these lesions. The use of meperidine during colonoscopy may decrease the ability to identify angiodysplasias because of a reduction in mucosal blood flow. Another study has identified that the use of a narcotic antagonist may increase the size of angiodysplasias and enhances the detection rate. On angiography, angiodysplasias appear as ectatic, slowly emptying veins or as arteriovenous malformations with brisk, early venous filling. More than half of angiodysplasias are localized to the right colon, and bleeding from angiodysplasia correlates with this distribution. Angiodysplasias may be associated with many medical conditions, including end-stage renal disease, aortic stenosis, von Willebrand's disease, and others. It is not clear whether this association reflects the greater tendency of angiodysplasias to bleed in these conditions or whether, in fact, angiodysplasias are more common structural findings in them.

**Colonic neoplasms**, including adenomatous polyps, juvenile polyps, and carcinomas, present in a variety of manners. Typically, bleeding from these lesions is slow, characterized by occult bleeding and secondary anemia. These neoplasms can bleed briskly, however, and in some series, up to 20% of cases of acute hemorrhage are ultimately found to arise from colonic polyps or cancers. Juvenile polyps are the second most common cause of hemorrhage in patients younger than the age of 20 years.

A wide variety of inflammatory conditions can cause acute lower gastrointestinal hemorrhage. Hemorrhage is rarely the presenting sign; rather, it develops in the course of the disease, and the cause is suspected based on the patient's history. Up to 20% of cases of acute lower gastrointestinal hemorrhage may be due to one of these inflammatory conditions. Most episodes of bleeding cease spontaneously or with

specific therapy directed at the cause. Hemorrhage complicates the course of ulcerative colitis in up to 15% of cases. Emergency colectomy for persistent hemorrhage accounts for 6% to 10% of emergency surgical colectomies in patients with this disease. Crohn's disease is less likely to cause massive colonic hemorrhage and occurs in roughly 1% of patients with this condition. Infectious causes include Escherichia coli, typhoid, Cytomegalovirus, and Clostridium difficile. Radiation injury is most common in the rectum after pelvic radiotherapy for prostate or gynecologic malignancies. Bleeding is most common 1 year after radiation treatments but may occur up to 4 years later. Patients with immunosuppression or acquired immunodeficiency syndrome (AIDS) are at risk for acute lower intestinal hemorrhage from a unique set of causes. Cytomegalovirus is the most common cause; Kaposi's sarcoma, histoplasmosis, and perianal fistulas and fissures are also problematic and are more likely to hemorrhage in patients with AIDS-induced thrombocytopenia.

**Vascular causes** of acute lower intestinal hemorrhage include the vasculitides (polyarteritis nodosa, Wegener's granulomatosis, rheumatoid arthritis, and others), which are associated with punctate ulceration of the colon and small bowel. Colonic ischemia with mucosal ulceration and friability may also result in acute hemorrhage, often in the setting of acute abdominal pain and sepsis. Acute mesenteric ischemia may be heralded by an episode of hematochezia in the context of severe abdominal pain, preexisting vascular disease, arterial embolism risk, or hypercoagulability. Although hemorrhage is an element in the clinical management of these patients, only rarely does the control of hemorrhage become the major focus of therapy. Rather, restoration of visceral perfusion is the primary therapeutic objective.

**Hemorrhoids** are usually noted on physical examination in more than half of patients with lower gastrointestinal hemorrhage. In fewer than 2% can the hemorrhage be attributed to these lesions, however. Unless unequivocal signs of bleeding are evident on anoscopy, investigation of the patient for another source of lower intestinal bleeding should be pursued. Patients with portal hypertension may develop massive hemorrhage from hemorrhoids, as can patients with HIV-associated thrombocytopenia with hemorrhoids.

**Uncommon Causes** Rare causes of lower gastrointestinal hemorrhage include solitary rectal ulcer, Dieulafoy's lesion of the colon, portal colopathy, NSAIDs, intussusceptions, or bleeding following colonoscopic biopsy or polypectomy.

The initial history and physical examination are directed to determining the potential source of the hemorrhage and the severity of initial hemorrhage. Most cases eventually are determined to result from angiodysplasia or diverticulosis, both of which are usually asymptomatic before initial hemorrhage. Nonetheless, the initial history should exclude other, less common causes of the bleeding. Specific inquiry should be made regarding use of NSAIDs or anticoagulants. Abdominal pain or recent diarrhea and fever may point to colitis, either infectious or ischemic. Patients with prior aortic surgery should be considered to have an aortoenteric fistula until proved otherwise. Prior radiation therapy for pelvic malignancy may indicate radiation proctitis. Recent colonoscopy may suggest bleeding from a biopsy or polypectomy site. The cause of previous episodes of bleeding should be elicited, as should the possibility of a history of inflammatory bowel disease. Family history of polyposis syndromes or colonic

malignancy may also be pertinent. Young patients—those less than 30 years of age—are at greatest risk for bleeding from Meckel's diverticulum or intestinal polyps. Physical examination should include measurement of orthostatic vital signs in patients without evident shock. All patients should be resuscitated, as outlined in the previous section. Pertinent findings on physical examination may include scars from previous abdominal incisions, the presence of abdominal masses, or skin and oral lesions suggestive of polyposis syndromes. Stigmata of cirrhosis suggestive of bleeding from hemorrhoids or varices secondary to portal hypertension should be considered. The rectal examination is important to identify any anorectal pathology, including tumors, ulcers, or polyps. The color of the rectal contents and the presence of formed stool or blood clot should also be noted. Anoscopic examination to exclude hemorrhage from hemorrhoids should be completed. A nasogastric tube should be inserted to look for blood or coffee ground–like material to exclude an upper gastrointestinal source. In patients with hematochezia and hemodynamic instability, emergency upper endoscopy is required.

**Diagnosis**

Today, emergency surgery is not a priority method for clarifying the source of bleeding. The first place is occupied by endoscopy (including medical), supplemented with hemostatic, antisecretory and replacement therapy. Local diagnostic protocols mostly depend on the diagnostic capabilities of a specific hospital. In addition to endoscopy, selective visceral angiography and scintigraphy with red blood cells labeled with 99mTc are also priorities.

**Colonoscopy** Since most intestinal bleeding stops on its own, a colonoscopy should be performed within the first 12 hours after hospitalization. In this setting, colonoscopy can be completed after colonic purging. Positive findings on colonoscopy include identification of an active bleeding site, identification of a nonbleeding visible vessel, clot adherent to a diverticular ulcerated orifice, clot adherent to a discrete focus of mucosa, or fresh blood localized to a colonic segment. Hemorrhage can be attributed only to lesions with clear stigmata of bleeding. Patients presenting with massive lower gastrointestinal hemorrhage are poor candidates for emergency colonoscopy. The procedure may be ineffective due to the impossibility of full visualization of the mucous membrane, despite attempts to clean it. Patients with massive hemorrhage have hemodynamic instability, precluding the use of sedation and increasing the risk for hypoxemia and complication. Further, resuscitation may be compromised during the procedure. Hence, colonoscopy is most appropriately used as the initial diagnostic procedure in patients presenting with acute hemorrhage that has ceased or in patients with a more moderate degree of bleeding.

**Selective Visceral Angiography** To perform diagnostic and therapeutic mesenteric arteriography in case of bleeding from the lower parts of the digestive tract, contrast is injected into one of the mesenteric arteries. Given the characteristic intermittent bleeding seen in lower gastrointestinal hemorrhage associated with diverticulosis, arteriovenous malformations, and other causes, bleeding may have ceased by the time of the study. Some radiologists have advocated evocative testing, including intra-arterial vasodilators, heparin, and fibrinolytic agents, in an effort to identify a bleeding source accurately. This approach does not appear to be warranted

except in patients with refractory intermittent episodes of hemorrhage in a fully staffed suite. Because 90% of cases of hemorrhage cease spontaneously, and only 10% rebleed, such evocative testing is inappropriate for most patients. Complications of angiography include the development of central (stroke) or peripheral (femoral artery) thrombosis or hematoma, renal failure, and paresis of the lower extremities. Their frequency reaches 10% of cases. Indications for performing this study in elderly people should be thoroughly substantiated (profuse bleeding).

**Technetium 99m-Red Blood Cell Scintigraphy** In this noninvasive nuclear medicine imaging procedure, the patient's red blood cells are labeled with a technetium isotope and reintroduced into the circulation. With each bleeding episode, labeled blood is shed into the colonic lumen, creating an isotopic focus that can be imaged with whole abdominal scintigraphy. After extravasation into the lumen, the blood moves through the colonic lumen, generally from the right colon to the left, but occasionally in retrograde fashion because of colonic contractions. If bleeding is present at the time of injection and initial imaging, $^{99m}$Tc-red blood cell scans can accurately identify a source of bleeding in up to 85% of cases. The informativeness of this method reaches 85% of cases with active bleeding, while at the same time, with stopped bleeding or its recurrence, this method is inferior to other methods.

It is indisputable that when deciding on surgical intervention, the diagnosis must be confirmed by one of two methods (endoscopically or angiographically).

**Treatment**

**Endoscopic Treatment** Thermal heater probes, electrocoagulation, and sclerotherapy have been used. Colonic diverticula bleeding was successfully stopped by electrocoagulation, although this approach has not been widely embraced. Endoscopic stoppage of bleeding from a diverticulum is dangerous due to the development of recurrence. Endoscopic hemostasis of angiodysplasias is effective in 80% of cases, while the recurrence rate is 15%. Must to avoid precipitating massive hemorrhage when treating angiodysplasias. Most endoscopists begin endoscopically to stop the bleeding from the periphery to the center. Endoscopic polypectomy at the height of bleeding showed good results. Recurrence of bleeding does not exceed 2% during the first two weeks after surgery.

**Angiographic Treatment** When using angiography as a diagnostic procedure for bleeding, you should try to use it also for a temporary therapeutic purpose (intra-arterial administration of the vasoconstrictor vasopressin is effective in 80% of patients). Rebleeding is common, however, after discontinuing the therapy. Complications are frequent and serious and include hyponatremia, myocardial infarction, edema of the lungs, and mesenteric thrombosis. Transarterial vasopressin is contraindicated in patients with chronic diseases of the cardiovascular system. Endovasal administration of vasoconstrictors is used as primary hemostasis before surgery or transcatheter embolization as final hemostasis at the height of bleeding (especially when surgery is contraindicated). For this, both gelatin sponges and microspirals are used. Given the weakness of the collateral blood flow of the large intestine, these procedures may be complicated by colonic infarction heralded by abdominal pain, fever, and sepsis. Hence, like vasoconstrictive therapy, this procedure should be limited to patients in

whom surgery is contraindicated and for temporary hemostasis when surgery is unavoidable.

**Surgery** The operation is indicated when endovascular manipulations are unsuccessful in the case of ongoing bleeding or its recurrence. Transfusion of more than 6 units of packed red blood cells, ongoing transfusion requirement, or persistent hemodynamic instability is an indication for colectomy in acute hemorrhage. Patients who develop recurrent lower gastrointestinal hemorrhage are also appropriately treated with colectomy. Hemicolectomy after stabilization of the patient's condition should be prioritized over subtotal colectomy, which is accompanied by a significantly higher postoperative mortality rate (up to 25%) and causes the development of a number of serious complications, especially in elderly patients (dehydration). There is no indication for a blind segmental colectomy, for which rebleeding rates as high as 75% are seen. Mortality after colectomy for acute lower gastrointestinal hemorrhage overall is less than 5%. As in upper gastrointestinal hemorrhage, in elderly patients, death occurs as a result of the development of such complications from the cardiovascular and respiratory systems, liver and kidney failure, and not directly from blood loss. Thoughtful timely management can lead to a successful outcome in most patients.

**RARE CAUSES OF GASTROINTESTINAL HEMORRHAGE FROM AN OBSCURE SOURCE**

The small bowel is a rare source of acute hemorrhage. Only 2% to 5% of patients with acute gastrointestinal hemorrhage are ultimately determined to have bled from a small intestinal source. This low frequency is fortunate because the small bowel is a difficult organ to visualize and precise detection of the bleeding lesion is characteristically delayed. Acute gastrointestinal hemorrhage from an obscure source has been reported to occur from a variety of conditions. These include radiation enteritis, small intestinal varices, Crohn’s disease, tuberculosis, syphilis, typhoid, histoplasmosis, vasculitis, small bowel ulcerated lesions in patients with gastrin-secreting tumors, and Dieulafoy’s lesions. Medical treatment is appropriate for most infectious causes and in patients with Zollinger-Ellison syndrome. Enterectomy is required in the other conditions. Disorders of the pancreas can cause acute gastrointestinal hemorrhage as blood is delivered into the duodenum through the pancreatic duct. Such bleeding has been reported in the setting of acute pseudoaneurysms after pancreatectomy and in pancreatic tumors. Bleeding is a rare complication of these disorders. Angiography may confirm the presence of a pseudoaneurysm and allow angiographic embolization for acute hemorrhage control. Pancreatic resection may be appropriate, depending on the clinical condition. The liver may also be the source of presumed acute gastrointestinal hemorrhage. Bleeding into the hepatic duct presents as gastrointestinal hemorrhage as blood enters the duodenum from the common bile duct, a condition known as hemobilia. Hemobilia has been reported to occur secondary to hepatic trauma with intrahepatic hematoma, hepatic aneurysms or other vascular malformations, hepatic tumors, hepatic abscess, or after hepatic resection or percutaneous liver biopsy. This diagnosis is usually considered when endoscopic visualization during acute hemorrhage shows blood entering the duodenum at the ampoule of Vater, depending on the clinical scenario. Selective

visceral angiography is usually required to define the source and often allows definitive management by intra-arterial embolization.

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**Tests**

1. The commonest cause of death in patients with alcoholic cirrhosis following portosystemic shunting is:
   A. Bleeding esophageal varices
   B. Hepatic failure with encephalopaty
   C. Malnutrition
   D. Hepatocellular carcinoma
   E. Cardiac failure with peripheral edema and ascites
2. A 30-year-old man with a duodenal ulcer is being considered for surgery because of intractable pain and a previous bleeding episode. Serum gastrin levels are found to be over 1000 pg/mL (normal 40-150) on three separate determinations. The patient should be told that the operation of choice is:
   A. Vagotomy and pyloroplasty
   B. Highly selective vagotomy and tumor resection
   C. Subtotal gastrectomy
   D. Total gastroctomy
   E. Partial pancreatectomy
3. All of the following problems commonly occur with the use of balloon tamponade for control of variceal bleeding EXCEPT
   A. Pneumonia
   B. Aspiration of nasopharyngeal secretion
   C. Rebleeding following removal of the tube
   D. Gastritis
   E. Esophageal ulceration or perforation
4. Omeprazole has been added to the H2-antagonists as a therapeutic approach to the management of acute bleeding gastric and duodenal ulcers. It acts by
   A. Blocking breakdown of mucosal-damaging metabolites of NSAIDs
   B. Providing a direct cytoprotective effect
   C. Buffering gastric acids
   D. Inhibiting parietal cell hydrogen-potassium-ATPase
   E. Inhibiting gastrin release and parietal cell acid production
5. What is the most reliable method for precisely locating an upper gastrointestinal lesion that is responsible for a bleeding?
   A. Upper GI series
   B. Exploratory laparotomy
   C. Upper GI endoscopy
   D. Arteriography
   E. Radionuclide scanning
6. The most common cause of massive upper gastrointestinal bleeding is
   A. Gastric ulcer

B. Erosive gastritis
C. Gastric carcinoma
D. Mallory-Weiss tear
E. Duodenal ulcer

7. A 50-year-old man is admitted with massive, bright red rectal bleeding. He recently had a barium enema that demonstrated no diverticular or space-occupying lesion. Nasogastric suction reveals no blood but does produce yellow bile. The patient continues to bleed. What is the next diagnostic step?
   A. Repeat barium enema.
   B. Colonoscopy.
   C. Upper GI series.
   D. Mesenteric angiography.
   E. Small bowel follow-through with barium.
8. All of the following statements regarding lower GI bleeding are true EXCEPT
   A. If bleeding is profuse, angiography may be useful.
   B. The mortality rate is about 10%.
   C. Persistent bleeding is an indication for surgery.
   D. Only 10%-15% of patients stop bleeding spontaneously.
   E. Blind total colectomy may be a necessary procedure
9. The correct surgical treatment for Mallory-Weiss tear of the esophagus is
   A. Transthoracic ligation of varices.
   B. Transthoracic antireflux procedure.
   C. Laparotomy, gastrotomy, and oversewing the bleeding vessel.
   D. Laparotomy and resection of the gastroesophageal junction.
   E. Antibiotics and observation.
10. Massive upper GI bleeding occurs in an otherwise asymptomatic, normal man following a violent episode of retching and vomiting without blood. The most likely cause of this man´s bleeding is:
    A. Hiatal hernia.
    B. Mallory-Weiss tear.
    C. Carcinoma of the stomach.
    D. Duodenal ulcer.
    E. Gastritis.

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| B | B | D | D | C | E | D | D | C | B |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of GI tract<br>– Physiology of GI tract<br>– Types of GI bleeding. | -To represent the methods of diagnostics of diseases of alimentary tract<br>-To make the flow diagram of mechanisms and diagnosis of GI bleeding. |

| | |
|---|---|
| Study:<br>– Stages of digestive bleeding.<br>– Methods of endoscopic hemostasis.<br>– Forrest classification<br>– Non-peptic ulcer causes of upper gastrointestinal bleeding. | -To conduct differential diagnosis with the inner bleeding of organs of abdominal cavity |

**6.2. Surgical aspects of gastric ulcer and duodenal ulcer. Etiology. Pathogenesis. Clinic of complications of peptic ulcer disease. Methods of diagnosis. Differential diagnosis. Therapeutic tactics.**

**Overview.**

Gastroduodenal ulcer is one of the most widespread diseases of organs of abdominal region. About 3,5 – 12% of population suffers from this pathology, thus 35 – 47% are people of working age. The morbidity of this disease in stomach and duodenum has increased morbidity for 38,4% for 10 years in the end of the XX century has grown in Ukraine.

Gastric outlet obstruction is the least frequent ulcer complication. Most cases are associated with duodenal or pyloric ulceration, with gastric ulceration accounting for only 5 percent of cases. Perforation of an ulcer is the formation of a hole in the wall of the stomach or duodenum, which can lead to peritonitis. In terms of frequency, this complication is inferior only to gastrointestinal bleeding (30%). Penetration of an ulcer is understood as its germination into neighboring organs or anatomical structures. The frequency of this complication does not exceed 20% among other complications of peptic ulcer. The incidence of malignancy in benign gastric ulcer is approximately about 1%.

**Educational aims:**

To understand the view and influence of ulcer disease on economic, social and biological factors on the dynamics of amount of amount of patients suffer from with complicated forms of ulcerous illness of stomach and duodenum.

On material of theme to develop the sense of responsibility for the timeliness of exposure of disease and rightness of professional actions for stopping the disease development of the operated stomach using pathogenetic grounded choice of method of surgical intervention in the complicated ulcerous illness of stomach and duodenum.

**A student must know:**

- Anatomo-physiological information about a stomach and duodenum, gastric secretion phases.
- Etiology and pathogenesis of gastroduodenal ulcer.
- Clinical view of gastric outlet obstruction and penetration.
- Modern methods of instrumental diagnosis of gastroduodenal ulcer.
- Basic principles for conservative treatment of the uncomplicated ulcerous illness.

- Basic principles for conservative treatment of the compensated pyloroduodenal stenosis, and preoperative preparation of patients with sub- and decompensated stenosis.
- Modern methods of surgical treatment for complicated peptic ulcer and duodenal ulcer.
- Informations about prophylaxis for postoperative complications, rehabilitation and health centre system of patients.
- Classification of diseases of the operated stomach.
- Causes of origin of dumping-syndrome, syndrome of abductive intestinal loop, agastric asthenia, digestive allergy, peptic ulcer of anastomosis and recurrent ulcer, gastro-intestinal fistula classification, pathological refluxes, clinics, diagnostics and methods of conservative and operative treatment.

**A student must be able to:**

- Collect and estimate information of anamnesis for a patient with complicated ulcer.
- Use physical methods of inspection for diagnostics of stenosis.
- Set the algorithm of patient's inspection and estimate the results of laboratory, instrumental inspection of patients.
- Determine optimum medical pathways (conservative, surgical), in the case of decompensated stenosis to be able to prove necessity of pre-operation preparation of patient.
- Collect and estimate information of anamnesis for a patient with the diseases of the operated stomach.
- Knowing the complaints, anamnesis, physiological and instrumental methods of inspection of patients to diagnose syndrome of dumping.
- Perform appropriate tests for diagnostics of dumping-syndrome, syndrome of efferent intestinal loop, alkaline gastritis.
- Set the algorithm of examination the patient with illness of the operated stomach and estimate the results of laboratory, instrumental inspection of patients.
- Define optimum medical options for a certain patient (conservative, surgical).

**Terminology.**

| Term | Definition |
|---|---|
| Peptic ulcers | are focal defects in the gastric or duodenal mucosa which extend into the submucosa or deeper |
| Classification of gastric ulcers by Johnson | according to classification gastric ulcers is divided on three types |
| Gastric outlet obstruction | results from fibrous scarring of chronic duodenal ulcer disease |
| Malignization | transformation of gastric epithelium in cancer |

| | |
|---|---|
| Ulcer penetration | refers to penetration of the ulcer through the bowel wall without free perforation and leakage of luminal contents into the abdominal organs |
| Perforated ulcer | is the formation of a hole in the wall of the stomach or duodenum, which can lead to peritonitis (inflammation of the abdominal cavity). |
| Diseases of the operated stomach | are the diseases which arise up after surgical treatment of peptic or duodenal ulcer or other pathologies of these organs. |

**Content:**

**PEPTIC ULCER DISEASE**

Peptic ulcers are focal defects in the gastric or duodenal mucosa which extend into the submucosa or deeper (Fig. 1).

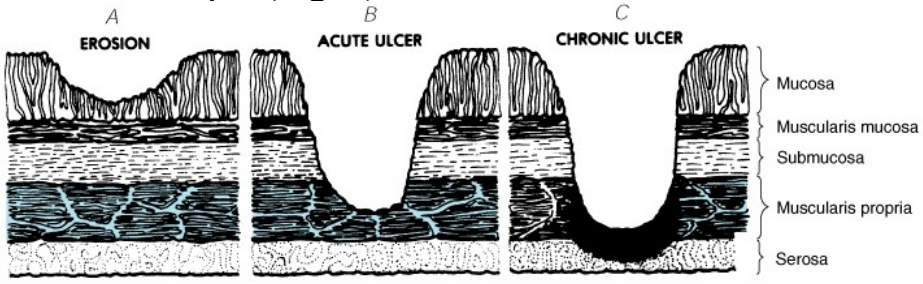



**Fig. 1. Diagram of gastric erosions and ulcers.**

They may be acute or chronic, and ultimately are caused by an imbalance between the action of peptic acid and mucosal defenses (Fig. 2).

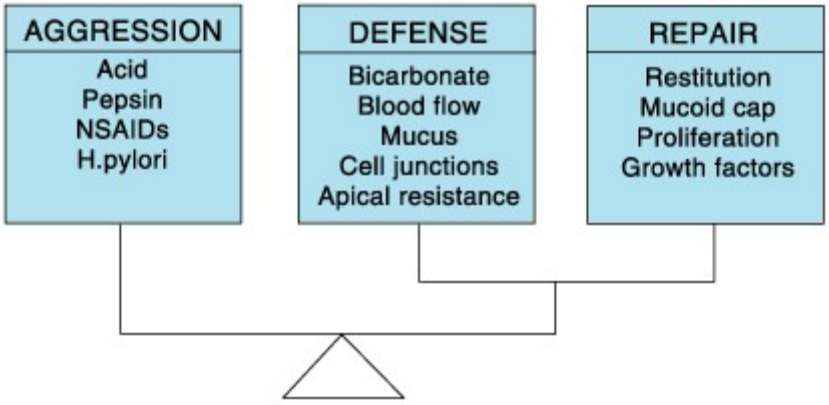



**Fig. 2. Balance of acid/peptic aggressive factors and mucosal defensive factors in the gastric mucosa.**

Peptic ulcer remains one of the most common diseases of the digestive tract. The frequency of such dangerous complications as perforation and bleeding does not tend to decrease, despite the success of modern endoscopic and medical techniques. The successful use of H. pylori-eradicating conservative treatment has faced the problem of antibiotic resistance, vagotomy has become a thing of the past, giving way to proton pump blockers, and the frequency of stress, cigarette and NSAID ulcers is decreasing.

Every year in the United States, more than 3 billion dollars are spent on patients with peptic ulcers. The frequency of peptic ulcer is 2%, and the mortality rate is 1.7 per 100,000 people. Moreover, more patients die from gastric ulcers than from duodenal ulcers. This is due to an increase in the incidence of stomach ulcers in the

elderly, especially in women (due to menopause). The death of this category of patients is associated with the development of complications such as perforation, bleeding, asymptomatic course and late referral to the hospital. Another aggessive factor is the use of NSAIDs due to concomitant cardiac pathology and the development of fatal complications (septic and hypovolemic shock) due to a decrease in immunity.

**Classification of peptic ulcer**

- acute or chronic,
- complicated or noncomplicated,
- gastric or duodenal ulcer

**Classification of gastric ulcers by Johnson (1965)**

- type I - ulcers of lesser curvature (3 cm higher than the pylorus);
- type II- double localization of ulcers simultaneously in the stomach and duodenum;
- type III - ulcers of pyloric end of stomach (not farther than 3 cm from the pylorus).

**Pathophysiology and Etiology**

The main pathogenetic factor in ulcer development is peptic acid. Among other factors of aggression, H. pylori infection and/or taking NSAIDs, smoking should be indicated. It is also important to reduce protective factors (mucosal-bicarbonate barrier, regeneration of the mucous membrane and its blood supply).

In the pathogenesis of duodenal ulcer, the influence of factors of aggression (hypersecretion) prevails, and in gastric ulcers, with the relative stability of factors of aggression (normal or reduced acidity), a decrease in the protective barrier is noted. Taking into account the role of H. pylori in the development of 80% of duodenal ulcers and 40% of gastric ulcers (eradication of *H. pylori* infection) allowed to reduce the frequency of such ulcer complications as penetration and stenosis. In the development of gastric ulcers of the I type according to Johnson, the main role belongs to the violation of the motor-evacuation function of the stomach (duodeno-gastric reflux (60% of patients) and gastrostasis (30%). Expanding knowledge about gastric oncogenesis and improving methods of morphological diagnosis of the results of polypositional gastrobiopsies made it possible to prove that the real frequency of ulcer malignancy does not exceed 1%. This made it possible to recognize that uncomplicated gastroduodenal ulcers require only conservative pathogenetic treatment.

Examples of the role of physiological and psychological stress are acute Curling's ulcer, which develops in burn patients, and Cushing's ulcer, which occurs in head injuries and after neurosurgery. Stress ulcers are often complicated by bleeding and perforation. Also, smoking increases, incl. drugs, as well as alcohol abuse (even beer) stimulate the production of hydrochloric acid. Smoking reduces the production of prostaglandins, which are a protective factor for the mucous membrane. A similar mechanism of ulcer development is also associated with coffee abuse, but these data are confirmed mainly in women.

**Clinical Manifestations**

Pain in the upper half of the abdomen worries the vast majority of peptic ulcer patients. At the same time, with duodenal localization of the ulcer, it occurs at night

(effect of the vagus nerve) and on an empty stomach (hunger pain). With gastric localization, the pain appears immediately after eating or an hour later, in this regard, patients avoid eating, losing weight, which is considered as a suspicion of malignancy of the ulcer. The diagnosis is established on the basis of exacerbations of the disease in spring and autumn (the influence of biorhythms that seasonally increase gastric secretion), taking and anamnesis of NSAIDs, smoking, infection with H. pylori, etc. Other symptoms include vomiting, which brings relief (due to a decrease in the concentration of hydrochloric acid), acid belching, heartburn, asthenoneurotic syndrome. Young men suffer from duodenal ulcer much more often, as the estrogen background of menstruating women prevents the development of this disease. With age, this ratio almost equalizes, however, due to the gastric localization of the ulcer.

**Diagnosis**

Symptoms of dyspepsia and/or pain in the upper half of the abdomen, especially in autumn and fall, require empirical antisecretory therapy (endoscopic examination is optional). However, after the age of 45, all patients in a similar situation must undergo an upper endoscopy to confirm the diagnosis and start pathogenetic treatment to prevent complications of the disease. Upper endoscopy is the main method of ulcer diagnosis to establish its localization, complications and treatment. For the differential diagnosis of an ulcer and cancer of stomach, a polypositional biopsy of various areas of the ulcer and its surrounding mucosa with further morphological research is important. Testing patients for H. pylori is mandatory (urease test, with a biological method (gastrobioptat) the sensitivity of the bacterium to antibiotics is also determined). X-ray examination can be useful for the diagnosis of such complications of the ulcer as perforation (phenomenon of pneumoperitoneum), pyloric stenosis (contrasts overstretched stomach, downcast into the small pelvis), and with gastric localization of the ulceration for differential diagnosis with diffuse (scirrhous) gastric cancer.

**Medical Treatment**

The conservative treatment of ulcer has the following objectives:

- pain suppression and creation of a comfort state of the patient,
- favoring a rapid epithelization in the ulcerous notch,
- prevention of major complications – hemorrhage and perforation.

The basic principles of treatment of gatroduodenal ulcer include:

- elimination of ulcer's morphological substrate (with medical treatment, the ulcer is scarred, with surgical treatment, its excision is performed);
- restoration of normal anatomy of the gastroduodenal zone (only surgically);
- decrease of gastric secretion (antisecretory therapy);
- elimination of the aggressive factors (smoking, alcohol, NSAID and H. pylori).

Positive compliance in treatment is important - the patient must want to be treated (regular intake of antisecretory drugs, antihelicobacter therapy), including by giving up smoking, drinking alcohol, replacing non-selective NSAIDs with other classes. Standard schemes for the eradication of H. pylori infection are given in table. 3.

**Table 3. Treatment Regimens for *Helicobacter pylori* Infections**

**Bismuth triple therapy**

- Bismuth, 2 tablets four times daily
- Metronidazole, 250 mg three times daily
- Tetracycline, 500 mg four times daily

**PPI triple therapy**

- PPI twice daily
- Amoxicillin, 1000 mg two times daily
- Clarithromycin, 500 mg two times daily
- Metronidazole, 500 mg two times daily

**Quadruple therapy**

- PPI twice daily
- Bismuth, 2 tablets four times daily
- Metronidazole, 250 mg three times daily
- Tetracycline, 500 mg four times daily

NOTE: Duration: 10–14 days. PPI – proton pump inhibitor.

Infectious disease consultation may be helpful in the compliant, symptomatic patient with persistent *H. pylori* infection following treatment; or another regimen could be tried (e.g., quadruple therapy). If initial *H. pylori* testing is negative, the ulcer patient may be treated with proton pump inhibitors and cytoprotector (sucralfate or misoprostol) for gastric ulcers may also be effective. If ulcer symptoms persist, an empiric trial of anti-*H. pylori* therapy is reasonable. Gastrokinetics (metoclopramide and domperidone) are prescribed to correct motor disorders. Conservative therapy is effective for 7-14 days (95% of duodenal ulcers heal). For gastric ulcers, the term is extended to 12 weeks.

**Surgical Treatment**

Indications for surgical intervention can be divided into vital, absolute and relative. Vital indications include conditions that lead to a fatal outcome in the absence of surgery in the near future: profuse bleeding) and perforation. Absolute indications include conditions in which the ulcer can only be removed surgically. Relative indications are conditions that, if conservative treatment is ineffective, can be eliminated surgically or a combination of complicated peptic ulcer disease with accompanying abdominal surgical pathology (cholelithiasis, hiatal hernia, mechanical duodenostasis).

**The indications for operation:**

The vital indications:

- profuse bleeding (ineffectiveness of medical or endoscopic hemostasis),
- perforation.

The absolute indications:

- gastric ulcer with severe epithelial dysplasy

– decompensative organic outlet obstruction

**Relative indications:** intractability or nonhealing (negative compliance).

The first surgical intervention that was proposed for the surgical treatment of peptic ulcer disease was gastrojejunostomy (Fig. 3).

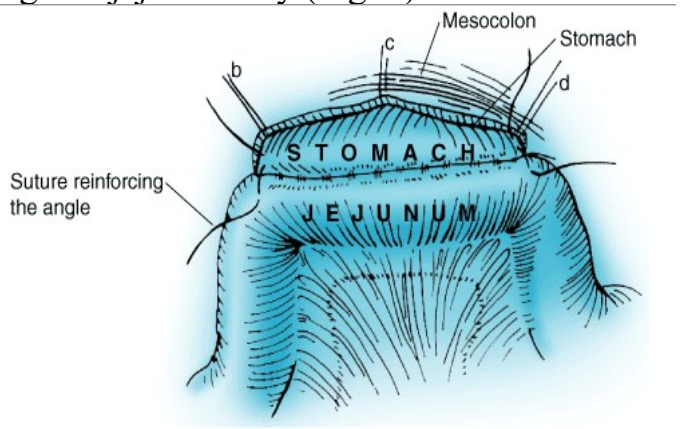



**Fig. 3. Retrocolic gastrojejunostomy**

The oncological mood caused the introduction of gastric resection. Among a number of modifications of this operation, resections according to Billroth are preferred. Two modifications of this operation are known: during resection according to Billroth 1, a gastroduodenal anastomosis is imposed (Fig. 4).

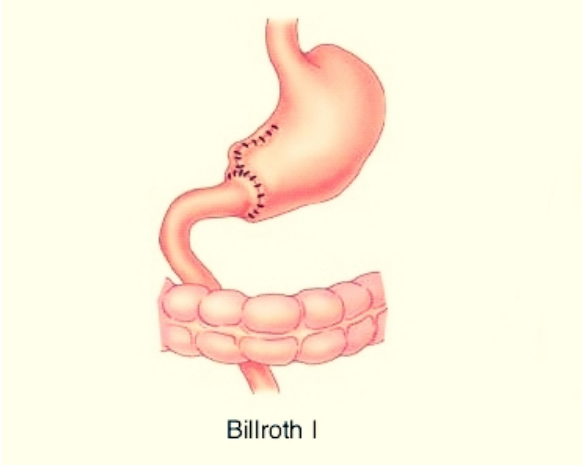



**Fig. 4. Billroth's operation I**

In the case of gastric resection according to Billroth 2, the reconstruction was performed by applying a gastroenteroanastomosis (Fig. 5).

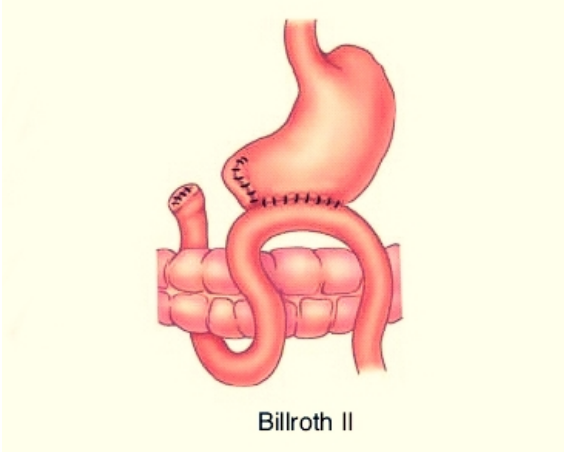



**Fig. 5. Billroth's operation II**

This surgical intervention was performed for both gastric and duodenal ulcers. A large number of post-gastroresection complications, which were accompanied by classic gastric resection, as well as the loss of a functioning stomach in patients with duodenal ulcer, prompted the development of an organ-preserving surgical intervention - vagotomy. The essence of the operation was parasympathetic denervation of the stomach, which led to a decrease in acid production. Depending on the level of crossing the vagus nerve, three modifications of vagotomy were distinguished: 1) trunk (subdiaphragmatic) vagotomy caused denervation of other organs of the abdominal cavity in addition to the stomach; 2) selective vagotomy, in which the vagus nerve was cut at the level of the stomach; 3) selective proximal vagotomy (HSV) included denervation of the body and the bottom of the stomach, that is, the area where acid-producing parietal cells are located (Fig. 6)

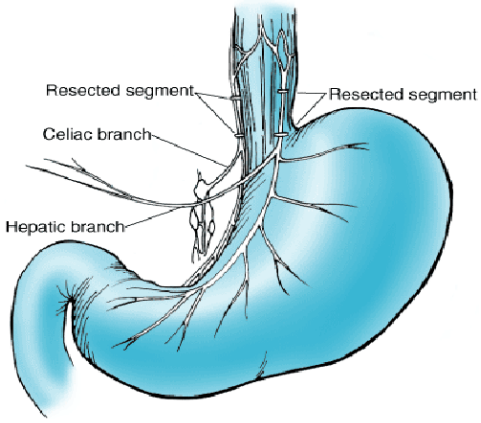



**Fig. 6. Schematic representation of various variants of vagotomy**

The introduction into medical practice of antisecretory drugs - H2 histamine receptors inhibitors, and a little later - proton pump inhibitors (PPI), narrowed the indications for the use of vagotomy. This was facilitated by the accumulation of data on the consequences of vagotomy, including its most physiological variant – HSV. Ulcer recurrence after various types of vagotomy reached 60%, which exceeded the negative consequences after the use of gastrojejunostomy (recurrence - 50%). This is due to the fact that during vagotomy, only the first (vagal) of the three phases of gastric secretion is eliminated. A few years after this operation, hypersecretion of hydrochloric acid occurs compensatingly due to the humoral and intestinal phases. At the same time, PPIs affect all three phases of gastric secretion, which allows them to be widely used for the treatment of acid-dependent conditions, and the presence of forms for parenteral administration - during surgery and in the early postoperative period. According to the basic principles of treatment of complicated duodenal ulcer, in addition to suppressing gastric secretion, the ulcer substrate must be removed. With a long history of ulcers and courses of anti-relapse treatment, the duodenum narrows during scarring of the ulcer, which requires appropriate correction if there are signs of stenosis. These two tasks (removal of the ulcer and restoration of the normal anatomy of the duodenal zone) are performed by surgery – duodenoplasty (Fig. 7).

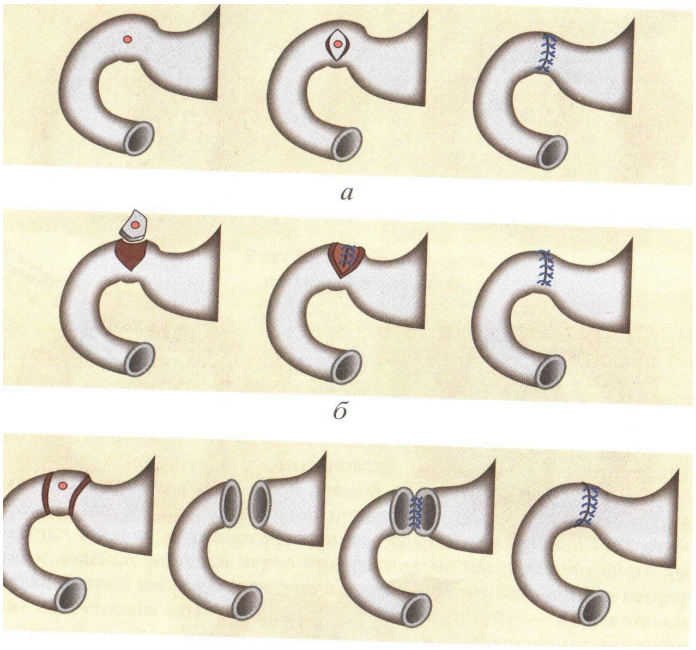
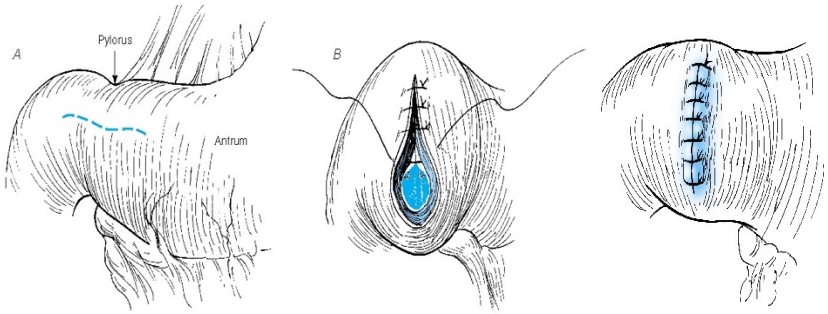



**Fig. 7. Different variants of duodenoplasty: a) typical, b) subcircular, c) circular**

When a duodenal ulcer is located near the pyloric sphincter or directly in its area, pyloroplasty is performed, or as we call it, «gastroduodenoplasty». The disadvantage of this intervention is the destruction of the anterior semicircle of the pylorus, which is why this operation is also called «hemipylorectomy». There are different variants of duodeno-ayo gastroduodenoplasty, but the most common is the Heineke-Mikulicz type, which closes a longitudinal transpyloric incision (A) in a transverse fashion (B,C) (Fig. 8).




a b c

**Fig. 8. Stages of the Heineke-Mikulicz duodeno- or gastroduodenoplasty**

Therefore, the operation of choice for complicated duodenal ulcer is duodenoplasty supplemented with PPIs.

An organ-sparing approach is also used for gastric localization of a complicated ulcer. The classic gastric resection was replaced by an partial gastric resection, during which all the main functions of the stomach (reservoir, evacuation, digestive and general trophic) are preserved (Fig 9).

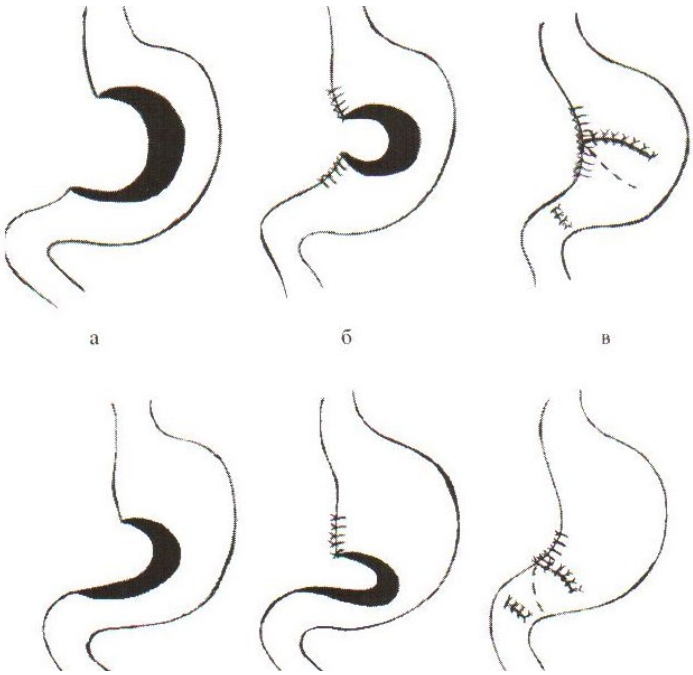



**Fig. 9. Partial gastric resection (the upper row - for ulcers of type 1 (pyloro-preserving), the lower row - for antral ulcers - pyloro-corrective).**

## GASTRIC OUTLET OBSTRUCTION

Gastric outlet obstruction results from fibrous scarring of chronic duodenal ulcer disease. Symptoms develop over a long period of time, occasionally more acutely due to edema caused by acute exacerbation of ulceration. But, even in the latter circumstance, preexisting scarring and stenosis are likely to be confirmed. Gastric outlet obstruction occurs less frequently than the complications of perforation and bleeding. It is likely that its incidence has decreased because of the advent of potent acid-reducing drugs and identification and eradication of H. pylori.

**Clinical classifications of ulcer stenosis**

- I - compensated;
- II - subcompensated;
- III - decompensated.

Essentials of the management are listed in Table 4.

**Table 4. Essentials: management of Gastric Outlet Obstruction**

Symptoms and signs
- Insidious clinical presentation
- Early satiety antedating vomiting
- Weight loss
- Fatigue

Biochemical goals of treatment
- Correction of hypovolemia and hypochloremic, hypokalemic, metabolic alkalosis

Diagnostic tests
- Barium meal
- Endoscopy

Conservative therapy (8–10 days)
- Continuous gastric decompression
- Suppression of acid secretion
- Nutritional support with parenteral or enteral nutrition (percutaneous feeding jejunostomy)
- Correction of anemia and vitamin K deficiency

Operative therapy
- Assess for "difficult" duodenum
  - If present, perform truncal vagotomy and gastrojejunostomy
  - If absent, perform truncal vagotomy and pyloroplasty
- Ancillary procedures
  - Feeding jejunostomy
  - Tube gastrostomy

**Clinical Presentation**. The symptoms of gastric outlet obstruction are usually insidious and accompanied by a chronic history of duodenal ulcer. The initial symptoms are early satiety, bloating, and halitosis. When vomiting eventually develops, it is usually after the last meal of the day. The vomitus may contain undigested food eaten 24 to 48 hours earlier. As the obstruction becomes more complete, vomiting may occur after any meal. Chronic weight loss, even emaciation, and chronic fatigue develop. Physical examination may show the presence of "succussion splash" (i.e., a splashing sound in the epigastrium when the patient is shaken from side to side). Infrequently, particularly in the emaciated patient, gastric peristalsis may be visible in the epigastrium.

**Diagnostic program**

- Complaints of patient and anamnesis of disease.
- Sounding of stomach and examination of gastric content.
- Gastroduodenoscopy, biopsy.
- Intragastric pH-metry.
- Study of motility of stomach.

- Roentgenologic examination of stomach and duodenum (structural features, passage).
- Sonography

**Investigation** A barium meal confirms gastric outlet obstruction by showing a dilated stomach and a small amount of barium entering the duodenum.

**Roentgenological stages of stenosis:**

- the compensation stage: evacuation of contents takes no more than 6 hours.
- the stage of subcompensation: evacuation take up to 24 hours.
- decompensation the stomach: the contrast stays for long to more than 24-48 hours.

The use of upper GI endoscopy is necessary to exclude antral cancer as the cause. The endoscope cannot be passed into the duodenum, and no gastric pathology may be found. Antral biopsy for histology and H. pylori studies should be obtained. The typical biochemical findings when prolonged vomiting is present are of hypochloremic, hypokalemic, and metabolic alkalosis. Vomiting results in loss of fluid, chlorides, and H+. Severe dehydration develops, and the kidneys attempt to compensate by retaining Na+. To accomplish this, potassium is initially exchanged, but as dehydration progresses and potassium stores become depleted, H+ is exchanged for Na+ in the renal tubules. Early in the evolution of biochemical derangements caused by gastric outlet obstruction, the urine is alkaline; however, paradoxic aciduria soon develops as H+ is lost in the urine, even as systemic metabolic alkalosis is developing. An electrocardiogram may show the typical peaked T-waves of hypokalemia. Starvation leads to hypoproteinemia and potential vitamin K deficiency. Therefore, nutritional status and coagulation factors need to be assessed.

**Treatment** of stenosis depends on its stage

*Compensated stenosis* is treated with conservative means (PPI, antihelicobacter therapy, gastrokinetics).

In *the subcompensated stage*, in addition to conservative treatment, endoscopic balloon pneumodilation is used (Fig. 10).

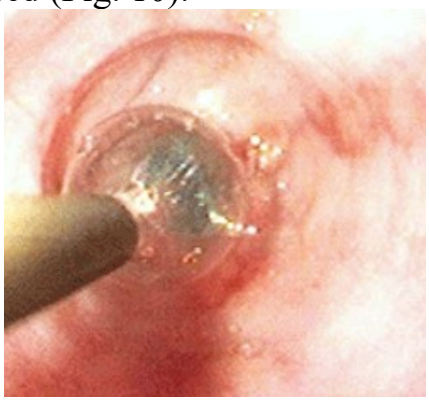


**Fig. 10. Endoscopic balloon pneumodilation**

At *the stage of decompensation* of stenosis, organ-sparing surgery is indicated.

In case of severe hypovolemic and electrolyte disturbances in decompensated stenosis due to constant vomiting, before the operation, the patient is given a course of short-term (3-5 days) infusion therapy (preferably in the intensive care unit under constant laboratory monitoring).

List of main treatment measures

- nasogastric suction (twice daily decompression and washing of stomach)
- intravenous hydration (transfusion of fluids up to 2,5-3 l per day (amino acid and glucose; plasma, albumen),
- electrolyte repletion (the ions K+, Na+, Ca++, Cl-),
- antisecretory medication (PPI).

The operation of choice for duodenal stenosis is duodenoplasty with PPI (Fig. 11).

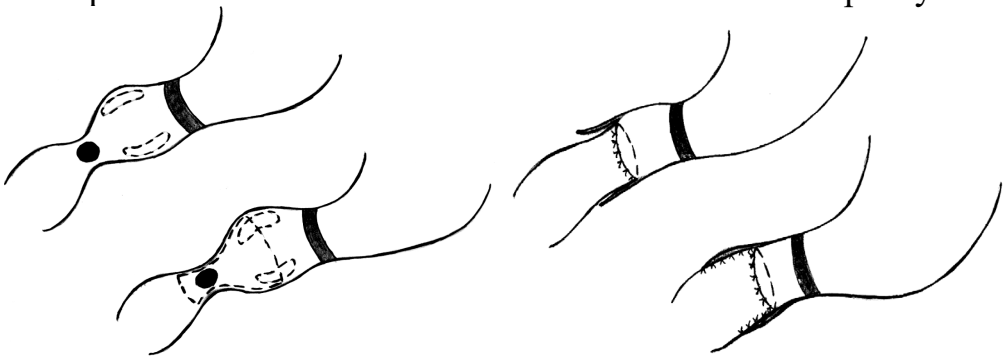


**Fig. 11. Stages of duodenoplasty (own development)**

We consider gastric resection for pyloroduodenal stenosis to be a harmful intervention. Gastroduodenoplasty with PPI is performed for pyloric stenosis.

**GASTRIC MALIGNANCY**

**Premalignant Conditions**

Information regarding premalignant conditions and other factors of gastric malignancy is summarized in Table 5.

**TABLE 5. Essentials: Gastric Malignancy**

Premalignant conditions
- *H. pylori* infection
- Atrophic gastritis and pernicious anemia
- Gastric polyps
- Gastric ulcer
- Hypergastrinemia
- Blood group A
- Previous gastric resection
- Ménétrier's disease

Carcinoma of the stomach
- Falling incidence
- Gross appearance: Polypoid, ulcerative, colloid, or infiltrative
- Surgical treatment: Bilroth II or total gastrectomy
- Early gastric cancer
  - No invasion of muscularis
  - 10% of gastric cancers in U.S.
  - 5-year survival of 70%–95%
- Advanced gastric cancer
  - Invasion of muscularis and/or lymph node metastasis
  - 80% of cases in U.S.

Gastric carcinoid tumors
- Classification
  - Type I: Associated with atrophic gastritis
  - Type II: Associated with MEN-I syndrome
  - Type III: Sporadic; most are malignant and metastasize to liver
- Treatment
  - Tumors <2 cm: Endoscopic excision
  - Tumors >2 cm: Resection with 1-cm margin

Gastric lymphoma
- Non-Hodgkin's lymphoma of B-cell type
- Significant association with MALT and *H. pylori* infection
- 40% present with bleeding, perforation or obstruction
- Cure rate of 65%–75% after curative resection and neoadjuvant therapy
- Treatment
  - Responsive to chemotherapy and radiotherapy
  - When confined to stomach: Curative resection followed by adjuvant chemo- or radiotherapy

*Abbreviations*: MALT, mucosa-associated lymphoid tissue; MEN-1, multiple endocrine neoplasia-1.

***Helicobacter pylori.*** Patients with H. pylori infection have a six- to nine-fold increased risk of gastric cancer. The pathogenesis is thought to proceed from gastritis to dysplasia to cancer. The incidence of mucosa-associated lymphoid malignancy is higher than adenocarcinoma.

***Atrophic Gastritis and Pernicious Anemia.*** The risk of developing adenocarcinoma is increased nearly six-fold in patients with atrophic gastritis and pernicious anemia. In a longitudinal prospective study,1 in 80 patients with pernicious anemia developed cancer. The achlorhydria that accompanies this condition favors bacterial proliferation, which generates carcinogenic nitrosamines from nitrates in food.

***Gastric Polyps.*** Adenomatous polyps, which represent about 10% of all gastric polyps, are significant risk for cancer. The cancer risk in small adenomatous polyps (<2cm) is 2%, but the risk rises to 24% in polyps 2cm or larger.

**Gastric Ulcer.** The incidence of malignant degeneration of a benign gastric ulcer is probably no higher than 1%. On the other hand, malignant lesions can masquerade as benign ulcers more frequently. Ulcer's malignization – characterized by permanent pain unrelated to food intake, anorexia, constant weight loss, permanent wasting, disappearance of localized pain, and installment of diffuse epigastric pain.

Regarding the true malignancy of a gastric ulcer or the presence of dysregenerative changes in the surrounding mucosa, the presence of severe epithelial dysplasia, especially with foci of metaplastic epithelium of the colonic type, indicates the presence of changes that can be considered "cancer in city" (Fig. 12).

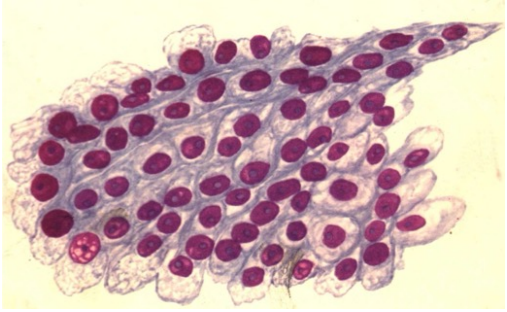


**Fig. 12. Epithelial dysplasia**

***Hypergastrinemia*** can be caused by gastrinoma, by prolonged achlorhydria that occurs as a result of atrophic gastritis, and by long-term therapy with proton-pump inhibitors. Hypergastrinemia results in hyperplasia of the ECL cells and a tendency to cause carcinoid tumors. Gastric carcinoids occur more frequently in patients with atrophic gastritis and the Zollinger-Ellison syndrome. Long-term therapy with proton-pump inhibitor has caused carcinoid tumors in mice, but there has been no reported incidence of carcinoid tumors in humans on long-term therapy.

