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GENERAL SURGERY AND SEMIOLOGY

LECTURE SUPPORT

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CONTENTS

I. SHORT HISTORY OF SURGERY

The word "surgery" means "hand work", "manual operation", or "craft". However the historical meaning of this word has become outdated. Nowadays, surgery is a high-professional medical specialty.

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 (1,600 B.C.) – probably the first surgical manuscript with description of 48 cases of wounds and other traumatic injuries treatment.

Code of Hammurabi from Babylon (1,790 B.C.) – is the first code of laws, which regulates the surgeon's professional 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 over 120 surgical instruments, 300 procedures.

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

Cornelius Celsus (Ancient Rome, 30 B.C.-37 A.D.) – the author of the first surgical treatise. He also described the 5 classical signs of inflammation.

Claudius Galen (129-210 A.D.) – he was the first, who proposed ligation of the bleeding vessel, and considered surgery as a separate specialty. His most important achievements belong to human anatomy and reconstructive surgery.

Abu Ali ibn Sina or **Avicenna** (980-1037), the author of "Canon of Medicine".

Abul Qasim Khalaf ibn al-Abbas al-Zahrawi or **Albucasis** (993-1064). He invented artery ligation during surgery, proposed operating room. He also described hereditary origin of hemophilia, and ectopic pregnancy for the first time.

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

Andreas Vesalius (Padua, Italy, 1515-1564), the first, who described properly the human anatomy in his book *"De corporis humani fabrica"* and is known as the first surgeon-anatomist.

Paracelsus (Switzerland, 1493-1541), being a military surgeon he improved the methods of wound treatment.

Ambroise Pare (France, 1517-1590), a military surgeon, invented hemostatic forceps, developed the technique of amputation, and described the treatment of gunshot wounds.

Dominique Jean Larrey (1766-1842) – the founder of modern military surgery. He developed a lot of new surgical procedures applied in case of traumatic injuries, and introduced the system of rapid transportation of injured soldiers to hospital.

Nicolai Pirogov (1810-1881) is considered the founder of surgery in Russia. He brought the technique of a lot of surgical procedures to perfection and developed the system of surgical care.

Professor **Nicolae Anestiadi** (1916-1968) is the founder of modern surgery in Moldova. He was a pioneer of modern thoracic surgery, cardiovascular surgery, abdominal surgery and anesthesiology in our country.

II. ANTISEPSIS

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.

Asepsis – this is a complex of methods, preventing entering of contaminants into the wound and whole human organism, in other words, creation of sterile surgical conditions.

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

(1) Empiric period;

(2) Antisepsis of the XIX-th century before the appearance of Lister's antisepsis;

(3) Period of Lister's antisepsis;

(4) Modern antisepsis.

Empiric period. Initial antiseptic methods are discovered in the scientific papers of doctors of the ancient world.

Antisepsis of the XIX-th century. Ignaz Semmelweis – use of 10% chloride of lime reduced greatly 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. 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 a system of measures to fight against infection.

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.

(1) Wound toilet. The bandage imbibed with exudation is removed; purulent exudation, infected clots, necrotized tissue are removed from the wound surface using a pincer with a gauze globule.

(2) Primary surgical processing of wound consists of dissection of wound, its pouches and excision of wound edges and bottom within the limits of healthy tissues.

(3) Secondary surgical processing of wound is performed in case when the wound infection has already developed.

(4) Other operations and manipulations – drainage of purulent foci.

PHYSICAL ANTISEPSIS

(1) Hygroscopic dressing material. Introduction of hygroscopic dressing material (gauze and cotton) into the wound increases significantly the volume of evacuated exudate.

(2) Hypertonic saline solution. Hypertonic solutions are used for improvement of wound flow-out, 10% solution of sodium chloride is used mostly.

(3) Sorbents. Substances, which absorb toxins, are introduced into the wound.

(4) Drainage. Latex tubes and bands are usually used for **passive drainage**. In **active drainage** the drainage tube is connected to a pump with negative pressure. In case of **flow-irrigative drainage** an antiseptic solution is introduced through one tube, and flows out through the other.

(5) Additional methods of physical antisepsis.

Drying – treatment in controlled abacterial environment.

Wound processing with **pulsatile water jet**.

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

High-energy (surgical) laser causes evaporation of tissues.

Ultraviolet rays.

Vacuum (VAC-system).

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 for the irrigation of purulent wounds and 2% solution for the disinfection of premises, 5-10% alcohol solution of iodine, iodine derivates (1% povidone and 1% solution of iodopirone).

(2) Salts of metals: 1-0.03% water solution of silver nitrate, nitrate salts: Collargol and Protargol.

(3) Alcohols: 70% and 96% solutions of ethyl alcohol.

(4) Aldehydes: formaldehyde, 1-3% Lysoform solution, Cidex (2% solution of glutaraldehyde).

(5) Phenols: carbolic acid, Ichtyol used in the form of ointment.

(6) Dyes: 1-3% alcohol solution of methylene blue, brilliant green, Rivanol.

(7) Acids: powder and 4% solution of boric acid, and formic acid.

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

(9) Oxidants: solution of hydrogen peroxide, Perhydrol contains 30% of hydrogen peroxide; 0.1% solution of potassium permanganate is used for wound irrigation.

(10) Detergents: chlorhexidine bigluconate in the form of 0.5% alcohol solution, and 5% water solution.

(11) Derivates of nitrofurane: Furacillin, Nitrofurantion, Furazolidone are uroantiseptics, Furagine is a substance for i.v. use.

(12) Derivates of 8-oxiquinoline: Nitroxaline (5-NOK) is an uroantiseptic. It is used in case of urinary tract infection. Enteroseptol, Interostopan are chemical substances used for enteric infection.

(13) Derivates of quinoxaline: Dioxidine is an antiseptic for external use.

(14) Derivates of nitromidazole: metronidazole (Metrogil, Trichopol) is a broad-spectrum chemotherapeutic substance. It is active against protozoa, bacteroid and anaerobic infections.

(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 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 affecting microorganisms and (2) different substances making better capacity of microorganism to fight against infection. Antibiotics, protein-degrading enzymes, bacteriophages and medical serums are biological antiseptics also.

(1) Antibiotics are widely used in treatment of purulent infections. The main principles of antibiotic therapy are:

- Antibiotics should be used according to doctor's prescription.
- It is very important to determine the sensitivity of microorganisms to antibiotics.

- Sensitivity testing to antibiotics should be performed.
- Antibiotics should be changed every 5-7 days in case of prolonged antibiotic therapy to avoid addicting of microorganisms to antibiotics.
- Combinations of antibiotics of different spectrum of action are used to increase antibacterial activity.
- Combination of administration ways is widely practised.
- Antibiotics should be used in combination with other antiseptic substances.

(2) Protein-degrading enzymes do not destroy microorganisms directly, but clean the wound from necrotized tissues, clots and pus quickly. Protein-degrading enzymes may be used in the form of powder, ointment, immobilized enzymes.

(3) Bacteriophages are substances, containing viruses, which can reproduce in bacterial cells and destroy them.

(4) Curative serums, which contain antibodies to main agents of surgical infection. They are used parenterally for passive immunization.

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

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

(2) Vaccines and anatoxins contain minimal dose of microorganisms or their toxins. They stimulate production of antibodies to certain microorganisms introduced in the organism (tetanus toxoid, staphylococcic anatoxin).

(3) Physical methods stimulate nonspecific resistance of organism. Ultraviolet and laser blood irradiation refer to this group of methods.

III. ASEPTIC TECHNIQUE IN SURGERY

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:
(1) Urinary tract infections;
(2) Pneumonia;
(3) Surgical-site infections (wound infections).

Definition of **surgical site infection** – 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 the place.

There are three types of surgical site infection:
(1) Superficial incisional infections (with affectation of skin and subcutaneous tissue);
(2) Deep incisional infections (involve fascia and muscles);
(3) Organ/space infections (localized in the organs or anatomical cavities).

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 (**exo**genous infection) and by treatment of chronic infections in the organism (**endo**genous 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

The most important measures for prevention of airborne infection include the following principles:
(1) Separation of patients with septic processes;
(2) Restricted access in operating rooms;
(3) Wearing surgical attire;
(4) Cleaning of operating rooms;
(5) Ventilating from the ceiling to the floor.

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 main principles of contact infection prevention are:
(1) Surgeon's hands scrub (thrice-repeated washing, drying with sterile towel and decontaminated with antiseptic solution during 2-5 minutes – alcohol, iodine/iodophors, chlorhexidine, and triclosan).
(2) Sterilization of surgical linens (gown, drapes and bandages) – sterilization in the autoclave for 45 minutes at 1.5 atm and temperature of 120ºC. The linens are loaded into Sheemelbush's container, which remains sterile during 72 hours after sterilization unless opened or damaged.
(3) Sterilization of surgical instruments includes three consecutive steps:
- Decontamination – instruments are immersed in 6% solution of hydrogen peroxide, 0.5% chlorine solution or other antiseptic.
- Cleaning – washing in detergent with brush and rinsing in warm running water.
- Sterilization – dry-heat sterilization in special electric oven under the following conditions: duration – 1 hour, temperature – 180ºC.

Delicate surgical items (optical, endoscopic) can be sterilized by cold methods – chemical sterilization using strong antiseptic solutions like glutaraldehyde (Cidex).

Control of quality of sterilization:
- Direct method – the obtained material is tested in the bacteriological laboratory;
- Indirect methods – using of heat-sensitive chemical indicators.

(4) Principal rules of decontamination of **operating field** (patient's skin):
- Shaving should be performed immediately before the operation;
- Skin must be cleaned more widely than the area of intervention;
- Cleaning must be performed from the center to periphery;
- More contaminated areas are cleaned later;
- Cleaning must be repeated three times before limitation of the operative field with drapes, prior to incision, before and after wound closure.

PREVENTION OF CONTAMINATION BY IMPLANTATION

Implantable device – is a device that is placed into a surgically or naturally formed cavity of the human body if it is intended to remain there for the **period of 30 days or more.** Surgical sutures, vascular grafts, artificial heart valve, orthopedic devices, synthetic meshes for hernia repair and other refer to implantable surgical objects.

Implantation of infected material in the human body inevitably results in development of septic–purulent process. Infection caused by an unsterile implant is resistant to antibacterial treatment and a standard surgical removal of infected item is required.

All implantable devices are sterilized at the factory by ionizing radiation and all these objects are disposable.

ENDOGENOUS INFECTION

Unlike external ways of infection, microorganisms can penetrate into the wound from different sources inside the patient's organism – chronic kidney infections, chronic bronchitis, chronic tonsillitis, caries and others.

There are two ways of spread of endogenous infection:
- Hematogenous;
- Lymphogenous.

Treatment of above-mentioned pathology prior to elective surgical intervention is strongly recommended.

ANTIBACTERIAL PROPHYLAXIS

Indications for antibacterial prophylaxis depend on the degree of contamination of surgical wound. In class 1 or clean wounds with no implantable device left in the ioperative site, the prophylactic administration of antibiotics is not indicated. If surgical implant was introduced into the body or if the wound is considered to be contaminated during surgery the application of antibacterial prophylaxis becomes mandatory. Infected or septic wound usually requires the association of prophylaxis with prolonged antibacterial treatment.

Selection of antibacterial drug for prophylaxis depends on pathogenic flora responsible for majority of surgical site infections. First generation cephalosporins and ampicillin–sulbactam refer to the first line drugs for antibacterial prophylaxis. These drugs should be combined with metronidazol in colorectal surgery. The optimal time for **administration** of preoperative doses is within 60 minutes before surgical incision by intravenous route. Normally, a single maximal dose of antibiotic is enough.

IV. HEMORRHAGE

Bleeding can be defined as a leakage of blood from the vascular system (blood vessels and cardiac cavities) caused by damage of its integrity or increased permeability of the vascular wall.

CLASSIFICATIONS OF BLEEDING
(1) Anatomical classification. In accordance with anatomical structure of damaged vessel hemorrhages are divided into:
- Arterial bleeding;
- Venous bleeding;
- Capillary (parenchymatous) bleeding.

