Nicolae Testemiţanu State University of Medicine and Pharmacy
Department of General Surgery and Semiology no.3

Eugen Guţu
Dumitru Casian

General Surgery and Semiology

3rd edition

Textbook for the 3rd-year students,
faculty of Medicine no. 2

Chișinău, 2023

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CZU: 617+616-07(075.8)
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Approved by the Quality Management Board of *Nicolae Testemițanu* State University of Medicine and Pharmacy, protocol no. 1 from 20 October 2022

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Guțu, Eugen.
General surgeryand semiology : Textbook for the 3rd-year students, faculty ofMedicine no. 2 / Eugen Guțu, Dumitru Casian ; Nicolae Testemițanu State University ofMedicine and Pharmacy, Department of General Surgery and Semiology no. 3. – 3rd ed. – Chișinău : S. n., 2023(Universul). – 213 p. : fig., tab. [10] ex.

ISBN 978-9975-47-241-8.

617+616-07(075.8)

Tipar: ÎS Editura UNIVERSUL, mun. Chișinău, str. V.Pîrcălab, 45

ISBN 978-9975-47-241-8.

CONTENTS

PREFACE

An “avalanche” of information is a characteristic phenomenon of XXI century and medicine of course is not an exception. In contrast with previous decades now the problem is not to find a good medical book but rather to select the best one from many and to carve out the time to read it. The topics included in the curriculum of General surgery and surgical semiology for III-rd year students usually are scattered as separate chapters among multiple comprehensive surgical textbooks which run from several hundreds to thousands of pages. Searching for optimal source of information requires a lot of experience, patience and time. To the student first time arrived in clinical environment, not familiar with many peculiarities of surgical patients and surgical care and always under “time pressure” this task can seem overwhelming.

This book is designed to assist our students endeavoring to learn the fundamental concepts and common aspects of surgical practice: aseptic technique, diagnosis and treatment of bleeding, perioperative management, principles of trauma care, infection control, art of patient examination and many others. Structure and content of the topics were adapted to recently modified curriculum of our discipline. Basing on successful format of previously published “course support” we have chosen not to extend the text too much, maintaining the focus upon most important issues. However, as surgical since progresses rapidly, we decided to complement current edition with new definitions, classifications and diagnostic methods widely implemented during the last years. Some particular topics that (from our teaching experience) represented more difficulties for students to learn now are explained comprehensively. The unique feature of this new manual is a

lot of high-quality illustrations aimed to support the understanding and memorizing of the material. We are confident that the first edition of “General surgery and surgical semiology” textbook will become a popular and valuable reference source for those beginning their mastering of clinical medicine.

Authors

I. SHORT HISTORY OF SURGERY

The word “surgery” consists of two Greek words: “hand” and “work” and, accordingly means “handicraft”, “manual operation”, or “craft”. However, the historical meaning of this word has become outdated. Nowadays, surgery is a high-professional medical specialty. The surgeon must not only possess the technique of surgical procedures, but also thoroughly know anatomy, physiology, pharmacology and other disciplines must be able to make the correct diagnosis, to determine the tactics of treatment.

The history of surgery is very interesting and long, but we will focus only on the main periods of its development and on contribution of the most prominent persons. Different surgical manipulations were performed in Ancient Egypt, India, Babylon, Ancient Greece and Rome, Byzantium and China 2-4 thousand years B.C.

Edwin Smith’s papyrus (Ancient Egyptian papyrus dated ca. 1,600 B.C.) – probably the first surgical manuscript with description in detail of 48 cases of wounds and other traumatic injuries and their treatment.

Code of Hammurabi from Babylon (1,790 B.C.) – is the first code of laws, which regulates not only the ordinary daily aspects of country life, but also the surgeon’s professional activity as well as responsibility.

In Ancient India the most famous surgeon was **Sushruta**. He lived on the bank of the river Ganges 600 B.C. In the manuscript *“Sushruta Samhita”* he described more than 120 surgical instruments, 300 procedures, for the first time dividing surgery into 8 separate areas.

Hippocrates (Ancient Greece, 460-377 B.C.), who is considered to be the father of medical art. Although from a

modern point of view he was a general physician, in his writings that have come down to our time there is a book called “During surgery”, which have had following parts: surgeon, assistant, instruments, light, method, time and place.

Cornelius Celsus (Ancient Rome, 30 B.C.-37 A.D.) – the author of the first surgical treatise and described 5 classical signs of inflammation, that have remained unchanged to our days.

Claudius Galen (Ancient Rome, 129-210 A.D.) – he was the first, who proposed ligation of the bleeding vessel, urged that surgery be considered as a separate medical specialty. His most important achievements belong to reconstructive surgery of soft tissues and human anatomy. He summarized results of his anatomical research in the treatise *“Omnia Opera”*, which became the only source of true anatomical knowledge for European surgeons over the next millennium.

It is necessary to emphasize the contribution of the Arab medical school to development of surgery:

Abu Ali ibn Sina or **Avicenna** (medieval Persia, 980-1037), the author of “Canon of Medicine”, which contains chapters on the treatment of wounds and local inflammatory processes.

Abul Qasim Khalaf ibn al-Abbas al-Zahrawi or **Albucasis** (Caliphate of Andalusia on the place of modern Spain, 993-1064). He invented artery ligation during surgery, proposed operating theater, as a separate room, intended only for performing surgical interventions. He also for the first time suggested the hereditary nature of hemophilia, described an ectopic pregnancy, and hip dislocation.

Doctors, who made an important contribution to the development of surgery in the Middle Ages, are:

Andreas Vesalius (Padua, Italy, 1515-1564), who, for the first time in post-antique history, questioned the correctness of Galen's anatomy, studied and properly described human anatomy in his book *"De corporis humani fabrica",* and is therefore considered the first modern surgeon-anatomist.

Paracelsus (Switzerland, 1493-1541), being a military surgeon he improved the methods of wound treatment. In addition, he insisted on the need to study in the universities that appeared in medieval Europe not only general medicine, but also surgery.

Ambroise Pare (France, 1517-1590), a military surgeon, invented hemostatic forceps, developed the technique of limb amputations, and described the treatment of gunshot wounds. The latter he realized in the form of a book written in French language and not in Latin. Thus, for the first time, a surgical manuscript was published in a modern language accessible to all doctors.

Dominique Jean Larrey (1766-1842) – considered the founder of modern military surgery. He developed a lot of new surgical procedures applied in case of traumatic injuries, described the clinical features of tetanus and pathophysiology of frostbite and hypothermia, and introduced the system of rapid transportation of injured soldiers into the hospital in order to provide them an emergency medical care.

It is impossible not to mention **Nicolai Pirogov** (1810-1881), who is considered the founder of surgery in Russia. Having thoroughly studied topographic anatomy using a special method of frozen cadavers, he brought the technique of many surgical procedures to perfection, and also developed a system for providing surgical care in war conditions. In particular, he developed the doctrine of wounded's triage, and

also proved the great importance of nursing care in the daily treatment of patients.

The beginning of modern era in the development of surgery became possible after the discovery and justification of the aseptic principles in surgical practice by **Joseph Lister** (Scotland, 1827-1912), and the introduction of inhalation ether anesthesia by **William Morton** (USA, 1819-1868). It made possible to perform large abdominal and thoracic surgeries; whereas the number of postoperative infectious complications has sharply decreased.

Since the end of 19th century, a number of surgeons around the world began to develop new methods of surgical treatment of various diseases, performed operations on all organs of the human body, and created modern surgical schools. Among them should be mentioned: **Theodor Billroth** (Austria-Hungary, 1829-1894), **Theodor Kocher** (Switzerland, 1841-1917), **William Halsted** (USA, 1852-1922), **William Mayo** (USA, 1861-1939), **Sergei Yudin** (USSR, 1891-1954).

The founder of modern surgery in Moldova is Professor **Nicolae Anestiadi** (1916-1968). He was a pioneer of modern thoracic surgery, cardiovascular surgery, abdominal surgery and anesthesiology in our country. The Association of Surgeons from Republic of Moldova bears his name, as well as one of the departments of surgery at the State University of Medicine and Pharmacy.

II. ASEPTIC TECHNIQUE IN SURGERY

SURGICAL SITE INFECTION

Preventing infection of the surgical site (wound) during surgical intervention is one of the main tasks facing the surgical team and the staff of the operating unit, and a number of activities and measures are aimed at solving this problem. Surgical site infection refers to **nosocomial infection** (Greek word *nosokomeion* – hospital). Nosocomial infection – any infection acquired during or as a result of hospitalization and treatment.

The most common **types of nosocomial infections** are as follows:

(1) Urinary tract infection;

(2) Respiratory tract infection (pneumonia);

(3) Surgical site infection (operating wound infections), accounting for about 15% of all types of nosocomial infections.

According to modern concepts, a **surgical site infection** is defined as an infection which occurs within **30 days** after the operation if no implant is left in the site or within **1 year** if an implant remains in place.

The **classification of surgical site infection** is based on anatomical principles and includes three levels of infection (Figure 1):

(1) Superficial incisional infections (with affectation of skin and subcutaneous tissue). These infections may be indicated by localized (Celsian) signs such as redness, pain, heat or swelling at the site of the incision or by the drainage of pus;

(2) Deep incisional infections (involve fascia and muscles). These infections may be indicated by the presence of pus or an abscess, fever with tenderness of the wound, or a

separation of the edges of the incision exposing the deeper tissues;

(3) Organ/space infections (localized in the organs or anatomical cavities). It involves any part of the anatomy other than the incision that is opened or manipulated during the surgical procedure, for example joint or peritoneum. These infections may be indicated by the drainage of pus or the formation of an abscess detected by histopathological or radiological examination or during re-operation.

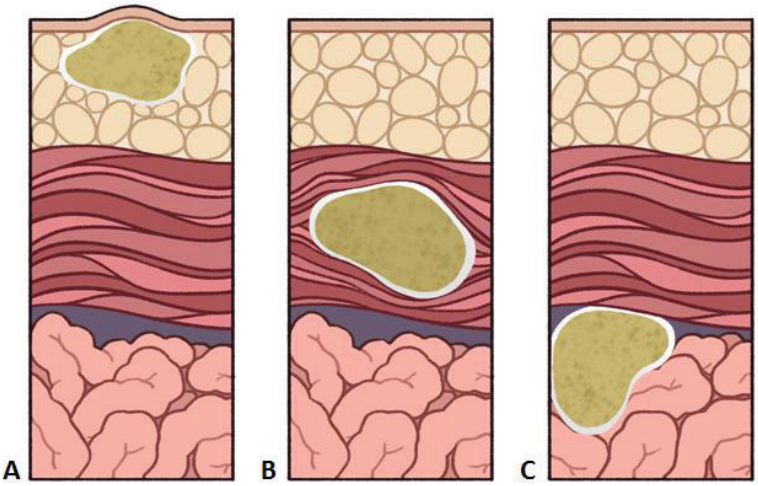

Figure 1. Types of surgical site infection: A – Superficial incisional infection; B – Deep incisional infection; C – Infection of organs and cavities.

The distribution of pathogens, isolated from infected surgical wound has not changed significantly during the years. *Staphylococcus aureus*, *Coagulase-negative Staphylococci, Enterococcus,* and *Escherichia coli* remain the most frequently isolated pathogens. An increasing proportion of surgical-site infections actually are caused by antimicrobial-resistant

pathogens, such as methicillin-resistant *Staphylococcus aureus* (termed MRSA infection) or by *Candida albicans.*

With aim to prevent the surgical site infection all surgeons around the world use aseptic technique. The main objective of **surgical asepsis** is to prevent the contamination of the open surgical wound by isolating the operative site from the surrounding non-sterile environment (**exogenous** infection) and by treatment of chronic infections in the patient's body (**endogenous** infection).

There are three ways of **exogenous contamination**:

(1) Airborne spread;

(2) Contact spread;

(3) Contamination by implantation of infected materials (implantation infection).

PREVENTION OF AIRBORNE INFECTION

In case of airborne way of contamination, pathogenic germs get into the wound from infected air. The air in operating room may be contaminated by infected droplets from respiratory tract of the patients and health-workers, droplets of wound discharge, infected clothes of patients and surgeons and infected dust, as well as pathogenic microorganisms from clothing and skin. The most important measures for prevention of airborne infection include the following principles:

(1) Separation of patients with septic processes from ones without them, what is achieved by the organization in any medical institution of at least two surgical departments – aseptic and purulent, as well as the allocation of an individual operating room for each of these departments;

(2) Restricted access in operating rooms for unnecessary staff and various kinds of visitors. In experiment was

determined that one person can eliminate in the air by respiration and speaking up to 10 pathogenic germs during one second. According to current standards, adopted by majority of European countries, the accepted level of bacterial contamination of the air in operating room should not exceed 200 pathogenic germs in one cubic meter of air;

(3) Wearing surgical attire. Although the specified set of clothes should not be sterile, however, masks, caps and shoe covers are used once, and the surgical suit (pants and a shirt) is washed every time after the end of the operating day;

(4) Cleaning of operating room. There are several types of cleaning: preliminary (every morning before starting work); current (during the operation, if necessary); postoperative (upon completion of surgery); final (daily at the end of the working day); and planned or general (usually at the end of the working week);

(5) Use of modern ventilation systems equipped with bacterial filters, with downward air flow from the ceiling to the floor in the operating room.

PREVENTION OF CONTACT INFECTION

The main sense of prevention of contact infection is - **"One and all that get into the contact with wound obligate to be sterile".**

The **Spaulding classification** categorizes medical devices based on the risk of infection involved with use:

(1) "Critical" items enter into tissues, vascular lumen or sterile cavities and spaces. These items include needles, vascular catheters, surgical instruments, sutures, gloves, gowns, drapes, bandages, and others.

(2) "Semicritical" items come in contact with mucous membranes and do not ordinarily penetrate sterile tissue. This

group includes endotracheal tubes for ventilation, flexible endoscopes, gastric tubes, vaginal speculum, and others.

(3) "Noncritical" items usually do not touch the patient or only come into contact with intact skin, for example, stethoscopes, thermometers, operating table, ground plate of electrosurgical equipment, and others.

There are **several levels** of antibacterial decontamination of surgical objects. **Sterilization** – destruction of all pathogenic microorganisms, including bacterial spores. Sterilization is achieved by various methods: using heat, ionizing radiation or chemicals. Sterilization is required for all "critical" items. **Disinfection** eradicates most (but not all) microorganisms and is commonly performed by using liquid chemical bactericidal antiseptics. **Cleaning** defined as the physical removal of organic material and/or soil, generally by using water with detergents.

There are 3 levels of disinfection depending on the degree of microbial elimination involved: **High-level disinfection** destroys microorganisms, mycobacteria, fungi, viruses, with the exception of some tolerant bacterial spores. For this, highly effective disinfectants are used at the correct dilution, temperature and contact time. High-level disinfection is required for "semi-critical" objects. **Intermediate-level disinfection** uses common medical disinfectants and is indicated for any item that touches mucous membrane or skin that is not intact. This kills all mycobacteria, vegetative bacteria, fungal spores, and some viruses, but not bacterial spores. **Low-level disinfection** (essentially "wipe-down") is adequate for "non-critical" accessories, which come into contact with intact skin. This level of disinfection destroys most bacteria, some fungi and some viruses, but does not inactivate resistant microorganisms.

All persons working in the operating room can be attributed to **sterile members** of surgical team (or so called "scrubbed" persons) and **nonsterile** (or "un-scrubbed") personnel. To "scrubbed" team refers surgeon, surgeon's assistants and scrub nurse. To nonsterile refers anesthesiologist, nurses and visitors (students, medical residents, consultants).

The **main measures** for preventing contact infection are as follows:

(1) Surgeon's hands scrub (washing with soap in running water, drying with a sterile towel and decontamination with an antiseptic solution for 2-5 minutes). As a rule, ethyl alcohol, chlorhexidine solution, iodine/iodophors antiseptic preparations, triclosan are used as an antiseptic.

(2) Mandatory use of sterile gowns and sterile gloves by surgeons and operating nurses during the surgical intervention. Actually, all surgeons use only disposable gloves sterilized at the factory by ionizing radiation. This should be kept in mind, that regardless to obligatory wearing of sterile gloves the preparation of surgeon's hands is of paramount importance because gloves are frequently damaged during the operation.

(3) Sterilization of textile surgical linens (gowns, drapes and bandages). Sterilization is carried out in an autoclave with steam at a temperature of 120°C under a pressure of 1.5 atmospheres for 45 minutes. The linens are loaded into Sheemelbush's container, which remains sterile during 72 hours after sterilization unless opened or damaged.

(4) Sterilization of surgical instruments includes three consecutive steps:

- Decontamination – instruments are immersed in a 6% hydrogen peroxide solution, 0.5% chlorine solution or other universal antiseptic (Anasept, Peroster).

- Cleaning – washing in detergent with brush and rinsing in warm running water.

- Sterilization – dry-heat sterilization in special electric oven under the following mode: duration – 1 hour, temperature – 180°C.

Delicate surgical items (flexible endoscopes, objects or instruments from plastic) can be sterilized by cold methods – chemical sterilization using strong antiseptic solutions like glutaraldehyde (Cidex), or ethylene oxide gas.

Quality control of sterilization of surgical material and instruments is an obligatory part of asepsis and can be carried out by two methods:

- Direct method – the obtained material is tested in the bacteriological laboratory. The main disadvantage of the method is its retrospective nature;

- Indirect methods – using of heat-sensitive chemical indicators. If the color of the indicator has changed, this means that the required temperature has been reached and the material or instruments are sterile.

(5) Proper preparation of patients' skin in the operating field area before surgery may reduce the risk of surgical site infection. Principal rules of decontamination of **operating field** (patient's skin) should be followed:

- A hygienic shower before the operation is desirable;

- Shaving should be performed directly (not earlier than 2 hours) before surgery;

- Skin must be treated with antiseptic more widely, and not just the area of the future skin incision;

- Cleaning must be performed from the center to periphery;
- More contaminated areas of the patient's body (groin, navel, purulent wound, intestinal stoma) are cleaned last;
- Cleaning must be repeated three times for one minute each until limitation of the operative field with drapes, prior to incision, before and after wound closure.

PREVENTION OF CONTAMINATION BY IMPLANTATION

Implantable materials and device – are medical devices that are placed into surgically or naturally formed cavity of the human body for a **period of 30 or more days.** Surgical sutures, vascular grafts, artificial heart valves, joint prostheses and osteosynthesis devices, synthetic meshes for hernia repair and other implantable surgical objects.

The implantation of infected materials into the human body inevitably leads to the development of a purulent-septic process. Implant infection is usually resistant to antibiotics due to the formation of so-called microbial biofilms (a limited accumulation of bacteria) and almost always requires removal of the infected device.

All implantable devices are sterilized by ionizing radiation at their place of manufacture. Re-sterilization is usually not allowed.

ENDOGENOUS INFECTION

With an endogenous infection, microorganisms enter the surgical wound from various foci of infection (most often chronic) inside the patient's body: chronic genitourinary and skin infections, chronic bronchitis, tonsillitis, caries, and others. The endogenous source of infection can play an important role in patients with compromised immunity and disturbed host-defense mechanisms.

There are two ways of endogenous infection spreading: hematogenous and lymphogenous.

To prevent endogenous infection of operating wounds, it is strongly recommended to treat chronic infectious processes prior to elective planned surgical interventions.

ANTIBACTERIAL PROPHYLAXIS

One from the most effective methods, equally effective against endogenous and exogenous infection is antibacterial prophylaxis, capable to reduce the rate of surgical site infection by 30%.

Indications for antibiotic prophylaxis are determined by the classification of surgical operations according to the degree of microbial contamination. Aseptic operations that are not accompanied by the introduction of an implant do not require prophylactic administration of antibiotics. In the presence of an implant, as well as in the case of conditionally infected and infected operations, antibiotic prophylaxis is mandatory. In purulent operations, prophylaxis is combined with prolonged postoperative antibiotic therapy.

Selection of antibacterial drug for prophylaxis depends on the type of pathogenic flora responsible for majority of surgical site infections. First generation cephalosporin's and ampicillin-sulbactam refer to the first line drugs for antibacterial prophylaxis. In colorectal surgery, these drugs should be combined with metronidazole.

The optimal time for **administration** of preoperative doses is within 60 minutes before surgical incision by intravenous route. Usually a single maximum dose of antibiotic is prescribed. As a rule, antibiotics are not administered after wound closure, and even if the doctor decides that repeated doses are necessary, the total duration of prophylaxis should not exceed 24 hours.

III. ANTISEPSIS

DEFINITION AND HISTORY

Antisepsis, being one of the important parts of general surgery, is a complex of measures used for destruction of microorganisms in the wound, pathological focus and in the organism as a whole. From the point of view of practical medicine, antisepsis is closely related to asepsis, and are often used simultaneously.

History of antisepsis and asepsis consists of **4 classic periods**:

(1) Empiric period;

(2) Asepsis and antisepsis of the 19th century, before the appearance of scientific antisepsis;

(3) Period of Lister's asepsis and antisepsis;

(4) Modern asepsis and antisepsis.

Empiric period. Initial antiseptic methods are discovered in the scientific papers of doctors of the ancient world. There are several examples: Ancient surgeons considered that a foreign body should be obligatory removed from the wound; It was forbidden to touch the wound in Hebraic legislation of Moses; Hippocrates advocated the principle of cleanness of surgeon's hands, used clean water and wine for management of wound, and affirmed, that a dressing material should be clean.

Asepsis and antisepsis of the 19th century. Medicine didn't know the causes of suppuration yet and the results of treatment were terrible. Postoperative mortality was more than 80% and was generally conditioned by purulent complications. In 1847 **Ignaz Semmelweis**, a Hungarian obstetrician-gynecologist, basing on his own experience, affirmed, that the cause of postnatal sepsis was introduction of contagious material from doctor's hands during

examination. Use of 10% chloride of lime reduced greatly above mentioned complications.

Joseph Lister, a British surgeon, applying the discovery of Louis Pasteur and analyzing the causes of postoperative mortality, made a conclusion that bacteria were the cause of complications. At the end of 19th century, he developed some methods of destruction of microorganisms in the air, on surgeon's hands, in the wound and on the objects, which get in touch with the wound. Carbolic acid was used as a disinfectant solution. Thus, the merit of Lister was that he developed an integral system of measures to control infection, that's why he is considered to be the founder of antisepsis. As a result of the widespread introduction of the Lister method, mortality after surgical interventions in Europe has decreased by 10 times.

Modern surgical antisepsis is closely connected with asepsis and is an integral part of the common system. Antisepsis is divided into several types according to the used methods: mechanical, physical, chemical, biological and combined antisepsis.

MECHANICAL ANTISEPSIS

Mechanical antisepsis is a mechanical removal of microorganisms from the wound. In practice, foreign bodies, non-viable and necrotized tissues, infected blood clots, purulent exudate, being an ideal medium for bacterial multiplication, are mechanically removed from the wound. For this purpose, a number of methods are used:

(1) Wound toilet is performed practically during any wound dressing. The bandage imbibed with dirty wound exudate is removed; purulent exudation, infected clots, necrotized tissue

are removed from the wound surface using some forceps with a gauze globule.

(2) Primary surgical debridement of wound – an important measure of mechanical antisepsis, which is used for recent wounds. It consists of dissection of wound, its pouches and excision of wound edges and bottom within the limits of healthy tissues. Surgical debridement is performed not so much for "sterilization by knife", but to reduce the volume of non-viable tissues in the wound, which are a favorable medium for development of microflora.

(3) Secondary surgical debridement of wound is performed in cases where a purulent process has already developed in the wound. It includes dissection of pockets and cavities, where pus and necrotic detritus accumulate, and the removal of non-viable tissues.

(4) Other operations and manipulations – drainage of purulent foci. In some cases, puncture with aspiration of the abscess can be effective (as in sinusitis or purulent pleurisy – accumulation of pus in the pleural cavity). With abscesses located deep in the patient's body, a puncture with needle aspiration is performed under the guidance of ultrasound or computed tomography.

PHYSICAL ANTISEPSIS

(1) Hygroscopic dressing material. Introduction of hygroscopic dressing material (gauze and cotton) into the wound increases significantly the volume of evacuated exudate. Usually gauze is used, from which tampons, turundas, meshes and sponges of various sizes and shapes are made.

(2) Hypertonic saline solution, which osmotic pressure is higher than osmotic pressure of blood plasma**.** Hypertonic

solutions are used for improvement of wound flow-out, 10% solution of sodium chloride is used mostly. When gauze meshes are imbibed with hypertonic solution, the outflow of exudate from the wound increases due to the difference in osmotic pressure.

(3) Sorbents. Substances, which absorb toxins, are introduced into the wound. Usually they contain substances of carbon in form of powder.

(4) Drainage is an important method of physical antisepsis used for the treatment of all types of wounds, as well as after surgical interventions on body cavities. There are three main types of wound drainage: passive, active, and flow-irrigative (Figure 2). Latex tubes and bands are usually used for **passive drainage**. On the drainage tube, several side holes are usually made. In **active drainage** the drainage tube is connected to a pump, creating negative pressure. In case of **flow-irrigative drainage** an antiseptic solution is introduced through one tube, and flows out through the other. Thus, a constant washing of the pathological focus is performed.

(5) Additional methods of physical antisepsis.

Drying – treatment in controlled abacterial environment (so-called open or bandage-free wound management). Any installation for treatment in abacterial environment consists of compressor for air insufflation, bacterial filter and an isolator with sterile environment, where the patient or the affected part of his body is placed. In this case, a scab is formed and wound cleaning and healing take place under it.

Wound processing with **pulsatile water jet**. A jet of liquid under high pressure washes away foreign bodies, pus and microorganisms. This kind of wound processing is carried out with a special device.

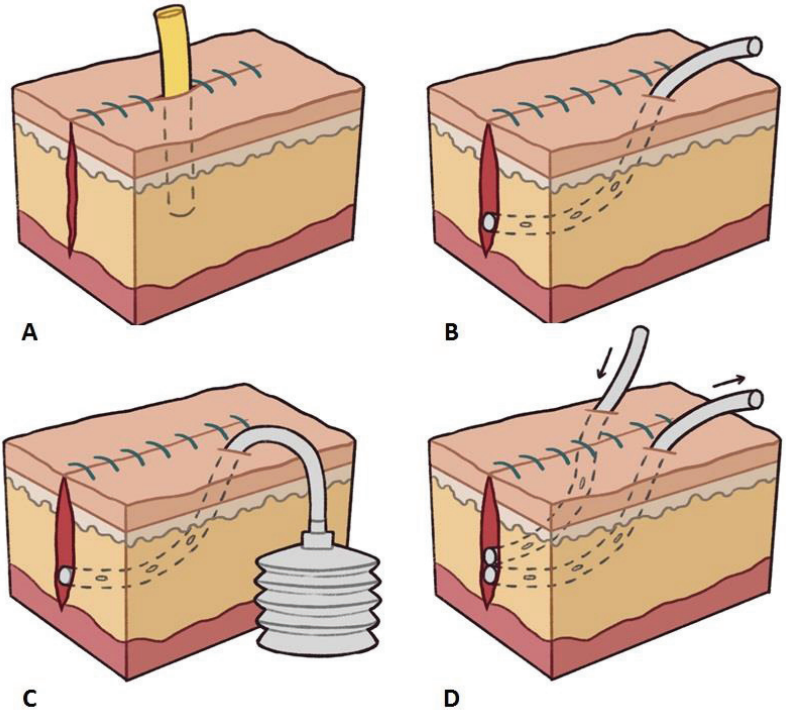

Figure 2. Types of wound drainage: A – Penrose type passive drainage; B – Passive drainage with tube; C – Active drainage with aspiration Redon type; D – Closed flow-irrigative drainage.

Ultrasound causes the effect of cavitation with unfavorable action upon organisms.

High-energy (surgical) laser leads to vaporization (evaporation) of tissue structures. As a result, there is a rapid simultaneous removal of purulent-necrotic tissues and sterilization of the wound surface. After such treatment, the wound becomes sterile, covered with a burn scab.

Ultraviolet rays. The bactericidal action of ultraviolet radiation is used to destroy microbes on the wound surface.

The method is especially indicated for superficial wounds and trophic ulcers of various origins.

Vacuum therapy (VAC – Vacuum Assisted Closure) is a system that promotes the healing of infected open wounds through the application of negative pressure. The method is based on insertion of polyurethane foam dressing cut to fit the wound cavity exactly (Figure 3).

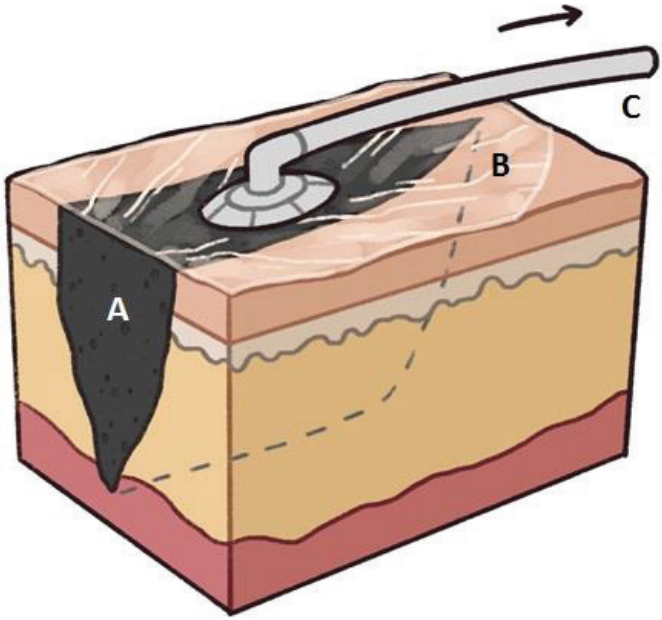

Figure 3. Vacuum therapy or VAC (Vacuum Assisted Closure) – system for the treatment of infected wounds: A – Sterile polyurethane sponge in the wound cavity; B – Adhesive sealed film; C – Tube connected to device that creates a constant or variable negative pressure of 50-125 mm Hg.

After that the wound is sealed with an adhesive dressing and negative pressure (125 mm Hg below atmospheric) generated by suction pump. Negative pressure reliably

removes liquid discharge, reduces swelling and bacterial colonization of the wound, increases local blood flow, promotes the rapid formation of granulation tissue, convergence of the wound edges and accelerates the healing process.

CHEMICAL ANTISEPSIS

Chemical antisepsis is a method of fight with infection in the wound, based on the use of chemical substances, with a bactericide and bacteriostatic action. There are local antiseptics and antiseptics for systemic administration. They are divided into the following groups:

(1) Haloids: 1-2% solution of chloramine is used for the irrigation of purulent wounds and 2% solution – for disinfection of non-biological surfaces, 5-10% alcohol solution of iodine, iodine derivates (1% povidone and 1% solution of iodopirone). Currently, the antiseptic agent povidone iodine (Betadine) is used widely. One milliliter of an aqueous solution contains 10 mg of active iodine and excipients. It is used for disinfection of the skin before injections, infusions, for disinfection of wounds, preoperative decontamination of the skin and mucous membranes.

(2) Salts of metals: 1-0.03% water solution of silver nitrate, nitrate salts: Collargol and Protargol. Effective for cauterization of hypergranulations and treatment of fistulas.

(3) Alcohols: 70% and 96% solutions of ethyl alcohol. It is widely used to decontaminate the edges of open wounds, the surgeon's hands and the operating field.

(4) Aldehydes: formaldehyde, 1-3% Lysoform solution, Cidex (2% solution of glutaraldehyde). Used for sterilization of catheters, flexible endoscopes, tubes and instruments not suitable to heat sterilization.

(5) Phenols: carbolic acid, Ichtyol used in the form of ointment. When applied topically, it has an antiseptic and anti-inflammatory effect.

(6) Dyes: 1-3% alcohol solution of methylene blue, brilliant green, Rivanol. They are weak antiseptics, but are traditionally used to treat superficial wounds, cuts and abrasions.

(7) Acids: powder and 4% solution of boric acid, especially effective in *Pseudomonas aeruginosa* infection of wounds. Formic acid – historically used to clean the surgeon's hands before surgery.

(8) Alkalis: 0.5% ammonia solution is an antiseptic for external use.

(9) Oxidants: solution of hydrogen peroxide. Hydrogen peroxide is discomposed in contact with the wound and releases oxygen in form of the foam. Antiseptic property of hydrogen peroxide is explained either by strong oxidative effect or mechanical cleaning of wound from purulent contents and foreign bodies. Perhydrol contains 30% of hydrogen peroxide and is intended for disinfection of surfaces and instruments. Potassium permanganate 0.1% solution is used for wound irrigation. Oxidants are especially effective in case of anaerobic and putrid infection.

(10) Detergents: Chlorhexidine bigluconate. It is one of the most widely used antiseptics at present, and is used both for treating the surgeon's hands and the skin of surgical field (0.5% alcohol solution), and for washing abdominal cavity for severe inflammatory processes (5% aqueous solution). In addition, detergents are widely used for pre-cleaning of instruments before their final sterilization.

(11) Derivates of nitrofurane: a specific group of drugs with different routes of administration. So, Furacillin is traditionally used topically, for treating wounds and washing cavities;

Furadonin and Furazolidone – uroantiseptics, administered enterally in the form of tablets; Furagine is an antibacterial substance for intravenous administration.

(12) Derivates of 8-oxiquinoline: Nitroxaline is an uroantiseptic, used in case of urinary tract infection. Enteroseptol, Interostopan are chemical substances used for intestinal infection.

(13) Derivates of quinoxaline: Dioxidine is an antiseptic for external use, for washing of purulent cavities.

(14) Derivates of nitromidazole: Metronidazole (Metrogil, Trichopol) is a broad-spectrum chemotherapeutic substance. In surgical practice, it is mainly administered intravenously. It is active against protozoa, bacteroid and anaerobic infections. It is important that with the widespread use of metronidazole, there is no resistance of microorganisms to it.

(15) Sulfanamides: Streptocide, Ftalazole, Sulfadimine, Biseptol are chemotherapeutic substances of bacteriostatic action. These substances in the form of pills, powders and unguents are used for suppression of purulent infection.

(16) Vegetable antiseptics: Chlorphyllipt (chlorophylls), Baliz (produced from saccharomycetes) and calendula (infusion of a medicinal plant) are usually used as antiseptics for external use for superficial wounds of skin or mucosa.

BIOLOGICAL ANTISEPSIS

Biological antiseptics are produced using (1) biological substances directly affecting microorganisms and (2) different substances making better capacity of human body to response upon infection. Antibiotics, protein-degrading enzymes, bacteriophages and medical serums are biological antiseptics also.

(1) Antibiotics are widely used in treatment of purulent infections. The basic principles of **antibiotic therapy** are as follows:

- Antibiotics should be used according to strict indications, avoiding the "symptomatic" prescription of antibiotics.
- It is very important to determine the sensitivity of microorganisms to antibiotics available at the hospital pharmacy.
- Sometimes sensitivity testing to antibiotics should be performed (only applies to penicillins and semi-synthetic penicillins).
- Antibiotics should be changed every 5-7 days in case of prolonged antibiotic therapy to avoid resistance development of microorganisms to antibiotics.
- Combinations of antibiotics of different spectrum of action are used to increase antibacterial activity.
- Combination of administration ways is widely practiced, although the parenteral route is preferred in surgical practice.
- Antibiotics should be used in combination with other antiseptic substances, such as metronidazole.

(2) Protein-degrading enzymes do not destroy microorganisms directly, but quickly clean the wound from necrotized and non-viable tissues, clots and pus. In addition, they have anti-inflammatory and anti-edematous effects. Protein-degrading enzymes are used in the form of powder, ointment, or dressings (the so-called immobilized enzymes).

(3) Bacteriophages are substances, containing viruses, which can reproduce in bacterial cells and destroy them. There are staphylococcic, streptococcic, pseudomonades, proteus bacteriophages. They are used topically for the treatment of purulent wounds and cavities.

(4) Curative serums, which contain antibodies to main agents of surgical infection. They are used parenterally for passive immunization. Anti-staphylococcal, anti-tetanus and anti-gangrenous serums are used.

Indirect (stimulating patient's immunity) biological antiseptics are: immune stimulating substances, vaccines, anatoxines and different physical methods.

Immunostimulating substances are thymolin, T-activine, interferon. They stimulate and modulate nonspecific immunity.

Vaccines and anatoxins contain minimal dose of microorganisms or their toxins. They stimulate production of antibodies by host organism to certain bacteria (tetanus toxoid, staphylococci anatoxin).

Physical methods stimulate nonspecific resistance of patient's organism.

IV. HEMORRHAGE

Bleeding can be defined as a leakage of blood from the vascular system, including blood vessels and cardiac cavities, caused by damage of its integrity or increased permeability of the vascular wall. Bleeding represents the extremely important problem of medicine and should be considered one from the most dramatic situations in surgery.

CLASSIFICATIONS OF BLEEDING

There are several classifications of bleeding based on various criteria. Each of these classifications reflects individual anatomical, etiological, pathogenetic and clinical features of the pathological process.