**PENETRATION**

Penetration of the ulcer means the germination of the ulcer into neighboring organs and anatomical structures. The frequency of this complication does not exceed 20% among other complications of peptic ulcer. Most often, a duodenal ulcer penetrates into the pancreas, and a gastric ulcer penetrates into the small omentum. Penetration of an ulcer into a hollow organ leads to the formation of a fistula (for example, with a gall bladder).

Features of the clinical symptoms of a penetrating gastroduodenal ulcer penetrating the pancreas:

- back pain is added to pain in the epigastric region;
- the rhythm of the pain is overshadowed - with a duodenal ulcer, the pain characteristic of pancreatitis is added to the night and hunger pain, which is relieved by taking antispasmodics and enzyme preparations, not PPIs.

Laboratory tests show changes characteristic of pancreatitis (increased blood amylase, urine diastase). Upper endoscopy (deep ulcer on the back wall of the duodenal bulb), ultrasound, CT scan help clarify the diagnosis.

As a rule, conservative therapy is effective for this complication, however, in cases where the symptoms increase, the ulcer does not heal, and especially when complications such as bleeding or perforation are added (as a rule, another ulcer of the front wall of the duodenum (kissing ulcers), duodenoplasty with extraterritorialization of ulcer (Fig. 13) for duodenal ulcer and economy resection for gastric ulcer operation is indicated.

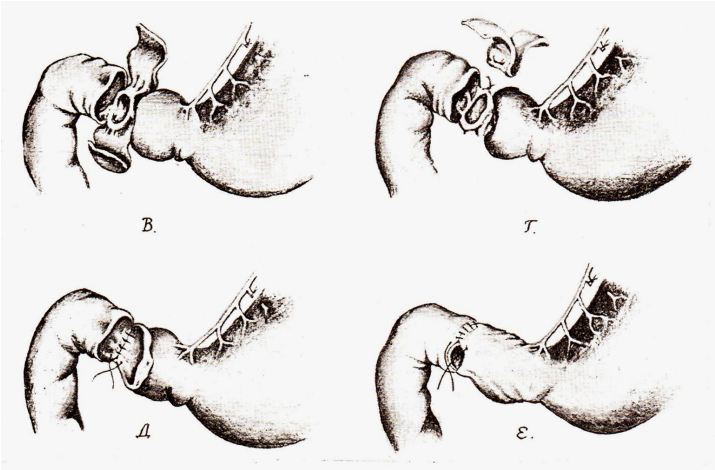



**Fig. 13. Stages of duodenoplasty with extraterritorialization of ulcer**

Since the excision of the ulcer, which penetrate into the pancreas, is impossible due to the development of profuse bleeding, the ulcer of the posterior duodenal wall is removed outside the duodenum, after which a circular duodenoplasty is performed. The ulcer, removed outside the duodenal contour in the absence of peptic factor, is reduced, being replaced by connective tissue.

**PERFORATION**

By localization, a perforated stomach ulcer (23%) and duodenum (77%) are distinguished.

There are three types of perforation:

- perforation in the abdominal cavity (88%);
- covered perforation (8%);
- atypical perforation (4%).

The vast majority of patients have *perforation into the free abdominal cavity or typical perforation*, which has a characteristic clinical picture. As with any acute abdominal pathology, a typical perforated ulcer goes through three stages (phases): 1) the phase of painful shock (duration - 24 hours), 2) transitory phase or imaginary well-being (duration - up to 72 hours), 3) phase purulent peritonitis (duration - more than 72

hours). The phase of pain shock is described with the help of the Mondor triad: 1) Dielafois symptom - sharp pain in the epigastric area (Dielafois compares it to pain due to a stab with a dagger) (95%); 2) palpation of the abdomen reveals the protective tension of the muscles ("wooden belly") - Cruvelier symptom (90%), 3) ulcer anamnesis (85%). Pain may be somewhat altered or absent in patients who systematically use painkillers, weakened patients (oncological, cachectic), as well as in a state of drug and alcohol intoxication. The protective tension of the abdominal muscles may also be absent in these categories of patients, as well as in elderly patients. A separate category of patients is morbidly obese, but muscle defense cannot be determined due to excessively developed adipose tissue. Some patients may not have an ulcer anamnesis, as the clinical picture of perforation is the first manifestation of the disease. For a typical perforation, percussing the abdomen in a horizontal position on the back (projection of the liver) reveals the absence of hepatic dullness due to the presence of free air (Clark's symptom).

*Covered perforation* was first described by A. Shnikler in 1912. Its mechanism is that during the entry of stomach contents and air into the abdominal cavity a perforating hole is covered with fibrin, omentum, or sometimes - food particles. Depending on the amount of contents that got into the abdominal cavity, muscle tension of the anterior abdominal wall and hepatic dullness may be absent. In patients with a covered perforation of the ulcer, not general, but local muscle tension (Ratner-Vicker's symptom) at the place of exudate accumulation is determined.

With *atypical perforation* of the back wall of the duodenum, the contents do not enter the abdominal cavity, and the retroperitoneal space (ulcers of the back wall of the duodenum), and with perforation of the back wall of the stomach - into the bursa omentalis.

**Diagnostics.**

To clarify the diagnosis, the patient undergoes an upper endoscopy, during which it is possible to visualize an ulcer with perforation. High informativeness is demonstrated by the inspection X-ray of the organs of the abdominal cavity, during which under diaphragm (1) air (2) in the abdominal cavity (pneumoperitoneum) is detected (Fig. 14). If it is difficult for the patient to be in an upright position (a), the X-ray examination can be performed in a horizontal position on the left side (lateroposition)(b,2).

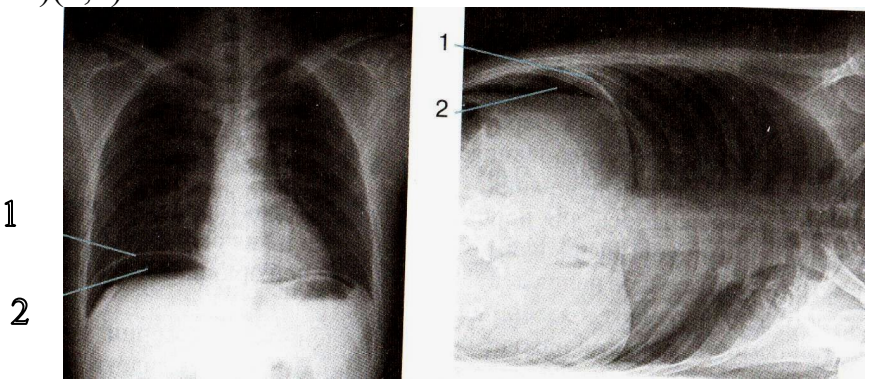
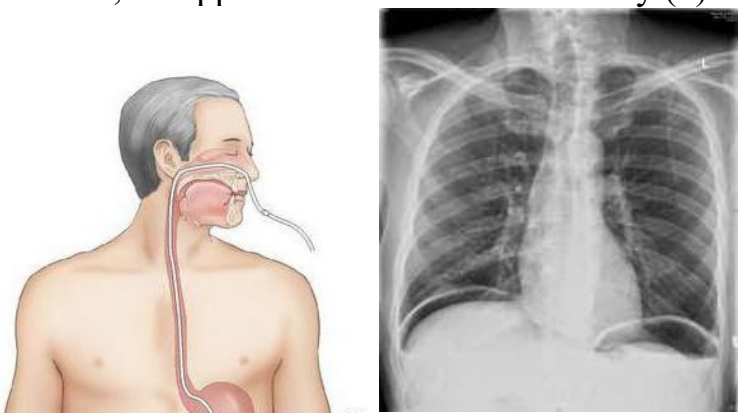



**Fig. 14. The phenomenon of pneumoperitoneum in a vertical and horizontal position**

As mentioned above, in the case of covered perforation of the ulcer, the amount of air that has entered the abdominal cavity may not be enough to be reflected on the X-ray. For this purpose, the technique of pneumogastrography (Fig. 15) is performed, which consists in introducing 500-1000 ml of air into the stomach through a nasogastric tube (a) using a Jeanette syringe (100 ml), after which the X-ray examination is repeated. At the same time, air appears in the abdominal cavity (b).



a b

**Fig. 15. The technique of of pneumogastrography**

Preliminary endoscopy can also be used as an option for pneumogastroscopy. In a number of clinics, the technique of "double contrast" is used, when water-soluble contrast in a volume of 40-60 ml is injected parallel to air through a probe.

During an ultrasound examination of the abdominal cavity, free fluid is determined (Fig. 16).

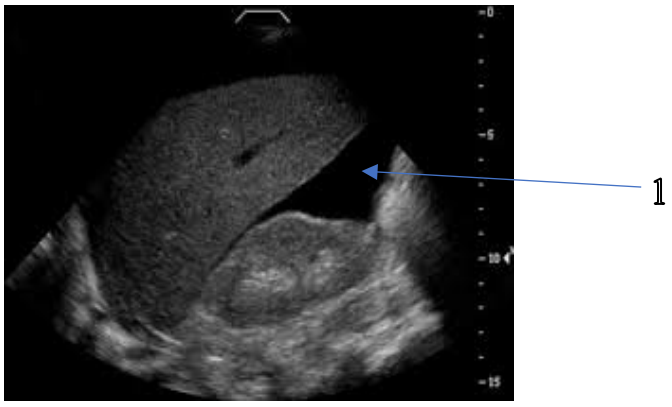



**Fig. 16. Signs of free fluid in the abdominal cavity (ultrasonography)**

Laparocentesis and laparoscopy should be included in the invasive methods of diagnosing a perforated ulcer. Laparocentesis is performed under local anesthesia using a trocar (Fig. 17).

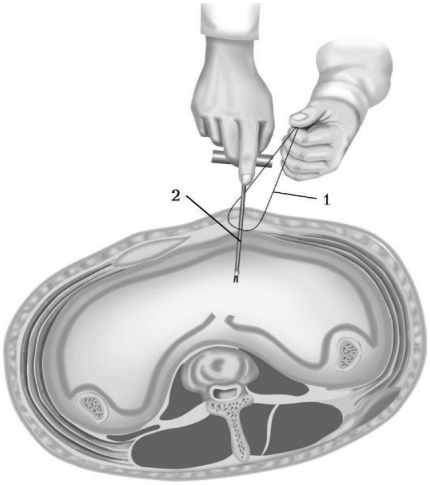



**Fig. 17. Laparocentesis (1) a ligature that tightens the anterior abdominal wall, 2) trocar)**

During laparocentesis, when obtaining abdominal effusion, it is not always possible to verify its nature. For this, the Neimark test is used, which consists in adding a 10% solution of iodine to the contents - the particles of the food that is in the contents under the influence of hydrochloric acid is broken down into starch, which is colored blue by iodine.

We also developed a technique for assessing peritoneal fluid, which consisted in adding a 50% ethanol solution to it (Fig. 18). The formation of a gel indicated the presence of exudate of the abdominal cavity (as a result of alcohol polymerization of exudate fibrin monomer). In the presence of transudate, the gel is not formed (sensitivity of the method was 95.8%, specificity - 83.3%).

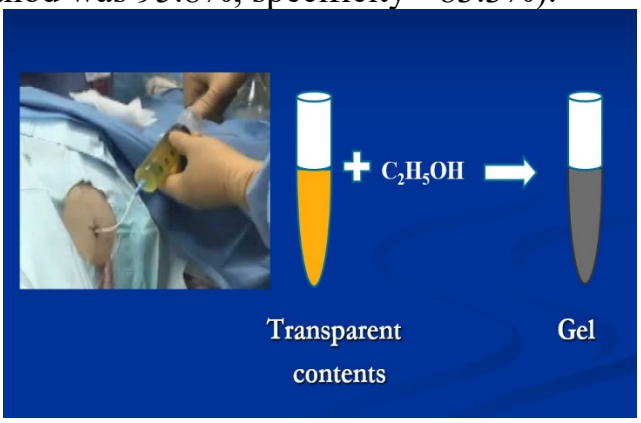



**Fig. 18. Ethanol test**

Laparoscopy (Fig. 19) in case of suspicion of a perforated ulcer allows to assess the nature of the contents of the abdominal cavity followed by its aspiration and drainage, to carry out an audit of the internal organs.

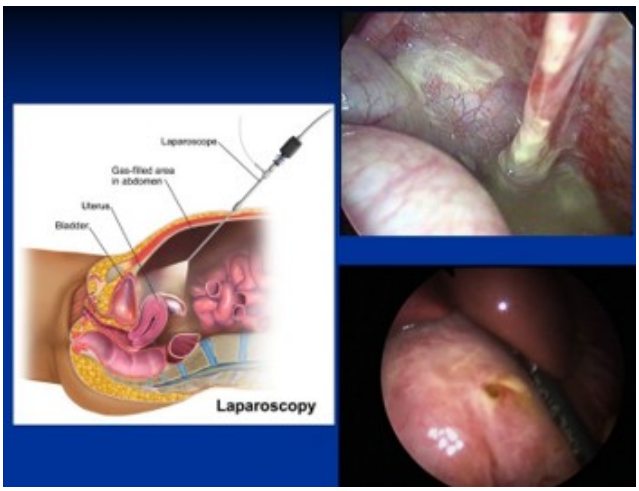



**Fig. 19. Laparoscopy**

**Treatment.**

The operation of choice for duodenal ulcer complicated by perforation is duodenoplasty supplemented with PPI, and for gastric ulcer - partial gastrectomy. In extremely severe patients, it is possible to perform a simple suturing of the perforating hole. However, this surgical intervention is accompanied by a high frequency of ulcer recurrence even with concomitant anti-relapse therapy (up to 40%), since the morphological substrate of the ulcer with a periulcerous infiltrate remains, there is no possibility of revision of the back wall of the duodenum and its decompression, it is impossible to restore the normal patency of the duodenum.

Taking into account the expansion of the possibilities of introducing laparoscopic interventions into urgent surgery, in the surgical treatment of a perforated ulcer, laparoscopic suturing of the perforated hole and endoplasty are used. Indications for laparoscopic suturing of a perforated ulcer after a mandatory preliminary endoscopy are an isolated ulcer without signs of bleeding and stenosis and a time from the moment to 8 hours.

It is also known about the method of conservative treatment of Taylor's perforated ulcer in patients in an extremely serious condition (acute myocardial infarction, acute cerebrovascular accident, comatose state, burns, etc.); for which general anesthesia is contraindicated, in case of long-term concealed perforation (more than 72 hours) in the absence of signs of spilled peritonitis, as well as in the case of the patient's categorical refusal of surgical treatment. The technique consists in constant aspiration of gastric contents, intravenous administration of PPI (80 mg), at least 3 broad-spectrum antibiotics, infusion therapy and x-ray or US examination of the abdominal cavity.

**DISEASES OF THE OPERATED STOMACH**

**Long-Term Sequelae of Ulcer Surgery** With the exception of proximal gastric vagotomy (PGV), any ulcer surgery may be associated with undesirable long-term sequelae. These and their pathophysiologic bases are summarized in Table 6.

**TABLE 6. Pathophysiologic Basis of Long-Term Complications of Ulcer Surgery**

| *Complication* | *Pathophysiologic basis* |
|---|---|
| Recurrent ulcer | Incomplete vagotomy<br>Inadequate gastric resection<br>Zollinger-Ellison syndrome<br>Retained antrum syndrome |
| Postvagotomy diarrhea | Unknown |
| Dumping syndrome | Rapid gastric emptying<br>Fluid shift into intestine, causing hypovolemia<br>Release of vasoactive peptides and amines (VIP, neurotensin, bradykinin, 5-HT) |
| Reactive hypoglycemia | Excessive release of insulinotropic peptides from the gut (GLI, GIP)<br>Secondary hyperinsulinemia |
| Gastroparesis | Unknown |
| Bile gastritis | Duodeno-jejuno-gastric bile reflux<br>Bile-induced mucosal injury |
| Iron-deficiency anemia | Decreased absorption of dietary iron<br>Chronic occult blood loss |
| Megaloblastic anemia | Decreased intrinsic factor secretion following radical subtotal or total gastrectomy |
| Malabsorption | Steatorrhea<br>Rapid intestinal transit<br>Blind-loop syndrome |
| Osteoporosis | Calcium malabsorption |
| Postgastrectomy carcinoma | Hypochlorhydria or achlorhydria |

*Abbreviations*: 5-HT, 5-hydroxytryptamine; GIP, gastric inhibitory peptide; GLI, glucagon-like immunoactivity; VIP, vasoactive intestinal peptide.

The operation least likely to cause undesirable side effects, PGV, is associated with the highest incidence of ulcer recurrence. On the other hand, the operation most likely to cure the ulcer problem, V & A, can lead to long-term complications.

**Ulcer Recurrence** The causes of ulcer recurrence are incompleteness of vagotomy, inadequacy of gastric resection, or both. Occasionally, however, ulcer recurrence is due to an undiagnosed gastrinoma. Recurrence following distal gastrectomy for gastric ulcer is uncommon.

***Classification of diseases of the operated stomach (by O.O. Shalimov and V.F. Saenko, 1987)***

*A. Postgastrectomy syndromes.*

I. Functional disturbance.

- Dumping syndrome.
- Hypoglycemic syndrome.
- Postgastrectomy (agastric) asthenia.
- Syndrome of small stomach.
- Syndrome of afferent loop (functional origin).
- Gastroesophageal reflux.
- Alkaline reflux-gastritis.

II. Organic disturbances.

- Peptic ulcer of anastomosis.
- Gastro-colic fistula.
- Syndrome of afferent loop (mechanical variant).
- Cicatricial deformation and narrowing of anastomosis.
- Mistakes in the technique of operation.
- Postgastrectomy accompanying diseases (pancreatitis, enterocolitis, hepatitis).

III. Mixed disturbances (combination with dumping or postvagotomy diarrhea).

*B. Postvagotomy syndromes.*

- Relapse of ulcer.
- Diarrhea.
- Disturbance of function of esophagocardial transition.
- Disturbance of emptying of stomach.
- Dumping syndrome.
- Reflux-gastritis.
- Gallstone disease.

The incidence of recurrent ulcer after surgery for duodenal ulcer is higher than other rates of recurrence and depends on the type of operative procedure used to treat the primary ulcer (Table 7).

**TABLE 7. Essentials: Recurrent Ulcer Following Surgical Therapy**

Causes
- Incomplete vagotomy
- Inadequate gastric resection
- Gastrinoma
- Uncommon following distal gastrectomy for gastric ulcer

Diagnosis
- Best established by endoscopy
- Assess for *H. pylori* infection and initiate eradication therapy if present
- Evaluate for completeness of vagotomy

Surgical treatment
- Necessary if medical treatment fails
- Choice of procedure depends on type of initial operation:
  - Antrectomy if PGV was initially performed
  - Re-vagotomy with or without antrectomy if truncal vagotomy was initially performed
  - Re-vagotomy with or without re-resection if V & A was initially performed

*Abbreviations*: PGV, proximal gastric vagotomy; V & A, vagotomy and antrectomy.

Following PGV and truncal vagotomy and pyloroplasty (V & P), the site of ulcer recurrence is usually the duodenum, although it may also be the stomach. Following truncal vagotomy and gastrojejunostomy (V & GJ), ulcer recurrence is nearly always in the jejunum, next to the stoma; hence, the names stomal and marginal ulcers. The evaluation of ulcer recurrence includes endoscopy, measurement of plasma gastrin levels, assessment of completeness of vagotomy, and tests for presence of H. pylori.

*Diagnosis with Upper Gastrointestinal Endoscopy*. Barium meal studies are usually not helpful in the diagnosis of recurrent ulcer. Upper GI endoscopy is the only reliable method of diagnosis.

*Diagnosis with Plasma Gastrin Levels.* Following all types of vagotomy not associated with antral resection, both basal and postprandial hypergastrinemia develop. Thus, elevated plasma gastrin levels following vagotomy must be interpreted with caution. If there is concern that a gastrinoma may be present, a "secretin test" should be performed to detect a paradoxical rise in plasma gastrin level following intravenous injection of secretin. If hypergastrinemia is demonstrated in a patient who has had antrectomy, either a gastrinoma or retained antrum syndrome is present. The latter syndrome develops after Bilroth II gastrectomy, when antral tissue is left in continuity with the duodenum. Chronic exposure of this tissue to alkaline secretion leads to G-cell hyperplasia and hypergastrinemia. Again, the secretin test is needed to rule out the diagnosis of gastrinoma.

*Assessment of Completeness of Vagotomy* Although a high basal acid output (>5mEq/h) is suggestive of an incomplete vagotomy, the sham feeding ("chew and spit") test is more definitive. It evaluates the cephalic phase of acid secretion.

*Diagnosis with Helicobacter pylori* Testing Evaluation for the presence of H. pylori may be accomplished with endoscopic biopsy, breath test, or serology. If H. pylori infection is present, eradication therapy is needed.

*Treatment* If infection with the bacterium H. pylori is diagnosed, eradication therapy should be started with the objective of effecting a permanent cure for the ulcer. Combination therapy with amoxicillin or with tetracycline, metronidazole and omeprazole is effective. A histamine H2-receptor antagonist or proton-pump inhibitor provides symptomatic relief but is unlikely to cure the recurrence of the ulcer. The type of operation needed if medical therapy fails depends on the primary operation that was performed.

**Postvagotomy diarrhea.** The incidence of incapacitating diarrhea following truncal vagotomy is 1% to 2%. The cause is unknown. Symptomatic treatment includes avoidance of certain foods and the use of bulk-forming agents (Kaopectate), codeine, and Lomotil. Postvagotomy diarrhea has no satisfactory treatment and is best avoided by performing PGV rather than truncal vagotomy as the primary procedure of choice. Surgical therapy for postvagotomy diarrhea is a last resort. If pyloroplasty was previously performed, pyloric sphincter reconstruction, which reverses the pyloroplasty, has had some success. If a gastrojejunostomy was performed, it can be taken down. The most controversial procedure is the interposition of a 6-inch segment of reversed jejunum between the stomach and duodenum or jejunum to slow intestinal transit. The reported results are not very encouraging and the procedure is rarely, if ever, recommended. Hence, the best form of treatment is prevention.

**Dumping Syndrome** Rapid entry of hyperosmolar chyme into the intestine as a result of destruction, resection, or bypass of the pyloric sphincter is the main cause of this side effect. Vagotomy, which interferes with gastric accommodation, contributes to rapid gastric emptying. Within 15 to 30 minutes of a meal, the patient experiences epigastric distress, sweating, flushing, and profound fatigue. Exaggerated bowel sounds (borborygmi) and sudden diarrhea may also be experienced. As described earlier, the underlying cause of the syndrome is the combination of fluid shift into the intestine, which causes hypovolemia, and the release of vasoactive substances from the intestine.

The best way to prevent dumping syndrome is to avoid, whenever possible, performing operations that are likely to cause it, including gastrectomy and truncal vagotomy and drainage. Pharmacologic and bacteriologic advances have nearly eliminated the need for elective ulcer surgery. In an emergency situation, the surgeon must decide whether to perform the quickest and safest operation at that moment as opposed to a lengthier operation with less undesirable side effects. Whenever the condition of the patient allows, particularly in young patients and women, PGV is a better choice than truncal vagotomy and drainage. In the setting of hemorrhage, control of bleeding is accomplished through duodenotomy, leaving the pyloric sphincter intact. When perforation is the indication for emergent surgery, PGV is again preferred if an acid-reducing procedure is to be done.

*Nonsurgical Management* Dietary measures often effectively control dumping syndrome. These include avoiding a high carbohydrate diet; eating small, frequent meals; not ingesting fluids with the meals; and lying down for about 60 minutes after

eating. Patients with severe symptoms have been successfully treated with the long-acting somatostatin analogue octreotide. The problem with this form of treatment is cost and the long-term need for injection therapy.

*Surgical Management* As always, surgical treatment for the dumping syndrome is a last resort. Some operative approaches are simple and have a chance to succeed. These include pyloric sphincter reconstruction when a pyloroplasty is present, or takedown of gastrojejunostomy when the stomach is otherwise intact. Other surgical options are more complex and should be undertaken only in extreme cases. These include conversion of Bilroth II gastrectomy to Bilroth I, and interposition of jejunum between the stomach and the duodenum. The latter procedures have had variable success.

**Reactive Hypoglycemia** Patients may develop typical signs and symptoms of hypoglycemia 90 to 120 minutes after a meal. In extreme cases, hypoglycemic crisis may develop. This side effect used to be called the “late dumping syndrome.”The pathophysiologic basis appears to be rapid absorption of glucose from the intestine, which leads to excessive secretion of insulin due to release of the insulinotropic peptide glucagon-like immunoactivity (GLI),which outlasts the hyperglycemic stimulus. Avoidance of carbohydrates in the diet is helpful. The long-acting somatostatin analogue octreotide is effective in controlling severe symptoms.

**Gastroparesis** A small percentage of patients may develop gastroparesis following vagotomy and/or gastric resection. The cause is unknown. Symptomatic therapy with prokinetic agents (e.g.,dopamine antagonists,cisapride) may be helpful. Some patients require repeated gastric resections, eventually necessitating total gastrectomy with Roux-en-Y esophagojejunostomy. In some patients with disabling symptoms, total gastrectomy is the only definitive and successful treatment.

**Bile Gastritis** Regurgitation of bile into the stomach invariably occurs when the pylorus is destroyed, resected, or bypassed. Some patients develop epigastric pain and bilious vomiting presumably due to the resultant gastritis. Medical therapy includes bile salt antagonists and prokinetic agents. Bile reflux can be prevented or minimized by inserting a 60-cm Roux-en-Y jejunal limb between the stomach and upper jejunum. Unfortunately, the early encouraging results of this operation have not been sustained over time.

**Chronic Anemia** Iron-deficiency anemia commonly occurs several years after gastrectomy, but it can also develop following truncal vagotomy. The causes may include chronic occult blood loss from gastritis and poor absorption of dietary iron. Megaloblastic anemia, due to vitamin B12 deficiency, may be seen after radical gastrectomy, indicating insufficient secretion of intrinsic factor. It can be successfully treated with monthly vitamin B12 administration parenterally.

**Malabsorption** Postgastrectomy patients often undergo weight loss and sometimes show signs of malabsorption of fat, carbohydrates, vitamins, and metals. Mild steatorrhea tends to occur after Bilroth II gastrectomy. Vitamin deficiencies may be related to blind-loop syndrome. Lactose intolerance is unmasked in patients who have a mild preoperative lactase deficiency. A significant long-term complication of gastric surgery is calcium malabsorption, which over years may lead to osteoporosis, particularly in women.

**Postgastrectomy carcinoma** A higher incidence of carcinoma of the stomach is seen in patients who had gastrectomy 20 years or more previously. The cause is unknown but may be related to hypoacidity favoring bacterial overgrowth and a generation of carcinogenic nitrosamines from food.

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**Tests**

1. A 30-year-old man with a duodenal ulcer is being considered for surgery. His serum gastrin level, however, is 150±10 pg/mL on three determinations. The surgeon should perform
   A. An arteriogram
   B. A secretin stimulation test
   C. A total gastrectomy
   D. A subtotal gastrectomy
   E. A highly selective vagotomy
2. Which of the following statements regarding stress ulceration is true?
   A. It is true ulceration, extending into and through the muscularis mucosa
   B. It classically involves the antrum
   C. Increased secretion of gastric acid has been shown to play a causative role
   D. It frequently involves multiple sites
   E. It is seen following shock or sepsis, but for some unknown reason does not occur following major surgery, trauma, or burns
3. A 72-year-old patient with an intractable type I ulcer along the incisura with a significant amount of scarring along the entire length of the lesser curvature. Select the appropriate surgical procedure for each patient.
   A. Vagotomy and antrectomy
   B. Antrectomy alone
   C. Vagotomy and pyloroplasty
   D. Vagotomy and gastrojejunostomy
   E. Proximal gastric vagotomy
4. Which vessel is most commonly associated with a posterior duodenal ulcer?
   A. Right gastroepiploic artery
   B. Common hepatic artery
   C. Gastroduodenal artery
   D. Superior mesenteric artery
   E. Middle colic artery
5. The lower esophageal sphincter pressure is increased by
   A. Glucagon
   B. Gastrin
   C. Emptying of the stomach
   D. Chocolate
   E. Acid in the stomach
6. Gastroesophageal reflux is best characterized by which of the following statements?
   A. It is synonymous with hiatal hernia
   B. It results from a higher than normal lower esophageal sphincter pressure

C. It may be associated with increased gastrin production
D. It is diagnosed by manometry and 24-hour monitoring of pH in the lower esophagus
E. It is a relative contraindication to esophagoscopy

7. The typical carcinoma that develops in association with Barrett's esophagus is
   A. Epidermoid
   B. Mucoepidermoid
   C. Small cell
   D. Adenocarcinoma
   E. Squamous cell
8. The blood supply to the stomach and duodenum arises from all of the following arteries EXCEPT
   A. Gastroepiploic artery
   B. Common hepatic artery
   C. Splenic artery
   D. Superior mesenteric artery
   E. Inferior mesenteric artery
9. What substance is secreted by the C cells?
   A. Gastrin
   B. Pepsin
   C. Pepsinogen
   D. Gastric acid
   E. Glucagon
10. 10. Gastric acid production is altered by all of the following hormones or actions EXCEPT
   A. Cholecystokinin
   B. Gastrin
   C. Vagal stimulation
   D. Secretin
   E. Glucagon

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| B | D | B | C | B | D | D | E | A | E |

**Materials on the independent teaching of students**

| Main tasks | Notes(instructions) |
|---|---|
| Repeat:<br>– Topography and stomach innervations<br>– Histological structure of stomach<br>– Physiological functions of stomach | -To represent the methods of diagnostics of diseases of digestive tract<br>-To make the flow diagram of gastric malignancy |
| Study:<br>– Types of duodenoplasty.<br>– Types of gastric resection.<br>– H. pylori and ulcer | -To conduct differential diagnosis with the decompensated stenosis and cancer of gastric outlet<br>-To conduct differential diagnosis of pathologies of the stomach |

| | |
|---|---|
| – Pathogenesis of gastroesophageal reflux disease (GERD) | |

## TOPIC 7. CLOSED AND OPEN ABDOMINAL TRAUMA

**Symptoms of damage to the abdominal cavity and retroperitoneal space. Instrumental methods of diagnosis. Therapeutic and diagnostic tactics. First aid for abdominal injuries. The scope of first aid. Treatment of gunshot and closed abdominal injuries. Wounds and closed injuries of the pelvis and pelvic organs. Classification of pelvic injuries. Symptoms and diagnosis of gunshot fractures of the pelvic bones with and without damage to the pelvic organs. First aid for wounds and closed injuries of the pelvis. Prevention of shock. Treatment of gunshot and closed pelvic injuries.**

**Overview**

Trauma means the change in the structure or function of organs and tissues resulting interaction with physical or chemical agents. Trauma encompasses a wide range of mechanisms of injury. It is the main cause of death in people under the age of 35 years and constitutes up to 20% of surgical patients visiting the hospital.

Globally, injury is the seventh leading cause of death, resulting in 5.8 million deaths in 2000. In the structure of peacetime trauma, blunt abdominal injuries make up 2-3% of all traumatic injuries, and the mortality rate ranges from 10 to 57%. Penetrating abdominal trauma makes up no more than 1-1.5% of the total number of injuries. The mortality rate in this group is 27.6%. However, if we take into account local wars and terrorist attacks, then penetrating abdominal injuries make up more than 50%, and the mortality rate is not less than 58-60%.

**Educational aims:**

- To teach students the diagnosis and basics of surgical treatment of abdominal injuries in their various manifestations.
- To familiarize students with the problem of penetrating and blunt peacetime and wartime abdominal injuries and wounds.
- To study methods of abdominal injury diagnosis and treatment.
- To show the complexity of the problem due to the presence in the abdominal cavity of many vital organs, damage to which leads to the rapid onset of peritonitis, bleeding, and the development of irreversible changes in organs and tissues up to multiple organ failure.
- To find out the types of traumas of abdomen, the clinical features, different variants of manifestation and complications.
- To make a patient examination plan for abdominal trauma.
- To formulate the diagnosis in patients with abdominal trauma and carry differential diagnosis.
- To estimate the workup in patients with suspected abdominal trauma.
- To determine the treatment approach to the abdominal trauma, and indications for surgery.
- To provide postoperative treatment for patients with abdominal trauma.
- To assess the prognosis of treatment of patients with abdominal trauma

**A student must know:**

- Anatomo-physiological information about abdomen cavity organs.

- Classifications of abdomen trauma.
- Mechanisms of injury of abdomen.
- Clinical picture of damages of abdomen.
- Methods of diagnostics of abdomen damages.
- Principles of treatment of abdomen trauma.
- Emergent abdominal exploration.
- Giving first aid for abdomen trauma.
- Features of surgical interventions for the different traumas of abdomen.
- General principles of operative management for abdomen trauma.

**A student must be able to:**

- Collect and estimate the complaints of patient with abdominal trauma, information of anamnesis, to conduct physical research and correctly interpret the results obtained.
- Define the rational volume of laboratory and instrumental methods of research.
- Correctly interpret the results of clinical analyses, peritoneal lavage, ultrasound diagnostics, computer tomography, X-ray.
- Give first aid for traumas of the abdomen.
- Define indications for operation and other methods of treatment of patients.
- Perform pre-operative preparation of patients.
- Conduct post-operative care.

**Terminology.**

| Term | Definition |
|---|---|
| Isolated trauma | traumatic action on organs and tissues within the limits of one anatomic area (thorax, abdomen, skull, extremities) |
| Combined trauma (polytrauma) | traumatic action within the limits of two and more of anatomic areas |
| Combination trauma | combination two or more, different mechanism of action of traumatic factors |
| Blunt damages of the abdomen | the mechanical damages of abdomen, when there is no violation of the skin covering the area |
| Penetrating trauma | is damage of abdomen, when there is violation of the skin, muscles and get into abdominal cavity |
| Hypovolaemic shock | is pathologic condition due to decreased circulating plasma volume |

**Content**

**Mechanisms of injury**

**Blunt trauma**

From a physical point of view in case of motor vehicle collisions and falls from heights victims are injured by fast decelaration. The destructive force of an impact depends on its strength, duration, and place of application. The physical deformities

that result from impact force are known as strains. Strains are divided into those that are tensile (stretching), shearing (opposing forces across an object), or compressive (crushing). When the elasticity (tendency to regain original condition) or viscosity (resistance to change in shape during motion) of a tissue or organ is exceeded by applied strains, disruption results. In biomechanical terminology, disruption occurs at the elastic limit or break point. Disruption causes injuries to the skin (abrasion, contusion, chop, puncture, incision, and laceration), buckling or fracture of bones, and visceral or vascular ruptures.

Other than impact force, factors that determine the magnitude of injury after blunt trauma include gender, impact resistance of body parts, fixation of body parts causing deformation during deceleration, and anatomic protection of body parts. The impact of gender is not clear at this time, but the lighter body skeleton and smaller muscular development of females may be important. In one study the fatality risk in motor vehicle crashes for females aged 15 to 45 years was 25 per cent greater than for males. Bones such as the first and second ribs, sternum, scapula, pelvis, and femur are fractured only when significant impact forces are applied. Therefore, associated injuries should always be suspected when such a fracture occurs. For example, associated injuries to the head, chest, and abdomen occurred in 53, 64, and 33 per cent of patients, respectively, with fracture of the first rib in one review. Fixation of the descending thoracic aorta to the ligamentum arteriosum, the liver to the falciform ligament, and the small bowel to adhesions, the ligament of Trietz, and the retroperitoneal caecum are thought to contribute to injuries occurring in these structures during blunt deceleration trauma. Finally, the close associations of the brain and skull, the right ventricle and the sternal area, and the spleen and ribs 9 to 11 are all related to injuries occurring in these structures.

**Penetrating trauma**

The kinetic energy of stab wounds is low, and death occurs only if a critical organ such as the heart or a major blood vessel is injured. Injuries from missile wounds are caused by a combination of factors including the following: (1) missile (weight or mass, shape, velocity, kinetic energy at impact); (2) medium (drag or resistance of the medium and coefficient of drag); and (3) pattern of flight of missile upon impact (whether yaw, tumbling, precession, or nutation is present). Direct mechanisms of injury from missile wounds or fragments include cutting or laceration and transfer of heat. Indirect mechanisms include longitudinal low-displacement shock or sonic pressure waves and temporary cavitations from transverse high-displacement shear waves. The low-displacement wave does not appear to cause damage to tissues, while the high-displacement wave associated with medium- or high-velocity missiles significantly increases indirect damage by causing cavitations. This is thought to be due to the transfer of kinetic energy causing alternating collapsing and reforming of the cavity after the missile passes. Tissues that are relatively inelastic such as the brain, liver, and spleen are damaged the most by cavitations. Other factors that increase the magnitude of injury include fragmentation of the missile after striking the victim or the creation of secondary missiles such as fragments of teeth or bone.

**Injury to the abdomen**

**Blunt injury**

Obvious indications for an emergency celiotomy in a patient who has suffered blunt abdominal trauma include hypotension in combination with a rigid distended abdomen, peritonitis, or evisceration. As is often the case in medicine, diagnostic problems arise when the physical examination is compromised by one of the following: (1) the patient is not available for productive contact due to brain injury, excessive use of alcohol or psychotropic substances; (2) Impaired sensation due to spinal cord injury (3) in addition to internal organs, surrounding structures (first of all skeleton) are also affected. In some situations, physical examination findings may be ambiguous. In the groups described, additional diagnostic tests are necessary to confirm a diagnosis of intra-abdominal injury. Hypotensive patients with possible intra-abdominal hemorrhage undergo either a diagnostic peritoneal lavage or surgeon-performed ultrasound. An open or closed diagnostic peritoneal tap/lavage is performed through an infraumbilical midline site (supraumbilical if there is pelvic fracture) after the insertion of a nasogastric tube and bladder catheter. The return of 10 to 20 ml of gross blood or of bile, succus entericus, stool, or food material is a positive ‘tap', and immediate celiotomy is indicated. In the hypotensive patient with a grossly negative ‘tap', the value of a subsequent time-consuming lavage with 1000 ml of normal saline solution is questionable.

Diagnostic peritoneal lavage is invasive, has an accuracy of 95 to 98 per cent in detecting intraperitoneal hemorrhage, and has a complication rate of 0.5 to 1 per cent. As an alternative to diagnostic peritoneal lavage, a surgeon-performed ultrasound is a noninvasive and rapidly-performed assessment of the abdomen in the injured patient. It is called FAST (focused assessment for the sonographic examination of the trauma patient). This investigation includes ultrasound of the pericardium which is extended onto the right and left subcostal, and suprapubic areas.

As an alternative to diagnostic peritoneal lavage, a surgeon-performed ultrasound is a noninvasive and rapidly-performed assessment of the abdomen in the injured patient. It is called FAST (focused assessment for the sonographic examination of the trauma patient). This investigation includes ultrasound of the pericardium which is extended onto the right and left subcostal, and suprapubic areas. To complete the “FAST” (focused assessment for the sonographic examination of the trauma patient) in hypotensive (or stable) patients. An anechoic image (fluid or blood) in subphrenic area, Morison's pouch, the splenorenal fossa, or the pouch of Douglas/pararectal areas in a hypotensive patient is essentially 100 per cent accurate in confirming the need for an emergency celiotomy. Hemodynamically-stable patients in whom the physical examination is compromised by any of the previously described factors are first evaluated by a surgeon-performed ultrasound. In the absence of any intraperitoneal fluid on a first and subsequent examination, the need for a follow-up examination of the abdomen with a CT scan is questionable - even in the intoxicated patient. The presence of intraperitoneal fluid on the ultrasound mandates a follow-up abdominal CT to localize the source of hemorrhage. Findings on an abdominal CT with contrast that indicate the need for an urgent celiotomy, even in a hemodynamically stable patient.

When an abdominal contrast CT is not available to evaluate the hemodynamically-stable patient, a chest radiograph, flat plate radiograph of the abdomen, or contrast study of the gastrointestinal or genitourinary tract will detect all of the injuries except those to the liver, spleen, or pancreas. As previously noted under ‘Patterns of injury', it is important to evaluate patients with marks of a restraint device across the lower abdomen after blunt deceleration/compression trauma carefully. In a former era, many of these patients would have died in a frontal collision. By surviving in the modern era of restraints, death is exchanged for deceleration/compression injuries in the abdomen. In one review of 61 children with a ‘linear ecchymosis across the abdomen' (seatbelt sign) after a motor vehicle crash, 14 children (23 per cent) injured a hollow viscous, (21 per cent) had an injury to the lumbar spine, and five (8 per cent) had injury to both.

**Penetrating trauma**

*The initial examination* (or ABCDE) of patients with penetrating abdominal trauma includes assessment of the following:

- Airway, breathing, circulation (ABC): includes vital signs
- Level of consciousness (D, capacity): to identify neurological deficits.
- Location of wounds (E, exposure): to examine the entire body surface and to document all penetrating wounds
- Type of penetrating weapon or object
- Blood loss.

In case of emergency surgery, all patients with penetrating abdominal trauma should undergo *basic laboratory tests:*

- Blood group and Rhesus
- Complete blood count
- Biochemical blood test (electrolytes, creatinine, urea, urea nitrogen)
- Blood glucose level
- Coagulogram (Prothrombin time (PT)/activated partial thromboplastin time (aPTT))
- Venous or arterial lactate level
- Complete urinalysis

*Treatment tactics*

The following diagnostic and therapeutic procedures should be performed in patients with penetrating abdominal trauma:

- nasogastric decompression of the stomach in intubated patients: to prevent aspiration
- Foley catheter placement: to monitor rehydration therapy, to control diuresis
- peritoneal lavage (open or closed): to detect damage to hollow organs or the diaphragm
- pleural drainage: to relieve hemothorax / pneumothorax
- local wound examination: a diagnostic aid to determine the path of penetration through the layers of the abdominal wall
- laparoscopy: to evaluate and treat intra-abdominal injuries, including stab wounds of the anterior abdominal wall with unspecified penetration.

Approximately 25 to 33 percent of patients with stab wounds of the anterior abdominal wall (between anterior axillary lines) do not have penetration of the peritoneal cavity. Therefore, in stable and cooperative patients without clear indications of a celiotomy (see below), it is necessary to inspect the wound and wound canal. The procedure can be performed by finger under local anesthesia under sterile conditions. If the finger passes into the peritoneal cavity, such an injury is considered as penetrating the abdominal cavity. The risk of damage to internal organs is very high. In addition, there is an infection of the abdominal cavity. Revision of the abdominal organs and sanitation of the latter is indicated. Usually, it is performed through a laparotomy. Recently, some authors have considered an alternative approach – diagnostic and curative laparoscopy.

Patients without penetration of the wound into abdominal cavity are discharged after the stab wound site is irrigated and closed. Penetration of the anterior fascia in large patients or the anterior peritoneum in thin patients mandates further evaluation. The most common option chosen around the world is serial physical examinations for 24 h by a surgeon or senior resident. This noninvasive approach results in a delay to definitive operation in only 5 to 6 per cent of patients with intra-abdominal injuries. A second option is to perform a standard diagnostic peritoneal tap/lavage with positive results being the same as described for blunt abdominal trauma. This invasive technique results in a certain number of false-positive results (bleeding from the site of the stab wound), occasional false-negative results (early lavage after small stab hole of midgut), and has an overall accuracy of 88 to 94 per cent. In the 45 to 50 per cent of patients who are originally asymptomatic despite having penetration of the anterior peritoneal cavity, 50 per cent of these will eventually come to a celiotomy based on a changing physical examination or on a positive tap or lavage.

Approximately 50 to 55 per cent of patients with anterior stab wounds penetrating the peritoneal cavity have the same obvious indications for an emergency celiotomy as do patients with blunt abdominal trauma. In addition, patients with the following should also undergo celiotomy: (1) new onset hematemesis, proctorrhagia, or hematuria; (2) evidence of a left-sided diaphragmatic defect on finger palpation prior to insertion of a thoracostomy tube; or (3) contrast radiography evidence of an injury to the kidney (significant injury), ureter, or bladder.

The management of stab or gunshot wounds to the flank (between anterior and posterior axillary lines from sixth intercostal space to iliac crest) or to the back (posterior to posterior axillary line from tip of scapula to iliac crest) has changed over the past 20 years. Because of the large bulk of muscles in this area in young males, the routine celiotomies that were performed in the past were often negative. In patients in whom a local wound exploration does not reveal the end of a stab wound track, either serial physical examinations or double (intravenous and oral) or triple (add rectal and colon) contrast CT is performed. Serial examinations result in a false-positive (unnecessary celiotomy)/ false-negative (delayed celiotomy) rate of 5 per cent. Examination of most or all retroperitoneal viscera and vascular structures using double or triple contrast CT has an overall accuracy rate of 96 to 97 per cent. Older data documented that gunshot wounds traversing the peritoneal cavity resulted in visceral or vascular injuries needing surgical repair in 96-98% of patients. Nowerday it is

considered that in 15- 30% of cases with gunshot wounds in proximity to the peritoneal cavity or visceral–vascular retroperitoneum actually have missile tracks that pass through the body wall or anterior–lateral extraperitoneal area, only. In addition, some centers are observing isolated gunshot wounds to the liver or kidney in stable patients in whom an emergency CT mostly rules out associated injuries to the gastrointestinal tract. When the hemodynamically stable patient without peritonitis presents with a possible extraperitoneal gunshot wound, serial physical examinations rather than an emergency celiotomy are appropriate. If available, a surgeon-performed ultrasound documenting intraperitoneal fluid (blood in the ‘asymptomatic patient') would be followed by celiotomy rather than serial examinations.

The following drugs may be used to treat patients with penetrating abdominal trauma:

- analgesics (e.g., morphine, fentanyl)
- anxiolytics (e.g., lorazepam, midazolam hydrochloride)
- antibiotics (e.g., cefotetan, metronidazole, gentamicin, vancomycin, ampicillin)
- neuromuscular blocking agents (e.g., succinylcholine, vecuronium bromide)
- immunostimulants (e.g., adsorbed tetanus toxoid)

**Urgent examination of the abdominal cavity**

Midline laparotomy provides the most optimal conditions for abdominal cavity revision, repair of internal organ damage, and abdominal cavity drainage. Cold steel is the fastest way to perform tissue dissection. The primary goal of surgery is to control massive intraabdominal bleeding. Only after this should attempts be made to prevent oozing from the abdominal wall in hemodynamically unstable patients.

Liquid blood accumulations can be evacuated with vacuum aspiration and clots – manually using pads. Massive bleeding is usually the result of damage to large vessels (aorta and its main branches, inferior vena cava and its main tributaries, and portal vein) or parenchymal organs (liver, spleen, kidneys, or pancreas). In many cases, to navigate and reduce the rate of blood loss, it is advisable to use measures to stop bleeding temporarily. If the source is located retroperitoneally, direct compression can be applied on the posterior peritoneum followed by high clamping of the aorta (in subdiaphragmatic space). After visualization of the source of bleeding, the clamp may be displaced caudally to avoid ischemic damage to the bowels and kidneys. In case of liver damage, this may be external packing and a Pringle maneuver, which means compressing the hepatoduodenal ligament. Penetrating trauma is not limited by the elastic properties of the tissue, and vascular injuries are far more common. While these concepts simplify the localization of injuries, unless the patient has exsanguinating hemorrhage, a methodical exploration should always be carried out.

Failure to detect damage to an internal organ can have fatal consequences for the patient. Therefore, the inspection must be carried out carefully. If one of the walls of a hollow organ is damaged, it is imperative to check the opposite wall. Difficulties arise when examining parts of the internal organs located retroperitoneally and in the mesentery of the intestines.

**Vascular Injuries**

Vascular damage is more typical of penetrating injuries than of blunt abdominal trauma. In the latter, the renal vessels are usually affected. Some techniques facilitate the revision of large vessels. Nowadays two methods are utilized to assess the arteries and veins in the retroperitoneal space. Exposure of inframesocolic retroperitoneal organs can be achieved by a right medial visceral rotation (the Cattell-Braasch maneuver). It includes Kocher's maneuver extended to the right colon mobilization.

A central supramesocolic retroperitoneal region can be investigated utilizing the Mattox maneuver, also called "a left medial visceral rotation". This is accomplished by incising the left lateral peritoneal reflection starting at the distal descending colon and extending the incision past the splenic flexure, around the posterior aspect of the spleen, behind the gastric fundus, and ending at the esophagus. This incision permits the left colon, spleen, pancreas, and stomach to be rotated toward the midline. The kidney can also be mobilized, but not obligatory, with the remaining viscera with both right and left medial rotations.

In case of bleeding due to the presence of a hematoma, methods of mobilizing the veins of the portal system, which are used during elective surgical interventions, may be problematic. Therefore, in trauma surgery, the neck of the pancreas is divided without hesitation.

**Diaphragm**

Injury of the diaphragm can be diagnosed by a chest x-ray. In case of penetrating trauma the rupture site may locate in any part of organ, many of these are subtle. Blunt trauma in the vast majority of cases (about 75%) leads to damage to the left dome of the diaphragm and may be wide. These injuries are usually corrected through a celiotomy by applying a running suture. Large deffects need mesh implantation.

**Spleen**

The spleen is an important organ of the immune system and plays an essential role in preventing infection and controlling the cellular composition of the blood. After its removal, the number of platelets increases significantly, which increases the risk of thromboembolic complications. Therefore, the current trend has become to try not to remove it in cases of trauma. Suturing a spleen defect and splenectomy require adequate organ mobilization. Splenectomy is indicated in hilar injuries, parenchyma crushing, and unsucsessful organ salvage and to patients with developed coagulopathy. Preservation of the spleen immunological function can be achived by replantation of its fragments within the leafs of the omentum. Technetium scans have confirmed their viability, and immunoglobulin M (IgM) levels have normalized. If one of the poles is destroyed, partial splenectomy can be used. Wound is closed by horizontal mattress sutures. Following ligation of the sutures and releasing compression, the spleen will expand slightly and further tighten the sutures. After splenectomy, vaccines against the encapsulated bacteria are administered. The (pneumococcal vaccine and vaccines effective against *Haemophilus influenza* and *Neisseria meningitidis* should be used if available).

**Stomach and Small Intestine**

Usually, injuries of the stomach pose no special problems or controversies.

However, it may be difficult to detect a gastric rupture in places where the gastric wall is extraperitoneal. To detect such lesions during surgery, the introduction of a solution of methylene blue through a probe into the stomach helps. In this case, it is advisable to compress the pylorus from the outside, which prevents the evacuation of the dye solution into the small intestine. It is necessary to find the place where the methylene blue leaks into the abdominal cavity.

It is necessary not to forget to check the posterior wall of the stomach when detecting a rupture of the anterior one. For this, it is necessary to dissect the gastrocolic ligament. Detected wall lesions must be sutured.

Small bowel ruptures in blunt abdominal trauma are quite difficult to diagnose even when using modern instrumental methods, such as CT and ultrasound. This is due to the fact that there may be no gas in the small intestine, and the amount of fluid is insignificant. It is difficult to detect even with CT. In order not to miss such lesions, it is necessary to continue monitoring the patient for 2-3 days in a hospital setting. Early signs of local peritonitis indicate a rupture of the small intestine. It has been shown that such conservative observational tactics do not worsen the results of treatment.

When inspecting the intestine during surgery, it is necessary to examine its walls from both sides, paying special attention to the places of fixation of the mesentery. Damage may be hidden there. Intestinal tears are sutured in the usual way. If there are a large number of defects in one place, it is advisable to resect the damaged segment..

**Pancreas**

Isolated injuries to the pancreas are rare due to its location. They are usually accompanied by injury to the surrounding organs: the stomach, intestines, duodenum, and surrounding vessels. Clinically, pancreatic damage manifests itself in the clinical picture of acute traumatic pancreatitis. In case of rupture of the gland, enzymatic peritonitis may occur due to leakage of pancreatic juice. CT helps to detect pancreatic damage, although there is evidence that it is of little information value in the first 6 hours after injury. During surgery, it is necessary to conduct a thorough inspection of the pancreas, macroscopically assess the condition of the parenchyma, the presence of edema, hematomas and clearly non-viable areas. It is very important to verify damage to the ductal system. It can be difficult to detect it during external examination. The feasibility of performing pancreatography through duodenotomy, resection of the tail of the gland and endoscopic retrograde pancreatography is debated. All these methods have many disadvantages. It is apparent based on the above options that no ideal method exists for identifying pancreatic ductal injuries that cannot be ruled out by direct exploration. This dilemma tends to encourage aggressive local exploration, which may create a ductal injury where none existed.Therefore, aggressive, traumatic examination should be avoided.

The extent of surgical intervention depends on the presence of a defect in the ductal system of the gland. In its absence, it is advisable to adequately drain the omental bursa. In case of detection of rupture of the main duct, it is advisable to consider the following options – distal resection of the pancreas (preferably with preservation of the spleen in the absence of its injury) or external drainage of the ducts.

Pancreatoduodenal injuries are caused by high-energy gunshot wounds. In patients with a pancreatoduodenal injury who also have an intrapancreatic bile duct

injury, it is possible to use the combination of pyloric exclusion and Roux-en-Y choledochojejunostomy to avoid a pancreatoduodenectomy. However, the complexity and unpredictable physiology of the combined procedures makes the pancreatoduodenectomy more attractive.

**Hepatic trauma**

Compressive injuries to the liver from the overlying ribs occur most frequently in frontal motor vehicle crashes in which the victim has an impact with the lower rim of the steering wheel or the dashboard. Compression against a shoulder belt restraint may be a cause, as well, particularly if the device is worn improperly under the right upper extremity. In 'T-bone' side impacts, the front seat passenger is at significant risk for a hepatic injury. Patients with penetrating wounds to the right thoracoabdominal area (nipples to costal margin and medial to right anterior axillary line) are at risk of a hepatic injury if the diaphragm is penetrated. This occurs in approximately 15 per cent of patients with penetration of the body wall by a stab wound and in 45 to 48 per cent of those with gunshot wounds.