(2) Classification of bleeding by **mechanism of occurrence:**
- *Per rhexin (Latin)* – hemorrhage due to mechanical injury of blood vessel;
- *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.
- Internal bleeding:
  a) intracavitary (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 (gastrointestinal tract from the esophagus to the rectum, respiratory tract, urinary tract, biliary ducts, nasal cavity and ear canal, uterus and vagina);
  c) intratissular (petechia, purpura, ecchymosis, hematoma).

(4) Classification of hemorrhage by **time of development:**
- Primary hemorrhage.
- Secondary hemorrhage:
  a) Early secondary hemorrhage,
  b) Late secondary hemorrhage.

(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 1500 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.

REACTIONS OF HUMAN ORGANISM 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 maintance of adequate blood supply of vital organs. There are two protective reactions, 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;
- Tachycardia;
- Centralization of blood circulation;
- Hyperventilation;
- Hemodilution;
- The renal mechanism with decrease of urine output (oliguria);
- Release of red blood cells from the "depot".

Pathological mechanisms of decompensation in hemorrhage are:
- Myocardial ischemia and disturbances of cardiac function;
- Debilitation of sympathetic nervous system and decentralization of circulation;
- "Blood sequestration";
- Brain ischemia;
- Disturbances of metabolism and exchange of gases;
- Systemic inflammatory response with the development of multiple organ dysfunction syndrome;
- Modification of microcirculation of white blood cells and platelets.

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.

Patients with **internal intraluminal bleeding** have**:**
- 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 diagnosis of **internal intracavitary bleeding** is the most difficult:
- 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.

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 $\times 10^{12}$/L);
- Hemoglobin level (normal values – 130-160 g/L);
- Hematocrit (normal values – 40-45%).

To assess the circulatory blood volume (CBV) 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 – 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.

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. 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.

Instrumental methods of diagnosis.

Instrumental methods of diagnosis are: 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 trocar and introduction of catheter (paracentesis, thoracentesis), direct visualization (thoracoscopy, laparoscopy).

V. BLOOD COAGULATION AND HEMOSTASIS

BLOOD COAGULATION

Hemostasis is defined as a complex of physiological mechanisms aimed to stop the bleeding. 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 favourable 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 fibres is realized with reinforcement of the fibrin clot under the influence of the fibrin-stabilizing factor XIII.

Mechanisms of limitation of local coagulation include:
- Vascular endothelium;
- Inactive state of clotting factors;
- Activation of antithrombin III;
- Natural anticoagulants – endogenous heparin;
- Fibrinolytic system of blood.

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.

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;
- Systemic damage of vascular endothelium;
- Direct activation of coagulation system by microbial enzymes;

- All these factors result in generalized 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.

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, the concentration of fibrinogen drops to the critical level, and D-dimers are significantly elevated.

Treatment.
- Correction of the causative pathology;
- Compensation of clotting factors (massive transfusion of fresh frozen plasma);
- Administration of heparin;
- Symptomatic therapy in case of dysfunction of organs and systems.

MEDICAMENTOUS AND SURGICAL HEMOSTASIS

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

Surgical hemostasis.

Methods of **temporary** hemostasis are:
- Application of compressive bandage or tight tamponade of the wound;
- Maximal flexion of extremity;
- Elevated position of extremity;
- Digital compression of bleeding vessel in the wound or along its trajectory;
- Application of hemostatic tourniquet;
- Hemostatic device XSTAT-30;
- Application of hemostatic forceps on the bleeding vessel.

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 (prosthetics);
- 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);
- Low temperature (liquid nitrogen and carbon dioxide);
- Ultrasound (Harmonic scalpel).

Chemical methods:
- Adrenalin;
- Ethanol and polidocanol;
- Cyanoacrylate.

Biological methods:

Biological methods are based on utilization of plasma derivates that stimulate local thrombogenesis. They are used as hemostatic sponges (Tachocomb), or as solutions (fibrin glue, Tissucol). Furthermore, there are haemostatic sponges on the basis of animal (bovine) collagen (Helistat, Surgispon), or cellulose (Surgicel).

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.

HISTORY OF BLOOD TRANSFUSION

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 haemagglutination and I-III blood groups (Karl Landsteiner, 1900);
- Discovery of the IV blood group (Jan Jánsky, 1907);
- Conservation of whole blood using sodium citrate (V.A.Iurevici and N.C.Rosergart, 1914);
- 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.

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;
- Grupa B(III) – individuals have the agglutinogen B on the surface of RBC, and α agglutinins – in plasma;
- Grupa 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.

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 haemolysis. There are different types of agglutination: iso-haemagglutination, hetero-haemagglutination, pseudo-haemagglutination and pan-haemagglutination.

The Ottenberg's rule:
- 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 is used.

Methods for determination (typing) of blood group:
- **Method with standard serum.** Standard serums of group 0(I), A(II), and B(III) are mixed with a small amount of examinated blood. The assessing of the results is performed after at least 5 minutes, by the presence or absence of agglutination;

Standard serum				Blood group
0 (I)	A (II)	B (III)	AB (IV)	
-	-	-	-	0 (I)
+	-	+	-	A (II)
+	+	-	-	B (III)
+	+	+	-	AB (IV)

- **Method with anti-A and anti-B monoclonal antibodies.** 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;

Monoclonal antibodies		Blood group
Anti-A	Anti-B	
-	-	0 (I)
+	-	A (II)
-	+	B (III)
+	+	AB (IV)

- Method with standard 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).

BLOOD TRANSFUSION

Indirect blood transfusion consists in its collecting, conservation and storage for a certain period at the temperatutre of 4-6°C. **Direct** transfusion is the introduction of donated blood to the recipient immediately after its collecting, that is, without the conservation and storage. **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;

- Acute preoperative normovolemic haemodilution;
- Reinfusion.

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

Blood components:

- **Packed RBCs (pRBC)**. 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. 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 50×10^9/L. In patients without risk of bleeding, the platelet transfusion is performed when the count is less than 20×10^9/L;

- **Granulocyte concentrate** is rarely used in surgery;

- **Fresh Frozen Plasma (FFP)**. 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 too. Transfusion of FFP is indicated in case of coagulopathies. Utilization of FFP for restoration of the circulatory blood volume is not rational.

Plasma derivates:
- Solution of albumin;
- Cryoprecipitate;
- Lyophilized thrombin;
- Immunoglobulin and gammaglobulin;
- Concentrates of coagulation and anticoagulation factors.

Blood substitutes (the main purpose of the use of blood substitutes is the correction of hypovolemia)**:**

- **Crystalloid solutions** (normal saline or 0,9% sodium chloride solution, Ringer's solution, Hartmann's solution). 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 Poliglucin, Dextran 40 or Reopoliglucin; starch derivatives – Hydroxyethyl starch (HES) 450 or Refortan). 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
- 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 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**.

Acute nonimmune reactions and complications: (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 reactions and complications: (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) transfusion-related acute lung injury or „TRALI" syndrome.

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

Late immune 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 its 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.

VII. 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 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 80kg 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. In case of local anesthetic toxicity the basic elements of treatment are oxygen and airway support. 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:
(1) Topical anesthesia;
(2) Tumescent anesthesia;
(3) Regional anesthesia:
- Peripheral neural block (large nerve or plexus);
- Central neural block (epidural or spinal).

TOPICAL ANESTHESIA

Application of topical anesthetics to the skin or mucosa to control pain associated with **minor invasive procedures** such as uretral 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 absorbtion 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 Ethyl 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 famous 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 adrenaline for vasoconstriction and prolongation of anesthesia;
- Alternate 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 Lidocain).

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 gynaecologic 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 (25-27 G) 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 vary 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.

BLOCKADES WITH LOCAL ANESTHETICS

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 anesthethic is a standard technique of "resuscitation" of ischemic intestinal loop during surgery for strangulated hernia. **Paranephric 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.

VIII. SURGICAL INTERVENTION. PRE- AND POSTOPERATIVE PERIOD

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.

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**.

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:

- Urinalysis and full blood count;
- Biochemical blood analysis (glucose, liver enzymes, creatinine, urea);
- Blood coagulation;
- Blood group and Rh;
- 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.

Risk of surgery can be stratified by ASA system:

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. 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;
- Correction of concomitant diseases;
- Measures for prevention of complications;
- Special preoperative care.

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 (III-IV generation Cefalosporins) are administered in the absence of infection, with the aim to reduce the rate of postoperative wound infection, which result from existing or potential contamination during surgery.

Routine procedure of **patient preparation** includes also:
- Administration of sedatives;
- Hygienic shower with cleaning and shaving of future surgical field;
- Digestive tract emptying;
- Catheterization of urinary bladder;
- Preparation of central venous access.

The most important details (argumentation of diagnosis, indications for surgery, planned volume and type of surgery, type of anesthesia and degree of risk) of preoperative period are summarized in the **preoperative conclusion** recorded in the patient's medical case history.

Position of patient on the surgical table depends on the type of surgery:
- Supine/dorsal recumbent (is typically used);
- Trendelenburg position;
- Reverse Trendelenburg position;
- Sitting or semi sitting position (Fowler position);
- Dorsal lithotomic position;
- Lateral position of patient.

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 the access to the affected organ.

There are **4 steps of surgical intervention:**
- Access;
- Exploration;
- Procedure;
- Check and closure.

Surgical intervention is considered to be finished when the last skin suture is applied.

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 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 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.

According to the **number of stages** surgeries are divided into one-stage surgeries and multiple-stage surgeriest.

There are 4 types of surgical interventions according to the **degree of contamination**:
(1) Absolutely clean – elective surgery not associated with opening of hollow organs (vascular surgery, cardiac surgery);
(2) Clean – opening of a little contaminated organs (stomach resection, biliary surgery);
(3) Infected – opening of a significantly contaminated organs (intestinal surgery);
(4) Purulent – surgery for purulent and septic processes (peritonitis, abscess).

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.

Physiological staging of postoperative period includes 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 thecavities;
- Urinary tract infection.

Complications of the remote postoperative period are:
- Recurrence of disease;
- Stenosis and occlusion of anastomosis;
- Vascular graft thrombosis.

IX. SURGICAL INSTRUMENTS. SUTURES AND KNOTS

SURGICAL INSTRUMENTS

Surgical instruments are very varied and comprise of numerous types of objects used for surgery. There is a common instrumentation, completed by a number of other special tools needed for interventions in other specialties (cardiac surgery, orthopedics, neurosurgery). The appearance of minimally invasive surgery has led to the creation of special instruments handled outside the body and reaching the working chamber through trocars.

Surgical instruments are made from durable materials (usually stainless steel, which provides elasticity, resistance to prolonged use and sterilization), which allows their cleaning and sterilizing. Instrumentation must be simple, easy to handle, must provide safety in use. The size and shape of the instrument are adapted special to surgical approach, and to region or organ need to be operated.

Normally, instruments used in general surgery are **divided into the following groups**: (1) for dissection of tissues, (2) for hemostasis, (3) for grasping of tissues, (4) retractors and dilators (5) for suture of tissues (6) for exploration, (7) different and special instruments, and (8) laparoscopic instruments.

Instruments for dissection of tissues are scalpels and scissors. Scalpel is a small and extremely sharp instrument used for surgery and anatomical dissection. Scalpels may be single-use disposable or re-usable. Disposable blades for scalpels are produced and sterilized in factory, and released into the hospitals in sterile sealed envelopes. Various types of blades by shape and size are labeled with specific number. Semioval flat blades are used for skin incision, and for other tissues small pointed or bent blades are used.

Surgical scissors may have different length and shape. Scissors length varies between 10 and 35cm, scissors with shorter and thicker blades are used for tissue dissection, thick-walled organs or sutures. Fine curve scissors are used in deep tissue dissection. In vascular surgery fine scissors of different shapes are used.

Amputation knife, wire saw Gigli, and different osteotomes are used for amputations.

Instruments for hemostasis. Various types of forceps adapted to operated region, depth of surgical field and vessel size are used for intraoperative hemostasis.

The main types of clamps used in general surgery are: Pean forceps – straight or curved active arms have no teeth, forceps Kocher – with the active arms curved or straight, with top teeth, forceps Mosquito – is a fine clamps, used in vascular and endocrine surgery.

From the inside of the arms is situated a locking system, rack type, composed of 2-7 teeth. Forceps are locked automatically by simple pressure on both arms of instrument.