(1) Anatomical classification. In accordance with anatomical structure of damaged vessel hemorrhages are divided into:

- Arterial bleeding. In this case the intensity of bleeding is determined by the diameter of injured artery and morphology of vascular damage (complete transection or lateral defect of the vessel);
- Venous bleeding. The volume of blood loss in case of venous bleeding can be significant, but manifestations of acute anemia develop more slowly comparing to the arterial hemorrhage;
- Capillary (parenchymatous) bleeding. This type of bleeding exteriorized blood has the mixed character due to concomitant injury of arterioles and venules of small diameter. If capillary bleeding originates from the parenchyma of the solid organs (liver, kidney, spleen, suprarenal gland, lung, and pancreas) it is referred to as parenchymatous bleeding.

(2) Classification of bleeding by **mechanism of occurrence:**

- *Per rhexin (Latin)* – hemorrhage occur due to mechanical injury of blood vessel. This is the most frequent type of bleeding;

- *Per diabrosin (Latin)* – bleeding caused by gradual destruction of vascular wall (erosion) by some pathological process;

- *Per diapedesin (Latin)* – bleeding which results from pathologically increased permeability of vascular wall.

(3) Classification based on the **site of bleeding:**

- External bleeding – into the external environment, usually arising from a wound.

- Internal bleeding (Figure 4):

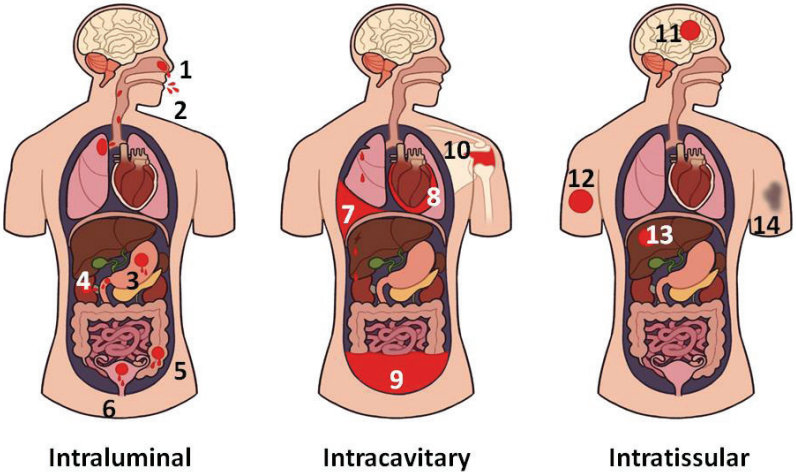

Figure 4. The main types of internal bleeding. Intraluminal: 1 – Epistaxis; 2 – Pulmonary bleeding with hemoptysis; 3 – Gastroduodenal bleeding; 4 – Renal bleeding; 5 – Intestinal bleeding; 6 – Uterine bleeding; Intracavitary: 7 – Hemothorax; 8 – Hemopericardium; 9 – Hemoperitoneum; 10 – Hemarthrosis; Intratissular: 11 – Intracerebral hematoma; 12 – Soft tissue hematoma; 13 – Liver hematoma; 14 – Ecchymosis.

a) Intracavitary – blood leaks into natural anatomical cavities of human body that have no communication with external environment (accumulation of blood into the peritoneal cavity is referred to as a hemoperitoneum, in the pleural cavity – as a hemothorax, in pericardial cavity – as a hemopericardium and in the articular cavity – as a hemarthrosis);

b) Intraluminal – leakage of blood in some natural cavities of human body communicating with external environment (gastrointestinal tract from the esophagus to the rectum, respiratory tract, urinary tract, biliary ducts, nasal cavity and ear canal, uterus and vagina);

c) Intratissular – diffuse imbibition or accumulation of effluent blood in tissues (petechia, purpura, ecchymosis, hematoma).

(4) Classification of hemorrhage by **time of development:**

- Primary hemorrhage – arising directly from vascular injury.
- Secondary hemorrhage:

a) Early secondary hemorrhage – represents the repeated bleeding that appears during the first hours or during the first day after stopping of primary hemorrhage, untill the moment of development of infection in the wound. The mechanism of this bleeding is explained by expulsion of thrombus or slippage of hemostatic thread from injured vessel;

b) Late secondary hemorrhage – occurs 5-7 days after the injury and development of suppuration in the wound. It is caused by erosion of the vessel wall or thrombus by bacterial enzymes and inflammatory mediators.

(5) Classification of hemorrhage **by evolution (intensity):**

- Acute bleeding;
- Chronic bleeding.

(6) Classification of hemorrhage **by severity:**

- Grade I (mild) – blood loss up to 750 mL, or up to 15% of circulatory blood volume (CBV);
- Grade II (moderate) – blood loss between 750 and 1,500 mL, or 15-30% of CBV;
- Grade III (severe) – blood loss from 1,500 ml to 2,000 mL, or 30-40% of CBV;
- Grade IV (very severe) – blood loss of more than 2,000 mL, that exceeds 40% of CBV, and in case of acute bleeding is considered to be fatal.

It should be mentioned that volumes of blood loss indicated above represent approximated values, calculated for adult male patient with body mass of 70 kg.

REACTIONS OF HUMAN BODY TO BLOOD LOSS

The complex of protective reactions of human organism in case of bleeding is primarily directed to equilibration of vascular resistance (capacity of vascular bed) with effective circulatory blood volume and maintains of adequate blood supply of vital organs. There are two protective reactions of human body, which develop in case of bleeding: (1) Physiological mechanisms of compensation and (2) Pathological mechanisms of decompensation.

Physiological mechanisms of compensation in hemorrhage are activated by hypovolemia and start when mean blood pressure decreases. Physiological mechanisms are:

- Increase of venous tone, what can compensate up to 10-15% of CBV loss;
- Tachycardia contributes to maintaining of minute cardiac output at the normal level;
- Centralization of blood circulation is realized by vasoconstriction (spasm) of arterioles, mainly in the skin, lungs

and digestive tract. At the same time, blood circulation of vital organs (coronary and cerebral) does not suffer for a long time;

- Hyperventilation aims to increase the oxygenation of blood and compensation of metabolic disturbances caused by tissue hypoxia;
- Hemodilution is caused by translocation of extracellular fluid into the vascular bed, what can restore up to 1 liter of intravascular liquid or 10-15% of CBV during one hour;
- The renal mechanism with decrease of urine output (oliguria) is triggered by hormone angiotensin, which promotes fluid retention in the body;
- Release of red blood cells from the “depot”. Although in physiological conditions up to 20% of all red blood cells are “stored” in the capillaries of skeletal muscles, liver and spleen, release of erythrocytes from the “depot” develops slowly and gradually.

Pathological mechanisms of decompensation in hemorrhage are:

- Myocardial ischemia and disturbances of cardiac function;
- Debilitation of sympathetic nervous system and decentralization of circulation, associated with loss of vascular tone and decreased peripheral vascular resistance;
- “Blood sequestration” – stagnation and exclusion of blood deposited in dilated vessels from effective circulation;
- Brain ischemia;
- Disturbances of metabolism and exchange of gases in organs and tissues;
- Systemic inflammatory response caused by tissue hypoxia and destruction of cells with subsequent development of multiple organ dysfunction syndrome;

- Modification of microcirculation of white blood cells and platelets, manifested by important abnormalities in function of immune system and coagulation properties of blood.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Any type of hemorrhage is characterized by general manifestations and local signs.

General symptoms of bleeding are determined by decrease of circulatory blood volume, tissue hypoxia, development of acute or chronic anemia, and do not differ in all types of hemorrhage.

- Subjective signs are: weakness, dizziness, blurred vision and photopsia, feeling of insufficiency of air, orthostatic hemodynamic instability.
- Objective symptoms include pallor of skin and mucosa, lips cyanosis, accelerated pulse of small amplitude, low arterial pressure, frequent respiration, disturbed psycho-neurological status and decrease of urine output.

Local symptoms.

In majority of cases the local signs of **external bleeding** allow not only diagnosing the fact of hemorrhage but also to determine its type (arterial, venous or capillary), volume and speed of blood loss.

In case of **internal intraluminal bleeding** blood will be exteriorized not immediately but after some time, undergoing some transformation during the passage inside of human body (through stomach, intestine, bronchus, urinary tract, etc.) The following symptoms may be seen**:**

- Hemoptysis (elimination of foaming blood from the mouth and nose) – in case of pulmonary bleeding;
- Epistaxis (bleeding from the nose) - in nasal bleeding;

- Vomiting with fresh blood, with blood clots or coffee-ground vomiting – in case of esophageal or gastric bleeding;
- Melena (semiliquid feces of black color) – sign of gastroduodenal bleeding;
- Hematochezia (bloody stool) – suggests bleeding from the colon or rectum;
- Hematuria – bleeding from the urinary tract, manifested by elimination of red or brown urine;
- Metrorrhagia – elimination of blood through the vagina, the source of bleeding is located inside the uterus.

The most difficult is the diagnosis of **internal intracavitary bleeding**. It is based on determination of blood accumulation into the closed cavities of organism and signs of dysfunction of injured organs compressed by blood:

- In case of intracerebral hematoma can be determined by bradycardia, face asymmetry, anisocoria;
- In hemothorax it is determined by dyspnea, tachypnea, skin cyanosis, reduced lung excursion, dullness on percussion, decreased or absent respiratory sounds, displacement of mediastinum;
- In hemopericardium it is determined by tachycardia, hypotension, skin cyanosis, distension of neck veins, enlarged heart limits on percussion, and muffled heart tones;
- Hemoperitoneum is characterized by abdominal pain, distension of the abdomen due to the accumulation of free fluid (blood), dullness of percussion sound at the flanks, decreased peristalsis and symptoms of peritoneal irritation;
- In hemartrosis – enlargement of the joint, acute pain, forced semiflexed position of extremity and impossibility to step on the affected leg.

Laboratory tests.

Determination of values of laboratory tests in case of bleeding has many aims: diagnosis of bleeding, estimation of blood loss, monitoring of patient state and decision making regarding the application of curative methods (blood transfusion, surgical intervention). The following laboratory tests should be studied in a patient with suspected or diagnosed acute bleeding:

- Number of red blood cells in the peripheral blood (normal values – 4.0-5.0 $x10^{12}$/L);
- Hemoglobin level (normal values – 130-160 g/L);
- Hematocrit (normal values – 40-45%).

DETERMINATION OF BLOOD LOSS VOLUME

To assess the circulatory blood volume, the following methods are used:

Allgower shock index: frequency of cardiac contraction (FCC) / systolic blood pressure (SBP).

- FCC/SBP = 0.5 – normal value;
- FCC/SBP = 0.6-0.8 – means loss of 10% of CBV;
- FCC/SBP = 0.9-1.2 – loss of 20% of CBV;
- FCC/SBP = 1.3-1.4 – loss of 30% of CBV;
- FCC/SBP ≥1.5 – loss of 40% of CBV.

The volume of blood loss can also be assessed by **red blood cell (RBC) count:**

- RBC count 4.5-3.5 mln, the volume of blood loss is 500 ml (15% deficit of CBV);
- RBC count 3.5-3.0 mln, the volume of blood loss is 1,000 ml (15-20% from CBV);
- RBC count 3.0-2.5 mln, the volume of blood loss is 1,500 ml (25-35% from CBV);

- RBC count <2.5 mln, the volume of blood loss is >1,500 ml (over 35% of CBV).

Intraoperative blood loss can be determined by **gravimetric method** (difference in the weight of saturated by blood and clean surgical textiles: bandages, meshes, tampons, drapes, gowns). The obtained value is increased by 50% and added to the volume of blood collected in the reservoir of surgical aspirator. Besides, the **special tables** were elaborated to provide average volume of blood loss for the most frequently performed surgical interventions. During surgery the volume of blood loss can be determined with highest grade of precision using the **values of patient hematocrit**.

Gross formula: $V\ (ml) = pq \times (Ht_1 - Ht_2) / (Ht_1)$

V – volume of blood loss; pq – estimated circulatory blood volume of the patient; Ht_1 – hematocrit before surgery; Ht_2 – hematocrit after surgery. So, the estimated CBV is determined on the basis of patient's weight in kilograms (p) and the coefficient (q) – the volume of blood per kilogram of weight, which in men is on average 75 ml/kg, and in women – 65 ml/kg.

Instrumental methods of diagnosis.

For diagnosis of internal bleeding, in addition to clinical and laboratory data, non-invasive and invasive imaging diagnostic methods are used: chest radiograph, ultrasound scanning, computed tomography and magnetic-resonance imaging, endoscopic methods (fibrogastroduodenoscopy, colonoscopy, bronchoscopy, cystoscopy, ureteroscopy, rhinoscopy), angiography, diagnostic fine needle aspiration (of pleural cavity, pericardial sac, peritoneal cavity, posterior vaginal fornix and articular cavity), centesis of cavity with needle (paracentesis, thoracentesis), and subsequent introduction of catheter (peritoneal lavage, thoracostomy), direct visualization (diagnostic thoracoscopy, laparoscopy).

V. BLOOD COAGULATION AND HEMOSTASIS

BLOOD COAGULATION

Hemostasis is defined as a complex of physiological mechanisms aimed to stop the bleeding. It can be said what spontaneous hemostasis is directed to the formation of the platelet plug, binded and fixed by fibrin (thrombus). The process of blood coagulation includes three main phases:

- **Phase I (vasoconstriction or vascular phase of hemostasis).** Trauma of the blood vessel leads to the contraction of smooth muscles of the media that results in prompt local reduction of blood flow and creation of favorable conditions for the development of thrombosis.

- **Phase II (platelet aggregation or cellular phase of hemostasis).** In case of endothelial injury, the thromboplastin (tissue factor) is exposed and stimulates the adhesion and aggregation of platelets to the collagen from subendothelial space. The phase ends with the formation of platelet plug.

- **Phase III (activation of coagulation cascade or plasmatic phase of hemostasis).** Although hemostasis can occur only due to vasoconstriction and platelet aggregation, the decisive role in the spontaneous stopping of bleeding belongs to the cascade of plasmatic coagulation with consecutive formation of thrombin and fibrin clot.

Hemostasis and fibrin clot formation develop in **intrinsic** and/or **extrinsic** pathways. Both of them lead to the activation of clotting factor X (Stuart-Prower factor). Extrinsic pathway plays a more important role in surgery and is associated with the release of tissue factor followed by the consequent activation of coagulation cascade. After activation of factor X, the coagulation process is continued by a single mechanism (**common pathway**). At the beginning prothrombin is

converted to thrombin and after that fibrinogen is transformed to fibrin. At the final stage, cross-linking of the fibrin fibers is realized with reinforcement of the fibrin clot under the influence of the fibrin-stabilizing factor XIII.

It should be mentioned that process of thrombus formation is limited to the site of vascular damage and has the temporary character. Hemostasis includes the complex interaction of pro-thrombotic, anticoagulant and fibrinolytic mechanisms which acts simultaneously. **Mechanisms of limitation of local coagulation** include:

- Vascular endothelium plays the role of mechanical barrier that isolates sub-endothelial pro-thrombotic factors (tissue factor, collagen) from the clotting factors circulating in the vascular system;
- Inactive state of clotting factors, whereas to initiate coagulation the activation of internal pathway through factor XII (Hageman) or activation of external pathway – through tissue factor is required;
- Activation of antithrombin III, which inhibits thrombin and Stuart-Prower factor and limits the coagulation process;
- Presence in the blood of natural anticoagulants – endogenous heparin, produced mainly by mast cells of the liver;
- Fibrinolytic system of blood, providing subsequent lysis and degradation of the fibrin clot.

SYNDROME OF DISSEMINATED INTRAVASCULAR COAGULATION

The physiological balance between pro-coagulant and anticoagulant systems of blood can be disturbed by several pathological conditions with the development of the so called **syndrome of disseminated intravascular coagulation** (DIC-

syndrome). This syndrome is also termed as “consumption coagulopathy” or “hemorrhagic syndrome”.

The **etiology** of DIC-syndrome includes:

- Severe bacterial and viral infections, sepsis;
- Severe traumatic injuries and burns;
- Major surgical procedures;
- Massive blood transfusion;
- Malignancies, especially acute leukemia;
- Certain obstetric complications, including premature placental abruption, intrauterine fetal death, amniotic fluid embolism.

Pathogenesis of DIC-syndrome is multifactorial and not fully studied. The main “triggers” for development of disseminated intravascular coagulation are:

- Activation of coagulation cascade by endogenous factors: tissue thromboplastin, products of tissue disintegration, tumoral procoagulants (in case of trauma or malignancy);
- Systemic damage of vascular endothelium with decrease of its antithrombotic properties (in case of infection, sepsis or trauma);
- Direct activation of coagulation system by microbial enzymes, especially of factor XII Hageman;
- All these factors result in generalized (but not local as usually) intravascular coagulation with formation of thrombi and blood microaggregates;
- The fibrinolytic system is acutely activated;
- Massive consumption of clotting factors leads to the depletion of their reserve with development of diffuse hemorrhages and complete loss of blood coagulability.

Clinical manifestations.

Acute, subacute and chronic forms of DIC-syndrome are distinguished. There are also two clinical and laboratory phases of this syndrome: **the phase of hypercoagulation** and **the phase of hypocoagulation**.

In the **first phase** of syndrome symptoms of main disease dominates in association with signs of generalized thrombosis, hypovolemia and disturbed metabolism. Frequently, the symptoms and signs of the first phase of DIC-syndrome are difficult to recognize or they are absent.

The **second phase** is characterized by the development of hemorrhagic complications. The syndrome is manifested by bleeding from at least three different sources: digestive tract, respiratory or urinary tracts, postoperative wound, sites of venous puncture. Patients may present with petechia, soft tissue hematoma, bleedings from mucous membranes, massive digestive hemorrhage, pulmonary and other bleedings, intracranial hematoma or other hematoma of other vital organs. Exteriorized blood has no tendency to clots formation.

Laboratory diagnosis.

Values of laboratory parameters show pronounced hypocoagulation: blood does not clot *in vitro*, severe thrombocytopenia, prothrombin time and activated partial thromboplastin time are increased, and concentration of fibrinogen drops to the critical level, and D-dimers are significantly elevated. The latter parameter indicates an increase in fibrinolysis.

For the purpose of more objective diagnosis of DIC, International Association of Thrombosis and Hemostasis proposed a scoring system:

Test	0 points	1 point	2 points	3 points
INR	≤1.3	1.3-1.7	>1.7	
Fibrinogen	>1.0 g/L	≤1.0 g/L		
D-dimers	<400 ng/ml		400-4,000 ng/ml	>4,000 ng/ml
Platelets	>100 $x10^9$/L	50-100 $x10^9$/L	<50 $x10^9$/L	
INR – international normalized ratio ≥5 points – positive diagnosis of DIC				

Treatment.

- Correction of the causative pathology that were the causes of DIC. In case of infection the antibacterial treatment is required, obstetrical complications should be corrected surgically, etc.;
- Compensation of clotting factors deficit (massive transfusion of fresh frozen plasma);
- Administration of heparin in the first phase of the disease stimulates release of antithrombin III and blocks the process of hemocoagulation;
- Symptomatic therapy in case of dysfunction of organs and systems.

MEDICAMENTOUS THERAPY

Medical treatment of major bleeding includes general measures, independently of the source and type of hemorrhage. These measures include:

- Mandatory hospitalization of patient with the major bleeding for diagnosis and treatment in the intensive care unit;

- Placement of large-bore venous catheter, preferable in the large central vein for massive transfusion therapy;
- Infusion of warmed crystalloid solutions in the volume that several times exceeds the volume of blood loss (usually 3:1) for fast compensation of circulatory blood volume;
- Blood transfusion, more exactly transfusion or red blood cells concentrate, is indicated to patients with level of haemoglobin below 70 g/L. It is recommended to combine the transfusion of red blood cells concentrate with transfusion of fresh frozen plasma at the proportion 1:1;
- Fresh frozen plasma should be transfused to patients with confirmed disorders of blood coagulation. It is important to know the minimal level of clotting factors, required for physiological hemostasis: prothrombin – 15-20% (normal values – 80-120%), fibrinogen – 1 g/L (normal values – 2-4 g/L), number of platelets – 50 $x10^9$/L (normal number – 180-320 $x10^9$/L).

TEMPORARY HEMOSTASIS

Methods of **temporary** hemostasis are:

- Application of compressive bandage or tight tamponade of the wound (Figure 5);
- Maximal flexion of extremity;
- Elevated position of extremity;
- Digital compression of bleeding vessel in the wound or along its trajectory;
- Application of hemostatic tourniquet;
- Application of hemostatic forceps on the bleeding vessel;
- Tamponade of the wound channel with a balloon catheter;

- Obturation of the lumen of the blood vessel with a Fogarty balloon catheter;

- A modern method of temporary hemostasis for massive intracavitary bleeding is the insertion through femoral artery into aorta of a special occlusive balloon (REBOA – Resuscitative Endovascular Balloon Occlusion of the Aorta).

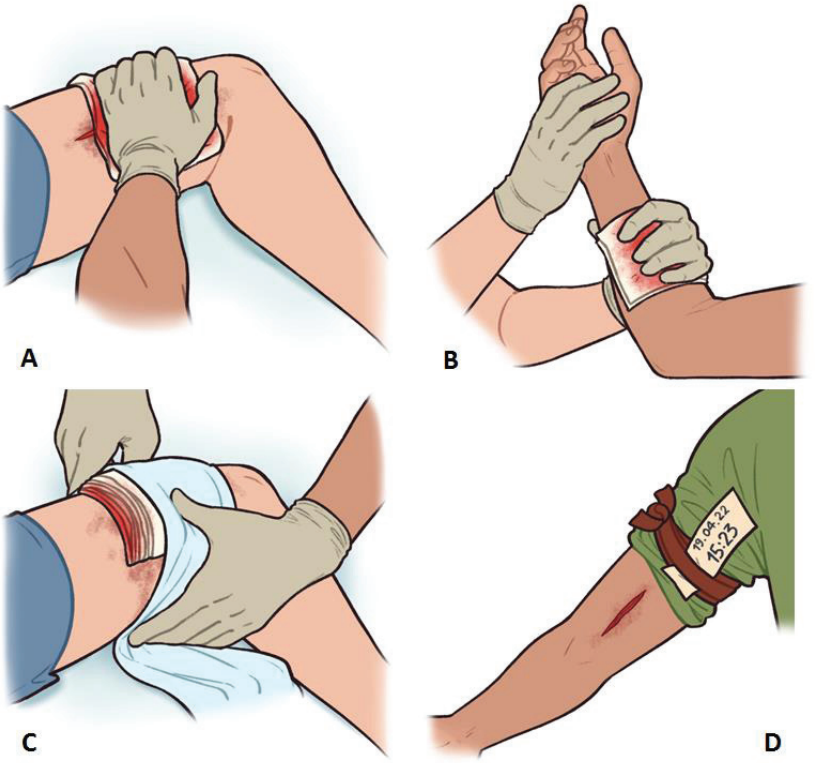

Figure 5. Methods of temporary hemostasis most commonly used in providing first aid to victims at prehospital stage: A – Manual compression of wound; B – Elevated position of the limb; C – Application of compressive bandage; D – Application of hemostatic tourniquet.

Application of hemostatic tourniquet – standard method of temporary hemostasis from arterial vessels of extremities realized by circular constriction with compression of the soft tissues together with major blood vessels. For correct application of hemostatic tourniquet, the following rules should be respected:

- Hemostatic tourniquet is applied only in case of severe arterial bleeding when the simple compressive bandage not results in an effective hemostasis;
- Hemostatic tourniquet should be applied superior (more proximally) to the bleeding site and as close as possible to the wound. If hemostasis is not complete the tourniquet can be reapplied at the level of hip or shoulder;
- For protection of skin from injury the tourniquet is applied over the patient's clothes or over some soft lining – towel, several layers of gauze;
- If tourniquet was applied correctly the pulse on peripheral artery should disappear, distal extremity becomes pale and bleeding ceases;
- Hemostatic tourniquet can be applied for no more than 1.5 hours, preventing the development of tissue necrosis;
- Because of this, the exact time of tourniquet application should be obligatory indicated on the special tag or in the medical documents of the patient;
- Traumatized patient with applied hemostatic tourniquet should be immediately transported to the surgical department for definitive hemostasis.

DEFINITIVE SURGICAL HEMOSTASIS

Methods of **definitive** hemostasis are classified according to their nature into mechanical, physical, chemical and biological.

Mechanical methods:

- Ligation of the bleeding vessel in the wound;
- Ligation or suturing of the vessel at a distance from the wound;
- Surgical repair of the vessel by application of vascular suture;
- Reconstruction of the injured segment of vessel by interposition of synthetic graft, autologous vein or artery;
- Long term wound tamponade (during several days);
- Clamping blood vessels with special titanium clips (in laparoscopic procedures);
- Clipping and ligation of bleeding esophageal varices (in endoscopic hemostasis);
- Embolization and stent-grafting of vessels (in endovascular surgery).

Physical methods:

- High temperature (monopolar and bipolar diathermocoagulation, laser photocoagulation, argon-plasma and radiofrequency coagulation). All these methods have the similar mechanism: the energy (electric current, laser) is transformed into the heat, elevates the local temperature up to 60-100°C, resulting in tissue destruction, coagulation and hemostasis;
- Low temperature (liquid nitrogen and carbon dioxide). Their action on biological tissue leads to the coagulation of proteins and blood clotting;
- Ultrasound (“Harmonic scalpel”) is used for concomitant dissection and coagulation of the tissue. In contrast with

diathermy, the harmonic scalpel uses the high frequency ultrasonic vibrations, which produces heat and results in denaturation of proteins.

Chemical methods:

- Adrenalin – causes vasoconstriction;
- Ethanol and polidocanol – leads to tissue sclerosis;
- Cyanoacrylate, which polymerizes instantly due to the contact with blood, converts from a liquid to solid substance and blocks the bleeding vessel.

Biological methods:

Biological methods are based on utilization of plasma derivates, that stimulate local thrombogenesis. These drugs contain fibrinogen and thrombin, which are activated upon contact with liquid blood. In open and laparoscopic surgery, they are used as hemostatic sponges ("Tachocomb"), or as solutions (fibrin glue, "Tissucol"). Furthermore, there are hemostatic sponges on the basis of animal (bovine) collagen ("Helistat", "Surgispon"), or cellulose ("Surgicel"), which stimulates the hemostatic process through the classical extrinsic pathway. After local application to the bleeding injury of parenchymatous organ the collagen sponges can stop the hemorrhage during the 2-5 minutes. The sponge can be left inside of organism being exposed to the complete spontaneous resorption.

VI. BLOOD TRANSFUSION

Transfusiology – is the branch of medicine which deals with transfusion of blood, its components and substitutes. **Blood transfusion –** administration of blood and/or its components into the circulatory system of the patient. Blood transfusion may be considered as a "transplant of liquid tissue" containing various blood cells with different action on the body, often negative.

HISTORY OF BLOOD TRANSFUSION

The use of the healthy person's blood to treat patients is one of the greatest achievement of medical science. Many centuries passed before this idea became a reality and won the universal recognition of world medicine. History of blood transfusion should be divided into the following periods:

Empirical era (XVII-XIX century):

- Transfusion experiments on animals (Richard Lower, 1665);
- First transfusion of blood from animals to humans (Jean-Baptiste Denys, 1667);
- First successful human to human blood transfusion (James Blundell, 1818).

Scientific era (XX century):

- Discovery of the reaction of hemagglutination and first three blood groups (Karl Landsteiner, 1900). Later, in 1930, Landsteiner would be awarded the Nobel Prize in Medicine and Physiology for this discovery;
- Discovery of the forth blood group and development of definitive blood groups classification (Jan Jánsky, 1907);

- The world's first direct blood transfusion, taking into account group compatibility (American surgeon George Crile, 1906);
- Conservation of whole blood, collected from donor, using sodium citrate (Vadim Iurevici and Nicolai Rosergart, 1914). This idea being the basis for the creation of blood banks and indirect blood transfusion method;
- Discovery of the Rhesus factor (Karl Landsteiner and Alex Wiener, 1937).

Post-war era (second half of the XX century):

- Wide indications for blood transfusion;
- Development of active blood donation and creation of blood banks;
- Discovery of the leukocyte and platelet antigenic blood systems.

Contemporary era:

- Reduction of indications for blood transfusion;
- Transfusion of blood components and plasma derivatives instead of whole blood;
- Predominant use of blood substitutes;
- Rejection of direct blood transfusion.

BLOOD GROUPS

Blood group (blood type) is defined as the presence or absence of an antigen (called agglutinogen) on the surface of red blood cells (RBC) and antibody (called agglutinin) in the serum. The blood groups are inherited according to the classical laws of genetics, remaining stable lifelong. The **AB0** and **Rhesus factor** systems are of the greatest clinical importance.

According to the AB0 system are distinguished 2 antigens – agglutinogens A and B, which are distributed separately or

together on the surface of erythrocytes, and 2 antibodies – agglutinins α and β. A unique property of the AB0 system is the presence as a norm of antibodies to antigens which are missed.

Thus, there are **four blood groups:**

- Group 0(I) – individuals do not have the agglutinogens on the surface of RBC, the agglutinins α and β are present in plasma;
- Group A(II) – individuals have the agglutinogen A on the surface of RBC, and β agglutinins – in plasma;
- Group B(III) – individuals have the agglutinogen B on the surface of RBC, and α agglutinins – in plasma;
- Group AB(IV) – individuals have the agglutinogens A and B on the surface of RBC, the agglutinins are absent in plasma.

Rh blood group system includes 49 antigens, but the only clinically important one is considered antigen D. The individuals who possesses the D-antigen are considered to be Rh (+) positive, representing about 85% of cases, and those who do not have this antigen – Rh (-) negative. In contrast to the AB0 system, the plasma of Rh-positive or Rh-negative individuals does not contain antibodies to D-antigen as a norm.

The immune reaction between RBCs antigens and antibodies is manifested by the appearance of **agglutination** – a cross-linking reaction of separated erythrocytes and specific antibodies with their hemolysis. There are different types of agglutination: iso-hemagglutination (agglutination as a result of the interaction between antigen and antibody of the same species), hetero-hemagglutination (interspecies agglutination of erythrocytes), pseudo-hemagglutination (the cross-linking of erythrocytes is not a result of interaction of agglutinogens and agglutinins, for example, at low temperature, bacterial

contamination of blood as well as in certain blood disorders and infections. Unlike the true agglutination, the false one disappears when several drops of saline solution are added to the tested blood) and pan-hemagglutination.

The reaction of iso-hemagglutination submits to the **Ottenberg's rule**, which is as follows:

- Only donor's erythrocytes of the transfused blood are agglutinated;
- Agglutinins of the transfused blood are diluted in vascular bed of patient, and they are not able to agglutinate the erythrocytes of the recipient;
- The 0(I) blood group – is “the universal donor”, the blood of group AB (IV) – is “the universal recipient”;
- The rule is valid only in significant (1:20 or more) dilution of transfused blood in the blood of the recipient;
- In case of transfusion of more than 500 ml of blood only the blood of the same group should be used.

Methods for determination (typing) of blood group:

- **Method with standard serum.** Blood group typing is performed at a temperature of +15-25°C. Standard serums of group 0(I), A(II), and B(III) are mixed with a small amount of examined blood. The assessing of the results is performed after at least 5 minutes, by the presence or absence of agglutination (Figure 6).

To avoid errors, standard sera are labeled and colored differently: serum 0(I) is colorless, the label is white; serum A(II) - bluish-green, label with a blue stripe; serum B(III) - pink, label with a red stripe; serum AB(IV) – yellow, label with yellow stripe.

- **Method with anti-A and anti-B monoclonal antibodies.** The monoclonal antibodies are typically colored: anti-A – blue, anti-B – pink. To determine the blood group, a large drop (0.1

ml) of monoclonal antibodies is applied on each dimple of plate and one drop of blood in the ratio of 10:1 (0.01 ml) is added. After mixing of the reagents with blood, the agglutination is expected for 2.5 minutes (Figure 7).

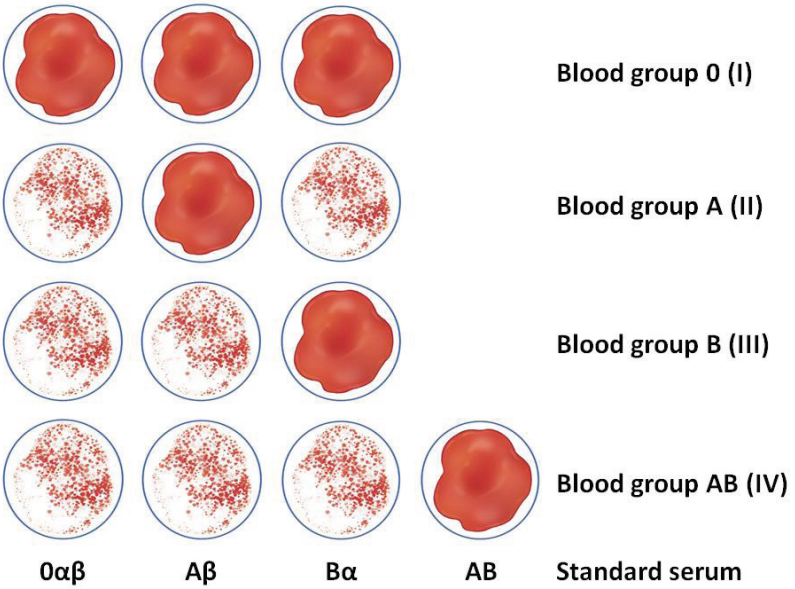

Figure 6. Determination (typing) of blood group: method with standard serum.

- **Method with standard erythrocytes**, that is, the determination of blood groups in a cross way – the detection of isohemagglutinins α and β in serum by standard erythrocytes. The method is based on interaction of natural serum agglutinins α and β and the corresponding antigens of standard AB0 erythrocytes.

Methods for determining (typing) of Rhesus factor:

- Method with anti-Rhesus serum (anti-D);

- Method with monoclonal antibodies anti-D;
- Reaction of conglutination using gelatin;
- Coombs test (indirect antiglobulin test).

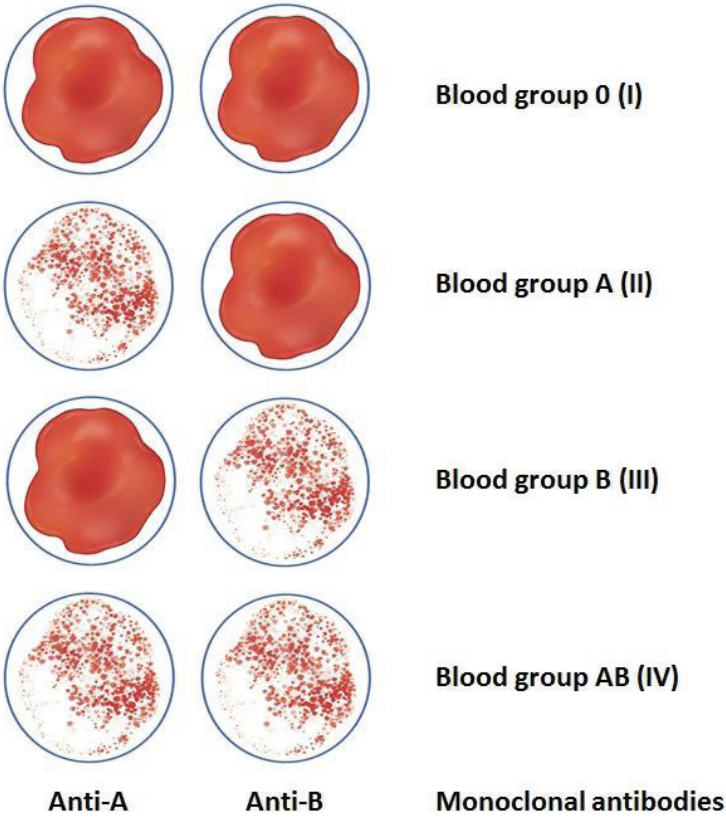

Figure 7. Determination (typing) of blood group: method with anti-A and anti-B monoclonal antibodies.

METHODS OF BLOOD TRANSFUSION

There are several methods of blood transfusion. **(1) Indirect** blood transfusion consists in its collecting, conservation and storage for a certain period at the temperature of 4-6˚C.

(2) Direct transfusion is the introduction of donated blood to the recipient immediately after its collecting, that is, without the conservation and storage. Nowadays, due to the high risk of viral infection of the recipient, the direct transfusion is performed only in exceptional cases. **(3) Autologous blood transfusion** consists in collecting and consequent transfusion of the patient's own blood back to his/her circulatory system.