**Diagnosis**

In hypotensive patients who have suffered blunt abdominal or multisystem trauma, either surgeon-performed ultrasound or a standard infraumbilical diagnostic peritoneal lavage is appropriate. Using a 3.5 mHz transducer in the right midaxillary line between ribs 10 and 11, the visualization of fluid (blood unless ascites is present) in Morison's pouch mandates a laparotomy in the absence of other overt sites of hemorrhage. An experienced surgeon-sonographer may visualize a hepatic injury, also. When no fluid is present in Morison's pouch, the ultrasound probe is moved to image the left subphrenic area/splenorenal recess and the pelvis. A diagnostic peritoneal tap that yields 10 to 20 ml of gross blood or a formal lavage whose effluent is cloudy enough to obscure the print on the bag of intravenous fluids mandates laparotomy in the hypotensive patient, also. In any patient undergoing emergency laparotomy after suffering blunt abdominal trauma, the most likely sources of hemorrhage are injuries to the liver, spleen, or mesentery. A patient who is hemodynamically stable and without peritonitis after suffering blunt abdominal trauma is evaluated by a spiral contrast CT if the physical examination is equivocal or compromised or if there is intra-abdominal fluid on the preliminary ultrasound. The volume of intraperitoneal fluid (blood), magnitude of injury to the liver or other organ, and the presence or absence of active hemorrhage on the contrast CT will determine whether nonoperative or operative management is chosen in the stable patient.

Penetrating wounds to the abdomen in patients with peritonitis, hypotension, or significant evisceration mandate laparotomy. Stab wounds to the right thoracoabdominal area in patients without fluid in the right subphrenic space or Morison's pouch on ultrasound undergo in-hospital serial physical examinations for 24 h after admission. An occasional stable patient with a gunshot wound to this area and minimal tenderness may be evaluated by a contrast spiral CT to determine the magnitude of hepatic and pulmonary injuries.

**Nonoperative management**

Approximately 80 to 85 per cent of all patients with hepatic trauma are stable upon arrival in the emergency center, and, in the absence of other indications for an

emergency laparotomy, nonoperative management is appropriate after a contrast spiral CT. Careful observation of patients who are on bed rest will allow timely detection of signs of internal bleeding or peritonitis. In this situation laparptomy is indicated. An increase in the size of the liver indicates the presence of a hematoma in the parenchyma without rupture of the organ capsule.

If a repeat spiral CT 5-7 days following trauma determine some healing, patient can be ambulated.

Approximately 2-7% of patients need to discontinue conservative treatment and proceed to surgery. The hepatic injury, itself, will be the cause in 50 to 75 per cent of the failures, and 65 to 85 per cent of the hepatic failures will be in patients with Grade IV or V injuries on the original CT. The same is gonoperative management of gunshot wounds of the liver. The success rate is similar to that described above for blunt trauma as missile tracks from civilian handguns are significantly smaller than many of the Grade IV or V hepatic injuries presently undergoing nonoperative management.

**General principles of operative management**

A midline incision is used, and blood and clots are evacuated manually or with a suction device. A vascular clamp is applied to the porta hepatis (Pringle maneuver) if a significant (Grade III, IV, V) hepatic injury is present. The injured lobe is compressed between laparotomy pads in the hands as the surgeon informs the anesthesiologist about the need to contact the blood bank. Also, the surgeon should request that an upper hand retractor, various sizes of metal clips, O-chromic sutures on blunt needles, and a 36–38 French thoracostomy tube be available in the operating room. When blood and appropriate equipment is available in the operating room, the packs around the liver are removed and the hepatic injury is inspected. Posterior lobar injuries or Grade III, IV, or V injuries are best visualized by division of the ipsilateral triangular ligament and the anterior coronary ligament at the edge of the liver. Folded dry laparotomy pads are then placed beneath the injured lobe to elevate it into the midline incision. In obese patients or in those with a high likelihood of an injury to the extrahepatic veins or retrohepatic vena cava (dark venous hemorrhage as the injured lobe is mobilized), a median sternotomy is also performed.

**Simple techniques of hemostasis**

Suturing the damaged parehima followed by external compression for several minutes, preferably with topical hemostatic is effective in approximately 90 per cent of penetrating injuries and 60 per cent of blunt injuries. Currently available topical hemostatic agents include oxidized regenerated cellulose, microfibrillar collagen hemostat, and fibrin sealant. Fibrin sealant, only recently available in the United States, contains human fibrinogen and thrombin, aprotinin, and calcium chloride. Five minutes of compression is performed after the application of a topical agent. After releasing compression, the electrocautery is used for any remaining bleeders when only Grade I or Grade II hepatic injuries are present. Suture hepatorrhaphy with O-chromic material is appropriate for Grade II and Grade III injuries. An interrupted or continuous suture technique is used, with the caveat that crushing sutures cause postoperative hepatic necrosis and ‘liver fever'. Drainage is not necessary in the absence of further hemorrhage or obvious leakage of bile.

**Advanced techniques of hemostasis**

Advanced techniques are necessary in 10% of penetrating wounds and in 40 per cent of blunt hepatic injuries. These patients have Grade III, IV, or V injuries.

***Extensive hepatorrhaphy***

Extensive hepatorrhaphy is indicated in ‘damage control' situations in which intraoperative hypothermia (<34–35°C), metabolic acidosis (pH < 7.1–7.2), and/or a coagulopathy (PT or PTT > 50 per cent normal) mandate a rapid operation. Large figure-of-eight sutures or a continuous O-chromic suture is used to reapproximate the sides of hepatic lacerations in the hope that hemorrhage from small hepatic arteries and low pressure hepatic veins or portal veins will be controlled by compression. Extensive postoperative hepatic necrosis is likely when such sutures are tied too tight in the presence of a prolonged Pringle maneuver.

***Hepatotomy with selective vascular ligation***

Gaining further exposure of a deep hepatic laceration or connecting the entrance and exit wounds of a penetrating wound with the finger fracture technique or the electrocautery is known as hepatotomy. Once completed, large Deaver or Harrington retractors are used to maintain visibility in the depths of the hepatotomy as selective vascular clipping or suture ligation of injured vessels is performed. This technique should be utilized prior to the onset of hypothermia and only by surgeons with sufficient experience in elective or traumatic hepatic surgery.

***Viable omental pack***

The gastrocolic omentum mobilized off the transverse colon with its blood supply intact is used to fill Grade III, IV, or V hepatic injuries or hepatotomy sites.

Intrahepatic omentum is effective in controlling venous hemorrhage, managing dead space, and in bringing mobile macrophages to the site of injury. While it does not appear to aid healing, postoperative bleeding and drainage of bile are much decreased in the experience of most trauma surgeons. The viable omental pedicle is held in place by compressing hepatic sutures tied under moderate tension.

***Resectional debridement with selective vascular ligation***

With disrupted hepatic tissue on the edge of an injured liver, the finger fracture technique or the electrocautery should be used to create a new fresh edge of the liver around the area of injury. Vessels and biliary ducts can then e clipped or suture ligated where they are intact, and all disrupted tissue outside this new line is then debrided. The application of a viable omental pedicle to this new raw surface is controversial, though this is appropriate when a coagulopathy makes hemostasis difficult.

***Absorbable mesh compression***

Wrapping an injured hepatic lobe in which all fragments are viable with a large sheet of absorbable mesh tailored around the porta hepatis and inferior vena cava hasbeen used in some centers. The technique is time-consuming, but eliminates the need for reoperation as when perihepatic packs are used for compression.

***Formal resection***

Anatomic lobectomy is used in approximately 3 per cent of patients undergoing operative management. No dissection is performed in the porta hepatis, and the lobectomy is performed with a Pringle maneuver in place using finger fracture or electrocautery and metal clips. The large right hepatic vein can usually be controlled

inside the liver as the lobectomy is completed. Anatomic segmentectomy is much more commonly utilized, especially with extensive lacerations beneath the falciform ligament mandating resection of Couinaud's segments II and III (left lateral segment).

***Selective hepatic artery ligation***

Selective hepatic artery ligation is used in about 1 per cent of patients undergoing operative management. It is indicated when arterial hemorrhage in a deep hepatic laceration cannot be directly controlled, but stops whenever a Pringle maneuver is applied. Extrahepatic ligation of the artery to the injured lobe in the porta hepatis will fail to control hemorrhage when the wrong artery is ligated or when intrahepatic or retrohepatic venous hemorrhage is present.

***Intrahepatic balloon tamponade***

The passage of a Foley or Fogarty balloon catheter into the hepatic track of a knife or missile may allow for balloon compression of the site of parenchymal hemorrhage. This technique is particularly useful when the novice trauma surgeon has little experience in completing an extensive hepatotomy through one or both lobes. The inflated balloon catheter is passed through the body wall away from the midline incision at the completion of the first laparotomy. After 48 to 72 h of balloon compression, the balloon is eflated and removed through the body wall in the surgical intensive care unit. Rebleeding is extraordinarily rare when a parenchymal track has been tamponaded for this period of time.

***Perihepatic packing***

The insertion of folded dry laparotomy pads over and, occasionally, below an injured hepatic lobe is used in approximately 5 per cent of patients undergoing operative management. Packs should be used to tamponade minor hepatic injuries or subcapsular hematomas when a damage control procedure is performed. They are also useful for any major hepatic parenchymal injury when advanced techniques of hemostasis fail secondary to intraoperative hypothermia or a coagulopathy. The use of a plastic sheet beneath the packs to prevent sticking to raw edges of parenchyma has been useful in the author's experience. Packs are removed at a reoperation 48 to 72 h after the original laparotomy when hypothermia, acidosis, and any coagulopathy are corrected and the cardiovascular, respiratory, and renal systems are stable. Perihepatic packs have also been used with success in patients with unruptured retrohepatic hematomas from presumed injuries to the retrohepatic vena cava.

***Atriocaval shunt***

Incertion of No.36 French thoracostomy catheter from the right atrium into the infrarenal part of the inferior vena cava provides temporary restoration of venous return from the lower body.

This causes a 40 to 60 per cent decrease in hemorrhage from an injury in the retrohepatic vena cava and should allow for a rapid repair. When there has not been a preoperative or intraoperative cardiac arrest from exsanguination, use of the atrocaval shunt has resulted in a 33 to 50 per cent survival in the modern era. Alternative approaches for injuries to the retrohepatic vena cava include direct approach behind an injured lobe, total hepatic vascular isolation, and deep hepatotomy.

**Drainage**

Closed suction drains above and below an injured lobe are used when an intraoperative coagulopathy or the extent of hepatic repair suggests that postoperative drainage of blood and bile is likely.

**Complications**

Postoperative hyperpyrexia occurred in nearly two-thirds of patients with Grade III, IV, or V injuries in one review. Early postoperative coagulopathies occur in 15 per cent of patients, while reoperations for persistent or late hemorrhage used to be necessary in 3 to 7 per cent of patients. Self-limited biliary fistulas occur in 8 to 10 per cent of patients, while intra-abdominal abscesses develop in 4 to 10 per cent. The embolization of disrupted intrahepatic arteries or pseudoaneurysms to control postoperative bleeding or late hemobilia by the interventional radiologist has caused a significant decrease in reoperations for hemorrhage. Reoperations for perihepatic abscesses have essentially disappeared in the modern era for the same reason.

**Mortality**

The liver-related mortality for patients undergoing nonoperative management has been 0 to 0.5 per cent in recent reports. Overall mortality in such patients is 8 to 9 per cent and is primarily due to associated intracranial injuries. In patients undergoing operation for blunt hepatic injuries, the overall mortality is 15 to 20 per cent. The mortality after operation for stab wounds and gunshot wounds to the liver is 2.5 and 10 per cent, respectively.

**Gallbladder and Extrahepatic Bile Ducts**

Damage to the bile ducts can be detected by performing intraoperative cholangiography or detecting bile leakage during compression of the gallbladder. If the duct is not completely transected, it must be sutured and external drainage of the bile ducts performed. If the duct is completely transected, a Roux-en-Y choledocho- or hepaticojejunal anastomosis is performed. Defect of gallbladder can be sutured, alternative is the cholecystectomy.

**Trauma damage control**

Patients with shock from exsanguination related to abdominal injuries and massive transfusion develop pre- or intraoperative hypothermia, persistent metabolic acidosis, and a coagulopathy. This so-called ‘vicious cycle of metabolic failure' is irreversible as long as the patient is in the operating room with the abdomen open during a prolonged procedure. Pre- or intraoperative markers that confirm ‘metabolic failure' and suggest that a ‘damage control' operation should be performed instead of a definitive laparotomy. The concept of ‘damage control' operations has now been used for over 15 years and includes three phases. Alternate closures of the abdominal incision or coverage of the open abdomen are used in most patients undergoing a ‘damage control' operation. Prospective randomized trials to confirm the value of ‘damage control' are unlikely to be performed as the concept is now widely accepted. Also, it appears to allow for the salvage of severely injured patients who died when prolonged definitive first celiotomies were performed.

**Colon**

The treatment of injuries of the colon has been debated for nearly a century. Finally, during the past decade, something resembling a consensus has been reached.

There are three methods for repairing colonic injuries. First one is the direct suturing of the defect or resection of the affected bowel. Second one is creating the colostomy, preferably loop colostomy with the resection of the injured colon. Exteriorized repairs are created by suspending a repaired perforation or anastomosis on the abdominal wall with an appliance after the fashion of a loop colostomy. If after 10 days the suture line does not leak, it can be descended into the abdomen under local anesthesia without subsequent risk of leakage. If the repair breaks down before 10 days, it is treated as a loop colostomy. Healing is successful in 50 to 60% of cases. The advantage is avoidance of an intraperitoneal suture line when it is at risk of leakage. Stomal complications similar to those of colostomies also can occur with the exteriorization. Exteriorized repair is probably no longer indicated since most patients who were once candidates for this treatment are now successfully managed by primary repair. Two methods have been advocated that result in 75 to 90% of penetrating colonic injuries being safely treated by primary repair. The first is to repair all perforations not requiring resection. If resection is required due to the local extent of the injury, and it is proximal to the middle colic artery, the proximal portion of the right colon up to and including the injury is resected and an ileocolostomy performed. If resection is required distal to the middle colic artery, an end colostomy is created and the distal colon oversewn and left within the abdomen. The theory behind this approach is that an ileocolostomy heals more reliably than colocolostomy, because in the trauma patient who has suffered shock and may be hypovolaemic, assessing the adequacy of the blood supply of the colon is much less reliable than in elective procedures. The blood supply of the terminal ileum is never a problem.

The other approach is to perform primary repair in almost all cases, reserving colostomy only for patients with severe fecal contamination and shock. Both of these approaches are reasonable and result in the majority of patients being treated by primary repairs.

**Kidneys**

There are several unique aspects to evaluate the renal injuries. Three imaging techniques, CT, intravenous (IVP), and arteriography, can be used to accurately evaluate the extent of a renal injury. However, the contrast material required for each is nephrotoxic and limits the number of studies that can be performed. Almost all blunt injuries of the kidneys are treated without surgery. Main symptom is hematuria. Diagnos can be confirmed by CT or IVP. Embolization can be helpful in the control of persistent hematuria. Persistent urinomas can be drained percutaneously. Surgical intervention is indicated only if the listed treatment methods are ineffective.

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**Tests**

1. Which of the following conditions is most likely to follow a compression-type abdominal injury?
   A. Renal vascular injury
   B. Superior mesenteric thrombosis
   C. Mesenteric vascular injury
   D. Avulsion of the splenic pedicle
   E. Diaphragmatic hernia
2. Blunt trauma to the abdomen most commonly injures which of the following organs?
   A. Liver
   B. Kidney
   C. Spleen
   D. Intestine
   E. Pancreas
3. Following blunt abdominal trauma, a 12-year-old girl develops upper abdominal pain, nausea, and vomiting. An upper gastrointestinal series reveals a total obstruction of the duodenum with a "coiled spring" appearance in the second and third portions. Appropriate management is
   A. Gastrojejunostomy
   B. Nasogastric suction and observation
   C. Duodenal resection
   D. TPN to increase the size of the retroperitoneal fat pad
   E. Duodenojejunostomy
4. An elderly pedestrian collides with a bicycle-riding pizza delivery man and suffers a unilateral fracture of his pelvis through the obturator foramen. You would manage this injury by
   A. External pelvic fixation
   B. Angiographic visualization of the obturator artery with surgical exploration if the artery is injured or constricted
   C. Direct surgical approach with internal fixation of the ischial ramus
   D. Short-term bed rest with gradual ambulation as pain allows after 3 days
   E. Hip spica
5. When operating to repair civilian colon injuries
   A. A colostomy should be performed for colonic injury in the presence of gross fecal contamination
   B. The presence of shock on admission or more than two associated intraabdominal injuries is an absolute contraindication to primary colonic repair
   C. Distal sigmoidal injuries should not be repaired primarily
   D. Right-sided colonic wounds should not be repaired primarily
   E. Administration of intravenous antibiotics with aerobic and anaerobic coverage has not been shown to decrease the incidence of wound infections after repair of colonic injuries

6. A 36-year-old man was admitted to the hospital with a gunshot wound to the right buttock. He is hemodynamically stable. There is no exit wound, and an X-ray of the abdomen shows the bullet to be located in the right lower quadrant. Correct management of a suspected rectal injury would include
   A. Barium studies of the colon and rectum
   B. Barium studies of the bullet track
   C. Endoscopy of the bullet track
   D. Angiography
   E. Sigmoidoscopy in the emergency room
7. Correct statements regarding blunt trauma to the liver include which of the following?
   A. Hepatic artery ligation for control of bleeding is associated with decreased morbidity and mortality
   B. The incidence of intraabdominal infections is significantly lower in patients with abdominal drains
   C. Intracaval shunting has dramatically improved survival among patients with hepatic vein injuries
   D. Nonanatomic hepatic debridement, with removal of the injured fragments only, is preferable to resection along anatomic planes
   E. Major hepatic lacerations that are sutured closed will result in intrahepatic hematomas, hemobilia, and bile fistulas
8. 18-year-old high school football player is kicked in the left flank. Three hours later he develops hematuria. His vital signs are stable. The diagnostic tests performed reveal extravasation of contrast into the renal parenchyma. Treatment should consist of
   A. Resumption of normal daily activity excluding sports
   B. Exploration and suture of the laceration
   C. Exploration and wedge resection of the left kidney
   D. Nephrostomy
   E. Antibiotics and serial monitoring of blood count and vital signs
9. An 18-year-old high school football player is kicked in the left flank. Three hours later he develops hematuria. His vital signs are stable. Initial diagnostic tests in the emergency room should include which of the following?
   A. Retrograde urethrography
   B. Retrograde cystography
   C. Arteriography
   D. Intravenous pyelogram
   E. Diagnostic peritoneal lavage
10. True statements concerning penetrating pancreatic trauma include
   A. Most injuries do not involve adjacent organs
   B. Management of a ductal injury to the left of the mesenteric vessels is Roux-en-Y pancreaticojejunostomy
   C. Management of the injury of the head of the pancreas with the damage of main pancreatic duct is pancreaticoduodenectomy

D. Small peripancreatic hematomas need not be explored to search for pancreatic injury
E. The major cause of death is exsanguination from associated vascular injuries

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| E | C | B | D | B | E | B | E | B | E |

**Materials for the self-study of the students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of organs of abdominal cavity, structure of the peritoneum and anatomy intestinum, features of blood circulation of liver and spleen.<br>– Physiology of peritoneum and organs of abdominal cavity.<br>– Pathogenesis of development of complications from abdominal trauma.<br>– Morphological changes in the organs of abdominal cavity. | -To represent the methods of diagnostics of diseases of abdomen and peritoneum as a table<br>-To make the flow diagram of mechanisms of damage of abdomen |
| Study:<br>– Techniques of hemostasis at injures of the liver.<br>– Types of accesses to the organs of abdominal cavity.<br>– Tactics at the thoracoabdominal trauma.<br>– Tactics at connections of head trauma and trauma of abdomen. | -To conduct differential diagnosis with the damages of organs of abdomen<br>-To conduct differential diagnosis of pathologies of the abdomen |

# TOPIC 8. SYNDROMES OF CHEST PAIN, RESPIRATORY AND HEART FAILURE IN THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE CHEST CAVITY. MODERN METHODS OF DIAGNOSIS AND SURGICAL TREATMENT OF CARDIOVASCULAR DISEASES. SURGICAL PATHOLOGY OF THE LUNGS AND PLEURA. DAMAGE TO THE CHEST CAVITY ORGANS

## 8.1. Syndromes of chest pain, respiratory and heart failure in the diagnosis and differential diagnosis of diseases of the chest cavity.

**Overview.**

Few symptoms are more alarming than chest pain. In the minds of many people, chest pain equals heart pain. While many other conditions can cause chest pain, cardiac diseases are so common and so dangerous that the symptom of chest pain should never be dismissed as being insignificant.

"Chest pain" is an inaccurate term. It is often used to describe any pain, pressure, squeezing, choking, numbness or any other discomfort in the chest, neck, or upper abdomen, and is often associated with pain in the jaw, head, or arms. It can last from less than a second to days or weeks, can occur frequently or rarely, and can occur intermittently or predictably. With such a broad definition, you can see why the term "chest pain" is of little help to doctors.

It is important to keep in mind that chest pain is merely a symptom, not a diagnosis. And because it can be a symptom of anything from a catastrophe to a trivial medical problem, when a person experiences chest pain it is important to try to characterize that pain as quickly as possible as being either completely benign, or possibly significant.

Chest pain is the presenting symptom in about 12% of emergency department visits and has a one year mortality of about 5%.

**Educational aims:**

- Interrogation and clinical inspection of patients with chest pain.
- To determine the etiologic and pathogenic factors of chest pain.
- To find out the types of chest pain, the clinical features, different variants of manifestation and complications.
- To develop a plan of examination of the patients with chest pain.
- To estimate laboratory data end instrumental examination of the thorax.
- To substantiate and formulate a diagnosis, conduct a differential diagnosis for the patients with chest pain.
- To prescribe the treatment for patients with chest pain.
- To determine the indications for operative treatment of patients with chest pain.
- To cure the patients with chest pain.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomo-physiological information about thorax.

- Classifications of chest pain.
- Mechanisms of chest pain.
- Clinical picture of chest pain.
- Methods of diagnosis of chest pain.
- Principles of treatment of chest pain.
- Features of surgical interventions for the different types of chest pain.

**A student must be able to:**

- Collect and estimate the complaints of patient with chest pain, information of anamnesis, to conduct physical research and correctly interpret the results obtained.
- Define the rational volume of laboratory and instrumental methods of research.
- Correctly interpret the results of clinical analyses, instrumental examinations
- Define indications for operation and other methods of treatment of patients.
- Perform pre-operative preparation of patients.
- Conduct post-operative care.

**Terminology.**

| Term | Definition |
|---|---|
| Respiratory Insufficiency: | A condition where the lungs are unable to function properly and maintain the normal processes of oxygen uptake and carbon dioxide elimination. |
| Stable angina | Chest pain follows a specific pattern, occurring when someone engages in hard physical activity or experiences extreme emotion. Other situations that bring on angina include smoking a cigarette or cigar, cold weather, a large meal and straining in the bathroom. The pain usually goes away when the pattern or trigger ends. |
| Unstable angina | This chest pain occurs at rest, during sleep or very often with minimal exertion. The discomfort may last and be intense. |
| Pneumothorax (pleural pneumothorax) | Is a collection of air or gas in the pleural cavity of the chest between the lung and the chest wall. |
| Hemothorax (or haemothorax) | Is a condition that results from blood accumulating in the pleural cavity. |
| Dyspnea or dyspnoea | (From Latin dyspnoea, from Greek dyspnoia from dyspnoos, shortness of breath) is a debilitating symptom that is the experience of unpleasant or uncomfortable respiratory sensations. |

**Content:**

Chest pain may be a symptom of a number of serious conditions and is generally considered a medical emergency. Even though it may be determined that the pain is non-cardiac in origin, this is often a diagnosis of exclusion made after ruling out more serious causes of the pain.

**Differential diagnosis**

The causes of chest pain range from non-serious to life threatening.

**Cardiovascular**

- Acute coronary syndrome
- Myocardial infarction ("heart attack")
- Pericarditis and cardiac tamponade
- Unstable Angina Pectoris - requiring emergency medical attention rather than primary intervention, as in myocardial infarction
- Aortic dissection
- Pericarditis and cardiac tamponade
- Arrhythmia - atrial fibrillation and a number of other arrhythmias can cause chest pain.
- Stable angina pectoris - it can be treated medically, and although it requires examination, it is not an emergency in its strict sense

**Pulmonary**

- Pulmonary embolism
- Pneumonia
- Hemothorax
- Pneumothorax and Tension pneumothorax
- Pleurisy - an inflammation which can cause painful respiration

**GI**

- Gastroesophageal reflux disease (GERD), other causes of heartburn
- Hiatus hernia (which may not accompany GERD)
- Nutcracker esophagus, achalasia, other neuromuscular disorders of the esophagus
- Functional dyspepsia

**Chest wall**

- Costochondritis or Tietze's syndrome - a benign and harmless form of osteochondritis often mistaken for heart disease
- Spinal nerve problems
- Fibromyalgia
- Chest wall problems
- Radiculopathy
- Precordial catch syndrome

**Chest conditions**

- Herpes zoster commonly known as shingles
- Tuberculosis

**Psychological**

- Anxiety
- Hypochondria
- Clinical depression
- Somatization disorder
- Panic attack

**Others**

- Da Costa's syndrome
- Precordial catch syndrome - another benign and harmless form of acute localized chest pain that is often mistaken for heart disease
- Bornholm disease - a viral disease that can mimic many other conditions
- Hyperventilation syndrome often manifests as chest pain and a tingling sensation in the fingertips and around the mouth
- Carbon monoxide poisoning
- Sarcoidosis
- Lead poisoning
- High abdominal pain may also mimic chest pain

**Diagnostic approach**

In the emergency department the typical approach to chest pain involves ruling out the most dangerous causes: heart attack, pulmonary embolism, thoracic aortic dissection, esophageal rupture, tension pneumothorax and cardiac tamponade. By elimination or confirmation the most serious causes, a diagnosis of the origin of the pain maybe made. Often, no definite cause will be found and reassurance is then provided.

As in any other medical condition, a thorough history and physical examination are essential to distinguish serious causes from trivial ones. To focus on the investigation, management of chest pain may be done in specialized departments (called medical examination departments). Rapid diagnosis can be life-saving and often requires no X-ray or blood tests (aortic dissection). It is helpful to focus on family history (premature atherosclerosis, cholesterol disorders), recent health changes, smoking, diabetes, and other risk factors.

**Diagnostic Evaluation**

The initial tests for patients with chest pain should include an electrocardiogram (ECG) and a chest x-ray.

The ECG may demonstrate regional ST segment depression/elevation indicating myocardial ischemia/ infarction, or may reveal the diffuse ST segment elevation of pericarditis. A chest x-ray may reveal rib fractures, focal infiltrates of pneumonia, wedge-shaped peripheral infiltrates of pulmonary emboli, or the radiolucency of a pneumothorax. It may also suggest aortic dissection (widened mediastinum), or hiatal hernia (stomach in the thoracic cavity).

If an acute coronary syndrome is suspected, medical therapy should be immediately started and serial ECGs and cardiac enzymes (creatine kinase and troponin) checked to confirm or exclude a myocardial infarction. For patients in whom the diagnosis remains uncertain but coronary artery disease (CAD) is suspected, a stress test can be performed for clarification. Chest pain associated with ST segment depression during a stress test is diagnostic of angina. Cardiac catheterization remains the gold standard for the diagnosis of coronary artery disease and may be necessary to rule out significant CAD in a subset of patients for whom other tests are unable to confirm or exclude the diagnosis.

In patients with pulmonary emboli, arterial blood gases usually reveal hypoxia and/or widened A-a gradient, and ventilation/perfusion (V/Q) scanning or spiral CT scanning may confirm the diagnosis. Patients suspected of having an aortic dissection should undergo an urgent transesophageal echocardiography, CT scanning with intravenous contrast, or magnetic resonance imaging (MRI). Patients suspected of having a gastroesophageal cause of their chest pain may need a barium swallow (esophageal reflux or rupture), endoscopy (esophagitis, gastritis, peptic ulcer disease), hepatobiliary hydroxyiminodiacetic acid (HIDA) scan or abdominal ultrasound (gall bladder disease), esophageal manometry (esophageal spasm), or continuous esophageal pH measurement (reflux) to confirm the diagnosis.

Depending on test results, patients may be referred to other specialized areas for additional testing and therapy. Common referral areas include:

- Endocrinology
- Gastroenterology
- Hypertension
- Lipid management
- Nicotine dependence
- Patient and health education
- Pulmonary medicine
- Sleep disorders
- Vascular medicine
- Weight/nutrition counseling

Visits to other areas are coordinated by the patient's primary physician. Results from these visits are returned to the primary physician to help develop a treatment plan.

**Angina Due to Coronary Artery Disease**

Angina is caused by ischemia (oxygen starvation) of the cardiac muscle. Coronary artery disease produces ischemia by narrowing the coronary arteries. Subsequently, when the heart tries to perform at a high level (such as during exercise), the narrowed artery is incapable of delivering the necessary blood volume to the working muscle. Ischemia ensues, and the resultant pain is called angina.

**Characteristics of pain:** Angina can be quite variable, but classically is described as a pressure-like, squeezing, crushing, or tight pain. Some patients with angina deny pain at all, but agree when the term "discomfort" is described to them. The discomfort often radiates to the jaw, shoulders or arms. It can be accompanied by nausea, sweating, shortness of breath, dizziness, weakness, or fatigue. Classically, angina is often provoked in a predictable manner by exercise, anger, or a large meal. But it can also occur unpredictably, without any obvious triggering factor, and at rest. Angina that is increasing in frequency or that occurs at rest is referred to as "unstable." Unstable angina often precedes a heart attack, and should be treated as a medical emergency.

**Evaluation:** If acute coronary syndrome ("unstable angina") is suspected, many people are admitted briefly for observation, sequential ECGs, and determination of cardiac enzymes over time are done. On occasion, further tests on follow up may determine the cause. TIMI score performed at time of admission may help stratify risk.

**Heartburn**

Heartburn is caused by acidic fluid from the stomach washing up into the esophagus.

**Characteristics of pain:** The pain of heartburn is often a burning discomfort directly beneath the breastbone. It is often accompanied by belching, or symptoms of bloating or gas. Sometimes an acid taste occurs in the mouth. Symptoms are often worse after a large meal, or after using tobacco, alcohol, or caffeine. Symptoms tend to improve with antacids.

**Evaluation:** The evaluation includes a careful medical history and physical examination, which often point to the diagnosis. The ECG is generally normal. Special swallowing tests are sometimes necessary to make the diagnosis.

**Benign Chest Wall Pain**

Benign chest wall pain is most likely a transient and fleeting irritation of the pleura, the slippery membrane that lines and protects the lungs. The pleura are very sensitive to pain, and for reasons that are usually not clear, some momentary irritation causes a painful sensation that can be quite severe, but that quickly subsides. This condition has no medical significance whatsoever. It is very common. Most people will experience these symptoms at some point in their lives.

**Characteristics of pain:** The pain is usually a sharp "catch" that interrupts a breath, and that returns with each breath for a few moments - then it subsides. It is not related to exercise, and generally can be localized to a specific small area (smaller than the palm of the hand) on the chest wall. It usually lasts for less than a minute, but can come back on and off for an hour or so. Frequently patients will describe recurrent symptoms every few weeks or months.

**Evaluation:** This condition can be evaluated by taking a careful history. The condition is very common, and very easy to diagnose by history - as long as the doctor is aware of it and understands it. The important thing to keep in mind is that this condition is completely benign, and is not related to any medical problem or any abnormality.

**Anxiety or Panic Disorder**

Anxiety or panic attacks are characterized by repeated episodes of intense anxiety and fear that occur without warning and generally without an identifiable cause. Panic attacks are often accompanied by chest pain, most likely caused by muscle contractions in the chest wall.

**Characteristics of pain:** The pain is usually localized to the chest wall, and can be fleeting and sharp, or can be a sharp "catch" that interrupts a breath. The chest wall can remain "sore" for hours or days after a panic attack. The severity of the chest pain is often magnified by the panic disorder itself.

**Evaluation:** This condition is usually apparent after taking a careful history. However, if the patient has risk factors for coronary artery disease, a noninvasive evaluation to rule out cardiac disease is often necessary. Anxiety disorders are often highly disruptive to an individual's life, and are highly treatable. Unfortunately, doctors all too often brush off patients presenting with chest pain who are found to have anxiety disorders. Patients with this diagnosis should seek active medical treatment from an understanding doctor.

**Asthma, Bronchitis, Pneumonia, Pleuritis**

Many varieties of lung problems can cause chest pain, including disorders of the airways such as asthma or bronchitis, infection of the lungs (pneumonia), and inflammation of the lining of the lungs (pleuritis, or pleurisy).

**Characteristics of pain:** Chest pain caused by lung problems can be localized or diffuse, constant or increasing with breathing, and mild or severe. Pleurisy, in particular, is characterized by chest pain that appears each time a person inhales, and nearly disappears with exhalation. A severe coughing spell or two can sprain the chest muscles, and leave them aching for several days.

**Evaluation:** Chest pain related to lung problems usually becomes quite apparent after the doctor takes a complete medical history, and performs a physical examination.

**Mitral Valve Prolapse**

In mitral valve prolapse (MVP), abnormalities of the mitral valve cause it to flop backwards into the left atrium as the left ventricle contracts. This flopping, or prolapse, is occasionally perceived as a form of chest pain.

**Characteristics of pain:** There is controversy as to how often MVP actually causes chest pain - it probably does so far less often than is alleged. When MVP does cause chest pain, the pain is most often described as a "catching" discomfort, and is often positional in nature (i.e., it may occur, for instance, only when the patient is lying on the left side.)

**Evaluation:** The evaluation of MVP consists of listening to the heart through the stethoscope and doing an echocardiogram.

**Pericarditis**

Pericarditis is inflammation of the pericardium, the membranous sac that encloses and protects the heart.

**Characteristics of pain:** Pericardial pain is usually localized to the area of the chest, and often increases during breathing.

**Evaluation:** The diagnosis of pericarditis is usually made by taking a medical history, doing a physical examination, and doing an ECG.

**Recent Chest Trauma**

Chest trauma of any type can cause chest pain by causing rib fracture or muscle strain or contusion. Not infrequently, a seemingly mild chest trauma (e.g.: sliding into second base during a weekend softball game) can be forgotten when the chest pain becomes apparent a day or two later.

**Characteristics of pain:** Chest pain caused by trauma is almost invariably localized, is often described as soreness or a sharp pain. It usually worsens with breathing, and often the chest wall is tender to touch or to pressure.

**Evaluation:** Chest pain caused by trauma usually becomes quite apparent after the doctor takes a complete medical history, and performs a physical examination. Sometimes a chest X-ray helps in making the diagnosis.

**Peptic Ulcer**

Peptic ulcer disease usually causes pain in the abdomen, but occasionally the pain can be perceived in the chest.

**Characteristics of pain:** The pain of peptic ulcers is often described as a burning or gnawing sensation. It is often relieved by eating a meal, and often made worse by

drinking alcohol, smoking, or ingesting caffeine. It can be accompanied by symptoms of bloating or gas.

**Evaluation:** The evaluation includes a careful medical history and physical examination, which often point to the diagnosis. The ECG is generally normal, and ulcer disease is only rarely confused with heart disease. Endoscopy is the favored method of diagnosis.

**Coronary Artery Spasm**

Angina is caused by ischemia (oxygen starvation) of the cardiac muscle. Coronary artery spasm produces ischemia by narrowing the coronary arteries. The narrowed artery is incapable of delivering the necessary blood volume to the working muscle. Ischemia ensues, and the resultant pain is called angina.

**Characteristics of pain:** Angina can be quite variable, but classically is described as a pressure-like, squeezing, crushing, or tight pain. Some patients with angina deny pain at all, but agree when the term "discomfort" is described to them. The discomfort often radiates to the jaw, shoulders or arms. It can be accompanied by nausea, sweating, shortness of breath, dizziness, weakness, or fatigue. While classical angina is often provoked in a predictable manner by exercise, anger, or a large meal, coronary artery spasm can occur at any time, and often occurs at rest.

**Evaluation:** The evaluation of angina includes a careful medical history and physical examination, which often point to the diagnosis. Testing always includes an ECG, and often a stress test or a cardiac catheterization. Coronary artery spasm should be suspected if the character of the pain and ECG changes are suggestive of angina, but the coronary arteries are normal on catheterization. If spasm is being considered, special steps should be taken during the catheterization to attempt to provoke spasm, thus documenting its presence.

**Prinzmetals Syndrome or Gabriel's Syndrome** is chest pain caused by spasms of coronary arteries. In most patients, there is coronary artery obstruction. In cases where there is obstruction, spasms may occur near the narrowing of the artery. With Prinzmetals Syndrome, chest pain may occur at rest. Some patients experience palpitations. In others chest pain is triggered by exertion. Medications, stents or surgery are treatment options. Once treated, the patient's prognosis is excellent and severe complications such as arrhythmias, heart attack or sudden death are rare.

**Coronary X Syndrome**

Angina is caused by ischemia (oxygen starvation) of the cardiac muscle. In Syndrome X, there are signs of ischemia on stress testing, but the coronary arteries are normal on cardiac catheterization. The cause of Syndrome X is unclear, but most authorities believe that patients with this syndrome have some sort of vascular abnormality involving the small branches of the coronary arteries - branches that are not visualized on cardiac catheterization. Fortunately, the prognosis of patients with Syndrome X appears to be quite good.

**Characteristics of pain:** Angina can be quite variable, but classically is described as a pressure-like, squeezing, crushing, or tight pain. Some patients with angina deny pain at all, but agree when the term "discomfort" is described to them. The discomfort often radiates to the jaw, shoulders or arms. It can be accompanied by nausea, sweating, shortness of breath, dizziness, weakness, or fatigue.

**Evaluation:** The evaluation of angina includes a careful medical history and physical examination, which often point to the diagnosis. Testing always includes an ECG, and often a stress test or a cardiac catheterization. In Syndrome X, the characteristics of the pain and the ECG are strongly suggestive of angina, but the coronary arteries are normal on catheterization. Further, classic coronary artery spasm cannot be provoked.

**Aortic Dissection**

Aortic dissection is a tearing of the lining of the aorta - the body's main artery that comes directly off the heart. It often leads to heart attack or stroke. When aortic dissection occurs acutely, it is often accompanied by sudden severe pain, sometimes occurring in the chest.

**Characteristics of pain:** The pain of aortic dissection is often very sudden and severe in onset. It is often described as a tearing pain, and is usually located beneath the breastbone. When aortic dissection is acute, there is usually no question in the patient's mind that something quite catastrophic has just occurred, and medical help is sought immediately. With subacute aortic dissection the tearing of the aorta occurs more slowly and gradually, and the pain tends to be somewhat less severe.

**Evaluation:** The evaluation is begun by taking medical history and doing a physical examination (which often reveals a new heart murmur). When dissection is suspected, the diagnosis is made by echocardiogram, MRI scan, CT scan or cardiac cath. If the dissection is mistaken as a "'routine" heart attack and thrombolytic drugs are given, the dissection can become suddenly worse and death can ensue.

**Treatment plans for chest pain** are individualized depending on the underlying cause, the presence of coexisting diseases, the age and medical history of the patient, and other factors. Treatment generally involves a multifaceted plan that addresses the cause, eases the pain, decreases the risk of developing serious complications, and helps a person to rest comfortably and live an active life.

If chest pain is suspected to be due to a respiratory, cardiac or serious gastrointestinal condition or disease, treatment generally involves supplemental oxygen. There are a variety of devices that are worn on the face to deliver different concentrations of supplemental oxygen. Oxygen is delivered to the lungs through a breathing tube and mechanical ventilation in cases where chest pain accompanies respiratory insufficiency, respiratory failure or cardiopulmonary arrest.

Chest pain caused by a heart attack is treated with supplemental oxygen, medications, such as nitroglycerin and thrombolytic drugs. Treatment also includes medication to dissolve a clot in the coronary artery that is causing the heart attack or a surgical procedure that opens up the clogged coronary artery (angioplasty).

When chest pain is caused by a viral infection, such as viral pneumonia, the treatment is rest, increased fluids and using a vaporizer. When chest pain is caused by a bacterial infection, such as in bacterial pneumonia, antibiotics are prescribed as well. Serious cases of pneumonia may require hospitalization.

When chest pain is caused by asthma, medications may include corticosteroids and bronchodilators, which are breathed in via inhalers and nebulizers. Other respiratory diseases that cause chest pain may require additional intravenous medications, hospitalization, intensive care and possibly life support measures. Chest

pain caused by the pain of such conditions as rib fractures may be helped by pain medications. Chest pain caused by hyperventilation or and anxiety attack can be treated by breathing into a paper bag and possibly anti-anxiety medication. Chest pain caused by trauma to the chest that results in a collapsed lung condition, such as pneumothorax or hemopneumothorax is treated by re-inflating the lung and draining the blood by insertion of a chest tube. Intensive care is also required. Treatment of chest pain caused by gastrointestinal diseases and conditions varies greatly depending on the specific cause.

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**Tests**

1. Tension pneumothorax has the following features, EXCEPT:
   A. Displacement of the heart on the side opposite pneumothorax.
   B. Distension of the neck veins.
   C. Arterial hypotension.
   D. All of the signs
   E. The blunt percussion sound on the side of the lesion.
2. What is the first aid in a patient with tension pneumothorax?
   A. Tube thoracostomy
   B. Pleural puncture in the $7^{th}$ intercostal space
   C. Pleural puncture in $6^{th}$ intercostal space
   D. Thoracostomy
   E. Bronchoscopy
3. A sharp left-sided chest pain appeared in a 35-year-old patient at the time of intense physical activity. Objectively: the patient is covered with cold sweat; breathing is difficult because of the pain. Auscultation: on the right side the breath sound is vesicular, on the left side - weakened. Tachycardia, heart rate -100. What is your diagnosis?
   A. Spontaneous pneumothorax
   B. Heart attack
   C. Myocardial infarction
   D. Intercostal Neuralgia
   E. Pneumonia
4. 48-years-old woman with ischemic cardiomyopathy have progressive attacks of stenocardia. On coronary angiography: anterior interventricular coronary artery is stenosed approximately 70% for 0, 7 sm. Other arteries are without hemodynamic changes. It is an indication for:
   A. Balloon angioplasty
   B. Coronary artery bypass grafting of one artery
   C. Coronary artery bypass grafting of 2-3 arteries
   D. Transplantation of heart

E. Medicinal therapy

5. The best test for establishing the diagnosis and the degree of myocardial dysfunction is
   A. Serial ECGs
   B. X-ray
   C. Creatine phosphokinase (CPK-MB) fractionation
   D. Echocardiography
   E. Radionuclide angiography
6. What statement about heartburn is not correct?
   A. Heartburn is caused by acidic fluid from the stomach washing up into the esophagus.
   B. The pain of heartburn is often a burning discomfort directly beneath the breastbone.
   C. It is often accompanied by burping, or symptoms of bloating or gas.
   D. Symptoms are often worse after a large meal, or after using tobacco, alcohol, or caffeine.
   E. Symptoms are often reduce after a large meal
7. What statement about pneumothorax is correct?
   A. Chest pain occurs at rest, during sleep or very often with minimal exertion.
   B. It is often accompanied by burping, or symptoms of bloating or gas.
   C. Symptoms are often worse after a large meal, or after using tobacco, alcohol, or caffeine.
   D. Chest pain follows a specific pattern, occurring when someone engages in hard physical activity or experiences extreme emotion.
   E. It is a collection of air or gas in the pleural cavity of the chest between the lung and the chest wall.
8. What statement about unstable angina is correct?
   A. Chest pain occurs at rest, during sleep or very often with minimal exertion.
   B. A condition where the lungs are unable to function properly and maintain the normal processes of oxygen uptake and carbon dioxide removal
   C. The pain is often a burning discomfort directly beneath the breastbone.
   D. Chest pain follows a specific pattern, occurring when someone engages in hard physical activity or experiences extreme emotion.
   E. This is a condition that results from blood accumulating in the pleural cavity.
9. What statement about stable angina is correct?
   A. Chest pain occurs at rest, during sleep or very often with minimal exertion.
   B. A condition where the lungs are unable to function properly and maintain the normal processes of oxygen uptake and carbon dioxide removal
   C. Symptoms often reduce after a large meal.
   D. Chest pain follows a specific pattern, occurring when someone engages in hard physical activity or experiences extreme emotion.
   E. Condition that results from blood accumulating in the pleural cavity.
10. A 27-year-old woman is admitted to the emergency department after a traffic accident. She has shortness of breath with respiratory rate of 60 breaths/min.

Severe muffled breathing on the right side. The first step in managing the patient should be to:

A. Take a chest x-ray
B. Draw arterial blood for blood gas determination
C. Decompression of the right pleural space
D. Perform pericardiocentesis
E. Administer intravenous fluids

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| E | A | A | A | A | E | E | A | D | C |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Surgical anatomy of the organs of chest<br>– Physiology of organs of chest<br>– Pathogenesis of development of complications in chest diseases<br>– Morphological changes in chest diseases<br>– Clinical displays and methods of diagnostics of chest diseases | -To represent the methods of diagnostics of chest diseases as a table<br>-To make the flow diagram of mechanisms of pain in chest diseases. |
| Study:<br>– Types of chest pain.<br>– Types of accesses to the chest.<br>– Tactics at trauma of chest.<br>– Tactics in mixed abdominal trauma with damages of chest. | -To conduct differential diagnosis with the damages of organs of chest.<br>-To conduct differential diagnosis of pathologies of the chest. |

## 8.2. Modern methods of diagnosis and surgical treatment of cardiovascular diseases. Surgical aspects of coronary heart disease and its complications. Modern methods of surgical and minimally invasive treatment.

**Overview.**

Acute cardiac infarction has become one of the most widespread reason of death all around the world. In 1990 6.3 million people died because of ischemic heart disease. The age of people who dead differs not only in different countries but inside them. Every year cardiac infarction develops in 900 000 people, nearly 225 000 from them die. Approximately in the half of these cases death comes during the first hour after appearance of symptoms of myocardial [cardiac] infarction, before the time patients can be delivered to the first aid department. Morbidity on the myocardial [cardiac] infarction increases with age both among men and among women, however in all of age groups this index higher among men and persons with less sufficiency. The resulted information allow to talk that modern achievements of clinical medicine in the area of

surgical (angioplasty and aortocoronary shunting) and pharmacological (trombolitics, inhibitors of enzyme) treatment not can radically decide this question, because more than half, but from some data, to 60% people perish from the myocardial [cardiac] infarction during the first hour from the beginning of disease to the medical care receiving. Almost 25% patients can have so less symptoms myocardial [cardiac] infarction, that it isn't noticed by them or their doctors.

**Educational aims:**

- Interrogation and clinical inspection of patients with the coronary (ischemic) heart disease
- To determine the etiologic and pathogenic factors of coronary (ischemic) heart disease.
- To find out the types of coronary (ischemic) heart disease.
- To develop a plan of examination of the patients with coronary (ischemic) heart disease.
- To estimate results of auscultation, ECG, echocardiogram, coronary angiography, laboratory data.
- To make a differential diagnosis, substantiate and formulate a diagnosis of the coronary (ischemic) heart disease.
- To determine the indications for treatment of patients with the coronary (ischemic) heart disease.
- To cure of the patients with the coronary (ischemic) heart disease.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomy and physiological information about heart.
- Classification of coronary (ischemic) heart disease.
- Mechanism of development of coronary (ischemic) heart disease.
- Clinical presentations of coronary (ischemic) heart disease.
- Methods of diagnostics of coronary (ischemic) heart disease.
- Foundations of treatment of coronary (ischemic) heart disease.
- Relief action at infarction.
- Features of surgical interferences at the different coronary (ischemic) heart disease.
- Technique of punction of pericardium.
- Deontological problems of heart replacement. Modern recourses of cardiosurgery at heart transplantation.

**A student must be able to:**

- Collect and estimate the complaints of patient with coronary (ischemic) heart disease, information of anamnesis, to conduct physical research and interpret the results which you have gotten.
- Define the rational quantity of laboratory and instrumental methods of research.
- Do electrocardiography without assistance and interpret the information.

- Interpret correctly the results of auscultation, ECG, echocardiogram, coronary angiography, laboratory data
- Do the closed-chest (cardiac) [external cardiac] massage.
- Do artificial pulmonary ventilation by methods “nose in nose” or “mouth in mouth
- Define the indications to surgery interventions and choice of the operation method for patients with coronary (ischemic) heart disease depending on the stage of heart disease and patient’s state.
- Prescribe post-operative preparation depending on patient’s state.

**Terminology.**

| Term | Definition |
|---|---|
| Coronary (ischemic) heart disease (CHD). | is decreasing or stop of providing myocardium with blood and oxygen, connecting with pathology in a system of coronary arteries |
| Cardiac arrhythmia (dysrhythmia) | is a term for any of a large and heterogeneous group of conditions in which there is abnormal electrical activity in the heart |
| Coronary arteries | is the heart's blood vessels |
| Angina pectoris | is severe chest paindue to ischemia (a lack of blood and hence oxygen supply) of the heart muscle, generally due to obstruction or spasm of the coronary arteries |
| Heart infarction | It is death of heart tissue due blocked coronary arteries |
| Coronary artery bypass grafting | It is forming of roundabout way for flowing of blood to the heart muscle |

**Content.**

***Coronary (ischemic) heart disease (CHD).***

The term CHD was recorded by the group of experts to denote the heart disease that has acute or chronic character and appears because of decreasing or stop of providing myocardium with blood, connecting with pathology in a system of coronary arteries.

**Etiology and pathogenesis.**

The main pathological process in coronary arteries of all patients is atherosclerosis. Other reasons, such as inflammatory sicknesses of coronary arteries (coronatritis) are mentioned more seldom, so they don’t have big pathological meaning. The narrowing of opening in coronary arteries that is caused with spasm as a rule is seen at primary atherosclerotic changes of coronary arteries intimae. From the patholophysialogical point of view CHD appears in usual or stress situations because of discordance between coronary blood circulation and myocardium request and the state of its metabolism. The most dangerous is lingering spasm of coronary arteries that makes acute ischemia of myocardium and its infarction. Steady clinical features of CHD appear when the opening of vessel is reduced to 70%. The disease happens more often with men than women. The most reliable factors of risk are disorder in lipidic

exchange, arterial hypertonic, smoking, heredity, and sex. The uniting of several factors increasing the danger of CHD.

**Classification**

New-York classification is the most popular. It can show general state of patient with this pathology and his reaction on the physical exercise(physical exercise stamina at all heart diseases). For CHD are given the assessment of painful syndrome(stable and instable angina) and anatomotopographical data of affection( place, grade and size of affective coronary vessels, sector cardiac beat and so on).

**Classification of ischemic heart disease (ICD-10)**

ISCHEMIC HEART DISEASE (I20-I25)

Included: with mention about hypertensia (I10-I15)

I20 Stenocardia [pectoral frog]

I20.0 Unstable stenocardia

I20.1 Stenocardia with the documentarily confirmed spasm

I20.8 Other forms of stenocardia

I20.9 Stenocardia unspecified

I21 Acute cardiac infarction

It is included: heart attack of myocardium, specified as acute or by fixed duration 4 weeks (28 days) or less after the appearanse of acute beginning. It is included: cardiac infarction:

- happened in the past (I25.2)
- subsequent (I22.-)
- specified as chronic or by duration of more than 4 weeks (more than 28 days) from the beginning (I25.8)
- some current complications after the acute cardiac disease (I23.-)

I21.0 Acute transmural cardiac infarction of front wall of myocardium

I21.1 Acute transmural cardiac infarction of lower wall of myocardium

I21.2 Acute transmuralheart cardiac infarction of other specified localizations I21.3 Acute transmural heart attack of myocardium of the unspecified localization

I21.4 Acute subendocardiac heart attack of myocardium

I21.9 Acute cardiac infarction unspecified

I22 Repeated cardiac infarction

Included: the reccurent cardiac infarction

Eliminated: cardiac infarction, specified as chronic or with the set duration more than 4 weeks (more than 28 days) from the beginning (I25.8)

I22.0 Repeated cardiac infarction of front wall of myocardium

I22.1 Repeated cardiac infarction of lower wall of myocardium

I22.8 Repeated cardiac infarction of myocardium of other specified localization

I22.9 Repeated cardiac infarction of myocardium of the unspecified localization

I23 Some current complications of acute cardiac infarction

Eliminated: listed states: - not specified as current complications of acute cardiac infarction (I31.-, I51.-)

- accompanying acute cardiac infarction (I21-I22)

I23.0 Hemopericardium as nearest complication of acute cardiac infarction

I23.1 Defect ofinteratrial septum as current complication of acute cardiac infarction

I23.2 Defect of interventicular septum as current complication of acute cardiac infarction

I23.3 Breaking of cardiac wall without hemopericardium as current complication of acute cardiac infarction

Eliminated: with hemopericardium (I23.0)

I23.4 Breaking of tendinous chord as current complication of acute cardiac infarction

I23.5 Breaking of papillary muscle as current complication of acute cardiac infarction

I23.6 Thrombosis of auricle, atrial appendage and ventricle of heart as current complication of acute cardiac infarction

I23.8 Other current complications of acute cardiac infarction

I24 Other forms of acute ischemic heart disease

Eliminated: transient ischemic heart disease of new-born (P29.4)

stenocardia (I20.-)

I24.0 Coronal thrombosis which does not bring to the heart attack over

Eliminated: a coronal thrombosis that is chronic or by the set duration more than 4 weeks (more than 28 days) from the beginning (I25.8)

I24.1 Syndrome of Dressler

I24.8 Other forms of acute ischemic heart disease

I24.9 Acute ischemic heart disease is unspecified

Eliminated: ischemic heart disease(chronic) (I25.9)

I25 Chronic ischemic heart disease

Eliminated: cardiovascular disease (I51.6)

I25.0 is Atherosclerotic cardiovascular disease, described in this way

I25.1 Atherosclerotic heart disease

I25.2 Heart attack of myocardium occured in the past

I25.3Aneurism of heart

I25.4 Aneurism of coronal artery

Eliminated: innate coronal (arteries) aneurysm (Q24.5)

I25.5 Ischemic cardiomiopathy

I25.6 Asymptomic ischemic disease

I25.8 Other forms of chronic ischemic illness of heart

I25.9 Chronic ischemic heart disease is unspecified

**Risk Factors**

Although the characteristics, locations, and severity of lesions in each person can vary, a number of established risk factors appear to predispose to atherosclerosis (4). These include advanced age, genetic predisposition, male sex, hypertension, diabetes mellitus, hyperlipidemia, and cigarette smoking. The presence of one factor increases the likelihood that the disease will develop at an earlier age, and the presence of more than one risk factor accelerates the process even further. The association of aging with the development of atherosclerotic coronary disease is complex; many of the other risk factors, such as hypertension, hyperglycemia, and hyperlipidemia, are also associated

with aging. Genetic factors play a major role, with direct effects on vascular endothelial biology and arterial wall structure. Indirectly, genetic factors predispose patients to risk factors with a genetic basis, such as hypertension, hyperlipidemia, and diabetes. Male sex is a well-documented major risk for the development of coronary disease. Men are three times more likely than women to have coronary disease, and angina or MI requiring treatment bypass surgery occurs 10 years earlier in affected men than in women.