Instruments for grasping of tissues. For grasping of tissue using anatomy (soft) forceps (pincettes) of different length depending on the depth of operator field. These are used for clamping soft tissues: the walls of the stomach, intestines or blood vessels. Free ends of surgical forceps (pincettes) are terminated with teeth, providing a strong grasping, and used to capture and presentation of resistant tissues (skin, fascia).

Intestinal Allis forceps have small fine teeth, which does not penetrate the bowel wall, vascular atraumatic clamp Satinsky has a long elastic, double curvature arms, and no teeth, used for hemostasis in the vascular wounds;

Retractors and dilators are required to remove the wound edges, tissues or organs from the surgical field. They may have different size and shape depending on the region to be operated on. They are managed by an assistant or mechanically.

Farabeuf retractor consists of a solid blade, bent at right angles at both ends, with one blade longer and wider and the other – shorter. They are used in pairs.

Volkman retractor is similar to the Farabeuf one, but has 2-6 sharp teeth on the end, used to remove resistant tissues (muscle, skin, tendons).

Abdominal valve consists of one straight or curved blade, which continues with a handle.

Mechanical retractors have a system to lock the valves, and generally are used in abdominal and thoracic surgery.

Instruments for tissue suture. It consists of surgical needle holders, needles and forceps.

Hegar needle holder is commonly used. It has the shape of long forceps, with thicker arms and short tip, which ensures its security. Mathieu needle holder has two arms articulated to the top, which is short and thick at the base and is provided with a lock system, consisting of three teeth on one arm and one tooth on the other.

Needles are made of steel and can be straight and semicurbed. Hagedorn needle has a swage, body and point. Hagedorn needles have 14 dimensions adapted to all organs and tissues which are sutured. Their curvature is 1/4, 3/8, 1/2, or 5/8 of circle. The needle point can be triangular or round. Those with triangular points are used for suturing the skin, fascia, tendons and muscles. Those with rounded point are used for suturing hollow organs or fine soft tissues.

The needle ear is fitted with two small side slats, with a narrow cleft in the "V"-shape between them, this system allows the loading of the needle with the thread push under tension.

A great progress was achieved with introduction of mechanical staplers for suturing. Numerous instruments, initially produced by the company AutoSuture, may be divided into linear staplers and circulars ones. Linear staplers are designed to apply 2 or 3 lines of staples to close quickly and safely pulmonary tissue or bowel lumen. A circular stapler allows performing anastomosis between two cavitary organs: to connect the colon, intestine or esophagus after their resection.

Instruments for exploration are stylets (probes), used for exploring trajectories of fistulas or normal anatomical structures, without risk of damaging surrounding tissues.

Special and different instruments (varicous vein stripper, working part of aspirator, electrocautery, forceps for fixing the operating field etc).

Laparoscopic instruments.

Operating room table. Requirements to the operating table are the following: stability and safety, functionality, easy cleaning and decontamination.

A surgical light. Requirements to the surgical light: bright, cold light, deep cavity illumination, minimal shadow interference. Surgical lamp should to be easily disinfected, but surgeons should have the power to change a field and adjust the pattern of light.

Surgical lamps could be mounted (ceiling or wall) and portable. In addition to this there are individual surgical light (named lighthead) placed on the head of surgeon.

SUTURE MATERIAL

The oldest sutures are from Egypt and China where 4,000 years ago suturing of skin and bowel with plant fibers were used. First absorbable suture material was catgut, invented by Galen, then Joseph Lister invented the first slowly absorbable material – chromic catgut. Natural silk came into use in the late XIX century, thanks to two great surgeons, Kocher and Halsted. Detailed studies of gut and silk properties have revealed multiple side effects of these materials: allergy, high reactivity and long-term resorption.

Subsequent progress in surgical suture material was marked by synthetic threads appearance: in the 20-s of the XX century – nylon (Polyvinyl) – the first synthetic material, in the 30-s of the XX century – kapron (Polyamide) and lavsan (Polyester), in

1956 – polypropylene, in the 40-s of the XX century – "supramid extra"– complex suture (braided or twisted multifilament kapron, coated with polymer), in 1971 – the first synthetic absorbable suture – Dexon (polyglycolic acid), and in 1974 – Vycril (polyglactin 910).

The ideal suture material should have the following characteristics: simplicity of sterilization, safety of knot, resistance to infection, absorbility, resistance of wire have to exceed that of the tissue, easy handling, universal application in various operations, the absence of carcinogenic activity, absence of allergic properties, and low cost.

Suture materials are classified by structure into three categories: monofilament (Prolene, PDS, Dermalon, Maxon, nylon), multifilament braided (Ethibond, Mersilene, Dexon II), and composites – polymer-coated multifilament (Vycril, Polysorbe, Supramide). According to the material from which they are produced, sutures are divided into the following categories:

(1) Biological suture materials: catgut, silk

(2) Synthetic suture materials

 A. Absorbable suture materials

- Derivatives of polyglycolic acid: Dexon, Vycril, Monocryl, Maxon;
- Derivatives of polydioxanone: PDS.

 B. Nonabsorbable suture materials

- Polyestere: lavsan, kapron, Mersilene, Polyester, Ethibond;
- Polyolefine: Prolene, polypropylene, polyethylene;
- Fluoropolymere (PTFE): Gore-tex;
- Polybuthilestere: Novefil.

(3) Metallic suture suture materials

If silk thread is used only two knots are made. Most threads are tied off with 3-4 knots (catgut, lavsan, kapron, Mersilene, Polyester, Dexon, Vycril etc.) Monofilament synthetic threads (polypropylene, PDS, nylon, Maxon, etc.) are ligated using 6-8 knots.

Atraumatic needle is an eyeless surgical needle with the suture attached to a hollow end. Currently atraumatic needles of various diameters and sizes are used in surgical practice. They are sterilized industrially and kept in sealed plastic envelopes. Labeling of enveloped atraumatic needle with thread includes: name of manufacturer and suture material, wire diameter and length, needle properties, the validity of sterilization period.

KNOTS AND SUTURES

The four nodes are commonly used in surgery:

(1) Simple (direct, square) knot – is the most applicable and easy;

(2) Surgeon's knot – is safer;

(3) Slip knot is used when one end of the thread is fixed;

(4) Tying instrument is applied when one end of the thread is short.

Skin wound suturing. The needle must penetrate the skin perpendicularly, and the force must always be applied along the direction of needle curvature; the needle is not left without control of any instrument; only fine manipulation of tissues are allowed; small "steps" of suture may cause rupture of wound edges; equal distance between the site of entry and exit of the needle, and equal distance between the sutures are required.

Sutures of skin are classified into interrupted (simple suture, suture of Blair-Donati and suture in U), and continuous (simple running suture and intracutaneous suture).

X. DRESSINGS AND BANDAGES

Dressing is a pad that is placed in direct contact with the wound. It should be large enough to cover the entire area of the wound and to extend at least 2-3 cm in every direction beyond the edges. If the dressing is not large enough, the edges of the wound are almost certain to become contaminated. The dressing material which covers wound should always be sterile. However, emergencies will sometimes arise when they are not available. In such a case, use the cleanest cloth available. Unfold these materials carefully so that you do not touch the part that is next to the skin.

Bandages are strips or rolls of gauze or other materials that are used for wrapping or binding any part of the body and to hold compresses in place. The types of most commonly used bandages are **(1) triangular bandage, (2) cravat bandage, (3) roller bandage, and (4) elastic net retention bandage**.

TRIANGULAR BANDAGES

Triangular bandages are usually made of cotton. They are made by cutting a 90 to 100cm square of a piece of cloth and then cutting the square diagonally, thus making two triangular bandages. The longest side of the triangular bandage is called the base; the corner directly opposite the middle of the base is called the point; and the other two corners are called ends.

Triangular bandage for shoulder. Cut or tear the point, perpendicular to the base, about 25cm. Tie the two points loosely around the patient's neck, allowing the base to drape down over the compress on the injured side. Fold the base to the desired width, grasp the ends, and fold or roll the sides toward the shoulder to store the excess bandage. Wrap the ends around the upper arm, and tie on the outside surface of the arm.

Triangular bandage for the side of chest. Cut or tear the point about 25 cm, perpendicular to the base. Place the bandage with the points up, under the arm on the injured side. Tie the two points on the top of the shoulder. Fold the base to the desired width, carry the ends around the chest, and tie on the opposite side.

Triangular bandage for foot. This bandage is used to retain large compresses on the foot. After the compresses are applied, place the foot in the center of a triangular bandage and carry the point over the ends of the toes and over the upper side of the foot to the ankle. Fold the excess of bandage at the side of the foot, cross the ends, and tie them in a square knot in front.

Triangular bandage for hand. This bandage is used to retain large dressings on the hand. After the dressings are applied, place the base of the triangle well up in the palmar surface of the wrist. Carry the point over the tips of the fingers and the back of the hand well up on the wrist. Fold the excess of bandage at the side of the hand, cross the ends around the wrist, and tie a knot in front.

CRAVAT BANDAGES

To make a cravat bandage, put the point of the triangular bandage to the middle of the base and continue to fold until a 5 cm width is obtained.

Cravat bandage for head. This bandage is useful to control bleeding from wounds of the scalp or forehead. After placing a compress over the wound, place the center of the cravat over the compress and carry the ends around to the opposite side; cross them, continue to pull them around to the starting point, and tie a knot.

Cravat bandage for elbow or knee. After applying the compress, and if the injury or pain is not too severe, bend the elbow or knee to a right angle position before applying the bandage. Place the middle of a wider edge over the point of the elbow or knee, and pull the upper end around the upper part of the elbow or knee, bringing it

back to the hollow, and the lower end entirely around the lower part, bringing it back to the hollow. Check that the bandage is smooth and fits comfortable; then tie a knot outside of the hollow.

Cravat bandage for arm, forearm, leg or thigh. The width of the cravat you use will depend on the length and area of the injury. Place a compress over the wound and center the cravat bandage over the compress for a small area. Bring the ends around, cross them, and tie over the compress. It may be necessary to make several turns around to use the entire bandage for tying for a small extremity.

If the wound covers a larger area, hold one end of the bandage above the compress and turn the other end spirally downward across the compress until it is secure, then pull it upward and around again, and tie a knot where the both ends meet.

Cravat bandage for axilla. This cravat bandage is used to hold a compress in the axilla. It is similar to the bandage used to control bleeding from the axilla. Place the center of the bandage in the axilla over the compress and pull the ends up over the top of the shoulder and cross them. Continue to pull the bandage across the back and chest, to the opposite axilla and tie them.

ROLLER BANDAGES

Roller bandage consists of a long strip of material (usually gauze or elastic) that is coiled into a cylindrical shape. Roller bandages are available in various widths and lengths. If the roller bandage have been sterilized, it may be cut off and used as compresses in direct contact with wounds. If you use a piece of roller bandage in this manner, you must be careful not to touch it with your hands or with any other unsterile object.

A piece of roller bandage may be used to make a four-tailed bandage. This is done by tearing the cloth from each end, leaving the center area as large as necessary. It is good for bandaging any protruding part of the body, because the center portion of the bandage forms a smoothly fitting pocket when the tails are crossed over.

The four-tailed bandage is often used to hold a compress on the chin or on the nose.

In applying a roller bandage, hold the roll in the right hand so that the loose end is on the bottom; the outside surface of the loose or initial end is then applied to the body part and held on it by the left hand.

The roll is then passed around the body part by the right hand, which controls the tension and application of the bandage. Two or three of the initial turns of a roller bandage should overlie each other to secure the bandage and to keep it in place.

In applying the turns of the bandage, it is often necessary to transfer the roll from one hand to another. Bandages should be applied evenly, firmly, but not too tight. Excessive pressure may cause disturbances of circulation and may lead to ischemic complications.

In bandaging an extremity, it is recommended to leave the fingers or toes exposed so the circulation of these parts may be permanently observed. It is likewise safer to apply a large number of turns of a bandage, rather than a few turns applied too firmly to secure a compress.