Types of autologous blood transfusion:

- **Autotransfusion.** Blood is collected from the patient a few weeks before the planned operation. Typically, it is performed several samplings of 400-500 mL of blood with an interval of one week. Last collecting is made no later than 72 hours prior to surgery. The collected blood is preserved, stored, and if it is necessary – is transfused to the patient during or after surgery;
- **Acute preoperative normovolemic hemodilution** consists in exfusion of 750-1,000 mL of patient's blood immediately before surgery. The collected blood is replaced by crystalloid and colloid blood substitutes. At the same time, the hematocrit of the patient decreases. During or after surgery, to compensate the blood loss, the collected blood is transfused back to the patient, maintaining the hematocrit level of 32-35%;
- **Reinfusion** is defined as an intravenous transfusion of patient's own blood, collected during operation from the anatomical cavities (peritoneal, pleural) in case of traumatic rupture of the lung, spleen, liver, or ectopic pregnancy. Currently, reinfusion is recommended to perform only using special devices such as “Cell-Saver”, engaged in aspiration, purification and filtration of effused blood. In case of extreme necessity, to save the life of the patient, the reinfusion can be performed manually. The blood is collected from the cavity in

a sterile container, filtered through eight layers of sterile gauze, they added heparin and transfused obtained blood intravenously.

BLOOD COMPONENTS AND SUBSTITUTES

Nowadays the transfusion of separated blood components and its derivates are used instead of whole blood.

Blood components:

- **Packed RBCs (pRBC)** are obtained by the centrifugation of whole blood and are stored in plastic containers at temperature of 4-6˚C. One container (1 dose) of pRBC contains 200-300 ml and has a hematocrit of 85-95%. It is transfused taking into account the compatibility of AB0 and Rh-system. Transfusion is always indicated when hemoglobin level is less than 70 g/L and is not recommended when the hemoglobin level is over 100 g/L. For intermediate values, the decision is taken on the basis of the severity of symptoms of hypoxia, stability of hemostasis, age and general condition of the patient. After transfusion of a single dose of pRBCs an increase of hemoglobin level by an average of 10 g/L and a 2% increase of hematocrit are expected;
- **Platelet concentrate (PC)**. A dose has a volume of 50-60 ml. It is recommended to transfuse one dose of PC per 10 kg/body weight (on average 6-8 doses). It is transfused taking into account the compatibility by AB0 and Rh-system. In case of bleeding the PC is transfused if platelet counts less than $50x10^9$/L. In patients without risk of bleeding, the platelet transfusion is performed when the count is less than $20x10^9$/L;
- **Fresh frozen plasma (FFP)** is obtained from whole blood by centrifugation or plasmapheresis. After the separation from blood cells, the plasma is frozen at a temperature of minus 40°C for 30 minutes. Quick freezing preserves the activity of

clotting factors. Before the transfusion, the plasma is defrosted at a temperature of 37°C. The usual dose is 15 ml per kilogram of patient's weight. Plasma must be compatible according to AB0 system, and in women of fertile age – according to Rh also. Transfusion of FFP is indicated in case of coagulopathies. Utilization of FFP for restoration of the circulatory blood volume is considered not rational.

At blood transfusion stations, from donor plasma are produced various preparations or so-called **plasma derivates:**

- Solution of albumin – is used as a substitute remedy in the treatment of hypoproteinemia, resistant to diuretics edema, ascites;
- Cryoprecipitate – is indicated in pathologies associated with clotting factors deficiency, especially fibrinogen;
- Lyophilized thrombin – has a local hemostatic effect due to the conversion of fibrinogen into fibrin and activation of coagulation factor XIII. It is produced in a lyophilized form, which prior to topical application is diluted with sterile saline solution;
- Immunoglobulin and gamma globulin – are used in the complex therapy of various infections and for the correction of immunodeficiency;
- Concentrates of coagulation and anticoagulation factors – are used for specific and rare indications, for example, in hemophilia A.

Blood substitutes represent an effective and safe alternative for blood transfusions. The main purpose of the use of blood substitutes is the correction of hypovolemia. There are two large groups of blood substitutes:

- **Crystalloid solutions** (normal saline or 0,9% sodium chloride solution, Ringer's solution, Hartmann's solution). After intravenous administration, their leave quickly the

bloodstream, and after transfusion of one liter of solution, the CBV increases with approximately 250 mL. To correct hypovolemia, crystalloid solutions should be administered in a volume of at least three times higher compared to the volume of blood lost;

- **Colloidal solutions** (gelatin preparations – Gelofusin, Ghemaxel; dextrans – Dextran 70 or Polyglucin, Dextran 40 or Reopolyglucin; starch derivatives – Hydroxyethyl starch (HES) 450 or Refortan). These blood substitutes are able to remain in the bloodstream up to 8 hours. Thus, in case of bleeding colloids are transfused in a 1:1 ratio to the volume of blood lost. The volume of transfused colloidal blood substitutes should not exceed 1.5 liters during 24 hours.

PROCEDURE OF BLOOD TRANSFUSION

The procedure of blood transfusion includes several mandatory steps:

- Determination of absolute indications for blood transfusion and argumentation in medical case of need for transfusion of each blood component or plasma derivate;
- Information of the patient about the need of transfusion therapy, possible risks and complications, alternative treatment and obtaining the patient's consent for blood transfusion;
- Determination of AB0 and Rh blood group of the patient;
- Filling the request with the indication of required blood component, its group and Rh type and the required number of doses. In a tube, labeled with personal data, 10 ml of patient’s blood are collected to check the compatibility with transfusional component. The request and blood sample are sent to the blood transfusion department;

- Check of container integrity, exterior appearance of blood component, as well as its name, term of validity, blood group and Rhesus factor indicated on the label of the container;

- In case of transfusion of packed RBCs and platelet concentrate the determination of blood group of each container is performed;

- Test for determination of individual compatibility of RBCs or platelet concentrate with patient's plasma (on plate agglutination);

- Biological test is mandatory for the transfusion of any blood component. After the installation of the system for transfusion 15 ml of blood component is administered intravenously streamly. Within 3 minutes, the condition of the patient is assessed. In the absence of symptoms of transfusion reaction, the test is repeated two more times;

- The transfusion itself is carried out in a maximum 4 hours from the needle puncture of the transfusion container. The pRBCs are transfused drop by drop at a rate of 40-60 drops per minute. The plasma and platelet concentrate are recommended to be transfused in jet. The patient's condition is assessed after 5, 15 minutes from the start of transfusion and after that - hourly;

- After completing transfusion, the results of all tests and patient's data are introduced in a special form for blood transfusion and in the patient's case history. The assessment of patient's condition is carried out in one hour, two hours and one day after the end of transfusion;

- The containers with residues of blood components (several milliliters) and tubes with plasma samples used for compatibility tests, are stored in the refrigerator for the following two days.

POSTTRANSFUSION REACTIONS AND COMPLICATIONS

The transfusion of blood components and plasma derivates is, in fact, a transplant of liquid tissue and may be accompanied by a variety of adverse effects and complications. The adverse effects of blood transfusion are divided into: **posttransfusion reactions** and **posttransfusion complications**. Posttransfusion reactions are short-term, do not lead to serious dysfunctions of organs and systems, and do not represent a threat to the life of the patient. Posttransfusion complications are severe and can have a fatal character. Posttransfusion complications and reactions are classified into **acute** or early (developing within 24 hours of transfusion) and **late** (after 24 hours), and also into **nonimmune** and **immune mediated**.

Acute nonimmune reactions and complications are as follows: (1) Acute sepsis and endotoxic shock; (2) Hypothermia; (3) Pyrogenic reactions; (4) Citrate toxicity and hyperpotassemia; (5) Air embolism, thromboembolism; (6) "TACO" – Transfusion Associated Circulatory Overload; (7) Massive transfusion syndrome.

Acute immune mediated reactions and complications are: (1) Acute hemolytic transfusion reactions or the so called transfusion shock; (2) Non-hemolytic febrile antigenic reactions; (3) Allergic reactions (urticaria); (4) Anaphylactic reaction; (5) "TRALI" syndrome – Transfusion-Related Acute Lung Injury.

Late nonimmune reactions and complications: (1) Hemotransmissibile infections (HIV, hepatitis B and C, cytomegalovirus, malaria, syphilis); (2) Posttransfusion hemosiderosis.

Late immune mediated reactions and complications: (1) Late hemolytic transfusion reactions; (2) Posttransfusion

thrombocytopenic purpura; (3) Posttransfusion „transplant versus host" disease; (4) Posttransfusion immunosuppression.

The most severe complication is **hemolytic** or **transfusion shock**. The cause is the transfusion of incompatible blood components according to AB0 system, Rh factor and, rarely, other erithrocitar antigens.

Periods of hemolytic shock:

- I period (or period of shock);
- II period (or period of acute renal failure). During the second period the phases of oligoanuria, polyuria and recovery of diuresis are distinguished;
- III period (or period of reconvalescence).

The **treatment of hemolytic shock** in the initial phase consists of the following actions: stop of transfusion, intravenous crystalloid transfusion with the addition of norepinephrine and dopamine, as well as glucocorticoid hormones (hydrocortisone). The patient is immediately transferred to an intensive care unit, and a container with a transfusion component is placed in the refrigerator for further analysis. Intensive care unit management includes: (1) Maintaining adequate respiration and oxygen therapy; (2) Introduction of blood substitutes in order to maintain stable hemodynamic; (3) Stimulation of diuresis; (4) Introduction of antihistamines and opioid analgesics; (5) The treatment of DIC syndrome.

Posttransfusion reactions

Depending on the clinical evolution and degree of fever, **3 grades of severity** of posttransfusion reactions are distinguished:

- Mild grade is characterized by increase of body temperature with 1°C, myalgia, headache, chills. Symptoms have a short duration and it's easy to treat;

- Moderate grade is characterized by 1.5-2°C elevation of body temperature, strong chills, myalgia and marked headache, tachycardia and increase of respiratory rate, sometimes rash;
- Severe grade is characterized by the body temperature increase of more than 2°C, there are intensive chills, cyanosis, terrible headache, vomiting, excruciating bone pain, lumbar pain, dyspnea, urticaria.

If a transfusion reaction is suspected (AB0 incompatability, haemolytic reaction, bacterial infection, severe allergic reaction, or transfusion-related acute lung injury):

- Stop the transfusion;
- Keep the IV line open with 0.9% saline;
- Record all observations, give supplemental oxygen;
- Double-check the blood unit label with the patient's identifiers;
- Send the unit of blood product and the transfusion set to the blood bank;
- Take 40 mL of blood: 10 mL send to blood bank; 10 mL – for urea and electrolytes; 10 mL – for coagulation screening; 10 mL – for blood cultures;
- Contact the blood bank directly by phone;
- Give broad spectrum antibiotic if infection suspected;
- Monitor fluid balance and urinary output.

VII. NUTRITIONAL DISTURBANCES. ENTERAL AND PARENTERAL FEEDING

Malnutrition is a nutrients deficiency, which is accomplished by increasing risk of complications. Although the disease process is usually the major cause of malnutrition (illnesses of digestive system, metabolic disorders, systemic inflammatory reactions), many patients lose additional weight during hospitalization because some diagnostic tests are done after an overnight fast and after majority of surgical procedures meals are limited. Additional risk factors are response to surgical stress and side effects of pharmacotherapy.

NUTRITIONAL ASSESSMENT

Nutritional assessment begins with the **history**. In most cases, the possibility of malnutrition is suggested by an underlying disease or by a history of recent weight loss.

Dietary history can give a good estimate of the patient's intake of calories, protein, amino acids, vitamins and trace metals.

The extent of malnutrition can be estimated based on **physical findings**. The amount of subcutaneous tissue on the extremities, abdomen, and buttocks and in the buccal fat pads reflects the status of caloric intake. The following signs of malnutrition may also be found:

Skin: dry skin, decreasing of elasticity, rash, delayed wound healing.

Nail: frailty and deformities.

Hair: increased quality and color, recent loss.

Teeth: erosions, abnormal loss, gingivitis.

Eyes: keratoconjunctivitis, blindness.

Lips: fissures and scars.

Tongue: brightly red, with prominent papillae (glossitis), mucosal atrophy.

Face: round and edematous, pallor.

Muscular system: reduced volume, weakness, pain, convulsions.

Bones: demineralization, contortion.

Extremities: reducing of muscle size and strength, symmetrical pedal edema.

Heart: chamber enlargement, murmurs.

Abdomen: hepatomegaly, abdominal mass, ostomy, enterocutaneous fistula.

Rectum: stool color, perineal fistula.

Neurological status: lethargy, apathy, depression, psychiatric disorders.

Laboratory tests. Complete blood count: increase of hemoglobin, hematocrit, RBC, WBC, thrombocytopenia.

Electrolytes. Abnormalities in serum electrolyte concentration is the result of external losses (diarrhea), decreased excretion (renal dysfunction), or overdosage of diuretics.

Liver function tests (AST, ALT, alkaline phosphatase, bilirubin, albumin, prothrombin) may be abnormal. The value of **serum albumin** less than 3.0 g/dL correlates well with body protein deficiency, and is one of the most important diagnostic criteria for malnutrition.

Immune function is frequently altered by malnutrition:

- **Delayed-type hypersensitivity** is anergy to common skin antigens.

- **Total lymphocyte count (TLC)** is calculated by the following formula: TLC = % lymphocytes x WBC/100. Where 1,500-1,800 mm^3 – mild depletion; 900-1,500 mm^3 –

moderate depletion; and less than 900 mm^3 – severe depletion of immunity.

ANTHROPOMETRICS

Anthropometrics is useful to compare an actual body weight of the patient with his **usual weight**.

Usual body weight p**ercentage** = Actual weight (100)/ Usual weight.

The grade of the body weight loss should be assessed in dependence on the time period. The body weight loss is divided into significant and severe ones.

Period	**Significant weight loss**	**High weight loss**
1 week	1-2%	>2%
1 month	5%	>5%
3 months	7.5%	>7.5%
6 months	10%	>10%

If a patient doesn't know his actual weight, an ideal weight can be used for the calculation:

Absolute weight loss = Actual weight (100)/ Ideal weight

The calculation of the **ideal body weight** is done using the following formula:

- For females: 45.5kg for height of 152 cm + 0.9kg for every 1cm over 152cm;
- For males: 48.1kg for height of 152 cm + 1.1kg for every 1cm over 152cm.

Body mass index (BMI): = weight (kg) / height $(m)^2$.

Classification of the body weight in accordance with BMI:

Insufficient weight	less than 18.5
Normal weight	18.5-24.9
Overweight	25-29.9
Obesity	30-34.9 (1^{st} grade)
	35-39.9 (2^{nd} grade)
Morbid obesity	40 and more (3^{rd} grade)

Body fat approximates the thickness of the **triceps skin fold (TSF)**. It is measured as follows: (1) the forearm should be relaxed down along the body; (2) measure the distance between the shoulder and the elbow, determine the midway; (3) to the midpoint of the arm on the posterior aspect grasp skinfold together with subcutaneous tissue, but without muscles; (4) measure the skinfold with calipers.

Protein, most of which resides in skeletal muscle, is estimated by correction the **mid-humeral circumference (MHC)** to account to subcutaneous fat tissue **(TSF)**, which gives the **mid-arm muscle circumference (MAMC).**

For measurement of the **mid-humeral circumference (MHC)**: (1) determine midway between the shoulder and the elbow and mark it; (2) measure the arm circumference on the level of middle point, without compression of soft tissues.

MAMC = MHC - (π) (TSF) / 10. Obtained data are compared with normal values for the patient's age and sex to determine the extent of depletion. A left shoulder circumference less than 23.5 cm usually corresponds to a BMI <20 kg/m^2, and a circumference greater than 32 cm corresponds to a BMI >30 kg/m^2.

Dietary history and anthropometric findings can be used to diagnose malnutrition. For this purpose, specially designed scoring systems are usually used, for example, MUST (Malnutrition Universal Screening Tool) or PONS (Pre-Operative Nutrition Score): BMI <18.5 kg/m^2 (<20 in patients over 65 years old) – 1 point; unintentional loss of more than 4 kg of weight during the last three months – 1 point; consumption of less than 50% of the usual diet during the last week – 1 point. A score of 1 indicates moderate and ≥2 indicates high risk of malnutrition.

NUTRITIONAL REQUIREMENTS OF PATIENT

Determination of patients' energy requirements is of high importance for assessments of malnutrition or over nutrition. Energy consumption may be assessed more exactly with direct or **indirect calorimetric measurements (Weir formula).** In this method the energy consumption is calculated by oxygen consumption and carbon dioxide delivery. However, this method is very difficult and labor consuming.

The basal energy requirements can be calculated using the **Harris-Benedict equation**.

Men = 66.5 + 13.8 (weight in kg) + 5 (height in cm) – 6.8 (age in years)

Women = 66.5 + 9.8 (weight in kg) + 1.8 (height in cm) – 4.7 (age in years)

For example, a 70-kg man with height of 170 cm, and the average age (40 years) consumes: 66.5 + 966 (13.8 x 70) + 850 (5 x 170) – 272 (6.8 x 40) = 1,610 kcal/day.

Approximate basal metabolic rate can also be calculated based on **body weight alone**. Although metabolic rate varies with age and sex, these factors are not major determinants. **Approximate basal metabolic rates** in adults: 50kg – 1,300 kcal/d, 60kg – 1,450 kcal/d, 70kg – 1,600 kcal/d, 80kg – 1,750 kcal/d, 90kg – 1,900 kcal/d, 100kg – 2,050 kcal/d.

These formulas help to calculate only the basal energy consumption. Real energy consumptions are increasing in surgical patients and significantly higher. After elective uncomplicated surgery the correction coefficient by Harris-Benedict formula is 1.1; in sepsis it is 1.2-1.5; after trauma – 1.4-1.6; and after major burns – 1.5-1.9.

ENTERAL NUTRITION

In general, the enteral rout is preferred over the parenteral rout. Enteral feeding is simple, physiologic, relatively inexpensive, and well tolerated by patients. Enteral feeding maintains the mucosal integrity, absorptive function and normal microbial flora of the gastrointestinal tract.

Enteral tube feeding is **indicated** for patients who have functional gastrointestinal tract, but are unable to sustain an adequate oral intake, as well as with severe malnutrition before surgery. Enteral feeding may be **contraindicated** in patients with intestinal obstruction, ileus, gastrointestinal bleeding, severe diarrhea, vomiting, enterocolitis, high-output enterocutaneous fistula.

Nasogastric, nasoduodenal, nasojejunal, and jejunal **feeding tubes** are available for the administration of enteral feeding products. With an expected length of tube feeding for more than 4 weeks, the placement of a percutaneous endoscopic gastrostomy is recommended (Figure 8).

Enteral feeding products. Standard solutions provide 1 kcal/mL; calorically concentrated solutions (>1 kcal/mL) are available for patients who require volume restriction. Currently available dietary formulation for enteral feedings can be divided into:

- Blenderized tube feeding can be composed from any food that can be blenderized. Caloric consumption of these formulas is equal to normal food.
- Nutritionally complete commercial formulas (standard enteral diets) vary in protein, carbohydrate and fat composition. Commercial formulas are convenient, sterile, and recommended for patients with normal gut function.
- Chemically defined formulas are commonly called elemental diets. The nutrients are provided in predigested and

readily absorbed form. They contain protein in the form of amino acids.

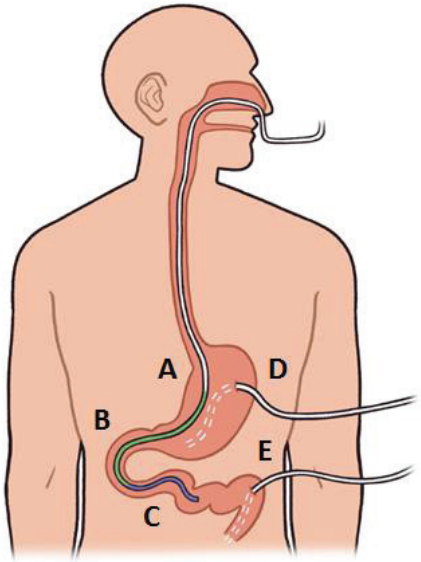

Figure 8. Routes of enteral feeding administration: A – Through nasogastric tube; B – Through nasoduodenal tube; C – Through nasojejunal tube; D – Through gastrostomy; E – Through jejunostomy.

- Modular formulas include special formulas that are used in specific clinical situations (pulmonary failure, renal or hepatic failure, immune dysfunction).

There are two **enteral feeding protocols:** bolus (fractional) and continuous infusion feeding. Bolus feeding is used in patients with nasogastric or gastrostomy feeding tubes. Feeding are beginning to administrate at 50-100 mL every 4 hours and are increased in 50 mL until the intake goal is reached (usually 240-360 mL every 4 hours). Continuous infusion is administrated by a pump is generally required for

nasojejunal, gastrojejunal or jejunal feeding tubes. With continuous administration, the initial infusion rate is 20-50 ml/hour, with subsequent correction depending on individual tolerance.

Metabolic complications. Hyperglycemia may occur in many patients, but it is particularly common in persons with diabetes. The serum glucose level should be determined frequently, and regular insulin should be administrated.

Tracheobronchial aspiration is a serious complication in patients with central nervous system abnormalities, and those who are sedated.

Diarrhea occurs in 10-20% of patients. Diarrhea may result from numerous causes: a too rapid increase in the volume of tube feeding, diet that is high in fat content or the presence of components not tolerated by the patient.

PARENTERAL NUTRITION

Parenteral nutrition is indicated for patients who require nutritional support, but cannot meet their nutritional needs through oral intake. Parenteral nutrition is indicated when the alimentary tract is obstructed (esophageal or gastric malignancies); when the alimentary tract is too short (after massive bowel resection); when the alimentary tract is fistulated (gastric or upper enterocutaneous fistulas); when the alimentary tract is inflamed (Crohn's disease and ulcerative colitis); when the alimentary tract cannot cope (ileus secondary to intra-abdominal inflammations such as acute pancreatitis).

Parenteral nutrition is divided into **(1) partial parenteral nutrition**, and **(2) total parenteral nutrition,** which provides complete nutritional support.

Total parenteral nutrition solutions are administered as a **“three-in-one”** admixture of:

(1) Protein, as amino acids (10%; 4 kcal/g),

(2) Carbohydrate as dextrose (50%-70%; 3.4 kcal/g), and

(3) Fat, as a lipid emulsion (20%; 9 kcal/g).

Total parenteral nutrition preparations provide total calories that are break down as 50-60% of carbohydrate, 25-30% of fat and 15% of protein. Electrolytes (sodium, potassium, chloride, calcium), trace elements (copper, chromium, zinc, iron) and vitamins (A, B, C, K) are added to the TPN.

Parenteral nutrition solutions must be administrated through a central venous catheter (Figure 9). They must be delivered into a high-flow system to prevent venous sclerosis because of the hyperosmolarity of solutions.

The three types of **complications** are mechanical, metabolic, and infectious.

Mechanical complications are: pneumothorax, air embolism, catheter embolism, and subclavian vein thrombosis.

The most common **metabolic** complication is hyperglycemia. Hyperglycemia may lead to coma and death. The serum glucose level should be monitored to prevent them.

Infectious complications may include subclavian catheter sepsis. Catheter sepsis is suggested by fever of no other obvious origin. In the presence of sepsis, the catheter should be changed or replaced.

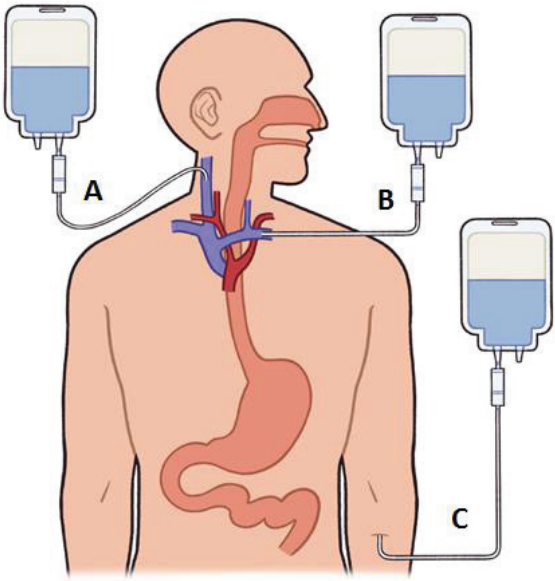

Figure 9. Methods of parenteral nutrition administration: A – Via central venous catheter in internal jugular vein; B – Via central venous catheter in subclavian vein; C – Via peripheral venous catheter.

OBESITY

Massively obese patients are those who weigh more than twice the calculated ideal weight, or who have BMI greater than 40. This degree of excessive weight has been termed **morbid obesity**.

Complications of morbid obesity are significant. The death rate is more than 10 times higher in morbidity obese young people than in people with average weight. The following complications are typical for obesity: cardiopulmonary effects, diabetes mellitus, joint diseases, cholelithiasis, fat induced liver diseases, thromboembolic disorders, endocrine dysfunction, and psychosocial problems. Nearly all medical

sequelae associated with obesity are reversible on resolution of the obese state.

Therapy always begins with reducing diets. Unfortunately, these measures are almost uniformly unsuccessful in patients with morbid obesity. Consequently, surgical therapy has assumed an important role in the treatment.

- **Jejunoileal bypass** involves anastomosis of the proximal jejunum to the terminal ileum. Weight loss results from malabsorption due to the shortened bowel. Follow-up showed a high rate of complications: protein deficiency, liver cirrhosis, renal stones.

- **Gastric bypass and gastroplasty.** The 3 types of gastric operations used to treat obesity are: (1) horizontal gastroplasty, (2) vertical banded gastroplasty, and (3) gastric bypass. Formation of a small (30-50 mL) pouch across the proximal stomach and a small (1 cm) channel for the passage of food is common for all procedures. In the first 2 years after gastric bypass brings the patients 30% of their weight. Thereafter the weight remains stable.

VIII. SURGICAL INTERVENTION. PRE- AND POSTOPERATIVE PERIOD

Surgery is an invasive procedure and may be associated with significant rate of perioperative morbidity and severe complications. Usually surgical intervention lasts no more than some hours, but patient is hospitalized for some days or even weeks. This time is required for preparation of patient for surgery and postoperative treatment. So, surgical intervention divides the treatment of patient into two periods – preoperative and postoperative.

PREOPERATIVE PERIOD

Preoperative period starts at the moment when a surgical disease was diagnosed and decision to operate was made. In case of acute surgical pathology, the beginning of the preoperative period in general corresponds with the moment of hospitalization of patient, whereas in chronic diseases – with the moment when patient addresses to his family doctor or goes to the primary medical consultation. Accordingly, the duration of the preoperative period is also different, which can vary from minutes to weeks.

There are two main **goals** of preoperative period:

(1) To reduce the surgical risk;

(2) To increase the curative effect of surgery.

There are also two **stages** of preoperative period: **diagnostic stage** and **stage of preparation**. During **diagnostic stage**, the diagnosis of a surgical disease is clarified on the basis of laboratory and instrumental studies. The data obtained allow the doctor to decide the optimal variant and aim of elected surgical intervention, as well as to determine the degree of surgical urgency.

Some specific tests may be helpful for prompt assessment of cardiorespiratory function: breath-holding test for maximal possible time after a full inhalation (**Shtanghe's test**) or exhalation (**Ghence's test**).

Prior to any surgical intervention a set of **routine diagnostic tests** is performed:

- Full blood count and urinalysis;
- Blood chemistry (total protein and albumin, glucose, bilirubin, liver enzymes, creatinine, urea);
- Coagulogram (prothrombin and fibrinogen);
- Blood group and Rh factor;
- Chest X-ray and ECG.

If some of the above mentioned test are found to be abnormal the consultation of specialist – e.g. cardiologist, endocrinologist may be necessary as well as more complex diagnostic tests.

The degree of operational risk is classified according to the ASA system (American Society of Anesthesiologists):

Grade I – patients with no systemic disturbances of vital organs function;

Grade II – patients with mild to moderate disturbances of vital organs function;

Grade III – patients with severe disturbances of vital organs function;

Grade IV – patients with life-threatening disorders of vital organs function;

Grade V – patients with little or no chance of survival with surgery or even without it (moribund).

The letter "E" is added to the assessed grade of risk in case of emergency.

Indications for surgery are divided into absolute and relative ones. Absolute indications to surgery exist in case of

life-threatening or incapacitating disease which can be treated only by means of operation. In case of an emergency the absolute indications can be named as vital because the surgery will be the life-salvaging. Relative indications for surgery can be in two situations: if a patient suffers of surgical pathology with no significant impact to the patient's health and life, or in case of diseases in which surgery and medical treatment either may give the good result.

In some cases, the risk of surgery can exceed the danger of disease and potential harm for patient's health. In such a case the surgery may be contraindicated. **Contraindications** are divided into absolute and relative ones as well.

The **stage of preparation** of patient for surgery includes the following tasks:

- Psychological support (providing to patient full information about his disease, the nature of proposed surgical procedure, its immediate and long-term results, possible postoperative complications and measures to prevent them, as well as existing alternative methods of treatment);
- Correction of concomitant diseases;
- Measures for prevention of complications;
- Special preoperative care or preparation.

If the patient agrees to be operated on he should sign the special document called the **informed consent** for surgery. The aim of general preparation of the patient for surgery is optimization of organ function which can be deteriorated by concomitant diseases: arterial hypertension, ischemic heart disease, diabetes mellitus, chronic obstructive pulmonary disease, renal diseases, anemia, liver diseases and coagulation disorders.

The most severe and dramatic postoperative complication which can be prevented is venous thromboembolism. The

most important factors **of venous thromboembolism risk** are: age, obesity, history of deep vein thrombosis, malignant tumors, major surgery and prolonged perioperative immobility.

The three basic components of prophylaxis are:

- Early postoperative ambulation;
- Elastic compression of lower limbs;
- Anticoagulation.

The next important stage in the preparation of patients for surgery is the administration of **prophylactic antibiotics**. Broad-spectrum antibiotics (cephalosporin) are administered in the absence of infection, with the aim to reduce the rate of surgical site infection, which result from existing or potential contamination during surgery. More detailed information about antibiotic prophylaxis is presented in the corresponding section of textbook.

Routine procedure of **patient preparation** includes also:

- Administration of sedatives before surgery;
- Hygienic shower with cleaning and shaving of future surgical field (not earlier than 2 hours before surgery);
- Digestive tract emptying: osmotic laxatives, cleansing enemas, restriction of oral nutrition (according to modern concepts, a 12-hour fast before a planned operation is not rational – solid food should be excluded 6 hours, and liquids – two hours before anesthesia);
- Catheterization of urinary bladder;
- Preparation of central or peripheral venous access.

The most important details of preoperative period (argumentation of diagnosis, indications for surgery, planned volume and type of surgery, type of anesthesia and degree of risk, availability of patients' informed consent) are

summarized in the **preoperative conclusion** recorded in the patient's medical case history.

The preoperative period ends with the transportation of the patient to the operating room. Usually patient is transported to the operating suite in supine position on the stretcher with assistance of at least 2 health workers.

Position of patient on the surgical table depends on the type of surgery:

- Supine/dorsal recumbent (is typically used; in this case the operating surgeon is staying on the right side of the patient, *vis a vis* with his assistants);
- Trendelenburg position (with elevated lower limbs, is used for surgery on pelvic organs);
- Reverse Trendelenburg position (with elevated head end – for surgeries on subdiaphragmatic zone);
- Sitting or semi sitting position (Fowler position) used in dental surgery;
- Dorsal lithotomic position (patient lies on his back with legs spread, elevated and fixed on supports, used for gynecological procedures and operations on the rectum);
- Lateral position of patient (for thoracotomy or kidney surgery).

SURGICAL PROCEDURE

Surgery can be defined as a mechanical influence upon tissues and organs, performed with diagnostic or curative purpose and usually associated with incision of teguments for access to the affected organ or to pathological process.

Surgical intervention starts with a skin incision, and is considered to be finished when the last skin suture is applied.

There are **4 steps of surgical intervention** (Figure 10)**:**

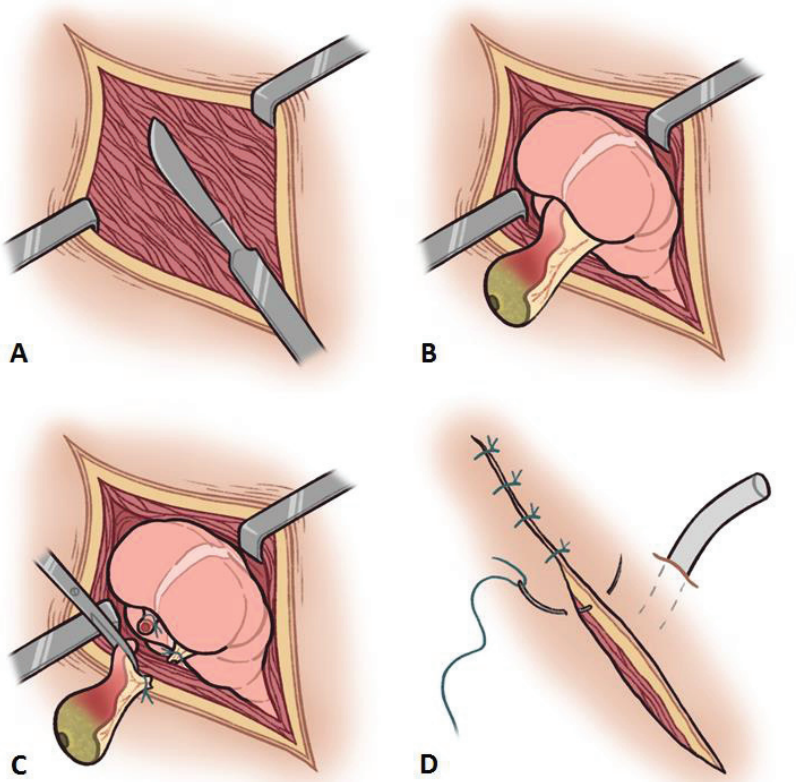

Figure 10. Basic steps of surgical intervention: A – Access (laparotomy); B – Exploration (confirmation of appendiceal inflammation); C – Procedure (appendectomy); D – Completion of intervention (drainage of abdominal cavity, wound closure).

(1) Access (should be wide enough, anatomical, less traumatic, physiological and aesthetic);

(2) Exploration (allows surgeon to confirm and definitively establish the location and extension of pathological process, as well as to clarify the plan of surgery);

(3) Procedure (drainage of pus collection, excision of mass, correction of existing pathological changes by removing an organ or part of it, applying anastomoses, implanting artificial prostheses);

(4) Check and closure (including quality of hemostasis and absence of foreign bodies, placement of drainage and wound closure by layers).

A broad spectrum of surgical interventions can be classified according to some principles. There are three types of interventions according to the **degree of emergency**:

(1) Immediate emergency – delay of surgery leads to the rapid death of patient (airway obstruction, profuse bleeding);

(2) Emergency – delay of surgery may result in severe complications (acute appendicitis, perforated gastric ulcer);

(3) Elective – time of surgery performance does not influence the results (varicose veins, benign tumors).

According to the **aim** of surgery interventions are divided into diagnostic (performed in order to determine the diagnosis or stage of disease) and curative. Curative surgical interventions may be radical or palliative. Palliative interventions are performed with the aim to prolong the patient's life or to improve the quality of life without definitive cure of pathology. As example may serve application of external intestinal fistula (stoma) for inoperable bowel cancer with intestinal obstruction, or gastrostomy for feeding in case of advanced esophageal cancer.

There are four types of surgical interventions according to the **degree of contamination**:

(1) Absolutely clean (aseptic) – elective surgery not associated with opening of hollow organs (abdominal hernia repair, cardiovascular and orthopedic surgery);

(2) Clean (conditionally aseptic) – interventions with opening of slightly contaminated organs (upper gastrointestinal tract, respiratory tract, urinary system);

(3) Infected – associated with opening of heavy contaminated organs or with a high risk of bacterial contamination (large bowel procedures or surgery for perforations of hollow viscus);

(4) Purulent (septic) – surgery for purulent and septic processes (peritonitis, abscess, phlegmon).

There are also the following **types of surgeries**:

According to the **number of stages** surgeries are divided into one-stage surgeries and multiple-stage surgeries, when for complete recovery of the patient it is necessary to perform several surgical interventions separated by a certain interval.

Repeated operations – surgical interventions performed on the same organ, for the same pathology or its complications, and during the same hospitalization (usually they carry the prefix "re-", as a relaparotomy, rethoracotomy);

Simultaneous operations – interventions performed on two or more organs, for different pathologies, but during one surgical procedure;

Combined operations – are interventions performed on different organs for the same disease.

POSTOPERATIVE PERIOD

The operation is finished with the application of last suture on the skin wound, and the postoperative period starts. Immediately after surgical intervention patients require a strict **postoperative monitoring**, usually performed in the intensive care unit:

- Blood pressure, heart rate, central venous pressure;
- Pulse oximetry;
- Urine output;

- Tubes and drains;
- Surgical wound;
- Function of operated organ.

Postoperative treatment includes: administration of analgesics, cardiovascular support, adequate oxygenation and respiratory support, restoration of blood volume and nutritional support. In the absence of contraindications, enteral nutrition (oral or tube feeding) is started as early as possible – on the first or second day after surgery.

Physiological staging of postoperative period includes three stages: the **catabolic** stage (5-7 days); the short **transitory** stage (3-5 days) and **anabolic** stage (3-4 weeks).

