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**STUDENT'S GUIDE
TO STATE EXAMINATION
QUESTIONS
FROM THE SURGERY**

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**MASARYKOVA
UNIVERZITA**

Dear Readers,

You now hold the result of the dedicated work of numerous physicians from a wide range of medical, not only surgical, departments. Each author is deeply committed to facilitating your orientation within surgical topics and fostering a comprehensive understanding to guide you effectively toward accurate surgical reasoning and action.

The aim of this work extends beyond merely reflecting the dynamic evolution of the field of surgery. Above all, it seeks to impart key insights rooted in the rich surgical tradition of Brno – to authentically highlight the historical significance of what we consider the "crown jewel" of surgery at the Faculty of Medicine, Masaryk University.

These materials are by no means a substitute for the mandatory and recommended literature; rather, they serve as an overview and supplement, designed primarily as a guide to assist you in preparing for questions on the state examinations in surgery (*the number of the official state exam question can be found in brackets after the chapter title*).

Finally, we kindly request your feedback – an evaluation of each chapter in terms of both scope and content. Your input is invaluable and will greatly contribute to the improvement of this resource. We welcome any suggestions for revisions, proposals for additions or improvements, whether by condensing or expanding particular sections. Beyond constructive criticism, we are, of course, equally appreciative of any questions or positive feedback.

Your feedback will be invaluable in guiding this text's further development and refinement. Please send your thoughts to our email address: **chirmuni@group.muni.cz**, and we will respond to each message.

We wish you joy in achieving grown understanding and the creative inspiration it brings as you use this resource to enhance your surgical knowledge and skills.

The Editors,

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RECOMMENDED LITERATURE:

PAFKO, Pavel. *Základy speciální chirurgie*. 1. vyd. Praha: Galén, 2008, 385 s.

ISBN 9788072624027.

ŠNAJDAUF, Jiří a Richard ŠKÁBA. *Dětská chirurgie*. 1. vyd. Praha: Galén, 2005, 395 s.

ISBN 807262329X.

HOCH, Jiří a Jan LEFFLER. *Speciální chirurgie: učebnice pro lékařské fakulty*. 2. vyd. Praha:

Maxdorf, 2003, 224 s. ISBN 8085912066.

MARKO, Ľubomír a Peter KOTHAJ. *Atlas miniinvazívnej chirurgie a chirurgickej endoskopie*. 1.

vyd. Banská Bystrica: Marko, 1998, 156 s. ISBN 8096807609.

KABELKA, Miroslav. *Dětská chirurgie: učebnice pro lékařské fakulty*. 1. vyd. Praha: Karolinum,

1992, 320 s. ISBN 8070665610.

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INTRODUCTION TO SURGERY

MEDICAL HISTORY AND PHYSICAL EXAMINATION IN SURGERY (15)

Veverková Lenka

Looking for clues– finding answers: Correct diagnosis, Adequate treatment

Right questions, Attention to details, keep it simple, Key is in the details, Communication
Patient-doctor relationship

Patient in pain, anxious, different social and mental status, new situation, non-specialist

Gaining the patient's trust is the essence

- ✓ Direct vs Indirect /Why? When? /
- ✓ monologue vs. dialogue /time/
- ✓ ideally, a combination of both

For doctors:

- ✓ Routine, lack of time, lack of energy, hopelessness...
- ✓ BUT can give you the first hint of diagnosis even before touching your patient
- ✓ Picture of patient's health status
- ✓ Probable need for the next step in the diagnostic algorithm
- ✓ Base a possible diagnosis
- ✓ General plan for the following treatment

Parts of medical history

In the case of an acute abdomen, we determine the day and time of the onset of the symptoms, how it changed over time, and what treatment has been given so far.

Whereas in the case of injuries, it is necessary to know the mechanism of injury.

Subjective symptoms: pain, vomiting, loss of gas and stool, loss of appetite, thirst, cough, shortness of breath, weight loss, difficulty urinating.

- a) Family history (FHx)
- b) Past Medical History (PMHx), in Past Surgical History (PSHx)
- c) Current treatment and medications
- d) Allergies
- e) Alcohol and smoking history
- f) Social history (SocHx)
- g) Obstetric and gynaecological history (OB/GYN)
- h) Review of systems (ROS)

Acute abdomen

- Severe abdominal pain with sudden onset, usually developed from full health. It can usually cause death if not treated.
- Complicated classification, many causes, numerous clinical signs

What should we ask about?

- a) Diet
- b) Stool and flatulence
- c) Fever
- d) Time
- e) Drugs used
- f) Urination
- g) Nausea, vomiting

PAIN- leading sign

Visceral x Somatic

Location, Irradiation, Type, Migration, Position of patient

Physical examination

- ✓ Inspection
 - a) Contour
 - b) Breathing movement
 - c) Colour
 - d) Scars, hernias, tumours
 - e) Skin lesions
 - f) Position of patient
 - g) General status
- ✓ Auscultation
 - a) Stethoscope
 - b) Intestinal movements – type, frequency, localisation...**4 quadrants**
 - c) Normal...frequent...silent...silence
 - d) High pitch noise
 - e) Mechanical obstruction, paralytic ileus, diffuse peritonitis, ascites, etc.
 - f) Lung + heart noises (differential diagnosis)

✓ Palpation

- a) Superficial vs. Deep /gently!!!/
- b) Start at the point farthest from the pain location
- c) Communicate with patient
- d) Bimanual palpation
- e) Pain, organs, tumours, hernias, pain, bowel movement, fluid (ascites)

Peritoneal irritation, peritoneal signs

Local vs. Diffuse

Severe finding – stay alert!!!

Often, this will mean surgery....

BUT only sometimes... (AP, localized diverticulitis...)