**Prevention**

Angina pectoris and MI are late manifestations of coronary artery disease. Because atherosclerosis, as evidenced by fatty streaks and early complicated lesions, been found in men as early as the second decade of life, primary prevention of this disease must begin early. The importance of understanding the risk factors for coronary disease and eliminating or modifying those that can be controlled cannot be overemphasized.

**Clinical Presentation**

The clinical manifestations of ischemic heart disease result from an imbalance between coronary arterial blood flow, myocardial oxygen demands, and the capacity the blood to transport oxygen. Atherosclerotic disease directly compromises coronary blood flow. When significant coronary obstructive disease is present, any of three interrelated ischemic clinical syndromes can result—angina pectoris, MI, and ischemic cardiomyopathy. The clinical presentation of coronary artery disease can take many forms. As many as 25% of patients who have positive results on exercise testing because of coronary occlusive disease have no clinical symptoms of typical angina pectoris. Similarly, some acute MIs are silent; patients may have electrocardiographic (ECG) or other evidence of past myocardial injury but no prior history of a clinical syndrome consistent with MI. In some patients, sudden death is the first and only manifestation of ischemic heart disease.

**Symptoms of Coronary Artery Disease**

Symptomatic angina pectoris is the classic presentation of coronary artery disease. The typical description of angina is pressure or heaviness felt in the middle of chest, sometimes radiating to the left shoulder and down the left arm. Patients typically clench their fists in the middle of the chest as they describe this discomfort.

Other, less typical syndromes may signal the presence of significant coronary obstruction and myocardial ischemia. Patients may complain of abdominal pain, nausea, or belching. Other symptoms include back pain or pain in one or both shoulders, jaw pain, or hand heaviness or numbness. Stable angina pectoris is brought on by reproducible increases in myocardial demand for oxygen. Patients report that certain levels of activity, emotional stress, or excitement can trigger angina, is promptly relieved by rest or relaxation.

The clinical presentations of patients with angina pectoris, therefore, vary considerably. The diagnosis of myocardial ischemia is suggested by the presence of angina pectoris but requires documentation of ECG changes of ischemia during chest pain or during exercise testing. The differential diagnosis of angina includes esophagitis secondary to gastrointestinal reflux, peptic ulcer disease, biliary colic, visceral arterial ischemia, pericarditis, pleurisy, thoracic aortic dissection, and many

musculoskeletal disorders. Furthermore, so-called angina equivalents develop in some patients with the onset of myocardial ischemia. These include shortness breath caused by sudden reductions in ventricular contractility and compliance. Other patients have episodes of silent or asymptomatic myocardial ischemia, documented only by continuous ECG monitoring.

In unstable angina, these symptoms may occur at rest or when the patient is sleeping, and myocardial ischemia typically develops without demonstrable changes in myocardial oxygen demand. In these cases, the *supply* of blood to the myocardium may be so marginal that spontaneous coronary reactivity alone may lead to symptoms. The term *unstable angina* also is applied to patients with new-onset angina pectoris or a marked increase in the frequency or severity of episodes of angina pectoris after a stable period.

A less typical form of angina is Prinzmetal's or variant angina. This type of angina occurs at rest or during sleep. It is thought to result from coronary arterial spasm. Such spasm may be mediated by the autonomic nervous system or by local vasoconstrictive agents. It may also result from smooth-muscle irritation or contraction caused by adjacent plaques. Spasm is almost always associated with underlying fixed atherosclerotic disease. Patients may have ST-segment elevation, rather than the more typical ST-segment depression that occurs during episodes of classic angina.

Physicians often grade angina according to the Canadian Heart Association scheme. Class I patients do not have symptoms. Class II patients have angina on significant exertion. Class III patients have angina on mild exertion, and class IV patients have symptoms at rest. A similar classification from the New York Heart Association is used to describe the severity of heart failure. Patients in New York Heart Association class I have no symptoms of heart failure. Class II patients have symptoms on significant exertion. Class III patients have symptoms on mild exertion, such as during normal daily activities, and class IV patients have symptoms at rest.

**Physical Examination Findings**

Usually, no signs of coronary artery disease are detected during the physical examination, but evidence of associated conditions may be found. Peripheral vascular disease may be manifested by a loss of pulses or the presence of bruits in the carotid arteries, abdomen, or femoral arteries. Other signs, such as ocular xanthomas or hypertensive retinal changes, may provide corroborative evidence in patients at risk for coronary disease.

**Diagnostic Studies**

Laboratory studies may be useful for detecting cardiac risk factors, such as diabetes mellitus, hyperlipidemia, or hyperthyroidism. Anemia in the presence of subcritical or borderline coronary obstruction may precipitate angina; myocardial ischemia results from the reduced oxygen-carrying capacity of blood.

The ECG pattern is frequently normal but may reveal evidence of old MI. Typically, these changes include Q waves or loss of R-wave progression in the precordial leads. Chronic ST-segment and T-wave changes may be suggestive of underlying coronary disease but are not specific.

Stress testing may be useful for detecting the presence of coronary disease or assessing the functional significance of coronary lesions. In the standard test, a patient

undergoes graded exercises on a treadmill with ECG monitoring. If signs or symptoms of angina pectoris develop in association with typical ischemic ECG changes, the test result is considered positive. The most diagnostic ECG changes are downward sloping ST-segment depressions. The accuracy of the test is reduced when the patient has underlying ECG abnormalities. Specificity may be improved if the test is combined with the administration of thallium. Thallium is a radioactive isotope that is distributed intracellularly, like potassium. When thallium is injected during exercise, if coronary ischemia develops, the involved area of myocardium fails to take up thallium and a defect is apparent on a myocardial scan. As the patient recovers from exercise and the ischemia is relieved, the previous defect fills in. In patients who cannot exercise, thallium imaging can be performed after the administration of dipyridamole. Dipyridamole, a coronary vasodilator, may reveal areas of relative underperfusion, and a thallium defect appears on scanning, as with exercise testing. When exercise or the administration of dipyridamole is considered unsafe, a rest-rest thallium myocardial scan may reveal evidence of borderline regional myocardial perfusion. In this test, scanning is performed early after injection with thallium and again several hours later. A defect noted on the early scan that fills in later is considered a sign of significant coronary obstruction. A defect that never fills in on thallium scanning is a sign of irreversibly scarred, nonviable myocardium.

Coronary arteriography, which is an invasive diagnostic procedure, is the only way to make a definitive diagnosis of significant coronary obstruction. Coronary arteriography is indicated for patients with atypical presentations and borderline or normal stress test results in whom a definitive diagnosis of coronary artery disease is needed. When classic anginal symptoms and ECG changes make the diagnosis of coronary disease fairly certain, patients should not undergo coronary angiography unless they are refractory to medical therapy or are candidates for revascularization. Regardless of symptoms, patients suspected of having severe coronary artery disease, such as stenosis of the left main coronary artery or severe proximal three-vessel coronary disease, should undergo coronary arteriography to document their condition because of the survival benefits that accrue with revascularization. Diagnostic coronary arteriography is also indicated when cardiac surgery is being planned for patients with other cardiac disease, such as valvular heart disease, in whom concomitant coronary disease is suspected. Examples include patients with aortic stenosis who have angina as part of the presentation. Patients with valvular heart disease who do not have angina but nonetheless have risk factors for coronary disease should also undergo angiography before surgery. These include men older than 45 to 50 years with one or more risk factors for coronary disease.

**Medical Management**

The medical management of coronary artery disease includes the identification and reduction of controllable risk factors. Obviously, patients can do little about a genetic predisposition for the development of coronary obstructions. Control of risk factors by weight reduction, smoking cessation, blood pressure control, and limitation of dietary fats is sensible. Patients with hyperthyroidism or anemia, which may exacerbate anginal symptoms, should have these underlying conditions corrected.

The goal of all therapy for angina pectoris is to decrease the imbalance between the myocardial oxygen supply and demand. Most of the medications that are useful in angina pectoris are more effective in reducing myocardial oxygen demand than in increasing supply.

***Nitroglycerin**,* one of the most commonly used agents, primarily dilates venous capacitance vessels, but at higher doses, it may also cause systemic arterial dilation. Although nitrate compounds do not appear to increase coronary blood flow in the normal heart, these drugs may dilate the coronary arterioles to some extent, so that coronary collateral blood flow improves in patients with extensive atherosclerotic obstructive disease. The primary benefit of nitrates, however, appears to be that they reduce myocardial oxygen demand by reducing ventricular work. This is the consequence of a reduction in systemic vascular resistance and dilation of venous capacitance vessels, which lowers ventricular filling pressures, ventricular wall stress or tension, and contractile work.

**b-*Adrenergic blockers*** also reduce myocardial oxygen demand by decreasing both cardiac contractility and heart rate. These agents may also reduce blood pressure and systemic vascular resistance, and so further reduce the work of the heart.

***Calcium channel blockers**,* such as nifedipine and diltiazem, have more complex cardiac and vascular effects; these include a reduction in ventricular contractility, variable degrees of vasodilation, and possibly a direct protection of myocytes when these cells become hypoxic. Calcium channel blockers may be particularly effective in patients with a component of coronary vasospastic disease.

***ACUTE MYOCARDIAL INFARCTION***

Acute MI is the direct result of an interruption in the blood supply to the myocardium. It is not the result of increased myocardial oxygen demand, but rather of loss of oxygen supply. It usually occurs after a coronary artery thrombosis at the site of a significant stenosis over a complicated plaque. The clot may form as a result of plaque rupture or hemorrhage that incites thrombus formation, or it may be secondary to coronary spasm, which further reduces luminal diameter, markedly decreases flow, and leads to thrombosis. Although the acute event associated with MI is thrombosis, studies in which cardiac catheterization was used have shown that about 20% to 30% of culprit coronary arteries are patent again within a few days of infarction. This is more common in nontransmural than in transmural MIs. One major determinant of the prognosis after acute MI is the amount of ventricular myocardium that undergoes necrosis. For post-MI patients with ejection fractions of more than 50%, the 3-year survival is nearly 90%, but when the ejection fraction falls below 37%, the 3-year survival rate is only 50%. The loss of 25% of the ventricular myocardium leads to symptomatic cardiac dysfunction, whereas the acute loss of more than 40% is frequently associated with cardiogenic shock and death. Efforts to treat patients who are experiencing MI are therefore focused on decreasing myocardial loss by improving flow to the area at risk as quickly as possible. Interestingly, collateral vessels, although unable to meet myocardial oxygen requirements completely, may supply enough blood to limit markedly the amount of myocardium lost. Thus, although well-developed collaterals may not prevent demand-induced angina, they may significantly diminish the loss of myocardium after an acute coronary occlusion.

**Presentation**

Pain is the most common presenting complaint in patients with MI. It is deep, visceral, and frequently described as heavy or crushing. However, pain is by no means universally present, and 20% to 25% of patients (most often diabetic or elderly patients) do not have symptoms. The combination of substernal chest pain lasting for more than 20 to 30 minutes and diaphoresis is strongly suggestive of MI. Interestingly, anterior MIs (usually involving the LAD coronary artery) result in sympathetic hyperactivity, with tachycardia and hypertension, whereas inferior MIs (involving the right coronary artery) frequently result in parasympathetic activity, with bradycardia and hypotension.

**Diagnosis**

The classic ECG picture of an acute MI is the development of Q waves and elevated, coved ST segments in leads reflecting the affected area.

Clinicians frequently characterize MIs by the associated ECG changes. Transmural infarctions usually cause Q waves, whereas subendocardial or nontransmural infarctions are characterized by transient ST-segment changes and evolving T-wave inversion, but not the development of Q waves. MIs are frequently referred to by these ECG changes and are called either *Q-wave* (transmural) or *non-Q-wave* (nontransmural or subendocardial) *infarctions*.

**Medical Management**

During the early phase of MI, it may not be clear whether the patient has unstable preinfarction angina or whether the symptoms indicate a process leading to irreversible myocardial injury. The ECG may be unrevealing, and cardiac isoenzymes may be unavailable. In this situation, oxygen should be administered, heart rhythm should be monitored, and lidocaine should be given to prevent ventricular fibrillation if warning arrhythmias occur. *Early evolving MI* is the term used to describe the condition of patients within 4 to 6 hours after the onset of continued chest pain. This state is important to recognize because ischemic myocardium may still be salvaged before irreversible necrosis develops.

The goal of initial treatment should be to control pain, most frequently with intravenous morphine. Reducing anxiety and pain may have a significant therapeutic effect by decreasing myocardial oxygen demand and limiting infarct size. Intravenous nitroglycerin, begun at a low dose of 0.2 mg/kg per minute to prevent the side effects of hypotension and headache, may diminish infarct size, prevent sudden death, and reduce the likelihood of congestive heart failure. The use of beta blockers is not uniformly agreed on, although they too have been shown to limit infarct size and decrease early mortality. Hypotension and bradyarrhythmias occur more frequently with beta blockers than with intravenous nitroglycerin. Giving them to patients with acute MI who have increased sympathetic tone, however, is probably a safe and beneficial practice. Unlike beta blockers, calcium channel blockers are of little benefit in the setting of acute MI.

Thrombolytic agents convert plasminogen to plasmin, a powerful thrombolysin. It was hypothesized that administration of thrombolytic agents would lead to the dissolution of coronary thrombi and reverse the process that leads to MI. In the late 1970s, a European trial of one thrombolytic agent, streptokinase, revealed a significant benefit when the drug was given within 12 hours of acute MI. Thrombolytic trials in

the 1980s involving thousands of patients established the benefit of this approach, showing that thrombolysis reopens acutely occluded coronary arteries in most cases, restoring flow and reducing mortality.

Although initial thrombolytic trials involved intracoronary administration of the drugs, the cumbersome necessity for emergency cardiac catheterization led to investigations of systemic intravenous administration, which allows virtually immediate therapy in the setting of acute MI. Three intravenous thrombolytic agents have been approved by the Food and Drug Administration: streptokinase, recombinant tissue-type plasminogen activator (rTPA), and anisoylated plasminogen streptokinase activator complex (APSAC). The most widely used is streptokinase, which has been effective in several large trials and is inexpensive. APSAC was developed to enable treating physicians to administer intravenous therapy as a bolus within a few minutes, with the effect maintained for a few hours, rather than as a continuous intravenous infusion, which is necessary with streptokinase and rTPA. However, results are not significantly better than with the other two drugs; furthermore, APSAC is expensive, and its prolonged half-life and thrombolytic effect can be a significant drawback rather than a benefit. Recombinant DNA techniques are used to produce rTPA, which is significantly more expensive than streptokinase. Although it generates less of a systemic fibrinolytic effect than either streptokinase or APSAC, its patency rates are higher.

Systemic intravenous thrombolytic therapy unquestionably decreases morbidity and mortality after MI. The earlier the treatment, the greater the impact, with the greatest benefit accruing in patients treated within 1 to 2 hours after the onset of symptoms. Furthermore, morbidity is decreased secondary to a reduction in arrhythmias and failure of ventricular power. Heparin and antiplatelet drugs such as aspirin provide an added benefit when combined with thrombolytic therapy, particularly in the case of rTPA, which has a short half-life and little antithrombin effect because it does not generate excessive amounts of fibrin degradation products. Complications of thrombolytic therapy include allergic reactions in patients exposed to streptococci or streptokinase in the previous year; reactions occur in fewer than 2% of patients. Hemorrhage is a major problem with all lytic agents, commonly developing at a site of vascular access. Stroke occurs in fewer than 1% of patients but may be catastrophic because of its hemorrhagic nature. Bleeding and stroke occur most frequently in elderly, female, hypertensive, and small patients.

**Indications for Mechanical Intervention**

The use of thrombolytic therapy with early recanalization of the culprit vessel responsible for the MI has had a tremendous impact on the treatment and prognosis of patients experiencing acute MI. The issue then becomes whether anything more need be done acutely; despite reperfusion, significant residual stenosis remain. The Thrombolysis in Myocardial Infarction phase II trial compared elective catheterization and percutaneous transluminal coronary angioplasty (PTCA) within the first 2 days of lytic therapy for MI versus cardiac catheterization and PTCA only if ischemia developed later in the hospital course ( 15). The more invasive approach failed to provide a benefit with respect to early or late mortality and, in fact, increased risk significantly. Based on the results of this and other trials, cardiac catheterization and

PTCA should be withheld in most patients who have no symptoms after thrombolytic therapy for an acute MI. A more invasive approach is justified in patients who exhibit residual ischemia during their hospital stay, either during convalescence or at a predischarge exercise stress test. PTCA may be appropriate, but if cardiac catheterization shows coronary artery disease in multiple vessels or anatomy more suitable for bypass than for PTCA, surgery should be carried out. The early and long-term results in patients operated on within 30 days of acute MI are excellent.

***Cardiogenic Shock***

The development of cardiogenic shock after MI is uncommon. In the multicenter investigation for the limitation of infarct size, cardiogenic shock developed in only 60 of 845 patients with acute MI (18). That group had a 65% mortality rate, whereas in the group in which shock did not develop, the mortality rate was only 4%. Infarct extension occurred in 23% of the shock group, and in 7% of the group without shock. More importantly, in 50% of patients, shock developed more than 24 hours after admission. Evaluation of these patients revealed that age above 65 years, ejection fraction below 35% on admission, a large MI as indicated by the magnitude of the CK-MB leak, a history of diabetes mellitus, and a history of previous MI are all risk factors for the development of shock. When three of these risk factors were present, the in-hospital mortality rate was 18%; when all five risk factors were present, the in-hospital mortality rate was 55%. Animal studies have shown that even in the face of prolonged regional myocardial ischemia, intervention with emergency revascularization may decrease the amount of damage sustained by the myocardium. These studies have focused on ways to decrease energy expenditure during early reperfusion and ways to tailor the initial reperfusate so as to decrease cellular swelling, provide intermediary cellular metabolic substrates, and decrease oxidant injury. In this way, myocardial damage resulting from an ischemic insult can be drastically reduced. A prospective study has evaluated the effect on mortality of emergency coronary bypass surgery in patients in cardiogenic shock after MI. Emergency coronary bypass was performed on 80 consecutive patients in cardiogenic shock who were being maintained on vasopressors and intraaortic balloon pumps after MI. When surgery was performed within 18 hours of the onset of shock, the mortality rate was 7%; when surgery was performed after 18 hours, the mortality rate was 31%. This represents a definite improvement over the results of medical therapy (65% mortality) for this severe complication of MI. In centers capable of performing surgery of this kind, it may be the ideal approach to patients in shock after MI. These results, which have not been duplicated by other institutions, must be viewed as preliminary.

***Ventricular Septal Defect***

Ventricular septal defects occur in about 2% of patients after MI. In general, this complication develops at a time when the myocardium is at its weakest, about 3 to 5 days after MI. It is more common in anterior than in posterior MIs, and with medical treatment the associated mortality rate is more than 90%. At greatest risk for the development of this complication are elderly hypertensive women with transmural infarction. Clinically, hypotension develops with congestive heart failure. Emergency cardiac catheterization reveals an oxygen step-up in the right ventricle, indicating a left-to-right shunt. Medical therapy involves decreasing the afterload as much as

possible; an intraaortic balloon pump is invariably used, in addition to vasodilator therapy if possible. The preload is optimized, and surgery should be performed immediately. Previous approaches involved the stabilization of patients for a prolonged period in the hope that the infarcted area of myocardium would become firmer and hold sutures better. During the 3 weeks that were generally allowed for this process, however, irreversible failure of multiple organ systems frequently developed as a result of shock and sepsis. Early operation before complications occur appears to carry a much better survival rate. Surgical opinion now favors early intervention for this complication.

***Acute Mitral Regurgitation***

Papillary muscle rupture with acute mitral regurgitation occurs infrequently, in fewer than 2% of patients. Like ventricular septal defect, it develops between the third and fifth days, when infarcted myocardium is at its weakest. Posterioinferior MIs lead to this complication more frequently than anterior infarctions, almost certainly because the circumflex artery and PDA provide the most crucial blood supply to the papillary muscles. Clinically, this complication can present with signs and symptoms similar to those of a ventricular septal defect. A new murmur and symptoms of congestive heart failure with hypotension develop. The pulmonary capillary wedge pressure tracing, however, shows prominent V waves, and no right ventricular oxygen step-up occurs. Immediate medical therapy involves decreasing the afterload with an intraaortic balloon pump. Surgery, although it poses an added risk, leads to a better survival than continued medical therapy and decreases the mortality from more than 90% to less than 50%. Evidence has shown that if total mitral valve excision can be avoided and all or part of the subvalvular mitral apparatus saved, the mortality rate can be decreased even further, from 20% with mitral valve replacement to 5% if the mitral valve apparatus is preserved with either repair or replacement. Long-term survival is also improved. In one series, the 4-year survival rate was 89% in the group of patients in whom the mitral apparatus was conserved; it was 59% in the group of patients who underwent mitral valve replacement with total excision of the native valve.

***Free Wall Rupture***

Ventricular free wall rupture after MI occurs also at a time when the myocardium is at its weakest, between the third and sixth days after infarction. The incidence is not well known, but the medical mortality rate is exceedingly high (> than 90%). The benefits of surgical intervention are undocumented. A variety of case reports cite the dramatic rescue of some patients, but circumstances must be ideal. The free wall rupture must be small and contained, so that time is available for diagnosis and operative intervention. Most commonly, free wall rupture leads to death. In some cases, it is contained and may go unrecognized until a pseudoaneurysm develops, which is diagnosed at a later date.

## MECHANICAL INTERVENTIONS

### Catheter-based Coronary Revascularization

Percutaneous transluminal coronary angioplasty is a cardiac catheterization technique designed to reduce the degree of myocardial obstruction and improve regional coronary blood flow. Under fluoroscopic guidance, a catheter is directed into the coronary artery to be treated. A guide wire is then placed across the obstructing

lesion. A balloon catheter is passed over the guide wire and the balloon positioned in the midportion of the obstructing lesion. Under fluoroscopic control, the balloon is inflated to a pressure of to 10 atmospheres for 20 to 60 seconds to reduce the degree of coronary obstruction. Balloon inflation may be repeated several times. It is unclear whether the beneficial effect of this treatment is compression or fracture of the plaque or fracture of the more pliable part of the coronary vessel circumference. After the balloon catheter is withdrawn, coronary angiography is undertaken immediately to assess the degree of dilation and to look for dilation-related complications, such as arterial dissection or acute thrombosis.

In addition to transluminal angioplasty, several other techniques have been developed that can be applied in percutaneous catheter-based systems. These include the placement of intracoronary stents, which are wire mesh cylinders similar in design to the stents placed in other locations, including stenotic major vessels. The systems have been miniaturized so that they can be deployed in the coronary system. The results of initial, nonrandomized studies are encouraging in regard to rates of acute thrombosis and stent failure. Stents appear to be more effective for treating coronary dissections and abrupt vessel closures in the catheterization laboratory.

On a long-term basis, the rates of restenosis appear to be lower with stents than with balloon angioplasty. Their long-term benefits are being evaluated. Atherectomy devices are useful in severely calcified coronary lesions in which inflation of a balloon is not effective. These are similar to high-speed rotating drill bits and literally drill a hole through obstructed and calcified coronary lesions over a guide wire. Multiple lasers have been developed for use in coronary revascularization. The results have been extremely varied to date, and as of yet, no laser has been accepted as a standard of care.

**Indications**

The indications for PTCA are the same as those for coronary artery bypass surgery, the main alternative revascularization technique. Patients with intractable symptoms and those with proximal coronary stenosis that place a large amount of myocardium at risk are potential candidates for angioplasty. The ideal lesion for angioplasty is a symmetric focal stenosis in an epicardial vessel. Long, asymmetric stenosis or those adjacent to bends in the artery or branch points are less likely to be treated successfully. In general, PTCA is contraindicated if significant disease is present in the left main coronary artery, the target coronary artery is less then 2 mm in luminal diameter, multiple significant obstructive lesions are present in the same artery, or the obstructive lesions are complex, such as those involving or straddling arterial bifurcations.

**Coronary Artery Bypass Surgery**

Coronary artery bypass grafting (CABG) is among the most commonly applied major surgical operations in the United States, with more than 250,000 procedures performed yearly. The goals of CABG are identical to the goals of medical treatment and PTCA—to treat ischemic heart disease by relieving the imbalance of myocardial oxygen supply and demand.

**Indications**

Patients are said to have *single-, double-*, or *triple-vessel* disease if significant atherosclerotic narrowing is present in one, two, or all three of the major arteries (i.e.,

LAD, circumflex, and right coronary arteries). In general, data from clinical trials and retrospective studies suggest that as the number of diseased major coronary arterial segments increases, the survival benefit of surgical therapy over medical therapy alone becomes greater.

Another well-accepted indication for CABG is the presence of significant stenosis of the left main coronary artery

Above all, the most common indication for CABG continues to be the relief of disabling angina refractory to medical therapy. Bypass surgery reduces or eliminates angina in more than 90% of patients, and those patients with the most severe anginal syndromes derive the greatest benefit.

In general, the occurrence of unstable (or crescendo) angina suggests that the patient is at risk for MI and death. These patients require aggressive medical therapy, including nitrates, b-adrenergic blockers, and calcium antagonists, in addition to heparin anticoagulation to forestall coronary arterial thrombosis. If the patient continues to experience unstable or rest angina despite maximal medical treatment, urgent coronary angiography is indicated in preparation for PTCA or surgery.

Emergency CABG is indicated as soon as it is apparent that an acute coronary occlusion has developed, an event heralded by the onset of chest pain, ECG changes, and often hemodynamic instability. It is usually possible to verify the presence and nature of the acute coronary occlusion by immediate repeated coronary angiography, which allows the diagnosis to be confirmed.

In most cases of an evolving MI, the ischemic injury is somewhat attenuated and hemodynamic stability is better if intraaortic balloon counterpulsation is established promptly in the catheterization laboratory before the patient is transported to the operating room. If severe hemodynamic instability develops despite balloon pump support, portable cardiopulmonary bypass perfusion with femoral arterial and venous cannulation may provide sufficient stabilization so that the patient can be transported to the operating room. In general, these patients should be placed on cardiopulmonary bypass as quickly as possible to initiate cardioplegic arrest and myocardial cooling and prevent further extension of the infarction.

**Standard Surgical Technique**

In coronary artery surgery, the diseased coronary artery is bypassed by creating an alternative conduit to deliver blood beyond the coronary stenosis. Grafts are constructed by making an end-to-side anastomosis to the coronary artery distal to the obstruction. The proximal end of a vein graft is usually sutured end-to-side to the ascending aorta. When the aorta is diseased, the origin of the innominate artery is sometimes used. The vein most commonly used as a graft is the greater saphenous vein, although the lesser saphenous vein is sometimes employed. The cephalic vein from the arm may be used, but its long-term patency is extremely poor.

The use of arterial grafts has increased. The most commonly used arterial graft is the left internal mammary artery (IMA). It is used most often as a pedicle graft, with its origin at the subclavian artery retained. The distal end is anastomosed end-to-side to the coronary artery. The artery most commonly grafted with the left IMA is the left anterior descending coronary artery. When multiple arterial grafts are desired, the right IMA can be used either as a pedicle graft or as a free graft, with the proximal

anastomosis made on the ascending aorta. More limited use has been made of the gastroepiploic artery, the radial artery, and the inferior epigastric artery.

Most patients require multiple-vessel grafting and typically have a combination of vein grafts and a mammary artery graft. The most common site for use of the left internal mammary artery is the left anterior descending artery.

When CABG is performed, if diffuse atherosclerotic changes are present or if the site chosen for a distal anastomosis is heavily diseased, the surgeon may need to perform an endarterectomy to allow for a more reliable graft-to-artery anastomosis. The data regarding the safety and efficacy of coronary endarterectomy are conflicting; endarterectomy sites are more prone to early thrombosis and reocclusion. Endarterectomy of the distal right coronary artery, the most common site for endarterectomy, appears to be safe and well tolerated, in part because the right coronary artery is often already nearly totally occluded.

***Disorders of rhythm of heart.***

All disorders of heart rate are divided into tachyarrythmia and bradyarrhythmia. Bradyarrhythmias include bradycardia, connected with the affection of pacemaker (syndrome of weakness of pacemaker and sino-auricular block), atrioventricular heart block and worsenings of introventricular conductivity.

Sick sinus syndromes (SSS) a complex of symptoms, which includes sine bradiocardia, stop of sine and blocking of synoartrial output.

**Etiology of weakness of pacemaker syndrom and sinoauricular block** of most often are such diseases as miocarditis, atherosclerotic hypoxiya, sometimes a traumatic damage of a pacemaker during cardiac surgery operations. There are also innate anomalies of development of a pacemaker.

**Symptomatic and clinical course**

Symptoms of SSS can be connected with tachycardia, bradycardia or with them both together, but most often is predefined by sudden changes in frequency of cardiac rythm, by a transition from one rhythm to other. The basic clinical display of this disease is the considerable diminishing of frequency of sinoatrial rate with possible stratification of whole syndroms: general weakness, rapid tiredness, dizziness, losing consciousness and others like that (Adams - Stokes disease). On ECG determine sinoatrial or auricle bradicardia.

**Treatment**.

At SSS and sinoauricular block of II–III of degrees the most effective method of treatment is implantation of artificial driver of rhythm. Permanent artificial drivers of rhythm are shown for symptomatic patients. Correlation of symptoms with bradycardia is needed, though it can be heavily, because of irregular nature of arrhythmias. At the intact function of A–V of a pacemaker the implantation of unicameral auricle drivers of rhythm is possible. In cases with oppression of conducting through A–V pacemaker is less than 120 imp. per 1 minute., bicameral pacemaker with fixing of electrodes in an auricle and ventricle is used.

***Atrioventricular blocks*** The sense of this type of block consists of violation of conductivity of impulses from atrium in ventricles. They can be slow or interrupted through the pathologically prolonged refractive period of atrioventricular connection of bundle of Hiss and both Hiss stalks.

**Etiology and pathogenesis of atrioventricular block (A–V block**) can be: 1) coronal atherosclerosis, often in combination with the heart attack of myocardium; 2) idiopathic atherosclerosis with calcenosis boundle of Hiss; 3) rheumatism, miocarditis or myocardial dystrophy; 4) the innitate defect of the explorer system; 5) traumatic damage of heart.

**Classification of atrioventricular block (A–V block**)

For A–V of block of I of degree lengthening of interval of PQ (more than 0,20 s) is typical.

At A–V block of the II degree some of impulses are not conducted from atrium to ventricles. Separate reductions of ventricles fall out hereupon, that disorders the rightness of cardiac rhythm.

At A–V block of Mobits-I a fall of single ventricular complexes is typical after the progressive lengthening of intervals PQ in a few successive reductions of heart.

A–V block of Mobits-II is characterized by the sudden blocking of auricle impulses and fall of single gastric complexes without the previous progressive increase of length of interval PQ. As a result of the complete breaking of conductivity of atrioventricular knot, bundle of Hiss or its stalks at complete A–V block even a single impulse from atrium (sine or ectopic) in ventricles is not conducted.

Blocking of all of supraventricles impulses at complete A–V block results appearance of ectopic replaceable rhythm with a center below places of block that controls reduction of ventricles. Complete atrioventricular dissociation comes as a result of such block, that reduction of atriums and ventricles takes a place, independently from each other. A basic factor that makes hemodynamic disorders is deceleration of ventricular activity with prolonged time of diastolic filling of heart and degree of myocardium affect. At the end of diastole, as a result of large volume of blood, fibers of myocardium overstretch, the end of diastolic pressure grows. Thus systolic pressure of blood rises and violation of blood providing of brain can come.diastole, as a result of large volume of blood, fibers of myocardium overstretches, and end-diastolic pressure grows. Thus systolic pressure of blood rises and violation of blood providing of brain can come.

**Symptomatic and clinical course of atrioventricular block (A–V block**)

Often patients with A–V blocks do not have any clinical displays. Basic subjective signs of disease at this category of patients are the periodic feelings of instantaneous stop of heart, easy tiredness, especially after the physical exercise, feeling of weight in the area of heart, headache, dizziness, sometimes is losing consciousness. A leading symptom here is bradycardia which is more expressed for people in years and in the cases of complete A–V block. Frequency of cardiac reductions for such patients varies from 15 to 40 per 1 minute. It is found out the syndrome of Morgania-Edems-Stoks in 25–60% cases. When a pause between reductions of heart is more than 8 sec or there is fibrillation of ventricles (8–10% cases) because of appearance of brain hypoxia there can be losing consciousness.

**Variants of clinical course and complications at atrioventricular block(A–V block**)

Depending on course, there are distinguished three forms of A–V of blocks.

- Proof (chronic, permanent) form, that most often happens at an atherosclerotic myocardiosclerosis and postmiocardic and traumatic damages.
- Transitional, acute (transient) form which takes a place in the acute stage of heart attack of myocardium, during intoxication by a foxglove and various myocarditises.
- Intermittent form shows the frequent periodic change of complete A–V block into incomplete or transitions them both in a sinus rhythm.

Depending on localization of ectopic hearth of activation, they distinguish the next variants of A–V of block.

- Proximal A–V block with a nodal rhythm. An auricle rhythm is correct, sinus - with frequency 90 per 1 minute, and a ventricular rhythm replaces nodal with frequency 30–40 per 1 minute. Ventricular complexes are nonenlarged (0.09 sec) and undistorted.
- Complete A–V block with idioventicular variable rhythm. Auricle abbreviates under the action of impulses from a pacemaker. Rhythm is satisfactory, with frequency 70 per 1 minute. Gastric rhythm – is independent of auricle, under the action of impulses from ectopic center in the left ventricle, idioventicular, correct, with frequency 12–30 per 1 minute. Gastric complexes are extended to 0.14 s.
- Complete A–V block with wrong activity of ventricles. Wrong reduction of ventricles arises up as a result of idioventicular rhythm which is consistently caused from ectopic centers in ventricles. One ventricular complexes remind the form of the left stalk block, others - block of right stalk of bundle of Hiss.
- Complete A–V block with extrasystolia. Ventricular complexes are extended, deformed, with frequency 30–36 per 1 minute, by the presence of the third extrasystolic reduction which violates the rightness of idioventicular rhythm. In this case there is a large danger of appearance ventricles flashing.

At complete A–V block mainly three types of complications can appear:

- Ectopic hearth in ventricles can decrease the frequency of initial impulses. It causes strong deceleration of ventricular reductions (oligosystole) or asystole with the attacks of Morgania-Edems-Stoks syndrome.
- Often, especially at the heavy ischemia of myocardium, attacks of syndrome of Morgania-Edems-Stoks happen. They are caused by fibrillation of ventricles
- Complete A–V block during its evolution results the decline of minute volume and development of ventricular insufficiency.

The foregoing types of complications make principal reason of deaths of sick people with complete A–V block and require urgent implantation of permanent electrocardioaccelerator.

**Differential diagnostics**

At a differential diagnosis people need to remmember all of types of bradycardia – sinus (weakness of pacemaker), nodal rhythm at the high degree of sinoauricular and atrioventricular block with correlation 2:1, 3:1, and 4:1. The basic diagnostic criterion of complete A–V of block is the permanent uncoordinated activity of atriums and

ventricles with expressed bradicardia (below 40 reductions per 1 minute) which practically does not change after the functional tests.

**Tactic and choice of treatment method at arrhythmia**

One of basic methods of treatment of arrhythmias is electropacing, which can be permanent and temporal.

*The indications for a temporary pacing are:*

- Atrioventricular block of III degree in the patients with myocardiac infarction;
- Urgent treatment of heart attacks;
- Complete atrioventricular block at drug toxicity (e.g., digitalis);
- Cardiosurgery with the use of artificial circulation during 7-10 days, as the prophylaxis of a sudden bradyarrhythmia in a postoperative period

**Methods of pacing**

***Transcutaneous Pacing*** is recommended for the initial stabilization. The procedure is performed by placing two pacing pads on the patient's chest

***Epicardial Pacing***

Temporary epicardial pacing is used during open heart surgery. The electrodes are placed in contact with the outer wall of the epicardium

***Permanent pacing***

Permanent pacing with an implantable pacemaker involves transvenous placement of one or more pacing electrodes within a chamber, or chambers

*Indications to permanent pacing*:

- Acquired, symptomatic, chronic or periodic complete A-V block.
- Complete innate A–V block with considerable bradycardia or symptoms which are caused by bradycardia.
- Symptomatic developed A–V block of the II degree.
- Bifascicular heart block or trifascicular block with A–V block of the II degree Mobits-II type, even without symptoms which are caused by bradycardia.
- Symptomatic (indisposition, cramps, dizzinesses or loss of orientation) sinus bradycardia (connection of symptoms with bradycardia must be necessarily documented).
- Disfunction of pacemaker, which includes the syndrome of bradycardia, tachycardia, sinoatrial blocking and stop of a pacemaker.
- Potentially dangerous for life is ventricular arrhythmia which arises up as a result of bradycardia;
- Asymptomatic acquired complete A-V block (permanent or intermittent) with a gastric rhythm, lower than 40 per 1 minute; asymptomatic A-V block of the II degree;

**The technique of implantation of permanent artificial pacemekers**

At endocardial method they use transvenous introductions of electrodes. Electrodes can be entered by punction or by the way of venesection. Most often v. cephalica, v. subclavia, v. jugularis ext. are used for this purpose. An electrode is inserted in right atrial appendage; venticular - in the apex of right ventricle. Electrodes are set under X-ray control, check up the threshold of stimulation, amplitude of intracardial signal of ECG, resistance of electrodes.

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**Tests**

1. The usual management of cardiac arrest should include all of the following protocols EXCEPT
   A. Immediate resuscitation, as irreversible brain damage will result after 3-4 minutes of diminished perfusion
   B. Establishment of an airway and ventilatory support
   C. Open-chest cardiac massage
   D. Defibrillation, if cardiac arrest is due to ventricular fibrillation
   E. Administration of cardiotonic agents
2. Stenosis coronary artery more then 75% is indication for:
   A. Baloon Angioplasty
   B. Stenting
   C. Coronary artery bypass grafting (CABG)
   D. Conservative therapy
3. Early complication of permanent cardio pacemaker:
   A. Competitive rhythm
   B. Displacement of electrode
   C. Breakage of electrode
   D. Refuse of work of cardiostimulator
   E. Heart attack of myocardium
4. Control of work of cardio pacemaker:
   A. ECG
   B. Sciagraphy
   C. Tomography
   D. X-ray
   E. Ultrasound
5. There are all method of cardiac arrhythmias EXCEPT:
   A. Antiarrhythmic drug
   B. Defibrillation
   C. Coronary artery bypass grafting
   D. Implantation of pacemaker
   E. Electrical cautery
6. For coronary artery bypass grafting use all shunt except:
   A. a.mammaria intern
   B. a. radialis
   C. v. saphena
   D. a. femoralis
   E. a. ulnaris
7. Syndrome of Morgan'I-Edems-Stoks:
   A. Asystole of auricles
   B. Asystole of ventricles
   C. An asequence is on the left fascicle of ventriculonector (fascicle of His)
   D. An asequence is on the right fascicle of ventriculonector (fascicle of His)

8. A 58-year-old man is in cardiogenic shock in the emergency department after sustaining an acute myocardial infarction (MI). An intraaortic balloon pump (IABP) is inserted. Which statement is TRUE about IABP?
   A. Single chamber pacemaker
   B. Cardioversion
   C. Dual chamber pacemaker
   D. Internal cardiac defibrillator (ICD)
   E. Greenfield filter
9. Which of the following statements is true concerning aortocoronary bypass grafting?
   A. It is indicated for crescendo (preinfarction) angina
   B. It is indicated for congestive heart failure
   C. It is not indicated for chronic disabling angina
   D. It is associated with a 10% operative mortality
   E. It is only indicated if significant triple vessel disease is documented ECG
10. The most useful incision in the operating room for patients with penetrating cardiac injury is:
   A. Left anterior thoracotomy
   B. Right anterior thoracotomy
   C. Bilateral anterior thoracotomy
   D. Median sternotomy
   E. Subxyphoid

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| C | C | B | A | C | D | B | E | A | D |

**Materials on the independent teaching of students**

| Basic tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of heart and pericardium<br>– Physiology of heart<br>– Conducting system of heart<br>– interpretation the ECG | - To sketch out the heart<br>-To represent the methods of diagnosis of diseases of heart as a table<br>-To sketch out normal cardiogram and it is at infarction |
| Study:<br>– Types of Coronary artery bypass grafting (CABG).<br>– Types of accesses are to the heart<br>– Tactic at cardiac arrhythmias<br>– Pathogenesis of development of complications after heart-surgery. | -To make indications to transplantation of heart<br>-To make differential diagnosis of cardiac arrhythmias<br>- To know method electrical cautery at cardiac arrhythmias |

## 8.3. Surgical pathology of the lungs and pleura. Principles of diagnosis and treatment.

**Overview.**

The modern human's deteriorating breathing hygiene has increased the disorder of the protecting mechanisms of the respiratory apparatus, increasing the frequency of immunodeficiency states, caused by diabetes, alcoholism and so on. This in turn has resulted in increased infections caused by antibiotic resistant cultures. The entire above mentioned are viewed as reasons that have contributed to the beginning of the infectious decomposition of the lungs. The possibility of developing lung abscesses is very high during pneumonia (2-5%), closed traumas of breast (1, 5-2%) and bullet wounds (1, 5%). In spite of great success in the treatment of these pathologies, lethality in lung abscesses reaches 10%, and in lung gangrene is still also very high – from 40 till 90%. Most often empyema of the pleura complicates the duration of severe pneumonia (5-8%), lung abscess (9-11%), lung gangrene (80-95%). Empyema of pleura is found in 3-5% cases during closed thoracic cage injury, and during sucking chest wound – in 10-15%.

**Educational aims:**

- To understand the effect of ecology, biology and social factors on the development of infectious destruction of lungs.
- According to the materials of the topic to develop the feeling of responsibility for timely diagnosis and revealing of diseases and accuracy of professional actions with the purpose of its prophylaxis, treatment and prevention of complications.

**A student must know:**

- Anatomy and physiology of the skin of different parts of the body.
- Anatomy and physiology of the lungs and pleura.
- Classification of suppurative diseases of the lungs and pleura.
- Up-to-date views on etiology and pathogenesis of lung and pleural diseases.
- Clinical picture of lung and pleura diseases.
- Treatment principles of lung and pleura diseases, statements to different methods of treatment.
- Peculiarities of surgical operations during lung and pleural diseases.
- Technique of executing a pleural puncture.
- Technique of "closed" drainage of pleural cavity.

**A student must be able to:**

- Deduce and estimate a patient's complaints from lungs and pleura diseases, anamnesis data, make physical examination, understand and interpret the results correctly.
- Define efficient content of laboratory and instrumental methods of the investigation.

- Understand and interpret the results of biochemical analysis of blood; exudates investigation, ultrasonic diagnostic, computed tomography, and X-ray.
- Define the indications to operations and other methods of treatment.
- Describe patients' pre-surgical preparation.
- Carry out postoperative period.

**Terminology:**

| Term | Definition |
|---|---|
| Abscessing pneumonia | Multiple destructive foci 0, 3-0, 5 cm in size, within 1-2 segments of lungs, which are not disposed to progression. The destruction of the lung is accompanied by expressed perifocal infiltration of a pulmonary tissue. |
| Lung abscess | Purulent or ichorous destruction of pulmonary tissue within one segment with formation of one or several cavities, filled by pus, and detached from adjacent parenchyma by a pyogenic capsule and expressed perifocal infiltration of surrounding pulmonary tissue. |
| Gangrenous abscess | Purulent, ichorous necrosis of pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma, with the liability to sequester formation. Depending on reactivity of the organism it can transform into a purulent abscess (after the lysis of sequestrations) or gangrene. |
| Lung gangrene | Diffuse purulent, ichorous necrosis of the tissue without the tendency to form defined demarcations with fast dynamics of spreading of necrotic zone and destruction of the parenchyma. It is characterized by grave intoxication, likely to form pleural complications and pulmonary bleeding. There is limited and wide-spread lung gangrene. |
| Pyopneumothorax | Is when a lung abscess bursts into pleural space, which is accompanied by purulent inflammation of pleural membranes with the collapse of lung. |
| Empyema | Pyogenic or suppurative infection of the pleural space (the most common is the exudative type of pleural effusion). |

**Content:**

Acute suppurative diseases of lungs include abscessing pneumonia, acute and gangrenous abscesses, focal and extensive lung gangrene. The agents of purulent pulmonary destruction are anaerobic non-clostridia microorganisms, staphylococci, Gram-negative bacteria, and mixed infection.

The predominant factors which cause the disease:

- disturbances of bronchial patency with the development of atelectasis;
- infectious inflammatory processes in a pulmonary tissue;

- Regional disturbances of blood supply with a further necrosis of areas of pulmonary parenchyma.

**Classification of acute suppurative diseases of the lungs**

*According to pathogenesis:*

- post pneumonic;
- aspirative;
- obturative;
- post traumatic;
- hematogenous or septic;
- lymphogenous;
- Thromboembolic.

*According to the character of purulent process:*

- single purulent abscesses;
- multiple purulent abscesses;
- bilateral purulent abscesses;
- gangrenous abscesses (single, multiple, uni- and bilateral);
- limited gangrene;
- Wide-spread gangrene.

*According to the localization* (with indication of affected segment or lobe)

*According to the stage:*

- 1st stage - necrotic pneumonia;
- 2nd stage - destruction and rejection;
- 3rd stage - cleaning and cicatrization.

*According to the duration of the disease:*

- acute;
- Chronic.

*Complications:*

- pulmonary bleeding;
- pyopneumothorax;
- pleural empyema;
- sepsis;
- Bronchogenic dissemination.

The clinical manifestation of acute purulent destruction of lungs depends on the size of the focus and the character of the destruction, reactivity of the organism, stage of the disease, peculiarities of the drainage of the purulent cavities and complications.

A ***lung abscess*** is a localized area of pulmonary parenchyma necrosis caused by an infectious organism. Tissue destruction results in the formation of a solitary or dominant cavity measuring at least 2cm in diameter. Less often, there may be multiple, smaller cavities (<2 cm). In that case, the infection is typically referred to as a necrotizing pneumonia. An abscess that is present for more than 6 weeks is considered chronic.

**Causes of Lung Abscess**

***I. Primary:***

A. Necrotizing pneumonia
- S. aureus, Klebsiella, Pseudomonas, and Mycobacterium
- Bacteroides, Fusobacterium, Actinomyces
- Entamoeba, Echinococcus

B. Aspiration pneumonia
- Anesthesia
- Stroke
- Drugs or alcohol

C. Esophageal disease (achalasia, Zenker's diverticulum, gastroesophageal reflux)

D. Immunodeficiency
- Cancer (and chemotherapy)
- Diabetes
- Organ transplantation
- Steroid therapy
- Malnutrition

***II. Secondary***

A. Bronchial obstruction:
- Neoplasm
- Foreign body

B. Systemic sepsis:
- Septic pulmonary emboli
- Formation of pulmonary infarct

C. Complications of pulmonary trauma:
- Infection of hematoma or contusion
- Contaminated foreign body or penetrating injury

D. Direct spread from extra parenchyma infection:
- Pleural empyema
- Mediastinal, hepatic, subphrenic abscess

Lung abscess results from lower respiratory tract infection only with organisms that cause necrosis. Organisms enter the respiratory tract by inhalation of aerosolized particles, aspiration of oropharyngeal secretions, or hematogenous spread from distant sites. Direct extension from a contiguous site is less common. Most primary lung abscesses are purulent bacterial infections secondary to aspiration. Risk factors for increased aspiration include states of consciousness, suppression of the cough reflex, esophageal motility disorders, and central neurologic disease (eg, stroke). During aspiration, the composition of the oropharyngeal flora determines the etiologic organisms; those organisms that are most abundant or virulent multiply and emerge as the sole or predominant pathogens. Secondary lung abscesses most often occur distal to obstructive bronchial carcinoma. Infected cysts or bullae are not considered true abscesses. Characteristic pathological features of aspiration pneumonia include alveolar edema and infiltration by inflammatory cells. Due to gravity, foci of infection tend to develop in the subpleural areas of the upper segments of the lower lobes and in the posterior segments of the upper lobes. The right lung is more commonly affected, presumably because of the less acute angle of the right main bronchus. Thus, the right

upper and lower lobes are most commonly affected, followed by the left lower lobe and the right middle lobe.

***Gangrene of the lung*** is the most severe form of extensive purulent destruction of a lung. Absorption of products of putrescent lung disintegration and bacteria toxins stimulates the production of inflammatory mediators and first of all pro-inflammatory cytokines (IL-1, IL-6, TNF, IL-8) and active radicals, which promote expansion of a destruction zone. In this connection the organism reaction on the inflammation accepts system character; control of immune system above occurring processes becomes weaker or lost. It results in acute intoxication of the organism, dysfunction of the vital bodies and threat of sepsis development, multisystem insufficiency or septic shock.

***Bronchiectasis*** is defined as a pathologic and permanent dilation of bronchi. This condition may be localized to certain bronchial segments or it may be diffuse throughout the bronchial tree, typically affecting the medium-sized airways. Overall, this is a rare clinical entity in the United States with a prevalence of less than 1 in 10,000. Development of bronchiectasis can be attributed to either congenital or acquired causes. The principal congenital diseases that lead to bronchiectasis include cystic fibrosis, primary ciliary dyskinesia, and immunoglobulin deficiencies (e.g., selective IgA deficiency). Congenital causes tend to produce a diffuse pattern of bronchial involvement. Acquired causes are categorized broadly as infectious and inflammatory. Adenoviruses and influenza viruses are the predominant childhood viral infections associated with the development of bronchiectasis. Chronic infection with tuberculosis remains an important worldwide cause of bronchiectasis. More significant in the United States is the occurrence of non-tuberculous mycobacterial infections causing bronchiectasis, particularly *Mycobacterium avium* complex. Noninfectious causes of bronchiectasis include inhalation of toxic gases such as ammonia, which results in severe and destructive airway inflammatory responses. Allergic bronchopulmonary aspergillosis, Sjögren's syndrome, and alpha$_1$-antitrypsin deficiency are some additional examples of presumed immunologic disorders that may be accompanied by bronchiectasis. The common pathway shared by all of these causes of bronchiectasis is impairment of airway defenses or deficits in immunologic mechanisms that permit bacterial colonization and establishment of chronic infection. Both the bacterial organisms and the inflammatory cells recruited to thwart the bacteria elaborate proteolytic and oxidative molecules, which progressively destroy the muscular and elastic components of the airway walls; those components are then replaced by fibrous tissue. Thus chronic airway inflammation is the essential pathologic feature of bronchiectasis. The dilated airways are usually filled with thick purulent material; more distal airways are often occluded by secretions or obliterated by fibrous tissue. The vascularity of affected bronchial walls increases, bronchial arteries become hypertrophied, and abnormal anastomoses form between the bronchial and pulmonary arterial circulation.

There are three principal types of bronchiectasis, based on pathologic morphology: cylindrical-uniformly dilated bronchi, varicose—an irregular or beaded pattern of dilated bronchi, and saccular (cystic)-peripheral balloon-type bronchial dilation. The saccular type is the most common after bronchial obstruction or infection.

***Pyopneumothorax*** is the result of a burst lung abscess into the pleural space, which is accompanied by purulent inflammation of pleural membranes with a collapse of lung.

Peripheral location of the purulent focus in pulmonary tissue results in destruction (fusion) of visceral membrane. As a result of, the pus and air penetrate into a pleural space that leads to purulent inflammation of parietal and visceral membranes of pleura. The disorder of a pleural continuity results in lung collapse.

Among other causes of pyopneumothorax are the chest trauma, which results in collapse of lung, infection and purulent inflammation of pleural membranes.

As the basic causes of pyopneumothorax are considered: acute abscess of lung; gangrenous abscess of lung; lung gangrene; purulent lung cyst; abscessing pneumonia; bronchiectatic disease; subphrenic abscess, which has burst into pleural space; injury of esophagus; mediastinitis; chest trauma; operation and diagnostic manipulations on chest organs.