In bandaging any extremity, it is advisable to include the whole extremity so that uniform pressure may be maintained. In bandaging a limb it is also mandatory, that the part is placed in the position it will take when the dressing is finally completed, because variations in the flexion and extension of the part will cause changes in the pressure of the bandage. The initial turns of a bandage on an extremity should be applied firmly, and when possible, around the part of the limb that has the smallest circumference. Thus, in bandaging the arm or hand, the initial turns usually are applied around the wrist. In bandaging the leg or foot, the initial turns are applied immediately above the

ankle. The final turns of a completed bandage usually are secured in the same manner as the initial turns i.e., by using two or more overlying circular turns. The terminal end of the completed bandage is turned under and secured to the final turns by either a safety pin or adhesive tape. When these are not available, the end of the bandage may be torn lengthwise for several centimeters, and the two resulting tails may be secured around the part by tying.

Roller bandage for elbow. A spica or figure-of-eight type of bandage is used around the elbow joint to retain a compress in the elbow region and to allow a certain amount of movement. Bend the elbow slightly, if you can do so without causing any additional pain or injury, or anchor a medium-width bandage above the elbow and encircle the forearm below the elbow with a circular turn. Continue the bandage upward across the hollow of the elbow to the starting point. Make another circular turn around the upper arm, carry it downward, repeating the figure-of-eight procedure, and gradually ascend the arm. Overlap each previous turn about two-thirds of the width of the bandage. Secure the bandage with two circular turns above the elbow and tie. To secure a dressing on the tip of the elbow, reverse the procedure and cross the bandage in the back.

Roller bandage for hand and wrist. A figure-of-eight bandage is ideal for the hand and wrist. Anchor the dressing, whether it is on the hand or wrist, with several turns of a medium-width bandage. If it is on the hand, anchor the dressing with several turns and pull the bandage diagonally upward and around the wrist and back over the wound. Make as many turns as necessary to secure the compress properly.

Roller bandage for finger. Place sterile dressing upon the wound and cover the dorsal surface of finger with free end of narrow roller bandage leaving the excess of approximately 40cm. Then pull the bandage downward covering the tip and palmar surface of finger and change the direction of bandage applying two-three circular turns around the finger. Repeat this technique using both ends of bandage. Continue as long as necessary and secure around base of finger.

Spiral reverse bandage for thumb. Anchor the bandage making two or three circular turns around the wrist. Then cover the finger downward to the tip, going around and making the reverse laps on each turning, overlapping about 1/3-1/2 the width of the previous turn. Continue as long as each turn lies flat. Then pull the bandage up, repeating the manipulation, and tie the end around the wrist.

Roller bandage for all fingers. Make two or three circular turns around the lower and smaller part of the limb to anchor the bandage and go downward cross the dorsal surface of hand to the tip of finger.

Roll the bandage around the finger upward, continue the bandage crossing the dorsal surface of hand and making several turns around the wrist, then make the same on the next finger. Start the bandage from the first finger on the right hand and from the fifth – on the left hand. Finish the bandage on the wrist.

Roller bandage for ankle and foot. The figure-of-eight bandage is also used for dressings of the ankle, and to fix the ankle in case of sprain.

While keeping the foot at a right angle, start a medium-width bandage around the instep for several turns to anchor it. Pull the bandage upward over the instep and around behind the ankle, forward and again across the instep and down under the arch, thus completing one figure-of-eight. Continue the figure-of-eight turns, overlapping 1/3-1/2 of its width, with an occasional turn around the ankle, until the compress is secured or until adequate support is obtained.

Roller bandage for heel. The heel is one of the most difficult parts of the body to bandage. Place the free end of the bandage on the external part of the ankle and pull the bandage under the foot and up.

Then pull the bandage over the instep, around the heel, and back over the instep to the starting point. Overlap the lower border of the first loop around the heel and repeat the turn, overlapping the upper border of the loop around the heel. Continue this procedure until the desired number of turns is obtained, and secure the bandage with several turns around the lower leg.

Roller bandage for arm or leg. Spiral reverse bandage must be used to cover wounds of the forearms and lower extremities; only such bandages can keep the dressing flat and smooth.

Make two or three circular turns around the lower and smaller part of the limb to anchor the bandage and start upward, going around and making the reverse laps on each turning, overlapping about 1/3-1/2 of the width of the previous turn. Continue as long as each turn lies flat. Continue the spiral and tie the end when completed.

Hippocrates' bandage is a complicate bandage of head. It is applied using a double-headed bandage, moving its heads in two perpendicular planes, periodically crossing them to form a bandage looking like Hippocrates' bandage.

Capeline bandage is a complicated bandage of head, applied using a medium-width bandage, crossing a band of gauze in that way changing its direction to cover completely the head in the form of a capeline.

ELASTIC NET RETENTION BANDAGES

Elastic net retention bandage is designed to secure other dressings in place without using adhesive. Possessing the elastic property, it fits tightly to any part of human body, and does not become loose during cutting and does not disturb movement. This type of bandage also allows monitoring the damaged area.

There are several sizes of elastic net retention bandage: size 1-3 - for toes, fingers, wrist or ankle; size 4-7 - for hands, feet, legs, arms, head and neck; size 8-14 - for torso, abdomen, perineum; size 20 - for large chest and abdomen. One can choose appropriate bandage to be applied on different part of the human body.

XI. MINOR SURGICAL PROCEDURES AND MANIPULATIONS

INJECTIONS

Parenteral route of drug administration is the most requested in surgical patients: intravenous, intramuscular and subcutaneous ways being more widely used in clinical practice.

Subcutaneous injections. Insert the needle at an angle of 90 degrees when using a small needle, or at an angle of 45 degrees, when subcutaneous tissue is less developed. Subcutaneous injections can be given in the arms, legs, or abdomen. It is extremely important to rotate (change) sites to prevent scarring and hardening of fatty tissue, and to keep the skin healthy.

Intramuscular injections. An intramuscular injection is chosen when a reasonably rapid systemic uptake of the drug (within 15-20 min.) is needed and relatively prolonged action is required. The amounts of solution that can be given depend on the muscle bed and range from one to 5 ml for adults. There are 5 sites that can be considered suitable for intramuscular injections: (1) middle-deltoid site, (2) dorsogluteal, (3) rectus femoris, (4) vastus lateralis and (5) ventrogluteal (the last two are the most recommended sites). Insert the needle into the patient's skin at an angle of 90° until approximately 1 cm of the needle is visible, and inject the solution at a rate of 1 ml per 10 seconds.

Intravenous injections. Intravenous administration of a drug ensures that bioavailability is complete. The antecubital fossa is the preferable site of access, needle being inserted in the vein at an angle of 30 degrees or less.

VASCULAR ACCESS

Advantages of central venous catheterization over peripheral access are: greater duration without infection, line security in situ, avoidance of phlebitis, larger lumens, multiple lumens for rapid administration of drugs, a route for nutritional support and central venous pressure monitoring. There are 2 main categories of central venous catheters: external or subcutaneous. In the **external line** one end of catheter is implanted into the lumen, but the other end exits the skin. The **subcutaneous line** is implanted entirely under the skin.

There are **3 basic methods of inserting the catheter:** (1) Over the needle; (2) Through the needle, and (3) Over guidewire (Seldinger technique). A guidewire is passed down the needle into the vein and the needle removed. The guidewire commonly has a flexible J-shaped tip to reduce the risk of vessel perforation and to help negotiate valves in the vein. Once the wire is placed in the vein, the catheter is passed over, until it is positioned in the vein. Routes for central venous cannulation include: the internal jugular, subclavian, femoral, antecubital and external jugular veins. The first three ways are commonly used for venous access. **Venesection** (or surgical venous incision) is an alternative way to place a central venous catheter. The vein is surgically exposed, clamped at two points and then opened with a small incision. The venous clamp is opened on one side and the catheter is then introduced into the vessel lumen.

Pulmonary artery catheter (Swan-Ganz catheter) is a central venous catheter with a small inflatable balloon at the end and thermistor (to measure temperature), introduced through a large vein, right atrium and ventricle to a branch of the pulmonary artery. Chest radiography is required to check the position of the pulmonary artery catheter and to assess pulmonary complications. Pulmonary artery catheter allows direct, simultaneous measurement of pressure in the right atrium, right ventricle, pulmonary artery, and the capillary filling pressure, oxygen saturations in the right heart.

THORACIC PROCEDURES

Thoracocentesis is a puncture of the pleural cavity to remove fluid or air for diagnostic or therapeutic purposes. Normally the pleural cavity contains less than 20 ml of clear fluid. A chest x-ray must be performed before the test (to select the best site for puncture) and after centesis (to confirm the absence of complications). The puncture is performed on the midclaviculare line, in the 2nd-3rd intercostal space in case of air accumulation, and on the midaxillary line in the 8th-9th intercostal space – in case of accumulation of fluid. The thoracentesis needle is inserted above the rib into the pleural space in order to prevent lesions of vessels. The aspiration should not exceed 1.5 L to prevent development of reexpansion pulmonary edema.

Thoracostomy (also known as a Bülau drain) is insertion of a drain through the wall of the chest into the pleural space. It is used to remove air or fluid from the intrathoracic space. The tube is commonly inserted into the 5th intercostal space slightly anterior to the mid axillary line. After local anesthesia and small skin incision a passage is made through the muscle into the pleural cavity over the rib that is below the intercostal level selected for chest tube insertion. Then the tube is placed through this passage and sutured to the skin (to prevent its falling out) and a bandage is applied. The outer end of the drain is attached to the underwater seal, below the level of the chest. This allows the air or fluid to escape from the pleural space, and prevents a reverse flow to the chest.

ABDOMINAL PROCEDURES

Laparocentesis is a procedure involving puncture and drainage of fluid from peritoneal cavity. It is commonly done for accumulation of free fluid (ascites), that may be caused by infection, inflammation, an injury, cirrhosis or cancer. Diagnostic indications: onset of ascites, ascites fluid of unknown etiology (suspected malignant ascites), clinically suspected ascites infections. Therapeutic paracentesis is indicated when ascites fluid causes respiratory compromise, abdominal pain, or worsening of existing hernias. The preferred site of entry into the peritoneal cavity is in the midline, inferior to the umbilicus. It is recommended to perform the procedure under ultrasound guidance. The patient is placed in supine position, with his head elevated at an angle of 45-60 degrees. After local anesthesia a large boring needle along with a plastic sheath is inserted to reach the peritoneal fluid. The needle is then removed, leaving the plastic sheath inside to allow drainage of the fluid. It is recommended that the upper limit of extracted fluid should be 1,500 mL, but patients with peripheral edema may tolerate larger volumes without hypotension (up to 5L). The latter case intravenous perfusion to prevent hypotension is necessary.

GASTROINTESTINAL PROCEDURES

Gastric intubation is the insertion of a plastic tube usually through the nose down into the stomach. Levin tube (one lumen) and Salem-sump tube (two lumens) are the most commonly used in surgical practice. There are diagnostic and therapeutic indications for nasogastric tube placement. **Diagnostic indications:** evaluation of upper gastrointestinal bleeding (presence, volume), aspiration of gastric content, administration of radiographic contrast to the gastrointestinal tract. **Therapeutic indications:** gastric decompression, relief of symptoms in bowel obstruction, aspiration of gastric content from recent ingestion of toxic material, administration of medication, feeding. After lubrication the tube is placed through the nostril along the floor of the nose and is advanced till the nasopharynx. The patient is asked to sip some water and to start swallowing it then the tube is advanced to the stomach.

Balloon tamponade is used in the management of upper gastrointestinal hemorrhage due to gastroesophageal varices. There are **3 main balloon tubes**: (1)

Sengstaken-Blakemore tube – has a gastric balloon, an esophageal balloon, and a gastric suction port; (2) Minnesota tube – has an additional esophageal suction port to prevent aspiration of esophageal contents; (3) Linton-Nachlas tube – has a single gastric balloon and is the most effective to control bleeding from gastric varices. **Indications:** acute life-threatening bleeding from esophageal or gastric varices that does not respond to medical therapy and endoscopic hemostasis or when the latter is unavailable. The tube can be passed through the nostrils or through the mouth. Inflation of gastric balloon is performed initially, pulling back the tube until resistance is felt. Then the esophageal balloon is inflated to the lowest pressure needed to stop bleeding. Generally, the esophageal tamponade tube is a provisional measure and it should not be left *in situ* for more than 24 hours (risk of perforation). When hemorrhage is controlled deflation of esophageal balloon is performed first. If there is no evidence of bleeding gastric balloon is deflated but the tube is left *in situ* for 24 more hours.