In the routine clinical practice, the postoperative period is usually divided into: **early** – 3-5 days after the surgery, **late** – 2-3 weeks after the surgery and **remote** – from 3 weeks up to 3 months after the surgery.

Postoperative period can be associated with **postoperative complications**.

Complications of the early postoperative period are:

- Bleeding;
- Shock (hypovolemic, toxic, cardiac);
- Cardiorespiratory insufficiency (myocardial infarction, pulmonary embolism);
- Anastomotic leakage.

Complications of the late postoperative period are:

- Wound infection;
- Pneumonia;
- Collections of infected contents in the cavities;
- Urinary tract infection.

Complications of the remote postoperative period are:

- Recurrence of disease;
- Stenosis and occlusion of anastomosis;
- Vascular graft thrombosis.

IX. LOCAL ANESTHESIA

Local anesthesia is a reversible loss of pain and other types of sensation in a limited area of the body of a conscious patient, associated or not with temporary absence of active movements, induced by injection of specific drugs – local anesthetics. There are many **advantages** of local anesthesia:

- Reduced surgical stress associated with local anesthesia (can be used in patients with comorbidities who are unfit for general anesthesia);
- Spontaneous respiration and airway maintenance with low risk of regurgitation and pulmonary aspiration of gastric content;
- Postoperative recovery is faster with early discharge of the patient;
- Patient can maintain the verbal contact with doctors during surgery;
- Suitable for out-patient surgery.

However, it should be mentioned that a good cooperation of patient and treating physician is imperative for local anesthesia (limited applicability in pediatric surgery) and sometimes the additional **intravenous sedation** may be required for adequate pain control.

LOCAL ANESTHETICS

The common mechanism of action of local anesthetics is a reversible block of the transmission of neural impulses when the drug is inserted on or near the nerve membrane. The nerve conduction is blocked due to stabilization of sodium channels in their closed state and prevention of propagation of action potentials along the nerve. Neural function returns spontaneously as the drug is metabolized or removed from the nerve by blood flow.

Drugs used for local anesthesia are divided into **two groups** based on their chemical structure: **amides** (lidocaine, bupivacaine) and **esters** (procaine or novocaine). These drugs differ by their physicochemical characteristics, speed of onset and duration of anesthetic effect (based on lipid solubility and tissue binding) as well as in typical doses used for anesthesia.

Amides. Lidocaine has a more rapid onset and its action is shorter than that of bupivacaine. Both drugs are widely used for tissue infiltration, regional nerve blocks, spinal and epidural anesthesia. Bupivacaine is more cardiotoxic than other local anesthetics. It has a direct effect on the ventricular muscle, and as it is more lipid soluble than lidocaine, it binds tightly to sodium channels (it is called the *"fast-in*, *slow-out"* local anesthetic). All amides are 95% metabolized in the liver, with 5% excreted unchanged by the kidneys.

Esters. Procaine, synthesized in 1905 as a nontoxic cocaine substitute, has a shorter duration comparing with amides and is used mainly for infiltration of tissues. Esters are hydrolyzed in the blood by pseudocholinesterase. Some metabolites have a greater allergic potential than the metabolites of amide anesthetics. However, true allergies to local anesthetics are relatively rare, several adverse reactions being usually caused by toxicity of drug.

Toxicity of local anesthetics is usually caused by overdosing or by rapid delivery of drug into the circulation due to accidental intravascular administration. Toxicity is first manifested by neurological disturbances and later – by cardiovascular effects. The early signs are: restlessness and complaints of tinnitus ("ringing of the ears"), followed by slurred speech, seizures, and unconsciousness. With increasingly elevated plasma levels of local anesthetics, progression to hypotension, increased P-R intervals on ECG, bradycardia, ventricular tachycardia and fibrillation or

complete atrioventricular heart block and cardiac arrest may occur.

The **toxic dose** of local anesthetics depends on the balance between the speed of absorption in circulation and speed of drug metabolization. Generally, the average toxic dose of lidocaine is considered approximately **5 mg/kg** and that of bupivacaine is approximately **3 mg/kg**. Calculation of the toxic dose before injection is mandatory. For example, in 80 kg patient the maximum dose of lidocaine is: 80 kg x 5 mg/kg = 400 mg. It is helpful to remember that the concentration (%) multiplied by 10 will provide the mg/ml for any solution. A 0.5% solution of lidocaine is 5 mg/ml. So, the allowed amount of anesthetic solution in our example would be: 400 mg / 5 mg/mL = 80 ml. Besides the dose calculation for prevention of local anesthetic toxicity it is recommended to detect the unplanned intravascular position of the needle by aspiration before injecting and by adding epinephrine, which slows drug absorption. Comparative characteristics of the main local anesthetics are presented in the table.

Local anesthetics	Start of action	Length of action		Maximum dose in adults	
		Adrenalin (+)	Adrenalin (-)	Adrenalin (+)	Adrenalin (-)
Amides					
Lidocaine	<1 min	60-400 min	30-120 min	7 mg/kg	4.5 mg/kg
Bupivacaine	2-10 min	240-480 min	120-240 min	3 mg/kg	2.5 mg/kg
Esters					
Procaine	5 min	30-180 min	15-90 min	14 mg/kg	10 mg/kg

In case of local anesthetic toxicity with an overdose of local anesthetics, the basic elements of treatment are oxygen, airway support, and pulmonary ventilation. For cessation of seizures benzodiazepine or thiopental is given. Cardiovascular support can be required in severe cases.

TYPES OF LOCAL ANESTHESIA

According to the level of neural block induced by administration of anesthetic drug local anesthesia used during surgical procedures is classified into (Figure 11):

(1) Topical anesthesia;
(2) Tumescent (infiltration) anesthesia;
(3) Regional anesthesia:
    - Peripheral neural block (large nerve or plexus);
    - Central neural block (epidural or spinal).

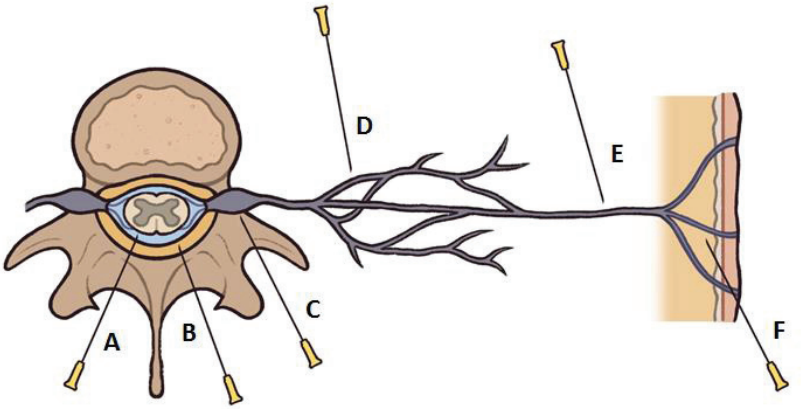

Figure 11. Site of anesthetic's injection for various types of local anesthesia: A – Subdural space (spinal anesthesia); B – Epidural space (epidural anesthesia); C – Nerve ganglia (paravertebral blockade); D – Nerve plexuses (blockade of the brachial plexus); E – Nerve trunks (truncular anesthesia); F – Soft tissues (tumescent / infiltration anesthesia).

TOPICAL ANESTHESIA

Application of topical anesthetics to the skin or mucosa to control pain associated with **minor invasive procedures** such as urethral catheterization, vein puncture in children, nasogastric tube insertion, endoscopic examination or laceration repair may avoid the need for drug injections. Various forms of topical anesthetics exist: gels, sprays, creams, ointments, patches. Usually anesthetics are used in relatively high concentration (2-4% for lidocaine). Due to time required for transdermal drug absorption the onset of anesthesia can be delayed for 30-60 minutes, especially in case of cream and ointment application. The most common topical anesthetics are: lidocaine spray, "LET" (mixture of lidocaine, tetracaine and epinephrine) and "EMLA" (Eutectic Mixture of Local Anesthetics – lidocaine and prilocaine). In sport medicine spray e*thyl chloride – the volatile substance that produces the skin cooling during vaporization, is used for temporary relief of muscle pain.*

TUMESCENT ANESTHESIA

Tumescent anesthesia is administered by delivering a large volume of dilute anesthetic solution to soft tissues (mainly subcutaneous adipose tissue) prior to incision until the tissue is firm and swollen or truly "tumescent". Although the method of tumescent anesthesia was presented and popularized by American Dr. Jeffrey A. Klein for liposuction in 1994, probably this is one of the oldest types of local anesthesia. The very similar method of anesthesia was invented by Russian surgeon Aleksandr Vishnevsky who published it in his book "Local Anesthesia by Creeping Infiltrate Method" in 1932.

The main principles of the **Vishnevsky local anesthesia technique** are as follows:

- The use of direct contact of anesthetic solution with terminal nerves that can be achieved by “tight infiltration” into fascial spaces and sheaths and by creation of “creeping infiltrates” under pressure, layer by layer;
- Hydraulic preparation of tissue;
- The use of large volume (up to 1800 ml) of weak 0.25-0.5% novocaine (procaine) solution mixed with epinephrine for vasoconstriction and prolongation of anesthesia;
- Interchangeable use of syringe and scalpel.

Actually, tumescent anesthesia is **mostly used** for interventions on soft tissues – treatment of small wounds, skin lesions, hernias, varicose veins or in plastic surgery. The typical anesthetic solution is 0.05% to 0.1% lidocaine mixed with 0.1% epinephrine (1 ml per 1 liter of solution) and sodium bicarbonate. The delivery of epinephrine to the tissue results in a profound vasoconstriction of capillaries, which delays the rate of absorption of lidocaine and substantially decreases the potential for blood loss during the procedure. Due to the slow absorption of lidocaine (up to 24-36 hours), dosages as high as 35 mg/kg to 55 mg/kg have been administered safely for tumescent anesthesia. The addition of sodium bicarbonate is aimed to minimize skin irritation and burning associated with the acidic pH of lidocaine solution.

The main **advantages** of tumescent anesthesia are: relative simplicity, immediate onset of anesthesia, reduced postoperative pain and decreased rate of surgical site infection (due to bacteriostatic and bacteriocidal properties of lidocaine).

REGIONAL ANESTHESIA

Peripheral neural block. Local anesthetic can be injected near a large nerve or plexus to provide anesthesia to a larger region of the body. The examples are: the brachial plexus for

surgery of the arm or hand, blockade of the femoral and sciatic nerves for surgery of the lower extremity, blockade of the cervical plexus for carotid endarterectomy, digit block for surgery of the fingers or toes (Oberst-Lukashevich technique) or intercostal block for analgesia of the rib fracture. Usually are relatively small amount of 1-2% solution of anesthetics is used and injected around the nerves. Precise injection requires a skilled anesthesiologist and, optimally, ultrasound guidance. **Delayed onset of anesthesia** (10-20 minutes) is characteristic of peripheral nerve block. Risks of peripheral regional nerve blocks depend on their location and include: nerve damage, puncture of big arteries or veins and pneumothorax.

Central neural block. Local anesthetic injected near the spinal cord, the so called spinal or epidural anesthesia, provides anesthesia for the lower half of the body. This type of anesthesia can be used for genitourinary or gynecologic surgery, inguinal hernia repair or lower limb procedures. Spinal and epidural anesthesia block the spinal nerves as they exit the spinal cord, which contains motor, sensory, and sympathetic components. Central block will cause sensory anesthesia, loss of motor function, and blockade of the sympathetic nerves distally from the level of injection.

In **spinal anesthesia** local anesthetic is injected directly into the dural sac surrounding the spinal cord. The level of injection is below L1-L2 (usually L3-L4), where the spinal cord ends in most adults. Puncture is performed with fine needle (25-27G) when the patient is in the sitting or lateral position with anterior flexion of the spine and bent knees to enlarge the intervertebral spaces. As the local anesthetic is injected directly into the cerebrospinal fluid surrounding the spinal cord, only a small dose is needed (approximately 2 ml) and the onset of anesthesia is rapid. The duration of spinal anesthesia

varies from 60 to 200 minutes depending on a drug, addition of epinephrine or major analgesics (fentanyl, morphine).

Complications of spinal anesthesia include hypotension (especially in hypovolemic patient), headache caused by leakage of cerebrospinal fluid from the puncture site in *dura mater*, backache, urinary retention, infection, epidural hematoma, and cephalad spread of anesthetic resulting in cardiorespiratory compromise. Injury of the nerves emanating distally to the spinal cord is very rare and results in the *cauda equina* syndrome manifested by pelvic organ dysfunction and lower limbs sensory and motor loss.

Absolute **contraindications** to spinal anesthesia are: generalized infection, infection at the site of puncture, uncorrected hypotension or severe hypovolemia, coagulopathy, high-dose anticoagulation, diseases associated with increased intracranial pressure, severe spine deformities and patient refusal.

Epidural anesthesia is applied in abdominal, thoracic, and lower extremity procedures. Much greater volumes of anesthetic are required compared to spinal anesthesia and the onset of the block appears only in 10-15 minutes. Local anesthetics, with or without opiates, are injected into the lumbar or thoracic epidural space via a long catheter inserted through the large (17-18G) needle using Seldinger technique. The presence of the catheter provides several advantages:

- Better control of anesthesia;
- Introduction of repeated doses provides anesthesia for lengthy procedures;
- Catheter can be used for postoperative analgesia.

Complications and contraindications associated with epidural anesthesia are similar to those of spinal anesthesia. However, due to a large needle, the headache caused by accidental puncture of *dura mater* is more severe and usually

requires treatment by “blood patch” method. The method consists in repeated puncture of epidural space in the same place where the needle was initially inserted and introduction of a small amount of the patient’s blood aimed to “seal” the leakage point in the *dura mater*. Placement and removal of epidural catheter in patients receiving anticoagulation is associated with a high risk of **epidural hematoma** – rare but catastrophic complication manifested by back pain, lower extremity sensory and motor deficit, and pelvic organs dysfunction. To reduce the risk of epidural hematoma, insertion and removal of catheter is performed in several hours after the last injection of heparin, and subsequent dosing is delayed for at least 2 hours.

CURATIVE BLOCKADES

In several pathological conditions intratissular injections of local anesthetics can be performed for the treatment, attempting to block the sensorial and autonomic nerves in site of trauma or inflammation. Most commonly 0.25-1% solutions of procaine are injected directly in the hematoma or surrounding facial compartments in case of **bone fractures** (tubular bones, ribs, pelvic ring). Anesthetic solution can reduce the severity of inflammatory process being administrated **retromammary** in case of mastitis or in **round ligament** in case of pancreatitis. Infiltration of intestinal **mesentery** with an anesthetic is a standard technique of “resuscitation” of ischemic intestinal loop during surgery for strangulated hernia. **Perinephric blockade** is widely used in the past as an adjunct for treatment of abdominal trauma and postoperative intestinal paresis. Nowadays it is replaced by epidural anesthesia.

X. WOUNDS

The doctrine of wounds has a long history. Wounds and injuries have accompanied human life since prehistoric times. The appearance and development of surgery itself is largely associated with the need to treat wounds, especially those received during wartime.

DEFINITION AND COMMON SYMPTOMS

Wound is an open damage of soft tissue (skin, mucosa, or profound tissue), caused by the action of traumatic agent. There are two ways of the influence of traumatic agents – external, which is more common and the internal one. Open fracture of extremity when the fragment of the broken bone perforates the soft tissue can be an example of internal influence.

Clinical manifestations of wound include local and general symptoms.

The **local manifestations** are the following: **pain** (*dolor*), **hemorrhage** (*haemorrhagia*), **wound dehiscence** *(hiatus)* and **functional disturb** *(functio laesa).* Severity of symptoms depends on the quantity of nerve endings in the zone of injury, the kind of traumatic agent, duration of its influence and neuropsychological status of patient, diameter of damaged vessel, site of the injury, hemodynamic status and the status of blood coagulation system, volume of damage of the major vessels and nerves, muscles, joints, bones and internal organs.

General clinical manifestations of wounds are conditioned by the bleeding severity and grade of anemia (weakness, somnolence, paleness, dizziness, tachycardia, and hypotension), injuries of inner organs, and infection (chills and fever).

CLASSIFICATION OF WOUNDS

(1) According to the **origin of trauma**, wounds are divided into: **surgical** wounds (in which pain is relieved by anesthesia, bleeding – by hemostasis and dehiscence – by stitch application), **accidental** wounds and **battle** wounds.

(2) Depending on **the nature of traumatic agent** wounds are divided into:

- Slash (or cut) wounds, caused by a sharp object (such as a knife, fragments of glass and others). They are characterized by a high risk of damage to blood vessels, nerves, and internal organs;
- Stab wounds, caused by a pointed object (for example a bayonet, a drill, and a needle). In diagnostic terms, they present significant difficulties, since the pain is insignificant, wound dehiscence is minimal, while external bleeding is not typical;
- Chopped wounds, caused by any heavy sharp object, which influence with high power perpendicular or sidelong to body surface (axe, saber, shovel). The regular borders and acute angles are similar to slash wound, but unlike to it there is considerable dehiscence and contusion of tissue, bone fractures and internal organs damage;
- Contusion wound – is a result of the action of any blunt object (a stone, stick, wheel, hammer). The borders of this kind of wounds are irregular, pain is significant, but bleeding is not intensive in consequence of fast thrombosis of superficial small vessels;
- Lacerated wound – produced by a blunt object, but directed at an acute angle to the surface of the body (industrial injuries, car crash accidents). Often are associated with detachment of the skin over a large area;

- Bite wound, produced by a dog, cat, men, or snake. These wounds are the most infected due to the highly virulent oral microflora, despite the small area of damage. Thus, there is a danger of development of infection from banal to anaerobe and right up to rabies. Sometime, bite wound may serve as a source of acute poisoning, snake or scorpions sting for example;
- Compound wound, which combines the properties of different wounds;
- Gunshot wound.

Unlike other types **gunshot wound** is characterized by a more severe evolution, a lot of complications and a high mortality rate.

There are the following features of **gunshot wound**:

a) Presence of three zones of tissue alteration. All other types of wounds are characterized by the presence of two zones of damage: the wound channel and traumatic necrosis. On impact stratum of air is forming in front and laterally of the bullet during its motion, forming so called bow shock wave. So called temporary pulsing cavity occurs in the tissue with pressure inside more than 1,000 atm, and pressure on the cavity walls is 120 kg/cm^2. When pressure arise, abrupt displacing of tissues, damage of vessels with subsequent thrombosis and ischemia are occurred. That is why, three zones of alteration are distinguished in gunshot wound:

- The first zone – is a wound channel;
- The second zone – the zone of primary traumatic necrosis;
- The third zone – the zone of molecular concussion.

b) Complicated anatomical character of damage and therefore, severity of injuries. The bullet can be deviated having collided with any durable obstacle. Different

contractility of damaged tissue also leads to deviation of wound channel. Therefore, damage to several body cavities (for example, abdominal and chest cavities) is often observed.

c) High degree of contamination. This occur due not only to high damaging potential, but also to significant contamination of the wound with oil and soot from weapons, small dirty objects, fragments of closure and ground.

(3) Classification according to the course of the wound channel (commonly applicable to gunshot wounds):

- Perforating (through) wound – has an incoming and outcoming holes;
- Blind wound – has only an incoming hole;
- Tangent wound – damage of superficial tissues only, and wound channel does not penetrate inside the body.

(4) According to the relation of wound channel to body cavities:

- Penetrating (in body cavities) wounds – with or without internal organs injuries; and
- Nonpenetrating wounds.

(5) According to the degree of contamination wounds are divided into:

- Aseptic wound – only surgical wound, produced in sterile conditions of operating room.
- Contaminated wound – each accidental wound is contaminated by microorganisms.
- Purulent wound – is also a contaminated wound, however in the purulent wound infection process has already developed. Commonly the purulent process develops in wounds in case of bacterial' concentration over 10^5 (100,000) microorganisms on 1 gram of tissue. This is the so-called "critical" level of bacterial contamination.

WOUND HEALING

Any wound (surgical, accidental) is followed by the so-called wound healing process. **Wound healing process** is a complex of consecutive changes in the wound and associated local and general reactions of the human organism.

There are **three phases** of wound process according to morphological modifications (Figure 12):

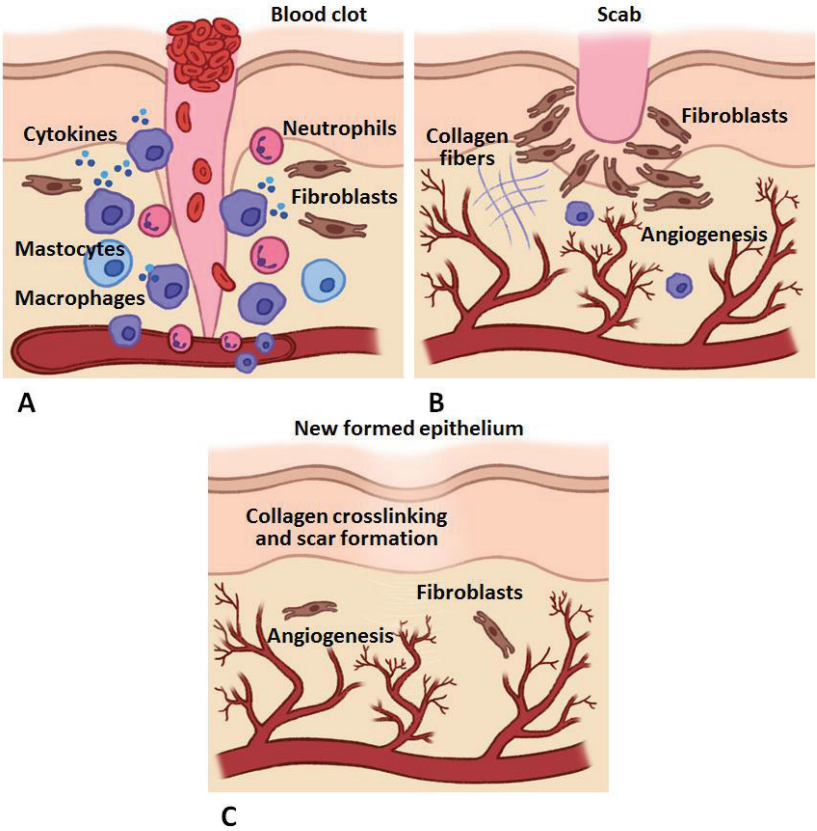

Figure 12. Morphologic characteristics of three phases of wound healing process: A – Phase of inflammation; B – phase of proliferation; C – Phase of epithelization and reorganization of scar.

(1) **Phase of inflammation** (duration of 1-5 days);

The first phase (inflammation) is divided into **two periods:** period of angiogenesis and period of wound cleaning from necrotic masses.

- **Period of angiogenesis.** Any wound generates the damage of tissue architectonics that results in bleeding. Vasoconstriction is an initial reaction of vessels to damage, which subsequently is replaced by paralytic vasodilatation and increased permeability of vascular wall. Increased permeability of vascular wall leads to migration of fluid and blood cells into the extracellular space. Within a few hours the wound is filled with polymorphonuclear neutrophils and lymphocytes.

- **Period of wound cleaning from necrotic masses.** Polymorphonuclear neutrophils phagocytize and destroy bacteria, damaged previously by granulocytes. The functions of macrophages are also the release of proteolytic enzymes and phagocytosis of necrotic tissues. Different types of lymphocytes take part in immune response against foreign materials, viruses and bacteria from the wound.

(2) Phase of proliferation (6-14 days). In this phase the principal role belongs to endothelial cells (proliferation of new blood vessels) and fibroblasts (responsible for collagen synthesis). All of these lead to intensive formation of **granulations** in the wound, i.e. fine conjunctive tissue with newly formed capillaries. As a result of granulation the tissue rapidly fills the bottom and wall of the wound, and the wound cavity is reduced.

(3) Phase of epithelization and reorganization of scar (since the 15-th day). The granulation tissue becomes more rigid while number of vessels, macrophages and fibroblasts decreases significantly. Collagen fibers are crosslinking and get

the fibrous structure. The newly formed fibrous tissue lines the bottom and walls of the wound, sealing and pulling its borders (wound contraction). Thus, the fine connective tissue is transformed into a dense scar, and epithelialization of the wound begins from the border to center.

TYPES OF WOUND HEALING

The character and duration of wound healing depends on the dimensions of space that should be filled with conjunctive tissue. There are **three types** of wound healing:

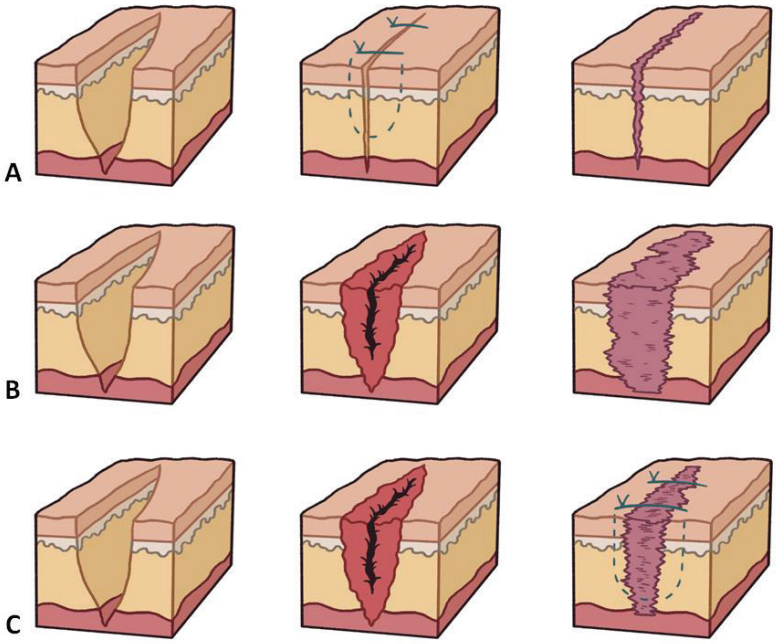

Figure 13. Types of wound healing: A – Healing by primary intention; B – Healing by secondary intention; C – Healing by tertiary intention.

- **Wound healing by primary intention** occurs in the wound with tightly adjusting of edges and absence of infection, within 6-8 days to form a thin relatively strong scar (Figure 13A). Surgical wounds are healed by primary intention.

- **Wound healing by secondary intention.** Wound healing after suppurative processes and filling the wound cavity with granulation tissue (Figure 13B). Wounds with a large skin defect, with the presence of foreign bodies, hematoma, or necrotic tissue are closed by secondary healing (intention). Secondary healing process can be long and last for several weeks. If granulating wound is sutured, this type of healing is sometimes referred to as tertiary intention (Figure 13C).

- **Wound healing under scab** is a special kind of healing, characteristic only of superficial wounds. The process begins with the clotting of blood, lymph and interstitial fluid in the damaged surface, and formation of scab (crust). The scab has a protective function and it should not be removed if there are no signs of inflammation. Epithelialization occurs under the scab.

COMPLICATIONS OF WOUNDS

The following complications can occur **during the first phase:** traumatic shock, bleeding, hemothorax, hemoperitoneum, different hematomas including pulsatile hematoma in case of major artery injury.

The following complications can occur **during the second phase:** development of banal purulent inflammation with abscess or phlegmon formation or anaerobic clostridial and non-clostridial infection, rabies (hydrophobia), and tetanus. Besides the development of erosive secondary hemorrhage, lung complications (pneumonia), wound cachexia and

suppuration of wound is possible.

The following complications can occur **during the third phase:** the dehiscence of wound (sometimes, with evisceration of intestinal loops, and considered as a severe complication of the healing process), local formation of ulcer or fistula, development of systemic complications – gastritis, peptic ulcer, hepatitis, reactive mental disorders.

WOUNDS TREATMENT

First aid in wounds

There are two basic rules of the first aid for wounds:

- Removal of early life threatening complications of wounds;
- Prevention of wound contamination.

Removal of early life threatening complications of wounds. The most dangerous complications are: (1) Bleeding; (2) Traumatic shock; and (3) Visceral injury. Basic information about the management of these early complications is provided in the relevant sections of this textbook.

Prevention of secondary contamination of the wound. Regardless of the type and location of injury, all accidental wounds are considered to be primarily infected. In addition, secondary penetration of bacterial agents into the wound from environment (various objects, surfaces, patient's skin) is possible. It is necessary to clean the surrounding skin of patient with any antiseptic, wash the wound and cover it with a sterile or clean dressing.

Further treatment depends on the degree of infection and according to this wounds are divided into three groups: (1) Surgical wounds (aseptic, sterile); (2) Contaminated wounds; (3) Purulent (septic) wounds.

Treatment of aseptic wounds, i.e. produced under sterile

operating room conditions, consists of surgical hemostasis and restoration of tissue integrity by sutures application. In the postoperative period the treatment includes:

(1) Analgesics (depend on the degree of traumatic influence of surgery, and may be provided by different methods and routs of administration);

(2) Prophylaxis of secondary infection (covering the wound with sterile bandage, its timely replacement and use of antiseptics);

(3) Acceleration of wound healing (by early mobilization of patients);

(4) Improvement of patient's general health status (correction of anemia, low serum protein level, blood circulation insufficiency, concomitant pathology, and others).

Treatment of contaminated wounds. The basic method of treatment of recently infected wounds is **primary surgical processing (debridement) of wound**. It includes the following steps (Figure 14):

- Dissection of tissue – is necessary for complete revision of wound channel and its extension;
- Exploration of wound channel – in order to establish a penetrating or non-penetrating in body cavities character of wound;
- Excision of wound borders and bottom – is performed to eliminate necrotic tissues, foreign objects and all wound surfaces, which are infected;
- Hemostasis – carried out carefully, to prevent the appearance of hematomas and other postoperative complications;
- Reconstruction of damaged tissues and structures – includes suture of nerves, vessels, tendons, reposition of bone fragments.

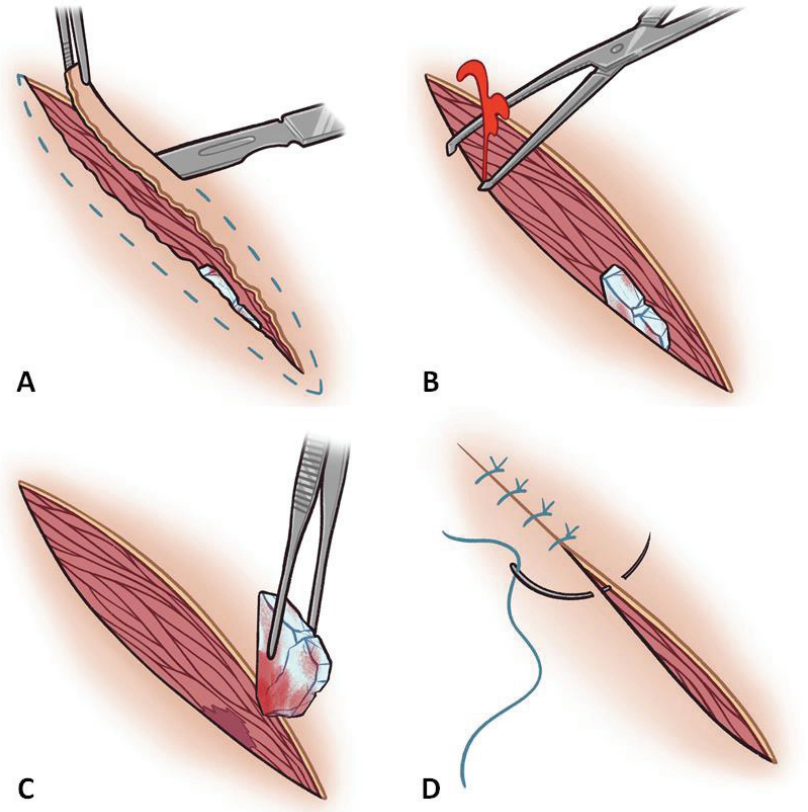

Figure 14. Basic steps of primary surgical debridement of contaminated wounds: A – Excision of margins and bottom of the wound in aseptic conditions; B – Careful hemostasis; C – Removing of foreign bodies and devitalized tissues; D – Application of primary or primary delayed sutures.

It should be emphasized that primary surgical debridement is not indicated for superficial cut wounds with smooth edges, without injuries of the neurovascular structures.

The primary surgical processing ends with **suture and drainage** of wound**.** There are several ways to end this

operation:

- Application of sutures with no drainage;
- Application of sutures with drainage;
- No suture – in case of high risk of infection development (with late primary surgical debridement; severe contamination of wound with soil; massive tissue damage; presence of concomitant diseases such as diabetes mellitus, anemia, immunodeficiency; localization of wound on the foot; gunshot wounds).

Kinds of sutures in wound closure. Wound healing by secondary intention has multiple disadvantages: large losses of fluid, proteins and electrolytes through the wound, a long period of healing and patients' disability, appearance of deforming scar, and an increased costs of treatment. Therefore, it is necessary to close the edges of the wound as early as possible by applying sutures. Sutures applied for wound closure are divided into primary and secondary types.

Primary sutures are divided into:

- Primary sutures are applied after the early primary surgical processing and commonly end with a wound healing by primary intention; and
- Primary delayed sutures are used when there is a significant risk of infection in the wound. This kind of suture is applied on the 5-6-th day after trauma, when inflammation is controlled. The primary operation can be finished by application of sutures but they are not tied.

Secondary sutures – suturing a granulating wound:

- Early secondary sutures are applied after the development of granulation, but before the formation of scar tissue on the 2-nd week of the disease; and
- Late secondary sutures are applied on the wound with scar tissue and developed phenomenon of contraction during

the 3-4-th week of disease. Contraction of wound borders is already present in 21 days, therefore it is necessary to excise wound borders prior to suturing. otherwise it is impossible to close the wound.

Treatment of purulent wounds. An advanced inflammatory process is already developed in the purulent wound, and the purpose of surgical intervention is to treat and not prevent wound infection. The basic component of purulent wounds' treatment is the **secondary surgical debridement (processing).** The latter is an independent surgical intervention, performing in the operating room, under adequate anesthesia by opening and drainage of purulent cavities and excision of necrotized tissues. It is important that after secondary surgical debridement, primary sutures are not applied to the wound, and the wound cavity is filled with gauze meshes imbibed with antiseptic solutions.

Additional physical methods of purulent wounds cleaning not replace, but only complement secondary surgical debridement and increase its positive effect. They include: (1) Pulsatile jet with antiseptics; (2) Ultrasound cavitation; (3) Surgical laser; (4) Treatment in controlled abacterial environment; (5) Vacuum therapy.

Local treatment of purulent wounds.

During the **first phase of wound healing** gauze meshes and towels moistened with liquid antiseptics: 3-5% solution of boric acid, 0.02% solution of chlorhexidine, 10% solution of NaCl (hypertonic saline solution) are used. Hydrophilic water-soluble ointments can be used from the 2^{nd}-3^{rd} days (Levosin, Levomikol, Mafenid-Acetat). Proteolytic enzymes (trypsin, hemotrypsin, hymopsinum) are also used for chemical processing of purulent wounds.

In the **second phase of wound healing process**, ointments, which contain antibiotics and stimulating substances (Solcoseril, Actoveghin, Tetracycline, Gentamycin, Vishnevsky unguents, synthomycin emulsion) should be applied with the purpose to protect the granulating tissue and to prevent the development of infection.

General treatment of patients with purulent wounds consists of:

- Antibacterial therapy;
- Detoxication;
- Immunomodulation;
- Parenteral and enteral nutrition;
- Symptomatic treatment.

Skin grafting. It is important that after secondary debridement primary sutures are not applied to the wound. The wound is closed with secondary sutures, most often leaving drainages in it. With extensive skin defects, long-term non-healing wounds and ulcers in the 2nd phase of the wound process, i.e. after cleansing the wounds from pus and the appearance of granulations, it is possible to close the defect using the so-called skin grafting:

- Full layer displaced flap;
- Walking stem (flap) according to Filatov;
- Full-thickness flap on a vascular pedicle using vascular anastomoses;
- Autodermoplasty with a free perforated flap (skin grafting).

XI. SURGICAL INFECTION

Infection – is the result of penetration and reproduction of microorganism in human body, which is manifested by the development of infectious disease. **The term "surgical infection"** combines two concepts: (1) Infectious process, which should be treated surgically; and (2) Infectious complications developing in the postoperative period.

CLASSIFICATION

There are some principles for the **classification of surgical infection**: by etiology, by clinical evolution, and by site of infection.

According to **etiological principle** surgical infections are divided into:

- Aerobic infection: Gram-positive: *Staphylococcus aureus, Streptococcus, Enterococcus, Pneumococcus,* and gonococci; and Gram-negative: *Escherichia coli, Proteus vulgaris, Proteus mirabilis, Pseudomonas aeruginosa, Klebsiella pneumoniae;*
- Anaerobic clostridial infection: *Clostridium perfringes, Clostridium edematiens, Clostridium histoliticum, Clostridium septicum, Clostridium tetani.*
- Anaerobic non-clostridial infection: Gram-positive: *Bacteroides fragilis, Peptococcus, Peptostreptococcus*; and Gram-negative: *Fusobacterium, Enterobacter*.
- Also distinguish mixed and fungal infection.