Blumberg's sign

Pleniés's sign

Rowsing's sign

Défense musculaire

✓ Percussion

Abdominal sounds heard upon percussion:

Tympany

A loud hollow sound should be obtained in the quadrants

They are loudest over the gastric bubble and intestines

Dullness

A short, high-pitched sound is heard over the liver

Spleen and distended bladder

Hyperresonance

Louder than tympany

Heard over air-filled or distended intestines

Flat

A very soft, short, abrupt sound

Heard when no air is present in the anatomical site due to the presence of muscle or tumour mass

✓ **Rectal exam**

perianal region (perianal abscess, fistula, fissure)

Internal bulks

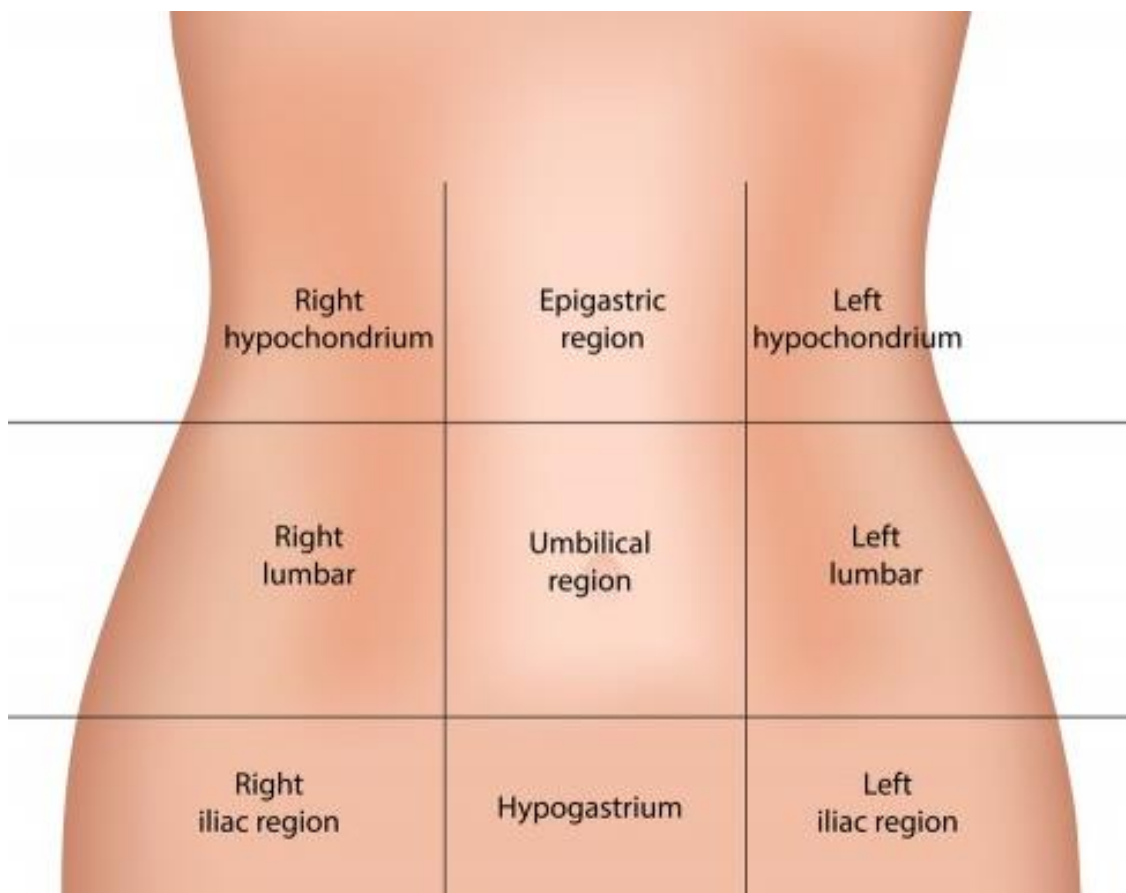
(polyps, tumours, haemorrhoids)

Stool (mucus or blood in stool, steatorrhea, consistency)

Intraabdominal pain

CONCLUSION:

- Ask appropriate questions
- Listen, take your time
- Combine information - symptoms-syndromes
- Get the patient's trust
- Gentle and thorough clinical examination – IAPPR
- Re-evaluate
- PC, CT, lab \neq head and hands



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EXAMINATION METHODS IN SURGERY (5)

Potrusil Martin

The examination algorithm of the surgical patient represents a key aspect in the process of differential diagnosis and subsequent adequate therapy. It is essential to proceed in an organized, deliberate and dynamic manner. **Investigation methods vary in ability to correctly diagnose different pathologies (sensitivity, specificity, accuracy) and invasiveness. The examination is indicated according to the suspected pathology. Sometimes, we do not need to have a precise diagnosis; it is sufficient to decide whether the patient must be operated on immediately (upper or lower GIT perforation), other times we need to know the type and extent of the disease precisely (Peripheral Artery Disease - PAD, or cancer). The basic investigation methods are medical history, physical examination, laboratory methods, imaging methods (contrast or no contrast), endoscopic methods, and diagnostic surgeries.**

Despite modern technology, medical history and physical examination are integral parts of the clinical assessment, and these steps must not be neglected or minimized. We always obtain family (hereditary and systemic diseases, malignancies, including age of diagnosis, e.g. carcinoma in those under 50 years of age, etc.) and personal history, focusing on known diseases, the nature and severity of their course, previous surgical interventions, traumas, gynaecological history, pharmaceuticals used, blood transfusions administered, allergies, risk factors (including abusive behaviour), occupation and social status, physiological functions.

The physical examination consists of the patient's inspection (general condition, consciousness, posture, mimicry, antalgic position, dyspnoea, cyanosis, icterus, erythema, oedema, ecchymosis, pallor, lividity, diaphoresis, nutritional status, trophic status, contours, disfiguration, scars, tissue vitality, etc.), auscultation (phonation, murmurs, respiratory phenomena, qualitative and quantitative character of peristalsis, etc.), palpation (systematically of all regions with a progression from quiet areas to painful areas, assessing superficial and available deeper structures, their consistency and sensitivity, pulsations, vortices, looking for resistance, fluctuation, crepitations, vibration, temperature changes, etc.), percussion (pain, sound phenomena), per rectum (sphincter tone, resistance, bowing, stenosis, sensitivity, cervix, prostate, ampulla content, etc.). A significantly defining symptom in surgery is **pain**. The onset of pain can be immediate (seconds), e.g. in perforation episodes, bleeding, torsion (adnexa, testes), progressive (minutes to hours), e.g. intussusception, strangulation, colic, acute pancreatitis), indolent (4 to 24h), e.g. appendicitis, colitis. Visceral pains (autonomic nerves) are difficult to localize (distension, spasms). Colicky pains are caused by distension of the hollow viscera, and the patient is restless and unable to find a position of relief. For somatic pains (somatic nerves),