Morphologically pus and air are present in pleural space. In lungs revealed sub-pleural purulent or necrotic foci, which connected with a pleural space through a pleuro-pulmonary fistula. From the outside the zone of disrupture is restricted by perifocal inflammation. In the draining bronchus it is possible to see manifestations of deforming, frequently polypous bronchitis.

***Classification***

**I. According to the etiological factor:**

- Specific.
- Nonspecific.

**II. According to the pathogenic factor**:

- Primary.
- Secondary.

**III. According to the clinical course:**

- Asymptomatic form.
- Mild form.
- Acute form.

**IV. According to extension of the process:**

- Localized pyopneumothorax:
  - parietal;
  - apical;
  - epiphrenic;
  - paramediastinal;
  - Polychamber.
- Subtotal pyopneumothorax.
- Total pyopneumothorax.
- Tension pyopneumothorax.

***Thoracic empyema*** is defined as a purulent pleural effusion. The most common causes are parapneumonia, but postsurgical or post traumatic empyema is also common.

***Classification***

**I. According to the etiological factor:**

- Specific.
- Nonspecific.

**II. According to the pathogenic factor:**

- Primary.
- Secondary.

**III. According to the clinical course:**

- Acute.
- Chronic.

**IV. According to extension of the process:**

- Focal.
- Wide-spread.

**V. According to the presence of lung destruction:**

- Empyema with destruction of pulmonary tissue.
- Empyema without destruction of pulmonary tissue.
- Pyopneumothorax.

**VI. According to communication with environment:**

- Closed pleural empyema;
- Open pleural empyema:
  - bronchopleural fistula;
  - thoracopleural fistula;
  - thoracopleurobronchial fistula;
  - Cribrate lung.

**Manifestation and diagnostics.**

According to the clinical course, there are such variants of the development of purulent diseases of lungs:

- *Favorable course.* The adequate treatment results in prompt positive clinical, roentgenological and laboratory dynamics, and ends by recovery.
- *Non-progressive course. P*oor drainage of the suppurative focus and permanent purulent intoxication results in development of the process into chronic form.
- *Progressing course. This is predetermined by the combination of a series of unfavorable factors like low resistance of the organism, autoimmune aggression, and high virulence of the infecting agent.* It is characterized by diffusion of the zone of necrosis and destruction with transferring in gangrene.
- *Encapsulated process.* Caused by the absence or complete obstruction of the draining bronchus under conditions of satisfactory resistance of the organism.
- *Complicated course.* Mostly is the result of progressive development of the pathological process.

The typical presentation of lung abscess may include fever (>38.9°C), productive cough, leukocytosis (>15,000 cells/ $mm^3$), chills, malaise, weight loss, pleuritic chest pain, fatigue, and dyspnea. Lung abscesses may also present more indolently, with weeks or months of cough, malaise, weight loss, low-grade fever, night sweats,

leukocytosis, and anemia. It usually takes 1 to 2 weeks for aspiration pneumonia to develop cavitation; 40 to 75% of such patients produce foul-smelling, putrid sputum. Severe complications, such as massive hemoptysis, endobronchial spread to other parts of the lung, rupture into the pleural space, and development of pyopneumothorax or septic shock and respiratory failure, are rare in the modern era of antibiotics. The mortality rate is 5 to 10%, except in the presence of immunosuppression, where rates range from 9 to 28%..

At gangrene of lungs a big amount of foul-smelling, foamy, three-layer phlegm that has a purulent - blood character begins to be separated, that shows anaerobic or a mixed kind of infection. The patients often have a repetitive painful cough. In the purulent process, as a rule, pleura are involved, that results the development of saprogenic empyema or pyopneumothorax. During the patient's examination, expressed respiratory insufficiency - anhelation, pallor of skin mucosa, cyanosis- is marked. During percussion a significant zone of reduction of percussion sound above the affected lung is defined, it is replaced by a bandbox sound above a destruction cavity that contains air and liquid. At auscultation a lot of moist rales of various calibers are listened. The clinic of pulmonary gangrene differs by terminal expression of signs. The state of the patients is critical. The patient is adynamic, exhausted, with oedema on legs. Dyspnea at rest, hemodynamic disturbances are evident. Dirty-grey or brown sputum with detritus, pieces of necrotic parenchyma and threads of blood excretes out with the cough up to 1 liter. An early pleural complication, such as pulmonary bleeding, is usual and may be profuse. Often it is associated with vital organ dysfunction and loss of consciousness. The intensive shadow which occupies a considerable area of lungs with visible cavities, that contain sequesters, fluid levels, is roentgenologically revealed. The shadow outline is irregular, but could be well defined if the process is within interlobar sulcus.

***Pulmonary bleeding*** arises suddenly, and associated with coughing out of a foamy, bright-red blood and clots by portions or continuous stream. The most often source of a pulmonary bleeding is the bronchial arteries and vessels of a pulmonary tissue. The clinical manifestation of a pulmonary suppuration is accompanied by dizziness, weakness, and dyspnea and chest pain. The hemodynamic disturbances depend on intensity of the bleeding. The auscultation of lungs reveals the moist rales (aspiration) on both sides. If the pulmonary destruction is present the plain film of the chest shows the localization of the source of bleeding. After hospitalization of the patient with this complication, exclusive information is obtained with a fibrobronchoscopy.

According to the degree, the pulmonary bleedings are classified (V.Struchkov, 1985):

**I degree - hemorrhage up to 300 ml.**

- Single hemoptysis.
- Multiple hemoptysis.

**II degree - hemorrhage up to 700 ml.**

- Single bleeding:
  - with falling of arterial pressure and decreasing of hemoglobin;
  - Without falling of arterial pressure and decreasing of hemoglobin.

- Multiple bleeding:
  - with falling of arterial pressure and decreasing of hemoglobin;
  - without falling of arterial pressure and decreasing of hemoglobin.

**III degree - hemorrhage exceeds 700 ml.**

- Massive bleeding.
- Fulminant, lethal bleeding.

The 1st degree of pulmonary bleeding manifests by coughing out sputum tinged with blood, the hemodynamic disturbance usually absent. The bleeding of 2nd degree is characterized by decreasing of arterial pressure by 20-30 mm Hg, tachycardia up to 100 beats/min and contents of hemoglobin within 60-80 g/1. The bleeding of 3rd degree is accompanied with sharp decreasing of arterial pressure, rapid pulse (more than 100-120 beats/ min), small, sometimes thread pulse, and even its disappearance on peripheral arteries, tachypnea to 40 per 1 min, hemoglobin to 50-60 g/1. Possibly the fulminant course up to the terminal state with prompt failure of cardiac activity and asphyxia by blood.

A daily persistent cough and purulent sputum production are the typical symptoms of bronchiectasis. The quantity of daily sputum production (10 mL to >150 mL) tends to correlate with the disease extent and severity. Often, patients with bronchiectasis may be asymptomatic or have a dry, unproductive cough ("dry bronchiectasis"). These patients are prone to upper lobe involvement. The clinical course is characterized by progressive symptoms and respiratory distress. Increasing dyspnea at rest and with exercise is the result of progressive airway obstruction. Acute exacerbations may be triggered by viral or bacterial pathogens. Hemoptysis may become more frequent as the disease progresses, and bleeding is caused by chronically inflamed, loose airway mucosa. In more advanced stages, massive bleeding may result from erosions of hypertrophied bronchial arteries.

The manifestation of pyopneumothorax depends on the size of the focus of destruction, which influences on the degree of lung collapse, and on amount of purulent content in a pleural space. The pain owing to the burst of destructive focus into pleural space often arises suddenly. The dyspnea occurs as a result of lung collapse owing to leakage of pus and air into pleural space. Its expression is in direct ratio to lung collapse. Therefore, dyspnea at rest is observed in a subtotal and total pyopneumothorax. It sharply amplifies even at minor physical activity. Auxiliary muscles take part in order to force respiration. The expectoration of sputum with ichorous smell is the outcome of destructive process in a pulmonary tissue. Its amount decreases after effusion of pus into pleural space. Hectic fever is caused by enlargement of the area of resorption. The patients are adynamic, flaccid. Some of them are unconsciousness. On objective examination the position of patients is forced, they sit leaning up on the bed (subtotal, total pyopneumothorax). The affected hemithorax takes no part in respiration. On palpation, there is diminished vocal fremitus on the side of lesion. Percussion reveals a sharp shortening of sound over the zone of exudate and bandbox sound above the region of collapsed lung. On auscultation there are no breathing sounds on the affected side. In case of localized pyopneumothorax ~ weakened or sharply weakened sound with a bronchial or amphoric tone.

The clinic of acute pleural empyema depends on extension of the process, reactivity of the organism and presence of complications (pain, dyspnea, cough, intoxication). A highly purulent pleural fluid with a foul odor makes the diagnosis of empyema obvious on visual examination at the bedside. Early, small to moderate turbid pleural effusion in the setting of a pulmonary process may warrant further pleural fluid analysis. Close clinical observation is also necessary to determine whether progression to empyema is occurring. Deteriorating clinical course or a pleural pH of less than 7.20 and a glucose level of less than 40 mg/dL indicates the need for drainage of the fluid. The forced patient's position and restriction of breathing should be considered as outcomes of a pain syndrome. The extension of pleural empyema causes the swelling of thoracic wall, smoothening of intercostal spaces. On palpation - diminished vocal fremitus on the part of lesion. The data of percussion and auscultation depend on the extent of the process and the amount of pus in the pleural space. By percussion over the exudate it is possible to reveal short sound with oblique upper contour. Above the exudate - tympanic sound resulting from consolidation of pulmonary tissue. On auscultation - diminished or absent sound in a great amount of exudate.

The chest radiograph is the primary tool for diagnosing a lung abscess. Its distinguishing feature is a firm mass or mass with a relatively thin-walled cavity. Air-fluid is often present within the abscess, suggesting communication with the tracheobronchial tree. Computed tomography is useful for clarifying the diagnosis when the radiograph is equivocal to help rule out endobronchial obstruction and to identify an associated mass or other pathological abnormalities. Cavitating lung carcinoma is often mistaken for a lung abscess. Other possible differential diagnoses include localized or interlobular empyema, infected lung cysts or bullae, tuberculosis, bronchiectasis, fungal infections, and noninfectious inflammatory conditions (eg, Wegener's granulomatosis).

Roentgenologically in the beginning of lung gangrene within the limits of one share or all lungs the extensive blackout comes to light which is increased every day. At occurrence of the message of a destruction cavity with bronchus on a background of infiltration of pulmonary tissue there are destruction cavities of various size both degree of filling by air and pus. Such complications as emptysis, bleeding, pyopneumotorax or empyema of pleura are the often parts that accompany gangrene of lung. At X-ray research of lungs and computer tomography in the largest cavities sequestration of different size come to light.

Roentgenological manifestations in localized pyopneumothorax not expressed and include horizontal fluid level, margin of partially collapsed lung and minor air in the pleural space.

The predominant roentgenological sign of a focal or wide-spread empyema is the presence of exudate. In localized acute pleural empyema observed a local intensive homogeneous shadow. Roentgenologically according to localization distinguished such types of a focal empyema: apical; paramediastinal; parietal; interlobar; epiphrenic. The wide-spread pleural empyema manifests by intensive homogeneous shadow in a basal parts with oblique upper contour (Damuaso' line). The diaphragmatic dome is failed to observe. The more pus is in pleural space, the higher upper border of the exudate.

The current gold standard for diagnosis is computed tomography of the chest, which provides detailed cross-sectional images of the bronchial architecture. Both mild and severe forms of bronchiectasis are readily demonstrated by this imaging modality. Chest radiography, although less sensitive, can reveal characteristic features of bronchiectasis, such as hyperinflation of the lungs, bronchiectasis cysts, and dilated, thick-walled bronchi that form tram-track patterns radiating from the throat of the lungs. Sputum culture can identify characteristic pathogens, including H. influenzae, S. pneumoniae, and P. aeruginosa. Sputum smears and acid-fast cultures should be performed to determine the presence of nontuberculous mycobacteria, which may be common in this setting. The degree of airway obstruction should be determined by spirometry, which can also assess the course of the disease.

**Bronchoscopy -** The debate over the relative merits of rigid versus fibre optic bronchoscopy has largely been resolved with the recognition that they are complementary, both having advantages and disadvantages. Any experienced bronchoscopist should be familiar with both types of instrument. Rigid bronchoscopy was and still is mainly the province of the thoracic surgeon. Although it can be performed under local anaesthesia, general anaesthesia is felt to be kinder and more appropriate. It retains several advantages over fibre optic bronchoscopy: the large calibre allows easier suction of retained secretions and better control of bleeding, foreign bodies can be removed more safely, and good control of ventilation and airway patency can be maintained. It can also be used to feel the mobility of the trachea and main bronchi, allowing assessment of operability of tumours. Whilst there is general agreement about topical anaesthesia for fibre optic bronchoscopy, the relative risks and benefits from sedation remain the subject of debate. Whatever approach is undertaken, it is essential that the patient be given a careful explanation of the procedure to avoid unnecessary fear. Lignocaine (lidocaine) is the most commonly used topical anaesthetic: there are a number of minor variations in technique used to achieve satisfactory anaesthesia for the nasal and oropharyngeal mucosa, larynx, and tracheobronchial tree. The nasal mucosa is anaesthetized with a 4 per cent spray or lignocaine (lidocaine) gel, the spray also being used for the oropharynx. Anticholinergics are now no longer regarded as necessary premedication for fibre optic bronchoscopy. Their use was largely based on anaesthetic practice for rigid bronchoscopy in order to dry secretions and reduce vasovagal activity, but any apparent benefits are marginal.

Fibre optic bronchoscopy is generally performed with a patient semi recumbent and the operator standing to one side and facing the patient. An alternative is to stand at the head of a supine patient, an approach that maintains the same spatial orientation of the bronchial tree as in rigid bronchoscopy. Insertion of the bronchoscope via the nose is preferred. In negotiating the nasal passages, the bronchoscope should be passed directly backwards and worked through by gentle manipulation, not force. In 5 to 10 per cent of patients the size of the nasal airway is inadequate and the oral route must be used. The bronchoscope should be protected by passing it through a mouth guard gently clasped between the patient's teeth. As the bronchoscope is advanced the nares, naso- and oropharynx, and vocal cords should be inspected and the mobility of the cords assessed during phonation. When entering the trachea, coughing can be

minimized by adequate local analgesia and avoiding contact with the mucosa as much as possible. Secretions can be aspirated, but if vision is impaired by blood or mucus on the distal lens it can usually be cleared by gently wiping the tip of the instrument on the bronchial mucosa.

Full inspection of the bronchial tree requires a methodical approach and detailed knowledge of bronchial anatomy. Individual segmental bronchi cannot be recognized without appreciating the route taken to that particular airway. Practice in manipulating the instrument on a lung model is strongly recommended: descriptions of normal and abnormal appearances are inadequate substitutes for practical experience. Pathological findings can be recognized by the presence of excess secretions, mucosal abnormalities or distortion of the normal anatomy, usually due to extrinsic pressure. Secretions may vary from excess amounts of normal clear mucus found in patients with chronic bronchitis to thick pus in patients with bronchiectasis or lung abscess. Thick, viscid plugs of mucus can be found in patients with asthma. Inflammation may be accompanied by erythema of the bronchial mucosa, which is friable and bleeds easily.

***Complications*** There are no absolute contraindications to fibre optic bronchoscopy. There are, however, increased risks in patients with ischaemic heart disease, respiratory failure, asthma, and bleeding diatheses. Bleeding, especially after biopsy, is the most frequent complication. Haemostasis may be achieved by wedging the bronchoscope *in situ*, and inflatable balloon catheters are also available. Resting arterial oxygen falls by an average of 2.5 kPa during fibre optic bronchoscopy. Adverse consequences are more likely to occur in patients with pre-existing respiratory failure, and hypoxia may be accompanied by ventricular dysrhythmias. Monitoring of oxygen saturation and supplemental oxygen provide appropriate safeguards. Radiological screening can help to reduce the likelihood of a pneumothorax following transbronchial biopsy. This occurs in approx. 5 per cent of procedures but rarely requires specific treatment. The risk of cross-infection is small provided that care is taken in cleaning and disinfecting the bronchoscope. Transmission of *Pseudomonas* and mycobacteria between patients has been reported. Cross-infection with human immune deficiency virus is likely to prove less of a problem but adequate safeguards need to be taken by staff performing the bronchoscopy. Gloves, gown, goggles, and mask should be worn by the bronchoscopist to prevent accidental cross-infection.

**Differential diagnostics between nonspecific inflammatory neoplasms and tuberculosis**

*Symptoms from* ***pulmonary neoplasms*** can be classified according to their etiology. Bronchopulmonary symptoms are those caused by direct tumor effects within the airway or parenchyma, including cough, hemoptysis, wheezing, dyspnea, or pneumonia. Recurrent pneumonia suggests partial bronchial obstruction. Alternatively, pneumonia characterized as a persistent infiltrate indicates complete bronchial obstruction or neoplastic consolidation (e.g. by bronchoalveolar cancer). Intrathoracic symptoms are those created by tumor extension outside the parenchyma. In most cases, these are signs of extensive disease and relative unresectability. Typical intrathoracic symptoms include:

- Hoarseness due to recurrent laryngeal nerve invasion, either directly or by metastasis to mediastinal lymph nodes;

- Phrenic nerve invasion with hemidiaphragmatic paralysis;
- Chest pain or pressure, due to parietal pleural or chest wall invasion;
- Superior vena cava syndrome, due to extrinsic compression by large hilar mass or bulky mediastinal lymph node metastases;
- Cardiac dysrhythmias, resulting from atrial or pericardial invasion;
- Dysphagia, due to compression or invasion of the esophagus in the posteroinferior mediastinum; and
- Vascular pain, typically central, severe, and unremitting, due to invasion of the aortic wall.

*Extrathoracic symptoms* are due, in most cases, to metastatic extension of the tumor to distant sites, with direct local effects. However, in 5 to 10 per cent of lung cancers, paraneoplastic syndromes may be identified.

*Chest radiography* is the basic diagnostic modality for neoplasms of the lung, with a high sensitivity at a low cost and minimal risk to the patient. Standardized techniques employ a high kilovoltage source and inspiratory posteroanterior and lateral views.

*Conventional laminar tomography* of the chest has limited application in the diagnosis of parenchymal pulmonary neoplasms, as computed tomographic scanning provides a more sensitive and precise examination.

*Magnetic resonance imaging (MRI)* has limited application for patients with pulmonary neoplasms.

*Positron emission tomography (PET)* scanning is a technique for biochemical characterization of tumors and their metastatic deposits.

*Cytologic evaluation of sputum* for neoplastic cells can diagnose malignant lung neoplasms in 60 per cent of cases, primarily when the tumor invades transmucosally. A lower yield is found for peripheral tumors, submucosal lesions, or when bronchial obstruction is present. Cell type can be specified in 80 per cent of cases, with a false-positive rate of less than 1 per cent. Sputum cytology should be obtained on any patient with a suspected lung tumor who has a productive cough or hemoptysis. Specimens should be collected in the morning for 3 days.

*Percutaneous needle aspiration* of pulmonary neoplasms is a safe, accurate method for histologic identification, particularly for those tumors located in the outer two-thirds of the pulmonary parenchyma.

***The tubercular cavern*** or ***tuberculoma*** is mainly located in the upper lobes of lungs, roentgenologically revealed on the background of characteristic changes of adjacent pulmonary tissue (fibrosis, petrifactions, dissemination, peribronchial lymphadenitis), sometimes a draining bronchus is detected. In the sputum mycobacteria of tuberculosis are frequently found. The considerable difficulties can arise during differentiation of the closed congenital cysts.

**Management**

The tactics in acute pulmonary destruction should be mainly conservative.

1. The adequate antibacterial, antiinflammatory therapy consists of intravenous introduction of antibiotics of B wide spectrum activity.

With the purpose of maximal concentration of drugs in the pathological focus applied:

- Injection of antibiotics in the vessels of pulmonary circulation by means of catheterization of central veins, pulmonary artery;
- Introduction of medicines into respiratory tracts (in the second stage) - through the endotracheal micro irrigator, nasogastric tube, during bronchoscopy, endoscopic catheterization of the abscess cavity through the draining bronchus, during aerosolic inhalations. The composition of medical admixtures includes: antibiotics, antiseptics (10% dimexid, dioxydin, microcid etc.), enzymes;
- Transcutaneously in the focus of destruction by means of puncture or draining with the usage of physical antiseptics.
- Intrapleural injections;
- By means of electrophoresis.

2. Evacuation of purulent content of the cavities:

- In natural way by an active sanation of tracheobronchial tree using repeated fibrobronchoscopies, aspirations through the endobronchial catheter, installations of medical agents through the microtracheostomy, aerosolic inhalations;
- Transthoracically by means of repeated punctures or external draining of peripheral cavities.

3. Detoxication therapy (intra- and extracorporeal).

4. Immune correction (under the control of immunogram):

- Active - staphylococcal anatoxin;
- Passive - specific gamma-globulins, hyperimmune plasma;
- Non-specific (pirimidine and purine derivates, drugs of thymus gland, splenin, levamisol,).

5. Homeostatic correction (oxygenotherapy, correction of anemia, hypoproteinemia, acidosis, microcirculatory disturbance).

6. Desensitizing, anti-inflammatory therapy, regulation of proteases activity: antihistamine, nonsteroid anti-inflammatory agents, inhibitors of proteases, antioxidants.

7. Correction of the organ and system dysfunction, prevention of complications, symptomatic therapy.

**Indications for operative management** in acute destructive processes of lungs:

- Pulmonary bleeding of II-III degree;
- Progression of the process despite active and appropriate therapy;
- Tension pyopneumothorax, which is failed to liquidate by the draining of pleural space;
- Impossibility to rule out the suspicion on a malignant tumour.

**Contraindications:** decompensation of the vital systemic functions in the terminal stage, bilateral purulent destruction of lungs, concomitant incurable malignant tumours.

Systemic antibiotics directed against the pathogen are the mainstay of therapy. For community-acquired infections secondary to aspiration, oropharyngeal streptococci and anaerobes are likely to be the causative agents. Penicillin G, ampicillin, or amoxicillin are the mainstay of therapy, but a beta-lactamase inhibitor or metronidazole should be added to cover the increasing prevalence of beta-lactamase-producing gram-negative anaerobes. Clindamycin is also the mainstay of therapy. For community-acquired infections, Staphylococcus aureus and aerobic gram-negative bacteria are common oropharyngeal flora. Piperacillin or ticarcillin with a beta-lactamase inhibitor (or equivalent alternatives) provide better coverage of the likely pathogens. The duration of antimicrobial therapy varies: 1 to 2 weeks for simple aspiration pneumonia and 3 to 12 weeks for necrotizing pneumonia and lung abscess. It is probably best to treat until the caries has resolved or until serial radiographs show significant improvement. Parenteral therapy is usually used until the patient becomes febrile and cannot demonstrate consistent enteral intake. Oral therapy may then be necessary.

Surgical drainage of lung abscesses is rare, as drainage usually occurs spontaneously through the tracheobronchial tree. Indications for intervention include failure of medical therapy; abscess under tension; abscess increasing in size with appropriate treatment; contralateral lung contamination; abscess greater than 4 to 6 cm in diameter; necrotic infection with multiple abscesses, hemoptysis, ruptured abscess, or pyopneumothorax; and inability to rule out cavitating carcinoma. External drainage can be accomplished by tube thoracostomy, percutaneous drainage, or surgical cavernostomy. The choice between thoracostomy and radiologic catheter drainage depends on the preference of the treating physician and the availability of interventional radiology. Surgical resection is required in less than 10% of patients with lung abscess. Lobectomy is the preferred intervention for bleeding from a lung abscess or pyopneumothorax. An important intraoperative consideration is to protect the contralateral lung with a double-lumen tube, bronchial blocker, or contralateral main stem intubation. Surgical treatment has a 90% success rate, with an associated mortality of 1 to 13%.

Acute infectious destruction diseases of lungs demand a complex treatment directed on struggle with an infection with the help of antibiotics of a wide spectrum of action, improvement of conditions of abscess catchment, elimination of albuminous, water-electrolytic and metabolic infringements, the maintenance of functions is cardiovascular and respiratory system, liver, kidneys, increase of organism resistance.

Standard therapy for bronchiectasis includes optimizing clearance of secretions from the tracheobronchial tree, using bronchodilators to relieve any airflow limitation, and correcting reversible underlying causes when possible. Chest physiotherapy based on vibration, percussion, and postural drainage is widely accepted as the mainstay of therapy. Acute exacerbations should be treated with courses of broad-spectrum antibiotics tailored to the culture and susceptibility profile. Typically, a 2- to 3-week course of intravenous antibiotics followed by oral therapy results in longer-lasting remission. Surgical resection of a localized bronchiectatic segment or lobe may benefit patients with refractory symptoms while on maximal medical therapy. Multifocal disease must be excluded before any attempt at surgery; any uncorrectable

predisposing factor (e.g., ciliary dyskinesia) also must be excluded. An important surgical tenet is to conserve as much normal parenchyma as possible. Patients with end-stage lung disease from bronchiectasis may be potential candidates for a bilateral lung transplant. Surgical resection is also indicated in patients with large hemoptysis secondary to hypertrophied bronchial arteries. Because resection may not always be clinically practical, bronchial artery embolization is an alternative.

The purpose of treatment of pyopneumothorax should include sanation of the destructive focus in pulmonary tissue and liquidation of complications; that means elimination of pus and air from a pleural space and prompt expanding of lung.

- Active sanation of tracheobronchial tree by means of tracheocentesis.
- Draining of pleural space, active aspiration of its content (air, pus) to expand the lung.
- Lavage of pleural space by antiseptic solutions.
- Appropriate antibacterial, anti-inflammatory and infusion therapy.
- The therapy for increasing of immunological resistance of the organism (Staphylococcal anatoxin, antistaphylococcal gamma-globulin, antistaphylococcal plasma).
- Endolymphatic introduction of immunity stimulators (thymalin, thymogen, T-activin).

The indications for operative management are the same, as in pleural empyema.

The presence of pus in a pleural space is the indication for its elimination. In the site of diagnostic thoracentesis performed the draining of empyema's cavity, its sanation by means of antiseptic solutions. In a focal empyema the aspiration of pus is carried out by thoracentesis and only in its inefficiency performed a draining of pleural space.

If there is a residual space, persistent pleural infection is likely to occur. A persistent pleural space may be secondary to a contracted but intact underlying lung; or it may be secondary to surgical resection of the lung. If the space is small and is well drained by chest tube, a conservative approach is possible. This involves leaving the chest tubes in place and connecting them to a closed-system drain until symphysis of the visceral and parietal surfaces occurs. At this point, the chest tubes can be disconnected from suction; if the residual pleural space remains stable, the tubes can be cut and removed from the chest within a few weeks. If the patient is stable, tube removal can often be performed on an outpatient basis, guided by the degree of drainage and the size of the residual space visualized on serial CT scans.

Intensive antibacterial and anti-inflammatory therapy should be immediately instituted. For general improvement, use detoxication therapy (infusion of saline solutions, hemotransfusion, transfusion of proteins, solutions of dextran, forced diuresis, hemosorption if necessary), for increasing of immunological resistance of the organism. The empyema's sanation decreases the amount of pus, which discharged out through the drainage. The optimal variant of such course is the liquidation of empyema’s cavity, and then the drainage must be removed.

Larger spaces may require open thoracotomy and decortication to attempt to re-expand the lung to fill this residual space. If re-expansion is unsuccessful or appears to

be too high risk, then open drainage, rib resection, and long-term consolidation with delayed closure with muscle flaps or thoracoplasty may be required.

Transition of the process into the chronic form (10-12 weeks) results in formation of residual empyema cavity, which is possible to reveal by means of pleurography - introduction of water-soluble contrast through the drainage with further X-radiography in two planes. Most chronic pleural space problems can be avoided by early consultation with a specialist thoracic surgeon and complete drainage of the empyema, allowing the space to be obliterated with a re-inflated lung.

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**Tests**

1. The condition shown in the x-rays below is compatible with which of the following manifestations?
   A. Difficulty swallowing solids but not liquids
   B. Higher-than-normal incidence of esophageal carcinoma
   C. Failure of the upper esophageal sphincter to relax in response to swallowing
   D. Normal pressure in the body of the esophagus
   E. Normal esophageal motility
2. At the time of operation on the patient in the earlier question, a firm, rubbery lesion in the periphery of the lung is discovered. It is sectioned in the operating room to reveal tissue that looks like cartilage and smooth muscle. The most likely diagnosis is:
   A. Fibroma
   B. Chondroma
   C. Osteochondroma
   D. Hamartoma
   E. Aspergilloma
3. A correct statement concerning bronchial carcinoid tumors is:
   A. They frequently metastasize
   B. They most commonly arise in peripheral terminal bronchioles
   C. They rarely produce the carcinoid syndrome
   D. They are radiosensitive
   E. Five-year survival is less than 50%
4. True statements about the lesion visualized on the film include which of the following?
   A. It is more apt to be metastatic breast carcinoma than primary lung carcinoma
   B. There is a 90% chance that this mass is malignant
   C. Since the diagnosis can only be established with certainty by resection, the mass should be excised

D. If the mass is malignant, the possibility for cure with excision is remote
E. The mass is most likely benign

5. Superior pulmonary sulcus carcinomas (Pancoast tumors) are bronchogenic carcinomas that typically produce which of the following clinical features?
   A. Atelectasis of the involved apical segment
   B. Horner syndrome
   C. Pain in the T4 and T5 dermatomes
   D. Nonproductive cough
   E. Hemoptysis
6. The chief diagnostic method for differentiation of pleural effusion is:
   A. X-ray
   B. Thoracentesis
   C. CT
   D. Ultrasound
   E. Bronchoscopy
7. The rupture of abscess of lung and presence of pus and air in the pleural cavity manifests with all of the following EXCEPT:
   A. Acute pain in the side
   B. Strong cough
   C. Shallow breath
   D. Tachycardia
   E. The compression of lung and displacement of mediastinum to the healthy side
8. What is the most effective way of treatment of patients with acute pleural empyema?
   A. The injection of antibiotics with proteolytic ferments inside the trachea
   B. Thoracotomy with pleurectomy
   C. Draining of pleural cavity by Bulough's system
   D. Draining of pleural cavity with active permanent aspiration
   E. Tapping of pleural cavity with removing of pus
9. A man of 40 has slept outside for 4-5 hours after being drunk. In 2 hours his body temperature increased, pains in chest appeared. Temperature gradually increased to 39. In 2 weeks during cough suddenly 200ml foul smelling pus appeared. What's the most possible diagnosis?
   A. Acute abscess of lungs
   B. Bronchiectatic disease
   C. Pleurisy
   D. Exacerbation of chronic bronchitis
   E. Cancer of lung with a development of pneumonitis
10. The patient enters to the emergency department with a high temperature (39º C), pain in chest, bad smell from the mouth, and productive cough with sputum (divides into three layers**)**. What disease can be firstly thought about?
    A. Bronchiectatic disease
    B. Pleurisy
    C. Acute abscess of lung
    D. Exacerbation of chronic bronchitis

E. Cancer of lung with development of pneumonitis.

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| B | D | C | C | B | B | E | D | A | C |

**Materials on the independent teaching of students**

| Main tasks | Notes(instructions) |
|---|---|
| Repeat:<br>– Lobar lungs structure and anatomy of pleural cavity, special features of blood circulation in lungs.<br>– Histological structure of hematoalveolar barrier.<br>– Normal physiology of pleura and lungs.<br>– Pathogenesis of pleural and lungs diseases.<br>– Morphological changes in pleura<br>– Clinical displays and methods of diagnostic of pleural and lungs diseases. | -Represent methods of lungs and pleural disease diagnostic using scheme.<br>-Make up a block diagram of lungs and pleural disease pathogenesis.<br>-Extract the schemes of conservative therapy of lungs and pleura inflammatory processes. |
| Study:<br>– Types of thoracoplasty.<br>– Types of the accesses to the thoracic cavity. | -To make a differential diagnostic with pneumonias.<br>-To make a differential diagnostic with pulmonary tuberculosis and its complications. |

## 8.4. Damage to the chest cavity organs. Clinic and diagnosis, differential diagnosis. Therapeutic tactics. Frequency and classification of wounds and closed injuries of the chest. Clinical manifestations and diagnosis of various types of wounds and injuries.

**Overview.**

Modern traumatism represents an important social problem. Recent observation shows an increase of major combined trauma, whose complications often result in death. Approximately 30% of patients presenting with significant trauma have chest wall injury. Trauma of the chest is usually accompanied by dysfunction of the vital organs. Therefore, it is necessary to constantly improve diagnostics and treatment of the patients who suffer from trauma.

Thoracic organs are vitally important and considerably different from one another in closeness, consistency, mobility and ability to withstand the impact of a force/trauma. Life threatening situations that may develop rapidly such as valvular pneumothorax, hemothorax, tamponade of heart and atelectasis of lungs requires a doctor's good knowledge of the clinics of this pathology, ability to quickly recognize it and make the most rational decision in relation to the character and maintenance of medical care. Closed trauma of the lungs accounts for 60% of cases, ribs (45, 4%),

heart (7, 7%), spine (4, 8%), diaphragm (3, 7%), large vessels (3, 0%) and large bronchial tubes (2, 6%).

Penetrating wounds of the chest are more life threatening for victims due to the possible damage of intrapectoral organs and development of internal bleeding, emphysema of mediastinum and increased lungs-cardiac insufficiency.

**Educational aims:**

- Interrogation and clinical inspection of patients with trauma of thorax.
- To determine the etiologic and pathogenic factors of traumas of thorax.
- To find out the types of traumas of thorax, the clinical features, different variants of manifestation and complications.
- To develop a plan of examination of the patients with trauma of thorax.
- To estimate laboratory data, results of thoracoscopy, ECG, bronchoscopy, diagnostic puncture, sciagrams of the thoracic organs.
- To draw a differential diagnosis, substantiate and formulate a diagnosis of the trauma of thorax.
- To determine the indications for treatment of patients with the trauma of thorax.
- To cure of the patients with the traumas of thorax after operations.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomo-physiological information about thorax and organs of mediastinum.
- Classifications of chest damage.
- Mechanisms of chest trauma.
- Clinical picture of damages of thorax.
- Methods of diagnostics of chest damages.
- Principles of treatment of chest trauma.
- Giving first aid for chest trauma.
- Features of surgical interventions for the different traumas of thorax.
- Technique of draining of pleural cavity.

**A student must be able to:**

- Collect and estimate the complaints of patient with the trauma of thorax, information of anamnesis, to conduct physical research and correctly interpret the results obtained.
- Define the rational volume of laboratory and instrumental methods of research.
- Correctly interpret the results of clinical analyses, pleural tapping, ultrasound diagnostics, computed tomography, X-ray
- Give first aid for traumas of the chest.
- Define indications for operation and other methods of treatment of patients.
- Perform pre-operative preparation of patients.
- Conduct post-operative care.

**Terminology.**

| Term | Definition |
|---|---|
| Isolated trauma | traumatic action on organs and tissues within the limits of one anatomic area (thorax, abdomen, skull, extremities) |
| Combined trauma (polytrauma) | traumatic action within the limits of two and more of anatomic areas |
| Combination trauma | combination two or more, different mechanism of action of traumatic factors |
| Blunt damages of the chest | the mechanical damages of chest, when there is no violation of the skin covering the area |
| Pneumothorax | the accumulation of air within the pleural space |
| Hemothorax | the accumulation of blood in the pleural space |
| Chylothorax | the accumulation of chyle in the pleural space after trauma to the thoracic duct or a major branch |

**Content:**

The opened damages can be impenetrable or penetrable, blind or through, without damage or with damage of internal organs or bones. As a rule, they are complicated opened or valvular pneumothorax, hemothorax, hemopneumothorax and hemopericardium.

The mechanical damage of chest is blunt, if there is no violation of safety of the skin covering this area. In a peacetime such damages occur 9-10 times more frequent than opened and accounts for 9% of the common traumas cases. It is known that 40-45% lost because of mechanical traumas. During the last decade, these types of damages have become more frequent, due to an increased rate of movement of transports, wide distribution of tall buildings and other reasons. Damages of the chest are direct reason of injury in every quarter of cases and accounts for pain in half of the cases of traffic accident.

**Classification of blunt damages of the chest.**

I. According to the injury of other organs:
- Isolated trauma.
- Combined trauma (cranio-cerebral, with the damage of abdominal organs, with the damage of bones).

II. According to the mechanism of trauma:
- Commotion.
- Fracture.
- Contusion.
- Compression.

III. According to the character of the damage of chest organs:
- Without damage of organs.
- With damage of organs (bronchi, lungs, heart, diaphragm, trachea, esophagus, vessels, etc.).

IV. According to the character of complications:
- Uncomplicated.

- Complicated:
  - Early (pneumothorax, traumatic shock, hemothorax, subcutaneous, mediastinal emphysema floatative rib fracture, asphyxia);
  - Late (posttraumatic pneumonia, suppurative diseases of lungs and pleura, posttraumatic pleurisy).

V. According to the state of cardiopulmonary system:
- Without phenomena of respiratory failure.
- Acute respiratory failure (of I, II, III degree).
- Without phenomena of cardiovascular failure.
- Acute cardiovascular failure (of I, II, III degree).

VI. According to the severity of trauma:
- Mild.
- Moderate.
- Severe

Trauma to the chest wall is common and can range from an isolated single rib fracture to flail chest. Approximately 30% of patients presenting with significant trauma have chest wall injury. Guidelines of the Advanced Trauma Life Support program (Airway, Breathing, Circulation, Disability, and Exposure) always should be followed in the preliminary assessment of these patients. This organized approach helps to rule out injuries to the underlying viscera such as the lungs, heart, liver, and spleen, all of which frequently are associated with chest wall injury.

***Blunt chest wall trauma*** commonly results in contusion with localized tissue swelling and hematoma formation. In severe cases, these injuries can progress to soft tissue infections or necrosis. Initially, it often is difficult to distinguish between deep muscle injury and bony fractures, given the pain that is caused by these injuries. When subcutaneous emphysema is palpable on the chest wall, injury to the airway or lung parenchyma leading to a pneumothorax or esophageal perforation should be suspected.

***Rib fractures*** are a common injury sustained after blunt chest wall trauma. A higher incidence of fractures is observed in the elderly owing to the loss of chest wall compliance from ossification of costal cartilage and osteoporosis. Symptoms include pain during inspiration and localized tenderness. The management of rib fractures depends on the number and location of the injuries. Upper thoracic rib fractures (T1-T5) are uncommon because of the relatively protected position of these ribs below the upper girdle musculature. Fractures of the first two thoracic ribs usually are seen in high-velocity injuries and can be associated with aortic disruption (6%). Similarly, fractures of the lower thoracic ribs (T11-T12) are uncommon because the ribs are short and less exposed. Frequently, fractures to ribs 11 and 12 are associated with injuries to underlying abdominal organs such as the spleen, liver, and diaphragm. Fractures to thoracic ribs 5 to 10 are most commonly reported. Flail chest is a unique injury in which rib fractures lead to an unstable chest wall that result in a paradoxical motion during respiration. The injuries must occur along the same rib to produce the free-floating segment. This injury arises from blunt chest wall trauma such as direct impact from a steering wheel.

***Pneumothorax*** is the accumulation of air within the pleural space. Pneumothorax may be spontaneous or occur secondary to a traumatic, surgical, therapeutic, or disease-related event. A pneumothorax compresses lung tissue and reduces pulmonary compliance, ventilatory volumes, and diffusing capacity. These pathophysiologic consequences depend primarily on the size of the pneumothorax and condition of the underlying lung. If air enters the pleural space repeatedly (as with inspiration) and is unable to escape, positive pressure develops in the pleural space, causing compression of the entire lung, shifting of the mediastinum and heart away from the pneumothorax, and severe respiratory compromise with hemodynamic collapse. This situation is called a tension pneumothorax.

Valvular pneumothorax occurs as a result of damage to the lung tissue or chest wall with the formation of a valve, when air enters the pleural cavity during inspiration, and does not exit during expiration, due to the closure of the valve. This is the most dangerous form of pneumothorax, leading to complete lung collapse, mediastinal shift, kinking of the great vessels and cardiac arrest.

It may be the consequence of a pneumothorax from many causes. Pneumothoraces may be classified as shown:

***Spontaneous***

- Primary
- Secondary
- Chronic obstructive pulmonary disease (COPD)
- Bullous disease
- Cystic fibrosis
- *Pneumocystis*-related congenital cysts
- Idiopathic pulmonary fibrosis (IPF)
- Pulmonary embolism
- Catamenial
- Neonatal

***Traumatic***

- Penetrating
- Blunt

***Iatrogenic***

- Mechanical ventilation
- Thoracentesis
- Lung biopsy
- Venous catheterization
- Postsurgical

***Other***

A primary spontaneous pneumothorax occurs without a known cause or evidence of diffuse pulmonary disease or from subpleural blebs. A secondary spontaneous pneumothorax occurs as the result of an underlying pulmonary process that predisposes to pneumothorax. Iatrogenic pneumothoraces are common and may be caused by thoracentesis, central venous catheterization, surgery, mechanical ventilation, or diagnostic lung biopsy. Patients with pneumothorax most commonly present with chest

pain. It is often sharp and pleuritic and may lead to severe respiratory embarrassment or become dull and persistent. Dyspnea is the second most common symptom in patients with pneumothorax. Less common symptoms include nonproductive cough and orthopnea.

***Hemothorax*** is the accumulation of blood in a pleural space. The cause of occurrence of this complication is the damage of vessels of the chest wall, pleura, lungs and mediastinum.

**Classification**

*I. According to extent:*

- Unilateral.
- Bilateral.

*II. According to the degree of hemorrhage:*

- Small (the loss is less than 10% of volume of circulating blood).
- Moderate (loss of 10-20% of volume of circulating blood).
- Great (loss of 20-40% of volume of circulating blood).
- Total (exceeds 40% of volume of circulating blood).

*III. According to the duration of bleeding:*

- With continued hemorrhage.
- With the stopped bleeding.

*IV. According to the presence of clots in a pleural space:*

- Coagulated.
- Non-coagulated.

V. *According to the presence of infection:*

- Non-infected.
- Infected (suppurative).

If hemothorax is a complication of blunt chest trauma, clinical manifestations depend on the severity of the injury and the degree of hemorrhage. Also, hemothorax itself leads to compression of the lungs and displacement of the mediastinum.

With a small hemothorax, clinical manifestations of hemorrhage are slightly pronounced or absent altogether.

With a moderate hemothorax, shortness of breath, cough, general malaise and dizziness are pronounced. The skin is pale. Hemodynamic disorders are observed: tachycardia, decreased blood pressure.

Large and total hemothorax are accompanied by an extremely serious condition. Patients are concerned about severe general malaise, dizziness, shortness of breath, difficulty breathing. In some cases, they end up in the hospital in a terminal condition. The skin is extremely pale. The peripheral pulse is impaired or absent. Tachycardia is noted, heart tones are weak, blood pressure is reduced.

***The coagulated hemothorax.*** When patients delay seeking medical attention or when bleeding is severe, clots form in the pleural cavity, and in some cases, all the blood that has accumulated in the pleural cavity forms a large clot on its own. Depending on the extent of the bleeding and, consequently, the size of the clot, patients complain of chest pain that worsens with breathing, shortness of breath, general malaise, and dizziness. As a rule, in 3-5 days a fever of 37, 5-38°C is observed.

***Chylothorax*** most commonly develops after surgical trauma to the thoracic duct or its major branch, but may also be associated with a number of other conditions (Table 1). It is usually unilateral; for example, it may occur on the right side after esophagectomy, where the duct is most commonly damaged during dissection of the distal esophagus. The esophagus is in close proximity to the thoracic duct as it enters the chest, starting from the abdominal cavity near the cisterna. Bilateral chylothorax may occur if the mediastinal pleura is disrupted on both sides. Left-sided chylothorax may develop after left-sided neck dissection, particularly at the confluence of the subclavian and internal jugular veins. Chylothorax may also occur after nonsurgical trauma, including penetrating or blunt chest or neck injuries, central line placement, and other surgical procedures. It is also seen in neonates, probably as a result of birth trauma. It can be observed in association with various benign and malignant diseases, which usually involve the lymphatic system of the mediastinum or neck. Given the significant variability of the course of the thoracic duct within the chest, some injuries are inevitable. The direct relationship of chylothorax to a surgical procedure, traumatic event, or neoplastic process may not always be obvious. Understanding the anatomy and course of the thoracic duct and some of its more common variants is helpful.

| **Table 1 Etiology of Chylothorax** |
|---|
| **Congenital** |
| Atresia of thoracic duct |
| Thoracic duct-pleural space fistula |
| Birth trauma |
| **Traumatic and/or latrogenic** |
| Blunt |
| Penetrating |
| Surgery |
| Cervical: excision of lymph nodes; radical neck dissection |
| Thoracic |
| Patent ductus arteriosus |
| Coarctation of the aorta |
| Vascular procedure reinvolving the origin of left subclavian artery |
| Esophagectomy |
| Sympathectomy |
| Resection of thoracic aneurysm |
| Resection of mediastinal tumors |
| Left pneumonectomy |
| Abdominal: sympathectomy; radical lymph node dissection |
| Diagnostic procedures |

| |
|---|
| Translumbar arteriography |
| Subclavian vein catheterization |
| Left-sided heart catheterization |
| **Neoplasms** |
| **Infections** |
| Tuberculous lymphadenitis |
| Nonspecific mediastinitis |
| Ascending lymphangitis |
| Filariasis |
| **Miscellaneous** |
| Venous thrombosis |
| Left subclavian-jugular vein |
| Superior vena cava |
| Pulmonary lymphangiomatosis |

SOURCE: Reproduced with permission from Cohen RG, et al: The pleura, in Sabiston DC, et al (eds): *Surgery of the Chest,* 6th ed. Elsevier, 1995.

The thoracic duct most commonly originates in the abdominal cavity from the cisterna chyli, which is located in the midline at about the level of the second lumbar vertebra. From this origin, the thoracic duct ascends into the thorax through the aortic hiatus at the level of Th10–Th12 and passes to the right of the aorta. Because the thoracic duct passes over the diaphragm, it most often remains in the right thorax, lying immediately posterior to the esophagus, between the aorta and the unpaired vein. The duct continues upward, running to the right of the spine. Then, at about the level of the fifth or sixth thoracic vertebra, it passes behind the aorta and aortic arch into the left posterior mediastinum. From there, it ascends again, remaining near the esophagus and mediastinal pleura as it exits the thoracic inlet. As it emerges from the thoracic inlet, it passes to the left, immediately posterior to the carotid sheath and anterior to the inferior thyroid gland and vertebral bodies. Medial to the scalene anterior muscle, it passes downward and empties into the junction of the internal jugular and subclavian veins. Given the extreme variability in the main duct and its branches, accumulation of chyle in the chest or flow from penetrating wounds may be seen after a variety of traumatic and medical conditions.

The main function of the duct is to transport fat absorbed from the digestive system. The composition of the normal hilum is fat with variable amounts of protein and lymphatic material (Table 2). Given the large volumes of hilum that flow through the thoracic duct, significant damage can result in leakage of more than 2 L per day; if left untreated, protein, volume, and lymphocyte depletion can lead to serious metabolic consequences and death. Diagnosis usually requires thoracentesis, which can be a serious clue; often the pleural fluid is milky and nonpurulent. However, unless the patient is not taking anything per os (NPO), the pleural fluid is unlikely to be a serious

pathology. Laboratory analysis of the pleural fluid shows a high lymphocyte count and high triglyceride levels. If the triglyceride level is greater than 110 mg/100 mL, a chylothorax is almost certainly present (a 99% accuracy rate). If the triglyceride level is less than 50 mg/mL, there is only a 5% chance of chylothorax. In many clinical situations, the accumulation of chyle may be slow, because of minimal digestive fat flowing through the gastrointestinal tract after major trauma or surgery, so the diagnosis may be more difficult to establish.

**Table 2. Composition of Chyle**

| Component | Amount (per 100 mL) |
|---|---|
| Total fat | 0.4–5 g |
| Total cholesterol | 65–220 mg |
| Total protein | 2.21–5.9 g |
| Albumin | 1.1–4.1 g |
| Globulin | 1.1–3.1 g |
| Fibrinogen | 16–24 g |
| Sugar | 48–200 g |
| Electrolytes | Similar to plasma |
| Cellular elements | |
| Lymphocytes | 400–6800/$mm^3$ |
| Erythrocytes | 50–600/$mm^3$ |
| Antithrombin globulin | >25% plasma concentrate |
| Prothrombin | >25% plasma concentrate |
| Fibrinogen | >25% plasma concentrate |

SOURCE: Reproduced with permission from Miller JI: Diagnosis and management of chylothorax. *Chest Surg Clin North Am* 6:139, 1996.

**Manifestation and diagnosis.**

After a severe mechanical trauma, patients usually complaint of severe pain on the damaged side of chest, increased during movements, cough and deep breathing. When there is damage of pulmonary tissues, shortness of breath, frequent cough and hemoptysis occurs. When there is a massive loss of blood there are characteristic symptoms in the pleural cavity: increased pulse decreased arterial pressure, pallor of skin, vascular and respiratory insufficiency, and shock.

In area of damage of soft tissues of breast and broken ribs a haematoma is often visible. At palpation of this area a sharp sickliness, especially expressed at the broken ribs, is marked. Crepitating of splinters of bones is sometimes felt. At the damage of pulmonary tissues it is possible to define hypodermic emphysema on characteristic crepitation of air, saved in soft tissues of thorax.

When there is a severe pleural damage of the framework of thorax, lungs and vessels the signs of loss of blood are distinctly determined, to respiratory and vascular insufficiency, pneumo- and hemothorax. Their clinical symptoms will be shown below.

A traumatic asphyxia is easily recognized by the characteristic navy blue coloring of overhead part of trunk, petechial spit of blood on a skin and shortness of breath. Sometimes there is a temporal loss of eyesight and ear in connection with spit of blood in these organs.

When subcutaneous emphysema is palpable on the chest wall, injury to the airway or lung parenchyma leading to a pneumothorax or esophageal perforation should be suspected. The diagnosis of flail chest is made on clinical examination.

Percussion reveals dullness in hemothorax. Auscultation reveals sharply weakened or absent breathing over the hemothorax site. Physical data in coagulated hemothorax (reduced or absent voice tremor on palpation, dullness on percussion, and sharply weakened or absent breathing on auscultation) indicate the presence of a pathological process in the pleural cavity.

Chest radiography and chest CT can be helpful in case of chest trauma.

***X-ray of the lungs***

The lungs should be reviewed individually and in comparison, matching zone by zone from apex to base. Before assessing the lung density the patient's position should be noted. Rotation of the patient is identified by observing the distance between the medial end of the clavicle and the adjacent margin of the vertebral body. It should be symmetrical in a correctly positioned patient. Rotation will alter the density between the lungs, the side closer to the film appearing blacker. Any discrepancy in density between the lungs in a patient who is not rotated must be regarded as pathological. Serial radiographs may show a variation in lung density, in the same subject depending upon the depth of respiration: the greater the degree of lung inflation, the more translucent the lungs appear.

The normal linear markings are composed of the pulmonary blood vessels and the fissures. An alteration in vasculature may be local or generalized, and should be interpreted in conjunction with an assessment of the hilar pulmonary vessels and the degree of lung inflation. Abnormal linear shadows may be identified from their compartment of origin, namely, the lung parenchyma, the interstitium, the bronchovascular bundles, or the pleura. Although it may be difficult to distinguish between parenchymal and interstitial origin, this method of evaluation is a useful aid to interpretation.

The silhouette sign is useful for identifying and localizing pulmonary disease. The cardiac and diaphragmatic outline is entirely dependent on adjacent air-filled lung for its visualization. If the lung opacifies, the outline will be obscured. Any intrathoracic abnormality that obliterates the contour of the heart or diaphragm is in anatomical continuity with these surfaces. Loss of the right heart border implies middle lobe disease or, on the left, disease of the lingula. A collapsed and opaque lower lobe, lying posteriorly, will not efface the cardiac border but will obscure the adjacent diaphragmatic contour. The pulmonary vessels should be examined from the periphery towards the hilum. This method prevents the more prominent hilar vessels from diverting one's attention from the less conspicuous vessels of the periphery. The vessels in comparable zones of the lung should be of similar size and number. The vessels should be clearly defined: marginal haziness indicates an abnormality of the perivascular connective tissue or lung parenchyma. The vessels in the upper zone of

the lungs are smaller than those of the lower zones at equivalent levels, because of preferential perfusion of the lung bases due to gravity. In the outer periphery (1 to 2 cm) of the lung, vessels are no longer visible. The pulmonary arterial system is invariably related to the bronchial tree; they branch together as they run in the bronchovascular bundles. The pulmonary veins lie within the interlobular septa and are thus separate from the bronchoarterial pathways. These vessels terminate medially into two superior and two inferior pulmonary veins. In the upper zones the veins lie lateral to the arteries, while in the lower zones they have a more horizontal course. The apical, segmental upper lobe bronchus may be visualized end-on, at the hilum. Comparison with an adjacent opaque artery is a useful assessment of the calibre of the pulmonary circulation: normally the artery is of a similar calibre to the bronchus. Redistribution of blood flow resulting in the upper lobe vessels being larger than the lower lobe vessels is a well recognized sign of increased pulmonary venous pressure.