According to **clinical evolution** surgical infections are divided into:

(1) Acute surgical infection:
   - Acute purulent infection;
   - Acute putrid infection;
   - Acute anaerobic infection;

- Acute specific infection (tetanus, anthrax).

(2) Chronic surgical infection:
- Chronic non-specific infection;
- Chronic specific infection (tuberculosis, syphilis, actinomycosis).

According to purulent **process location** surgical infections are divided into**:**

- Soft tissue infections (skin, subcutaneous tissue, fascia, muscles);
- Bones and joint infections;
- Brain and its covers infections;
- Thoracic organs infections (lung, pleura, mediastinum);
- Abdominal organs infections;
- Infections of other organs and tissues (hand, breast).

PATHOGENESIS

Purulent surgical infection – this is an acute pathological process with different localization and character, caused by pyogenic (pus-forming) microorganisms. The development of surgical infection is produced by three elements: (1) Infectious agent (pathogenic microorganism); (2) Site of infection penetration; and (3) Response reaction of human body.

Infectious agent (pathogenic microorganism). The role of different microorganisms in the development of infection varies with time, depending on the constantly increased resistance of individual strains to the antibacterial treatment. In evolution of purulent surgical infections great values have biological characteristics of microorganisms: invasiveness, toxicity, virulence, as well as degree of contamination.

Site of infection penetration. Microorganisms are widely distributed in external environment, and also in a large number on the surface of human skin, gastrointestinal and

respiratory tract mucosa. Breakdown of their integrity represents a site, through which infectious agents may penetrate inside the tissues. There are exogenous and endogenous routes of infection penetration.

Response reaction of human body. Penetration of infecting agent doesn't result in development of purulent process obligatorily. Significant roles have response reactions of the human body, in which non-specific and specific defense mechanisms are distinguished:

(1) **Nonspecific** protective mechanisms:

- Protective and bactericidal properties of the skin and mucous membranes;
- Saprophytic microflora of the human body, which exhibit antagonistic activity against exogenous microorganisms;
- Humoral factors contained in plasma (leikin, β-lysine, lysozyme, complement system);
- Cellular mechanisms of nonspecific defense (inflammation, phagocytosis). Inflammation is the leading reaction of human body in purulent infection. It aimed to localize the purulent process and to prevent spreading of bacteria. Phagocytes – are polymorphonuclear leukocytes and mononuclear phagocytes (monocytes, hystiocytes, macrophages of lung, spleen, liver, etc.) They absorb and destroy microbial bodies, their fragments, and also release cytokines;

(2) **Specific** mechanisms of protection include humoral and cellular immune response**.** Humoral response consists of recognition of foreign antigen and producing of antibodies by B-lymphocytes. Cellular immune response is realized by T-lymphocytes. Some of them have a direct action on antigen (killer cells), others affect indirectly, producing immune response mediators (lymphokines)

A number of factors can **diminish the activity of protective mechanisms:** the age of patients (children and elderly persons); concomitant diseases accompanied by immunodeficiency (diabetes, renal or hepatic insufficiency, malignancy, AIDS); anemia and hypoproteinemia; use of certain medications (immunosuppressant, cytostatic, antibiotics) and radiation therapy.

SEMIOLOGY AND DIAGNOSIS

Clinical manifestation of acute purulent surgical diseases consists of general and local signs and symptoms.

In acute purulent infection **local reaction** is manifested by classical signs of inflammation, described by Cornelius Celsus 2 thousand years ago:

- *Rubor* (hyperemia, redness) is caused by vasodilatation in the area of inflammation;
- *Calor* (local hyperthermia) is caused by enhancement of local catabolic reactions with energy release;
- *Tumor* (swelling, edema) is caused by increasing of the vascular wall permeability, and extravasation of fluids;
- *Dolor* (pain) is explained by influence of the vasoactive substances and interleukins, as well as tissue edema and compression of the nerve endings;
- *Functia laesa* (functional disturb) is caused by both pain and edema.

Specific clinical symptoms are used to **diagnose** the accumulation of pus inside an inflammatory infiltrate: softening, fluctuation, and diagnostic needle puncture. Softening, which is determined by palpation in the middle of a dense inflammatory infiltrate suggests the accumulation of pus. The fluctuation is defined as follows: with his one hand doctor pushes on one side of the inflammatory infiltrate and

with his other hand feels the wave of liquid (pus) on opposite site.

Imaging diagnostic methods, used to reveal the profound purulent focus are the following: X-ray examination; ultrasound scan; computed tomography, and others.

General manifestations of acute surgical infection are similar to general symptoms of inflammation: subjective (fever, chills, headache, weakness, dizziness, loss of appetite), and objective (high body temperature to 39-40°C, tachycardia, dyspnea, cold sweats, enlargement of the spleen and liver, and sometimes – jaundice of sclera). It should be noted, all of these symptoms are reversible and disappear after successful surgical treatment of purulent process.

Laboratory data: leukocytosis, leukocyte formula left shift, appearance of young forms of white blood cells (myelocytes), lymphopenia, monocytopenia, increased erythrocyte sedimentation rate, toxic anemia. Biochemistry may show increased serum creatinine and urea levels.

GENERAL PRINCIPLES OF TREATMENT

The management of acute surgical infection is difficult, and includes general and local measures. The basic principles of **local treatment** are the following:

(1) Surgical debridement (evacuation) of purulent collection. The technique of surgical debridement of purulent accumulation is the following. It is preferably to perform intervention under short-term general or regional anesthesia. The surgical treatment involves abscess opening, removal of pus, inspection of residual cavity (visual or with finger), dividing adhesions, and excision of necrotic tissues. If surgery is performed radical, healing process is faster and the complications are fewer.

(2) Local processing with antiseptics. Irrigation of purulent cavities with antiseptics: 3% hydrogen peroxide, 3.2% solution of boric acid, an aqueous solution of chlorhexidine, iodine povidone, and others.

(3) Adequate drainage of residual cavity. All possible means of physical antiseptics are used for this purpose: passive drainage (gauze meshes and pads, rubber strips, drainage tubes), active, and flow-irrigation drainage, vacuum therapy, and others.

(4) Immobilization. In the acute period of purulent process, it is necessary to keep the affected segment immobilized, particularly in case of its location on extremity.

Methods of **general treatment** of surgical infection include: antibacterial therapy and detoxification, immunomodulation, and symptomatic treatment.

COMMON PURULENT PROCESSES OF SOFT TISSUES

A furuncle (boil) is a *Staphylococcus aureus* infection of an obstructed hair follicle (Figure 15A). Three stages are distinguished in the evolution of furuncle: (1) Infiltration, (2) Abscess formation and rejection of necrotic core, and (3) Scarring. Constitutional symptoms and fever are not common. Antibiotic therapy is not usually necessary. **Cavernous sinus thrombosis** is a rare but very serious (and often fatal) complication of a furuncle on the lateral side of the nose or infraorbital area. Therefore, these patients need in-hospital treatment and, besides of local measures, they should receive antibiotics and anticoagulants.

A carbuncle is a large abscess (almost always staphylococcal) extending from several infected hair follicles into the subcutaneous fat (Figure 15B). It is often seen at the nape of the neck, or on the back. Carbuncles are more

common in diabetic patients. There are two phases of evolution: (1) Infiltration and (2) Abscess formation. If pus has been formed, surgical cruciate incision, thorough excision of necrotic tissues and drainage of the abscesses is required, usually leaving wound open to heal by secondary intention.

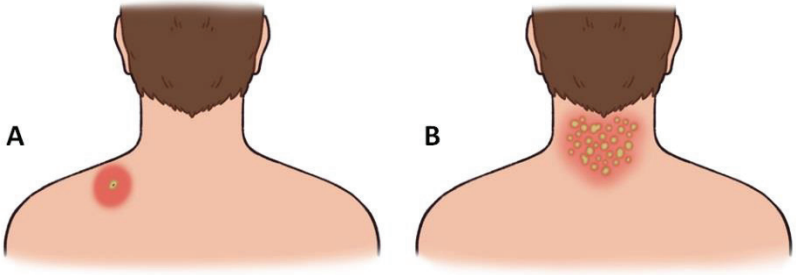

Figure 15. Acute purulent processes of soft tissues: A – Furuncle; B – Carbuncle.

Hidradenitis suppurativa. This indolent, chronic, suppurative infection of apocrine sweat glands is fortunately rare. It is often due to *Staphylococcus aureus*, and occurs mainly in adults. It results in recurrent crops of abscesses, leading to sinus formation and extensive scarring in the axillary, perineal and/or genital areas. When abscess is formed, softening, fluctuation and spontaneous eruption of pus occurs. Oral anti-staphylococcal therapy combined with moist compresses and surgical drainage of fluctuant lesions is usually effective.

Abscess is a limited collection of pus in different tissues or body cavities (Figure 16A). It may appear due to microbial penetration into the tissue through abrasion, injections, wounds, or as a complication of various inflammatory processes (appendicitis, peritonitis, pneumonia). A specific feature of condition is the presence of pyogenic capsule, which limits a further spread of pus. Commonly, the severity

of local and general symptoms greatly depends on the location of the abscess. The diagnosis of abscess is an absolute indication for surgical treatment.

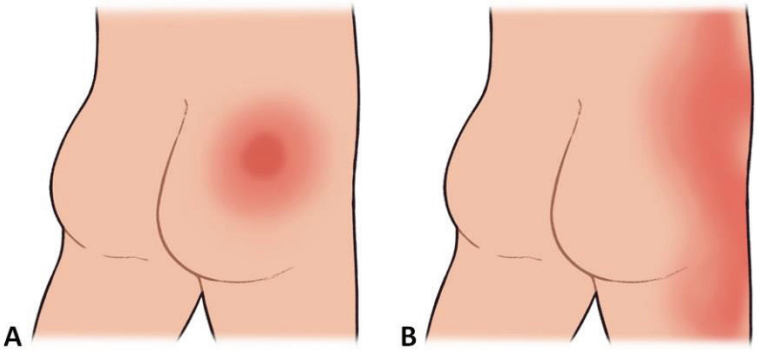

Figure 16. Acute purulent processes of soft tissues: A – Abscess; B – Phlegmon.

Phlegmon is an acute diffuse, not limited suppurative inflammation of cellular spaces: subcutaneous, intermuscular, retroperitoneal (Figure 16B). Phlegmon can be a separate primary disease, as well as a complication of various suppurative processes (furuncles, carbuncles, abscesses). Clinical presentation is characterized by rapid appearance and spreading of a painful swelling, redness of skin, pain, motion disturbances in the affected part of the body, fever and symptoms of intoxication. The urgent surgical treatment is mandatory.

Mastitis is an inflammation of the breast parenchyma. In the vast majority of cases develops in postpartum period (lactational or puerperal mastitis). Lactational infections are thought to arise from entry of bacteria through the nipple into the duct system. Mastitis is classified according to the phase of inflammation (serous-infiltrative, abscess form, gangrenous), and localization of suppurative focus

(subcutaneous, intramammary, retromammary, subareolar). The treatment is complex – surgical drainage of abscess, antibiotic therapy, frequent emptying of the breast and suppression of lactation, and physiotherapy.

Acute paraproctitis (anorectal suppuration) – is a purulent inflammation of the perirectal adipose tissue. Acute paraproctitis is classified according to the anatomical location of purulent focus into: submucosal, subcutaneous, ischiorectal, pelviorectal, and retrorectal. Local and general symptoms appear simultaneously. The disease starts with weakness, headache, chills, and high fever. At the same time pain in the rectum or pelvis appears and gradually increases. It is aggravated by defecation or in sitting position.

Erysipelas is a primary skin infection, almost always caused by *Streptococcus pyogenes***.** Erysipelas is classified into erythematous, bullous, phlegmonous, and necrotic forms (Figure 17). The disease begins with symptoms of severe intoxication, chills, fever up to 39-41˚C and leukocytosis. Skin lesions are bright red, painful, associated with increased local temperature. The spreading inflamed area is very well outlined, with the margin above the normal skin. Therefore, local changes in erysipelas are compared with “flame” or “geographical map”.

Erysipeloid is an occupational disease of persons employed in meat or fish production, as the causative agent (a gram-positive rod – *Erysipelothrix rhusiopathiae*) is found in many wild and domestic animals. The infection develops as an indolent, purple, swollen, nonpurulent area at the site of inoculation, which spreads slowly outwards. Penicillin is the antibiotic of choice.

Lymphangitis is particularly likely to complicate group A streptococcal infection of the skin of a limb. The clinical signs

are red lines of inflammation corresponding to the lymphatic channels, extending to the regional lymph nodes. The lymph nodes themselves are tender and swollen and edema of the limb may develop. Fever is common. Untreated lymphangitis is likely to lead to bacteremia.

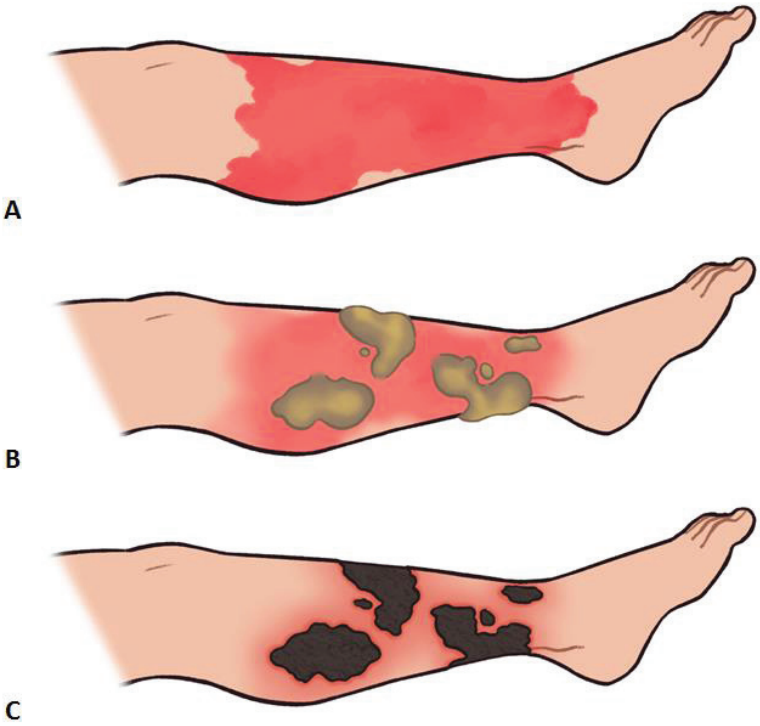

Figure 17. Forms of erysipelas: A – Erythematous; B – Bullous; C – Necrotic.

Pilonidal cysts and abscesses are common in young hirsute men, and are found at the upper end of the intergluteal cleft. Pilonidal cysts have a long indolent course with chronic or intermittent purulent discharge to the skin surface via one or more orifices. Periodic acute exacerbations may progress to

abscesses. Pilonidal abscess causes acute pain, swelling and redness of skin. Pilonidal abscesses are often multilocular. The standard operation is to excise an elliptical wedge of tissue, incorporating the mass of cysts and overlying skin.

An ingrowing toenail occurs when the distal edge of the nail persistently cuts into the adjacent nail fold. The problem almost always affects the great toe. In effect there is a laceration which cannot heal because of the presence of a foreign body (the toenail). The infection by a mixture of local bacterial and fungal flora complicates the clinical picture. Swelling aggravates trauma caused by the nail edge. Urgent surgical treatment involves avulsion of the whole nail, or one side of the nail. This immediately removes the “foreign body” and permits rapid resolution.

Bedsore is a necrosis of soft tissues as a result of constant pressure, accompanied by local circulatory and nervous disorders. Bedsores develop in patients with limited mobility, in areas of bone prominence. Classification: stage I – Intact skin with erythema and reactive hyperemia; stage II – Partial damage of skin, involving the epidermis and derma; stage III – Impairment of the skin of full thickness with extension in the subcutaneous tissue, but not through the muscular fascia; stage IV – Complete tissue necrosis, extending into muscle, bone, tendon or joint capsule. Treatment of pressure ulcers includes non-operative and surgical methods. Principles of conservative treatment: pressure reduction; control of infection; wound care and regular dressings; negative pressure therapy; use of growth factors.

Fistula is an abnormal connection between body cavities or hollow organs to each other or to the external environment. Sometimes fistulas are created surgically for therapeutic purposes. In relation to the external environment, are

distinguished external and internal fistulas. In turn, external fistulas are divided depending on the nature of discharge, such as urinary, fecal, biliary, mucous, purulent. Local and general symptoms, as well as the evolution of disease, vary significantly depending on the type of fistula. To clarify the diagnosis, probing, radiography with a contrast agent (fistulography), as well as other imaging methods (ultrasound, computed tomography, magnetic resonance, radiography, endoscopy) are used. Treatment varies depending on the cause and location of the fistula.

XII. FELON AND HAND PHLEGMON

ANATOMICAL FEATURES OF THE HAND

The skin of the palmary surface of the fingers and hand is strong, thick and not elastic, which explains the low probability of spontaneous rupture of purulent focus localized in subcutaneous fat. Adipose tissue of the palmary surface of the hand is divided by the fibrous septa into separate cells, which are attached to the periosteum and aponeurosis. This causes the spread of infection in the depth, rather than in the width of tissues. High pressure in limited fibrotic cells can impair venous outflow and leads to local compartment syndrome with compression of the nerves (pain) and thrombosis of blood vessels (ischemia, necrosis).

Tendon sheaths of the II, III and IV fingers begin from the nail bones and end at the level of the distal metacarpal bones, they are separated from each other and are not connected with the synovial forearm bursae. The flexor tendon sheath of the thumb communicates with the radial synovial bursa, and the flexor tendon sheath of the V finger – with the ulnar bursa. In 80% of individuals, the communication exists between the radial and ulnar bursae. Thus, the inflammatory process involving the tendon sheath of the I and V fingers can spread to the forearm cellular space (Pirogov-Parona's space).

The palmar surface of the hand is separated by palmar aponeurosis into two spaces:

- Superficial space (subcutaneous fat); and
- Deep space.

The transverse septum to the metacarpal of the middle finger divides the deep space into:

- The radial thenar space;
- The ulnar midpalmar space; and

- The small hypothenar space, separated by the medial fibrous septum.

The palmar aponeurosis continues on the fingers and palms to the distal part of the II-V-th metacarpophalangeal joints, and forms three holes (commissures), which connect the superficial and deep spaces.

On the dorsal part of the hand the subcutaneous (superficial) and subaponeurotic (deep) spaces are distinguished. Unlike the palmar surface, skin and subcutaneous tissue of the dorsal side of the hand are thin, not fixed by a deep fascia and swell easily. Therefore, swelling of the dorsal surface of hand is often a sign of infection of the deep palmar space.

FELON

Felon is an acute purulent process, localized in the soft tissues of the palmar surface of fingers, in the area of nail, as well as in the bones and joints of fingers. Suppurations occurring on the dorsal surface of finger (with exception of nail area) do not refer to a felon. Wooden splinters, thorns, or minor cuts are common predisposing causes, yet no history of injury exists in one half of patients. Infection also may spread from a lancet-induced trauma from fingertip blood glucose measurements. The most common causative organism is *Staphylococcus aureus*. Gram-negative and anaerobic organisms have been reported in immunosuppressed patients. It should be noted, that historically the term *felon* is applied only to purulent diseases of hand fingers.

Classification of felon

Inflammatory processes of the finger are divided into superficial and deep forms (Figure 18).

Superficial forms:

- Cutaneous felon (*panaritium cutaneum*);
- Subcutaneous felon (*panaritium subcutaneum*);
- Paronychia (*paronychium)*.

Deep forms:

- Pyogenic flexor tenosynovitis (*panaritium tendinosum*),
- Bone felon (*panaritium ossale*),
- Articular felon (*panaritium articulare*)
- Pandactylitis *(pandactylitis)* also refers to deep forms.

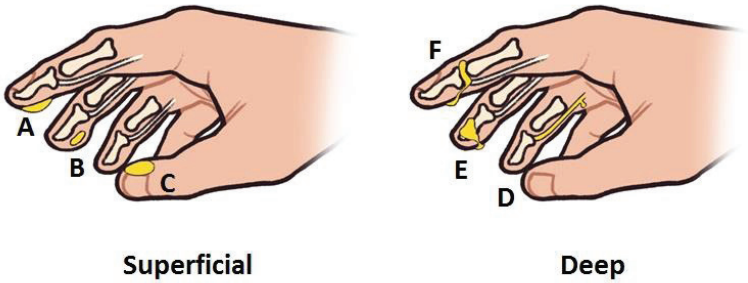

Figure 18. Forms of panaritium. Superficial: A – Cutaneous; B – Subcutaneous; C – Paronychia; Deep: D – Tenosynovitis; E – Bone; F – Articular.

SPECIAL FORMS OF FELON

Cutaneous felon. On the palmar surface of finger, a superficial vesicle with pus, surrounded by a thin strip of hyperemia is formed. Pain is not characteristic. The treatment of cutaneous felon consists in removing the affected epidermis and drainage of pus, followed by application of antiseptic dressings. Antibiotics are usually not required, except the cases complicated by lymphangitis or lymphadenitis.

Subcutaneous felon. Patients complain of acute pulsating pain in the finger (palmar surface of nail phalanx), which

increases gradually. Active and passive movements are limited and painful. The finger is swollen and tense. Hyperemia is not characteristic, but it occurs occasionally on the dorsal surface of finger. Palpation of finger (with probe or the tip of forceps) reveals an area of marked tenderness, corresponding to the site of tissue necrosis and accumulation of pus. Radiographs should be obtained if there is a history of penetration with a radiopaque foreign body. Adequate early conservative treatment of a felon (including warm compresses, elevated position of hand, immobilization of finger, and antibiotics), can prevent abscess formation and other serious complications. The first sleepless night due to acute pain in the finger is an absolute indication for surgery (Voyno-Yasenetsky's rule). With the progression of disease, the underlying bone, joint or flexor tendons may become infected.

Principles of surgery for felon are the following:

(1) Surgery should be performed in operating room, with the use of special instruments;

(2) Surgery must be performed under Oberst-Lukashevich local anesthesia. Note, that local anesthetic containing a vasoconstrictor such as adrenaline must never be used in digits' surgery because of the risk of ischemic necrosis;

(3) Surgery should be performed bloodless (after application of tourniquet at the level of basal phalanx).

(4) Pus or any fluid obtained from a wound should be sent for bacteriological examination.

For drainage of purulent collections with subcutaneous felon, the most preferred is the use of small incisions made on the lateral surfaces of fingers. This avoids damage to the joints, blood vessels and nerve endings, as well as subsequent formation of scars that could limit the mobility of the fingers and disturb tactile sensitivity. Sometimes, two lateral incisions

are made to drain the purulent collection, which allows lavage and through-out drainage of the abscess using a small tube or a rubber strip fashioned from the surgical glove.

Paronychia is an acute purulent inflammation of the lateral nail fold; it is the most common infection of the finger and usually results from a trauma to the eponychial or paronychial region. Initially in the nail fold mild pain, swelling and redness appear. When pressing under the nail fold a drop of pus is released. Although paronychia typically starts as a cellulitis, its progression to abscess formation is not uncommon. Occasionally, infection can spread under the nail plate itself, resulting in subungual abscess. Initial treatment includes warm compresses, elevated position of hand, immobilization of finger, and antibiotics. When abscess develops, surgical drainage is required. Abscess can be drained through Clapp incision (arcuate, parallel to the edge of the nail). Another method of surgical treatment for paronychia is the detachment of nail fold and evacuation of purulent collection. The operation is performed under local anesthesia. Subsequently, for several days the conservative therapy is administrated: ointment dressings, compresses.

Purulent tenosynovitis is an infection process that involves the fluid-filled sheath (called synovium) that surrounds the tendon. The most common cause of tenosynovitis is a puncture wound of the finger. Pyogenic flexor tenosynovitis is the most common in the index, middle, and ring fingers and can be formed as early as 6 hours after the initial penetration of infection. Four classic **signs of Kanavel** indicate the presence of infection (Figure 19):

(1) Tenderness along the course of the flexor tendon,

(2) Exquisite pain at the slightest attempt of passive extension,

(3) Symmetrical fusiform swelling of the entire finger, and
(4) Flexed resting posture of finger.

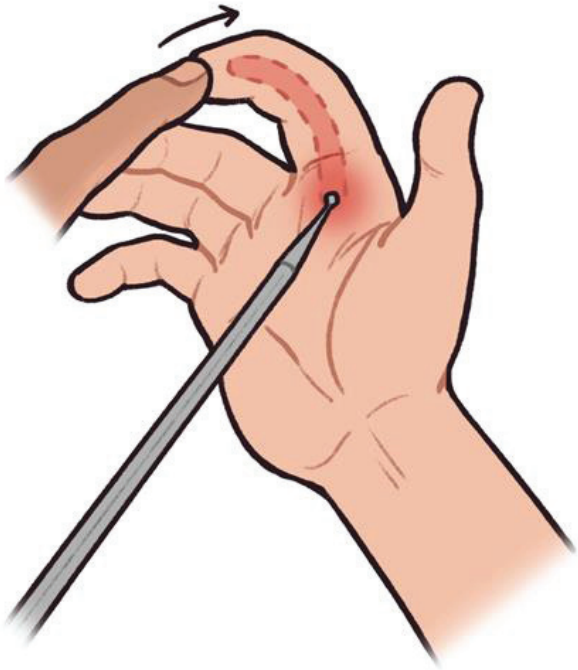

Figure 19. Determination of Kanavel's signs in the case of purulent tenosynovitis.

These symptoms are usually accompanied by high fever and intoxication. The treatment includes surgical drainage, antibiotic therapy, pain relief, and elevation and immobilization of the hand. Surgery for purulent tenosynovitis requires wide opening of synovial sheath and subsequent assessment of tendon viability. In case if the tendon is viable, topic antiseptics and drainage with perforated tube of infected cavity are performed. In purulent tenosynovitis of the

I-st and V-th fingers, it is necessary to open deep cellular spaces, including in forearm. Sometimes the incision may continue from affected finger toward the palm and forearm.

Bone and articular felon. Felons that are untreated, incorrectly treated, or have a prolonged course may lead to osteomyelitis and septic arthritis. Characteristic clinical manifestations are the following: dull pulsating pain, marked edema, increase of pain on percussion along the axis of the finger. Radiographs are informative in 10 days after the onset of disease and can show bone destruction, joint disintegration, presence of sequestration or pathological fracture. The treatment of bone and articular felon is only surgical. A wide excision of all necrotic tissues is performed under general anesthesia, with subsequent prolonged drainage, immobilization and antibiotic therapy. If improvement does not occur, amputation of the phalanx may be the final decision.

Pandactylitis is an inflammation of all tissues of finger, including skin, subcutaneous tissue, tendon, joint, and bone, As a rule, this is an outcome of untreated felon rather than primary disease. Unfortunately, the amputation of finger is practically inevitable.

HAND PHLEGMON

Suppurative processes of the hand are characterized by severe evolution, especially on their localization on the palmar surface. Infectious agents can get into the tissues through direct penetrating injury, or spread from the fingers.

In accordance with localization of the purulent process in certain cellular spaces, phlegmons of hand are **classified** as follows:

Dorsal surface:

- Superficial (subcutaneous) phlegmon;
- Deep (subaponeurotic) phlegmon.

Palmar surface:

- Superficial phlegmon: cutaneous abscess; supraaponeurotic phlegmon; interdigital (commissural) phlegmon;
- Deep phlegmon: phlegmon of thenar; phlegmon of midpalmar space; and phlegmon of hypothenar.

Semiology of hand phlegmon

Superficial forms: moderate edema and hyperemia, more pronounced on the dorsal surface of hand; flexed position of fingers with exquisite pain, associated with their extension; indisposition, sleeplessness and high fever.

In some cases, inflammatory process may spread through interdigital apertures toward the dorsal surface of hand (so-called commissural phlegmon). The characteristic symptoms of the disease are painful swelling in commissural zone, and impossibility to bring fingers together.

Deep forms:

- Usually develop as a complication of tenosynovitis due to the spread of infection through the flexor tendon sheaths;
- Are characterized by acute onset with severe pain and high fever;
- Significant swelling not only of the dorsal, but also of the palmar surface of hand is revealed;
- Flexed position of fingers with restriction of movements are characteristic;
- On attempt of fingers extension or palm palpation patient feels intolerable pain;
- The general condition of patients worsens dramatically.

Surgical treatment of hand phlegmon includes the sequential implementation of the following measures: opening and drainage of purulent collection (abscess), excision or debridement of necrotic tissues, thorough hemostasis, wound lavage with antiseptic solutions, dressing with antiseptic solutions, and splint immobilization of affected arm. Surgery should be supplemented with antibiotics (usually within 7-10 days), anti-inflammatory drugs and analgesics. Incisions for drainage of hand phlegmon are placed in accordance with the anatomical location of the abscess. At the same time, they should not affect important anatomical structures: arteries, nerves and synovial bursa. Sometimes, incision may be necessary from both dorsal and palmar side of the hand.

XIII. ANAEROBIC INFECTION

Anaerobic infection – is a severe toxic wound infection, caused by anaerobic microorganisms with preferential affection of muscle, fat and conjunctive tissues. Anaerobic infection is seldom in peacetime, but in the time of war and natural disaster it is more frequent. Anaerobic infection develops in case of dirty, smashed, and gun-shot wounds. There are three kinds of anaerobic infection: classical clostridial infection, non-clostridial infection, and tetanus.

ANAEROBIC CLOSTRIDIAL INFECTION

Gas gangrene and clostridial myonecrosis are interchangeable terms used to describe an infection of muscle tissue by toxin producing clostridia.

Etiopathogenesis. Gas gangrene is caused by an anaerobic, gram-positive, spore-forming bacillus (*Clostridium*). The most common anaerobic infection is caused by *Clostridium perfringens* (50-85%), *Clostridium septicum* (10-30%), *Clostridium oedomatiens* (5-10%), *Clostridium sporogenes, Clostridium hystoliticum*. These organisms are ubiquitous in soil and dust. Bacterial multiplication and production of exotoxins require a low oxygen tension. The most important toxin is alpha toxin – lethal, necrotizing, hemolytic, and cardiotoxic. Toxins lead to edema and necrosis of muscles and conjunctive tissue. Another feature is the ability to cause hemolysis, thrombosis, affection of myocardium, liver and kidney. Forming of gas is classical characteristic of anaerobic clostridial infection.

The incubation period of gas gangrene is short (less than 24 hours). Reproduction of microorganisms begins in damaged tissues and is characterized by forming a lot of exotoxins.

Edema causes the increase of pressure in the fascial compartment which leads to ischemia. Vein thromboses disturb blood circulation. All this leads to ischemia and creates anaerobic conditions in the tissues. Production of gas dissects tissues along muscle bellies and fascial planes. Microorganisms are spread with edematic fluid through perivascular and intermuscular spaces. All these cause a massive spreading of infection.

Systemically, exotoxins may cause severe hemolysis. Hemoglobin levels may drop and, when occurring with hypotension, may cause acute tubular necrosis and renal failure.

Classification. Gas gangrene is classified into posttraumatic, postoperative, or spontaneous types. Posttraumatic gas gangrene accounts for 60% of the overall incidence, mostly in peacetime associated with road traffic injuries.

According to clinical features anaerobic infection is divided into several groups: myonecrosis (predominant damage of muscles) – classic form, necrotizing fasciitis and cellulitis (predominant damage of connective and subcutaneous adipose tissue) – edematic form, and combined form.

History. Most patients with posttraumatic gas gangrene have sustained serious injury to the skin or soft tissues or have experienced open fractures. Patients with postoperative gas gangrene have frequently undergone recent surgery of the gastrointestinal or biliary tract. In contrast, the history is usually unremarkable in patients with occult malignancy–associated spontaneous gas gangrene.

Clinical manifestations. Patients complain of severe pain, sensation of compression, caused by edema. The skin is cyanotic, with blood imbibition and hemorrhagic bullae, local temperature is low (Figure 20). Muscles look like “boiled

meat", and are edematous. Edema is developed quickly. To objectify fast progressive edema, measure the circumference of the limb in dynamics or perform Melnikov's test: a thread is placed loosely round the affected extremity, and it penetrates into the skin in 20-30 min. Palpable crepitus is caused by gas formation. Percussion reveals hyperresonance with "metallic" sound. Accumulation of gas may cause the sign of "champagne cork" during evacuation of gauze dressing.

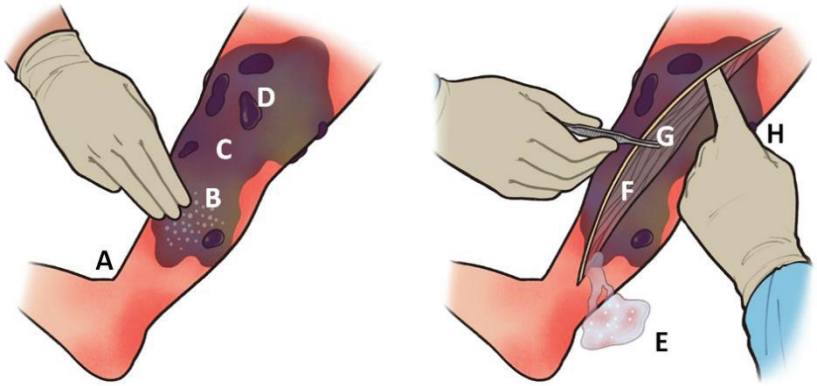

Figure 20. Local signs suggestive for anaerobic soft tissue infection: A – Limb edema; B – Subcutaneous crepitation; C – Hyperemia and cyanosis of the skin; D – Hemorrhagic bullae; E – Exudate in form of „slop" or „dish-water"; F – Muscles with aspect of „boiled meat"; G– Absence of muscle contraction on mechanical irritation; H – Positive „finger test".

Tachycardia disproportionate to body temperature is common. Late signs of gas gangrene include hypotension, renal failure, and an altered mental status.

Diagnosis. X-ray examination and CT scan reveal accumulation of gas in soft tissue (Crauze's sign). Despite

serious infection, WBC counts may not show leukocytosis. A Gram stain of the exudate reveals large count of gram-positive bacilli without neutrophils. Rapid (in as little as 2 hours) detection of alpha toxin in infected tissues through enzyme-linked immunosorbent assay (ELISA) is a potential diagnostic tool. Histopathologic findings in gas gangrene consist of widespread myonecrosis, destruction of other connective tissues, and a paucity of neutrophils in the infected area. Leukocyte aggregates are found in the border regions.

Treatment. The combination of aggressive surgical debridement and effective antibiotic therapy is the determining factor for successful treatment of gas gangrene.

General treatment consists of administration of highly effective antibiotics: a combination of penicillin and clindamycin, or a combination of clindamycin and metronidazole. Patients with gas gangrene frequently require intensive care. Hyperbaric oxygen therapy is used.

Surgical care includes large transection of soft tissues in the affected area or longitudinal incisions on extremity (fasciotomy). The aim of incisions – to improve access of air to wound channel and to decrease compartment and tissue ischemia. Daily surgical debridement of the wound is necessary (excision of necrotic and visually non-viable tissues). In case of unfavorable evolution amputation of extremity may be necessary and life-saving. Abdominal involvement requires excision of the body wall musculature.

Prophylaxis consists of early surgical debridement of the wound. Avoid suturing wounds due to a crush injury or open fractures with devitalized muscle and soil contamination.

ANAEROBIC NON-CLOSTRIDIAL INFECTION

Non-clostridial anaerobic infection is more frequent. These bacteria are present in the normal gastrointestinal flora of human body. They consist of *Peptococcus, Peptostreptococcus, Eubacterium, Fusobacterium, Bacteroides, Bacteroides fragilis, Micrococcus,* and others.

Clinical manifestations are presented by phlegmona and necrosis with affection of subcutaneous adipose tissue, fascia, and muscles. Wide, rapid, and progressive spreading is the feature of this infection. For an objective assessment of the infection' progression, it is recommended to mark the boundaries of inflammation zone on the skin. Severe tenderness, hyperemia or cyanosis of the affected area (sometimes with detachment of epidermis and appearance of hemorrhagic blisters), intense edema, often hypoesthesia of skin and crepitus due to accumulation of gas are noted. General manifestations include fatigue, fever, hypotension, oliguria, and high leukocytosis. The discrepancy between the severity of the general condition and local symptoms may also indicate in favor of an anaerobic non-clostridial infection.

During the surgical debridement of the infectious process, attention is paid to the presence of the following signs: (1) The specific character of exudate in form of "slop"; (2) No or little bleeding when cutting tissues; (3) Lack of contraction of muscles when they are irritated, for example, during diathermocoagulation; (4) The minimum resistance of tissues when trying to separate them with a finger (the so-called "finger test").

Diagnosis. Computed tomography scan allows to visualize the presence of gas and exudate in soft tissues and determine the extension of pathological process. Although in most cases the diagnosis is made basing on clinical manifestations, culture

of anaerobic bacteria is important to confirm the type of infection. Percutaneous direct-needle aspiration of pathological focus is the best method. Transportation of specimens should be prompt in special oxygen-free environments. Cultures should be immediately placed under anaerobic conditions and should be incubated for 48 hours or longer. Gram stain of the specimen or wound discharge provides important preliminary information. Gas-liquid chromatography is informative but rarely performed, not being a widely accessible method.