the patient can specify locally (peritoneum). Peritoneal irritation causes constant pain, worsening with every movement, so the patient seeks a stable position of relief. Pain irradiation is caused by convergent nerve afferentation from different areas. Localization of pain in the epigastric region (greater splanchnic nerves and vagus nerve - nn. splanchnici maiores and n.vagus) originates from organs embryonically differentiated from the foregut, i.e., stomach, D1-2 duodenum, liver, pancreas. Mesogastrium (lesser splanchnic nerves and vagus nerve - nn. splanchnici minores and n.vagus), organs of the embryonic midgut supplied from the basin of the a.mesenterica sup. (e.g. appendicitis - pain around the umbilicus with transfer to the right hypogastrium). Hypogastrium pains (pelvic sympathetic nerves and lesser splanchnic nerves - pelvic sympaticus and nn. splanchnici minores) originate from organs nourished by the inferior mesenteric artery (a.mesenterica inf.) ranging from the aboral transversal colon (c.transversum) to the oral rectum, including the testes and ovaries. Pain in the perineum (internal iliac arteries - aa. iliacae int.) may originate from the aboral rectum.

Unique physical examination findings may be associated with specific pathologies. Murphy's sign of acute cholecystitis is positive when inspiration during palpation in the right upper quadrant produces sudden pain at the palpation site. Défense musculaire is a painful contraction of the abdominal wall muscles during palpation based on diffuse peritonitis. Percussive pain on the abdominal wall during peritonitis is a Plenies' sign. Many signs will help localize the area of ongoing peritonitis: Blumberg sign is rebound tenderness at the site of inflammation following adduction of the palpating hand (peritonitis), Rovsing sign is positive for pain at the McBurney point elicited by palpation in the left hypogastrium (acute appendicitis), and obturator sign is a pain in the hypogastrium caused by flexion and external rotation of the right thigh in supination (pelvic inflammation/abscesses), the iliopsoas symptom is pain on elevation and extension of the lower limb against resistance (appendicitis/retrocecal abscess), the Chandelier symptom is characterised by severe pain in the hypogastrium and pelvis during cervical movements (pelvic inflammation). Charcot triad is a pain in the right upper quadrant, icterus and fever (cholangitis), Courvoisier sign is palpable hydroptic gallbladder and concomitant icterus (periampullary tumours), Cruveilhier sign (caput medusae) is varicose and dilated venous plexus in the umbilicus (portal hypertension), Cullen sign is the finding of a periumbilical hematoma (hemoperitoneum /severe acute pancreatitis), Grey-Turner sign is bruising on the lateral sides of the abdomen (bleeding in acute pancreatitis), Kehr sign is left shoulder tenderness on palpation in the left upper quadrant (hemoperitoneum, esp. in splenic haemorrhage).

Laboratory tests are a routine part of the diagnostic algorithm in the surgical patient. Interpretation of the results is always with respect to the current clinical status of the patient and his dynamics. A basic biochemical panel from a blood sample includes the following parameters: renal function - urea, creatine, CKD-EPI, ionogram (sodium, potassium, chloride, in indicated cases calcium, phosphorus,

magnesium), serum osmolarity, liver function - bilirubin (total, conjugated), ALT, AST, GGT, ALP, (ammonia), pancreas - lipase, amylase (3 times increased level compared to the norm in acute pancreatitis). Total plasma protein, albumin, prealbumin, cholesterol, and triglycerides are evaluated to assess nutritional status. Myoglobin and troponin T (myocardium) may be helpful in muscle ischemia states. Interpretation of acid-base balance requires ions, pH, pCO₂, HCO₃, base deficit, O₂ saturation - in arterial blood vs. venous blood, as well as lactate level and oxygenation index (PaO₂/FiO₂). Markers of severity of inflammation - routinely CRP, leukocytes in blood count (additionally procalcitonin, presepsin, IL-6, TNF α in serum, ICIS score). Carbohydrate metabolism - plasma glucose (indicated HbA1c - glycated haemoglobin, protein C). Blood count - leucocytes, erythrocytes, haemoglobin, haematocrit, platelets (differential budget where appropriate). Coagulation - prothrombin time, fibrinogen, aPTT, and D-dimers (thrombosis/ischemia) indicated further antithrombin, anti-Xa, anti-IIa and others). Other specific parameters include antibiotic levels (e.g., vancomycin, linezolid), hCG (pregnancy), and toxicology. Haemoculture - aerobic, anaerobic (from the periphery and central catheter). Serology - panfungal antigens, viruses, parasites. Tumour markers - CA 19-9, CEA, CA 125, CA 72-4, AFP, CYFRA, PSA, etc. Urinary examination - urinary sediment, culture, fractional excretion of ions, if necessary, urinary proteins, osmotically active substances (tubular function). Punctate examination - markers (e.g., in ascites - colour, appearance, leukocyte concentration, polymorphonuclear concentration, total protein, glucose, lactate dehydrogenase), culture, mucin (present at GIT communication), 3 times higher amylase concentration (pancreatic fistula), 3 times higher bilirubin concentration (biliary fistulation), urea, creatine (concentration in punctate vs. serum level - urinary admixture). Microbiological microscopy - early Clostridia (wound impression on glass), mycobacteria, fungi, parasites (aspirate, tissue sample).

Modern surgical diagnostics uses a wide range of **imaging modalities**. The appropriate modality is indicated based on anamnestic data and careful physical examination. Methods of first contact with the surgical patient include skiagraphy (X-ray), ultrasonography (US), or computed tomography (CT). Other radiological studies are selective.

Skiagraphy

Plain or contrast imaging. Positive contrast (barium sulfate, iodine derivatives of benzoic acid), negative contrast (air, water), and double contrast (positive and negative). Importance of different projections and patient position. Functional imaging - forced joint positioning (indirect signs of soft tissue damage), defecography, transit-time, intravenous urography, cholangiography (PTC, peri-operative), ERCP, angiography, etc. Skiagraphy in traumatology. The limitation of X-ray examination is limited specificity. Diagnosis: pneumonia, pleural effusion - fluidothorax, pneumothorax, emphysema, tumour staging, hiatal/para oesophageal herniation, perforation of the digestive tube

(pneumoperitoneum), obstruction of the small and large intestine, ileus, perforation of the digestive tube (pneumoperitoneum), foreign bodies.