***Computed tomography of the chest***

CT is used routinely in the assessment of benign and malignant lesions throughout the chest wall, lung fields, or mediastinum. CT may also be used in patients with thoracic trauma, to define or confirm an abnormality detected or suspected on the standard chest radiograph. Cross-sectional imaging overcomes the difficulties of interpretation due to superimposition of structures on the chest film. CT is particularly sensitive in detecting pleural abnormalities, air, and fluid collections, which may pass unnoticed on the supine radiograph. It allows discrimination of blood from other pleural fluids due to the differences in density and is valuable in the assessment of mediastinal widening. Patients with high velocity deceleration injury and suspected trauma to the aorta or brachiocephalic arteries must always be studied by angiography to locate the site of injury precisely. However, in stable patients with mediastinal widening in whom there is a low index of suspicion of aortic injury; CT with intravenous contrast is advised. The mediastinal widening may be elucidated as being due to a fractured sternum, unfolded thoracic aorta, congenital vascular anomaly, a paramediastinal pleural collection, or a paraspinal haematoma related to a vertebral fracture. Pulmonary injury, including laceration, haematoma, and contusion is clearly defined. CT contrast studies allow differentiation of contused lung from adjacent collapsed lung or haematoma. Pulmonary abnormalities noted on CT are usually more extensive than those recognized on the plain chest radiograph. Pleural lesions, haematoma, and fractures may be demonstrated by CT but are usually apparent on clinical examination and on the plane radiograph. CT may be helpful in the evaluation of myocardial injury. The ability to demonstrate tracheobronchial or esophageal disruption is uncertain. Contrast examination of the esophagus remains the investigation of choice in suspected esophageal rupture. In patients with severe lower thoracic injury, CT scanning of the liver and spleen following intravenous contrast should be performed.

***Rib radiographs*** can help to confirm the diagnosis of rib fracture in an acute setting but cannot completely rule out this injury. 3 to 6 weeks after injury, callus formation around the fracture site is evident on repeat films.

The diagnosis of primary spontaneous pneumothorax usually is established by history and physical examination and confirmed with chest radiography. Patients are

often tall, thin men from 25 to 40 years of age. Physical findings may be normal if the pneumothorax is less than 25%. Characteristic physical findings include diminished chest excursion and hyperresonance on percussion of the affected side. Breath sounds are diminished to absent. Rarely, subcutaneous emphysema may be palpated or pneumomediastinum auscultated on cardiac examination. A pneumothorax usually is seen on the standard posteroanterior chest radiograph with displacement of the visceral pleura from the parietal pleura by air in the pleural space. The area appears hyperlucent with absent pulmonary markings. An end-expiratory chest radiograph may appear to increase the size of the pneumothorax because of reduction in lung volume during forced expiration. Recognition of a pneumothorax may be difficult on portable supine or semirecumbent chest radiographs obtained in trauma or critically ill patients because of both the location of the least dependent pleural spaces (anterior, subdiaphragmatic) and associated radiographic findings. Patients with bullous disease also may have chest radiographs that are difficult to interpret; chest CT may be useful in these situations. The routine use of CT in patients with spontaneous primary pneumothorax is not warranted because the confirmation of apical blebs does not change treatment recommendations. The occurrence of apical blebs and bullae in these patients has been found to be greater than 85% in most recent surgical series.

The radiological picture of hemothorax is quite specific. There is an intense homogeneous shadow on the side of the lesion with an oblique upper contour (Damoiseau line). The costal sinus is not visualized. In case of small hemothorax, depending on the degree of intrapleural bleeding, the shadow is observed only in the sinus area. In case of medium degree, hemothorax reaches the angle of the scapula (on the posterior surface) or the 5th rib on the anterior surface of the chest wall. In case of large hemothorax, this shadow reaches the 3rd rib, and in case of total hemothorax it is characterized by a complete shadow of the pleural fissure, and in some cases - by a shift of the mediastinum to the healthy side. On the chest radiograph of coagulated hemothorax, an intense shadow is detected, sometimes heterogeneous (with illumination and multilevelness).

At the small tears of bronchial tubes or trachea the state of patients some time can remain satisfactory, and the first symptoms appear pretty lately because of formation of scar, causing stenosis of the damaged bronchial tube. It is diagnosed on the basis of information of bronchoscopy at which detects the rupture of the tracheal wall.

***Thoracoscopy***

Conventional wisdom relates minimal access to limited exposure, but with the advent of videoscopic surgery this is no longer true. The thoracoscope is attached to a video camera unit and it provides a magnified view of the surgical field with high resolution for details. The chest is the most suitable body cavity for the minimal access approach not only because thoracotomy is a very painful incision but also because once the lung is collapsed (with selective one-lung ventilation) there is plenty of room for instrument manoeuvring. The use of carbon dioxide insufflations, and hence valved ports, is unnecessary. Conventional thoracic instruments can be placed directly through small wounds into the chest.

**Management**

Soft tissue infections or necrosis require antibiotic therapy and debridement. Circumferential burns to the chest wall require escharotomy to allow adequate chest wall expansion.

Fractures to thoracic ribs 5 to 10 are most commonly reported. Injury to three or more ribs often requires hospitalization for analgesia and monitoring of respiratory status. Splinting from improperly controlled pain can lead to atelectasis, retained secretions, and pneumonia. This is a particular problem in the elderly population. Analgesia can be provided using oral, intravenous, or intramuscular opioid analgesics for mild-to-moderate injuries, or epidural analgesia or intercostal nerve blocks for more severe injuries. Pain relief in closed trauma of the chest is achieved by means of different blocks:

- Vagosympathetic block.
- Alcohol - novocaine block of the site of fracture.
- Paravertebral block.

On the 2nd to 3rd day it is desirable to administrate electrophoresis with Novocaine. For the prophylaxis of congested phenomena in a pulmonary tissue used respiratory gymnastics, forced ventilation of lungs and inhalations.

Pulmonary contusion is the most commonly associated injury. Maintenance of adequate ventilation is the goal of therapy. Stabilization of the chest wall has been attempted using weights and rib binders, as well as fixation devices such as pins and plates. The methods of renewal of the skeleton of the flail chest are divided onto three groups:

- External fixation of a movable segment by means of suturing for intercostal muscles and traction during 2-3 weeks,
- Intermedullary costal osteosynthesis;
- Mechanical ventilation (often with positive end-expiratory pressure).

Mechanical ventilation with positive-pressure ventilation also occasionally is used to treat injuries in the elderly or in those patients with underlying pulmonary disease. Some centers report a more rapid wean from mechanical ventilation with the use of internal fixation.

The treatment of a first-time spontaneous pneumothorax depends on the size of pneumothorax, associated symptoms, and pulmonary history. Small pneumothoraces (<20%) that are stable may be monitored if the patient has few symptoms. Follow-up of a pneumothorax should include a chest radiograph to assess stability within 24 to 48 hours. An uncomplicated pneumothorax should reabsorb at a rate of approximately 1% per day. Indications for intervention include progressive pneumothorax, delayed pulmonary expansion, or development of symptoms. Moderate (20% to 40%) and large (>40%) pneumothoraces nearly always are associated with persistent symptoms that cause physical limitations and require intervention. Simple needle aspiration of a pneumothorax may relieve symptoms and can promote faster lung reexpansion. It also may help to determine whether the initial fistula that caused the pneumothorax has sealed or if there is an ongoing air leak that requires chest tube insertion. This method is carried out using a standard thoracentesis kit and either an evacuated bottle or hand aspiration via a three-way stopcock and syringe. The needle generally is placed either

anteriorly or laterally. The needle aspiration may be repeated, or a chest tube or needle catheter/thoracic vent drainage system may be inserted. It provides excellent management of iatrogenic pneumothoraces after central venous access or lung needle biopsy. This approach conservatively treats a sealed pneumothorax and identifies those with an active air leak for chest tube insertion. Emergent needle decompression for tension pneumothorax is carried out on the affected side by placing an 18-gauge needle or angiocatheter into the hemithorax *at the midclavicular line in the second anterior intercostal space.* This emergency maneuver relieves the tension created within the thorax. It does not treat the pneumothorax; subsequent chest tube insertion is required. Tube thoracostomy (chest tube insertion) and underwater seal drainage are the mainstays of treatment for spontaneous pneumothorax.

Full re-expansion of the lung, even in the presence of a continuous leak, usually can be achieved with the application of suction to the thoracostomy drainage system. The classic location for chest tube insertion is the same as for emergency needle decompression because the tube can be inserted quickly and easily without the need for patient positioning. The preferred approach is through the fourth, fifth, or sixth intercostal space in the mid-to-anterior axillary line. This can be done under local anesthetic employing rib blocks or under intravenous procedural sedation. The chest tube should be directed upward to the apex of the hemithorax. Care should be taken to avoid the subcutaneous placement of a chest tube. Digital pleural dilatation is recommended to confirm entrance into the chest cavity, appreciate any adhesions, and allow passage of the chest tube without need for a stylet, which can cause damage to the lung or other intrathoracic structures. Needle catheter/thoracic vent drainage systems may be used for the treatment of pneumothorax. This system is comparable to a chest tube and drainage system, although the tube is of much smaller diameter and is inserted by means of the Seldinger technique or stylet. The end of the needle catheter drain is modified to be completely compatible with the many underwater seal drainage systems available. Many kits also include a Heimlich valve (also available separately), which can be used in conjunction with either a catheter drain or conventional chest tube. The Heimlich valve and thoracic vent function as a one-way valve that lets air escape from the hemithorax, similar to an underwater seal. Patients may be discharged with these in place, to be removed at a later time after the leak has stopped.

Complications of chest tube insertion for pneumothorax are uncommon but include puncture of the underlying lung with air leak and pneumothorax; subdiaphragmatic entry with injury to the liver, spleen, or other intra-abdominal organs; secondary bleeding from injury to an intercostal vessel or, more commonly, to a larger vessel; and even puncture of the heart. Other technical complications include loss of catheter, guidewire, or fragment in the pleural cavity and infection. Occasionally, rapid drainage of a large effusion may be accompanied by dyspnea, clinical instability, and a phenomenon called post-expansion pulmonary edema. For this reason, it is recommended to drain only up to 1 L initially. Most complications can be avoided by consulting with a clinician experienced in pleural drainage techniques.

Re-expansion pulmonary edema is a rare complication that can be seen after treatment of a pneumothorax. Risk factors for this complication have not been consistently identified. Although re-expansion pulmonary edema is thought to be

secondary to a sudden increase in capillary permeability, the exact mechanism of this increased permeability is unknown. Most cases have been reported after rapid lung re-expansion. An air leak may be present for a variable amount of time after tube thoracostomy. Should the air leak persist for more than 72 hours or the lung does not completely re-expands, surgical intervention is compulsory. Primary spontaneous pneumothorax tends to recur with increasing frequency after each episode. The risk of first-time recurrence is about 25% to 30%.

Surgery is recommended for a recurrence or the development of a contralateral pneumothorax. Surgical intervention for a first-time pneumothorax is recommended in situations that include bilateral simultaneous pneumothoraces, complete (100%) pneumothorax, pneumothorax associated with tension, and borderline cardiopulmonary reserve and in patients in high-risk professions or activities involving significant variations in atmospheric pressure, such as pilots or scuba divers. Surgery for complications of pneumothorax (empyema, hemothorax, or chronic pneumothorax) also is recommended in patients with first-time spontaneous pneumothorax. Surgery for primary spontaneous pneumothorax has evolved over recent years from open thoracotomy (axillary or posterolateral) to a minimally invasive video-assisted technique. The surgery carried out is identical, despite the differences in approach. Apical blebs are resected. The parietal pleura over the apex of the hemithorax can be removed (pleurectomy), abraded (mechanical pleurodesis), or treated with talc or tetracycline-like agents (chemical pleurodesis or poudrage). The recurrence rate for these procedures, performed open or closed, is less than 5%.

Treatment options for primary and secondary spontaneous pneumothorax are similar. However, patients with secondary pneumothorax generally are debilitated from a respiratory standpoint and may have other significant comorbid diseases. Treatment with tube thoracostomy alone has a high recurrence rate. Effective treatment must be individualized but should include chemical or surgical pleurodesis in combination with complete lung reexpansion and effective sealing of air leaks.

The tremendous success of video-assisted thoracic surgery in the treatment of primary spontaneous pneumothorax has led to earlier referral by physicians and increased acceptance by patients for surgery. Stapled resection of apical bullas followed by mechanical pleurodesis remains the most frequently used technique, although more cost-effective means of eliminating the bullas (like suturing or looping) have been developed. While cases of primary spontaneous pneumothorax are easily approachable by video-assisted thoracic surgery, treatment of secondary spontaneous pneumothorax (with established lung pathology like emphysema or pneumoconiosis) requires more clinical judgment. Patients with difficult adhesions to take down may be more suitable for thoracotomy, while those who are elderly with multiple comorbidities may benefit more from a chemical pleurodesis (we prefer talc slurry) if the lung can be fully re-expanded.

The treatment of small hemothorax requires needle aspiration or drainage of pleural space and elimination of *blood at the mid-to-anterior axillary line in the sixth or seventh intercostal space*. The manipulation is carried out in VI-VII intercostal spaces in the postaxillary or scapular lines. Total, great or moderate hemothorax with persistent bleeding (positive Revilour-Greguar's test) requires thoracotomy for

liquidation of a bleeding source. The bleeding wounds of lungs are sewed up by twist suture. If the pleural space contains liquid blood, the surgeon carries out its reinfusion. The clots are removed from pleural space.

In case of the coagulated hemothorax the needle aspiration obtains small amount of a liquid hemolyzed blood and small bloody thrombi (consequently to the inner diameter of the needle).

The treatment plan for any chylothorax depends on the cause, the amount of drainage, and the patient's clinical condition. In general, most patients are managed with a short period of chest drainage, administration of NPO, total parenteral nutrition (TPN), and observation. Chest drainage should be sufficient to allow competitive lung expansion. Some authors have advocated somatostatin with varying results. If significant chylothorax drainage (>500 mL/day in adults, >100 mL in infants) persists despite TPN and good lung expansion, early surgical ligation of the duct is recommended. Ligation is best approached by right thoracotomy, and in some experienced centers, right VATS. Chylothorax caused by malignancy often responds to radiation and/or chemotherapy, and is therefore less likely to require surgical ligation. Untreated chylothorax is associated with significant nutritional and immunological depletion, leading to significant mortality. Before the introduction of surgical thoracic duct ligation, the mortality rate from chylothorax exceeded 50%. With the availability of TPN for nutritional supplementation and surgical ligation for persistent leaks, the mortality rate from chylothorax is less than 10%.

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**Tests**

1. A previously healthy 20-year old man is admitted to a hospital with acute onset of left-sided chest pain. The electrocardiographic findings are normal but chest X-ray shows a 40% left pneumothorax. Treatment consists of which of the following procedures?
   A. Observation
   B. Barium swallow
   C. Thoracotomy
   D. Tube thoracostomy
   E. Thoracostomy and intubation
2. A 50-year-old salesman is on a yacht with a client when he has a severe vomiting and retching episode punctuated by a sharp substernal pain. He arrives in your emergency room 4 hours later and has a chest film in which the left descending aorta is outlined by air density. Optimum strategy for care would be:
   A. Immediate thoracotomy
   B. Serial ECGs and CPKs to rule out myocardial ischemia
   C. Left chest tube and spit fistula (cervical esophagostomy)
   D. Flexible esophagogastroscopy to establish diagnosis
   E. Nasogastric tube, antibiotics, close monitoring

3. A sharp left-sided chest pain appeared in a 35-year-old patient at the time of intense physical activity. Objectively: the patient is covered with cold sweat; breathing is difficult because of the pain. Auscultation: on the right side the breath sound is vesicular, on the left side - weakened. Tachycardia, heart rate -100. What is your diagnosis?
   A. Spontaneous pneumothorax
   B. Heart attack
   C. Myocardial infarction
   D. Intercostal Neuralgia
   E. Pneumonia
4. Tension pneumothorax has the following features, EXCEPT:
   A. Displacement of the heart on the side opposite pneumothorax.
   B. Distension of the neck veins.
   C. Arterial hypotension.
   D. All of the signs, without exception.
   E. The blunt percussion sound on the side of the lesion.
5. What is the first aid in a patient with tension pneumothorax?
   A. Tube thoracostomy
   B. Pleural puncture in VII intercostal space
   C. Occlusive bandage
   D. Thoracotomy
   E. Bronchoscopy
6. A 35-year-old patient has been admitted to a district hospital a week after a vehicle accident with the clinical signs of a convoluted hemothorax. What is the feasible therapeutic tactics in terms of prevention of acute pleural empyema in the patient?
   A. Pleural puncture
   B. Complex haemostatic conservative therapy
   C. Surgical removal of a convolute hemothorax
   D. Tube thoracostomy with passive drainage
   E. Thoracostomy with active drainage
7. Which of the following situations would be an indication for performance of a thoracotomy in the emergency room?
   A. Massive hemothorax following blunt trauma to the chest
   B. Blunt trauma to multiple organ systems with obtainable vital signs in the field but none on arrival in the emergency room
   C. Rapidly deteriorating patient with cardiac tamponade from penetrating thoracic trauma
   D. Penetrating thoracic trauma and no signs of life in the field
   E. Penetrating abdominal trauma and no signs of life in the field
8. A 19-year-old boy fell off his bicycle and was run over by a truck. When he was brought to the emergency room, he was awake, alert, and appeared frightened but not in any pain. The chest x-ray showed fluid in the lower left lung field, and the nasogastric tube appeared to be curling upward into the left chest. The next best step in management is:
   A. Placement of a left chest tube

B. Immediate thoracotomy
C. Immediate celiotomy
D. Esophagogastroscopy
E. Removal and replacement of the nasogastric tube; diagnostic peritoneal lavage

9. A 61-year-old man who smokes cigarettes and has chronic obstructive pulmonary disease fell and fractured the 7th, 8th, and 9th ribs in the left anterolateral chest. Chest x ray is otherwise normal. Appropriate treatment might include:
   A. A. Strapping the chest with adhesive tape
   B. Immobilization with sandbags
   C. Tube thoracostomy
   D. Peritoneal lavage
   E. Surgical fixation of the fractured ribs
10. A 25-year-old woman arrives in the emergency room following an automobile accident. She has acute dyspnea with a respiratory rate of 60 breaths/min. Breath sounds are markedly diminished on the right side. The first step in managing the patient should be to:
   A. Take a chest x-ray
   B. Draw arterial blood for blood gas determination
   C. Decompress the right pleural space
   D. Perform pericardiocentesis
   E. Administer intravenous fluids

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| D | A | A | E | A | C | E | C | D | C |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of organs of mediastinum, lobar structure of lungs and anatomy of pleural cavity, features of blood circulation of lungs<br>– Physiology of pleura and lungs<br>– Pathogenesis of development of complications from thoracic trauma<br>– Morphological changes in the organs of mediastinum | -To represent the methods of diagnostics of diseases of lungs and pleura as a table<br>-To make the flow diagram of mechanisms of damage of thorax |

| | |
|---|---|
| Study:<br>– Types of thoracoplastics.<br>– Types of accesses to the organs of pectoral cavity.<br>– Tactics in thoracoabdominal trauma<br>– Tactics at connections of head trauma and trauma of thorax. | -To conduct differential diagnosis with the damages of organs of mediastinum<br>-To conduct differential diagnosis of pathologies of the pleura |

# TOPIC 9. LIMB ISCHEMIA SYNDROME. THROMBOSES AND EMBOLISMS OF VESSELS. THROMBOEMBOLISM OF THE PULMONARY ARTERY

## 9.1. Limb ischemia syndrome

**Overview.**

Atherosclerosis became the cause of chronic arterial occlusive disease of the lower extremities. The arterial narrowing or obstruction that occurs as a result of the atherosclerotic process reduces blood flow to the lower limb during exercise or at rest. Epidemiological studies indicate that up to 5% of men and 2.5% of women 60 years of age or older have symptoms of intermittent claudication. The prevalence is at least threefold higher when sensitive noninvasive tests are used to make the diagnosis of arterial insufficiency in asymptomatic and symptomatic individuals. The symptoms of chronic arterial insufficiency of the lower extremities progress rather slowly over time. Thus, after 5 to 10 years, more than 70% of patients report either no change or improvement in their symptoms, while 20% to 30% have progressive symptoms and require intervention, and less than 10% need amputation. Despite the relatively benign prognosis for the affected limb, however, symptoms of intermittent claudication should be viewed as a sign of systemic atherosclerosis. This explains why, compared with age-matched controls, patients with intermittent claudication have a threefold increase in cardiovascular mortality

Acute arterial ischemia more frequent is an outcome of acute thrombosis of existent stenotic arterial segment (in 60%) or embolism (30%). Differential diagnostics of these two pathological states is important, as these circumstances influence on tactic of treatment and prognosis. Among other reasons of arterial ischemia is a trauma, iatrogenic damage, aneurysm, defects of heart.

A risk of development of thromboses after surgical operations: Prosthetics of hip joint - 60-65%. Amputation of hip 20%. Prosthetics of a knee-joint 25%.

**Educational aims:**

- Interrogation and clinical inspection of patients with limb arterial occlusion, nervous system damage and pathology of locomotive system.
- To determine the etiologic and pathogenic factors of limb arterial occlusion, lesions of the nervous system and damage of locomotive system.
- To find out the types of limb arterial occlusion, lesions of the nervous system and pathology of locomotive system, the clinical features, different variants of manifestation and complications.
- To develop a plan of examination of the patients with limb arterial occlusion, lesions of the nervous system and pathology of locomotive system.
- To estimate laboratory data end instrumental examination of the arteries.
- To draw a differential diagnosis, substantiate and formulate a diagnosis for the patients with limb arterial occlusion, nervous system damage and pathology of locomotive system.
- To prescribe the treatment for patients with limb arterial occlusion, nervous system damage and pathology of locomotive system.

- To determine the indications for operative treatment of patients with limb arterial occlusion, nervous system damage and pathology of locomotive system.
- To cure of the patients with limb arterial occlusion, lesions of the nervous system and pathology of locomotive system.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomo-physiological information about arteries.
- Classifications of acute and chronic ischemia of extremities.
- Mechanisms of limb arterial occlusion, nervous system damage and pathology of locomotive system.
- Clinical picture of limb arterial occlusion, nervous system damage and pathology of locomotive system.
- Methods of diagnosis of limb arterial occlusion, nervous system damage and pathology of locomotive system.
- Principles of treatment of limb arterial occlusion, nervous system damage and pathology of locomotive system.
- Features of surgical interventions for the different types of limb arterial occlusion, nervous system damage and pathology of locomotive system.

**A student must be able to:**

- Collect and estimate the complaints of patient with limb arterial occlusion, nervous system damage and pathology of locomotive system, information of anamnesis, to conduct physical research and correctly interpret the results obtained.
- Define the rational volume of laboratory and instrumental methods of research.
- Correctly interpret the results of clinical analyses, instrumental examinations
- Define indications for operation and other methods of treatment of patients with limb arterial occlusion, nervous system damage and pathology of locomotive system.
- Perform pre-operative preparation of patients with limb arterial occlusion, lesions of the nervous system and pathology of locomotive system.
- Conduct post-operative care.

**Terminology.**

| Term | Definition |
|---|---|
| Obliterating endarteritis | Is the disease of vessels of neurohormonal genesis which begins from the affection of peripheral vessels, mainly arteries, and results in the obliteration of their lumen. |
| Atherosclerosis obliterans of the lower extremities | Is a widespread disease, with a specific lesion of arteries of elastic and muscular types as a focal growth of connecting tissue with a lipid infiltration of the tunica intima. |

| | |
|---|---|
| Acute arterial occlusion | The sudden block of blood flow in a major artery caused by an embolism or thrombosis, which result from other diseases. |

**Content:**

Endarteritis obliterans is a segmental, inflammatory, obliterating disease affecting primarily small and medium-sized arteries and develops predominantly in young adults, usually men, who smoke.

**Etiology and pathogenesis**

In etiopathogenesis of endarteritis obliterans is considerably played by the consecutive or simultaneous influence of such factors like temperature, tobacco intoxication, mechanical traumas etc. The damage to the vascular walls results in autoimmune processes, which considerably intensifies the proliferative processes of the vascular intima. First of all, the vessels are affected. It results in intracapillary malnutrition with a hypersensitivity of the vascular walls, which provokes occurrence of pain. The latter causes the spasm of regional vessels, sluggish blood flow, hypercoagulability and finally necrosis of tissues.

**Pathology**

The morphological basis of endarteritis obliterans is the intimal hyperplasia predominantly of medium-sized or small vessels of the extremities, which leads to stenosis and obliteration of arterial lumen: latter causes hypoxia of the extremities, which leads to necrosis.

**Classification**

In the course of endarteritis obliterans such stages are distinguished (according to A. Shabanov, 1983):

- ischemic;
- of trophic changes;
- ulcerative-necrotic;
- Gangrenous.

**Forms of clinical course and complications**

The course of the disease and expressiveness of signs depends on its stage.

In **ischemic** stage fatigability of legs during walking, coldness, paresthesias and muscular cramps are observed. The main signs are discoloration and temperature changes of the skin of feet, lability of vascular response, impaired pulsation and blanching of capillaroscopy pattern. Angiogram is without pathological changes.

The stage of trophic changes is characterized by extremely fatigability and coldness of legs, expressed paresthesias and appearance of pain when walking as "intermittent claudication". Intermittent claudication (Latin: claudicatio intermittens) is a clinical diagnosis given for muscle pain (ache, cramp, numbness or sense of fatigue), classically in the calf muscle, which occurs during exercise and is relieved by a short period of rest. Objective examination reveals blanching or cyanosis of feet, thickened and deformed nails, skin atrophy and coldness, impaired or absent of arterial pulsation on foot. On capillaroscopy in these patients it is possible to note pathological changes of the pattern: decreasing of capillaries amount and spasm of arterial part of a capillary loop. On arteriogram an occlusion of arteries of the leg is observed.

For ulcerative-necrotic stage is characteristic the constant resting pain, which amplifies when the patient is supine. The ulcers appear on toes and foot. The walking is limited, the sleep is disturbed. Rather frequently, the phenomena of accompanying thrombophlebitis and lymphangitis join the ulcerative process. There are expressed atrophy of muscles, blanching of skin (cyanosis in the region of ulcers), coldness of skin and absence of arterial pulsation on foot. The trophic changes exist not only on skin, but also in bones (spotty osteoporosis). On arteriogram it is possible to note the occlusion of two or even of three arteries of the leg. On capillaroscopy the blanched or cyanotic background, lack of capillaries and their deformity are revealed.

The gangrenous stage is characterized, first of all, by the signs of toxemia with its influence on mentality, cardiovascular system, kidneys and liver. The patients in this stage are sleepless either day, or night. The gangrene can be either wet or dry. Thus the necrosis of soft tissues, and frequently of bones, edema of the leg, ascending lymphangitis, thrombophlebitis and inguinal lymphadenitis takes place. The temperature of skin in dry gangrene is usually decreased, in wet - can be normal. On angiogram observed the occlusion of arteries of foot, leg, and quite often the femoral artery.

Among the complications of endarteritis obliterans most frequently: arterial thrombosis and gangrene of extremity. The dry gangrene often develops in the regions with lack or absence of muscles and subcutaneous fat. The demarcation line in these cases is well defined with a slight expressed zone of inflammation on its edge. The wet gangrene develops when the chronic ischemia is complicated by vascular thrombosis. It can develop not only in arteries of foot, but also in arteries of the legs. The extremity in such patients is swollen, with tense skin. The demarcation line in such situations, as a rule, is expressed rather weakly. The leading sign of wet gangrene are the symptoms of general toxemia.

**The diagnostic program**

- Complaints, anamnesis.
- Examination of extremities.
- Palpation, auscultation of vessels.
- Rheovasography.
- Dopplerography of vessels.
- Aorto-arteriography.
- Biochemical blood analysis.
- Coagulogram.

**Tactics and choice of treatment**

***Conservative therapy.*** The goal of the treatment of obliterating endarteritis may be changed depending on the stage of disease; nevertheless the main purpose should be the renewal or improvement of capillary circulation. This problem could be solved by: 1) improving of blood rheology; 2) improving of peripheral macrohemodynamics, particularly by reducing of the arterio-venous dumping of blood (thus the application of spasmolytics is categorically contraindicated); 3) normalization of interaction between endothelium and formed elements of blood.

The most effective is the synthetic prostaglandin El - Vasaprostan. Its prompt therapeutic efficiency is caused by inhibition of free radicals of oxygen and lysosome enzymes of activated leukocytes in ischemic conditions, block of thrombocyte activity, elevation of erythrocyte lability, decreasing of their aggregation and diminishing of blood viscosity.

Among the other vasoactive drugs, pentoxyphillin is often of great value. Besides the expressed block of thrombocytes, it stimulates decreasing of aggregation of erythrocytes, elevating of their synthetic properties, and also inhibits anti-inflammatory effect of various cytokines.

Desensitizing agents (dimedrol, pipolphen, diazolin, suprastin, tavegil) block the influence of histamine and result in vasodilating effect, thus reducing the permeability and fragility of the vascular wall.

The drugs, which influence on the blood rheology, are dextrans (rheopolyglucin, polyglucin). They diminish blood viscosity, prevent aggregation of formed elements of blood, reduce peripheral resistance, and increase fibrinolytic activity of blood. Anticoagulants (of direct and indirect action) and antiaggregants (aspirin) also improve the blood rheology.

The stimulators of metabolism are also used in the therapy of arterial ischemia. These drugs are nicotinic acid, solcoseril, actovegin.

In the complex of treatment physiotherapeutic agents and oxygenotherapy are also included.

The special attention thus should be paid to the blockade of ganglions, nervous trunks and plexuses. Taking into account a stage system of the regulation of vascular tonus, such blockade can be performed on different levels of vegetative nervous system: blockade of thoracic sympathetic ganglions and ganglions of a lumbar part of sympathetic trunk. Particular application has also paranephral, epidural and paraarterial blockade.

If conservative therapy is failed, the surgical treatment is necessary. A sympathectomy is considered to be the most effective operation in the patients with endarteritis obliterans.

The ganglion sympathectomy solves such problems: completely removes vasomotor spasm, liquidates or reasonably relieves the pain. Operation is especially effective in onset of the disease. In later stages the sympathectomy loses its anesthetizing action. Operation is carried out on the background of conservative treatment, which should last and in postoperative period. A lumbar sympathectomy requires removing 1-3 sympathetic ganglions.

***Contraindications for sympathectomy are***: 1) atony of capillaries; 2) lack of the effect at blocking ganglions; 3) complete obstruction of popliteal artery; 4) duration of reactive hyperemia exceeds 3 min; 5) anatomic and functional failure of collaterals.

Amputation should be deferred until conservative treatment has been given a thorough trial. In some instances it may be best to allow digits that are entirely or partially gangrenous or to slough spontaneously without amputation. It is almost useless to delay amputation of the leg when gangrene extends well into the foot, and it is inadvisable to delay amputation if pain is severe and cannot be controlled or if severe infection or toxicity supervenes. When amputation of a leg is necessary, an attempt

should be always be made to carry out the procedure below the knee. Only in rare instances it is necessary to do an amputation above the knee in patients with endarteritis obliterans.

***Atherosclerosis obliterans of the inferior extremities***

The inner lining of arterial blood vessels is normally smooth, allowing blood to flow easily. In lower extremity arterial disease, the lining becomes damaged, leading to build up of cholesterol and other lipids, causing the arterial wall inner lining to become rough and thickened. This accumulation is called atherosclerosis, or "hardening of the arteries." As the atherosclerotic process of the lower extremity arteries increases, the arteries become narrowed or blocked, causing blood flow to decrease. This can lead to discomfort, cramps, or pain in the hips, thighs or calves with walking. This is called claudication.

Claudication typically occurs during physical activity such as walking and is promptly relieved by a brief resting period (2-5 minutes).Normally; blood flow can increase up to ten-fold to meet the increased need for additional oxygen in exercising muscles. However, when the leg arteries are blocked, blood flow cannot increase in response to exercise and pain develops. Claudication pain always involves the same muscle groups, usually the calves, and does not change from day to day. The vascular surgeon relates the onset of claudication pain to a particular walking distance in terms of street blocks (e.g. "2-block claudication") or distance travelled before the symptom occurs. This helps to provide a standard of measuring if there has been any change before and after therapy has been initiated.

As atherosclerosis progresses and blockage becomes more severe, pain may occur in the feet even when at rest. This pain, known as rest pain, occurs because the arteries of the leg can no longer deliver adequate blood flow to the feet, even at rest. Rest pain generally worsens when the legs are elevated, such as when lying in bed at night. Relief from this pain may occur only when the feet are dangled. Gangrene or "death of tissue" may occur when nutrition needed for normal growth and repair can no longer be provided because of extensive arterial narrowing (stenosis) or complete blockage (occlusion) of lower extremity arteries.

Currently, atherosclerosis affects up to 10% of the Western population 65 years or older. When claudication is used as an indicator of lower extremity arterial disease, estimates are that 2% of the population aged 40 to 60 years and 6% older than 70 years of age are affected. With the elderly population expected to increase to 22% by the year 2040, lower extremity arterial disease will be even more common.

***Risk Factors***

The risk factors for atherosclerosis affecting the lower extremities are the same risk factors associated with coronary artery disease or cerebrovascular disease. These risk factors include:

- Smoking
- High blood pressure (hypertension)
- High levels of blood cholesterol or triglycerides (hypercholesterolemia, hyperlipidemia)
- Obesity

- Sedentary lifestyle
- Diabetes
- Family history of heart disease or arterial disease

Smoking is the most influential of all the risk factors. Although the mechanism by which smoking causes or worsens atherosclerosis is unclear, it is known that the degree of damage to the arterial wall lining is directly related to the amount of tobacco used. Quitting smoking is essential in the battle against atherosclerosis progression.

**Classification (according to A. Fountain, 1954)**

I stage - complete compensation (coldness, fatigue, paresthesias);

II stage -functional circulatory insufficiency (a leading sign - intermittent claudication);

III stage - ischemia of extremity at rest (a leading sign - resting night pain);

IV stage - considerably expressed destruction of tissues of the distal parts of extremity (ulcers, necrosis, and gangrene).

**Signs and Symptoms**

Essential to the management of a patient with leg pain is a comprehensive lower extremity examination including palpation of peripheral pulses. Signs and symptoms that advanced lower extremity arterial disease is causing the leg pain include:

- Discoloration of the affected leg or foot when dangling (from pale to bluish-red)
- Decreased hair growth on the legs and feet
- Diminished or absent pulses in the affected leg or foot
- Temperature difference in affected leg or foot (cooler than other extremity)
- Change in sensation (numbness, tingling, cramping, pain)
- Presence of non-healing wound on affected lower extremity
- Shrinking of calf muscles
- Presence of thickened toenails
- Development of gangrene
- Other diseases that must be considered

Other disorders that can cause leg pain are:

***Arthritis*** - Arthritic pain is variable from day to day and may be aggravated by certain weather patterns or physical movements. Unlike claudication, rest does not provide relief.

***Varicose veins*** - The pain associated with varicose veins is a dull aching, typically occurring at the end of the day or after prolonged periods of standing. Pain from venous disease is not exacerbated by exercise.

***Venous thrombosis*** - Swelling and leg pain associated with venous thrombosis typically occurs with walking and is relieved by extremity elevation, unlike arterial disease.

***Spinal stenosis*** - narrowing of the spinal canal due to a ruptured disk or arthritis of the back causes leg pain when standing and is not relieved by brief resting periods. Relief of pain often occurs by leaning forward against a stationary object (e.g. a tree) or sitting.

***Diabetic Neuropathy*** - Pain associated with this complication of diabetes is usually present in both legs. It often presents with numbness and diminished sensation in the lower extremities.

**Diagnostic Testing**

If you are suspected of having lower extremity arterial disease or your symptoms are worsening, the vascular surgeon will ask you certain questions, examine you, and order either non-invasive or invasive diagnostic tests.

Non-invasive testing: These tests are performed in the clinic setting or in a vascular laboratory, most often on an outpatient basis. They are virtually painless methods of examining the blood flow to the extremities, with essentially no side effects or risks.

**Pulse examination**: This is the primary assessment of the circulation. Normal findings by an experienced examiner make the likelihood of lower extremity arterial disease remote.

**Arterial blood pressures**: Using an ultrasound stethoscope (Doppler), the blood pressure in your arms and legs is measured, and the pressures are compared. This test gives the vascular surgeon a generalized assessment of the severity of the lower extremity arterial disease.

**Duplex scanning:** This test is useful for detecting blockages in an artery and measuring the size of the artery. It may also be used to measure the size of a vein that may be used as a bypass, a means of re-routing blood flow around a blocked or occluded area.

**Magnetic resonance angiography (MRA):** This test is also useful for imaging extremity vessels.

Invasive Testing: Tests in this category involve the injection of a contrast dye directly into your arteries under X-ray guidance.

**Angiogram**: This test is the most helpful imaging study used to direct treatment of symptomatic lower extremity arterial disease. Dye is injected directly into the artery and special x-rays are taken to reveal the exact location of the arterial blockage. An angiogram is only necessary when interventional or surgical treatment is being considered.

**Medical Management**

The treatment of leg pain due to claudication is primarily medical management of the symptoms, with surgery reserved for severe exercise induced pain which negatively impacts an individual's lifestyle. While atherosclerosis cannot be totally cured or prevented, the progression of the disease can be controlled through risk factor modification. This involves changing one's lifestyle to include healthy habits.

**Smoking:** Tobacco in any form should be avoided. Continued smoking is the most common adverse risk factor associated with progression of lower extremity arterial disease in patients experiencing claudication. The nicotine found in tobacco products causes the blood vessels to constrict, further narrowing them, preventing blood from reaching the intended targets (e.g. body organs, extremities), increasing the risk of atherosclerosis. In addition, smoking also decreases the amount of oxygen in the blood and can cause the blood to clot more readily.

**High blood pressure:** Untreated or uncontrolled high blood pressure (hypertension) causes the heart to work harder and creates additional stress on the arteries. Blood pressure should be monitored regularly because often hypertension occurs without symptoms. Take your medications as prescribed as long as your physician instructs you to, even if it normalizes and you "feel good."

**Diet:** The risk of atherosclerosis can also be reduced by carefully monitoring cholesterol (found in organ and red meats, dairy products and egg yolk) and saturated fats (found in animal fat and plant oils) in the diet. Polyunsaturated fats (found in corn, safflower and olive oils) are the fats to incorporate in your diet. In addition, a salt-restricted diet will help control high blood pressure and fluid retention associated with weight gain. If you are overweight, a general weight reduction diet is advantageous. Checkups with the doctor should include the monitoring of serum cholesterol. If it remains high (>200) despite the above diet then medication to reduce the cholesterol should be considered.

**Exercise:** Exercise plays a vital role in the treatment of atherosclerosis in patients with claudication. Patients with intermittent claudication often voluntarily reduce their daily walking because of pain and the fear of causing further tissue damage. This leads to an increasingly sedentary lifestyle that complicates the picture even more. Increase your walking distance gradually, stopping to rest when the leg pain develops. When it disappears, begin walking again. Measurable improvement ranged from 80-234% in controlled studies that incorporated a regular walking program into the daily routine. A regular walking program of 45-60 minutes/day is recommended.

**Diabetes:** Due to the important role that diabetes mellitus plays in the earlier onset and accelerated rate of atherosclerosis progression, it is crucial to follow the advice of the health care team regarding diet, medications, and treatment. Early treatment and meticulous management is paramount to controlling the effects of diabetes on arteries.

**Foot care:** When blood flow to the lower extremities is decreased, delayed healing of sores, serious infections, and gangrene (tissue death) of the feet or toes can occur after seemingly minor injuries (e.g. hang-nail, superficial laceration). Care must be given to avoid any situation that might cause injury to the foot. Inspect your feet daily. Immediately report to your physician the detection of any foot injuries or sores.

**Pharmacologic therapy:** In addition to others prescribed by your physician, the following medications may be added to your regimen:

Anti-Platelet Agents These drugs decrease the overall risk of heart attacks (angina, myocardial infarction) or strokes (cerebrovascular accidents or transient ischemic attacks) in persons with atherosclerosis. They also may improve walking distance by enhancing blood flow and overall circulation. Two examples of anti-platelet agents are:

- Aspirin - One aspirin tablet (81-325 mg) daily is the main antiplatelet agent utilized.
- Clopidogrel bisulfate (Plavix) – Plavix is a newer anti-platelet agent that appears to be gaining in popularity. Studies are in the process of determining which patient populations would most benefit from using this agent.
- Anticoagulation Agents – These drugs inhibit clot formation. Examples of these medications include:

- Warfarin (Coumadin) – The amount of this pill that is prescribed is based on specific blood test results.
- Lovenox – This medication, given as an injection, is used to achieve adequate anticoagulation, often while waiting for the warfarin dose to reach a therapeutic level.

Other Agents – These medications improve the circulation in the lower extremity. Treatment for at least one month is required to produce noticeable results. Examples of these types of medications include:

- Trental
- Pletal

**Surgical Management**

In cases where diligent medical therapy is not sufficient in resolving the symptoms or the symptoms progress at a very fast rate and have become lifestyle restricting, surgical therapy should be considered.

The first in providing surgical care is to determine exact location of the arterial blockage in the leg. Although this can be performed with an ultrasound, the most accurate test is called an arteriogram. An arteriogram is an outpatient procedure where a small needle and catheter are inserted into your artery. It is similar to having an IV started. A biologically safe dye is then injected into the arteries and pictures are taken of the blood flowing down each leg. This provides a roadmap of all of the normal and abnormal segments of arteries and allows the surgeon to identify the areas of concern.

Once the areas of arterial blockage is determined, two treatments options are possible; angioplasty or open surgery. An angioplasty is when small balloon is used to dilate a narrowed segment of an artery. Typically, the balloon is inserted into the artery and placed exactly at the area of arterial narrowing. The balloon is then inflated; smashing the plaque that was inhibiting the blood flow. This procedure can be performed at the same time as the arteriogram and usually requires less than a 24-hour hospital stay.

If there is too much blockage in the arteries to treat with balloon angioplasty, an open bypass operation must be undertaken. A bypass operation involves finding a suitable blood vessel above and below the area of blockage and routing blood flow between the two vessels with the aid of a bridge (graft) carry the blood between the two vessels. The graft may be constructed from a vein in the leg or a synthetic material. The procedure is rather tedious and usually requires 2 to 5 hours of surgery. A hospital stay of 3 to 7 days after surgery can be expected. A blood transfusion is needed in less than 10% of cases.

Angioplasty and open surgical repair are very safe procedures with excellent results. Factors that may diminish the success of each procedure include:

- the amount of arterial blockage present,
- the overall health status of the patient and,
- Adherence to risk factor control after the intervention.

The most significant risk factor that contributes to early failure after intervention is smoking; therefore lifelong cessation is paramount.

**Conclusion**

Patients with lower extremity arterial disease may present with symptoms ranging from minor claudication to extensive gangrene and threatened limb loss (amputation). While studies of large groups of patients with claudication reveal that amputation is uncommon, it continues to be a significant fear. How rapidly arterial disease progresses to limb loss largely depends on the number and severity of risk factors (e.g. smoking, hypertension, obesity, diabetes). Timely and regular medical evaluations and patient compliance with smoking cessation, diet and blood pressure control, daily exercise, and adherence to prescribed treatment modalities can markedly improve the claudication symptoms and ultimate outcome associated with lower extremity arterial disease.

Acute arterial occlusion may result from embolism from any source, acute thrombosis, or arterial trauma.

| Causes of Acute Arterial Occlusion | | |
|---|---|---|
| Embolus | Thrombosis | Trauma |
| Cardiac source<br>Atrial fibrillation<br>Myocardial infarction<br>Endocarditis<br>Valvular disease<br>Atrial myxoma<br>Prosthetic valves | Vascular grafts<br>Atherosclerosis<br>Thrombosis of aneurysm<br>Entrapment syndrome<br>Hypercoagulable state<br>Low flow state | Blunt<br>Penetrating<br>Iatrogenic |
| Arterial source<br>Aneurysm<br>Atherosclerotic plaque | | |
| Paradoxical embolus | | |

***Arterial embolism.***

Most arterial emboli originate in the heart and travel to the arteries of the extremities. The lower extremities are affected much more frequently than the upper extremities. Most such emboli occur in patients with cardiac disease. Often, the severity of the patient's underlying cardiac disease can increase the risk of surgery and limit the ability to restore blood flow to the ischemic limb. Sources of embolism include thrombus formation in the ventricles of the heart after myocardial infarction and atrial thrombus in patients with atrial fibrillation. Very often, patients with lower limb embolism have a history of recent myocardial infarction, arrhythmias, or atrial fibrillation.

20 percent of peripheral embolisms are embolizations of a thrombus or plaque originating from aneurysms or atherosclerotic lesions of the arteries. Emboli usually lodge where there is an acute narrowing of the artery, such as an atherosclerotic plaque or a branching point of the vessel; therefore, the bifurcations of the common femoral, common iliac, and popliteal arteries are common locations. Among arterial emboli, the following are most common:

- Femoral artery — 28 %
- Upper limb arteries — 20 %

- Aortoiliac segment — 18 %
- Popliteal artery— 17 %
- Visceral and other arteries — 9 % each

Compared with thromboembolism, fat embolism is less likely to cause symptoms of acute arterial ischemia. Fat emboli are usually irregular in shape and do not extend along the bloodstream; as a result, they tend to become incompletely occluded with subsequent secondary ischemic atrophy of the limb.

***Arterial thrombosis***—thrombosis of a stenotic artery is a well-known complication of atherosclerosis. Thrombosis of atherosclerotic vessels can occur by two mechanisms:

- Progressive atherosclerotic narrowing of the artery with low blood flow, stasis, and possible thrombosis
- Rupture of atherosclerotic plaque, intraplaque hemorrhage, and local hypercoagulation

The ischemia that occurs in the limb with arterial thrombosis on the background of underlying atherosclerosis is usually less severe than after acute embolism. This difference is primarily due to the collateral circulation that develops over time in patients with atherosclerosis and chronically stenosed vessels. The symptoms of chronic ischemia in atherosclerosis are often so pronounced that patients do not notice any changes in thrombosis, or only a slight increase in the symptoms of chronic ischemia when the main atherosclerotic vessel is completely blocked.

Arteritis, hypercoagulable states can lead to arterial thrombosis, occlusion, and acute limb ischemia. Although these conditions more commonly affect venous circulation, certain diseases accompanied by hypercoagulability contribute to arterial thrombosis (e.g., antiphospholipid antibodies and hyperhomocysteinemia).

**Arterial trauma** - Acute arterial thrombosis, which complicates vascular or cardiac diagnostic and interventional procedures, has become a common cause of acute limb ischemia. The incidence of arterial complications after interventional cardiac catheterization has been reported to range from 1.5 to 9 percent by various sources. Although acute arterial occlusion occurs in less than one percent of interventional catheterization procedures, this complication requires immediate surgical correction. Thromboembolism can also develop at the sheath site or at the catheter tip.

**Clinical evaluation** History taking and physical examination are the first steps in the evaluation of a patient with acute limb ischemia.

**Pain** caused by acute ischemia is usually localized distally in the limb, gradually increases and progresses with increasing duration of ischemia, and spreads in a prosimian direction. Later, pain may decrease due to ischemic nerve damage.

The history is important to determine whether the patient had symptoms of chronic ischemia before the acute condition. Patients with emboli usually have no previous symptoms of ischemia, and can often determine the exact time when symptoms associated with acute ischemia began. Sudden onset of ischemic symptoms in a previously asymptomatic patient is most consistent with embolism, whereas gradual increase in symptoms in a patient with chronic ischemia suggests thrombosis on the background of atherosclerotic vascular damage.

**Pulse.** The quality and pattern of peripheral vascular pulsation should be assessed. If a pulse is not palpable, Doppler ultrasound should be used. It is extremely rare to have life-threatening limb ischemia without significant pulse deficit. The pulse status of the contralateral limb is also important. The presence of a pulse deficit in an asymptomatic contralateral limb is a sign of underlying chronic arterial occlusive disease, more often atherosclerosis, that acute thrombosis has already occurred in the affected vessel. Conversely, the presence of a normal pulse in the contralateral limb indicates the absence of chronic occlusive disease and increases the likelihood that a cardiac embolus is the cause of the acute occlusion.

**The skin** of both the normal and affected limbs should be examined for temperature and color. The skin of the ischemic limb is usually cool and pale with delayed capillary refill. The level of arterial obstruction is usually visually one joint above the line of demarcation between normal and ischemic tissue. Both extremities should also be examined for signs of chronic ischemia, such as skin atrophy, hair loss and thickening of the nails, trophic ulcers, and necrotic areas.

**Neurological examination.** A thorough neurological examination should be performed, including reflex testing. Subjective sensory deficits, such as numbness or paresthesias, are signs of early neural dysfunction secondary to ischemia. Loss of sensory or motor function is a sign of progressive ischemia. Sensory deficits on the dorsum of the foot are often the earliest neurological sign of vascular insufficiency.

**CLASSIFICATION OF ACUTE EXTREMITY ISCHEMIA**

The Society of Vascular Surgeons (SVS) and International Society of Cardiovascular Surgeon (ISCVS) have developed a standardized method for categorizing and reporting acute limb ischemia based upon clinical examination. Extremities are placed in one of three categories based upon these clinical findings to help judge the severity of ischemia:

| SVS/ISCVC Classification of Acute Extremity Ischemia | | | |
|---|---|---|---|
| | Viable | Threatened | Nonviable |
| Pain | Mild | Severe | Variable |
| Capillary refill | Intact | Delayed | Absent |
| Motor deficit | None | Partial | Complete |
| Sensory deficit | None | Partial | Complete |
| Arterial Doppler | Audible | Inaudible | Inaudible |
| Venous Doppler | Audible | Audible | Inaudible |
| Treatment | Urgent work-up | Emergency surgery | Amputation |

• **Viable limbs** mean that there is no risk of tissue loss or atrophy.

• **Threatened** limbs have reversible ischemia, but immediate relief of the arterial occlusion is required if the extremity is to be salvaged and major amputation avoided.

• **Nonviable limbs** have irreversible tissue necrosis and require amputation regardless of the prescribed therapy. Revascularization of the nonviable limb may be performed to allow stump healing or to allow amputation at a lower level. According to the clinical course of disease (V. Savelyev and all. (1973)). IE- (ischemia at exertion) Lack of signs of ischemia at rest, (occurrence them only at exertion).

| Level of ischemia of extremity | Main feature |
|---|---|
| IT (ischemia of tension) | The absence of ischemia feature at rest (They appear only at exercising) |
| I A | The feeling of numb, cold, paresthesia |
| I B | Pain |
| II A | Parethis |
| II B | Plegium |
| III A | Subfascial swelling of muscles |
| III B | Partial contraction |
| III C | Total contraction |

**Diagnostic tests.** Arteriography is the primary diagnostic procedure that provides the most complete information in the setting of acute arterial occlusion. Arteriography can clearly delineate the site of the lesion, and arteriography can usually differentiate thrombosis from embolism.

An embolus on arteriography demonstrates a sharp contrast cutoff with a rounded reverse meniscus. An embolus may also be visible as an intraluminal filling defect, with contrast flow around it if the vessel is not completely occluded. Other findings most consistent with embolus include the presence of normal vessels without stenotic areas, the absence of collateral circulation, and the presence of multiple filling defects.

Arterial thrombosis usually appears as a sharp or narrowed mass in the lumen of the vessel, but not a rounded section, on arteriography. These patients usually have diffuse atherosclerosis with well-developed collateral circulation.

Although arteriography is the gold standard for diagnosis, it is not feasible for every patient with suspected acute arterial ischemia. Patients with a threatened limb, when there are signs of irreversible tissue damage, cannot tolerate the several-hour delay in revascularization while arteriography is performed. Thus, patients with a viable limb can usually undergo arteriography, whereas patients with a threatened limb should have immediate surgical revascularization with intraoperative arteriography if necessary on a case-by-case basis.

**Treatment.** The literature describes many treatment modalities for acute ischemia, which are difficult to compare. Acute limb ischemia is associated with high mortality and high rates of limb loss. The rate of limb loss is as high as 30 percent, and the in-hospital mortality rate for patients with this pathology is as high as 20 percent.

The best method of preventing limb loss is to initiate therapy promptly. After the diagnosis of acute arterial ischemia is established based on the history and physical examination, and before the initiation of instrumental methods of investigation, the Fifth ACCP Consensus Conference on Antithrombotic Therapy recommends that the patient immediately receive 10,000 units of intravenous heparin, followed by a

continuous infusion of heparin. The anticoagulant prevents further progression of the thrombus and suppresses thrombosis and stasis. Time is of the essence; the decision to administer heparin is based on clinical judgment and should not be postponed while waiting for diagnostic procedures and preparation for them.

Once heparin is initiated, treatment varies depending on the viability of the limb and the ability to perform revascularization. Treatment options include surgery and thrombolytic therapy.

Patients presenting with acute limb ischemia secondary to thrombosis or embolism should undergo urgent surgical revascularization. Most of these patients develop embolism, and irreversible limb tissue damage can occur within four to six hours of the onset of ischemia. Although pharmacological thrombolysis can successfully dissolve thromboemboli, the time required for this procedure is usually too long to be an adequate alternative to surgery.

Usually, thrombus or embolectomy is all that is needed to remove the occlusion and restore adequate blood flow to the limb. Most surgeons perform an intraoperative final arteriography after embolectomy to assess the adequacy of distal blood flow and the surgical procedure performed. Intraoperative thrombolytic therapy may also be used if there are small emboli in distal blood vessels to avoid additional surgical access. Depending on the duration and severity of ischemia and the severity of limb edema, fasciotomy may be necessary to prevent compartment syndrome. Patients with viable limbs. Thrombolysis has recently become a common alternative to surgery in patients with ischemic but viable limbs without evidence of necrosis. The success rate of thrombolysis in such cases approaches 70 percent with this technique, and limb salvage rates similar to those of surgery have been reported. However, the efficacy of thrombolytic therapy is limited by the severity of ischemia and the limited time from the onset of ischemia, the so-called therapeutic window, required to achieve thrombus dissolution.

Re-embolization occurs in approximately 7% of patients who are continuously anticoagulated versus 21% of those who are not. The most important initial decision is to focus on the viability and potential salvage of the ischemic limb.