Treatment. The goal of treatment is to provide early and radically as possible, debriding necrotic tissue, draining pus, improving circulation, and increasing tissue oxygenation. Hyperbaric oxygen therapy and antimicrobial therapy should be used. The *Bacteroides fragilis* group is almost uniformly susceptible to metronidazole, carbapenems, chloramphenicol.

SURGICAL ASPECTS OF TETANUS

Pathophysiology. Tetanus results from infection with *Clostridium tetani*, spore-forming, anaerobic, gram-positive bacillus. This bacillus is found in soil, dust, clothing, skin, and in human gastrointestinal tract. The spores are very stable and need tissue with the proper anaerobic conditions to germinate; the ideal media are wounds with tissue necrosis. Under anaerobic conditions, the spores of *Clostridium tetani* germinate and produce 2 toxins: tetanolysin (with not recognized pathologic activity) and tetanospasmin, which is responsible for the clinical manifestations of tetanus.

The source of infection is usually a wound (≈65%), which often is minor (wood or metal splinters, thorns). Chronic skin ulcers are the source in approximately 5% of cases.

Frequency and mortality. Although rare, the disease has not been eradicated. Reports show up to 1 million cases annually. The mortality rate of severe tetanus may be as high as 60%. The disease is not transmitted from one person to the other.

Classification. The types of tetanus are: generalized, local, cephalic, and neonatal.

- **Neonatal tetanus** is a major cause of infant mortality in underdeveloped countries. The infection results from umbilical cord contamination during unsanitary delivery, associated with a lack of maternal immunization. The mortality rate of neonatal tetanus exceeds 90%.
- **Cephalic tetanus** is uncommon and usually occurs as a result of head trauma.
- Patients with **local tetanus** present with persistent rigidity in the muscle group close to the injury site.
- Patients with **generalized tetanus** present with *trismus* ("lockjaw"), which is the inability to open the mouth secondary to masseter muscle spasm. Dysphagia (difficult swallowing) is also an early complaint. *Risus sardonicus* results from facial muscle involvement. As the disease progresses, patients have generalized muscle rigidity. Tonic contractions cause *opisthotonus* (ie, flexion and adduction of the arms, clenching of the fists, and extension of the lower extremities). During these episodes, patients feel severe pain. The spasms can cause fractures, tendon ruptures, and acute respiratory failure.

Clinical manifestations and diagnosis. Most cases occur in patients with a history of only partial immunization. Symptoms usually begin 8 days after the infection. Commonly the first signs of tetanus are headache and lockjaw: inability to open the mouth secondary to masseter muscle spasm.

Patients may report a sore throat with dysphagia (difficulty swallowing). Patients are afebrile. Severe tetanus results in opisthotonus, periods of apnea resulting from the spasm of the intercostal muscles and diaphragm.

Laboratory findings and imaging studies are not diagnostically valuable in tetanus.

Treatment and prevention. To prevent reasons physicians must clean thoroughly wounds and remove dead or devitalized tissue. The suspected wound should be treated in opened way, and not close it with sutures. However, in most cases when disease has been already developed, the wound responsible for tetanus is clear upon presentation. In these substances surgical debridement of primary wound has no benefit for tetanus.

Passive immunization with human tetanus immune globulin (TIG) shortens the course of tetanus and may lessen its severity. The treatment may require intensive care unit admission and ventilatory pulmonary support. To treat muscle spasms, the following medicines can be administrated: diazepam, magnesium sulfate, sedatives, narcotics, neuromuscular blocking agents, and muscle relaxants. Metronidazole has a good antimicrobial activity.

Prevention is the ultimate management strategy for tetanus. Programmed prophylaxis consists of active immunization. An effective vaccine called tetanus toxoid (anatoxinum) can be administered in combination with diphtheria toxoid and pertussis vaccine (DTP) to children. Revaccination is performed every 10 years.

XIV. SURGICAL SEPSIS

An infection is a microbial phenomenon characterized by a local inflammatory response to the presence of microorganisms. Sepsis is an infection accompanied by an acute inflammatory reaction with systemic manifestations associated with release of numerous endogenous mediators of inflammation into the bloodstream. The fundamental difference between sepsis and a local inflammatory process is an abnormal or unregulated response of the patient's body to infection, leading to damage of its own tissues and the development of life-threatening dysfunction of internal organs (definition III of the International Consensus on Sepsis, 2016). Clinically, sepsis is manifested in the same way although the causal microorganisms are different.

TERMINOLOGY

Historically, sepsis has been viewed as a “generalized” infection accompanied by bacteremia – the presence of pathogenic microorganisms in systemic circulation. Sustained bacteremia, in contrast to transient bacteremia, may result in a sustained febrile response. The term “septicemia” refers to the multiplication of bacteria in the bloodstream, and “septicopyemia” – is bacteremia with purulent metastasis into distant organs.

In 1992, a group of international experts developed clinical and laboratory criteria for the diagnosis of sepsis and its separate forms:

Sepsis subgroup	Definition
Systemic inflammatory response syndrome (SIRS)	The presence of two or more of the following criteria: - Temperature >38˚C or <36˚C - Heart rate >90 beats/min - Respiratory rate >20 breaths/min - Altered WBC count >12,000/mm^3 or <4,000/mm^3
Sepsis	The systemic inflammatory response to documented infection
Severe sepsis	Sepsis associated with organ dysfunction (multiple organ failure)
Septic shock	Sepsis induced hypotension persisting despite resuscitation, presence of hypoperfusion abnormalities or organ dysfunction

In subsequent decades, the distinction between “sepsis” and “severe sepsis” was eliminated, and dysfunction of vital organs became as a diagnostic criterion for sepsis.

The use of the word “sepsis” is a source of confusion because it is used in different clinical situations (from “wound sepsis” – infected wound to septic shock). Note that the term sepsis is not synonymous with infection and should be reserved for the systemic process.

INCIDENCE AND MORTALITY

The annual rate of sepsis is 300 cases per 100,000 people, while the total mortality that exceeds 30%. Mortality rates are the highest (50-80%) in patients with septic shock. Every year 18 million cases of severe sepsis are recorded.

Sepsis is one of the most common causes of death from infection in intensive care units. The increasing incidence of sepsis is due to aging population; better survival of patients who are at high risk due to chronic diseases representing an increased risk of sepsis, such as diabetes; wide use of invasive devices for monitoring and treatment of critically ill patients; the increasing use of therapies that result in immunosuppression; and increasing resistance of bacteria to antibiotics.

PATHOPHYSIOLOGY

The pathogenesis of sepsis is complex and, despite significant advances, still not well understood. Sepsis is the result of the interaction between pathogenic microorganisms and host factors. Following initial tissue injury, a local inflammatory response occurs with cytokine induction. Released cytokines include tumor necrosis factor (TNF), interleukins (IL-1, IL-2, IL-6) and platelet activating factor (PAF). Cytokines are proteins, which control specific and nonspecific immunological reaction of organism. They are produced by leucocytes, macrophages, and monocytes.

The primary response to local infection and release of cytokines is to mobilize macrophages and neutrophils which diapedese into the tissues (Figure 21). Cytokines signal systemic elements of inflammation resulting in activation of endothelium, complement system and blood coagulation, which amplifies the primary inflammatory response. This is normal and appropriate. However, if the infection is severe or persistent, the localized reaction may spill over excessively into the systemic circulation, producing the sepsis syndrome.

The effects of cytokines on the human organism are generalized neutrophil-endothelial cell adhesion and

endothelial injury, increased capillary permeability, coagulopathy with capillary leak and microthrombi, tissue hypoxia and ischemia, all of which can lead to the development of multiple organ dysfunction syndrome.

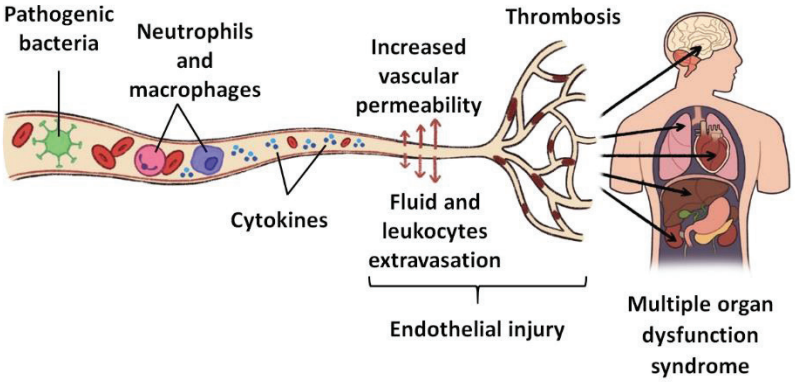

Figure 21. Basic mechanisms in pathophysiology of surgical sepsis.

Additional mechanisms for the development of multiple organ failure are: shunting of capillary blood flow with cessation of oxygen delivery to tissues (distributive defect), as well as impaired intestinal barrier function and translocation of intestinal bacteria and endotoxins into the portal and systemic circulation.

CLASSIFICATION

Most cases of sepsis are caused by gram-negative bacilli or gram-positive cocci. According to the type of bacteriological agent sepsis is classified into: sepsis caused by *Staphylococcus*, by *Streptococcus*, by *Escherichia coli* sepsis, by *Klebsiella*, anaerobic sepsis (*Bacteroides fragilis*). Rarely, it is caused by *Candida*.

The source of infection that leads to sepsis may be acquired (intra-abdominal abscess, limb gangrene) or endogenous (from the patient's own bowel due to translocation). According to localization of primary focus of infection there are: abdominal sepsis (14%), sepsis of soft tissue, pulmonary (44%), urological (18%), gynecological, blood (endocarditis, central venous catheterization), and neonatal types of sepsis.

According to clinical evolution sepsis is classified into: fulminate form sepsis (develops within 5-7 days), acute sepsis (2-4 weeks), subacute sepsis (6-12 weeks), chronic sepsis. Although, the existence of this last form of sepsis remains controversial.

There are practically no specific morphological signs of sepsis. However, in the most severe cases can be determined septicopyemia (the presence of secondary purulent metastases in various tissues and organs).

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The basis for clinical diagnosis of sepsis are: classical signs of surgical infection in combination with symptoms of SIRS and / or signs of multiple organ failure.

The signs of multiple organ failure include: low cardiac output and hypotension; low partial oxygen saturation (PaO2 <400 mm Hg) or need in pulmonary ventilation; bilirubin, AST and ALT increased; oliguria less than 30 ml per hour (or <500 ml/day); thrombocytopenia less than 100,000/mm^3; adynamic intestinal obstruction and gastric stress ulcer bleeding; consciousness disturbances and others.

To quantify the listed signs, it is recommended to use a special scale SOFA (Sequential Organ Failure Assessment). As a simplified version, a “fast” scale is used – qSOFA, which includes only three criteria: (1) respiratory rate ≥22 per

minute, (2) systolic pressure ≤100 mm Hg, (3) impaired mental status of the patient. The presence of two or more criteria indicates organ dysfunction. An objective diagnosis of septic shock requires two criteria: (1) the need for vasopressors to maintain mean arterial pressure (1/3 systolic + 2/3 diastolic) ≥65 mm Hg, (2) blood lactate level >2 mmol/L.

Detection of microbial infection is necessary. However, local clinical signs of infection are often absent. In such situations, it is necessary to use instrumental diagnostic methods: X-ray of the lungs, ultrasonography, computed tomography or magnetic resonance, thoracocentesis, paracentesis, etc.

On admission blood cultures of patients are obtained, optimally – before the start of antibiotic therapy. Leukocytosis with left shift is an important, but nonspecific diagnostic finding. Recently biochemical markers of sepsis have been proposed: cytokines (IL-1, IL-6, TNF), C-reactive protein and procalcitonin. Their level in sepsis is increased, which can facilitate diagnosis, but they are also not specific. Ultimately, diagnosis of sepsis is predominantly clinical.

TREATMENT

The management of patients with sepsis includes general and local treatment. Local treatment consists of early control of the infection source: surgical debridement and excision of necrotic tissues, drainage of abscesses, local wound treatment.

Antimicrobial therapy using broad-spectrum antibiotics is the mainstay of general treatment. Antibiotics are given empirically immediately after the diagnosis of sepsis is made. The mortality rate is substantially lower in patients who

receive prompt (during the first 1-3 hours) and appropriate antimicrobial therapy.

The presence of organ dysfunction dictates the need for intensive therapy and syndromic correction of systemic disorders (respiratory, renal, liver failure). With hypotension, the introduction of large volumes of crystalloids (Ringer's and Hartman's solutions), colloidal blood substitutes, and albumin is indicated. Objective indicators of the effectiveness of intensive therapy are: mean arterial pressure, diuresis and blood lactate level.

XV. GENERAL SEMIOLOGY AND DIAGNOSTIC PROCESS

PHASES OF DIAGNOSTIC PROCESS

Diagnosis (Gr.) means discernment, ability to distinguish. Diagnostics is a complex cognitive process and completes with analysis. In order to establish the correct diagnosis, the doctor must have the following qualities: knowledge, attention, systematic approach, logic, critical analysis of obtained data and the use of accumulated experience.

The **diagnostic process** has a 3 phases:

1st phase: detection of specific symptoms and syndromes.

A **symptom** (Gr.) means accident, misfortune (pain, vomiting, dyspnea, swelling). The existence of symptom indicates a deviation from normal condition and means the presence of pathology or abnormality. The term is sometimes also applied to physiological states outside the context of disease, as, for example, when referring to "symptoms of pregnancy". The symptom cannot be measured quantitatively, and its presence is described as "positive", and its absence – as "negative". The terms "asymptomatic condition", as well as "asymptomatic course of disease" are used sometimes to emphasize absence or lack of subjective manifestations of underlying disease. In English medical literature the word symptom is defined as any feature which is noticed by a patient. A sign is usually noticed by a physician.

Syndrome (Gr.) means together, amongst others. It is a complex of symptoms that have a common genesis and characterize a certain pathological condition (acquired immunodeficiency syndrome, systemic inflammatory response syndrome, acute abdomen, intestinal obstruction, acute limb ischemia, etc.)

The science that studies symptoms and syndromes of a disease is called semiotics, or **semiology**. Another definition: semiology is the art of interpretation of symptoms. So, semiology includes collection of patient's complaints, history

of disease *(anamnesis morbi),* history of life *(anamnesis vitae)* and documented underlying diseases. Determination of objective signs by inspection, palpation, percussion, and auscultation. Some of the special tests, not requiring any sophisticated equipment (thermometry, and anthropometry, functional tests of veins using tourniquet, monofilament thread for testing sensitivity in peripheral neuropathy) also relate to semiology. In other words, semiology is equal to the first phase of diagnostic process and ends with establishing of preliminary diagnosis.

2nd phase of diagnostic process: laboratory tests, instrumental imaging studies (non-invasive or invasive) that can confirm or clarify the preliminary diagnosis.

3rd phase of diagnostic process includes comparison of subjective and objective data with the results of laboratory and imaging studies, and differential diagnosis.

When the diagnostic process is finished, the clinical diagnosis is made. On the basis of this diagnosis, the further curative policy is determined. It may include conservative treatment or surgery. The final diagnosis is based on the results of surgery, morphological and bacteriological data, and it is made on the moment of the patient's discharge.

SURGICAL HISTORY OF DISEASE

History of disease – is the documentation of diagnostic process, evolution of disease, and efficacy of treatment (surgery). The form of history of disease is well known. Surgical history differs by the presence of specific parts: **local status** *(Status localis),* which describes visible suppurative processes of soft tissues, masses and lumps, hernias, and location, form, depth and other characteristics of wounds. Preoperative conclusion, surgical protocol and postoperative monitoring are also distinctive parts of surgical history of disease.

XVI. SEMIOLOGY OF BREAST DISEASES

Surgical semiology is a keystone of primary diagnosis and differentiation of the breast diseases. Breast diseases are **classified** into congenital and acquired. Congenital abnormalities are malformations (polytelia, atelia, amastia, and aberrant breast). Acquired diseases are: (1) Inflammatory – nonspecific diseases (acute lactational or puerperal and nonlactational or non-puerperal mastitis) and specific diseases (tuberculosis and syphilis of the breast); (2) Dyshormonal diseases (mastopathy, gynecomastia); and (3) Tumors of the breast (benign or malignant). Although, additional laboratory and imaging examinations are often necessary to establish definitive conclusion, basic information is obtained by complex assessment of history and physical examination data.

HISTORY

History has an important role: pregnancy and delivery, lactation, preexistent inflammatory diseases, hormonal contraceptives intake, menopause, gynecological pathology. High fever and local pain in the breast may suggest inflammatory process, premenstrual fullness, tenderness and pain – mastopathy, palpable mass – tumor. It is of high importance to establish risk factors for breast cancer: increasing age, prior cancer on the opposite breast, presence of tumor in patient's mother or sisters, late or no pregnancy, early menarche and late menopause, exposure to ionizing radiation (radiation therapy), obesity, and also intake of estrogens contraceptives.

PHYSICAL EXAMINATION

Inspection detects congenital anomalies of the breast. The most common is **polytelia** – one or more extra breasts, located along the so-called "milk line", which passes from nipples to inguinal regions. Only a small nipple and areola are usually present, they are often mistaken for a common mole. As a rule, glandular tissue is absent. Polytelia has no pathologic significance.

Atelia (absence of nipples), **amastia** (absence of breast), **polymastia** (supernumerary breasts) refer to rare abnormalities. **Aberrant** breast is commonly located in the axillae. It consists of functional glandular tissue and increases during lactation. All conditions mentioned above, generally, present cosmetic problems.

Routine physical examination of the breast is the easiest during the first 1-2 week after menses. Initial inspection is done with the patient in sitting position, with her arms at her sides. On inspection note the following signs (Figure 22):

- **The size and symmetry** of the breasts.
- **Skin color.** Redness of skin may suggest infection or carcinoma. A presumed infection of the breast generally clears promptly and completely with antibiotic therapy. If erythema or edema persists, inflammatory carcinoma is considered.
- **Thickening and edema of skin**. Unusually **prominent skin pores** ("orange peel" sign) is characteristic of breast cancer. Its mechanism is explained by accompany lymphatic vessels obstruction with tumor's cells.
- **The contour** of the breast. Local protrusion or, in contrast, flattering of the contour may suggest inflammatory or cancerous mass.

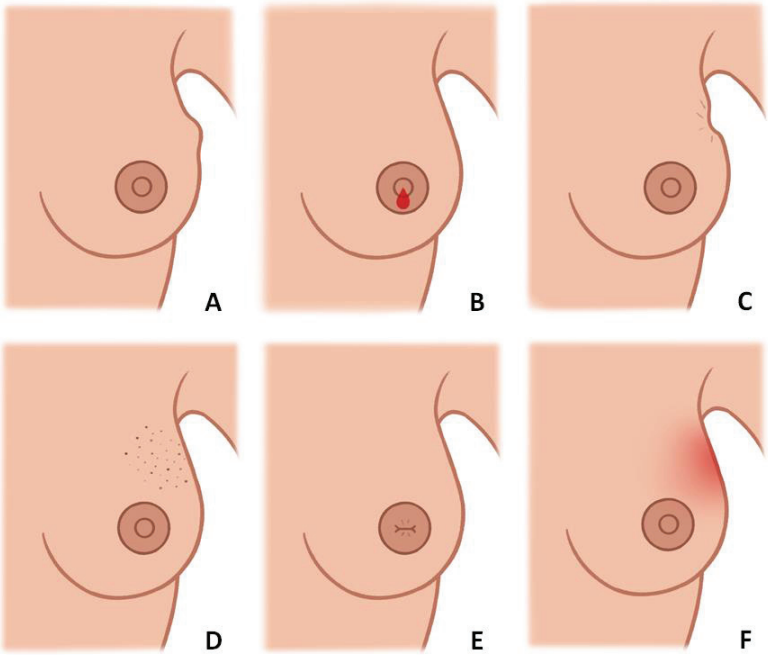

Figure 22. Pathological signs detected on inspection, suggesting possible malignant neoplasm of the breast: A – Local protrusion; B – Pathological discharge from the nipple; C – Skin dimpling; D – Local swelling of skin ("orange peel" sign); E – Unilateral nipple retraction; F – Local hyperemia of skin.

- **Skin dimpling.** As breast cancer advances, it causes fibrosis. Shortening of the fibrotic tissue produces retraction signs, involving skin and nipple.

To detect the retraction that may be invisible the patient is asked: to raise her arms over her head; and then to press her hands against her hips. Repeated inspection of patient with raised arms allows detecting prior invisible signs of retraction. Pressing against the hips contracts the pectoral muscles.

When cancer or its associated fibrous strands are attached to the fascia overlying these muscles, pectoral contraction can draw the dumpling.

- **Size and shape of nipples.** Occasionally, nipples are inverted and depressed below the areolar surface. Recent and unilateral flattering or depression of the nipple, which may deviate the areola into two parts, are typical for an underlying tumor.

- **Any rashes or ulceration of areola and nipple** may suggest a rare form of breast cancer – Paget's disease. The disease starts as a scaly, eczema-like lesion. The skin may also weep, crust or erode. Paget's disease should be suspected in any persisting dermatitis of the areola and erosions of nipple.

PALPATION

Palpation of the breast is done in lying position of a patient with her arm rested over her head. This helps to spread the breast across the chest and make it easier to find nodules. Palpation is performed with all fingers flat on the breast, compress the tissue gently in a rotary motion against the chest wall (**Velpeau method**). Proceed systemically, examining the entire breast including the periphery and areola. It determines:

- **The consistency of tissue.** Normal consistency varies widely, depending on the proportion of soft fat and firmer glandular tissue. Physiologic nodularity may be present and may increase premensrually. Tender and painful cords suggest mammary duct ectasia (enlargement) with inflammation around them; it is a benign disease called fibrocystic **mastopathy**. In breast conjunction tissue and cysts with fluid are developed. Most common symptom is pain in the breast, which has a periodical character and increases till the 5-7-th

days before menses. Breast palpation reveals tender, nodular breasts, more often located in the lateral regions. With mastopathy, a Kőnig's sign is determined: palpable mass in the upper right position disappears in repeated palpation in lying position.

- **Tenderness** caused by cysts, inflamed areas can be determined in the premenstrual period. Sometimes pain may be a manifestation of cancer.

- **Mass**. Any lump or mass that is qualitatively by its density different from the rest of the breast tissue is suspected. If one or more nodules are present, it should be described:

(1) Their location, by quadrant of breast (superior-external, superior-internal, inferior-internal, inferior-external) or the clock method, and their distance from nipple in centimeters;

(2) Number of nodules;

(3) Size in centimeters;

(4) Shape (round or discoid);

(5) Contour (regular or irregular);

(6) Consistency (fluid, soft, elastic, firm or hard);

(7) Tenderness on palpation;

(8) Delimitation in relationship to surrounding tissues (well circumscribed or not). Delimitation in relationship to surrounding tissues (well circumscribed or not). So, hard, irregular, poorly circumscribed nodules, fixed to the skin or underlying tissue, strongly suggest cancer.

(9) Mobility, with reference to the skin, pectoral fascia, and the underlying chest wall. For determination of mobility, can try to move the skin over the mass or move the mass itself.

- **Subareolar nodules.** Palpation is finished with gentle compression of the areola for the detection of subareolar nodules, such as intraductal papilloma.

- **The character of discharge** may also be established. Small amount of milky discharge may persist for long periods after lactation. Milky discharge unrelated to a prior pregnancy or lactation is called **nonpuerperal galactorrhea**. It is generally of hormone and drug related origin. A non-milky discharge suggests local benign or malignant breast disease. Serous discharge is caused by benign lesions, purulent discharge – by mastitis, bloody discharge – by intraductal papilloma, or underlying cancer.

- Because the lymphatics of much of the breast drain toward the axillae, assessment of the axillary **lymph nodes** is an important part of breast surgical semiology. The central axillary nodes are commonly palpable. They are usually located high in the axillae and midway between the anterior and posterior axillary folds. If the central nodes fell large, hard, or tender, or if there is a breast mass suspicious, the other groups of lymph nodes should be detected: the pectoral (or anterior) lymph nodes, subscapular (or posterior) lymph nodes group, lateral group are palpated along the upper humerus. Lymph drains from the central axillary nodes to the infraclavicular and supraclavicular nodes.

Malignant breast tumors metastasize to the brain, lungs, bone, liver, as well as the opposite breast.

ADDITIONAL METHODS

The routine breast examination should end with a discussion of the elements of **breast self-examination**. Breast self-examination is best carried out just after menstruation ends or monthly in nonmenstruating women. Breast self-

examination should include observation in a mirror as well as palpation in both upright and supine positions.

When benign or malignant diseases of breast are suspected imaging and instrumental investigation methods are the following:

Mammography – non-contrasted X-ray examination of the breast. Mammography is also used for screening. The aim of screening is to detect breast cancer at an early stage, when any changes in the breast would be too small to feel or to detect on physical examination.

Ductography is indicated in the presence of pathological discharges from nipples. Contrasted material is introduced into the cannulated duct.

Ultrasonography may differentiate firm mass from fluid collection.

Thermography is based on the fact, that the temperature of malignant tumor is 1.5-2°C higher than the temperature of surrounding tissues. It is performed with assistance of special thermal probes.

Fine needle aspiration and biopsy permits rapid, minimally invasive cytological and histological diagnosis of many palpable detected breast masses.

MALE BREAST

It is necessary to inspect the nipple and areola for nodules or ulceration, which may suggest **breast cancer**. The adult male breast remains a rudimentary structure with ductal elements and nipple-areola complex. Cancer may develop from ductal elements in the presence of hormonal disorders. Clinical signs of breast cancer in men are similar with in women: mass which involves areola and nipple, bloody

discharge, hyperemia of skin, retracted nipple, and dimpling signs.

Gynecomastia – is a pathological enlargement of glandular tissue, caused by estrogens-androgens imbalances. If the breast appears enlarged, distinguish between the soft fatty enlargement that may accompany obesity and the firm, well circumferented, mobile disc of tissue, palpable immediately beneath the nipple–areola complex, and called gynecomastia. It is commonly idiopathic, but may be associated with use of drugs, systemic diseases (liver dysfunction, Klinefelter syndrome, testicular tumor). Idiopathic gynecomastia requires surgery.

XVII. SEMIOLOGY OF ACUTE ABDOMEN

GROUPS OF DISEASES

Abdominal pain is one of the most common condition which calls for prompt diagnosis and treatment. Usually, other symptoms accompany the pain, but in most cases of acute abdominal disease the pain is the main symptom and complaint. The term **“acute abdomen”**, which is constantly applied to such cases, is a general definition but, in the same time, a syndrome, and it signifies the need for prompt diagnosis and early treatment. Patients with acute abdomen need urgent admission into the surgical department. However, the term “acute abdomen” should never be equated with the invariable need for surgery.

Lesions, which give rise to the syndrome of “acute abdomen”, are:

(1) **Inflammatory diseases** of the abdominal organs (acute appendicitis, acute cholecystitis, cholangitis, diverticulitis, acute pancreatitis, Crohn’s disease, inflammatory diseases of uterus and annexes, etc.);

(2) **Perforation of a hollow organ** into the peritoneum (gastric or duodenal perforated ulcer, gastric or bowel tumor perforation, appendiceal perforation, gall bladder perforation, spontaneous rupture of esophagus, rupture of urinary bladder, traumatic injury or rupture of hollow organs);

(3) **Intestinal obstruction** (adhesions, large occlusive bowel tumor, intussusception, lumen obstruction by biliary stone or ascaris impaction, strangulated hernia, outer compression by cyst or tumor, etc.);

(4) **Intraperitoneal hemorrhage** – spontaneous (rupture of a tubal gestation, ruptured Graafian follicle, ruptured aorta

aneurysm), and traumatic (rupture of spleen, liver, kidney, mesentery, large vessels);

(5) **Extraabdominal diseases** may also be accompanied by severe abdominal pain (myocardial infarction, pneumonia, alimentary tract infections, renal colic, non-compensated diabetes mellitus, systemic vasculitis, etc.)

COMPLAINTS AND HISTORY

The necessity of making a thorough history taking and physical examination in every acute abdominal case is undoubted. Radiologic or ultrasound examinations, computed tomography scanning and the vast number of laboratory tests will not compensate for a poor or incomplete history and physical examination. A large number of acute abdominal conditions can be diagnosed by considering carefully the history of onset. Should be noted:

Patient's age. The incidence of certain conditions is limited within a particular range of years. Acute intussusception with intestinal obstruction occurs generally in infants under two years of age. Cancerous obstruction of the large intestine is relatively common in persons over 60 years of age. Perforated gastroduodenal ulcer is rare under 15 years of age, and acute pancreatitis is seldom under the age of 20.

Exact time of onset. Patients with perforated gastroduodenal ulcer can fix the exact time and even minute, at which the pain started. It is also important to determine whether the condition began immediately after some injury. Hernia strangulation generally develops after a sudden physical workout, weightlifting, change of body position.

Acuteness of onset. Perforation of gastric or duodenal ulcer, acute pancreatitis, and ruptured aortic aneurysm are

the only abdominal conditions which lead to collapse. In women the rupture of ectopic gestation also causes fainting. Majority cases of intestinal obstruction and inflammatory abdominal organs diseases have a gradual onset. Also gradually, in correspondence on progression of pathological process, increase symptoms in case of inflammatory abdominal organs diseases.

It is important to know, that according to its origin, pain is divided into visceral and somatic ones.

Visceral pain arising from the gastrointestinal organs, due to distention, gaseous dilatation or spasm of intestine, gall bladder, urinary bladder and ureter, and is distributed by celiac nerves. Visceral pain has diffuse character, and is mainly localized around of the umbilicus. Often patients cannot define an exact site of pain. Pain is often felt by patients in the form of compression, cramps and colic.

Somatic pain arises from the parietal peritoneum, mesentery of the small and large bowel, omentum and retroperitoneal space. Somatic pain is strictly limited, very intensive and continuous. Commonly, the origin of pain is due to inflammatory processes.

The **abdomen is often divided** for descriptive purposes into four quadrants by imaginary lines crossing on the umbilicus: the right upper, right lower, left upper and left lower quadrants. Another system divides the abdomen into nine regions (3 parts). They are epigastric (includes right and left hypochondrium, and epigastric area itself); mesogastric (includes the periumbilical area, left and right lateral flanks); and hypogastric (suprapubic area, right and left iliac fossa) (Figure 23).

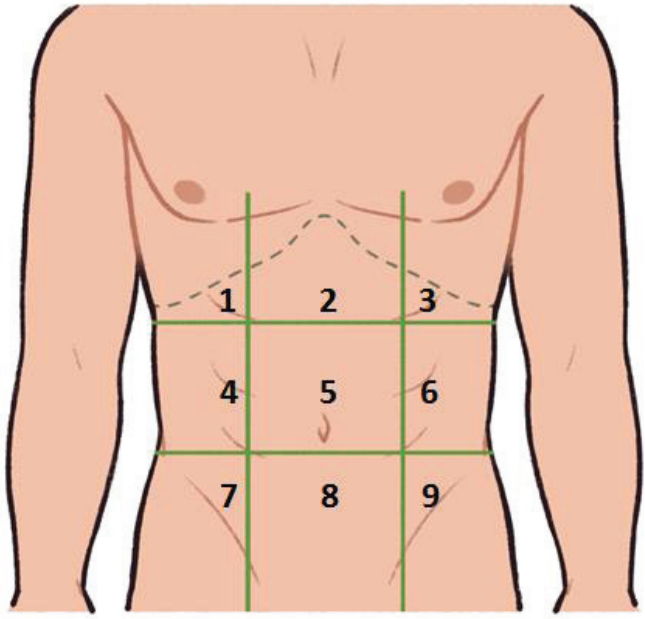

Figure 23. Nine zones of the abdomen: 1 – Right hypochondrium; 2 – Epigastric area; 3 – Left hypochondrium; 4 – Right mesogastric region (right abdominal flank); 5 – Periumbilical area; 6 – Left mesogastric region (left abdominal flank); 7 – Right iliac fossa; 8 – Hypogastric (suprapubic) area; 9 – Left iliac fossa.

Initial pain location. Pain arising from the small intestine is always felt first in the umbilical areas of the abdomen. The pain due to large gut affections is felt first in the hypogastrium. Pain in the inguinal regions may suggest strangulated hernia (Figure 24).

Shifting of pain. Shifting of pain in the right iliac fossa some hours after acute epigastric pain is usually due to appendicitis, and presents a classical sign of disease (Kocher's sign). When severe pain is first felt in the thorax, but later it is felt more in

the abdominal cavity, one must consider the possibility of dissecting aortic aneurysm.

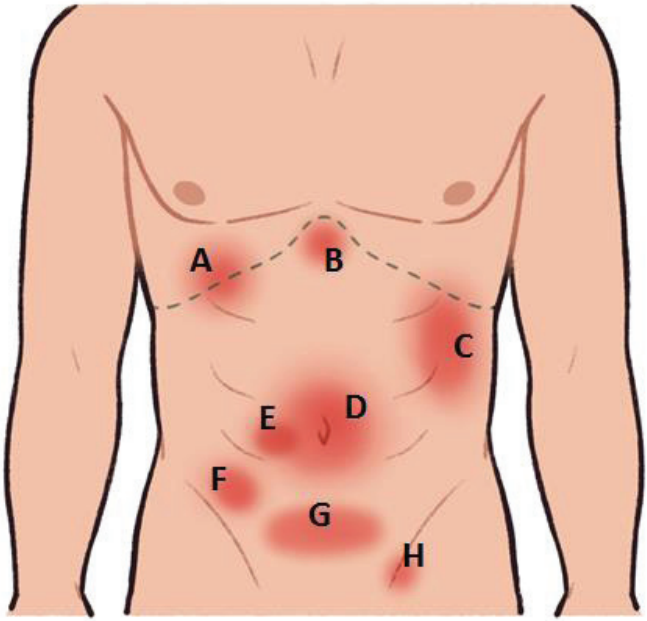

Figure 24. Common site of abdominal pain in some diseases: A – Acute cholecystitis; B – Acute pancreatitis; C – Renal colic; D – Pathology of the small intestine; E – Crohn disease; F – Acute appendicitis; G – Pathology of the large bowel; H – Strangulated inguinal or femoral hernia.

The character of pain is often a help to determine the nature of condition. General burning "knife-like" pain is characteristic of perforated gastric ulcer; agony continuous pain occurs in acute pancreatitis; sharp constricting pain which takes breath away – in biliary or renal colic; tearing pain – in dissecting aneurysm, gripping pain – in intestinal obstruction; acute constant pain – in acute appendicitis; dull pain – in pyelonephritis.

Radiation of pain. In biliary colic the pain frequently radiates to the inferior angle of the right scapula, while renal colic – in the testicle and femur of the same side. In many conditions of upper abdomen, the pain radiates to the top of the shoulder. In acute pancreatitis the pain typically radiates to the right or left loin, or has "like a belt" character. Uterine and rectal pains radiate back into the sacral area.

Increase of pain on movement or respiration. Pleuritic pain is usually maximal on deep inspiration. Inflammation of the gallbladder may cause inhibition of movement of the diaphragm, and the pain may be increased by a forced respiration. Pain accentuated by reclining and relieved by an upright position is often of retroperitoneal origin, as in pancreatitis. In patients with appendicitis pain increases on walking, especially, in step on the right leg.

Vomiting is a typical complaint of patients. In acute abdominal lesions vomiting is always due to one of these causes: (1) Reflex mechanisms (2) Irritation of the nerves of the peritoneum and mesentery; (3) Mechanical obstruction of the intestines.

Frequency of vomiting. A single vomiting episode at the very onset of disease is characteristic for acute appendicitis. In obstruction of the small intestine vomiting is usually frequent and copious in quantity. Frequent vomiting also occurs in patients with acute cholecystitis and acute pancreatitis. There are, however, many serious abdominal conditions in which vomiting infrequent or absent. Internal hemorrhage from a ruptured ectopic gestation or ruptured spleen is often accompanied by no nausea and vomiting. Also vomiting is uncommon for perforated gastric or duodenal ulcer. In obstruction of the large intestine vomiting is a late or infrequent symptom.

Character of vomit. In acute gastritis the vomit consists of gastric contents mixed with a little bile. Frequent vomiting with bilious fluid is characteristic of acute cholecystitis and acute pancreatitis. In intestinal obstruction the character of vomiting material varies. First the gastric contents, then bilious material, then greenish-yellow, yellow, and finally orange or brown feculent-smelling fluid is ejected. The so called "feculent" vomit is pathognomonic of obstruction of small intestine.

Constipation and diarrhea. The occurrence of constipation for several days is a serious symptom of large bowel obstruction. Diarrhea is common in acute gastroenteritis and in cases of pelvic appendicitis. The presence of blood and mucus in the rectum is a classical sign of intussusception.

Thirst is especially evident in advanced peritonitis, severe pancreatitis and acute intestinal obstructions, which are associated by severe hydro-electrolyte imbalances.