Ultrasonography

2D/3D imaging. Doppler evaluation of blood flow. Duplex ultrasound combines 2D imaging and Doppler. Intravenous contrast ultrasonography, e.g. SonoVue (focal liver lesions). Imaging of solid and cystic lesions (e.g. thyroid, kidneys), abnormal tissue masses (e.g. tumours, parenchymal metastases), fluid collections and free fluid (e.g. ascites, pleural effusion, abscess), visualization of motion (e.g. aneurysm pulsation, cardiac echo), assessment of blood flow, detection of dilatation (e.g. bile ducts, hydronephrosis), size measurement, examination of the biliary system (lithiasis, wall thickening/inflammation), breast examination, navigation of percutaneous interventional procedures (aspiration, biopsy), using special probes - endosonography (EUS), transrectal (TRUS), ultrasound elastography. Ultrasonography is limited by body habitus (thick layer of adipose tissue), bone shielding and gas.

Computed tomography (CT)

Native vs. oral or intravenous contrast examination dose-dependent and timing of phases (arterial, venous, late arterial and venous. Caution (Cave)!! Allergy to IV iodine contrast and Acute Kidney Injury (AKI) or Chronic Kidney Disease (CKD) with high levels of urea and creatinine. Spectral CT. Examination of deep-seated organs and structures, e.g. brain, spine, lungs, mediastinum, retroperitoneum, urgent imaging in polytrauma mode (whole-body spiral CT), additional examination in the sudden abdominal episode, localization and staging of tumours, examination of topographic relationships of organs before planned surgery (e.g. tumour invasion, extent and size of abdominal aortic aneurysm, anatomical anomalies), CT angiography (vascular pathology, bleeding, ischaemia, embolization), CT guided interventions (puncture, drainage, biopsy, ablation, etc.), CT virtual endoscopy.

Magnetic resonance

High magnetic field (1 to 5 Tesla). IV contrast for MRI is called gadolinium. Caution (Cave)!! Metallic foreign bodies and claustrophobia are contraindications for MRI. Visualization of the CNS (brain, spinal cord), soft tissue tumours (trunk, extremities), biliary anatomy, intestinal stenoses, strictures, fistulations (MR enterography), pelvic organs (e.g. staging of rectal tumours), musculoskeletal system (e.g. knee joint), evaluation of blood flow (MR angiography)

Endoscopy, diagnostic laparoscopy/video-assisted thoracoscopy, diagnostic laparotomy, thoracotomy.

ANTISEPSIS, ASEPSIS, STERILISATION (37)

Veverková Lenka

Although the following slides may appear theoretical, infection prevention is a fundamental aspect of medical practice.

Asepsis – Refers to the complete absence of pathogenic organisms, including viruses, bacteria, yeast, spores, and fungi.

Antisepsis – A comprehensive set of procedures and measures designed to neutralize infectious agents on the body's surface, mucous membranes, and within human tissues.

Antisepsis is achieved by applying antiseptic substances to the skin, body cavities, or wounds. These substances effectively destroy pathogens while remaining non-toxic to tissues and should not exhibit allergenic, mutagenic, teratogenic, or carcinogenic properties.

Disinfection – The process of eliminating infectious agents (all pathogens) through physical and chemical methods.

This approach focuses on reducing most microorganisms in the environment and on non-living objects, such as in rooms, air, equipment, and instruments.

Disinfectants may, however, exhibit tissue toxicity.

Objective: It aims to interrupt the transmission route from the source of infection to a susceptible individual.

- **Preventive** (anticipated infection)
- **Repressive** (existing infection)
- **Focal disinfection** (ongoing, terminal)
- **Full disinfection** – effective against viruses, spores, and helminth eggs
- **Partial disinfection**

Principles:

- Knowledge of the hygienic-epidemiological situation
- Measures proposed by a physician and carried out by qualified personnel
- Understanding of the infection source
- Understanding of the transmission route

Disinfection Methods:

- **Physical desinfection:**
 - **Heat:** Boiling for 30 minutes or 20 minutes with increased pressure
 - **Radiation:** UV lamps for operating rooms and clinics, air filtration

- **Chemical disinfection:**

- **Factors:** substance concentration, duration of action, ambient temperature
- **Solutions:** immersion, wiping, evaporation into the environment
- **Aerosols:** continuous spraying

Chemical Disinfectants:

- Phenols
- 70% - 75% Ethyl or Isopropyl Alcohol
- Halogens
- Glutaraldehyde

Hand Disinfection:

- Sterillium classic pure, Septoderm solution, gel, and spray, Promanum – isopropyl ethanol

Skin Disinfection:

- Betadine solution – iodinated povidone in aqueous or aqueous-alcohol solution
- Braunol (PVP) – used for disinfecting skin, mucous membranes, open wounds, and body cavities
- Braunoderm, Jodisol – 2%-3%, Gutasept F, G spray – quaternary ammonium compound with propanol and benzalkonium chloride, Mesosept, Septonex, Softasept

Disinfection of Surgical Instruments:

- Chirosan, Chirosan Plus – a powder-based preparation with peracetic acid, offering a broad bactericidal spectrum, including spores, used for disinfecting instruments and equipment
- Chiroseptol – an aldehyde-based solution, free of formaldehyde

Surface Disinfection:

- Deconex, Bacillol AF, Hexaquart

Floor Disinfection:

- Desam GK, Desam OX – oxygen-based solution
- Chloramine – chlorine-based solution, which can irritate skin, eyes, and respiratory system

Sterilisation – A set of procedures aimed at destroying or removing all microorganisms, including spores and viruses, from the environment.

- **Physical Sterilisation:**

- **Boiling under pressure:** Boiling for 20–30 minutes at a temperature of 135°C and pressure of 0.3 MPa; used for metal instruments, glass, and suture material.
- **Steam sterilisation** – In autoclaves, where steam at a temperature of 125–140°C and pressure of 0.07–0.25 MPa is used for sterilising linens, dressings, and instruments.