Anoscopy is an examination of anal canal using a small, rigid, tubular instrument – anoscope. The rectum should be emptied before the procedure using laxative or en enema. Topical anesthesia or intravenous medications may be administered for analgesia. The most common position of the patient is the lateral decubitus with the contralateral leg bent at the knee and hip, or the knee-shoulder position.

Sigmoidoscopy is the minimally invasive examination of the large intestine from the rectum through the last part of the colon (35cm distally from the anus). There are **2 types of sigmoidoscope**: flexible and rigid. The colon must be completely empty thus the patient must drink only clear liquids for 12-24 hours beforehand. The night before or right before the procedure, the patient takes a laxative and is given an enema. The patient must lie on the left side on the examining table. The tube is lubricated and inserted with obturator into the rectum and slowly guided into the colon removing the obturator.

UROLOGICAL PROCEDURES

Urethral catheterization is the insertion of a tube into the bladder via urethra. Indications: acute or chronic urinary retention, collection of urine for analysis, the need for accurate monitoring of fluid volume output, benign prostatic hyperplasia, incontinence of urine, and after various surgical interventions involving the bladder and prostate. There are some **types of catheters**: Foley catheter (with a balloon at the tip); Robinson catheter (without balloon, used for a short-term drainage of urine); Coudé catheter (with a curved tip that makes it easier to pass through the curvature of the prostatic urethra). The male patients should be in the supine position with legs slightly apart, and with the legs apart and the knees bent for female patients. When the catheter passes into the bladder, urine will be seen coming through the catheter. At this point, it is pushed forward 1-2 cm more and the balloon (of Foley catheter) is inflated with 5 ml of sterile solution.

Percutaneous suprapubic cystostomy is a puncture and drainage of bladder. It is performed when transurethral catheterization is not possible or is contraindicated. If possible, the bladder is initially distended with fluid. The bladder access is made approximately 2-3cm above pubic symphysis. Cystostomy may be performed under ultrasound guidance using the trocar technique when the bladder is fully distended. When the bladder is not well distended, the needle is initially inserted into the bladder under sonographic guidance, ensuring that no bowel loops are injuried accidentally.

XII. WOUNDS

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 include:
- General symptoms; and
- Local symptoms.

The **local manifestations** are the following: **pain** (*dolor*), **hemorrhage** (*haemorrhagia*), **wound dehiscence** *(hiatus)* and **functional disturb**. 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 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, internal organ alteration and infection.

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);
- Stab wounds, caused by a pointed object (for example a bayonet, a drill, and a needle);
- Chopped wounds, caused by any heavy sharp object;
- Contusion wound is a result of the action of any blunt object (a stone, stick, wheel, hammer and other);
- Lacerated wound;
- Bite wound, produced by a dog, cat, men, or snake;
- Gunshot wound; and
- Compound wound, which combines the properties of different wounds.

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.
- 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.

c) High degree of contamination.

(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 wounds – with or without internal organs injuries; and
- Nonpenetrating wounds.

(5) According to the degree of contamination wounds are divided into:
- Aseptic wound – surgical wound, produced in sterile conditions.
- 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 developes in wounds in case of bacterial' concentration over 10^5 (100,000) microorganisms on 1 gram of tissue.

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:

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

The first phase (inflammation) is divided into **two periods:**

- **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.** Polymorphnuclear neutrophils phagocytize and destroy bacteria, damaged previously by granulocytes. 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). Epithelization 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:

- **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. 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. 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.
- **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 haematoma 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 cachexy 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), 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.

Prevention of secondary contamination of the wound.

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 consists of surgical hemostasis and restoration of tissue integrity by sutures application. In the postoperative period the treatment includes:
(1) Analgesics (different methods of administration);
(2) Prophylaxis of secondary infection (use of sterile bandages and antiseptics);
(3) Acceleration of wound healing (by early mobilization of patients);
(4) Improvement of patient's general state (correction of anemia, low serum protein level, blood circulation insufficiency, concomitant pathology).

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:
- Dissection of tissue;
- Exploration of wound channel;
- Excision of wound borders and bottom;
- Hemostasis;
- Reconstruction of damaged tissues and 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).

Kinds of sutures in wound closure. 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:
- 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. Cicatrisation 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.

Additional physical methods of purulent wounds cleaning include: (1) Pulsatile jet with antiseptics; (2) Ultrasound cavitation; (3) Surgical laser; (4) Treatment in controlled abacterial environment.

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 chlorhexidinum, 10% solution of NaCl (hypertonic saline solution) are used. Hydrophilic water-soluble ointments can be used from the 2nd-3rd days (Levosin, Levomikol, Mafenid-Acetat). Proteolytic enzymes (trypsin, hemotrypsin, hymopsinum) are also used for chemical processing of purulent wounds.

Granulating tissue is developed during the **phase of regeneration**.

Ointments, which contain antibiotics and stimulating substances (Solcoseril, Actoveghin, Tetracycline, Gentamycine, Vishnevsky ungvents) 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.

XIII. 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; coli bacilli; proteus (vulgaris, mirabilis, morgagni, rettgeri, inconstans); clebsiella; pseudomonas aeroginosa; and Gram-negative: *Escherichia coli, Proteus vulgaris, Proteus mirabilis, Pseudomonas aeruginosa.*
- Anaerobic clostridial infection: *Cl.perfringes; Cl.edematiens; Cl.histoliticum; Cl.tetani.*
- Anaerobic non-clostridial infection: Gram-positive: *Bacteroides fragilis, Peptococcus, Peptostreptococcus*; and Gram-negative: *Fusobacterium, Enterobacter.*
- 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;
- Bones and joint infections;
- Brain and its covers infections;
- Thoracic organs (lung, pleura, mediastinum, etc) infections;
- Abdominal organs infections;
- Infections of other organs and tissues (hand, breast, etc).

The development of surgical infection is produced by three elements:
- **Infectious agent (pathogenic microorganism)**;
- **Site of infection penetration**;
- **Response reaction of the human body**.

In evolution of purulent surgical infections great values have biological characteristics of microorganisms: **invasiveness**, **toxicity, virulence,** as well as **degree of contamination**.

There are two mechanisms **of response reactions** of the human body:

(1) **Nonspecific** protective mechanisms:
- Protective and bactericidal properties of the skin and mucous membranes;
- Saprophytic microflora of the human body;
- Humoral factors contained in plasma (Leikin, β-lysine, lysozyme, complement system);

- Cellular mechanisms of nonspecific defense (inflammation, phagocytosis).
(2) **Specific** mechanisms of protection include humoral and cellular immune response**.**

A number of factors can **diminish the activity of protective mechanisms:** the age of patients (children and elderly persons); sex (protective reaction of the female body is more perfect); concomitant diseases accompanied by immunodeficiency (diabetes, renal or hepatic insufficiency, malignancy, AIDS); anemia and hypoproteinemia; use of certain medications (immunosuppressants, cytostatics, 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:
- *Rubor* (hyperemia, redness);
- *Calor* (local hyperthermia);
- *Tumor* (swelling, edema);
- *Dolor* (pain);
- *Functia laesa* (functional disturb).

Specific clinical symptoms are used to **diagnose** the accumulation of pus inside an inflammatory infiltrate: softening, fluctuation, and diagnostic puncture.

Imaging diagnostic methods, used to reveal the profound purulent focus are the following:
- X-ray examination;
- Ultrasound;
- Computed tomography.

Purulent processes may be **complicated locally** by the development of:
- Necrosis;
- Lymphangitis;
- Lymphadenitis (adenophlegmon).

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. Middle molecules are considered to be a universal marker of intoxication.

GENERAL PRINCIPLES OF TREATMENT

The basic principles of **local treatment** are the following:

(1) Surgical debridement (evacuation) of purulent focus. 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.

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

(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.

(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. Three stages are distinguished in the evolution of furuncle: (1) infiltration, (2) abscess formation and rejection of necrotic core, and (3) scarring. **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.

A carbuncle is a large abscess (almost always staphylococcal) extending from several infected hair follicles into the subcutaneous fat. 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.

Hydradenitis 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 appear.

Abscess is a limited collection of pus in different tissues or body cavities. 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.

Phlegmon is an acute diffuse (not limited) suppurative inflammation of of cellular spaces (subcutaneous, intermuscular, retroperitoneal). 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: (1) submucosal, (2) subcutaneous (3) ischiorectal, (4) pelviorectal, and (5) 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 (1) erythematous, (2) bullous, (3) phlegmonous, and (4) necrotic forms. 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 – signs of "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.

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. Untreated lymphangitis is likely to lead to bacteremia.

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 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.

XIV. FELON AND PHLEGMON OF THE HAND

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.

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. The most common causative organism is *Staphylococcus aureus.*

Classification of felon

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.

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. The first sleepless night due to acute pain in the finger is an absolute indication for surgery (Voyno-Yasenetsky's symptom). 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 anaesthetic containing a vasoconstrictor such as adrenaline must never be used in digits surgery because of the risk of ischaemic necrosis; (3) Surgery should be performed bloodless (after application of tourniquet at the level of basal phalanx).

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).

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: (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. 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.

Bone and articular felon. 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. 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).

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 revieled;
- 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.

XV. SEMIOLOGY OF SKELETAL SYSTEM: TRAUMA AND INFECTION

FRACTURES

Fractures and dislocations are the most common musculoskeletal traumatic injuries of the limbs.

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 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 epiphysial 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.

DISLOCATIONS

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 (> than 4 weeks). Irreducible dislocation can not 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 of fracture/dislocation includes application of splint (medical device for immobilization), dressing (if open), adequate analgesia and intravenous fluid replacement. An attempt to reduce the fracture/dislocation should not always be made. The diagnosis of fracture/dislocation should be confirmed with biplane X-ray. Hospital treatment of fracture/dislocation consists of reduction and immobilization.

There are some specific diseases related to orthopedic infection – osteomyelitis, arthritis, and bursitis.

OSTEOMYELITIS

Osteomyelitis means the bone infection. Although the suffix *'myelitis'* denotes inflammation of the bone marrow, we use this term to include infection of all parts of the bone. There are **two ways of bone contamination**: haematogenous way, which results in primary acute osteomyelitis, and contact way – posttraumatic or surgery-related osteomyelitis. According to the evolution of disease, osteomyelitis may be classified as acute or chronic.

Posttraumatic osteomyelitis develops more often as a complication of open bone fracture or due to septic complication of elective orthopedic surgery – implant related infection.

Acute haematogenous osteomyelitis develops in childhood and is caused by infection spread through blood vessels from a septic focus. Osteomyelitis is usually metaphyseal and frequently involves the long tubular bones. Purulent inflammation

starts from bony marrow and develops until the detachment of periosteum and formation of subperiostal abscess. Subperiostal abscess may break into the soft tissue with formation of intermuscular and subcutaneous phlegmon.

There are **three clinical forms** – toxic, septic and local. The sudden onset of disease, high fever, intoxication, weakness, pallor, clouding of consciousness, repeated vomiting, dyspnea and tachycardia are characteristic of the toxic form. Local manifestations are indistinct – hyperesthesia during palpation or movement of the affected extremity. The similar clinical evolution is characteristic of the septic form with the formation of metastatic abscesses. The most common kind of acute haematogenous osteomyelities is the local form. There are clear local signs: severe permanent pain in the affected bone and restriction of movements.

The consecution of clinical examination: inspection of extremities, evaluation of active and passive motions, palpation and percussion of bone.

X-ray signs appear in two weeks after the onset of disease: exfoliation of periosteum, thickening of periosteum, lysis of normal bone structure.

Chronic osteomyelitis develops as the outcome of acute process. Rarely primary chronic forms (atypical forms) may develop: albuminous osteomyelitis, Brode's abscess and Garre's sclerozing osteomyelitis. The sequestration (formation of necrotic fragment of bone included into the so-called "sequester box") is the main sign of chronic osteomyelitis. The other typical sign is the formation of external fistula. Clinical manifestations depend on the stage of disease. Fever, local pain and abundant purulent discharge from fistula occur in the phase of exacerbation. In the phase of complications the pathological fracture, contraction of extremity, deformation and false joint formation may develop. Sequestration or the above mentioned complications on the X-ray confirm the diagnosis.