Past history. It is well to inquire a patient concerning any previous illness. Pain which develops in 2-3 hours after meal would suggest exacerbation of duodenal ulcer. Right hypochondriac pain related to meals suggests the presence of gallstones.

PHYSICAL EXAMINATION

General appearance. One should always take the time to watch the patient quietly for several minutes. The pale or gray face covered with cold sweat suggests perforated gastroduodenal ulcer, acute pancreatitis or acute strangulation of gut. Deathly pallor face in a woman with internal hemorrhage from rupture of tubal gestation is typical. The earthy-gray color of the skin covered with cold sweat, pointed facial features, shiny eyes, make up the classic mask-

like appearance of the face named “Hippocratic face” or “abdominal face” characteristic of advanced peritonitis.

Behavior in bed. Restlessness of those suffering from severe colic contrasts with the immobility of those suffering from peritonitis. In extensive peritonitis the knees are frequently drawn up to relax the abdominal tension. In cases of ruptured spleen or liver the patient lies quietly on his side (“tilting doll” symptom).

Skin color does not change in the majority of acute abdominal diseases. However, jaundice is often observed in acute cholecystitis, cholangitis and pancreatitis, pallor – in peritoneal hemorrhage, cyanosis – in thrombosis of mesenterial vessels.

Pulse. An increase in the frequency of pulse is a constant accompanying symptom of the advanced stages of peritonitis and peritoneal bleeding. In late peritonitis the pulse is rapid (so as to be almost uncountable) and weak (so as cannot be determined).

Blood pressure. The decrease of blood pressure suggests internal hemorrhage, and circulatory failure following intestinal obstruction.

Respiration rate is important in differentiating between abdominal and thoracic conditions. If the respiration rate is raised to double to normal on the onset of illness, the causative lesion is probably thoracic in origin.

Temperature. Subnormal temperature (35-36˚C) is recorded in the diseases, associated by shock: pancreatitis, perforated gastroduodenal ulcer or intraperitoneal hemorrhage. At the onset of appendicitis, the temperature is usually normal, but within a few hours it rises up to 37.3-37.5˚C. When appendiceal perforation occurs the temperature may achieve 38˚C. It should be remembered that very high

fever is quiet unusual in the early stages of acute abdominal surgical diseases. If patient with abdominal pain is found to have a temperature of 40-41˚C at the onset of illness, the thorax or the kidney is very likely the seat of the disease.

Inspection of the abdomen. On inspection the size and symmetry of the abdomen should be assessed. Symmetric distension of the abdomen is caused by obesity, ascites or general intestinal distension by gas. Asymmetric distension is determined in case of intestinal obstruction, when one or few severe distended loops may be seen through the anterior abdominal wall, or in the presence of a large tumor. In patients with tumors of pancreatic head a **Courvoisier rule** as a visible protrusion in the upper right abdomen, caused by an increase in its volume gallbladder can be detected.

All hernias orifices must be inspected as a routine, and special attention directed to the femoral canal, where in an obese subjects a small hernia is easy to overlook.

In the case of a retroperitoneal hematoma caused by acute pancreatitis, ruptured abdominal aortic aneurysm, or blunt abdominal trauma, physical examination may reveal bruising around the umbilicus (**Cullen's sign**), or in the flanks and lumbar region (**Gray Turner's sign**).

ABDOMINAL PALPATION

Abdominal palpation is performed when a patient is in lying position, with his arms relaxed down along the sides of the body, and knees – slightly bent. This allows relaxation of the anterior abdominal wall muscles. Gentleness is essential to succeed in palpation. Palpation should begin in the part removed from the point of maximum pain. Palpation determines the extent and intensity of the muscular rigidity,

any site of pain, areas of hyperesthesia, peritoneal signs, and the presence of any swelling.

Rigidity (or tenderness, muscular resistance, defense, contraction) is a reflex related to involuntary muscular rigidity in response to peritoneal inflammation or irritation. Muscular contraction may be firm, continuous and extend on the whole abdomen, which releases a classical view of the "board-like" abdomen in perforated gastroduodenal ulcer. However, more often tenderness is localized in a certain area: the right upper quadrant – in acute cholecystitis; the epigastrium – in acute pancreatitis; the right iliac fossa – in appendicitis. Rigidity is often absent in the pelvic inflammatory lesions, is diminished and is difficult defined – in peritoneal bleeding, and is not characteristic – in intestinal obstruction.

In some conditions, muscular resistance may be very slight even in the presence of serious peritonitis: (1) When the abdominal wall is very fat and flabby, (2) In patients with severe toxemia, (3) In patients with shock, and (4) In elderly patients.

Hyperesthesia. May be tested by light and superficial sliding movement with the outer tips of fingers. Skin hyperesthesia in the right iliac fossa may be determined in acute appendicitis. However, its presence has a relatively little diagnostic value in the majority of acute abdominal pain.

Peritoneal irritation. Classical symptom – is the Blumberg's sign: the fingers are pressed gently but deeply over the inflamed focus (Figure 25A) and then the pressure is withdrawn. In case of peritoneal inflammation, the patient experiences a sudden severe pain (Figure 25B).

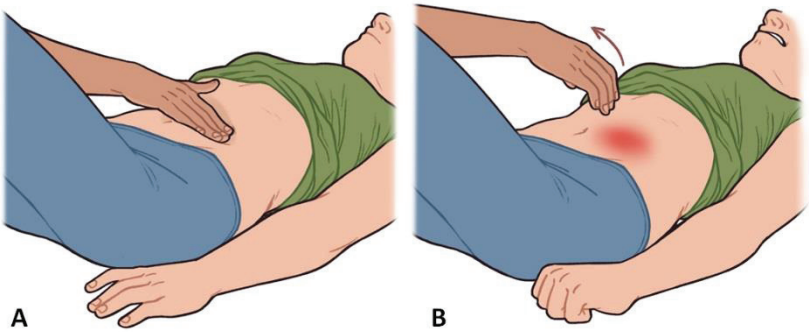

Figure 25. Positive Blumberg's sign.

Determination of iliopsoas rigidity. Hip flexion increases a severe pain – it is called psoas symptom. In another variant, the patient lies on the opposite side and extend the hip (Cope's sign).

Palpation of the loins may be done in a bimanual manner. In bimanual palpation the physician brings one hand anterior, and another – posterior of the loin. Thus he can feel between the two hands any loin swelling or mass. A pyonephrosis or hydronephrosis or a lumbar abscess can thus be detected.

PERCUSSION AND AUSCULTATION

Percussion of the abdomen. One can assess the amount of gaseous distention of the gut, pattern of dull area – in the presence of free fluid by percussion of the abdomen.

Liver dullness. If on percussion a resonant sound is obtained in the normally dull liver area there is likely to be pneumoperitoneum due to the rupture of the stomach or intestine. Disappearance of liver dullness may be observed in large bowel intestinal obstruction.

Free fluid. Free fluid in the abdomen may be serum, pus, blood, bile, or urine. For the determination of free fluid one flank is percussed while the patient is lying on the back, and after he has been turned over onto the opposite side again.

Auscultation. A surgical aphorism says, that a quiet abdomen means peritonitis and a loud borborygmi (peristalsis) suggests intestinal obstruction. Besides, in intestinal obstruction an auscultative symptom – "plash sound" is also determined over the affected gut loop, containing large amount of fluid and gas. Aortic bruits should be heard in the point, located just above the umbilicus on the left.

Rectal examination. Sometimes digital rectal examination may reveal a stricture of the rectum due to cancer, or the apex of intussusception. Pressing on the anterior rectal wall may produce tenderness and sudden pain, which suggest pelvic peritonitis or pathological fluid accumulation in the Douglas pouch.

XVIII. TRAUMA. INJURIES OF HEAD, CHEST, ABDOMEN AND SKELETAL SYSTEM

DEFINITION AND INCIDENCE

Trauma is a breakdown of morphological integrity and functions of tissue, organs and systems of organism, which develops due to the action of external force or energy. Traumatic injuries take the second place in mortality structure during peacetime, and are the main causes of death among people aged 1-50.

Trauma-related deaths occur in **three periods** after injury. About half of all deaths occur within seconds or minutes of injury and are related to lacerations of the aorta, heart, brain, and spinal cord. The second mortality peak occurs within hours of injury (30%), which are caused by hemorrhage and by injuries to the central nervous system. As many of these deaths can be prevented by early treatment during the "golden hour" after injury, the development of trauma treatment systems, including rapid transportation is of high importance. The third mortality peak (20%) includes deaths that occur in the period from 1 day after injury to weeks later. Late mortality is usually attributed to infection and multiple organ failure.

BIOMECHANICS AND CLASSIFICATION OF TRAUMA

Injury is categorized as either **penetrating** or **non-penetrating (blunt).** In penetrating trauma (or wounds), the injury is produced by crushing and separation of tissues along the path of the penetrating object. In blunt trauma, the injury is produced as the tissues are compressed during impact. Changes in speed (acceleration or deceleration) have a main role in biomechanics of the blunt trauma. In blunt trauma a

special place takes so called **katatrauma**, which are injuries produced by falls from heights.

Traumas may also be divided **according to the character of damage agent** into: mechanical, thermal, chemical, biological, radiological, electrical, psychic traumas, barotraumas – commonly due to the blasting wave, etc.

Trauma may be received under **production** (manufacture, agriculture) **and non-production conditions** (car crash accidents, sport trauma, child trauma etc.) Physicians are particularly concerned about **iatrogenic injuries** – are those, which are got during curative or diagnostic procedures (for example, urethral injury during bladder catheterization).

Traumas can also be classified into **superficial injuries** – contusions and wounds of soft tissue, muscular ruptures, dislocations; and **injuries of internal organs**.

If only one organ was damaged this type of injury is called **isolated** trauma. A damage of two and more organs from one anatomic system results in a **multiple** trauma (for example, simultaneous damage to the liver and spleen, fracture of the femur and humerus). Injury of organs from different anatomical systems is classified as **associated** or **polytrauma** (brain concussion, ribs fracture and ruptured spleen). In addition, injuries caused by the action of two or more etiological factors (for example, mechanical and thermal) produce **combined** trauma.

TRAUMATIC DISEASE

Traumatic disease is a breakdown of the human body functions and consequence of its compensatory reactions, developing after severe injuries. There are **4 periods** of traumatic disease:

(1) The period of traumatic shock lasts from several hours to 2 days and it is caused by primary structural damage and acute blood lost;

(2) The period of early manifestations (from 2 to 10 days). Massive resorption of tissue toxins into the blood stream (metabolites of tissue necrosis) and bacterial toxins (produced by microorganisms from infected wound) are produced in this period. Clinically, acute toxemia is characterized by high fever over 38°C, in contrast to the period of shock, when core hypothermia is common;

(3) The period of late disturbances is characterized by dystrophic and sclerotic processes in the inner organs (kidneys, liver), ankyloses, contractures, etc.;

(4) The period of convalescence may last for months and years. However, approximately 60% of patients who suffered from traumatic disease become handicaps.

HEAD INJURIES

Brain injury is the most common cause of death in trauma. The injuries are usually a result of blunt trauma.

Traumatic injury to the brain involves a **(1) Primary brain injury** that occurs on blow and leads to disruption of brain substances and blood vessels. In addition, **(2) Secondary brain injury** may result from hypoxia, hypotension, the effects of increased intracranial pressure, and altered cellular biochemical processes.

Primary examination begins with palpation of the skull and the head to identify hematomas, lacerations, and fractures. Scalp lacerations can cause significant blood loss and should be closed with a full-thickness running suture to provide hemostasis. The findings of ecchymosis over the mastoid

process, otorrhea, rhinorrhea, and periorbital ecchymosis (raccoon's sign) often indicate basilar skull fracture.

Neurological examination includes determination of general and focal signs. Besides hypertension, hyperthermia, bradycardia and bradypnoe, headache and dizziness, nausea and vomiting, retrograde amnesia (inability to remember events before trauma), sleepiness and depression, loss of consciousness and neurologic coma refer to general signs. **Glasgow Coma Scale,** which is based on three easily defined parameters: (1) Eye opening, (2) Verbal response, (3) Motor response; has become an international standard used to assess level of consciousness after head injury.

In head-injured patients' **focal signs** are the following: hemiparesis (weakness of voluntary movements) or paralysis (loss of muscle function), aphasia, loss of sensitivity, anisocoria, and loss of light reflex.

Specific types of head injury are:

(1) **Skull fractures** are divided into linear, depressed, open, and basal skull fractures. Are common injuries, but do not, by themselves, cause neurologic disability. Many severe brain injuries occur without skull fractures, and many skull fractures are not associated with brain injuries.

(2) **Concussion** – it is a relatively mild form of brain injury, accompanied by only a brief loss of neurologic function (loss of consciousness and retrograde amnesia).

(3) **Diffuse axonal injury** often called brain stem injury. It is similar to severe concussion and is characterized by prolonged coma.

(4) **Cerebral contusion** – is a focal injury of brain. Contusion can occur beneath the area of impact (coup contusions) or in the areas remote from impact (countercoup

contusions). The contusion itself may produce focal neurologic deficit.

(5) **Cerebral compression** is caused by intracranial hematomas: subdural, epidural, or intracerebral. Neurologic signs may differ due to great variations in location, size, and rapidity of bleeding. Its clinical picture always includes an association of general and focal signs. In case of hematoma, little can be done therapeutically to change the magnitude or location of the primary injury after it has occurred, and the main method of treatment remains immediate surgical intervention.

CHEST INJURIES

Chest injuries are common after trauma and are frequently severe. Primary inspection of a patient should include details of the circumstances of injury. Common complains are chest wall pain, dyspnea and weakness. Physical examination begins with the inspection to detect the presence of contusions, penetrating wounds, and asymmetry of the chest. Breathing may be superficial and accelerated, with unilateral impairment of the chest wall excursion. Gentle palpation of the chest wall may reveal a localized pain, areas of instability, crepitus of rib fragments.

To classify an injury, the **thorax is divided into four anatomic zones**: (1) Chest wall; (2) Pleural space; (3) Pulmonary parenchyma; and (4) Mediastinal structures.

(1) Injuries to the chest wall. Rib fractures are the most common and often significant injuries of the thoracic cage, and more often caused by blunt trauma. Pain experienced on motion results in splinting of the thorax. A palpable and/or visible deformity suggests rib fractures. Localized pain, tenderness on palpation and crepitus are present.

The most severe chest wall injury is **flail chest**, in which a "flail" segment of the wall does not have bony continuity to the rest of the rib cage. Clinical signs include abnormal instability of the segment, crepitus, as well as the presence of paradoxal motion (with inspiration the chest wall segment moves inwardly, and outwardly – with expiration). Breath excursion is limited seriously, and leads to hypoxia.

Sternal fractures are frequently associated with a significant blow to the anterior chest. The diagnosis of sternal fracture is made by palpation of the sternum (pain, deformity and crepitus). A lateral chest radiograph reveals sternal fractures and the degree of posterior displacement.

(2) Pleural space injuries include various types of pneumothorax and hemothorax.

Simple (closed) pneumothorax is the presence of air in the pleural space (Figure 26A). Air may collapse lung tissue. The patient with pneumothorax has dyspnea, decreased breath excursion of the affected hemithorax. Percussion of the chest shows hyperresonance, breath sounds are usually decreased. Simple pneumothorax requires tube thoracostomy when it is large enough to be seen on plain chest radiograph, to prevent further complications (such as hypoxia, shock, or tension pneumothorax).

Tension pneumothorax – this condition develops when a "one-way valve" air leak occurs either from the lung or through the chest wall defect (Figure 26B). Air is forced into the thoracic cavity without any means to escape, collapsing completely the affected lung. The mediastinum and trachea are displaced to the opposite side, interfering with venous return. Tension pneumothorax is identified by severe respiratory insufficiency, unilateral absence of breath sounds, distended neck veins, and diffuse cyanosis. Tension

pneumothorax requires immediate decompression with large-bore needle. This maneuver converts the injury to a simple pneumothorax.

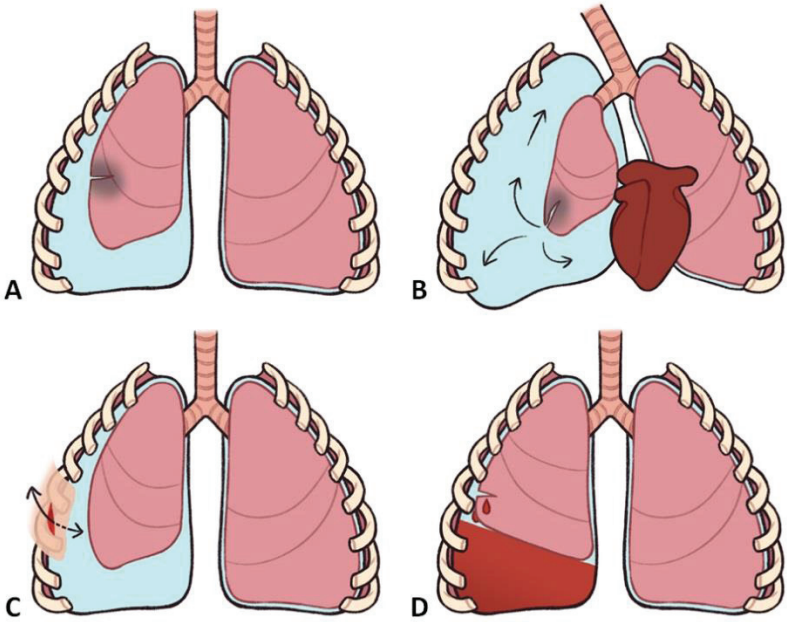

Figure 26. Pleural space injuries: A – Closed pneumothorax; B – Tension pneumothorax; C – Open pneumothorax; D – Hemothorax.

Open pneumothorax or sucking chest wound, is an uncommon injury that produces a large chest wall defect (Figure 26C). The defect allows equilibration of intrathoracic and ambient pressures, leading to collapse of the lung. The diagnosis can be made on simple inspection of the chest and hearing the flow of air through the wound.

Hemothorax is the accumulation of blood in the pleural space (Figure 26D). Patients complain of dyspnea. Physical examination reveals decreased breath sounds and dullness on

percussion of the injured side. The treatment of hemothorax begins with tube thoracostomy to evacuate the blood and reexpand the lung. Only patients with massive hemothorax or persisting bleeding need urgent thoracotomy.

Caked or clotted hemothorax – is identified when pleural cavity is filled by clots, and the lung cannot be expanded despite the adequate, large-bore tube thoracostomy.

(3) Pulmonary parenchymal injuries include pulmonary contusion, laceration, hematoma, and pneumatocele.

(4) Mediastinal injuries.

Tracheobronchial injuries. Patients complain of dyspnea, cough, or hemoptysis. Physical examination reveals subcutaneous emphysema due to leakage of air through traumatic defect. Diagnosis is definitively confirmed by bronchoscopy.

Cardiac tamponade – is accumulation of blood into the pericardial sac, which restricts cardiac activity and interferes with cardiac filling. The classic Beck's triad consists of muffled heart tones, decline in arterial pressure, and jugular venous distention. A high central venous pressure (over 20-25 cm of the water column), an enlarged cardiac shadow, and blood on pericardial aspiration are diagnostic.

Traumatic aortic rupture is associated with a very high mortality. The most common site is the aortic arch, just distal to the origin of the left subclavian artery. Specific symptoms include severe chest or back pain, upper extremity hypertension, and asymmetry of pulses in the upper and lower extremities (pseudocoarctation).

Traumatic diaphragmatic hernia – is the rupture of diaphragm with displacement of abdominal organs into the pleural cavity, which is associated with lung collapse, and mediastinum shift to the opposite side. The main symptoms

are dyspnea, progressive cardiovascular failure, dullness on percussion, decrease of breath and appearance of peristaltic sounds over the left chest.

Esophageal injury. Most penetrating injuries (wounds) of the esophagus arise from the lumen; many of them are iatrogenic by nature. The main causes include: esophagoscopy, esophageal dilatations, pressure injuries produced by Blakemore tube, chemical burns, and surgical procedures. Early symptoms include chest pain and dysphagia, presence of blood in nasogastric aspirate. Late findings include subcutaneous emphysema, and fever.

ABDOMINAL INJURIES

Abdominal trauma is classified into **(1) Blunt** and **(2) Penetrating** (wounds). Additionally, blunt abdominal injuries are divided into 2 types: (1) Trauma with injuries of the abdominal wall and (2) Trauma with injuries of the inner organs. Wounds are divided into: (1) Non-penetrating wounds (when a wound channel does not affect the parietal peritoneum), and (2) Penetrating wounds: (a) without abdominal organs injuries and (b) with abdominal organs injuries (Figure 27).

In abdominal trauma the objective of physical examination is to identify rapidly the signs of inner organs injuries. Precise definition of specific organ injury is unnecessary, rather should be established that there are intra-abdominal injuries and surgical treatment is indicated.

To better understand the symptomatology of trauma, organs of the abdominal cavity can be divided into **solid and hollow**. The liver, spleen, pancreas, kidney, and major vessels refer to solid organs; the stomach, duodenum, small intestine, and colon refer to hollow organs. **Two main clinical**

syndromes may occur in abdominal trauma with inner organs damage: **hemorrhagic** (most common for injuries of solid organs) and **peritoneal** (common for injuries of hollow viscera). They association is also possible.

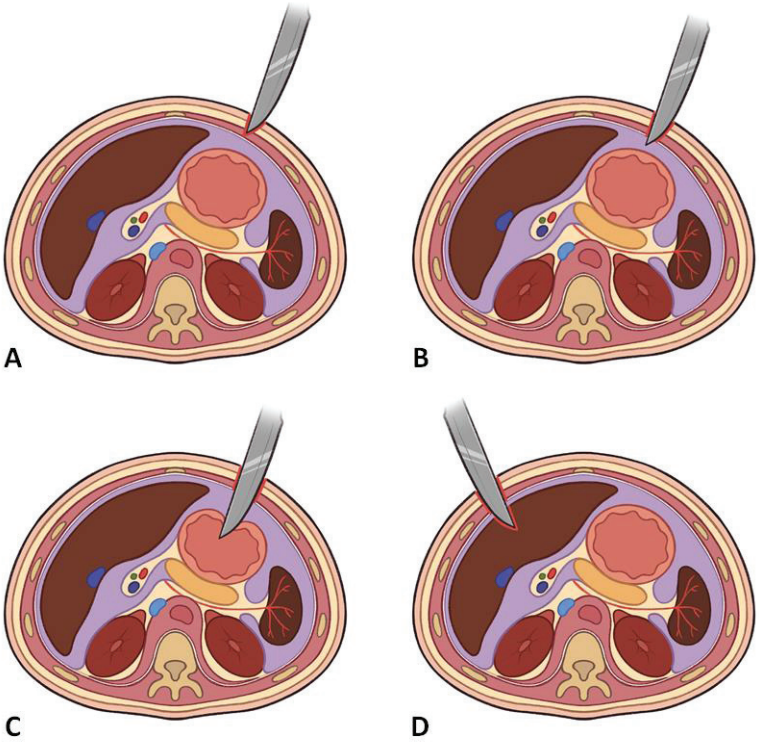

Figure 27. Types of abdominal wounds: A – Non-penetrating wound (parietal peritoneum is not injured); B – Penetrating abdominal wound without injury of internal organs; C – Penetrating abdominal wound with injury of hollow viscus (usually manifested with peritoneal syndrome); D – Penetrating abdominal wound with injury of parenchymatous organ (usually manifested by hemorrhagic syndrome).

Patients with abdominal organs injuries may present with abdominal pain, weakness, dizziness, and hemodynamic

instability. The abdomen should be inspected for contusions, and penetrating wounds. Abdominal palpation may reveal tenderness and peritoneal signs. The purpose of rectal examination is to study integrity of walls, and search for blood. In pelvic injuries the rectal wall should also be palpated for fractured bony elements.

Local exploration reveals if the wound is penetrating. Diagnostic paracentesis and peritoneal lavage is a critical step in the evaluation of blunt abdominal trauma (presence of blood and enteric contents in peritoneal cavity). A hemodynamically stable patient can undergo radiographic and laboratory studies, ultrasound and computed tomography scan, and also laparoscopy.

Patients with gunshot abdominal wounds do not require any local wound exploration and additional diagnostic tests. According to statistical data, 90-95% of them are intraabdominal injuries and, hence, they have indications for urgent operation.

FRACTURES AND DISLOCATIONS

Fractures and dislocations are the most common musculoskeletal traumatic injuries of the limbs and are observed in more than 70% of patients with trauma.

A **fracture** is defined as a linear deformation or discontinuity of bone produced by forces that exceed the ultimate strength of the material. Usually bone injuries are associated with external forces – **traumatic fractures**. Many diseases can gradually lead to osteoporosis, demineralization and, weaken a bone and result in **pathologic fracture**.

Classification of fractures. Fractures may be either closed or open. Fractures can affect different anatomic zones of the bone: diaphyseal, metaphyseal and epiphyseal fractures.

Fractures are also classified according to the position, number, and shape of the bone fragments: transverse, oblique, spiral, “greenstick” (common among children), compression, depressed, impacted, comminuted, avulsion. There are some types of displacement of bone ends – edgewise, lengthwise, angular and rotational.

Symptomatology. Deformity or unnatural position of the limb, false or unnatural motion over injured extremity and a grating sound (crepitus) during palpation of the limb are **absolute** symptoms of fracture. Pain, shortening of injured limb, difference in size or shape, ecchymosis on the skin and swelling are **relative** symptoms of fracture. The arterial pulses distal to the fracture site and capillary refill should be assessed.

A **dislocation** is the displacement of a bone end from its articular surface, sometimes with associated tearing of the ligaments. Dislocations are **classified** in traumatic and pathologic; acute (<48 hours), long-standing (3-4 weeks) and chronic (more than 4 weeks). Irreducible dislocation cannot be reduced without surgical intervention due to interposition of soft tissues between articular surfaces. Habitual (relapsing) dislocation - develops as a result of severe damage of ligaments and consequently chronic instability of joint.

Symptoms of dislocation are pain, loss of motion of the joint, deformity and moderate to severe swelling.

Prehospital care for fracture and/or dislocation includes application of splint (medical device for immobilization), dressing (if open), adequate analgesia and intravenous fluid replacement. An attempt to reduce the fracture and dislocation should not always be made. The diagnosis of fracture and dislocation should be confirmed with biplane X-ray. Hospital treatment of fracture and dislocation consists of reduction and immobilization.

XIX. SEMIOLOGY OF VASCULAR DISEASES OF EXTREMITIES

NOSOLOGY OF DISEASES

Vascular diseases of extremities can be divided into arterial pathology, venous and lymphatic disorders.

Diseases of arteries in the majority of cases are manifested clinically as a syndrome of arterial insufficiency, caused by the obstruction of arterial lumen (ischemic syndrome), and a syndrome of aneurysmal dilatation of the artery. Arterial insufficiency can develop suddenly (in case of arterial trauma, arterial thrombosis or embolism) – referred to as acute ischemia, or it may progress gradually (in case of peripheral arterial disease caused by atherosclerosis, thromboangiitis, or nonspecific aortoarteriitis) being termed in these cases as chronic ischemia.

In case of **acute limb ischemia**, the term “thrombosis” used to describe the situation when intraluminal blood clot forms *in situ* – at the site of endothelial injury provoked by complicated atherosclerotic plaque, inflammation of arterial wall or mechanical damage. Thrombus may obstruct the arterial lumen completely or partially. The state of hypercoagulability contributes to the development of thrombosis. The term “embolism” (from the Greek word *embolos* – plug) means the complete obstruction (occlusion) of the arterial lumen, caused by the material passing from the upstream parts of cardiovascular system – thrombotic masses, fragments of arterial plaque or tissue of a cardiac tumor. The blood clots originating from the left hart chambers in patients with atrial fibrillation, myocardial infarction and artificial heart valves represent the most frequent cause of peripheral arterial embolism.

Chronic limb ischemia usually develops as a result of the progressive narrowing (arterial stenosis) and subsequent complete obliteration of the arterial lumen with atherosclerotic plaque.

Arterial aneurysm is referred to as an 50% increase in the of normal diameter of the artery or an eccentric bulging of the arterial wall. The enlargement of artery can be caused by traumatic injury – so-called false aneurysm or be a result of inflammation and degenerative changes of the arterial wall – true or atherosclerotic aneurysm. False aneurysms are mostly saccular in shape and the wall of aneurysm sac is represented by fibrotic tissue forming around primary hematoma. Atherosclerotic aneurysms are usually spindle-shaped while the wall of aneurysm preserves the structure of a normal artery: intima, media and adventitia. True aneurysm of the popliteal artery and abdominal aortic aneurysm are most frequently diagnosed.

Diseases of venous system are determined by disturbances of blood outflow from extremity. Impairment of venous return can develop acutely as a result of acute vein thrombosis or chronically – causing chronic venous insufficiency.

Acute vein thrombosis may affect superficial veins – usually the saphenous veins in patient with lover limb varicosities or deep veins – popliteal, femoral, iliac, subclavian. It should be noted, that in contrast to deep vein thrombosis the thrombosis of superficial veins is not associated with significant disturbances of venous circulation in affected extremity. The most threatening complication of vein thrombosis is a pulmonary artery embolism.

Chronic venous insufficiency is caused by incompetence of venous valves leading to the retrograde flow of the venous blood (“venous reflux”), chronic obstruction of major veins

(usually post-thrombotic) or by their combination. Reflux represents the main pathogenic mechanism in patients with varicose veins of lower limbs while secondary venous obstruction is characteristic for patients who suffered with deep vein thrombosis in the past. Both mechanisms are responsible for venous hypertension in the distal part of the limb, fluid extravasation, migration of polymorphonuclear cells into the soft tissue and development of inflammation and hypoxia.

Diseases of lymphatic vessels of extremities (congenital anomaly, trauma or inflammatory obliteration) lead to the impairment of lymph outflow and as a result to the development of lymphedema.

COMPLAINTS AND HISTORY

Complaints typical for patients with vascular diseases of extremities are pain, muscular weakness and fatigability, sensory changes, restriction of mobility, sense of pulsation, heaviness in extremity, edema, and muscular cramps.

Pain. Sudden severe sharp pain in extremity is typical for acute ischemia. Most acute – dramatic onset is especially characteristic for arterial embolism. Pain is constant, focused in the distal part of extremity and not influenced by changes of posture.

In the early stage of chronic ischemia, patients develop pain only during walking. After some minutes of rest, the pain disappears spontaneously and reappears again after passing the same distance (200-500 meters in the mild cases and less than 200 meters – in severe cases). Most frequently pain is localized in the calf muscles and described by patient as a “cramp”. Rarely pain is precepted in the hip muscles and buttocks. This symptom is named **“intermittent claudication”**,

that basically represents a misnomer because patient not actually limps. While arterial obliteration progresses the "pain-free" walking distance becomes shorter, reaching several dozens of meters.

In the advanced stage of chronic ischemia pain is localized mostly in the forefoot, becomes constant, very severe or intolerable, increasing at nighttime and deprives the patient of sleep. Dependent position of affected limb reduces the pain slightly because of moderate increase in blood inflow. For this reason, patients often sleep sitting up in the chair or hanging leg down from the bed. This symptom named **"rest pain"** testifies a forthcoming gangrene of extremity – terminal stage of chronic ischemia.

Deep vein thrombosis is accompanied by moderate or minimal pain. The pain is constant, localized in the calf, popliteal fossa and along big vessels. In case of superficial thrombosis of varicose veins pain is perceived by patient in the site of thrombus location and secondary inflammation of the soft tissue. The pain is not a characteristic clinical symptom of chronic venous insufficiency.

Neurologic disturbances. Sensory changes can be represented by **paresthesia** (unusual sensation of pricking, burning), decreased tactile sensation or **complete sensory loss**. Patients with vascular pathology can also complain to motor disturbances: restriction (**paresis**) or absence (**paralysis**) of active movements in extremity. It should be beard in mind, that neurological abnormalities are specific for acute ischemia, indicate the direct threat to limb viability and mandates the revascularization of extremity without any delay.

Limb edema – is a typical complain in case of many vascular and systemic diseases. Swelling of only one limb is more

characteristic for vascular pathology. Marked edema, progressing during a day or two and involving the entire limb is specific for ilio-femoral deep vein thrombosis. Limb swelling caused by chronic venous or lymphatic insufficiency increases gradually – during months or years, affects the foot and lower leg, becomes worse after prolonged standing and improves with limb elevation. Foot edema can be observed also in advanced stages of acute and chronic limb ischemia.

Feeling of pulsation along the trajectory of the major arteries of limb may be a sign of an arterial aneurysm.

Feeling of heaviness in the lower limbs ("heavy legs") is typical for early stages of chronic venous insufficiency.

Anamnesis. It is necessary to analyze the time and circumstances of the onset of disease, the sequence of symptoms development and the treatment actions performed so far. Acute ischemia can be provoked by trauma to the limb, dehydration, abandoning or change in the regimen of taking antithrombotic and antiarrhythmic drugs. The development of venous thrombosis is often preceded by prolonged immobility, trauma or surgery, pregnancy and use of hormonal contraceptives. The following data should also be analyzed during history taking of a patient with vascular diseases of extremities: heavy smoking, coronary artery disease, cardiac infarction and arrhythmia (atrial fibrillation), wounds in the region of big arteries, surgical interventions.

INSPECTION

In case of **acute ischemia,** the extremity is pale, the skin is mottled ("marmoreal" limb), and subcutaneous veins are collapsed. Active movements in the distal parts of extremity (flexion, extension and separation of toes / fingers) are limited

or impossible. The flexion contracture of extremity develops in the advanced stage of acute ischemia.

Chronic ischemia is associated with atrophy of calf muscles, loss of hair growth and thickening of the toenails. In stage of "rest pain" the color of the skin is pale, purple or cyanotic. Changes in the foot color, caused by chronic limb-threatening ischemia, can be demonstrated by so called **Buerger-Ratschow's sign**. Within a minute after elevation of the affected extremity the foot becomes deadly white and with dependent position of the limb the foot slowly becomes dusky red or blue as blood returns to the tissues.

Both acute and chronic ischemia, invariably lead to the development of gangrene if untreated. The term of **gangrene** denotes the specific type of necrosis which is characterized by:

(1) It develops only in the tissue which is in contact with external environment;

(2) Affected tissue becomes black-colored;

(3) It affects an organ or an anatomic region as a whole.

There are two types of limb gangrene – dry and damp (moist) gangrene.

Dry gangrene develops slowly and is characterized by dehydration of affected tissue, decrease of tissue volume, clearly outlined borders of necrosis (so called "demarcation line"), absence of infection and general reaction of the organism. **Damp gangrene** is characterized by marked edema, exfoliation of epidermis and formation of bullae, absence of "demarcation line", fast spreading, association of infection and severe intoxication.

In case of **peripheral arterial aneurysm,** the inspection reveals presence of pulsatile mass.

Deep vein thrombosis is associated with marked edema of thigh and shin, moderate cyanosis of distal parts of extremity,

extension of subcutaneous veins. The most severe form of deep vein thrombosis is called **“blue phlegmasia”** (*phlegmasia cerulea dolens)* and can result in damp venous gangrene.

In case of **superficial veins thrombosis,** the inspection reveals dilated and tortuous veins with skin hyperemia above them. Unlike non-thrombosed veins, venous trunks and clusters filled with thrombus do not collapse in recumbent position.

In **chronic venous insufficiency** the appearance of leg is characterized by presence of varicose veins, edema of malleolar region and brown-colored spots on the internal surface of the shin (hyperpigmentation), venous ulcer. It should be remembered that examination of patients with venous diseases should be performed in upright position of the patient.

The term **trophic ulcer** denotes the defect of soft tissue which has not any tendency to heal during 6 weeks and more. Typically, **venous trophic ulcer** develops on the medial surface of the lower third of the shin and usually is preceded by the so called “white atrophy” – zone of affected skin similar to the drop of stearin. Venous ulcers gradually increase in size and may involve the entire leg circumference. The borders of the ulcer are irregular, surrounding skin is chronically inflamed (eczema), the ulcer base is friable and depending on phase of inflammation is covered with pus, fibrin or granulations. Venous ulcers are characterized by large volume of exudate. **Arterial (ischemic) ulcers** usually are small, painful and typically localize on the toes or in interdigital spaces. Ischemic ulcers are covered by black-colored necrotic eschar or a dens layer of fibrin, whereas the granulation and exudation are absent.

Lymphedema is usually associated with significant increase of limb volume, visible limb deformity and marked hypertrophic skinfolds (“elephant leg” or “elephantiasis”). The skin frequently affected by eczema and other dermatological conditions.

PALPATION

Palpation possess the high diagnostic value in case of peripheral arterial diseases, because the **pulse** is absent or diminished distally to arterial occlusion or stenosis (Figure 28).

In patients with arterial disease of **lower limbs** pulse should be palpated on:

- Femoral artery (immediately below the middle of inguinal ligament);
- Popliteal artery (with both hands in popliteal fossa);
- Posterior tibial artery (behind the medial malleolus); and
- Dorsalis pedis artery (on the dorsum of the foot, in proximal part of first metatarsal space).