- **Hot air sterilisation:** Conducted at temperatures of 140–220°C; used for glass, porcelain, powders, oils, and certain metal instruments. Performed in metal boxes or thin-walled glass containers, with duration depending on temperature (20 minutes at 180–200°C).
- **High-vacuum infrared sterilisers:** Operate at 280°C for 7 minutes.
- **Radiation sterilisation:** Utilises gamma rays or accelerated electrons, enabling sterilisation of heat-sensitive items (e.g., needles, syringes).
- **Chemical Sterilisation:**
 - Based on the sterilising effects of chemical agents at temperatures of 20–100°C, suitable for items that cannot withstand higher temperatures (e.g., rubber, plastics).
 - **Formaldehyde:** A gas with bactericidal and virucidal properties, acting on surfaces only (lacks penetrating ability) and released through evaporation.
 - **Ethylene oxide:** A volatile liquid whose vapours are highly flammable and explosive and can irritate the respiratory tract and conjunctiva. Suitable for instruments, textiles, catheters, optical devices, and suture materials.

TAKE HOME MESSAGE:

Asepsis Basics

- **Personnel**

Use of clean and dirty zoning, wearing protective equipment, Hand washing, wearing a sterile coat and gloves, Glove changing

- **Patient**

Preparation of patient and operating field, Washing the skin, Shaving, Operational field disinfection, If necessary - prophylaxis - antibiotics

Antisepsis: A term sometimes used synonymously with asepsis but also refers specifically to the use of antiseptics.

Antiseptics are agents that chemically reduce or destroy pathogens and are applied to skin and wound surfaces.

Disinfectants are chemical substances applied to inert surfaces.

WOUNDS - CLASSIFICATION, TREATMENT, HEALING, COMPLICATIONS (13)

Pospíšil Jan, Mitáš Ladislav

Introduction

A wound is an injury to the integrity of tissue - skin, mucous membrane or even hard tissue. We describe for each wound's the location, size, shape, direction, edges and depth.

Distribution of wounds

Wounds can be divided in several ways, e.g. according to the damage to the skin cover, the depth of the damage, penetration through the body cavities, the extent of the damage, aetiology, etc.

1. According to the damage to the skin cover

- a. Open wound
- b. Closed wound

2. According to the depth of the disability

- a. Superficial (simple) – affecting the skin and subcutaneous tissue
- b. Deep (complicated) – involving muscles, tendons, vessels, nerves, etc.

3. According to the relationship to the body cavities

- a. Non-penetrating
- b. Penetrating (through parietal peritoneum, pleura, dura mater, synovial membrane)

4. According to contamination of the wound with impurities

- a. clean wound (a non-traumatic wound that is without inflammation, without a breach of aseptic catheters, e.g. after hernioplasty)
- b. clean, contaminated wound (non-traumatic wound where one of the systems - gastrointestinal, respiratory, urinary tract was opened, e.g. after a planned colectomy)
- c. Contaminated, dirty wound (traumatic wound with the opening of some non-sterile systems (e.g. gastrointestinal) with massive contamination)
- d. dirty, infected wound

5. According to the length of healing

- a. Acute
- b. Chronic – healing for more than 6 weeks

Types of wounds according to the type of damage

1. **Mechanical aetiology**

a. **cut wound** (vulnus scissum) – caused by pressure and pull of a sharp object (cut - incision), bleeding and burning pain. It tends to be shallower at the edges than in the middle part

- b. **secant wound** (vulnus sectum) – caused by the impact of a sharp object, deeper structures can also be bruised. It has the same depth throughout the entire extent of the wound
- c. **gunshot wound** (vulnus sclopetarium) – caused by a bullet or projectile. It has a shot, a firing channel, and possibly shot itself. It tends to be contaminated with gunpowder, which complicates healing
- d. **puncture wound** (vulnus punctum) – caused by the penetration of a sharp object into the depth of the body, with the risk of damage to deep structures and organs and infection. It always has a puncture and a puncture channel
- e. **bite wound** (vulnus morsum) - can imitate a stab wound or contusion, infection and poor wound healing often occur
- f. **contusion wound** (vulnus contusum) – caused by the compression of soft tissues between a solid object and a person's own skeleton.
- g. **laceration wound** (vulnus lacerum) – is caused by pulling and subsequent tearing of the skin, it bleeds little, it can also be contaminated
- h. **contusion and laceration wound** (vulnus contusolacerum) - is caused by overcoming the elasticity of the skin by the pressure and pull of a blunt instrument on the surface of the body.

2. Thermal etiology

This category includes **burns** (combustiones) and **frostbite** (congelationes)

3. Chemical etiology

This includes necrosis caused by **acids** (coagulation necrosis) and **alkalis** (colliquation necrosis).

4. Radiation damage ev electric current

Wound healing

Wound healing can be primary or secondary.

Primary wound healing (by primary intention; per primam intentionem) - takes place in 6 phases – coagulation and inflammation, fibroplasia, angiogenesis, epithelization, maturation of collagen fibres and then completion of healing. The duration of healing is approximately 18 months.

Secondary wound healing (by secondary intention; per secundam intentionem) - especially gaping wounds or wounds healing in unfavourable conditions - infection, foreign body, poor blood circulation, diabetes, anaemia, immunodeficiency, hypoproteinaemia, deficiency of trace elements and vitamins, etc. Wounds are covered with fibrin, then granulation and epithelialization

Wound treatment

First aid

For extensive wounds, first ensure vital functions. Subsequently, a sterile bandage and temporary stoppage of bleeding (pressure bandage, pressure points, tourniquet) were applied.

Definitive wound treatment

The treatment takes place under aseptic conditions (possibly under local anaesthesia or general anaesthesia). First, revision of the wound, ascertaining the functionality of tendons, the integrity of blood vessels, nerves or other tissues, or their treatment.

A primary suture can be performed to clean wounds. If contamination or infection in the wound is suspected, a primary delayed suture can be performed (in 3-7 days). In case of clear contamination or infection in the wound, it must be left for secondary healing. Subsequently, after its cleaning, a secondary suture can be performed (early - after 2 weeks, possibly delayed - after more than 3 weeks).

During secondary healing, it is advisable to maintain its specific environment in the wound and keep it clean (regular debridement and toileting of the wound). It is possible to use a wide range of different types of wet healing (preparations with silver, etc.) or perform negative pressure therapy.