The treatment of osteomyelitis includes: antibacterial therapy, analgesics, detoxification, stimulation of immunity, immobilization, and surgery (debridement, drainage, sequesterectomy, bone resection).

ARTHRITIS AND BURSITIS

Arthritis is an acute or chronic inflammation of articulation. Infection can penetrate into the articulation from surrounding tissues (primary arthritis), in case of open damage (posttraumatic arthritis) and by means of blood or lymphatic circulation (secondary or metastatic arthritis). Serous, fibrinous and purulent forms are destinguished depending on the character of exudation.

General symptoms are high fever and intoxication. Local signs include: pain, painful and restricted motion, swell, half-bent position of extremity, local elevation of skin temperature, reddening, and occasionally fluctuation.

The treatment of arthritis requires immobilization, antibacterial therapy and repeated needle aspiration of joint. In case of severe purulent arthritis arthrotomy should be performed.

Bursae represent the endothelium-lined pillows that contain little fluid. Being situated between the paraarticular tissues that slide against each other, the bursae decrease the existing frictional forces. If the bursae are overloaded, they become inflamed, swollen and very painful. Such condition is termed **bursitis**.

Symptoms are red, warm, swollen and painful periarticular tissues confined to the one part of joint. The movement within normal values is usually possible.

The management consists of rest, ice, anti-inflammatory drugs and a compression dressing. Cases resistant to treatment require needle aspiration and steroid injection. Purulent bursitis and chronic bursitis may require surgical excision.

XVI. SEPSIS AND ANAEROBIC INFECTION

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.

Terminology. Sustained bacteremia, in contrast to transient bacteremia, may result in a sustained febrile response. Septicemia refers to the multiplication of bacteria in the bloodstream. Septicopiemia is bacteremia with purulent metastasis into the organs.

The experts of the ASCCM provided the practical definitions of "sepsis":

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, e.g. renal failure
Septic shock	Sepsis induced hypotension persisting despite resuscitation, presence of hypoperfusion abnormalities or organ dysfunction

Multiple organ dysfunction syndrome (MODS) is present if SIRS is associated with organ dysfunction, e.g. oliguria, hypoxia.

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.

Severe sepsis is one of the most common causes of death 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; the 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 of sepsis. Sepsis is the result of the interaction between microorganisms and host factors (cytokines). Released cytokines include tumour necrosis factor (TNF), interleukins (ILI1, ILI2, ILI6) and platelet activating factor (PAF).

The primary response to initial tissue injury is to mobilize macrophages and neutrophils. Cytokines signal systemic elements of inflammation resulting in activation of endothelium, complement system and blood coagulation. This is normal. 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 microtrombi, tissue hypoxia and ischemia, all of which can lead to the development of multiorgan system failure.

Classification of sepsis. 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, Streptococcus* sepsis, *E.coli* sepsis, *Klebsiella* sepsis, anaerobic sepsis (*B.fragilis*). Rarely, it is caused by *Candida*.

The source of infection that leads to sepsis may be acquired (intra-abdominal abscess) or endogenous (from the patient's own bowel). According to localization of

primary focus of infection there are: abdominal (14%), soft tissue, pulmonary (44%), urological (18%), gynecological, blood, and neonatal types of sepsis.

According to clinical evolution sepsis is classified into: fulminate form sepsis (5-7 days), acute sepsis (2-4 weeks), subacute sepsis (6-12 weeks), chronic sepsis.

According to pathological findings sepsis is subdivided into: bacteremia, septicemia, septicopyemia.

Clinical manifestations. Classical signs of surgical infection in combination with symptoms of SIRS and signs of MODS are the basis for clinical diagnostics of sepsis.

The signs of MODS are: low cardiac output and hypotension, need in pulmonary ventilation or ARDS, AST and ALT increased twice, oliguria less than 30 mL per hour, thrombocytopenia less than 100,000/ mm3, adynamic intestinal obstruction and stress bleeding, consciousness disturbances and other.

Diagnosis. Detection of microbial infection is necessary. On admission blood cultures of patients are obtained. Leukocytosis with left shift is a nonspecific diagnostic finding. Recently biochemical markers of sepsis have been proposed: cytokines (L-1, IL-6, TNF), C-reactive protein and procalcitonin. Ultimately, diagnosis of sepsis is clinical.

Treatment. The management of patients with sepsis includes general and local treatment. Local treatment consists of surgical processing of primary purulent focus. Antimicrobial therapy using broad-spectrum antibiotics is the mainstay of treatment. The mortality rate is substantially lower in patients who receive prompt and appropriate antimicrobial therapy.

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, but in the time of war it is more frequent. Anaerobic infection develops in case of dirty, smashed, gun-shot wounds. There are three kinds of anaerobic infection: clostridial infection, non-clostridial infection, tetanus

ANAEROBIC CLOSTRIDIAL INFECTION

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

Ethiopathogenesis. Gas gangrene is caused by an anaerobic, gram-positive, spore-forming bacillus (*Clostridium*). 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.

The incubation period of gas gangrene is short (<24h). 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. Production of gas dissects tissues along muscle bellies and fascial planes. Microorganisms are spread with oedematic 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.

According to clinical features anaerobic infection is divided into several groups: myonecrosis – classic form, necrotizing fasciitis and cellulitis – edematic form, and combined form.

Clinical manifestations. Patients complain of severe pain, sensation of compression, caused by edema. The skin is cyanotic. Muscles look like "boiled meat", and are edematous. Edema is developed quickly. 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.

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. Detection of alpha toxin in infected tissues through ELISA is a potential diagnostic tool.

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. 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), daily debridement. 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 processing of wound.

ANAEROBIC NON-CLOSTRIDIAL INFECTION

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

Clinical manifestations are presented by phlegmon with affection of subcutaneous adipose tissue, and muscles. Wide, rapid, and progressive spreading is the feature of this infection. Clinical manifestations are hyperemia of the affected area, and edema; muscles look like "boiled meat". Anaerobic infection often is manifested as suppuration, thrombophlebitis, abscess formation, and gangrenous destruction of tissue associated with gas. General manifestations are fatigue, fever, hypotension, oliguria, and high leucocytosis.

Diagnosis. Collection of specimens of anaerobic bacteria is important for the documentation of infection. Direct-needle aspiration is the best method. Transportation of specimens should be prompt in oxygen-free environments. Gram stain of the specimen provides important preliminary information. Gas-liquid chromatography is often used.

Treatment. The environment is controlled by debriding necrotic tissue, draining pus, improving circulation, and increasing tissue oxygenation. Hyperbaric oxygen therapy and antimicrobial therapy should be used. The B fragilis group is almost uniformly susceptible to metronidazole, carbapenems, chloramphenicol.

SURGICAL ASPECTS OF TETANUS
Pathophysiology. Tetanus results from infection with *C 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 *C 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"). Dysphagia 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. 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. Patients may report a sore throat with dysphagia. 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. However, surgical debridement has no value for tetanus.

Passive immunization with human tetanus immune globulin (TIG) shortens the course of tetanus and may lessen its severity. The treatment may require ICU admission and ventilatory 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.

XVII. DIAGNOSTIC PROCESS

Diagnosis (Gr.) means discernment, ability to distinguish. Diagnostics is a complex cognitive process and completes with analysis. 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 ist 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 (intestinal obstruction, portal hypertension, AIDS, SIRS 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 (functional tests of veins using tourniquet, thermometry, and anthropometry) also relate to semiology. In other words, semiology is equal to the first phase of diagnostic process and ends with preliminary diagnosis.

2nd phase of diagnostic process: laboratory tests, paraclinical (imaging) studies.

3rd phase of diagnostic process: 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 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.

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, 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.

XVIII. SURGICAL SEMIOLOGY OF HEAD AND NECK

Although the head and neck are relatively small parts of the total body area in adults, many vital important organs are located in this area, and both benign and malignant diseases may occur in all of them.

SEMIOLOGY OF THE HEAD

Headache is an extremely common symptom in patients with head pathology. In surgical practice headache may be caused by brain tumors, skull injury, cerebral abscess, and cerebral circulation impairment. Location, quality and severity, onset and duration of pain and associated symptoms such as nausea, vomiting, vertigo, neurologic and mental abnormalities should be taken in consideration for the assessment of headache. Examination of head should include the visual inspection and palpation. Knowledge of anatomy is helpful in process localization and description of pathological findings. Anatomical regions of the head are called according to the underlying bones. For example, we can divide the head into the frontal area, parietal area, temporal area, occipital area and so on.

The examination should start from **visual assessment** of the following:

(1) Position of the head. The forced position of the head may be a sign of diseases: anterior flexion occurs in ankylosing spondylitis (so called Behterew's disease), posterior flexion – in case of meningeal syndrome (skull injury, bacterial meningitis) and lateral flexion accompanied with face deformity – in case of congenital torticollis.

(2) Presence of any visible tumors. Local swelling of the head may be caused by cranial hematoma (subcutaneous or subaponeurotic accumulation of blood), benign and malignant tumors. Usually, a benign tumor such as lipoma and ateroma are mobile, round, have regular borders and do not fuse with the surrounding tissue. The malignancies such as basal cell carcinoma, melanoma and squamous cancer are relatively rare and have the opposite signs.

(3) Presence of any inflammatory process. An acute inflammatory process develops more often on the face (in nasal and upper lip region especially) due to plenty of subcutaneous fat. The typical example of acute local inflammatory process is a furuncle of face. Note that furuncles in the region of the upper lip may be complicated by acute thrombosis of intracranial cavernous sinuses.

NECK EXAMINATION

Visual inspection of the neck includes the assessment of length and volume of neck, color of skin, state of subcutaneous veins, and presence of any scars, visible lymph nodes and tumors. The length of neck depends on constitution – it is short in hypersthenics and it is long in asthenics persons. The volume of the neck depends on constitution too. Furthermore, neck volume can increase essentially in case of pathological mass, compression or thrombosis of the superior vena cava (so-called Superior Vena Cava syndrome) or in case of pathologically enlarged thyroid gland (so called goitre or struma). The volume of neck should be measured with a flexible tape measure.

Palpation of the neck allows the assessment of lymph nodes conditions in this region. The submandibular, cervical and supraclavicular lymph nodes should be investigated thoroughly. Physicians can assess by palpation the size, mobility and any tenderness of a node. Small, mobile, discrete, painless nodes are frequently found in normal persons. Small, invisible tumors can be detected by palpation too.

Palpation and auscultation are both applicable for the diagnostics of carotid artery diseases. Auscultation of systolic murmur or disappearance of pulse testifies to carotid

stenosis or occlusion. If these pathologies are suspected the angiography and ultrasound Duplex scan of carotid arteries should be performed mandatory. Atherosclerotic occlusive diseases of carotid artery are widely spread in the elderly and are responsible largely for a great part of acute cerebral vascular accident (stroke).

COMMON NECK ABNORMALITIES

Congenital defects and diseases. The following abnormalities can be found in the neck:

Torticollis (Grisel's disease or so-called stiff neck, wry neck). This is a congenital deformity of the neck with the following symptoms – fixed position of the head in posterior – lateral flexion, rotation of face in the opposite side, cicatrisation and contraction of the sternocleidomastidian muscle.

Medial cyst of the neck. This is a congenital disease caused by incomplete obliteration of thyrolingual duct. This kind of cyst is of a small size, grows slowly and usually is located between the hyoideus (sublingual) bone and the larynx.

Lateral cyst of the neck is the same type of cyst located however near the anterior border of the sternocleidomastoidian muscle. Inflammation and infection of cyst, both medial and lateral, may result in the formation of cervical fistula (medial or lateral respectively).

Cervical lymphadenopathy. The major lymph node groups are located along the anterior and posterior sides of the neck and in the submandibular area. If the nodes are quite big, you may be able to see them bulging under the skin, particularly if the enlargement is asymmetric (i.e. it will be more obvious if one side is larger than the other). To palpate, use four fingertips as these are the most sensitive parts of your hands. Examine the both sides of the head simultaneously, walking your fingers down the area in question while applying steady, gentle pressure. The major groups of lymph nodes as well as the structures that they drain, are listed below. The description of drainage pathways are rough approximations as there is frequently a fair amount of variability and overlap.