As described in detail above, patients with ischemic but viable limbs should undergo urgent arteriography during clinical examination to plan surgical or medical revascularization. Arteriography allows to determine the exact localization of the thrombus or embolus and the severity of the lesion. During arteriography, attention is paid to several indicators that are crucial in order to accurately determine whetherthrombolytic therapy or surgical revascularization is the most appropriate treatment. These include:

- Etiology of acute ischemia (embolus or thrombus)
- Localization and extent of arterial lesion
- Duration of ischemic symptoms
- Availability of a suitable vein for bypass surgery
- Presence of contraindications (e.g., concomitant diseases) on the part of the patient for surgical intervention or thrombolysis.

Thus, a proximal embolus at the bifurcation of the common femoral artery is an ideal localization for embolectomy. On the other hand, a tibial artery embolus is best treated with a thrombolytic agent because this vessel is of smaller caliber.

Patients with nonviable limbs. Patients with nonviable limbs should have the necrotic segment of the limb amputated immediately. Arteriography is not usually performed because the level of amputation is determined by clinical findings and tissue viability at the time of surgery. Every effort should be made to preserve as many joints as possible to facilitate the prosthetic procedure, increase the function of the remaining limb, and increase the chances of successful rehabilitation. Delay in amputation of a nonviable limb may result in infection, myoglobinuria, acute renal failure, and sepsis.

**Basic literature:**


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**Tests**

1. According to the clinical course of disease (V.Savelyev and all., 1973), I B stage is:
   A. Sensation of numbness, coolness, paresthesia
   B. Pain
   C. Paresis
   D. Plegia
   E. Subfascial muscular edema
2. According to the clinical course of disease (V.Savelyev and all., 1973), II A stage is:
   A. Sensation of numbness, coolness, paresthesia
   B. Pain
   C. Paresis
   D. Plegia
   E. Subfascial muscular edema
3. According to the clinical course of disease (V.Savelyev and all., 1973), II B stage is:
   A. Sensation of numbness, coolness, paresthesia
   B. Pain
   C. Paresis
   D. Plegia
   E. Subfascial muscular edema
4. According to the clinical course of disease (V.Savelyev and all., 1973), III A stage is:
   A. Paresis
   B. Plegia
   C. Subfascial muscular edema
   D. Partial muscular contracture
   E. Total muscular contracture

5. According to the clinical course of disease (V.Savelyev and all., 1973), III B stage is:
   A. Paresis
   B. Plegia
   C. Subfascial muscular edema
   D. Partial muscular contracture
   E. Total muscular contracture
6. According to the clinical course of disease (V.Savelyev and all., 1973), III C stage is:
   A. Paresis
   B. Plegia
   C. Subfascial muscular edema
   D. Partial muscular contracture
   E. Total muscular contracture
7. Acute arterial occlusion is the indication for:
   A. Conservative treatment
   B. Urgent operative treatment
   C. Elective operative treatment
   D. None of the above
   E. All of the statements is true
8. The absolute contraindications for operation in patients with acute arterial occlusion include all of the following EXEPT:
   A. An agonal state of the patient,
   B. Total ischemic contracture of extremity (acute ischemia III C stage),
   C. Grave condition of the patient with slight degree of ischemia (acute ischemia IA-IB stage).
   D. Acute myocardial infarction
9. The relative contraindications for operation in patients with acute arterial occlusion include all of the following except
   A. Acute myocardial infarction
   B. Stroke
   C. Inoperable tumors
   D. Slight degree of ischemia without signs of its progression
   E. Grave condition of the patient with slight degree of ischemia (acute ischemia IA-IB stage)
10. The commonest cause of death following arterial reconstruction of the lower extremity is
   A. Graft infection
   B. Cerebrovascular accident
   C. Myocardial infarction
   D. Systemic sepsis secondary to skin necrosis
   E. None of the above

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| B | C | D | C | D | E | B | D | E | C |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Anatomy of extremities vessels<br>– Physiology of artery<br>– Pathogenesis of arterial occlusion<br>– Morphology of arteries<br>– Clinical displays and methods of diagnostics of arterial occlusion | -To represent the methods of diagnostics of diseases of arterial occlusion as a table<br>-To make the flow diagram of arterial occlusion |
| Study:<br>– Types of arterial occlusion.<br>– Tactics in acute arterial occlusion. | -To conduct differential diagnosis with of other arterial diseases |

**9.2. Thromboses and embolisms of vessels. Classification, clinic, diagnosis and differential diagnosis. Methods of conservative and surgical treatment. Thromboembolism of the pulmonary artery. Classification, clinic. Emergency care, methods of diagnosis, treatment and prevention.**

**Overview**

Hypercoagulation disorders, or thrombophilias, are most frequently encountered in surgical practice as deep venous thrombosis (DVT) or, less often, pulmonary embolism (PE). Although a ready explanation based on Virchow's triad may be available for the majority of surgical patients, other potential causes must be considered.

Disseminated intravascular coagulation (DIC) is not a separate disease, but a syndrome that occurs in the context of other diseases. Authors disagree on the diagnosis and treatment of this syndrome. Although DIC is usually considered to belong to the group of hemorrhagic disorders due to the bleeding that accompanies it, it is important to consider the signs of microvascular thrombosis and thrombosis of large vessels, which accompany DIC and lead to organ failure and death.

The management of acute extremity ischemia remains a major surgical challenge. Even with optimal surgical management, acute lower extremity ischemia resulting from thromboembolic disease continues to cause significant morbidity and mortality. Limb loss rates of 8% to 22% and perioperative mortality rates of 10% to 17% continue to be reported. Maximization of limb salvage, although simultaneously minimizing associated morbidity and mortality, requires expeditious diagnosis and restoration of perfusion.

Acute DVT is one of the most common causes of mortality in inpatients, especially in surgical patients.

Pulmonary embolism (PE) is a relatively common cardiovascular emergency. By occluding the pulmonary arterial flow, it may lead to acute life-threatening but

potentially reversible right ventricular failure. PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. However, early diagnosis is fundamental, since immediate treatment is highly effective. Acute pulmonary embolism may occur in 25% of general surgery patients with great majority asymptomatic and leads to 25% of postoperative deaths. Risk of fatal pulmonary embolism is 0.2 – 0.5% in moderate risk patient. Risk of fatal pulmonary embolism is 1 – 5% in high risk patient. Among patients with proximal deep vein thrombosis (DVT), about 50% have an associated, usually clinically asymptomatic PE at lung scan. In about 70% of patients with PE, DVT can be found in the lower limbs if sensitive diagnostic methods are used.

**Educational aims:**

- Interrogation and clinical inspection of patients with the thromboembolic diseases.
- To determine the etiologic and pathogenic factors of the thromboembolic diseases.
- To find out the types of Pulmonary Embolism (PE).
- To develop a patient examination plan with arterial and venous thrombosis and embolism.
- To estimate results of auscultation, ECG, echocardiogram, coronary angiography, laboratory data, duplex investigation of the vessels.
- To make a differential diagnosis, substantiate and formulate a diagnosis of the thrombosis and embolism of arteries and veins.
- To determine the indications for treatment of patients with the thromboembolic diseases.
- To cure of the patients with the thromboembolic diseases.
- To estimate efficiency of treatment and prognosis of disease.

**A student must know:**

- Anatomical and physiological data about arterial and venous systems and lungs.
- Main causes of arterial and vein thromboses.
- Mechanism of development of Pulmonary Embolism (PE).
- Mechanism of blood coagulation.
- Clinical presentations of thromboembolic disorders
- Methods of diagnosis of acute vessels' diseases.
- Main principals of surgical interferences at the different thrombotic and embolic complications.

**A student must be able to:**

- To collect and estimate the complaints of patient with the thromboembolic diseases, gather anamnesis, to conduct physical research and interpret their results.
- To define the rational quantity of laboratory and instrumental methods of investigation.

- Be able to do artificial pulmonary ventilation by methods “nose in nose” or “mouth in mouth
- To define the indications for surgical interventions and to choose the operation method in case of thrombosis or embolism.
- Prescribe post-operative treatment depending on patient’s state.

**Terminology.**

| Term | Definition |
|---|---|
| Thromboembolism | A condition in which a blood vessel is obstructed by a clot (thrombus) carried in the bloodstream from its site of formation. |
| Thrombus | An aggregation of platelets, fibrin, clotting factors, and cellular elements of the blood attached to the interior wall of vein or artery, sometimes occluding the lumen of the vessels. |
| Pulmonary Embolism (PE) | The blockage of pulmonary artery by fat, air, tumor tissue, or thrombus that usually arises from the peripheral vein (most frequently one of deep veins of the legs). |
| Pulmonary Infarction (PI) | Necrosis in part of a lung caused by an obstruction in branch of a pulmonary artery. |
| Thrombophilia’s | Hypercoagulation disorders |
| Embolectomy | A surgical incision into an artery for the removal of an embolus or clot, performed as emergency treatment for arterial embolism. |

**Content:**

**THROMBOTIC DISORDERS**

Thrombophilia may be caused by a decrease in antithrombotic proteins or an increase in prothrombotic proteins. The former includes antithrombin deficiency, protein C deficiency, and protein S deficiency. The above also includes factor V, prothrombin gene mutation (G 20210A), and and increased levels of factors VII, VIII, IX, and XI or vWF.

*Antithrombin* deficiency is an autosomal dominant genetic disorder that affects an estimated 1.1% of unselected patients with venous thromboembolism. Antithrombin levels range from 40% to 70% of normal, with as many as 85% of affected patients suffering a thrombotic event by 50 years of age. These patients are generally believed to be at greater risk than patients with other types of thrombophilia.

*Protein C* is a vitamin K–dependent glycoprotein synthesized by the liver that inactivates factors Va and VIIIa. Protein C deficiency is also an autosomal dominant genetic disorder that affects 3.2% of unselected patients with venous thromboembolism. Up to 50% of affected patients will experience a thromboembolic event by the age of 50.

*Protein S* is also a vitamin K–dependent glycoprotein that acts as a cofactor to inactivate factors Va and VIIIa. Protein S deficiency affects 2.2% of unselected patients with thromboembolism, with up to 50% experiencing their first event by 25 years of age.

*Factor V Leiden* is a single–base pair mutation (Arg506→Gln) of the factor V gene that results in activated protein C resistance. Factor V Leiden is found in 4% to 6% of the general population and in 6% to 33% of unselected patients with venous thromboembolism. There is a threefold to sevenfold increase risk for thromboembolism, with 30% suffering an event by the age of 60.

*The prothrombin gene* mutation G20210A is a glycine-to-arginine mutation in the factor II (prothrombin) gene. This mutation is identified in 6.2% of unselected patients with thrombotic events. A substantial number of these patients also carry the factor V Leiden mutation.

Increased levels of factors VII, VIII, IX, and XI or vWF (>150 IU/dL) are associated with a 2.2- to 4-fold increase risk for thrombotic events and a 1.08 relative risk for each 10-IU/dL increase. The causes of these elevations remain unclear but may have a genetic basis.

In addition to these inherited disorders, acquired disease processes associated with thrombotic events include pregnancy; cancer; sepsis; trauma; major operations, particularly those involving the pelvis; nephrotic syndrome; myeloproliferative disorders; medications like oral contraceptives, hormonal therapy, and chemotherapy; and malnutrition, including folic acid and vitamin $B_{12}$ and $B_6$ deficiency. Arterial thromboembolic events are difficult to separate from underlying atherosclerotic disease. Disorders include hypercysteinemia; paradoxical embolism due to a patent foramen ovale in the heart or due to a defect in interatrial septum; and inherited thrombophilia. Treatment of thrombotic events includes correction of the underlying process and anticoagulation. The duration of anticoagulation remains controversial and depends on the magnitude of the event, ongoing risk for thrombosis and treatment-associated risks, and anticipated future circumstances. Prophylaxis for patients at risk is based on the underlying disorder, magnitude of the risk, and anticipated requirements.

**DISSEMINATED INTRAVASCULAR COAGULATION**

DIC is a syndrome characterized by signs of activation of the coagulation system, fibrin deposition, fibrinolytic activation, sharp consumption of blood clotting factors and platelets, as well as multiple organ failure.

In acute DIC, there is a hemorrhagic syndrome, which is characterized by numerous ecchymoses (hemorrhages), bleeding from the mucous membranes and a decrease in the number of platelets and blood clotting factors. On the other hand, chronic (latent) DIC is less pronounced and is manifested by thromboembolism, which is evidence of activation of the coagulation system. In chronic DIC, clotting factors may be normal, increased or slightly decreased, and the number of platelets may also be altered.

Pathological conditions that may be accompanied by DIC include massive blood transfusion, amniotic fluid embolism, placental abruption, abortion, sepsis, viremia, burns, crushing and massive tissue destruction, poisoning with various poisons or snakebites, malignant tumors, liver disease and various inflammatory and autoimmune diseases, including vasculitis, Crohn's disease and similar diseases, heparin-induced thrombocytopenia with thrombosis, purpura fulminans in newborns (homozygous protein C deficiency).

The diagnosis of acute DIC can be made without performing all laboratory tests in most cases of this syndrome. This is especially true when the clinical picture is consistent with DIC and all routine test results are abnormal (i.e., platelet count, prothrombin time, partial thromboplastin time, fibrinogen level).

Numerous other laboratory parameters are associated with DIC, including prolonged thrombin time and decreased levels of antithrombin III, protein C, plasminogen, and alpha-2-antiplasmin. However, these same changes can be seen in severe liver disease and severe bleeding that results in plasma loss. The only test that can distinguish DIC from liver disease is the D-dimer test. This test is usually negative in liver disease, except in the case of massive necrosis, which can cause DIC.

Another laboratory finding of chronic DIC is a shortened activated partial thromboplastin time. Platelet levels may be normal, mildly decreased, or elevated. Furthermore, platelet levels may increase with heparin therapy and decrease dramatically with heparin withdrawal in the presence of a hypercoagulable state or chronic DIC.

Treating the underlying disease that caused it is crucial for successful treatment of DIC. Treating the hypercoagulability that causes organ failure is also important.

Acute DIC is treated with blood products that control bleeding well. Some medications (such as tissue factor concentrate, inhibitors of antithrombin III, protein C, or thrombomodulin) have shown success.

Prolonged bleeding may be due to depletion of clotting factors, but the indiscriminate administration of blood products, especially those containing fibrinogen, may exacerbate the syndrome. Washed red blood cells, platelets, AT-III concentrate, crystalloid and colloid solutions may be used to increase circulating blood volume. If other therapeutic measures are ineffective, fibrinolysis may be attempted. ε-aminocaproic acid may be administered in conjunction with heparin. Despite improved diagnostic and therapeutic methods, mortality from DIC remains high.

## ACUTE THROMBOEMBOLIC DISEASE

### Pathophysiology

Compared with other organs and tissues, the extremities are relatively resistant to the effects of ischemia. Unlike the brain, in which irreversible changes occur after 4-8 minutes of ischemia, or the myocardium, in acute infarction after 17-20 minutes, the lower limb can be fully saved even after 5-6 hours of deep ischemia.

Assessing the impact of ischemia on a limb is complicated by the fact that the various tissues that comprise the extremity have different susceptibilities to ischemic injury, and they manifest this injury in different fashions. Skin and bone are relatively resistant to the effects of ischemia and may survive injuries that, by their effect on other tissues, have rendered the limb painful and useless. Nervous tissue is generally the most sensitive component of the extremity to the effects of ischemia. Significant morbidity may therefore result from isolated ischemic nerve injury in an otherwise intact limb.

Skeletal muscles are the main structural component of the limb and play a key role in the pathophysiology and pathomorphology of limb ischemia.

Although skeletal muscle has a relatively slow resting metabolic rate compared with other tissues, it accounts for 90% of the metabolic activity of the lower extremity. Skeletal muscle receives 71% of the resting lower extremity blood flow and a larger

proportion during reperfusion hyperemia. Skeletal muscle plays a pivotal role in the numerous local and systemic manifestations of extremity ischemia-reperfusion injury.

**Reperfusion Syndrome**

The profound effects of revascularization of the ischemic lower extremity were described as early as the 1950s by Haimovici. As ischemic skeletal muscles reperfuse, a variety of intracellular ions, structural proteins, enzymes, and other components are released through the damaged sarcolemma into the circulation. The resulting myonephropathic syndrome, with its associated hemodynamic instability, lactic acidosis, and hyperkalemia, is well recognized by surgeons. Myoglobin released from injured muscle cells into the circulation is cleared through the kidneys, resulting in dark urine (without red blood cells). Myoglobinuria may persist for 2 to 4 days after reperfusion. Acute renal failure may ensue from myoglobin casts developing in the renal tubules as well as direct toxic effects of the myoglobin on the tubules. Serum creatine phosphokinase levels may increase dramatically (to >10,000 units) after reperfusion. Against the background of electrolyte disturbances, bradycardia or arrhythmias may be observed. During ischemia, cell membrane dysfunction occurs. In this case, the reperfusion phase is characterized by the development of both intracellular and interstitial edema. Interstitial edema occurs as a result of increased permeability of the microvascular membrane for ions, water, and proteins. Such edema may appear within a few minutes, progressing significantly over the next 24 hours. The degree of edema depends on the duration of ischemia, the disease that caused the ischemia, and the adequacy of revascularization. When muscle edema occurs within the bone-fascial sheath, interstitial pressure continues to increase. Acute compartment syndrome occurs when the pressure rises above the capillary perfusion pressure (30 mm Hg) and tissue perfusion is impaired. If not recognized and decompressed with fasciotomy, compartment syndromes will lead to further tissue ischemia despite apparent successful revascularization.

Similarly, prolonged vascular occlusion can lead to thrombosis of small vessels in the muscle and skin, preventing tissue reperfusion when blood flow is restored in larger vessels. Although current understanding of the effects of limb revascularization and compartment syndrome is well established, understanding of ischemia-reperfusion injury at the tissue level is only beginning to be explored. The pathophysiology of ischemic injury is complex and involves a number of factors, including inadequate oxygen and nutrient delivery, changes in ionic charge, and membrane permeability.

Most of the tissue damage in reperfusion syndrome is thought to be caused by oxygen-derived free radicals that are generated during the rapid reperfusion of ischemic tissue. These oxygen-derived free radicals are produced by neutrophils via the enzyme NADPH oxidase. These radicals are reactive species that result from the reduction of monovalent molecular oxygen. These unstable species attack unsaturated fatty acid bonds in phospholipid membranes, causing both mechanical and functional damage to biological tissue.

**Etiology *of embolism***

In 70% of cases, acute arterial ischemia of the limb is cardiac in nature, i.e. the primary thrombus is formed in the left heart chambers, and then in the form of a thromboembolus it enters the arterial bed of the limbs, causing acute arterial ischemia.

The primary cause of arterial thromboembolism in such cases is atrial fibrillation, left ventricular aneurysm, parietal thromboses and endocarditis.

Potential sources of embolism include thrombus formation in the ventricles of the heart after myocardial infarction and atrial thrombus in patients with atrial fibrillation. Up to 75 percent of patients with lower extremity embolism have a history of recent myocardial infarction or atrial fibrillation. Embolization of thrombus or plaque originating from aneurysms or atherosclerotic lesions accounts for 20 percent of peripheral embolisms. Emboli usually lodge where there is narrowing of the artery, such as atherosclerotic plaque or a branching site; the most common locations are the bifurcations of the common femoral, common iliac, and popliteal arteries. In a large series of arterial embolisms, for example, the following rates were noted:

- Femoral artery—28 percent
- Upper limb arteries—20 percent
- Iliac artery—18 percent
- Popliteal—17 percent
- Visceral and other—9 percent each

Acute arterial occlusion has become the more common cause of acute limb ischemia, complicating diagnostic and interventional vascular or cardiac procedures. The incidence of arterial complications after interventional cardiac catheterization (including hematomas, arteriovenous fistulas, pseudo aneurysms, arterial occlusion, and cholesterol embolism) has been reported to range from 1.5 to 9 percent.

Although acute arterial occlusion occurs in less than one percent of interventional catheterization procedures, this complication requires immediate surgical correction. Thromboembolism can also develop at the sheath site or at the tip of the catheter, with embolization occurring during sheath removal.

***Thrombosis***

Acute thrombosis usually occurs in vessels that are already affected by atherosclerosis. In such cases, the resulting ischemia is often less severe than in acute embolism. The most common vessel of the extremity affected by acute thrombosis is the superficial femoral artery, which is often affected by long segments of atherosclerosis. Also, patients with popliteal artery aneurysms have a higher tendency to thrombosis and can lead to more severe ischemia, especially in connection with subsequent embolization of the tibiae vessels.

A particularly severe form of ischemia occurs due to distal thrombosis of the vessels of the extremities, which can occur against the background of sepsis, or in hypercoagulable states such as DIC syndrome.

Prolonged exposure to heparin can lead to thrombosis in patients with heparin-induced antibodies. These antibodies cross-react with the heparin-platelet factor 4 complex on the platelet surface, leading to the formation of granular white platelet thrombi and subsequent thrombocytopenia. Acute thrombosis following arterial bypass grafting can also lead to recurrent ischemia. The degree of ischemia depends on the location of the shunt and the previous surgery. Early shunt occlusions (within 2 months of surgery) are usually caused by technical or diagnostic errors. Intermediate shunt

occlusions (within 2 years) are usually associated with the formation of intimal hyperplasia at the anastomoses or within the graft (for venous grafts).

**Presentation and Evaluation**

Clinical manifestations of acute arterial ischemia of the extremities are included in the principle of "five Ps":

1. Pain - severe pain.
2. Pallor - pallor of the limb.
3. Paresthesias - parasthesia in the affected limb.
4. Paralysis - paralysis of the limb segment.
5. Pulslessness - absence of pulsation in the main vessels of the limb.

Sometimes a "sixth P" is added to the principle of "five Ps": poikilothermia (cold blood) or "perishing cold". The most frequent symptom that forces the patient to seek medical help is severe pain syndrome in the limb of ischemic genesis. The most frequent localization of arterial thromboembolism of the lower limb is the bifurcation of the common femoral artery.

Often, along with pain in the limb and the absence of arterial pulsation distal to the site of thromboembolism, the patient has sensory disorders on the affected limb. The absence of arterial pulsation on both lower limbs is observed with saddle thromboembolism of the abdominal aorta bifurcation. The presence of pulsation on the femoral artery and the absence of a pulse on the popliteal artery and distally indicates either distal thromboembolism of the common femoral artery or thromboembolism of the superficial femoral and popliteal arteries. Thromboembolism of the “popliteal trifurcation” is manifested by ischemia of the lower leg and the absence of pulsation on the arteries of the foot with possible preservation of the pulse on the popliteal artery. In any case, the absence of arterial pulsation on the main vessel of the limb on one side (in the presence of a corresponding contralateral pulsation) should alert the doctor to search for arterial thromboembolism of this limb.

Patients with suspected acute arterial ischemia should undergo Dopplerography of the arteries of the lower extremities as a matter of urgency. The purpose of the examination: to clarify the localization of the thromboembolus, its size and to develop an optimal plan for surgical intervention.

The evaluation of each patient should be individualized to provide sufficient information to effectively treat the patient without compromising the limb.

When the history and physical examination suggest an embolus as the source of occlusion, further preoperative workup is straightforward. Routine preoperative blood work and a chest x-ray are performed, as is an electrocardiogram, which is performed to document atrial fibrillation, cardiac ischemia, or myocardial infarction.

Because arterial emboli are removed by direct arterial dissection and embolus removal (as described below), preoperative arteriography is usually not necessary. When the diagnosis of embolism is in doubt or the location of the thromboembolus is uncertain, a preoperative arteriogram may be useful to define the anatomy and guide the revascularization procedure.

Arteriograms of intraarterial emboli demonstrate an abrupt loss of contrast in the artery with a rounded meniscus at the site of the embolus. Conversely, an embolus may appear as an intraluminal defect with partial blood flow around it. After surgery, when

the embolus has been removed and the limb is revascularized, the patient is stable, and the evaluation is completed with documentation of the source of the embolus. In most cases, this includes transesophageal echocardiography.

**Management**

Drug therapy. In the absence of significant contraindications, a patient with acute arterial ischemia of the limb should immediately begin anticoagulant therapy with low molecular weight heparins. Surgical treatment tactics. In the case of acute arterial thrombosis of the arteries of the lower extremities, the patient is urgently required to undergo open surgery in the volume of open thromboembolectomy. If the thromboembolus is localized in the common femoral artery or its bifurcation, inguinal access to the vessel is performed. A transverse arteriotomy is performed, using a Fogarty catheter, thrombotic masses are removed from the lumen of the artery until the distal arterial blood flow is completely restored. After thromboembolectomy, if technically possible, it is advisable to perform a final intraoperative arteriography for the presence of residual thrombotic masses in the distal arterial bed of the limb. The arteriotomy site is sutured and anticoagulant therapy is prescribed.

In the case of arterial thrombosis of the popliteal segment, the femoral approach is used in most cases. In case of unsuccessful attempts to remove thrombotic masses from all three infrapopliteal vessels using a Fogarty catheter, thrombus extraction is performed under fluoroscopy control from the inguinal approach (fluoroscopic visualization) using a metal catheter for thrombectomy (over-the-wire thrombectomy catheter).

In the postoperative period, it is important to establish the source of thromboembolism of the limb arteries as early as possible by performing echocardiography and CT scanning of the descending thoracic aorta and abdominal aorta. In more complex situations, at the time of acute arterial ischemia of the limb, the patient already had OZANC or thrombosis occurred against the background of destruction of atherosclerotic plaque. In such cases, the embolectomy catheter, as a rule, cannot be passed distal to the area of arterial occlusion. In such cases, the surgeon has two options: imposing a bypass arterial anastomosis or passing a catheter to the site of thrombosis to perform selective thrombolytic therapy. In both cases, the patient is shown arteriography to clarify the extent of thrombosis and assess blood flow in the distal arterial vessels and their suitability for bypass grafting. Based on the results of angiography, one of the possible surgical treatment options is selected. Both with bypass grafting and with selective thrombolytic therapy, long-term results are equivalent.

Re-embolization occurs in approximately 7% of patients who are continuously anticoagulated versus 21% of those who are not. The most important initial decision is to focus on the viability and potential salvage of the ischemic limb.

Occasionally, patients have such prolonged and severe ischemia that irreversible ischemic damage to the limb (manifested as gangrenous changes in the leg) occurs. Such cases are best treated with primary amputation of the limb.

***Complications of reperfusion in acute arterial ischemia.***

The manifestations of reperfusion of an ischemic limb vary greatly in their pathophysiological effects and directly depend on the duration of ischemia and the

volume of affected tissues. The most severe postreperfusion syndrome occurs with a saddle thrombus at the bifurcation of the abdominal aorta. While patients with distal arterial thromboembolism and timely surgical revascularization may not have clinically significant manifestations of postreperfusion syndrome at all. It should be remembered that even a small volume of acute arterial ischemia can cause significant postreperfusion complications due to the presence of premorbid cardiovascular pathology.

One of the manifestations of postreperfusion syndrome may be recurrence of thrombosis of the vessels of the lower extremities. The main direction of therapy of post-reperfusion syndrome is alkalization of diuresis by intravenous administration of bicarbonate solutions. Alkalization of blood allows to increase solubility of myoglobin in urine, which prevents its crystallization into tubular structures, which can obstruct the lumen of the ureters and initiate the development of acute post-renal renal failure.

***Compartment syndrome.*** Compartment syndrome occurs in post-reperfusion syndrome, which was preceded by prolonged arterial ischemia. In this case, significant interstitial edema of the soft tissues of the limb occurs, which is due to increased transudation of fluid at the level of the microcirculatory bed of the ischemic area. Interstitial edema of tissues in closed rigid fascial sheaths of the lower limb causes a significant increase in intratissue pressure. When the intratissue pressure in a closed fascial sheath exceeds the capillary perfusion pressure, oxygen transport to the tissues stops, which causes the development of tissue acidosis and ischemia, even with preserved pulsation in the main vessels. Most often, compartment syndrome in acute limb ischemia develops in the anterior fascial sheath of the thigh. The most common early manifestation of compartment syndrome is numbness in the space between the first and second toes as a result of compression of the deep peroneal nerve. To clarify the diagnosis of compartment syndrome, invasive measurement of intratissue pressure in all fascial spaces of the limb is indicated. Intratissue pressure in the limb is measured by inserting an intraarterial catheter into the corresponding fascial sheath with subsequent recording of blood pressure values in it. A diagnostically significant criterion for compartment syndrome is a diastolic pressure of more than 30 mm Hg. The presence of compartment syndrome is an indication for open surgical fasciotomy of the corresponding compartments of the limb.

## DEEP VENOUS THROMBOSIS OF THE LOWER EXTREMITY

Venous thromboembolism, including deep vein thrombosis and pulmonary embolism, is a national public health issue. Deep vein thrombosis affects more than 250,000 patients worldwide each year.

Thrombosis is the end product of two interrelated processes, platelet activation and the blood coagulation pathway. Thrombin is key to both of these processes. Homeostatic mechanisms balance these processes, but changes in internal or external factors can lead to an imbalance.

In the late 1800s, Virchow developed a simple model that laid the foundation for understanding the factors that cause thromboembolism: stasis, endothelial damage, and impaired coagulation. It is generally believed that most thrombi develop in the lower extremities and that a large percentage of them arise in the calf veins. With the increasing use of central venous catheters during long-term antibiotic therapy in the

upper extremities, there has been an increase in the incidence of thrombosis. Another source of thrombosis in the upper extremities is compression of the subclavian vein with structural narrowing in the thoracic region. Complications of deep vein thrombosis are common. Thrombi can also occur in the right side of the heart.

Thrombosis develops when the balance between coagulation and fibrinolysis shifts to favor coagulation. The thrombotic process can proceed via the intrinsic or extrinsic pathway. The extrinsic pathway begins with local cell damage, leading to the release of tissue factor and exposure of the collagen matrix, which promotes platelet aggregation. Factor VII is activated, as well as factors IX and X. Coagulation proteins assemble on the surface of the platelet membrane. Platelet adhesion is stimulated by von Willebrand factor. As a result, platelets accumulate in the form of a platelet plug. In the presence of the prothrombinase complex (factors Xa and Va, calcium, and prothrombin), thrombin is catalyzed, leading to the cleavage of fibrin peptides A and B and the activation of factor XIII, which in turn catalyzes the cross-linking of fibrin monomers. The result is a clot of activated platelets and factors Va and VIIIa.

Coagulation occurs along the intrinsic pathway by contact activation, where factor XI is converted to XIa, which in turn catalyzes the activation of factor IX to IXa and activates this conversion of factor X to Xa. Acting together on platelets, factors VIII, IXa, X, and calcium catalyze the activation of factor X to Xa and fuse with the prothrombinase complex.

Several mechanisms of anticoagulants balance clotting. Antithrombin III stops the cleavage of fibrinopeptides A and B, stops the activation of factors V and VIII, and inhibits platelet aggregation and activation, as well as factors IXa, Xa, and Xia. Activated protein C inactivates factors Va and VIIIa and reduces the acceleration of the rate of thrombin generation. Heparin cofactor II regulates thrombin generation. An extrinsic pathway inhibitor is an agent, also known as a tissue tumor inhibitor, that inactivates the activation of factor X by tissue factor VIIa but does not affect factor IX.

Plasmin is the main fibrinolytic enzyme, whose substrates are fibrin, fibrinogen and coagulation factors, which act to disrupt platelet adhesion. Plasmin creates two fragments: E and D. It is the D fragment that is measured in the D-dimer ELISA test and serves as an indicator of fibrinolysis and thrombosis. Plasmin is the product of the interaction of plasminogen and tissue plasminogen activator. Plasminogen is activated exogenously by streptokinase or urokinases, and by intrinsic factors. The balance in this system depends on thrombus formation, thrombus inhibition and fibrinolysis.

Risk factors can generally be divided into three categories: disease-specific, iatrogenic, and hereditary.

Patients with cancer, sepsis, and lower extremity fractures are examples of individuals with risk factors directly related to a disease or condition.

Iatrogenic factors include those interventions used during treatment, such as central venous catheters, prolonged immobilization, or lengthy surgery.

There are also congenital defects in the coagulation system that increase the risk of developing deep vein thrombosis. These include problems with antithrombin III, proteins C and S, factor V, and resistance to activated protein C.

Patients with a family history of deep vein thrombosis or recurrent deep vein thrombosis at a young age are candidates for specific laboratory testing. These include

NTT and platelets, ATIP antibody tests and antibody tests, protein C levels, lupus antigen mixture, fibrinogen levels, plasminogen activity, and platelet aggregation.

The thrombotic process that begins in a vein may, in the absence of timely treatment or in the presence of inadequate anticoagulation, spread to more proximal segments of the deep venous system, leading to edema, pain, and limb immobilization. The most serious complication of acute DVT is pulmonary embolism, a condition with potentially high mortality. A late consequence of DVT, especially if thrombosis occurs in the iliac-femoral veins, may be CVI and, ultimately, post-thrombotic syndrome, as a result of valvular dysfunction in the presence of persistent obstruction of the venous lumen.

***Clinical diagnosis***

The diagnosis of DVT requires close attention to threatening symptoms. Most are familiar with the Homans sign, which refers to calf pain during dorsiflexion of the foot. Although the absence of this sign does not necessarily rule out the presence of a venous thrombus, a positive Homans sign prompts additional diagnostic testing. The extent of venous thrombosis in the lower extremity is an important factor in the presentation of symptoms. For example, most calf thrombi may be asymptomatic unless they have spread proximally. Only 40% of patients with venous thrombosis have any clinical manifestations of the disease.

Disseminated venous thrombosis involving the ileofemoral venous segment results in severe swelling of the leg with marked pain and pallor, a condition known as phlegmasia blanca. As the disease progresses, such massive swelling may occur that arterial inflow may be compromised by subfascial edema. This condition results in a painful, blue leg, a condition known as phlegmasia blanca. In this course of the disease, venous gangrene may develop if blood flow is not restored.

***Diagnosis.***

Venography using contrast medium injected into the venous system is the most accurate method for confirming and localizing DVT. The superficial venous system must be occluded with a tourniquet, and the veins of the foot are injected to visualize the deep venous system. Although this is a good test for detecting occlusive and nonocclusive thrombus, it is also invasive.

Impedance plethysmography measures the change in venous capacity and rate of venous emptying during temporary occlusion of the venous system. A cuff is inflated around the upper thigh until the electrical signal ceases. When the cuff is deflated, rapid outflow and volume reduction are observed. In venous thrombosis, a lengthening of the outflow wave is noted. It is not very useful clinically for detecting calf vein thrombosis and is also a poor method in patients with previous venous thrombosis.

The D-dimer test measures cross-linked degradation products that are a surrogate for plasmin activity on fibrin. It has been shown that, when combined with clinical assessment and evaluation, the sensitivity exceeds 90–95%. The negative predictive value is up to 99%. In the postoperative patient, the D-dimer level may be elevated due to previous surgery, so a positive D-dimer test is of no use in assessing DVT in such patients. However, a negative D-dimer test in patients with suspected DVT has a high negative predictive value in this category of patients as well.

Duplex ultrasound

The current gold standard of choice for diagnosing DVT is duplex ultrasound, a method that combines Doppler ultrasound and color scanning. The advantage of this test is that it is noninvasive, comprehensive, and does not involve the risks of contrast angiography. This test is also highly dependent on the interpretation of the results by the physician performing it, which is one of the potential disadvantages. Detailed imaging begins at the calf with imaging of the tibial veins, and then proximally over the popliteal and femoral veins. A properly performed examination assesses blood flow with distal compression, which increases blood flow, and with proximal compression, which should interrupt blood flow. If any segment of the venous system being examined is not responsive to compression testing, venous thrombosis is suspected.

**Treatment**

Immediately after the diagnosis of deep vein thrombosis, a treatment plan must be drawn up. Complications of DVT include proximal thrombus spread in one-third of hospitalized patients, pulmonary embolism, and postthrombotic syndrome. In addition, the recurrence rate of lower extremity DVT without adequate treatment is 30%.

Treatment is initially initiated with intravenous heparin. Oral anticoagulation with warfarin is then initiated for months. The heparin dose should be adjusted to maintain the patient at a therapeutic level, with a RNT of 1.5 to 2.5 times. The total daily dose may be 30 to 40,000 IU/24 h. The effect of heparin on coagulation is to inhibit the activation of factors V and VIII by thrombin. Side effects of heparin treatment include bleeding, thrombocytopenia, osteoporosis, alopecia, and hypersensitivity.

Systemic intravenous unfractionated heparin is usually given for five days. Because of the risk of bleeding, which may occur in 10% of patients during the first five days of treatment, continuous monitoring is necessary. Advantages of intravenous unfractionated heparin include reduced bleeding, antiplatelet activity, reduced heparin-induced thrombocytopenia, less interference with protein C, less risk of osteoporosis, and, importantly, a more predictable dose response. Because intravenous unfractionated heparin can be administered subcutaneously and the dose adjusted according to weight, patients can be treated on an outpatient basis without requiring continuous monitoring except in special cases such as renal failure, morbid obesity, and possibly pregnancy. Warfarin should be initiated only after therapeutic anticoagulation with heparin to prevent warfarin-induced skin necrosis. Warfarin has an international normalized ratio (INR) of 2.0 to 3.0. Use is recommended throughout life in the absence of contraindications.

The widespread use of thrombolysis for the treatment of other pathological thrombosis has led to an increase in interest in thrombolysis in DVT. The supposed advantage of thrombolysis in DVT is the preservation of deep vein valve function with a subsequent reduction in the chances of developing CVD and post-thrombotic syndrome. However, the use of thrombolytic therapy in DVT is still controversial today.

One exception is the patient with phlegm, in whom thrombolysis is recommended to relieve significant venous obstruction. In this condition, thrombolytic therapy will lead to more rapid relief of symptoms and eliminate long-term consequences better than heparin anticoagulation alone. An alternative for this condition is surgical venous thrombectomy.

A common surgical method for removing blood clots from deep veins is the three-catheter method of surgical thrombectomy. The operation is performed from an incision in the area of the anterior surface of the thigh in its upper third, which makes it possible to isolate the saphenofemoral mouth, the common femoral, and the initial segment of the superficial and deep veins of the thigh. A catheter for thrombectomy is inserted proximally through the phlebotomy hole. On the contralateral limb, the saphenofemoral mouth and the common femoral vein were isolated along the anterior surface of the upper third of the thigh, and one catheter was inserted through the venotomy incision to occlude the inferior vena cava and the other to occlude the common iliac vein. The following manipulations are performed in the following sequence: catheter thrombectomy from the iliac segment affected by the thrombotic process and release of the mouth of the deep vein of the thigh. Subsequent manipulations: removal of the occlusion of the contralateral common iliac vein to obtain retrograde blood flow through the iliac segment free of thrombotic masses. After performing thrombectomy from the iliac veins, if necessary, thrombectomy from the femoral vein and the mouths of all tributaries is performed.

Regardless of the initial treatment chosen, long-term anticoagulant therapy is indicated. In most cases, a minimum duration of 3 months is recommended. However, if the patient has established hypercoagulability or has already had episodes of venous thrombosis, lifelong anticoagulant therapy is necessary in the absence of contraindications under periodic INR monitoring (INR range is 2.0 to 3.0).

***Prevention***

Patients who have undergone major abdominal or orthopedic surgery, major trauma, or prolonged immobilization for other reasons (>3 days) are at increased risk of developing DVT and venous thromboembolism.

Prevention methods can be mechanical or pharmacological. The simplest method is the patient's gait. Early activation and walking are advisable for patients in the postoperative period. Activation of the calf pump mechanism is an effective means of prevention. A patient who is activated within 24-48 hours after surgery has a low risk of developing venous thrombosis. The practice of "getting out of bed and into a chair" is one of the most thrombogenic events that can be imposed on a patient. Sitting in a chair with the legs down causes venous stasis, which in the postoperative period can easily become a predisposing factor for the development of thromboembolism.

The most common method of prevention in the surgical world has traditionally revolved around sequential compression devices that periodically compress the calves and essentially replicate the calf bellows mechanism. This has statistically significantly reduced the incidence of venous thromboembolism in surgical patients. Surgical patients are also prescribed compression hosiery, which they wear during surgery and until the time of activation in the postoperative period.

Another popular method of thrombus prevention is the use of low-dose unfractionated heparin. The classic dose that is prescribed is 5000 units of unfractionated heparin every 12 hours. However, an analysis of studies comparing placebo with this dose of heparin shows that the indicated dose of 5000 units when subcutaneously administered heparin every 8 hours, rather than every 12 hours, reduces the development of pulmonary embolism. Low-molecular-weight heparin inhibits the

activity of factors Xa and IIA, with the ratio of anti-factor Xa activity to anti-factor IIA activity ranging from 1:1 to 4:1. Low-molecular-weight heparin has a longer plasma half-life and is much more bioavailable. There is a much more predictable anticoagulant response than fractionated heparin. Various analyses, including a large meta-analysis, have clearly shown that LMWH provides equivalent, if not superior, efficacy with significantly less bleeding. In addition, LMWH shows a significant reduction in the development of venous pulmonary embolism compared with other methods. In short, LMWH is considered the optimal prophylaxis method in moderate- and high-risk patients.

## DEEP VENOUS THROMBOSIS OF THE UPPER EXTREMITY

DVT of the upper extremities is much less common than DVT of the lower extremities, accounting for only about 5% of all documented DVTs. Although not as common, it is a serious problem; pulmonary embolism occurs in about one-third of all patients with DVT of the upper extremities. DVT of the upper extremities usually occurs with thrombosis of the axillary or subclavian veins. The condition can be divided into two categories: primary/idiopathic and secondary. Etiological factors for primary DVT of the upper extremities include Paget-Schroetter syndrome and idiopathic DVT of the upper extremities. Patients with Paget-Schroetter syndrome develop thrombosis during physical exertion of the limb due to compression of the subclavian vein. The classic case is a young athlete who uses the upper limb in repetitive movements, such as swimming, which causes repeated external compression of the subclavian vein. These patients have congenital anatomic abnormalities, such as cervical ribs or myofascial bands, that may cause venous compression.

Treatment with initial thrombolysis to dissolve the thrombus followed by resection of the first rib is indicated in these patients. Idiopathic upper extremity DVT is sometimes ultimately explained by an occult malignancy, and therefore the diagnosis of idiopathic upper extremity DVT requires investigation for an undetected malignancy.

Secondary upper extremity DVT is more common and etiologies include an indwelling central venous catheter, pacemaker, thrombophilia, or malignancy. Classic physical examination findings include unilateral edema, pain, limb discomfort, erythema, and sometimes palpable tenderness. The diagnosis is confirmed by duplex ultrasound. Because the clavicle occludes the midline of the subclavian vein, venography or magnetic resonance venography may be necessary and are second-line imaging modalities. Treatment of upper extremity DVT includes anticoagulant therapy. Therapeutic dosing parameters are the same as for lower extremity DVT. Long-term complications of upper extremity DVT include recurrence and postthrombotic syndrome. Postthrombotic syndrome is treated with elevation of the extremities and compression hosiery.

## SUPERFICIAL THROMBOPHLEBITIS

Superficial thrombophlebitis may occur spontaneously in patients with varicose veins, in pregnant women or postpartum women, or in patients with rare disease states such as thromboangiitis obliterans. It may also occur at the site of venous catheters and at sites of trauma.

The presence of superficial thrombophlebitis may sometimes indicate the presence of an abdominal tumor, such as pancreatic carcinoma (Trusseau's sign).

Patients typically present with localized pain and redness of the extremities. Patients have distinct areas of induration, erythema, and tenderness consistent with dilated and thrombosed superficial veins. Over time, the induration progresses along the affected vein. Unless deep veins are involved, patients do not have significant swelling of the extremity. The presence of fever suggests septic/purulent thrombophlebitis.

Superficial thrombophlebitis should be distinguished from a number of conditions, including ascending lymphangitis, cellulitis, and erythema nodosum. In contrast to these and other disorders, superficial thrombophlebitis is localized along the superficial vein.

The primary treatment for superficial thrombophlebitis is the administration of nonsteroidal anti-inflammatory drugs, venotonics, oral anticoagulants, compression therapy, or elastic bandaging.

In most cases, symptoms improve within 7 to 10 days. Removal of the involved vein is recommended for recurrent phlebitis in the same vein segment, although the procedure is technically simpler and the incisions are usually smaller if the inflammation of the vein can be resolved (usually at least six months) before surgical removal is considered. However, surgical removal is performed immediately if septic/purulent thrombophlebitis is the indication for removal. If there is progressive proximal extension involving the saphenofemoral junction (lower limb) or the cephalosubclavian junction (upper limb), vein ligation and resection should be performed.

Alternatively, full anticoagulation may be initiated and monthly duplex scanning of the veins may be performed.

Septic thrombophlebitis requires treatment with broad-spectrum intravenous antibiotics. If rapid thrombus breakdown occurs, a short course of antibiotics and standard treatment for superficial thrombophlebitis should not be given. However, if the patient develops sepsis, the infected vein should be removed.

The vast majority of patients with uncomplicated superficial thrombophlebitis respond to conservative treatment.

**PULMONARY EMBOLISM**

Pulmonary embolism (PE) is the blockage of pulmonary artery by fat, air, tumor tissue, or thrombus that usually arises from the peripheral vein (most frequently one of deep veins of the legs).

***Etiology and pathogenesis.***

A number of causes can cause pulmonary embolism. Air embolism can occur during the placement or removal of central venous catheters during major venous surgery. Amniotic fluid embolism can occur during active labor. Fat embolism due to bone fractures causes a syndrome characterized by respiratory failure, coagulopathy, encephalopathy, and a petechial rash of the upper body. Other less common causes of pulmonary embolism include septic embolism, tumor embolism from atrial myxoma, or parasitic embolism, among others. However, DVT remains the most common source of pulmonary embolism. Approximately 60% of patients with untreated proximal DVT

of the lower extremity may develop pulmonary embolism. In less than 10% of cases, thromboembolism may cause pulmonary infarction. The pathophysiology of PE depends on both the size and frequency of emboli and the condition of the lung. Obstruction of the large pulmonary arteries leads to increased pulmonary artery pressure and acute right ventricular failure, but many of the clinical manifestations of pulmonary thromboembolism result from the release of vasoactive amines, which cause significant pulmonary vasoconstriction. The latter leads to increased physiologic dead space and systemic hypoxia due to right-to-left shunting. Reflex bronchial vasoconstriction also occurs.

The pathophysiological basis of pulmonary embolism is its obstruction by an embolus or thrombus, which causes the development of hypoxemia and pulmonary arterial hypertension. As a result, right heart overload develops and acute right ventricular failure develops. The pathophysiological basis of pulmonary infarction is also obstruction of the pulmonary artery and its small branches in the absence of formed bypasses of blood flow. In a number of cases, pulmonary infarction is apparently formed with obstruction of the bronchial arteries and their branches. In clinical practice, pulmonary embolism and its branches are more often observed without pulmonary infarction. Pulmonary infarction usually develops 24-48 hours after the moment of pulmonary embolism. Within 6-9 days, its organization begins, which in 90% of cases leads to the development of pneumonia. In 30% of patients, atelectasis of the lungs is observed, and in 5-7% - their abscessation with the formation of pleural empyema. The specified pathophysiological mechanism of the disease development determines the formation of a number of severe syndromes - acute respiratory and cardiac failure.

The development of acute respiratory failure (ARF) and acute circulatory disorders in PE is based on impaired pulmonary perfusion, which directly depends on the size and localization of the embolus. This leads to the formation of pulmonary arterial hypertension, a significant violation of ventilation-perfusion ratios and deterioration of gas exchange.

However, mechanical obstruction of pulmonary circulation by an embolus is not the only one at the basis of the pathogenesis of ARF and determining the severity of the patient's condition. The reactions of the lungs and circulatory system to the embolus itself and the production by it and blood cell aggregates of biologically active substances (serotonin, histamine, prostaglandins, eicosap-noids, etc.) are of great importance. In response to this, generalized arterio-spasm occurs in the small circle of blood circulation and collapse of the large circle of vessels. Clinical signs of this are an increase in pressure in the pulmonary artery against the background of a decrease in systemic arterial pressure. As a direct reaction to biologically active substances, the formation of bronchiolospasm occurs - the Euler-Lillestrand reflex, which leads to obstructive pulmonary disease. Acute right ventricular failure is formed due to its intense work to overcome high pulmonary resistance. Impaired pulmonary circulation leads to a decrease in the filling of the left ventricle and a syndrome of small cardiac output. This causes a collaptoid state, causing a decrease in coronary blood flow, which can manifest as severe myocardial ischemia, up to the development of cardiac fibrillation.

The embolus is affected by the fibrinolytic system of the lungs, which contributes to its dissolution. In case of violation of the coagulation properties of the blood, the reverse phenomenon may occur, when the size of the embolus increases due to the deposition of new layers of fibrin on them.

The role of reflex reactions in the pathogenesis and development of severe cardiopulmonary failure is undeniable, as evidenced by the following data: with massive PE, only 16.2% of patients have its fulminant form, acute - 56.5% and a quiet course - in 27.3% of patients, while with thromboembolism of small branches, 20-30% of patients develop a clinic of acute pulmonary heart, which cannot be associated with the volume of lung tissue excluded from the circulation. The combination of mechanical factors and reflex spasm of pulmonary vessels leads to a decrease in blood pressure due to a decrease in blood flow to the left heart. These mechanisms of pathogenesis of PE lead to the formation of acute pulmonary insufficiency and circulatory disorders. Every second patient with PE is in a state of shock.

***Clinical.***

The signs and symptoms associated with PE are notoriously vague. Dyspnea and chest pain are present in 75% of patients with PE. However, these symptoms are nonspecific, especially in patients who may have cardiopulmonary disease. Tachycardia, tachypnea, and altered mental status are very alarming signs in a high-risk group. The classic triad of dyspnea, chest pain, and hemoptysis is present in only 15% of patients with pulmonary embolism. Pleural friction rubs and S1Q3T3 morphology on electrocardiography are even less common.

To determine the treatment tactics and predict its results, it is advisable to use the classification of PE of the European Society of Cardiology, according to which thromboembolism of the branches of the LA is divided into non-massive and massive. The diagnosis of massive thromboembolism is established in the presence of arterial hypotension or cardiogenic shock.

By localization, one or two are distinguished. By the nature of the clinical course:

- acute (lasts hours);
- subacute (days)
- recurrent.

Massive PE is considered to be a lesion of more than 50% of the pulmonary vascular bed, and submassive - less than 50%.

***Diagnostics.***

Diagnosis of PE begins with a preliminary assessment of the level of probability for all patients. The Wells criteria (or modified/dichotomous Wells criteria - Wells scale) and the Geneva criteria are two of the most widely known and approved diagnostic scoring systems. They use a combination of physical examination, medical history, and vital signs to predict the likelihood of developing PE.

Chest X-ray. The pathognomonic sign of PE is Westermark's sign - an increase in the transparency of the lung tissue due to the impoverishment of the vascular pattern. However, data on the frequency of its detection are quite contradictory and range from 6 to 90%. The reasons are both the difficulties of subjective assessment of lung transparency, especially against the background of concomitant pulmonary pathology, and the transient nature of this sign. It has been experimentally proven that

Westermark's sign disappears 24-36 hours after PE, despite the ongoing occlusion of the pulmonary arteries. Specific for massive PE is the appearance of signs of cor pulmonale: dilation of the pulmonary trunk and main pulmonary arteries, dilation of the right heart cavities, dilation of the vena cava. The degree of dilation corresponds to the volume of the pulmonary bed excluded from the circulation and the magnitude of pulmonary hypertension. These signs are more stable than the Westermark symptom and are detected in approximately 2/3 of patients regardless of the occlusion period. One of the manifestations of thromboembolism of the main or partial arteries is considered to be a decrease in the size of the pulmonary field and a high position of the dome of the diaphragm due to bronchospasm, pleurisy and stagnation in the liver and spleen. However, this symptom is unstable, it is observed in 39% of patients. A frequent radiological finding is pulmonary infarction or infarct pneumonia. For the defeat of small branches of the pulmonary artery, unilateral or bilateral pleural exudate, disc-shaped atelectasis, basally located obscurations are characteristic. A significant increase in the sensitivity of the method in the diagnosis of PE can be achieved through the use of special computer programs for automated assessment of lung tissue transparency on conventional radiographs, especially during dynamic observation. However, even in this case, an X-ray examination cannot be a reliable method of topical diagnosis.

Electrocardiography: ECG changes in PE are caused by acute overload of the right ventricle and consist in the deviation of the electrical axis of the heart to the right, the shift of the transition zone to the left, the appearance of deep Sb Q3, negative T3, a tendency to ST3 elevation and ST1, 2 depression (McGinn-White syndrome), the formation of negative TV1/V3, blockade of the right bundle branch block, the appearance of rhythm disturbances and atrioventricular conduction. The sensitivity and specificity of ECG during the diagnosis of PE does not exceed 60-70%. Simultaneous registration of several electrocardiographic signs of acute or chronic pulmonary cor pulmonale indicates the presence of pulmonary embolism, while their absence does not deny this diagnosis and requires the use of other methods.

Arterial blood gas analysis indicates hypoxia and often respiratory alkalosis or an increased arterio-alveolar oxygen gradient.

D-dimer levels are elevated in both pulmonary embolism and acute DVT, but this test is not specific enough to be the first line of diagnosis. D-dimer testing is used to rule out PE in patients with a low probability of DVT. The use of D-dimer in the diagnosis of PE requires the use of a highly sensitive assay (e.g., an improved turbidimetric assay or ELISA).

Cardiac troponins may be elevated in acute massive or submassive PE that results in hemodynamic instability (systolic blood pressure <90 mm Hg) and right heart deformity leading to myocardial ischemia. Troponin is released from damaged myocardial cells. In acute PE, elevated troponins indicate myocyte ischemia and microinfarction due to acute cardiac deformity of the right ventricle. Approximately 30% to 50% of patients with massive PE will have elevated troponins I and T. These indicate poorer right ventricular function and a high incidence of complications. Normal troponin T levels have a 97%-100% negative predictive value for in-hospital mortality. These two biomarkers are not part of routine algorithms.