Complications of wound treatment

Wound infection – a common complication of wound healing. It manifests itself with standard symptoms of inflammation. The source is most often pyogenic bacteria (e.g. *St. aureus*, *S. pyogenes*, *Pseudomonas aeruginosa*, *E. coli*, etc.). The basic treatment is dissolution of the wound, drainage and flushing with antiseptics. In case of more extensive inflammation, we start antibiotics (ABX) therapy – first, a broad-spectrum ABX, then, according to its sensitivity.

The most feared are Clostridium infections - e.g. *Cl. tetani* (it is necessary to think about vaccination or passive immunization) or *Cl. perfringens* (causing flatulence, which has a high mortality despite maximal therapy)

Gap (dehiscence of the wound) - if it is an uncomplicated dehiscence, it is possible to perform a secondary suture. If an infection cause the dehiscence, local treatment and ABX are necessary first..

Morning collection

- Seroma – a collection of serous fluid – it can only be aspirated
- Haematoma – if it is expanding, it must be evacuated. There is also a risk that the hematoma becomes infected.

Snakebite

Treatment of a wound after a snake bite has its specifics. It is necessary to wash the wound and temporarily increase the bleeding by pressure centrally. Subsequently, immobilization, serum (in the case of a viper bite, it is already used minimally) + central analeptics, cardiotoxic and corticoids are necessary. Possibly also strangle the affected limb and move the "choke" 10-20 cm more centrally for 15 minutes.

SURGICAL INFECTION (ABSCESS, PHLEGMON, CARBUNCLE, FURUNCLE, PANARITIUM) (18)

Veverková Lenka

Surgical infections are associated with substantial morbidity and mortality. Patients with SSI are twice as likely to die, 60% more likely to be admitted to the intensive care unit, and more than five times more likely to be readmitted to the hospital after discharge

Definitions -Surgical infection

- ✓ **Infection:** the process whereby organisms (e.g. bacteria, viruses, fungi) capable of causing disease gain access and cause injury or damage to the body or its tissues
- ✓ **Pus:** a yellow/green foul-smelling viscous fluid containing dead leukocytes, bacteria, tissue and protein
- ✓ **Abscess:** localized collection of pus, usually surrounded by an intense inflammatory reaction
- ✓ **Cellulitis:** a spreading infection of subcutaneous tissue

Necrotising and non-necrotising infections

- according to the presence of necrosis - necrotic, non-necrotic
- according to aetiology - monobacterial (mostly streptococci and clostridia), polybacterial - aerobic + anaerobic

Necrotising fasciitis

- Post-operative wound infection (SSI) resulting in necrotic fascia and aponeuroses in the wound (often accompanied by thrombosis of the skin veins resulting in necrosis of the skin covering).
- mixed infection, the main pathogens being streptococci, but also G- aerobic and anaerobic bacteria, staphylococci.
- Clinically: general symptoms (fever, chills, shivering), local wound pain, the wound is tense, swollen, reddened, at revision purulent secretion is expelled and necrotic fascia is revealed, at its disintegration eventration occurs - then it is Heal as a laparostomy with mesh, wound healing per secundam - wait for separation of necrotic fascia or necrectomy, preferably after necrectomy of the VAC system.- After healing, hernias in the scar are common.

Anaerobic necrotising soft tissue infection

- Clostridial myonecrosis, anaerobic cellulitis, streptococcal myonecrosis, - mixed anaerobic infections (Fournier's gangrene, Plaut-Vincent angina, Meloney's ulcer)
 - Local treatment (excision of necrotic tissue, oxidative solutions in the vagina) and systemic treatment (combination of ATBs - most often with metronidazole - basic ATB against anaerobes).

Pyogenic infections

Purulent infections caused by staphylococci - inflammation and the formation of abscesses), streptococci - tends to cause unbounded spread of inflammation, phlegmon, and pneumococci:

1. abscess -- Central necrosis and colic, the cavity - typical of staphylococci.
2. phlegmon -- Infiltrating, unbounded inflammation - typical of streptococci
3. Empyema -- Pus in a preformed cavity

Putrid infection

Caused by putrefactive bacteria,

typical is brownish, putrid smelling pus, pungent to feculent, typically early infections (abdominal surgery, diabetic defects, pressure ulcers...

Pathogens:

1. enterobacteria EC, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, genus *Proteus*, *Acinetobacter*, *Serratia*, *Morganella morganii*, including causative agents of infectious diarrhoea (genera *Salmonella*, *Shigella*).
2. less virulent clostridia (G+ anaerobes - *Clostridium tertium*, *Fallax*, *Putrificum*, *Aerophoetidum*...).

Anaerobic infections

are characterised by gas formation:

1. Clostridium (G+ rods - *Clostridium perfringens welchii*, *Cl.oedematiensis*, *septicum*, *histolyticum*...)
 - *Clostridial myonetrois* (gas gangrene) - Muscle necrosis (under fascia)
 - Epifascial phlegmon (anaerobic cellulitis) - infection of necrotic tissue above the fascia.
2. G+ anaerobic streptococci, peptococci, peptostreptococci - streptococcal myonetrois
3. anaerobic G-infections (*Bacteroides fragilis*, *Fusobacteria*, *Leptotrichia*) - mixed infections (Spirilli), purulent processes.

INFECTION-SPECIFIC

- They are characterised by a specific macro- and microscopic picture:
- Tuberculosis (pulmonary, extrapulmonary) - *Mycobacterium tuberculosis* (Koch's bacillus)
- Stage 3 syphilis - *Treponema pallidum*
- Leprosy (lepromatous, tuberculoid) - *Mycobacterium leprae* (Hansen's bacillus)
- Based on clinical manifestations, also actinomycosis and anthrax (its cutaneous form - has differential diagnostic importance).