Nodes are generally examined in the following order:
- Anterior cervical nodes (both superficial and deep). Drainage: the internal structures of the throat as well as part of the posterior pharynx, tonsils, and thyroid gland;
- Posterior cervical nodes. Drainage: the skin on the back of the head. Also frequently enlarged during upper respiratory infections (e.g. mononucleosis);
- Tonsillar nodes. Drainage: the tonsilar and posterior pharyngeal regions;
- Submandibular nodes. Drainage: the structures in the floor of the mouth;
- Submental nodes. Drainage: the teeth and intraoral cavity;
- Supraclavicular nodes. Drainage: part of the thoracic cavity, abdomen.

Enlarged or tender lymph nodes suggest inflammation which may be caused by different inflammatory processes of the neck (such as furuncle, carbuncle, abscess, osteomyelitis and so on). Infected lymph nodes tend to be firm, tender, enlarged and warm. Inflammation can spread to the overlying skin, causing it to appear reddened. Malignancies may also involve the lymph nodes, either primarily (e.g. lymphoma) or as a site of metastasis. In either case, these nodes are generally firm, not tender, matted (i.e. stuck to each other), fixed (i.e. not freely mobile but rather stuck down to underlying tissue), and increase in size over time. Remember, that hard and fixed painless lymphatic nodes usually suggest malignancy. The location of the lymph node may help to determine the site of malignancy. Diffuse, bilateral involvement suggests a systemic malignancy (e.g. lymphoma), while those limited to a specific anatomic region are more likely to be associated with a local problem. Enlargement of supraclavicular nodes, especially on the left, suggests possible metastasis of abdominal malignancy,

commonly of gastric cancer. This symptom borrows the name of Virchow's metastasis. Unexplained enlargement of lymph nodes requires a careful inspection of the regions they drain. To make the differential diagnosis of regional and generalized lymphadenopathy the close visual assessment of nodes located in any anatomical area is necessary.

SEMIOLOGY OF THYROID GLAND

Inspection. Prior to palpation, look at the thyroid region. If the gland is quite enlarged, you may actually notice it protruding underneath the skin. To find the thyroid gland, first locate the thyroid cartilage, which is a mid-line bulge towards the top of the anterior surface of the neck. The thyroid gland lies approximately 2-3 cm below the thyroid cartilage, on either side of the tracheal rings, which may or may not be apparent on visual inspection. If you are not sure, give the patient a glass of water and on its swallowing watch this region. The thyroid tissue, along with all adjacent structures, will move up and down with swallowing. Note that normal thyroid is not visible.

On **palpation** of the thyroid gland the best position is behind the patient. From behind, place the fingers of both hands on the patient's neck so that the inde (II finger) are just below the cricoid cartilage. The patient's neck should be extended. Ask the patient to swallow. In this moment the thyroid gland rises under the doctor's fingers. The size, shape and consistency of the gland as well as the presence of any nodules or tenderness should be noted. Goitre, thyroid tumor and other may cause abnormalities discovered during thyroid examination.

XIX. SEMIOLOGY OF THORAX AND SPINE DEFORMITIES

DEFORMITIES OF THORAX

Cross section of the thorax in adult has an oval form, and its lateral diameter is larger than its anteroposterior one. Deformities of the thorax may be **congenital** and **acquired**. Pectus excavatum, pectus carinatum ("bird chest"), sternal fissure, and Poland's syndrome refer to congenital deformities.

The most common congenital deformity is **pectus excavatum.** The inspection of patient reveals the deformity, in which the body of the sternum is displaced posteriorly to produce a funnel-shaped depression. The depression is centered at the xiphisternal junction. Besides cosmetic deformity, patients have an exercise intolerance, atypical chest pain, dyspnea, bronchospasm, poor feeding, and arrhythmias.

Pectus carinatum – the protrusion deformities of the sternum. Most of patients are asymptomatic. The deformity usually is maximal below the nipple level.

Sternal fissure. The superior, inferior and complete sternal clefts are distinguished. The most common is superior sternal cleft, in which the defect is U- or V-shaped, usually extending down to the fourth costal cartilage.

Poland's syndrome is a unilateral absence or hypoplasia of the pectoralis muscles, breast, soft fat tissue and partial absence of the costal cartilages.

Emphysematous chest, paralytic chest and "boat" chest refer to acquired deformities.

Emphysematous or "barrel" chest has an increased anteroposterior diameter, with extension of the intercostal spaces. In inspiratory contraction neck muscles and abdominal muscles take part. It develops due to obstructive pulmonary diseases.

Paralytic chest is observed in patients with lung tuberculosis and in malnutrition. The atrophy of chest muscles and asymmetry of the clavicles and scapulas are noted.

"Boat" chest is characterized by deep depression into the superior and medium parts of sternum. This condition occurs in patients with syringomielia.

DEFORMITIES OF SPINE

Inspection of spine should be performed in two positions: (1) from side and (2) from behind of patient.

Viewed laterally anteroposterior spine deformities are easy to establish. Convexity (posterior direction) of spine is named kiphosis, concavity (anterior direction) – lordosis. Thoracic kyphosis, such as cervical and lumbar lordosis are normal.

Flattering of the spinal curvature (*dorsum platum*) is often caused by muscle spasm or by decreased spinal mobility.

Hyperlordosis – an accentuation of the normal lumbar curve – develops to compensate for the protuberant abdomen of pregnancy or marked obesity.

Hyperkyphosis – a rounded thoracic convexity – is common in aging.

Gibbus is an angular deformity of a collapsed vertebra and is caused by vertebral body fractures, metastatic cancer and tuberculosis of the spine.

Viewing the patient **from behind** every lateral deformity of spine should be noted in relation to an imaginary line, dropped from the spinous process of Th1 through the gluteal cleft. Lateral twist of the spine is called **scoliosis**. It should be distinguished from a tilt of the spine, caused by painful muscle spasms. In tilt of the spine a plumb line falls to one side of the gluteal cleft. In scoliosis the body has a compensated lateral curvature of the spine and a plumb line from Th1 drawn through the gluteal cleft.

To continue the examination patient is asked to bend forward to touch the toes. **Structural scoliosis** is associated with rotation of the vertebrae upon each other, and the rib cage is deformed respectively.

Functional scoliosis may develop to compensate other abnormalities such as unequal leg length. It involves neither vertebral rotation nor thoracic deformity. Functional scoliosis disappears with forward flexion.

Palpation of the spine is done when a patient is in standing and lying position. The spinous processes are palpated with a thumb.

XX. SEMIOLOGY OF 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).

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 beast 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 "milk line". 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.

Physical examination of the breast is the easiest during the 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 size and symmetry** of the breasts.
- **Skin color.** Redness of skin may suggest infection or carcinoma.
- **Thickening and edema of skin**. Unusually **prominent skin pores** ("orange peel" sign) is characteristic of breast cancer.
- **The contour** of the breast. Local protrusion or, in contrast, flattering of the contour may suggest inflammatory or cancerous mass.
- **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 (1) to raise her arms over her head and then (2) to press her hands against her hips. (1) Repeated inspection of patient with raised arms allows detecting prior invisible signs of retraction. (2) 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.

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 (**Velpeau method**). It determines:

- **The consistency of tissue.** Tender cords suggest mammary duct ectasia with inflammation around them; it is a benign disease called **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-th-7-th days before menses. Breast palpation reveals tender, nodular breasts. Kőnig's sign revealed: palpable mass in the upper right position disappears in repeated palpation in lying position.

- **Mass**. Any mass that is qualitatively 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 or the clock method;
  (2) Number of nodules;
  (3) Size in centimeters;
  (4) Shape (round or discoid, regular or irregular);
  (5) Consistency (fluid, soft, elastic, firm or hard);
  (6) Delimitation in relationship to surrounding tissues (well circumscribed or not);
  (7) Tenderness;
  (8) Mobility, with reference to the skin, pectoral fascia, and the underlying chest wall.

- **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. Serous discharge is caused by benign lesions, purulent discharge – by mastitis, bloody discharge – by intraductal papilloma, or underlying cancer.

- Assessment of the axillary **lymph nodes** is an important part of breast 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 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 (BSE)**. BSE is best carried out just after menstruation ends (monthly in nonmenstruating women). BSE 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.

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. It is commonly idiopathic, but may be associated with use of drugs, systemic diseases (liver dysfunction, Klinefelter syndrome, testicular tumor). Idiopathic gynecomastia requires surgery.

XXI. 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;
(2) Perforation of a hollow organ into the peritoneum;
(3) Intestinal obstruction;
(4) Intraperitoneal hemorrhage;
(5) Extraabdominal diseases may also be accompanied by severe abdominal pain.

COMPLAINTS AND HISTORY

Patient's age. Acute intussusception 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 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 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.

According to its origin, pain is divided into visceral and somatic pains.

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, mezogastric and hypogastric.

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.

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.

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 pain occurs in acute pancreatitis; sharp constricting pain which takes breath away – in 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. Pain accentuated by reclining and relieved by an upright position is often of retroperitoneal origin, as in pancreatitis.

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 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.

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 duodenal ulcer. Right hypochondriac pain related to meals suggests the presence of gallstones.

PHYSICAL EXAMINATION
General appearance. The pale or gray face covered with cold sweat suggests perforated 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 mask-like appearance of the face is named "Hippocratic face" or "abdominal face".

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.

Temperature. Subnormal temperature (35-36°C) is recorded in the diseases, associated by shock: pancreatitis, perforated ulcer or intraperitoneal hemorrhage. At the onset of appendicitis, the temperature is usually normal, but within a few hours it rise up to 37.3-37.5°C. When appendiceal perforation occurs the temperature may achieve 38°C. Very high fever is quiet unusual in the early stages of acute abdominal surgical diseases.

Inspection of the abdomen. 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.

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 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.

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.

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

Determination of iliopsoas rigidity. Hip flexion increases a severe pain – it is called psoas symptom.

Palpation of the loins may be done in a bimanual manner.

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.

XXII. TRAUMA. INJURIES OF HEAD, THORAX AND ABDOMEN

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 **nonpenetrating (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. 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 etc.

Trauma may be received under **production and non-production conditions**. **Iatrogenic injuries** are those, which are got during curative or diagnostic procedures (for example, perforation of the urinary bladder on 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. Injury of organs from different anatomical systems is classified as associated trauma. In addition, injuries caused by the action of two or more etiological factors induce 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:
(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 resorbtion 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;
(3) The period of late disturbances is characterized by dystrophic and sclerotic processes in the inner organs (kidneys, liver), ankilosis, 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. 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, hypertermia, bradycardia and bradypnoe, headache and dizziness, nausea and vomiting, retrograde amnesia, sleepiness and depression, loss of consciousness and neurologic coma refer to general signs. **Glasgow Coma Scale** has become an international standard used to assess level of consciousness after head injury.

In head-injured patient's **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.
(2) Concussion – it is a relatively mild form of brain injury, accompanied by only a brief loss of neurologic function.
(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 (contrecoup 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.

CHEST INJURIES

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 injuries of the thoracic cage. 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.

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).

(2) Pleural space injuries include various types of pneumothorax and hemothorax.

Simple (closed) pneumothorax is the presence of air in the pleural space. 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. 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.

Open pneumothorax or sucking chest wound, is an uncommon injury that produces a large chest wall defect. 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. Patients complain of dyspnea. Physical examination reveals decreased breath sounds and dullness on percussion of the injured side.

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.

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 CVP, 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 penetraiting 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 Blakmore 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.

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.

Organs of the abdominal cavity are 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.

Patients with abdominal organs injuries may present with abdominal pain, weakness, dizziness, and hemodinamic 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 peritoneal lavage is a critical step in the evaluation of blunt trauma (presence of blood and enteric contents). A hemodynamically stable patient can undergo radiographic and laboratory studies, ultrasound, CT, and 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 operation.

XXIII. SEMIOLOGY OF VASCULAR DISEASES OF EXTREMITIES

Vascular diseases of extremities can be divided into **arterial pathology, venous and lymphatic disorders.**

Diseases of arteries can occur with the syndrome of arterial insufficiency (or ischemic syndrome) and syndrome of aneurysmatic dilation of artery. Arterial insufficiency may develop suddenly (in case of arterial trauma, arterial thrombosis) and is called acute ischemia or gradually (in case of atherosclerosis, thromboangiitis, or nonspecific aortoarteriitis) being called chronic ischemia. Arterial aneurysm is referred to as an over 50% dilation of artery normal diameter. The enlargement of artery can be caused by traumatic injury – false aneurysm or be a result of inflammation and degenerative changes of arterial wall – true aneurysm.