At the level of **upper limb**, the pulse is palpated on:

- Axillary artery (in the axillary fossa, at the level of humeral head);
- Brachial artery (medial to the biceps brachii muscle by applying pressure to the medial edge of the humerus or medial to the biceps tendon in the antecubital fossa);
- Radial artery (on the ventral surface of the wrist, between the flexors tendon and radial bone); and
- Ulnar artery (on the ventral surface of the wrist, between the flexors tendon and ulnar bone).

Palpation of the arteries should be performed carefully, slowly, in a warm room environment and requires a certain experience from the examiner. The most difficult is the palpation of the popliteal artery, which lies deep in the

popliteal fossa. It should also be remembered that in approximately 10% of healthy individuals, the pulse on the dorsalis pedis artery cannot be palpated due to anatomical variability of the vessel.

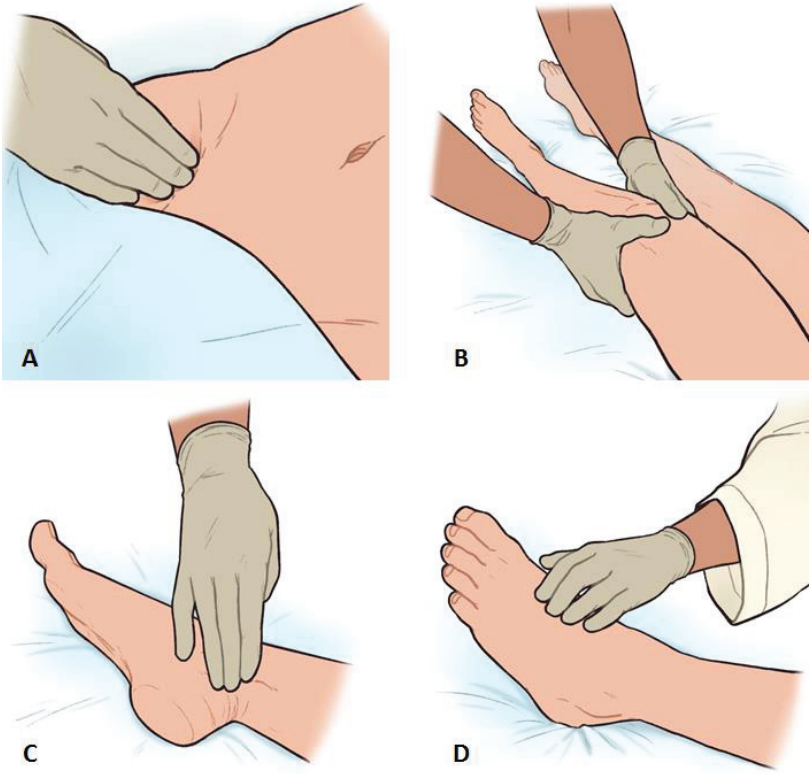

Figure 28. Points and technique of pulse palpation on lower limb: A – Femoral artery; B – Popliteal artery; C – Posterior tibial artery; D – Dorsalis pedis artery / anterior tibial artery.

On palpation, the **presence or absence of a pulse** is noted, as well as the **degree of its filling (amplitude)**. The absence of a pulse indicates occlusion of the more proximal arterial segment, its weakening – indicates stenosis, and increased

pulsation at a certain point suggests a possible aneurysmal dilatation.

In addition to palpation of the pulse in patients with suspected limb ischemia, the skin temperature of the affected limb, sensorial function and range of motion are evaluated.

In case of acute ischemia, the **"6P" complex of symptoms** can be determined:

(1) Pain,
(2) Pallor,
(3) Pulselessness,
(4) Poikilothermy ("perishing cold"),
(5) Paresthesia,
(6) Paralysis.

Pulsatile mass is a symptom of arterial aneurysm. In case of arterio-venous fistula (pathologic communication between an artery and a vein caused by trauma or malformation) the **systolic thrill or "purr of cat"** can be felt. The compression of fistula results in decrease of heart rate up to 10-15 beats/min (positive **symptom of Branham**).

In case of superficial venous thrombosis, careful palpation can reveal painful infiltrate along the affected vein and dense clots into the venous lumen. The thrombosed varicose vein will not collapse on compression with a finger. For diagnosis of deep vein thrombosis two symptoms can be used: tenderness during squeezing of the calf muscles in antero-posterior direction (**Mozes sign**) and pain elicited by passive dorsiflexion of the foot (**Homans sign**). However, the diagnostic precision of these clinical tests is extremely low.

Hackenbruch test (cough test) and **Trendelenburg test** (tourniquet test) can be used to determine the reflux at saphenofemoral junction and along the great saphenous vein. Permeability of deep veins is indirectly assessed using **Delbet-**

Perthes test. Currently these tests are performed exceptionally rare due to the widespread use of duplex ultrasound for investigation of the peripheral venous system. In advanced stage of chronic venous insufficiency palpation reveals hardening of subcutaneous fat on the lower leg (lipodermatosclerosis) as a result of chronic inflammation.

Palpation can distinguish venous and lymphatic edema. Inability to pinch a skin fold at the base of the second toe is a symptom characteristic for lymphedema (**Stemmer sign**).

AUSCULTATION

Normally there are no any sounds above peripheral blood vessels. Arterial stenosis or aneurysm both lead to the appearance of systolic bruit. Shunting of blood through arterio-venous fistula leads to the appearance of permanent **systolic-diastolic bruit ("machine sound")**. There are not any sounds in case of complete arterial obstruction.

In patients with suspected peripheral arterial disease of lower limbs the auscultation of the arteries is performed along the imaginary line joining the point of femoral artery pulsation in the groin and umbilicus (point of projection of aortic bifurcation). At the upper limb the systolic bruits can be heard in supraclavicular fossa.

DETERMINATION OF ANKLE-BRACHIAL INDEX

In order to assess the severity of ischemia in patients with peripheral arterial disease, the ankle-brachial index (ABI) should be measured. For this purpose, a tonometer and a hand-held Doppler with a frequency of 8-10 MHz are used (Figure 29).

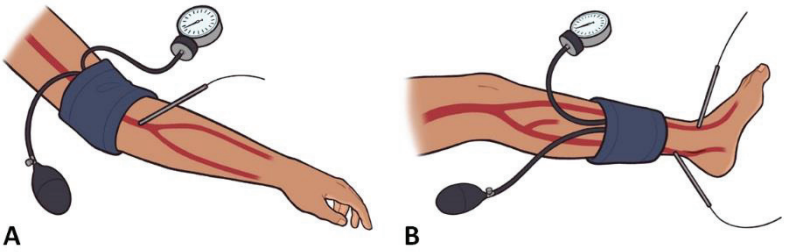

Figure 29. Technique of blood pressure measurement using a manual pressure cuff and a continuous wave Doppler: A – Determination of systolic blood pressure at the distal brachial artery (denominator of ankle-brachial index); B – Determination of highest systolic pressure at the dorsalis pedis artery or posterior tibial artery (numerator of ankle-brachial index).

- In the supine position of the patient, the cuff of the sphygmomanometer is applied on the shoulder and, using the hand-held Doppler, a clear arterial signal is found on the brachial artery in the antecubital fossa. The cuff is inflated until the sound signal disappears and, during slow deflation the pressure that corresponds to the moment of restoration of blood flow is noted.
- Then a pressure cuff is applied in the lower third of the leg and the systolic pressure on the dorsalis pedis artery and posterior tibial artery is determined in the same way.
- To calculate the ABI, the systolic pressure at the foot is divided by the systolic pressure determined at the brachial artery. ABI values greater than 0.9 and less than 1.4 are considered normal. In case of severe ischemia, the ABI decreases to less than 0.5.

XX. DIABETIC FOOT

INCIDENCE AND DEFINITION

Diabetes mellitus is a severe metabolic disease with a steady trend of increasing incidence worldwide. It is expected that in the following years there will be about 500 million people with in the world living with diabetes. Approximately 10-15% of diabetic patients develop plantar ulcers during their life. In diabetics the risk of amputation is fifteen times higher in comparison with non-diabetic population. Up to 70% of all amputations performed worldwide are done in patients with diabetes mellitus. The prognosis of the disease is unfavorable – about half of patients who have undergone a major amputation for diabetic foot lose the contralateral limb or die during the next few years.

According to the WHO definition of 1999, **diabetic foot** is a foot of diabetic patients with ulceration, infection and/or destruction of the deep tissues, associated with neurological abnormalities and various degrees of peripheral vascular disease in the lower limb.

CAUSES AND FORMS

Disturbed metabolism of glucose provokes at least **3 negative effects** upon the lower limbs:

(1) **Atherosclerosis of peripheral arteries** begins early in patients with diabetes mellitus – at a young age, occlusions and stenosis of the vessels develop more quickly and involve a large number of arteries, including mostly the calf and foot vessels. Occlusive and stenotic arterial lesions lead to the decreased perfusion of affected limb and development of chronic ischemia.

(2) In diabetes affection of the peripheral nervous system includes somatic and autonomic neuropathy. Motor deficit as a part of **somatic neuropathy** results in weakness of plantar muscles and development of osteoarticular deformations but sensorial deficit leads to hypoesthesia or anesthesia of the foot skin. Sometimes neuropathy may cause pain in the leg.

(3) **Dysfunction of autonomic nervous system** is responsible for decreased sweating, hyperkeratosis and formation of calluses in the pressure points. Dry skin may crack with consequent infection. Autonomic neuropathy is also responsible for loss of capillary tone, venous stasis, opening of the arterio-venous shunts, and increased osteolytic activity resulted in osteoporosis.

All above mentioned mechanisms are considered to be the cause of diabetic foot development. Basing on prevalence of one or another pathogenic mechanism **3 forms of diabetic foot** are distinguished:

(1) **Neuropathic form** (diagnosed in approximately 70% of cases);

(2) **Ischemic form** (10%); and

(3) **Mixed** or **neuro-ischemic form** (20%).

In the development of diabetic foot and especially plantar ulcers the role of **disturbed plantar biomechanics** is important. Flexor contracture of the toes (clawed digits), declined metatarsal heads and midfoot collapse (Charcot foot, “rocker bottom” foot) lead to the excess of pressure in the points of bony prominence and subsequent soft tissue necrosis. The critical level of plantar pressure, which results in formation of diabetic ulcer, is considered to be 7 kg/cm^2. Plantar callus serves as a foreign body focusing and concentrating pressure in a small area.

All forms of diabetic foot are frequently associated with infection. The high frequency of **infectious complications** can be explained by a number of factors:

(1) Open wounds and tissue defects (ulcers, cracks) act as entry gates of infection;

(2) The lack of protective pain sensation predisposes to the development of traumatic foot injuries and delay of the diagnosis;

(3) Prolonged hyperglycemia results in compromised immunity – suppression of phagocytosis, decreased function of monocytes and compliment system;

(4) Arterial obliteration leads to tissue ischemia and hypoxia, which contributes to the multiplication and spread of pathogenic microorganisms.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Patients with diabetes require a prophylactic examination of the feet (diabetic foot screening) at least once a year. The examination should include an analysis of complaints and anamnesis, a thorough visual inspection of the foot (including interdigital spaces), palpation of the foot pulses and assessment of skin sensation.

There are some groups of **complaints** important for the diagnosis of diabetic foot: ischemic related complaints (severe pain during the night time and after leg elevation); neuropathic related ones (paresthesia, unusual sensation, decreased sensibility); infection related complaints (pulsatile pain, fever).

During the **examination**, physician is looking for foot deformities, calluses, skin lesions, trophic ulcers and gangrene. If pathological findings are noted, the exact location and size

of the tissue defect are recorded (it is recommended to take a photograph the affected areas).

Absence of pulse on the foot arteries is highly suggestive for peripheral arterial disease with chronic ischemia and serves as an indication for instrumental assessment of foot perfusion.

To confirm the **sensory loss**, the Semmes-Weinstein 10 g monofilament test should be performed. Sensibility to touch with standardized monofilament is tested on the plantar surface of the toes and in the region of metatarsal bones heads. The time of pressure with the monofilament is one second. It is important not to ask the patient about sensations during the test but to instruct him before the test to report any touch when he feels it. If sensation is absent at least in one-point – neuropathy should be diagnosed and the patient referred to a neurologist.

The simplest **diagnostic method**, usually used as a screening tool of arterial pathology is systolic pressure determination in plantar arteries by **hand-held Doppler** (technique is described in previous chapter). In case of diabetic foot, the plantar pressure may be false elevated due to medial-calcification of the tibial arteries (so-called Mönckeberg's arteriitis).

Diabetic foot circulation and tissue oxygenation can be assessed by transcutaneous determination of **O_2 pressure on the foot (TcpO2)** and determination of toe systolic pressure by means of **photoplethysmography**. The toe systolic pressure below 30 mm Hg or oxygen pressure less than 30mm Hg may predict non-healing foot lesions (critical tissue ischemia) and the highest risk of amputation. If ischemia is confirmed by any of above-mentioned methods patient will require the urgent

vascular imaging of lower limb arteries (**duplex ultrasound, angiography**).

Bacteriology. There are several pathogens mostly responsible for the development of diabetic foot infection: *Staphylococcus aureus* is the most common and the most virulent microorganism; *Streptococcus β-haemoliticus*; non-clostridial anaerobes *(Bacteroides, Peptostreptococcus)* are characteristic of deep ulcers and severe infection in ischemic foot. In case of long-standing tissue defects, the above-mentioned bacteria are often revealed in association with *Escherichia coli, Proteus mirabilis* and *Pseudomonas aeruginosa*.

Clinical picture of diabetic foot infection includes local (pain, skin redness, edema, induration, fluctuation) and general (fever, nausea, vomiting, tachycardia, disturbed mental state, oliguria) signs.

Various methods can be used for the **confirmation of diabetic foot infection**:

- The simplest method is probing of ulcer or wound bottom with a sterile probe. Finding of direct “probe to bone” contact signifies a 90% probability of underlying osteomyelitis.
- Diagnosis of osteomyelitis of diabetic foot may be confirmed by imaging techniques: biplane X-ray (repeated with 2-3 weeks’ interval), computed tomography scanning with leukocytes labeled with indium (111In) and magnetic resonance imaging. The last method has been considered as a method of choice.
- As with other surgical infections, laboratory markers of inflammation – leukocytosis, increased number of neutrophils, accelerated ESR, high levels of C-reactive protein and elevated procalcitonin have an important diagnostic role.

CLASSIFICATION

In order to take evidence-based clinical decisions and develop an individual treatment plan for a patient with diabetic foot syndrome, the use of specifically designed classifications is mandatory.

Generally, infections of diabetic foot can be divided into:

(1) **Superficial** – which affects the skin and subcutaneous adipose tissue; and

(2) **Deep** with the affection of muscles, tendons, bones and joints.

Internationally recognized classification of diabetic foot infection includes 2 more grades: grade 0 or absence of infection and grade IV – the most severe infection. The diagnosis of grade IV diabetic foot infection is done in any type of foot infection if it is associated with clinical signs of systemic inflammatory response syndrome (SIRS).

The most widely known classification of diabetic foot, based on grade of tissue destruction is **Wagner-Meggitt classification** (Figure 30):

- Grade 0 – absence of foot tissue defect (completely epithelialized ulcer, hyperkeratosis, callus);
- Grade 1 – superficial full-thickness ulcer (not extending through the subcutis);
- Grade 2 – ulcer with exposed tendon or bone without osteomyelitis / abscess;
- Grade 3 – deep ulcer with osteomyelitis / abscess formation;
- Grade 4 – localized gangrene of toes or forefoot; and
- Grade 5 – foot with extensive gangrene.

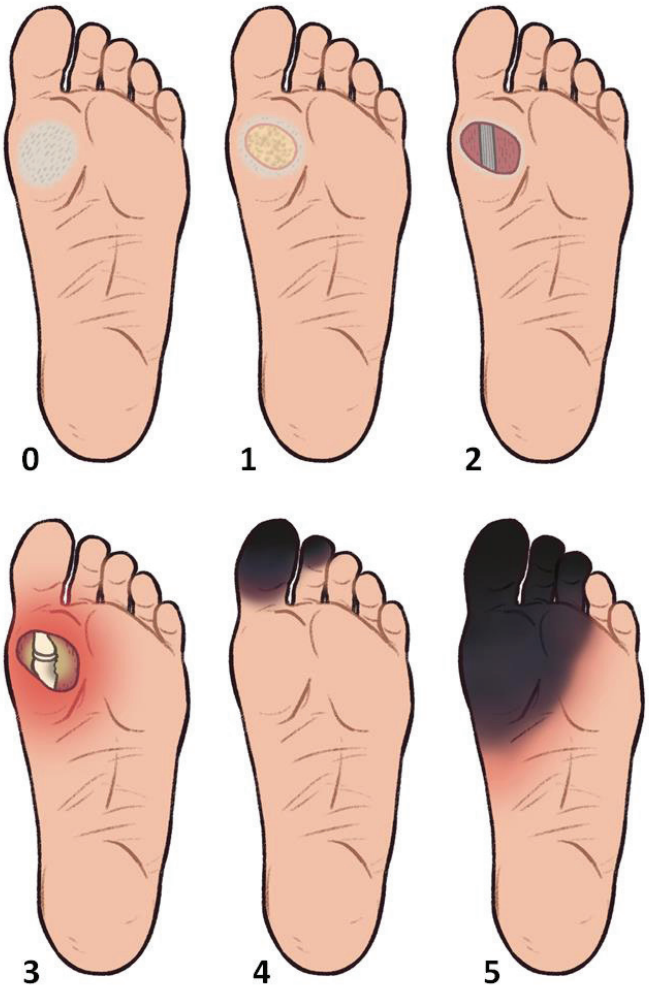

Figure 30. Wagner-Meggitt classification of diabetic foot.

The main disadvantage of the above-mentioned classifications is the isolated description of infection and necrosis, as well as the lack of consideration of severity of ischemia – one of the most important pathogenetic factors of

the disease. To address these shortcomings, more sophisticated classification systems for diabetic foot were developed and implemented in clinical practice: The **University of Texas classification**; **S(AD)SAD classification** (*Size, Sepsis, Arteriopathy, Denervation*); **PEDIS classification** (*Perfusion, Extent, Depth, Infection, Sensation*) and other.

Currently, the **WIfI system** (***W****ound,* ***I****schemia,* ***f****oot* ***I****nfection*) is recognized by the majority of experts in the field as a most appropriate, allowing to quantify three key components: (1) Wound or extent of tissue defect (from the absence of an ulcer to an extensive tissue loss or gangrene, requiring proximal foot amputation); (2) Degree of ischemia (defined by parameters of ankle systolic pressure, ankle-brachial index, toe pressure measured by plethysmography, and transcutaneous oxygen pressure); and (3) Severity of foot infection (ranging from no evidence of infection to infection involving deep anatomical structures and accompanied by systemic reaction).

Basing on these three main criteria and using special tables, the WIfI stage can be determined (from stage 1 – "very low risk of limb loss" to stage 4 – "high risk of limb loss"), reflecting the risk of major amputation of the limb, the likelihood of wound healing and potential benefit from surgical or endovascular revascularization procedures. It is recommended to incorporate the WIfI stage in the clinical diagnosis of all patients with diabetic foot at various steps of medical care.

TREATMENT

Management of diabetic foot is universally complex and depends on the clinical form of disease.

- The primary treatment goal always is an adequate **correction of hyperglycemia** by means of oral antidiabetic drugs and insulins.

- For **prevention of major acute cardiovascular complications** (myocardial infarction, stroke, acute limb ischemia) – the leading cause of mortality among diabetic population**,** the following drugs are prescribed obligatory: antithrombotic drugs (aspirin, clopidogrel), statins (simvastatin, atorvastatin, rosuvastatin) and antihypertensive medication.

- Patients with ischemic and neuro-ischemic form of the diabetic foot require urgent **revascularization**. The revascularization procedures can be done by either endovascular percutaneous transluminal angioplasty with stent implantation or via open surgical by-pass. To date, it is considered the wrong practice to perform a major (above or just below the knee) amputation in a patient with ischemic diabetic foot without prior angiography proving the impossibility of performing an operation to restore the sufficient blood flow to the affected limb.

- The cornerstones of infection control are long term **antibacterial treatment** (cephalosporins, fluoroquinolones, until 2-6 weeks in case of osteomyelitis) and, of course, urgent **adequate surgical debridement**.

- **Local treatment** includes: removing of pus, infected and devitalized tissue, as well as application of moist dressings with chemical antiseptics and proteolytic enzymes. Fragments of tissues removed during surgery are sent to a bacteriological laboratory for isolation of pathogenic microorganisms and determination of its sensitivity to antibiotics.

- Perspective method for the treatment of chronic purulent wounds within the diabetic foot is **vacuum therapy**,

the technique and positive effects of which have been described in the previous sections.

- In patients with diabetic foot ulcers local treatment should be combined with **foot off-load** realized by patient bed rest, plastic orthoses and plaster cast application or using of crutches and wheel-chair.

- The presence of necrotized tissue requires its removing by means of **minor amputations or exarticulations** finalized with the application of primary or secondary sutures. Significant tissue defects without tendency to spontaneous healing require plastic reconstructions: skin grafting, soft tissue transfer (local, distant or free). It is important to note, that all types of plastic surgery have a chance of success only if adequate control of infection and ischemia is achieved.

- **Other treatment modes.** In the last few decades several more methods of diabetic foot treatment have been proposed and extensively studied. They are: local administration of growth factors (PDGF – *platelet derived growth factor*), covering tissue defects with bioengineered skin (Dermagraft – neonatal fibroblasts fixed on biodegradable mesh), systemic introduction of granulocyte-colony stimulating factor (Filgrastim) and larval wound therapy with sterile maggots of *Lucilia sericata*.

Extensive experience accumulated in the treatment of diabetic foot proves that success in this extremely difficult cohort of patients may be achieved only by multidisciplinary or so-called team-approach. The vascular surgeon, plastic surgeon, orthopedic surgeon, endocrinologist, neurologist, cardiologist, food and shoe-specialists should be involved in the treatment of patient.

Prevention of major amputations caused by diabetic foot includes several important measures.

- First of all, it is a good metabolic control with glucose level less than 7 mmol/L or (more sensitive) level of glycosylated hemoglobin HbA1C <7% should be achieved. The level of HbA1C is considered as a gold standard for metabolic control in diabetic foot care.

- Correction of associated risk factors requires lipid control, maintaining of blood pressure not more than 140/90 mm Hg and obligatory smoking cessation.

- High-quality medical treatment should be combined with close follow-up of diabetics, patient education and foot protection by orthopedic footwear.

XXI. BASIC TRANSPLANTOLOGY

Developments of medicine, and especially of surgery in the last time, led to that the majority of diseases are cured completely, while others pass in stable remission. However, there are a number of diseases, such as diffuse cardiac sclerosis, chronic renal failure, diffuse pulmonary fibrosis, liver cirrhosis, which cannot be cured by conservative or conventional surgical methods. In such situations, to avoid patients' death, the only is possible to transplant a healthy organ instead of the affected and not functioning one.

Transplantology is a science that studies the theoretical premises and the practical possibilities of replacing nonfunctional organs or tissues to other organs or tissues, taken from another individual or another part of the same body.

HISTORY

Since early times, the idea of tissue and organ transplantation occupied the imagination of successive generations, and over the centuries have been described numerous attempts of organ transplantation. However, the modern era of transplantology began with perfection of the blood vessels connection technique. Vascular suture was developed by Alexis **Carrel** at the beginning of the XX century. Nevertheless, the problems of reject reactions in case of organs and tissues transplantation were were not yet known.

Ukrainian scientist **Iurii Voronoi** in the 30s of the XX century in Kherson performed a number of kidney transplants taken from deceased donors. Although all the patients died, the transplanted cadaveric kidneys functioned normally for some time. The first successful transplantation of kidney from

a living donor between two identical twins was carried out by **Joseph Murray** and colleges in 1954 in Boston.

In 1959 American scientist **Robert Schwartz** proved that the anti-cancer drug 6-mercaptopurine has an immunosuppressive effect. Its use increased greatly transplant compatibility after transplantation. After that, English professor **Roy Calne** proposed to use another medication – azathioprine for immunosuppression. With these discoveries, the transplantology gained widespread development, and indeed is considered the miracle of modern medicine.

COMMON TERMS

It is necessary to define some general terms that are used in organ and tissue transplantation.

A **donor** is considered to be an individual or an area of the body, which is the source of tissue or organ for transplantation to another person or to another area.

A **recipient** is considered to be an individual or an area of the body, which will receive the extracted tissue or organ.

Transplantation is a surgical procedure of replacement of a damaged organ or tissue with others.

The following **types of transplantation** are distinguished:

- **Autologous transplant (autograft)** – when the donator and recipient is one and the same person;
- **Syngeneic transplant (singraft)** – when the donator and recipient are monozygotic identical twins;
- **Allograft transplant** – transplantation of organs or tissues from one human to another;
- **Xenotransplantation (xenograft)** – when a donor organ or tissues are taken from an animal for transplantation to human.

Depending on the location where the transplanted organ or tissue may be placed, the transplantation is divided into orthotopic and heterotopic (Figure 31).

- In case of **orthotopic transplantation** the transplanted organ is placed to its normal anatomical position (as it is done in transplantation of heart, lungs or liver).
- **Heterotopic** transplant is considered the placement of the donor organ in the area other than its normal anatomical location (kidney, or pancreas transplant).

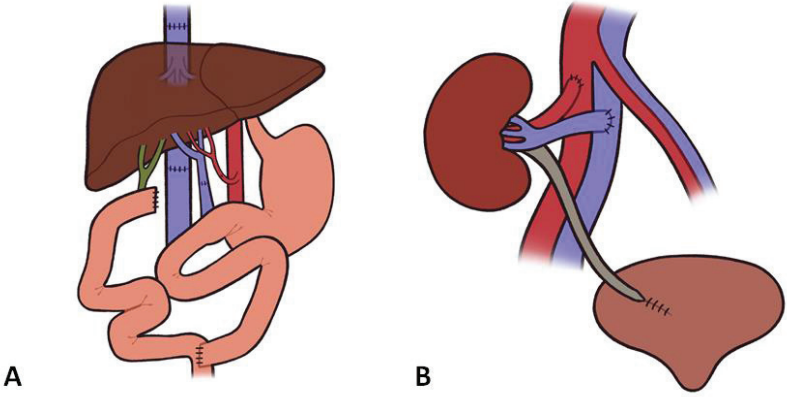

Figure 31. Examples of orthotopic (A) and heterotopic (B) organ transplantation: A – Affected liver of the recipient is removed and liver harvested from the deceased donor is transplanted in the normal anatomic position with anastomosis of blood vessels and bile duct; B – Kidney harvested from the living or deceased donor is transplanted into the iliac fossa of the recipient with the anastomosis between the renal vessels and iliac vessels; the diseased kidney of the patient usually is not removed.

Prosthetics is the use of non-organic or synthetic materials for replacement of tissues and organs. These technologies are increasingly used in cardiac and vascular surgery, orthopedics,

ophthalmology, otorhinolaryngology, surgery for abdominal wall hernias and hand.

Replantation – is a surgical reattachment of a limb or its segment, separated from the body. Replantation is performed using microsurgical techniques: are sutured sequentially damaged arteries and veins, and connected nerves and bones of the cut off limb segment.

Transplantation on the vascular stalk provides dissection of the tissue flap with preservation of the vessel supply; subsequently in the transplanted tissue newly formed vessels grow. This type of transplant is designed to close extensive tissue defects formed after removal of malignant tumors, or as a result of traumatic injuries.

In **free flap transplantation or free grafting**, the prepared flap (usually skin) loses initially its vascular connection with the donor site. Free skin grafting is often used to cover the superficial defects of tissues resulting from burns or trophic ulcers. The skin flap usually will be taken from unaffected areas of the body. For transplantation use the surface layer of the dermis with the preservation of the basal one, in order the donor site will regenerate by itself.

TRANSPLANT REJECTION

Full compatibility of transplanted organs or tissues is achieved only in autologous and syngeneic transplant. In case of allogenous transplantation the tissue incompatibility reaction, or so called **transplant rejection** may occur.

Graft rejection occurs as a result of the immune response, rather than due to non-specific inflammatory response. The main role in transplant rejection belongs to T-lymphocytes. The essential roles in transplant rejections have the same mechanisms, which normally protect the human body from

pathogens. In other words, there are no specific mechanisms responsible exclusively for transplant rejection reaction.

There are three categories of **histocompatibility antigens** relevant for transplantation:

- AB0 antigen system;
- Human leukocyte antigens (HLA), which are considered the major histocompatibility antigens;
- Histocompatibility antigens of minor significance.

Antigenic AB0 system is significant in transplantation, because the same antigens are located not only in blood cells, but are also present in other types of cells. It is important for successful allograft transplantation, as the recipient will receive the tissue or organ compatible by ABO system. Ottenberg transfusion law is also acceptable for transplantation.

Human leukocyte antigens (HLA) have a major role for tissue compatibility, in view of their importance in the immune response. Their physiological function is to recognize polypeptide fragments of antigenic foreign protein, thus to help in their further identification by T-lymphocytes.

Histocompatibility antigens of minor significance are polymorphic proteins, which, after transplantation, are presented as antigenic peptides by recipient antigen-presenting cells.

There are three types of **transplant (allograft) rejection:**

(1) **Hyperacute rejection** occurs immediately after transplantation due to AB0 incompatibility or as a result of the presence of recipient formed anti-HLA antibodies in the blood. Discribed situation may arise as a result of previous blood transfusion, organ transplants or pregnancy. This kind of rejection is characterized by the development of intravascular thrombosis.

(2) **Acute rejection** usually develops within the first 6 months after transplantation, and is caused mainly by T lymphocytes. Acute rejection is characterized by mononuclear cell infiltration of the graft. The majority of acute rejection episodes can be reversed under the influence of additional immunosuppressive therapy.

(3) **Chronic rejection** usually occurs after the first 6 months after transplantation. This type of rejection is the major cause of failure in transplantations. The pathophysiology of chronic allograft rejection is still poorly understood, although it is known that nonimmune factors are responsible for pathogenesis of chronic rejection. Myointimal proliferation of graft arteries is characteristic, which leads to ischemia and organ fibrosis. Immunosuppressive therapy has a little effect in prevention of chronic rejection.

Prevention of transplant rejection is carried out both before and after surgery.

Measures, which should be applied before transplantation include:

- Determination of compatibility according to the AB0 system;
- Determination of compatibility according to the HLA system;
- Cross-matching test to determine the antibodies.

Among the measures, which should be performed after transplantation, there is a prolonged immunosuppressive therapy in order to prevent acute rejection.

Treatment of transplant rejection.

Treatment of **hyperacute rejection** is very difficult, and the known conservative measures (plasmapheresis, cyclophosphamide, prostaglandin E) are unlikely to be

efficient, and hence a retransplantation is necessary in most cases.

Treatment of **acute graft rejection** consists in the administration of Solu-Medrol (methylprednisolone) in pulse therapy mode (1 gram for 3 days). Refractory cases should be treated with monoclonal antibodies or antilymphocytic serum. In severe cases, a retransplantation is required.

The treatment of **chronic rejection** is complex and difficult. Sometimes only replacement of cyclosporine to tacrolimus or sirolimus can prevent rejection, but in most cases an organ loss occurs and retransplantation is in need.

In addition to suppressing immune system, immunosuppressants have many side effects that can worsen the patient's condition, and should be taken into account when prescribing or changing therapy regimens.

ORGANS DONATION

There are two categories of donors: **living donors**, which can donate paired organs (for example, kidneys) or parts of organs such as the liver, pancreas, kidney, intestine or lung; as well as those **who are not able to live (with preserved cardiac function and with cerebral death)**, which are the most common. They are the only ones who can be donors of heart or cornea. **Dead donors without cardiac activity ("asystolic" or "non-heartbeating" donors)** are rarely used, although historically for a long time they remained the only source of donor organs. The main problem accompanying organ donation from non-heartbeating donors is warm ischemia, which inevitably develops and damages all organs after circulatory arrest. Therefore, the main attention of transplantology is focused on donors with preserved cardiac

activity, but with brain death. The establishment of irreversible brain death is a very responsible procedure, as it means that curative measures aimed to maintain the patient's vital functions have no perspective and can be stopped.

Signs of brain death.

Brain death is a state of cessation of cerebral function wherein the proximate cause is known and is considered irreversible. The American Association of Neurology (AAN) has defined brain death with three cardinal signs, cessation of the functions of the brain including the brainstem, coma or unresponsiveness and apnea.

Brain death is recorded in the absence of the cerebral nerve reflexes, which include: pupillary reflexes, corneal reflexes, pharyngeal (gag) reflex and tracheal (cough) reflex, reflex movements of the eyeballs. In case of brain death, there should be no motor reflexes to pain stimuli in the head or face, and the absence of spontaneous breathing. The presence of spinal reflexes does not preclude brain death.

Rules for organ/tissue extraction for transplantation:

- Extraction of organs is performed in sterile conditions;
- The organ is removed, preserving the maximum possible length of vessels and ducts;
- After extraction the organ is perfused with a preservation solution of low temperature (+1-4°C);
- After extraction the organ is either transplanted in short time to recipient or is stored in a sealed plastic bag at a temperature of +1-4°C.

The **period of cold ischemia** of organ starts with clamping of the aorta and introduction the ice cold (+1-4°C) solutions

(UW – University of Wisconsin, or Custodiol, Celsior) into the donor's vascular system.

The **period of warm ischemia** starts after removing the organ intended for transplantation from a cold preservation solution.

The main tasks in the field of organ transplantation are as follows:

- Providing the long-term vital activity of the transplanted organ;
- Prevention of organs rejection;
- Technically perfect execution of transplantation;
- Determining the optimal volume of post-operative intensive care;
- Monitoring of patients after transplantation throughout their life;
- Further development of effective immunosuppressive therapy.

ACKNOWLEDGEMENT

The Department of General Surgery was formed simultaneously with the foundation of Chisinau Medical University in 1945. The first head of department was prof. Savely Rubashov (from 1945 to 1947). From 1947 to 1950 the department was headed by prof. Alexey Lvov. Thereafter, the Department of General Surgery was headed by such eminent surgeons as prof. Petr Hohlov (1950-1952), assoc. prof. Leonty Shulyak (1952-1954), prof. Martiros Arutyunyan (1954-1957), prof. Nikolai Gladyrevsky (1957-1962), prof. Constantin Tsabarna (1962-1979), acad. Gheorghe Ghidirim (1979-1991), assoc. prof. Nicolae Curlat (1991-1994), prof. Silviu Sofronie (1994-1995), prof. Evstafie Cicala (1995-2003), assoc. prof. Ivan Parus (2003-2005), prof. Eugen Guţu (2005-2022). Now Department is leaded by assoc. prof. Dumitru Casian. We are proud to keep history of department and are grateful to our predecessors for setting high standards and preserving the tradition of teaching surgery to medical students for many generations.

We also sincerely thank the leadership of the *Nicolae Testemitanu* State University of Medicine and Pharmacy, represented by the former rector acad. Ion Ababii and present rector prof. Emil Ceban for the democratic style of administration and entire adherence to the principle of academic freedom. In the context of constant changes in the nosology of leading diseases and rapid modernization of surgical specialty, we found full support for our initiatives to change some topics and reform the content of educational program in general surgery and surgical semiology. In this regard, we are also grateful for the constant help and assistance of vice-rector prof. Olga Cernetchii, head of the educational department, assoc. prof. Silvia Stratulat, as well as to the deans of the Faculty of Medicine no.1 (prof. Gheorghe

Placinta) and the Faculty of Medicine no.2 (assoc. prof. Mircea Betiu).

Over the past two decades, the staff of Department of General Surgery and Semiology no.3 has undergone some changes associated with both natural causes and some reorganizational decisions. However, at different stages and in different circumstances, each of the staff members expressed some new ideas and brought their intellectual contribution to improving the content of the course and writing this manual. We thank them all:

Associate professor, Ph.D. Ivan Parush
Associate professor, Ph.D. Traian Beschieru
Associate professor, Ph.D. Alexei Zaporojan
Associate professor, Ph.D. Gheorghe Cristalov
Associate professor, Ph.D. Vladimir Iacub
Associate professor, Ph.D. Victor Curca
Associate professor, Ph.D. Gheorghe Popa
Associate professor, Ph.D. Vasile Culiuc
Associate professor, Ph.D. Ion Isac
Assistant, Ph.D. Marcel Sochirca
Assistant, Ph.D. Serghei Chitic
Assistant, Ph.D. Tudor Ababii
Assistant, Ph.D. Ghenadie Mocanu
Assistant, Ph.D. Vasile Guzun
Assistant, Ph.D. Roman Targon
Assistant, Ph.D. Florin Bzovii
Assistant Serghei Cumpata
Assistant Luminita Vescu
Assistant Andrei Shcureac
Assistant Serghei Moroz

We express our deep gratitude to our colleagues, without whose daily work the edition of this textbook would not have been possible.