Hospital-associated infections

Infections related to hospitalization /hospital- related infection/

- ✓ Patient in the time of admitting to hospital without signs of infection

- ✓ Infection after dismissal of patient, evidently related to hospitalization
- ✓ 5 – 10% of in-door patients
- ✓ Polyresistant bacterial agents
- ✓ I.C.U. patients
- ✓ Difficult and costly therapy

Most frequent HAI

- ✓ **urinary** (UC, retention – pain) (40%)
- ✓ **respiratory** (pain, ventilation) (20%)
- ✓ **SSI** (superficial, deep) (25%)
- ✓ **Blood stem** (septicemia, catheter associated)
- ✓ **Bed sores**
- ✓ **Gastroenteritis** (post-ATB)

Wound infections

A wound infection is defined by the US Centre for Disease Control and Prevention (CDC) as surgical site infection (**SSI**). This is further defined as:

- **Superficial incisional SSI** – infection involves only skin and subcutaneous tissue of incision.
- **Deep incisional SSI** – infection involves deep tissues, such as facial and muscle layers.
- **Organ/space SSI** – infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during the operation. **Infection must occur within 30 days** of the surgical operation.

And at least one of the following is present:

- Purulent discharge from the surgical site
- Purulent discharge from wound or drain placed in wound
- Organisms isolated from aseptically obtained wound culture
- Must be at least one of the signs and symptoms of infection – pain or tenderness, localised swelling, or redness/heat.

Other factors that play a role in wound infection:

- **General patient characteristics**
 - Age, obesity, malnutrition
 - Endocrine and metabolic disorders
 - Hypoxia, anaemia
 - Malignant disease
 - Immunosuppression

- **Wound characteristics**
 - Nonviable tissue in wound
 - Foreign bodies
 - Tissue ischaemia
 - Haematoma formation
- **Operative characteristics**
 - Poor surgical technique, long operation time (>2 hours)
 - Intraoperative contamination, Prolonged preoperative stay
 - Hypothermia

Treatment of infections

We combine a variety of modalities, which should be appropriately related and mutually reinforcing

Surgical treatment

- Removal of the lesion - excision, ectomy (appendectomy, cholecystectomy, amputation)
- incision and drainage with removal of pus, necrectomy
- according to the rule "**ubi pus ibi evacua**".
- For sinus infections, a similar effect is achieved by exposing them to the outside - laparostomy, pleurostomy.

ANTIBIOTIC TREATMENT STRATEGY IN SURGERY (36)

Laššáková Zuzana, Ledvina Tomáš, Musilová Karolína, Horváthová Beáta, Horváth Teodor.

The antibiotic treatment strategy in surgery follows the general principles of antibiotic policy. The aim is to use antibiotics efficiently, safely (watch out for allergies) and cost-effectively, while minimising the emergence of antibiotic resistance. The basis of rational antibiotic therapy is 1/ the correct choice of antimicrobial agent (according to the proven or suspected agent), 2/ the dose of the antibiotic (ATB), 3/ the route of administration (oral, intravenous, topical), 4/ the time interval between doses and 5/ the optimal duration of therapy. It is important to monitor the results of therapy and possibly change or extend it (into a combination).

In surgery, we use antibiotics prophylactically or therapeutically. Prophylactic administration of ATBs means their administration in an attempt to prevent infectious complications. Prophylaxis is divided into 1/ primary (we want to prevent the emerge of infection, i.e.: administration of antibiotics before surgery), and 2/ secondary (prevention of recurrence or reactivation of a pre-existing infection, e.g.: cyclic therapy with rifaximin (Normix) in a patient with recurrent diverticulitis). Primary prophylaxis is the most widely used in surgery, based on the principles of Malek's protected coagulum. We administer ATB before the procedure, well in advance, and repeatedly for longer procedures, using both i.v. and p.o. forms of ATB. We choose it for procedures where infectious complications are 1/ severe, e.g. when foreign material is used (osteosynthetic material, vascular endoprostheses, mesh in hernia surgery), in thoracic surgery, or 2/ frequent, e.g. in intestinal surgery, heavily contaminated wounds. Two examples from practice: 1/ Half an hour before osteosynthesis, we prophylactically administer i.v. cefazolin (it acts mainly on gram-positive cocci), because we are afraid of wound infection from the skin, where there is a predominance of these microorganisms. 2/ Before elective resection of the intestine, which we first mechanically prepare (laxatives and infusions), we use p.o. rifaximin (it acts locally in the intestine) and half an hour before surgery broad-spectrum antibiotics i.v. (amoxicillin-clavulanate) or a combination of antibiotics (ceftriaxone + metronidazole), because we expect a diverse bacterial flora including anaerobes.

The therapeutic use of ATB can be divided according to the known pathogenic agent into 1/ empirical 2/ presumptive and 3/ targeted. In empirical therapy, we do not know the pathogen or its sensitivity to ATBs, but we try to estimate the most likely agent and its sensitivity to ATBs according to the type of infection, localization and local epidemiological situation. For presumptive therapy, we have only limited data on the pathological agent; we do not know everything. E.g. microscopy result from haemoculture (G+ cocci), without culture and sensitivity. In targeted ATB therapy, we initiate antibiotic treatment exactly according to the cultured agent and its sensitivity (one way to determine

sensitivity is to determine the so-called MIC = minimum inhibitory concentration). According to the phase of treatment, antibiotic therapy can be divided into 1/ initial antibiotic therapy and 2/ de-escalating ATB therapy. In initial ATB therapy, we often do not know the exact causative agent, so it is an empirical therapy; we use broad-spectrum ATBs, often a combination of several ATBs to broaden the spectrum of action and increase the potency of the ATB; we use bactericidal ATBs; in severe infections we choose parenteral administration (more effective) and rather higher doses of ATBs. In de-escalation therapy, we already have the culture result and sensitivity, so we can substitute broad-spectrum ATBs with targeted, narrower-spectrum ATBs (they have fewer adverse effects, e.g. on the gastrointestinal tract, and lower potential for resistance), or switch from combination to monotherapy, or reduce the dose or dosing interval.

The division of ATBs into three groups based on their effect (so-called PK/PD targets) is essential for a properly guided therapy: 1/ time-dependent ATB (for the effect it is important to maintain a sufficient concentration of ATB for the longest possible time above the MIC of the pathogen, e.g. 2/ concentration-dependent ATBs (for the effect is important to reach concentration of the ATB above the MIC, and a decrease to zero concentration before the next dose, e.g. aminoglycosides, fluoroquinolones), 3/ exposure-dependent ATBs (both concentration and time above the MIC are important for the effect - i.e. total exposure, most other ATBs). For time-dependent ATBs, it is typical to use shorter dose intervals (according to the half-life of the drug), prolonged infusions, or even continuous administration of ATBs. However, for concentration-dependent ATBs, administration of higher doses at longer time intervals is more typical. ATBs with a narrow therapeutic window (i.e. risk of underdose or, on the other side, toxicity, e.g. aminoglycosides, vancomycin) are routinely monitored by periodic level sampling (TDM, usually in collaboration with a clinical pharmacist).