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. Chronic venous insufficiency is usually caused by disturbed function of venous valves and loosed tone of vein wall. These abnormalities are either primary (varicose disease), or secondary as a result of vein thrombosis in the past.

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 fatiguability, sensory changes, restriction of mobility, sense of pulsation, heaviness in extremity, edema, and muscular cramps.

Sudden sharp pronounced pain in extremity is typical for acute ischemia. In the early stage of chronic ischemia, patients develop pain only during walking. After some minutes of rest, the pain disappears. This symptom is called "intermittent claudication". In the late stage of chronic ischemia, the pain becomes constant, increasing at nighttime and deprives the patient of sleep. Dependent position of affected limb reduces the pain because of moderate increase of blood flow. The "rest pain" testifies a forthcoming gangrene of extremity.

Deep vein thrombosis is accompanied by moderate pain. The pain is constant, localized in the calf, popliteal fossa and along big vessels. In case of thrombosis of varicose veins pain is located in the site of inflammation. The pain is not a leading clinical symptom of chronic venous insufficiency.

Sensory changes can be represented by paresthesia or by decreased sensibility. Patients with vascular pathology can complain of restriction (paresis) or absence (paralysis) of active movements in extremity. Neurological disorders are typical for acute ischemia.

Constant feeling of pulsation along the arteries of extremities may be a sign of arterial aneurysm.

Heaviness in the lower limbs (heavy legs) is typical for early stages of chronic venous insufficiency.

Swollen limb is a common symptom of many vascular diseases – final stage of chronic and acute ischemia and vice versa it is an early sign in case of deep vein thrombosis. Edema caused by chronic venous insufficiency and lymphedema increases gradually during several months or years, and become worse in upright position and decreases in recumbent position.

The following data should be analyzed during history taking of a patient with vascular diseases of extremities: smoking, coronary artery disease, cardiac infarction and arrhythmia, wounds in the region of big arteries, surgical interventions.

INSPECTION

Chronic ischemia is associated with atrophy of calf muscles, loss of hair growth and thickening of the toenails. In severe stage the color of the skin is extremely pale or cyanotic. Changes in the foot color, caused by critical 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.

In case of **acute ischemia,** extremity is pale, the skin is mottled ("marmoreal" limb), and subcutaneous veins are collapsed. Active movements in the distal parts of extremity are limited or impossible. The flexion contracture of extremity develops in the advanced stage of acute ischemia.

Both acute and chronic ischemias, invariably lead to the development of gangrene if untreated. The term of **gangrene** denotes the specific type of necrosis which is characterized by:
- It develops only in the tissue which is in contact with external environment;
- Affected tissue becomes black-colored;
- It affects an organ or an anatomic region as a whole.

There are two types of gangrene – dry and damp (moist) gangrene.

Dry gangrene 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, absence of demarcation line, fast spreading and association of infection.

In case of **peripheric arterial aneurism** the inspection reveals some 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 *"phlegmasia cerulea dolens"* and can result in damp venous gangrene.

In case of **thrombosis of superficial veins,** inspection reveals dilated and waved 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 varicose veins, edema of malleolar region and brown-colored spots on the internal surface of the shin (hyperpigmentation), venous ulcer.

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. Arterial ulcers are small, painful and localized in the toes.

Lymphedema is usually associated with significant increase of limb volume, limb deformity and marked skinfolds (elephantiasis).

PALPATION

Palpation is of diagnostic importance in peripheric arterial diseases, because the pulse is absent or diminished distally to arterial occlusion or stenosis.

In case of acute ischemia the "6P" complex of symptoms can be determined – pain, pallor, pulselesseness, poikilothermia, paresthesia, paralysis.

Pulsatile tumor is a symptom of arterial aneurysm. In case of arterio-venous fistula 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 – symptom of Branham.

In case of superficial venous thrombosis, careful palpation can reveal painful infiltrate along the vein and dense clots into the venous lumen. Deep vein thrombosis is associated with two symptoms: pain during squeezing of calf muscles and during dorsal flexion of foot (Mozes's and Homans's symptoms).

Hackenbruch's test (cough test) and Trendelenburg test can be used to determine vertical reflux through the saphenous vein. Permiability of deep veins is tested by Delbet-Perthes test. In advanced stage of chronic venous insufficiency palpation reveals hardening of subcutaneous fat on the shin (lipodermatosclerosis).

Palpation can distinguish venous and lymphatic edema. Inability to pinch a skin fold at the base of the second toe is a symptom characteristic of 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.

XXIV. DIABETIC FOOT: THE SURGICAL ASPECTS

INCIDENCE

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.

Approximately 10-15% of diabetic patients develop plantar ulcers during their life. In diabetics the risk of amputation is 15 times higher in comparison with non-diabetic population. Up to 70% of all amputations performed worldwide are done in patients with diabetes mellitus.

CAUSES AND FORMS

Disturbed metabolism of glucose provokes at least **3 negative effects** upon the lower limbs. (1) **Atherosclerosis** of peripheric arteries begins early in patients with diabetes mellitus – at a young age, occlusions and stenoses of the vessels develop more quickly and involve a large number of arteries, including the calf and foot vessels. Occlusive and stenotic arterial lesions lead to the decreased perfusion of affected limb and development of chronic ischemia. In diabetes affection of the peripheric nervous system includes somatic and autonomic neuropathy. (2) Motor deficit as a part of **somatic neuropathy** results in weakness of plantar muscles and development of osteal-articular 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, 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 – **neuropathic form** (diagnosed in approximately 70% of cases), **ischemic form** (10%) and mixed or **neuro-ischemic form** (20%).

In the development of diabetic foot and especially plantar ulcers the role of disturbed plantar biomechanics is importnant. 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.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

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 (paresthesias, unusual sensation, decreased sensibility); infection related complaints (pulsatile pain, fever). Absence of pulse on at least one artery requires a consultation of vascular specialist. To confirm the sensory loss, the Semmes-Weinstein 10g monofilament test should be performed. Sensibility to touch with 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. In case of diabetic foot, the 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 O2 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.

Bacteriology. Any form of diabetic foot is often associated with infection. There are several pathogens responsible for 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), CT, scanning with leukocytes labeled with indium (111In) and MRI. The last method has been considered as a method of choice.

CLASSIFICATION

Generally, infections of diabetic foot can be divided into **superficial** – which affects the skin and subcutaneous adipose tissue and **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 frequently used classification of diabetic foot, based on grade of tissue destruction is **Wagner-Meggitt classification**: Grade 0 – completely epithelialized ulcer, 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.

In 2000, in the **Texas University** (USA) another classification system of diabetic foot was developed. This classification is based on the variants of clinical association of various grades of diabetic foot destruction with ischemia or infection. There are 4 grades of foot lesions: grade 0 – epithelialized lesions; grade 1 – superficial lesions not involving tendon, capsule or bone; grade 2 – lesions penetrating to tendon or capsule; grade 3 – lesions penetrating to bone or joint. Each grade may be combined with one of 4 categories: A – no infection and ischemia; B – association with infection; C – association with ischemia; D – association with infection and ischemia.

The current comprehensive classification of diabetic foot was proposed in 2004 by K.A.Treece and has the acronymic name **S(AD) SAD**: Size (Area and Deep) of lesion, Sepsis – the presence of infection, Artheriopaty – the presence of circulatory disturbances and Denervation – symptoms of diabetic neuropathy. Each component of classification is categorized according to the severity of pathology from 0 to 3 points. S(AD) SAD classification is an excellent instrument for patient stratification in scientific research.

TREATMENT

Treatment of diabetic foot is always complex and depends on the clinical form of disease. For **correction of disturbed circulation,** the following drugs are used: antithrombotic (Aspirin, Clopidogrel, Ticlid), prostanoids E1 (Vasaprostan), statins (Simvastatin, Atorvastatin). **Surgical revascularizations** are done by either endovascular percutaneous transluminal angioplasty with stent implantation or open surgical by-passes. Results of surgical arterial interventions among diabetics are worse than in non-diabetics due to diffuse nature of disease, prevalence of distal lesions and vascular wall calcification.

The cornerstones of infection control are long term **antibacterial treatment** (Cefalosporine, Fluorchinolone, until 2-6 weeks in case of osteomyelitis) and, of course, adequate surgical debridement. **Local treatment** includes application of moist dressings with chemical antiseptics and proteolytic enzymes. Any type of treatment should be combined with foot off-load realized by patient bed rest, plaster cast application or using of crutches or wheel-chair. New and perspective method of chronic purulent wounds treatment is vacuum assisted aspiration (VAC). The method is based on insertion of polyurethane foam dressing cut to fit the wound cavity exactly. After that the wound is sealed with an adhesive dressing and negative pressure (125 mm Hg below atmospheric) generated by suction pump. VAC results in increased local blood flow, reduced edema and bacterial colonization, rapid formation of granulation tissue and wound contraction.

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). 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 (ex. PDGF), 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.

Success of diabetic foot treatment 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 glycolizated 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.

XXV. 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, 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.

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, entero-cutaneous 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.

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 percentage = 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 month	7.5%	> 7.5%
6 month	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 (1st grade)
	35-39.9 (2nd grade)
Morbid obesity	40 and more (3rd 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.

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) = 1610 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 - 1300 kcal/d, 60kg - 1450 kcal/d, 70kg - 1600 kcal/d, 80kg - 1750 kcal/d, 90kg - 1900 kcal/d, 100kg - 2050 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 feeding is **indicated** for patients who have functional gastrointestinal tract, but are unable to sustain an adequate oral intake. Enteral feeding may be **contraindicated** in patients with intestinal obstruction, ileus, gastrointestinal bleeding, severe diarrhea, vomiting, enterocolitis, high-output enterocutaneous fistula.

Nasogastric, nasoduodenal, nasojejunal, gastrostomy and jejunal **feeding tubes** are available for the administration of enteral feeding products.

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.
- 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.

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 pancreatitis).

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

TPN solutions are administered as a 3-in-1 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).

TPN 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. 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: pneumotorax, 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.

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.

XXVI. BASIC TRANSPLANTOLOGY

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

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.

Ukrainian scientist **Iurii Voronoi** performed a number of kidney transplants taken from deceased donors. The first successful transplantation of kidney from a living donor was carried out by **Joseph Murray** and colleges in 1954.

In 1959 **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.

COMMON TERMS

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.

Depending on the location where the transplanted organ or tissue may be placed, the transplantation is divided into orthotopic and heterotopic.
- 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).

Prosthetics is the use of non-organic or synthetic materials for replacement of tissues and organs.

Replantation – is a surgical reattachment of a limb or its segment, separated from the body.

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.

In **free flap transplantation or free grafting**, the prepared flap (usually skin) loses initially its vascular connection with the donor site.

TRANSPLANT REJECTION

Full compatibility of transplanted organs or tissues is achieved only in autologous and syngeneic transplant. In case of allogenous and xenogenous 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.

There are three categories of **histocompatibility antigens** relevant for transplantation:
- ABO antigen system;
- Human leukocyte antigens (HLA), which are considered the major histocompatibility antigens;
- Histocompatibility antigens of minor significance.

Antigenic ABO 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 successfull 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 ABO incompatibility or as a result of the presence of recipient formed anti-HLA antibodies in the blood. 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. Nonimmune factors are responsible for pathogenesis of chronic rejection. Myointimal proliferation of graft arteries is characteristic, which leads to ischemia and organ fibrosis.

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 ABO 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 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 a organ loss occurs and retransplantation is in need.

ORGANS DONATION

There are two categories of donors: **living donors**, which can donate a kidney or parts of organs such as the kidney, liver, pancreas, 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** are rarely used.

Signs of brain death.

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;
- After extraction the organ is either transplanted in short time 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 body.

The **period of warm ischemia** starts after removing the organ intended for transplantation from a cold preservation solution.

In organ transplantation the main problems are the following:
- Providing the vital activity of the transplanted organ;
- Prevention of organs rejection;
- Technically perfect execution of transplantation;
- The volume of post-operative intensive care;
- Monitoring of patients after transplantation throughout their life;
- An effective immunosuppressive therapy.