CT angiography is now the best imaging method for diagnosing PE, due to its wide availability, sensitivity and specificity. CT angiograms (CTA) are more accurate than ventilation-perfusion scanning, and compared to pulmonography, CTA is a less invasive and less expensive method. Among the methods for diagnosing PE, the second most informative is lung perfusion scintigraphy using human albumin microspheres labeled with Tc99 with a half-life of 6.5 hours. Perfusion scintigraphy provides the ability to quantitatively assess the degree of pulmonary perfusion disorders. Perfusion deficit is defined as the product of the degree of decrease in radioactivity and the area of the accumulation defect as a percentage of the total lung area. In this case, the area of the right lung is 55%, the left lung is 45%. If there are several defects, their sum is determined. To level the topographic and anatomical features of the lungs, the authors of the method proposed to take its arithmetic mean as the total perfusion deficit. The main disadvantages of the given method are its cumbersomeness and subjective nature, therefore, for the quantitative assessment of scanograms, it is recommended to use computer software. Sensitivity and specificity of perfusion scintigraphy in the diagnosis of PE increases to 98%. Due to the low sensitivity and specificity, transthoracic and transesophageal echocardiography have limited diagnostic value for PE. Echocardiography is indicated for patients with suspected massive PE to assess the function of the right heart for hemodynamically significant PE, when the introduction of a contrast agent is undesirable. For critically ill, non-transportable patients, echocardiography can be an auxiliary method in the diagnosis of PE, indicating right ventricular dilatation or hypokinesia. Acute changes in right ventricular pressure, size, and function are usually seen, indicating both increased right ventricular tension and pulmonary artery pressure. These changes are suggestive of PE in the absence of alternative diagnoses. While of limited value in the diagnosis of PE, echocardiography has great prognostic value in risk stratification of patients with acute PE. Right ventricular dysfunction or dilatation in acute PE is associated with worse outcomes, including increased mortality.

Pulmonography is performed in patients with acute massive PE, during interventions on the inferior vena cava, when planning pulmonary thrombectomy or thrombolysis. However, because angiography is an invasive procedure, the risk of complications and mortality is increased. The mortality rate from angiography has been reported to be 0.5%, while 1% of patients may have serious complications, including arrhythmias, hypotension, bleeding, and nephrotoxicity. In the absence of a higher standard to which to appeal, it is not possible to discuss the specificity and sensitivity of pulmonary angiography based solely on generally accepted statements. Pulmonary angiography is not recommended except in certain circumstances, such as poor visualization with perfusion scintigraphy or catheter-directed thrombolysis.

Among the methods proposed for quantitative assessment of angiopulmonograms, the most widespread is the Miller angiographic index (1971), which simultaneously takes into account the localization of emboli, topographic and anatomical features of the lungs and the intensity of contrast of distal pulmonary vessels. The value of the Miller index consists of two parts. The first determines the level of PE, based on the number of segmental branches distal to the lesion. In the left lung, 7 segments are distinguished, in the right lung 9 segments. The presence of

occlusion or filling defect in the segmental artery is counted as one point, partial - from 2 to 4, in the left main pulmonary artery - as 7, in the right - as 9 points. Damage to the pulmonary artery trunk is 16 points. The second part of the Miller index takes into account the level of peripheral perfusion in the upper, middle and lower parts of the lungs. The absence of contrast-enhanced vessels is equal to 3 points, a significant decrease in their number is equal to 2, and a moderate decrease is equal to 1. Thus, the maximum value of the perfusion index is 18, and the Miller angiographic index is equal to 34 (16+18) points.

Recently, the effectiveness of magnetic resonance angiography (MRA), magnetic resonance venography (MRV), or a combination of these two methods in the diagnosis of acute PE has been studied. In general, MRA and MRA-MRV have been recognized as uninformative methods for the diagnosis of PE. After a multicenter study, it was found that PE was diagnosed in 57% of patients using MRA. MRA and MRI in combination have a diagnostic sensitivity of 92%, but only about half of the patients had adequate imaging of the study results. Such results are associated with technical difficulties in identifying the abrupt cessation of contrast in a thrombosed vessel and in obtaining unclear images of the thoracic vessels due to respiratory movements of the chest. Thus, MRA and MRI can be recommended for use only in medical institutions with extensive experience in this field.

But, despite the rather widespread opinion that magnetic resonance imaging (MRI) cannot be used at all for the study of pulmonary pathology, experience shows that it can be used to detect emboli in the trunk of the pulmonary artery and its branches of the I-II order in the form of low-intensity intravascular structures. However, it should be noted that the capabilities of MPT and the detail of these images are inferior to CT. In addition to topical diagnosis of PE, MRI can be used to search for the source of embolism in peripheral veins. MRI has proven to be a fairly effective method for the diagnosis and differential diagnosis of chronic pulmonary hypertension. The optimal methods for this are pulsed spin-echo sequence and cine-MRI, which provide high-quality visualization of all parts of the heart and large vessels. Characteristic features of chronic postthromboembolic pulmonary hypertension are right ventricular dilatation and hypertrophy (wall thickness >7 mm), pulmonary artery and branch dilatation, high systolic signal intensity in the pulmonary artery on spin-echo images, tricuspid and pulmonary regurgitation. However, in general, MRI is inferior to CT in diagnosing PE.

**Treatment.**

The main tasks of PE treatment:

- eliminate the threat of death from cardiopulmonary failure in the acute phase of the disease;
- normalize lung perfusion and prevent severe pulmonary hypertension in the long term;
- ensure reliable prevention of recurrent embolism.

Currently, the following methods of PE treatment are the most justified:

1. Conservative therapy:
   - anticoagulant;
   - thrombolytic.

2. Surgical treatment:
   - Thrombectomy under conditions of artificial blood circulation (AC).
   - Thrombectomy without the use of AC.
   - X-ray endovascular method.
3. Combination of conservative and surgical therapy.

The main treatment method for most patients with PE is conservative therapy. Anticoagulant therapy is the method of choice in the treatment of patients with non-massive PE. In massive PE, thrombolytic therapy is indicated.

The appointment of low-molecular-weight heparin is started as soon as the diagnosis of PE is established after the initial stabilization of the patient with ventilatory support and vasopressor therapy. Low-molecular-weight heparins allow the body's fibrinolytic system to function unhindered. However, conventional therapy with heparin and indirect anticoagulants only prevents further thrombus formation, without affecting the degree of pulmonary artery occlusion.

Thrombolysis is indicated for large clots, severe respiratory failure, hemodynamic instability, or right-sided heart failure. Compared with heparin, thrombolytic therapy in the first 24 hours accelerates the resolution of pulmonary embolism. The disadvantages of thrombolytic therapy are its high cost and increased risk of serious bleeding. The use of thrombolytic therapy in PE has several advantages. Thrombolysis has been shown to reduce right ventricular strain, pulmonary hypertension, and perfusion deficits that persist after standard heparin anticoagulant therapy in massive PE. Catheter-directed thrombolysis may be safer than systemic administration in PE, although data on this are limited.

The prognosis of PE largely depends on the degree of obstruction of pulmonary blood flow. In patients who did not receive thrombolytic therapy, the total number of deaths and recurrence of PE in the near future is about 10%, and the mortality rate during the first year is 19%. In approximately 50% of cases of massive PE, death occurs in the first 15 minutes. About 2/3 of patients die in the first 2 hours due to cardiogenic shock. Therefore, the decisive factor is not so much the method of treating patients as the time of initiation of treatment. In later terms, the main cause of death is infectious and cardiovascular complications. Even a partial reduction in the size of thrombotic occlusion in massive or submassive PE with obstruction of more than 50% of blood flow in the pulmonary artery allows you to significantly improve the condition of patients and the prognosis of the disease.

However, the recommended multi-hour infusion of streptokinase (100,000 U/h for 24 h) or urokinase is not effective enough and can cause hemorrhagic complications. Recently, intensive research has been conducted on the use of tissue plasminogen activator for thrombolytic therapy in patients with PE. Tissue plasminogen activator is used in the form of intravenous administration of 100 mg over 2 h as an alternative to long-term infusion of urokinase and streptokinase. The efficacy and safety of bolus administration of tissue plasminogen activator have been studied in several multicenter projects. The results show that bolus administration of 50 mg of tissue plasminogen activator is accompanied by a less pronounced decrease in fibrinogen levels and a smaller increase in its content compared to infusion of 100 mg of tissue plasminogen activator for 2 hours, with the same frequency of hemorrhagic

complications. According to the results of clinical studies, tissue plasminogen activator has a number of advantages compared to streptokinase, first of all, faster dissolution of the thrombus. Of particular importance is the absence of antigenic properties of tissue plasminogen activator. With its use, the risk of allergic reactions disappears and the risk of developing symptomatic arterial hypotension, which quite often occurs with the introduction of streptokinase, is significantly reduced. In addition, tissue plasminogen activator is the drug of choice in patients with repeated administration, in those who have previously used streptokinase or its analogues (within the previous 2 years).

Before starting therapy, in addition to cannulation of the vein, the patient must be connected to a cardiac monitor. Despite the large number of available thrombolytic drugs and those that are at the testing and development stage, streptokinase and tissue plasminogen activator are mainly used in clinical practice. In the case of using streptokinase, 1,500,000 IU of the drug is diluted in 200 ml of saline or 5% glucose solution and administered intravenously drip for 60 minutes. Prophylactic use of glucocorticosteroids in patients with PE is ineffective.

When using tissue plasminogen activator (actilize), the generally accepted regimen is an accelerated administration regimen, which involves the administration of the first 15 mg of the drug intravenously as a bolus, after which intravenous infusion is started at a dose of 50 mg (or 0.75 mg/kg, but not more than 50 mg) over the first 30 minutes and the last 35 mg (or 0.5 mg/kg, but not more than 35 mg) is administered over the next 60 minutes. In patients with a body weight of less than 65 kg, the total dose of the drug should not exceed 1.5 mg/kg. Treatment tactics for PE are determined by the severity of the patient's condition, which, in turn, depends on the size of the thrombus and the degree of reduction in blood flow in the LA.

The fulminant form of PE with the onset of shock requires urgent intensive therapy, including the infusion of plasma substitutes, sympathomimetics and thrombolytic drugs. In such cases, bolus intravenous administration of streptokinase at a dose of 3 million units or 50 mg of tissue plasminogen activator is recommended. If the use of the drug does not give the desired result: Blood pressure remains below 90 mm Hg, partial tension of oxygen in arterial blood is less than 60 mm Hg, and diuresis is less than 20 ml/h, it is worth considering the possibility of surgical intervention.

In case of submassive PE with obstruction of about 50% of the pulmonary vascular bed, the condition of patients is usually considered severe. For proper monitoring of such patients, careful monitoring of hemodynamic parameters (blood pressure, heart rate, central venous pressure, pressure in the right ventricle and pulmonary artery) and indicators of blood gas and acid-base composition is required. In the absence of hypercapnia, inhalation of oxygen in high concentrations (4-8 l/min) should be used to combat hypoxia.

Morphine is prescribed with caution to eliminate pain. Systemic hypotension in PE is an absolute indication for the use of sympathomimetics, noradrenaline or dopamine, starting with low doses of 2-5 μg/kg/min. Glucocorticosteroids are prescribed. A slow infusion of dextran is performed to increase cardiac output and improve microcirculation. Antithrombotic therapy is started with a bolus injection of heparin at a dose of 10,000-15,000 U followed by an intravenous infusion of 1,000-1,500 U/h. If the patient's condition does not stabilize as a result of the measures taken

within 2 hours, thrombolytics should be used. In cases where the clinical diagnosis of PE is beyond doubt, the administration of thrombolytic drugs is started without angiopulmonographic confirmation. The time window for thrombolysis in patients with PE is not precisely defined, but it is believed that the use of thrombolytic agents can be effective within 2 weeks of the onset of the first symptoms of the disease. The initial loading dose of streptokinase is 250,000 U, followed by an infusion of 100,000 U/h for 12-24 hours. The use of urokinase is started with a bolus dose of 4,400 U/kg, then switched to an infusion of 4,400 U/kg/h for 12-24 hours. However, the most effective means of thrombolytic therapy for PE today is tissue plasminogen activator. The drug is prescribed in a dose of 100 mg, which is administered intravenously over 2 hours. Heparin infusion is continued for 7-10 days. In the last 2-4 days, indirect anticoagulants are prescribed in parallel under the control of the prothrombin index. These drugs are used for 3-6 months.

Contraindications to thrombolysis:

- Acute bleeding and postoperative condition.
- Hemorrhagic stroke of any age.
- Intracranial tumor or aneurysm.
- Ischemic stroke less than 2 months old.
- Traumatic brain injury within 1 month of therapy.
- Hemorrhagic diathesis.
- Suspected aortic dissection.

These contraindications should be considered in each individual case in the context of the risk-benefit ratio. In patients with severe clinical PE, most contraindications are considered relative, with the exception of those related to malignant neoplasms, especially brain tumors.

Complications of thrombolytic therapy:

- bleeding;
- hypotension;
- allergic reactions.

Currently, the method of choice in patients with thromboembolism of small branches of the pulmonary artery is a bolus administration of heparin 10,000-15,000 units with subsequent intravenous infusion of 1000-1500 units/hour for several days. Such therapy requires careful monitoring of hemostasis parameters, primarily the partial thromboplastin time (PTT), which should increase by 1.5-2 times from the initial value or the upper limit of normal. Further tactics include the appointment of indirect anticoagulants for 3-6 months under the control of the prothrombin index. In the presence of contraindications to anticoagulant therapy or recurrence of PE, despite ongoing heparin therapy, the method of choice is the implantation of a filter in the inferior vena cava. If the predicted effectiveness of thrombolysis does not seem undeniable, before starting it, the doctor should compare the expected effect of treatment with the risk of possible complications and relative contraindications.

If the source of PE is the inferior vena cava system, implantation of a cava filter is recommended. In patients with floating thrombi, this procedure is performed before the start of thrombolysis. Preference is given to modern cava filters of the Greenfield,

Gunther or Osot type, which can be installed through the jugular vein under local anesthesia.

Installation of a cava filter is indicated for the enlargement of the venous thrombus despite adequate anticoagulant therapy, in the presence of contraindications to anticoagulant therapy, in cases of complications of anticoagulant therapy, in patients with recurrent deep vein thrombosis or PE despite anticoagulant therapy. Temporary or permanent cava filters can also be installed for prophylactic purposes in high-risk patients in the presence of serious injuries or inoperable oncological diseases.

However, the installation of cava filters does not completely eliminate the risk of PE, moreover, it may increase the risk of deep vein thrombosis, and does not prevent the risk of post-phlebitis syndrome at all. Studies have shown that patients with installed cava filters had a significantly higher risk of developing deep vein thrombosis than patients who received only a course of anticoagulant therapy. There was no difference in the frequency of post-phlebitis syndrome in the study groups. However, in the group of patients with installed cava filters, PE was registered much less often than in the comparison group (patients receiving only anticoagulant therapy). Current global recommendations provide for the appointment of a standard course of anticoagulant therapy to patients with cava filters in the absence of contraindications.

Alternative venography methods have been successfully used to place filters, in particular, filter placement under ultrasound guidance, both intravascular and transabdominal. Kassavin and colleagues recently described the use of intravascular ultrasound-guided placement of cava filters in 20 patients, which, compared with traditional techniques, reduces the risk of prolonged radiation exposure and exposure to nephrotoxic contrast agents, thus also reducing the duration and overall cost of the procedure.

Surgical treatment of PE is indicated in the ineffectiveness of conservative therapy in patients with massive PE, hypotension, requiring the introduction of vasopressors and in the case of the formation of post-thromboembolic pulmonary hypertension. Also, percutaneous or open thrombus extraction is indicated for hemodynamically unstable patients in the presence of contraindications to thrombolysis or the latter has been ineffective. The open technique of pulmonary embolectomy is used in patients with uncontrolled hypotension, when it is impossible to perform transcatheter pulmonary embolectomy, in the presence of foreign bodies and tumor embolism. The catheter technique includes mechanical thrombolysis or removal of an intact pulmonary embolus using an aspiration device.

**Prognosis.**

PE is one of the most common causes of in-hospital mortality, which can be prevented and prevented. Prevention of acute venous thrombosis and early diagnosis through selective screening of high-risk patients are important steps towards reducing the incidence. The installation of cava filters in a certain group of patients prevents the occurrence of pulmonary embolism, but has no relation to the treatment of the underlying disease (venous thromboembolic disease) and does not prevent the further development of acute venous thrombosis and post-thrombotic syndrome.

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**Tests**

1. Earliest sign of deep vein thrombosis is:
   A. Calf tenderness
   B. Rise in temperature
   C. Swelling of calf muscle
   D. Homan’s sign
   E. Varicose veins
2. A 64-year-old man is admitted 14 months after a femoropopliteal bypass graft procedure with a cold foot and no graft pulse. Urokinase infusion was started. Which of the following statements regarding management is true?
   A. Clot lysis is accomplished in 25% of patients
   B. After successful clot lysis, surgical revision of the opened graft should be considered only if early reocclusion occurs
   C. Urokinase is less successful in lysing acute thromboses of prosthetic grafts than those of vein grafts
   D. Streptokinase is the preferred thrombolytic agent when treating graft occlusions
   E. With optimal treatment, a 20% reocclusion rate is expected within 1 year
3. Cockett’s and Todd’s operation is for:
   A. Burger’s disease
   B. Lymphedema precoxa
   C. AV fistula
   D. Varicose veins
   E. Deep veins thrombosis
4. The most reliable sign of deep vein thrombosis is:
   A. Swelling of a limb distally
   B. Positive Homan’s sign
   C. Calf tenderness
   D. Dilation of superficial veins
   E. Teleangiectasia
5. Brodie-Trendelenberg test is for:
   A. Testing sapheno-femoral junction incompetence
   B. DVT
   C. Mid thigh perforators in varicose veins
   D. Calf Perforators
   E. Thrombosis of superficial veins
6. White leg is due to:

A. Femoral vein thrombosis and lymphatic obstruction
B. Deep femoral vein thrombosis
C. Lymphatic obstruction only
D. None of the above
E. Superficial veins obstruction

7. Brodie-Trendlenburg test demonstrates:
A. Mid-thigh perforators
B. Deep vein thrombosis
C. Sapheno-femoral incompetence
D. Calf perforators
E. Superficial veins thrombosis
8. Most common source of pulmonary embolus:
A. Iliofemoral veins
B. Deep veins of leg
C. Deep veins of calf
D. Popliteal veins
E. Major saphenous vein
9. All of the following are correct about axillary vein thrombosis except:
A. May be caused by a cervical rib
B. Treated with IV anticoagulant
C. Embolectomy is required in almost all cases
D. May occur following excessive exercise
E. Often is catheter-induced
10. Best method of diagnosing deep venous thrombosis is:
A. Venography
B. Duplex study
C. Plethysmography
D. Radionuclide scan
E. Rheovasography

**Keys for tests**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| C | E | D | C | A | B | C | C | C | B |

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Normal Anatomy of arterial and venous systems.<br>– Normal peripheral blood circulation<br>– Pathophysiology and etiology of acute ischemic syndrome.<br>– Mechanism of blood coagulation.<br>– Special investigations in patients with thrombosis. | Make a scheme of arterial and venous systems of lower extremities.<br>Draw a scheme of normal blood outflow from the legs.<br>Make a scheme of the pathogenesis of acute ischemic syndrome.<br>Prepare a scheme of normal blood coagulation. |

| | |
|---|---|
| | Indications to the different methods, their sensitivity and specificity. |
| Study:<br>– Clinical symptoms and signs of acute ischemia syndrome.<br>– Clinical examination and special investigations in patients with acute thrombosis.<br>– Diagnostic value of D-dimer evaluation.<br>– Principles of surgical treatment of patients with vascular thromboses.<br>– Principals of anticoagulant therapy.<br>– Complications of thrombolytic therapy. | Make a scheme of acute ischemia syndrome.<br>May a list of indications and contraindications to various instrumental investigations.<br>Positive and negative predictive value of D-dimer assessment.<br>Make the schemes of surgical accesses to vessels for thrombectomy and embolectomy.<br>Make the scheme of anticoagulant therapy. |

## TOPIC 10. IATROGENIES IN SURGERY.

**Relevance of the topic.**

Recently, an increase in iatrogenic diseases has been recorded, of which 45-48% are associated with surgical treatment, especially in abdominal surgery. This is due to the growing operational activity and more aggressive interventions. Knowledge of typical iatrogenies and their legal bases will help to avoid or minimize them in practice.

**Goal:**

- Learn the basic terminology and classification of iatrogens in treatment
- Know the causes of iatrogens and their consequences
- To master the main types of iatrogenic surgical and resuscitation profiles
- Master the main types of iatrogenia of prophylactic and diagnostic profile
- Learn the mechanisms of iatrogens in the treatment of patients
- Know the legal basis of iatrogens.

**The student should know:**

- Definition of the concept of iatrogeny.
- Basic classifications of iatrogens.
- The main iatrogenies are related to the diagnosis of diseases.
- The main iatrogenies are related to the treatment of patients.
- The main iatrogenies are related to resuscitation measures.
- The main iatrogenies are related to radiation therapy.
- Legal basis of iatrogenia.

**The student should be able to:**

- Interpret iatrogenies depending on their detection.
- Operate with basic terminology and classification.
- Minimize complications in treatment.
- Choose tactics to minimize the negative consequences of treatment

**Content of educational material.**

| Term | Definition |
|---|---|
| Iatrogenies | These are pathological conditions (diseases, complications, injuries) caused by medical interventions. |
| Surgical iatrogenies | Iatrogenic diseases that arose as a result of surgical intervention. |
| Idiosyncrasy | Pathological reaction of the body to the action of medicinal drugs, which has a non-specific mechanism. |
| Reanimation iatrogenies | A type of iatrogenic pathology, the development of which was caused by resuscitation or intensive therapy of terminal conditions. |

**Topic content:**

**Iatrogenies** are pathological conditions (diseases, complications, injuries) caused by medical interventions. Thus, iatrogenic means the negative effects of medical

influences, which are expressed in the emergence of new diseases in the patient or in complications and worsening of an already existing disease.

The term "iatrogenia" began to be widely used in medical practice after it was published in 1925. Bumke's article "The doctor as a cause of mental disorders".

The term iatrogenia is of Greek origin: iatros (physician) and genes (origin). This term means a psychogenic illness or neurosis arising under the influence of the doctor's actions, behavior or words.

At the beginning of the 20th century Swiss psychiatrist E. Bleuler wrote that the disease can be aggravated, complicated or arise as a result of "undisciplined thinking of the medical worker".

This is most often observed during the examination of the patient, when early signs of the disease are detected and the patient is worried about the change in well-being and is very sensitive to the words of the medical worker.

By the middle of the 20th century Iatrogeny meant diseases arising as a result of the careless statements of a medical worker. Over time, a tendency to call all diseases resulting from a medical error iatrogenic was noticed.

However, not all diseases resulting from medical interventions can be attributed to medical errors. Iatrogeny includes diseases or deaths that occur as a result of the side effects of pharmacological drugs.

Iatrogenic diseases and reactions are listed in the International Classification of Diseases (ICD), where they can be found both in three-digit headings and in an additional classification.

When analyzing iatrogenic pathology, the following features are determined: - medical-biological (pathogenetic); - medical and social; - legal

**Medical and biological features of iatrogeny.** The development of iatrogenies depends on the individual characteristics of the patient, his reactivity, stress resistance, individual inadequacy to the actions of pharmaceuticals, other methods of treatment and diagnosis.

The medico-biological features of iatrogenia include the honest mistake of the doctor, which is connected with his insufficient qualifications, as well as when new methods of diagnosis or treatment are introduced into medical practice.

**Medical and social features of iatrogens.**

The development of iatrogenia is possible due to errors in the diagnosis of the disease associated with the use of old equipment that has expired.

With insufficient financing of the health care industry, such iatrogenies are no longer a rare case in medical practice.

Legal features of iatrogenic diseases are related to the need to compensate for the damage caused to human health as a result of medical intervention.

Illness or death of a patient caused by medical negligence also refers to iatrogenic pathology. In this case, we should be talking not only about economic sanctions, but also about criminal liability. Complications or development of the disease as a result of failure to provide medical assistance are not considered iatrogenic.

**Classification of iatrogens**

To date, there is no single, generally accepted option for the classification of iatrogens, therefore, in the field of health care, several options for the classification of iatrogens are used:

- according to the etiology of the disease;
- according to the International Classification of Diseases;
- according to Kalytiaevsky;
- according to Rykov.

**Classification by etiology of the disease.** In the classification, iatrogenic pathologies are distinguished, based on the etiology of the disease, the importance for the course of the disease and thanatogenesis. According to this scheme, the following classes of iatrogens are distinguished:

- Iatrogenies associated with preventive measures.
- Iatrogenies related to the diagnosis of diseases.
- Iatrogenies associated with the use of pharmacological drugs.
- Iatrogenies caused by radiation diagnosis or treatment.
- Iatrogenies associated with the use of medical tools and materials (introduction of alloplastic materials into the cardiovascular system, vascular catheterization, use of pacemakers, etc.).
- Complications that occur during blood transfusion.
- Death due to narcosis.
- Iatrogenies that arise as a result of surgical interventions.
- Iatrogenies of a deontological nature.

**Classification according to the International Classification of Diseases.** The classification of iatrogens was developed based on the provisions of the International Classification of Diseases and is widely used in a number of countries. The following classes of iatrogenic diseases are distinguished:

- Iatrogenies arising from surgical diseases and surgical interventions, specifying the disease and the nature of the surgical intervention.
- Iatrogenies caused by medical treatment.
- Iatrogenies caused by preventive measures.
- Iatrogenies of diagnostic measures.
- Death from anesthesia, including with premedication.

**Classification according to Kalytiaevsky.** The peculiarity of this classification is that each class of iatrogenic pathologies is divided into subclasses.

I. Iatrogenies associated with treatment.

1.1. Medicinal iatrogenes.

G1.1. Iatrogenies' caused by the side effect of medications or their individual tolerance.

G1.1. Iatrogenies' caused by the side effect of medications or their individual tolerance.

G1.2. Iatrogenies caused by inadequate or erroneous use of pharmacological drugs.

1.2. Surgical iatrogenies.

G2.1. Iatrogenies, which are due to the risk and difficulty of surgical intervention or anesthesia.

I.2.2. Iatrogenies caused by errors in the technique of surgery or anesthesia, incorrectly selected tactics or method of surgical intervention.

1.3. Physical methods of treatment.

I. 3.1. Side effect of radiation and other types of physical treatment methods and their individual tolerance.

I.3.2. Iatrogenies caused by inadequate and erroneous use of radiation and other physical methods of treatment, equipment malfunctions.

1.4. Other iatrogenic diseases associated with treatment.

II. Iatrogenies are related to the diagnosis of diseases.

II.1. Diseases caused by the risk of using the diagnostic method itself or the diagnostic tools used.

II.2. Diseases caused by errors during diagnostic manipulations, malfunctions in the equipment. Redundant diagnostic examination.

III. Iatrogenies associated with preventive measures (vaccinations).

III.1. Iatrogenies due to the risk of side effects of the drug or the method itself.

III.2. Diseases associated with errors during preventive measures.

IV. Information iatrogenies.

IV. Diseases caused by the actions of a medical worker on the patient's mental state.

IV. Diseases caused by the actions of a medical worker on the patient's mental state.

IV.. Self-medication (use of pharmacological drugs that were not prescribed by a doctor).

V. Iatrogenic pseudodiseases.

VI Diseases that were registered by medical statistics, however, did not cause unwanted consequences in patients.

VII Iatrogenies, which were the result of an erroneous diagnosis, which caused harmful consequences for the patient's health.

VIII. Other iatrogenies.

**Classification according to Rykov**

In connection with the possible introduction of insurance medicine and the development of the private sector of the health care industry, the classification of iatrogenes proposed by V.A. Rykov. This classification stipulates economic sanctions in the event of an iatrogenic pathology.

- **1st group**. Iatrogenies that occur at the previous stages of treatment and therefore are not related to the medical and preventive institution that established the diagnosis of iatrogenic disease.
- **2nd group**. Iatrogenies caused by an abnormal reaction of the body to pharmacological drugs and other methods of treatment. Iatrogenies of this group do not belong to "doctor's mistakes", so no economic sanctions are foreseen for the medical institution.

- **3rd group**. Iatrogenies caused by diagnostic and treatment errors. The medical institution must fully bear economic or other responsibility. In Ukraine, the mechanism of economic sanctions has not been developed.
- **4th group**. Iatrogenies that do not play a certain role in the pathogenesis of the disease, that is, an iatrogenic disease is superimposed on a background or concomitant disease. The majority of specialists adhere to the point of view of the lack of economic responsibility of a medical institution when a patient is diagnosed with iatrogeny of the 4th group.
- **5th group**. Iatrogenies arising as a result of self-medication. There are no economic sanctions against the medical institution.

**Iatrogenies associated with preventive measures**

Most often, iatrogenic pathologies associated with preventive measures are caused by side effects of vaccines or the risk of side effects of the vaccination method itself. Reactions can be unexpressed and mild, but sometimes dangerous conditions for life and health occur: encephalopathy, anaphylactic shock. This group of iatrogens also includes complications that arise when using substances to fight disease carriers and pathogens. Iatrogenies associated with preventive measures can occur even with the correct use of high-quality vaccines. This is observed with high individual reactivity, idiosyncrasy to vaccines or serums. Iatrogenies can occur with careless organization of preventive measures.

This group of iatrogenic pathologies includes diseases associated with:

- wrongly chosen route of administration of vaccines and serums;
- using expired biological material for vaccinations;
- neglect by the medical staff of the patient's allergic or general history;
- administration of the drug without prior desensitization;
- overdose.

Iatrogenies associated with preventive measures can be observed due to overdose of vitamin D during the prevention of childhood rickets. Severe iatrogenic poisonings are observed due to violations of safety techniques when using toxic substances.

**Iatrogenies associated with the diagnosis of diseases**

There are two main groups of iatrogenic diseases associated with the diagnosis of diseases:

- iatrogenic pathologies, which are the result of establishing a false diagnosis;
- iatrogenic diseases caused by complications from the diagnostic procedure.

False diagnoses may not lead to iatrogenic pathology. Most often, this happens as a result of a false diagnosis of appendicitis and, as a result, appendectomy. According to statistical data, up to 20% of patients were incorrectly diagnosed with appendicitis. In most such cases, iatrogenic pathology does not occur.

In other cases, a false diagnosis leads to iatrogenic consequences. Errors in the diagnosis and, as a result, erroneous treatment negatively affect the course of the true disease and can lead to the emergence of a "second" disease due to unproven therapy.

The first group of iatrogens caused by a false diagnosis includes "computer-generated" pseudo-diseases. During mass examinations with the help of a number of general computer programs, without taking into account the individual characteristics

of the organism, false diagnoses are often established. When, with the help of a computer program, the researcher gives information about the immune reactivity of the body based on the indicators of the cardiovascular system, an error in the diagnosis is a consequence of an incorrect diagnostic method. In recent years, the number of private diagnostic centers aimed at such "general computer diagnostic methods" has been increasing catastrophically for the health care industry.

The second group of iatrogenic diseases related to diagnosis includes:

- complications from angiography;
- complications from endoscopic diagnosis;
- complications from hepatic cholangiography;
- complications of puncture biopsies.

During the diagnostic procedure, iatrogenic pathologies are most often detected immediately and are non-infectious in nature. Mostly it is bleeding or anaphylactic shock.

For example, during angiography, iatrogenia can be caused by the action of radiopaque substances - urographite, hexobryx. Individual sensitivity of the body to these drugs can lead to anaphylactic shock or hyperosmolar coma.

During endoscopic diagnosis, iatrogenia may occur as a result of bleeding.

**Iatrogenies caused by patient treatment**

Iatrogenies associated with the use of medicinal products

Iatrogenic pathologies in a large number of cases arise as a result of the wrong choice of drugs, their dose, complications that arise with increased individual sensitivity to a pharmacological drug. In the event of an erroneous diagnosis, the doctor may prescribe drugs that are not only unnecessary for the body, but may also cause iatrogeny.

In recent years, there has been a trend towards the mass use of pharmacological drugs in Ukraine, which was stimulated by advertising in the press, on radio and television. This leads to the mass use of drugs for the purpose of self-medication, underestimation of the consequences of the toxic effects of pharmacological drugs. Physical and chemical pollution causes disturbances in the metabolic processes of the body, tension in the work of the system that ensures the homeostasis of the body.

Irresponsible prescription of drugs: antibiotics. sulfonamides, psychotropic drugs, hormonal and immunostimulating drugs led to the fact that drug-induced iatrogenicity began to be called "drug-induced disease".

Medical iatrogenies arise as a result of:

- erroneous prescription of the drug by the doctor, without taking into account the patient's age and condition;
- overdose of the pharmacological action of the drug;
- failure to take into account the side effect of a medicinal product or the cross-effect of drugs during complex chemotherapy, the introduction of drugs without taking into account their cumulative effect.

Mechanisms of drug-induced iatrogens are different. Conventionally, they are divided into the following groups:

- Mutagenic, carcinogenic and teratogenic effect of medicines.

- Pathological (allergic) immune reactions caused by drugs.
- Idiosyncrasy.
- Toxic effect of a pharmacological drug.
- Combined mechanism of side effect of medicines.

**1. Mutagenic, carcinogenic, and teratogenic effects of drugs** cause iatrogenic pathology, which manifests itself several months or even years after the therapeutic course. Thalidomide teratogenesis and coumarin embryopathy are the most well-known in the world.

**2. Pathological (allergic) immune reactions** caused by drugs. The vast majority of medicinal products are complex high-molecular compounds that have antigenic properties. Other drugs that do not have a large molecule can act as haptens, forming antigenic complexes with proteins, poly-nucleides, polysaccharides.

An allergic reaction to medicinal products can occur in the form of an acute humoral reaction with the development of anaphylactic shock. Allergic reaction to medicinal preparations has in its pathogenesis a preliminary sensitization of the body. However, sensitization can be cross, passive and hidden, and then an allergic reaction to the drug can occur "unexpectedly". For example, anaphylaxis to the first administration of penicillin may be the result of drinking milk stabilized by the addition of antibiotics. The second way of development of pathological immune conditions under the influence of drugs is the formation of immune complexes that lead to the emergence of serum sickness. The third way to the development of immune pathology caused by drugs is a delayed allergic reaction. For example, the Koch reaction. The fourth of the possible mechanisms of drug iatrogenesis consists in the development of an autoimmune reaction to substances that denature the body's own cells, which are formed under the influence of drugs.

Алергійна реакція на лікарські засоби може протікати по типу термінової гуморальної реакції з розвитком анафілактичного шоку.

**3. Idiosyncrasy** - a pathological reaction of the body to the action of medicinal preparations, which has a non-specific mechanism. Idiosyncrasy and drug allergy refer to different types of iatrogenic pathology. Drug allergy has an immune mechanism. Idiosyncrasy is a hereditary deficiency of some enzymes involved in drug metabolism. An example of such an idiosyncrasy is primaquine hemolytic anemia, which occurs as an iatrogenic disease when taking antipyretic drugs and painkillers. Such anemia is associated with a deficiency of the enzyme glucose-6-phosphadehydrogenase in erythrocytes. In such persons, the use of these drugs can lead to hemolysis of erythrocytes. There is a large list of enzymopathies that can cause the development of an iatrogenic disease in response to the administration of drugs.

**4. Toxic effect of medicines**. Iatrogenies caused by the toxic effect of drugs are caused by two factors:

- exceeding the dose of the medicinal product;
- accumulation of the drug-toxin in the body.

The degree of toxic effect of the drug on the body is different: from damage to microsomal structures to complete cell necrosis.

**5. Combined effect of medicines.** The cause of an iatrogenic disease can be a combination of two idiosyncrasy reactions and an allergic reaction to taking a medication. For example, with gluten enteropathy due to a deficiency of the intestinal peptidase enzyme, gluten does not fully break down and "pieces" of this complex protein compound cause the development of an allergic reaction.

**Iatrogenies associated with surgical intervention**

Iatrogenic diseases that arose as a result of surgical intervention occur much less often than other pathological conditions caused by medical factors. However, this diagnosis of iatrogenic pathology is the easiest to establish, because iatrogenic factors are more obvious during surgical intervention. It is easier to find out which errors of the surgeon affected the course of the disease and led to the development of iatrogenic pathology.

Among surgical iatrogenes, the following occur most often:

- unreasonably wide operations;
- accidental damage to organs and blood vessels;
- long-term catheterization of large venous vessels, etc.

If the surgical intervention led to the development of adhesion disease, dumping syndrome, violation of carbohydrate metabolism, then such pathological processes are also considered as iatrogenic pathology.

In the surgical hospital, it is possible to diagnose iatrogenic pathology, which is not directly related to the activities of the surgeon and is an accompanying surgical iatrogeny. Medical factors that lead to severe complications during surgery include anesthesia and analgesia, resuscitation measures. Long-term parenteral nutrition and hemodialysis can lead to the development of iatrogenic pathology.

Thus, iatrogenic pathologies caused by surgical intervention have two main causes. The first is due to the great risk and complexity of the surgical intervention, which are complicated by anesthesia and anesthesia. When an iatrogenic pathology occurs in such conditions, neither the doctor nor the medical institution should bear legal and financial responsibility. Of course, there can be no legal liability only in those cases where informed consent was obtained from the patient before the operation.

If the operation is not widely implemented in medical practice and can be considered as scientific research, then informed consent from the patient must be obtained in writing. In such a document, it is necessary to indicate the possible development of iatrogenic pathology as a result of the proposed surgical intervention.

The second reason for the development of iatrogenic pathology due to surgical intervention is due to errors in the technique of surgery or anesthesia, wrongly chosen method or tactics. This type of iatrogenic pathology is most often associated with insufficient qualifications of the doctor. In this case, the court may order the payment of compensation to the patient or his relatives.

**Reanimation iatrogenies**

Reanimation iatrogenies - a type of iatrogenic pathology, the development of which was caused by resuscitation or intensive therapy of terminal conditions.

Reanimation iatrogenies can be conditionally divided into the following groups:

**1st group** - iatrogenies caused by errors of medical staff during surgical manipulations:

- heart puncture;
- puncture or catheterization of veins;
- tracheotomy;
- other manipulations.

**2nd group** - iatrogens caused by hypoxic damage to organs and tissues that develop after successful resuscitation.

When determining thanatogenesis, significant difficulties arise when separating the main pathological process from iatrogeny, which was a consequence of resuscitation measures. However, in a significant number of cases, the cause of death is not resuscitation iatrogeny, but ineffective resuscitation.

When determining thanatogenesis, significant difficulties arise when separating the main pathological process from iatrogeny, which was a consequence of resuscitation measures. However, in a significant number of cases, the cause of death is not resuscitation iatrogeny, but ineffective resuscitation.

Therefore, from an ethical point of view, when it comes to diseases that develop as a result of resuscitation measures, it is more correct to speak not about iatrogenic pathology, which is caused by medical factors, but about resuscitation complications.

Resuscitation complications include:

**traumatic complications:**

***related to manipulations on the heart***:

- damage during heart massage;
- damage during heart puncture;
- damage during catheterization of large veins;

***related to manipulations on the respiratory organs***:

- complication of tracheal intubation;
- complications during tracheotomy;
- complications during artificial lung ventilation;

**non-traumatic complications:**

***complications from transfusion therapy:***

- acute dilatation of the heart;
- air embolism;
- thrombosis and embolism;
- post-transfusion shock;
- citrate intoxication;
- pyrogenic reactions;
- syndrome of massive transfusions;
- others;

***complications during detoxification of the body:***

- gastric lavage;
- intestinal dialysis;
- hemodialysis;
- peritoneal dialysis;
- complications during hyperbaric oxygenation;

- development of osmotic nephrosis when using methods of forced diuresis;
- septic complications during venous catheterization;
- diseases of body revitalization:
  - postanotoxic encephalopathy;
  - cardiopulmonary syndrome;
  - liver-kidney syndrome;
  - postanotoxic endocrinopathy.

**Iatrogenies associated with radiation therapy**

Radiation iatrogenic pathology - pathological processes in the body, organs or tissues that are caused by medical interventions of a radiation nature. Radiation methods are used in medicine for both diagnostic and therapeutic purposes. They distinguish:

- photon (quantum);
- corpuscular radiation therapy.

Quantum radiation therapy is carried out with X-rays (low-voltage, ortho-voltage and megavoltage) and gamma radiation.

Corpuscular radiation therapy - alpha, beta rays, electrons, neutrons, pi-mesons, protons.

Iatrogenic radiation pathology can occur at different times after radiation therapy or diagnosis. The degree of development of iatrogeny depends on the dose of ionizing radiation and the radiosensitivity of the body, organs and tissues to which the action of the rays was directed.

Iatrogenic radiation pathology, which occurs immediately after a radiation therapy session, has the form of radiation burns.

Iatrogenic radiation sickness can appear several months or even years after a course of radiation therapy and be caused mainly by changes in blood vessels, fibers, and connective tissue cells. Damage to small vessels and capillaries cause microcirculation disorders. Conducting a course of radiation therapy rarely leads to pathological changes in large vessels: thrombosis, sclerosis or obliteration. The consequence of pathological changes in blood vessels is circulatory hypoxia, which increases necrotic and dystrophic processes, which lead to the development of secondary sclerosis of tissues. Radiation damage leads to the development of autoimmune processes and disruption of innervation.

The clinic of radiation iatrogenic pathology most often manifests itself in the development of the reaction of the hematopoietic system: leukocytopenia; thrombocytopenia; agranulocytosis. With radiation damage to the skin, radiodermatitis can develop, which can turn into wetting with swelling and small erosions. In the late period, an iatrogenic radiation ulcer may develop.

After a course of radiation therapy for breast cancer, osteonecrosis of the clavicle or ribs can develop, which is often complicated by a fracture. Radiation iatrogenic pathologies are most often manifested as pericarditis, radiation pneumonia, radiation esophagitis, hepatitis, proctitis, nephritis, cystitis and other diseases.

The causes of the development of iatrogenic radiation pathology are conditionally divided into three classes:

- Iatrogenic pathology associated with increased radiosensitivity of the body. In this case, the medical institution bears no legal responsibility for the development of an iatrogenic disease.
- Iatrogenic pathology caused by overdose, errors of medical personnel when working with equipment, violation of safety rules. In such cases, the medical institution bears legal and financial responsibility for the development of an iatrogenic disease. Not only the chief doctor, but also the head of the relevant unit should be responsible for insufficient control of the knowledge of medical workers who work with radiation exposure equipment.
- Iatrogenic pathology occurs as a result of insufficient control over the reaction of the patient's body to the course of radiation therapy. In this case, the medical institution also bears legal and financial responsibility for the development of iatrogenic pathology. However, in this class of iatrogenic liability, along with the chief physician, the head of the department to which the patient was hospitalized, or the head of the clinical laboratory, if the iatrogenic disease arose due to errors in monitoring the condition of the blood picture, are responsible.

**Other iatrogenies caused by medical intervention**

Iatrogenic pathology caused by medical intervention includes manipulative iatrogenies, the development of which is caused by medical procedures, as a result of which bleeding and suppuration occurred, which led to the complication or development of a concomitant disease.

The group of iatrogenies caused by medical intervention includes diseases, the development of which is due to inadequate or erroneous use of various treatment methods or the use of faulty equipment: blood diseases, cystitis, burns, etc. Other types of iatrogenic pathology of this group include cases of granulomatous serositis caused by foreign bodies (talc, starch), myospherulosis caused by the use of ointments and solutions made on a non-sterile basis; complications due to pacemaker implantation, etc.

**Iatrogenic factors associated with deontological errors of medical professionals**

Iatrogenic diseases can occur as a result of deontological errors of the medical staff of treatment and prevention facilities, imprudent, careless statements or actions.

Many examples are known when medical workers caused psychogenic disorders in patients with their careless words or ill-considered behavior. One careless question or observation during history-taking can foreshadow impending disaster. The development of an iatrogenic disease can be caused even by the communication of medical personnel witnessed by the patient. For example, a patient can often hear the phrase: "This is a serious (interesting) case, the patient must be shown to the professor" or "Just look at his cardiogram, there is an extrasystole."

Most people who have experienced the first signs of the disease are characterized by increased emotionality and anxiety. Therefore, they pay considerable attention not only to the doctor's words, but also to his behavior, intonation, and facial expression.

In medical practice, categories of patients who are at risk of developing psychogenic iatrogenic diseases should be identified.

The degree of reaction of patients to deontological errors of medical personnel depends on a number of factors:

- psychological and physiological features of the patient's personality;
- gender;
- age;
- education.

Depending on the type of higher nervous activity, the patient will react differently to the doctor's behavior, his words, silence, communication with colleagues. Of course, in a patient with a melancholic temperament, the doctor's incomprehensible phrase: "Dear, you should pay more attention to your health. You have myocardial dystrophy..." can cause significant psychological stress, the result of which can be neurotic reactions. Among this category of patients, cancerophobia or cardiophobia often occurs, in which even an uncontrolled facial expression of a doctor or an incomprehensible medical term can have a pathogenic meaning.

The risk of developing iatrogenic diseases is not the same in patients of different ages, sexes and education.

In women, psychogenic iatrogenic diseases develop more often than in men.

Age groups at risk of iatrogenic diseases are patients of transitional age:

- teenagers, whose hormonal changes in their body make them emotionally vulnerable to the environment, as well as to changes in their own body;
- patients at the age of menopause, women with pathological menopause are especially vulnerable;
- elderly patients, some of whom are afraid of old age and diseases that are characteristic of elderly people, they have a fear of a fatal prognosis even of a mild illness.

Iatrogenic factors also include the spread of medical information. Of course, the popularization of medical knowledge is of great importance for the prevention of diseases, providing first aid to the victim, etc. However, in this case there is "the other side of the coin". If a person has a phobia for any disease, such knowledge can become an impetus for neurotic disorders.

Iatrogenies caused by deontological errors of medical workers are manifested mainly by neurotic reactions in the form of various variants of autonomic dysfunction. their development is determined by the increased emotionality and vulnerability of the patient.

Depending on the nature of the psychotrauma and the psycho-physiological features of the individual, autonomic disorders can have a generalized nature and are expressed by a predominant dysfunction of the cardiovascular system (heart arrhythmia, changes in blood pressure, etc.), the digestive system (heartburn, vomiting, diarrhea, etc.) and other body systems in combination with senestopathy, negative affective background.

From a legal point of view, it is very difficult to prove the relationship between a neurosis and an iatrogenic disease.

In order to prevent the occurrence of this type of iatrogenic diseases, it is necessary to learn the basic principles of medical ethics and deontology, which are based on a sensitive attitude and a sense of empathy.

**Legal principles of iatrogeny**

According to the current legislation of Ukraine, the definition of iatrogeny is consistent with the legal norms "damage to health", "recognition of the degree of damage to health", "improper provision of medical care", which are defined in the Civil Code, the Criminal Code of Ukraine, the Law of Ukraine "Basics of Legislation" of Ukraine on health protection".

The legal principles of causing harm to health by a doctor are laid down even in the laws of ancient states. In particular, in ancient Babylon, in the Code of King Hammurabi, the high social and financial status of a doctor was combined with great responsibility for his work. For causing damage to health as a result of treatment, it was necessary to pay a large monetary fine. And in paragraph 218 of the codex of King Hammurabi, it is stated: "If a doctor performs a serious operation on someone with a bronze knife and causes death to the patient, or if he removes a cataract from someone's eye and damages the eye, then he is punished by cutting off his hand."

The Bamberg Codex (1507) and the Criminal Code (contract), known as "Carolina" (1532), defined legal proceedings for the mistakes of doctors.

Foreign experience suggests that one of the most important means of legal control over the provision of medical services is the responsibility of the doctor and/or medical institution to the patient for the occurrence of diseases and pathological processes as a result of medical intervention.

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**Tests**

1. What classification of iatrogens does not exist?
   A. by the etiology of the disease;
   B. according to the International Classification of Diseases;
   C. according to Kalytiaevsky;
   D. by links of provision of honey. help
   E. according to Rykov.

Option of the correct answer: D, today there is no single generally accepted option for the classification of iatrogens, therefore, in the field of health care, several options for the classification of iatrogens are used:

- according to the etiology of the disease;
- according to the International Classification of Diseases;
- according to Kalytiaevsky;

– according to Rykov.

2. How many groups does Rykov's classification include:
   A. 1
   B. 2
   C. 3
   D. 4
   E. 5

Option of the correct answer: E. In this classification, economic sanctions are stipulated in the event of iatrogenic pathology: 1st group. Iatrogenies that occur at the previous stages of treatment and are therefore not related to the medical and preventive institution that established the diagnosis of an iatrogenic disease, 2nd group. Iatrogenies caused by an abnormal reaction of the body to pharmacological drugs and other methods of treatment. Iatrogenies of this group do not belong to "doctor's mistakes", therefore no economic sanctions are foreseen for the medical institution, the 3rd group. Iatrogenies caused by diagnostic and treatment errors. The medical institution must fully bear economic or other responsibility. In Ukraine, the mechanism of economic sanctions has not been developed, the 4th group. Iatrogenies that do not play a certain role in the pathogenesis of the disease, that is, an iatrogenic disease is superimposed on a background or concomitant disease. The majority of specialists adhere to the point of view of the lack of economic responsibility of a medical institution when a patient is diagnosed with iatrogeny of the 4th group, 5th group. Iatrogenies arising as a result of self-medication. There are no economic sanctions against the medical institution.

3. Which of the following factors are the most typical causes of surgical iatrogenes?
   A. Incorrect method of diagnosing pathologies.
   B. Not a full set of medical personnel during the operation.
   C. Unreasonably broad operations.
   D. Intentional crossing of organs and blood vessels.
   E. Poor security of the operating room

Correct answer option: C, iatrogenic diseases caused by surgical intervention often occur as a result of unnecessarily extensive operations, accidental damage to organs and vessels, as well as long-term catheterization of large venous vessels.

4. What actions most often lead to resuscitation iatrogenies?
   A. Caused by errors of medical personnel during surgical manipulations.
   B. Caused by hypoxic damage to organs after successful resuscitation.
   C. Which arise as a result of a malfunction of the equipment in the operating room.
   D. Caused by incorrect selection of anesthesia doses.

Correct answer option: D, resuscitation iatrogenies can be divided into groups, among which one of the groups is those caused by hypoxic damage to organs and tissues after successful resuscitation.

5. Iatrogenies are
   A. Pathological conditions (diseases, complications, injuries) caused by medical interventions, i.e. negative effects of medical influences, which are expressed

in the emergence of new diseases in the patient or in complications and worsening of an already existing disease.

B. Deliberate harm to the patient due to the doctor's dislike for him.

C. Long-term complications after extensive surgical interventions that did not appear due to the fault of the doctor and surgical tactics.

D. Complete recovery of the patient after treatment of the pathology.

E. The presence in an individual of several diseases that have a synchronous course in different phases and stages of their development, both related and not related to each other by pathogenesis and genetically.

Option of the correct answer: A, according to the definition of the concept of iatrogeny, these are pathological conditions caused by medical interventions, that is, negative effects of medical influences, which are expressed in the emergence of new diseases in the patient or in complications and worsening of an already existing disease.

6. What types of radiation therapy are used in medicine:

A. Photon (quantum)/corpuscular radiation therapy.

B. Radical/palliative.

C. Primary/ secondary.

D. Aimed/ general.

E. Wave/ straight.

Option of the correct answer: A, Radiation methods are used in medicine for both diagnostic and therapeutic purposes. There are: photon (quantum), corpuscular radiation therapy. Quantum radiation therapy is carried out with X-rays (low-voltage, ortho-voltage and megavoltage) and gamma radiation. Corpuscular radiation therapy - alpha, beta rays, electrons, neutrons, protons.

7. The clinic of radiation iatrogenic pathology is most often manifested in:

A. Development of intestinal phenomena.

B. Development of the reaction of the hematopoietic system

C. Development of cardiovascular lesions.

D. Development of malabsorption syndrome.

E. Without manifestations (hidden course).

Variant of the correct answer: B, the clinic of radiation iatrogenic pathology most often manifests itself in the development of the reaction of the hematopoietic system: leukocytopenia; thrombocytopenia; agranulocytosis.

8. Complications may develop after a course of radiation therapy for breast cancer:

A. Necrosis of superficial tissues.

B. Bone marrow lesions.

C. Osteonecrosis of the clavicle or ribs.

D. Sepsis.

E. Menopause.

Option of the correct answer: C, one of the most common complications of improperly selected radiation therapy is osteonecrosis of the clavicle or ribs, with the possibility of their fracture.

9. Illness caused by the actions of medical workers:

A. Carcerophobia.

B. Retrogeny.

C. Iatrogeny
D. Hepernosognoria.
E. All of the above.

Variant of the correct answer: C, cancerophobia is not a disease, but a phobia of cancer, that is, a pathological fear of cancer, retrogeny - the lower jaw is placed behind the upper jaw, iatrogeny is a disease caused by the actions of medical workers, hypernosognosia is a subjective overestimation of the severity of the disease, its possible complications and the probability of failure.

10. For the first time, the term "iatrogeny" began to be used in medical practice in:
A. 1925
B. 1944
C. 1987
D. 2000
E. 1920

Correct answer option: A, the term "iatrogenia" began to be widely used in medical practice after it was printed in 1925. Bumke's article "The doctor as a cause of mental disorders".

**Materials on the independent teaching of students**

| Main tasks | Notes (instructions) |
|---|---|
| Repeat:<br>– Pathophysiology of basic surgical diseases.<br>– Anatomy of organs of abdominal and thoracal cavity<br>– Morphological changes in the organs at disease<br>– Types of miniinvasive and endoscopic techniques | -To determine the best radiological methods depending on types of pathology<br>-To select more suitable miniinvasive methods depending on types of pathology |

**Навчальне видання**

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# Навчальний посібник з хірургії для студентів-медиків (6 курс).

## 2-ге видання, перегляну́те/ за ред. проф. І.В. Колосовича та доц. А.М. Циганка

**(англійською мовою)**

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