If an ongoing infectious disease is suspected, the surgeon will perform physical and laboratory examinations (CRP, leu, procalcitonin, urinary sediment), imaging (X-ray for osteomyelitis, pneumonia, ultrasonography or CT scan for abscess) and take a sample for bacteriological examination before administering the antibiotic. He determines the most likely pathological agents and accordingly deploys empirical ATB therapy. He monitors its efficacy (clinically, laboratory, imaging, local findings) and possibly adjusts it accordingly to the results of culture, sensitivity and success of the empirical therapy. It is always necessary to bear in mind the possibility of a viral aetiology of the infection (enteritis, pneumonia, herpes zoster, meningitis, encephalitis). The most common causes of antibiotic therapy failure are incorrect choice of ATB, failure to achieve a sufficient concentration of ATB (PK/PD target) due to incorrect dosage and/or interval, inappropriate dosage form, or premature termination of ATB therapy. Given the globally increasing trend of antibiotic resistance, it is necessary

to use interdisciplinary collaboration between clinicians, clinical microbiologists and clinical pharmacists.

COMMENTS AND CONTROVERSIES:

Eradication is the treatment of colonization (using therapeutic ATB regimens) to prevent the colonization from becoming an infection or the disease from progressing to a chronic stage, e.g.: eradication of *Helicobacter pylori*).

The division of bacteria can be viewed from different perspectives (clinical, microbiological).

The older way of dividing bacteria into 1/ Typical, 2/ Atypical, 3/ Spirochetes and 4/ Mycobacteria, is used more in clinical practice. It is based on the clinical and paraclinical manifestations of diseases caused by these groups of bacteria, types of used antibiotics usually also differ.

The newer division is more microbiologically relevant. Bacteria are divided according to their relationship to the atmosphere, Gram staining and cultivability into 1/ Gram-positive (aerobic and anaerobic), 2/ Gram-negative (aerobic and anaerobic), 3/ mycobacteria (tuberculous and non-tuberculous), 4/ other (mycoplasmas, chlamydia, rickettsiae, spirochetes).

BASIC SURGICAL PROCEDURES (12)

Gregora Jakub, Horváth Teodor

Surgery uses medical knowledge to treat diseases with hands, instruments and technology. This is done under aseptic conditions in the process of surgical intervention - surgery. We distinguish between 1/ **invasive** surgical procedures, i.e. interventions in which the patient's tissues are cut or the wound is sutured. 2/ In **non-invasive procedures**, the integrity of the body surface is not disturbed. Examples are the reduction of fractured bones or the application of a bandage.

Division of surgical procedures

Surgical procedures are generally divided according to several criteria:

A. Indication

Absolute - there is no other treatment option of the disease, for example, bleeding from an injured blood vessel or choking from foreign body aspiration. The most serious is the **vital indication** when failure to perform surgery is immediately life-threatening, and without surgery, the patient would certainly die, for example, peritonitis from the perforation gastrointestinal (GI) tract.

Relative - there are other treatment options, but surgery gives a better and more lasting result than conservative treatment

Prophylactic - surgery prevents possible complications of the disease in the future, e.g. mastectomy in women with BRCA1 or BRCA2 mutation, i.e. removal of the organ before the malignancy develops.

B. Purpose

Diagnostic - to detect the underlying disease or to find out its extent, e.g. diagnostic laparoscopy

Therapeutic - cure of the patient or relief from difficulties, i.e. surgery 1/ **radical resection** - complete removal of the cause of the disease and cure of the patient, 2/ **palliative treatment** - achieving relief from symptoms of the disease - e.g. gastro-enteric anastomosis in duodenal obstruction by an inoperable tumour.

C. Time considerations

Emergency (urgent, unplanned) - for conditions arising suddenly, often from full health with a rapid onset and life-threatening course. These are divided into 1/ **Urgent, which** must be performed immediately, e.g. haemoperitoneum for splenic injury, and 2/ **Acute**, which can be performed in matter of hours, e.g. acute appendicitis.

Elective - after thorough examination and preparation of the patient, e.g. surgery of inguinal

hernia, varices, uncomplicated cholecystolithiasis (gallstones).

D. Minimally invasive vs open surgery

Minimally invasive surgery using a video camera, images projected on a monitor and special endoscopic surgical instruments inserted into preformed cavities through small incisions via working inputs (= **ports**). The compound names of these procedures are formed by the first part of the word describing the space in which the operation takes place, e.g. **laparoscopy** (abdomen), **thoracoscopy** (chest), and **arthroscopy**(joint). Additionally, minimally invasive methods also include **robotically assisted surgery**.

Open (traditional) surgery - access to the operated area is achieved through a surgical incision of the skin and tissues at a particular anatomical site, e.g. laparotomy, thoracotomy, or craniotomy.

Nomenclature of surgical procedures

Ablation - removal of a superficial part of the body, organs e.g. nail, polyp, breast

Amputation - cutting off or removal of the end part of the body or an organ, e.g. a finger, a leg, a rectum.

Anastomosis - the mutual connection between two hollow organs or structures (vascular, intestinal), depending on the method, there are three types of anastomosis: *end-to-end*, *side-to-side* or *end-to-side*

Biopsy - taking a tissue sample for histological examination - either 1/ **needle biopsy** under imaging control under local anaesthesia (ultrasound or CT guided biopsy), e.g. transparietal biopsy (transabdominal liver or transthoracic lung biopsy) or 2/ **surgical biopsy**, e.g. excision from a retrosternal mass by the parasternal route in the diagnosis of lymphoma or skin excisional biopsy in the diagnosis of inflammatory breast cancer.

Dissection - splitting of tissue or anatomical structure, e.g. fistula, adhesion.

Enucleation - excision of a well-defined structure or deposit, e.g. cyst, eyeball

Evacuation - draining e.g. haemarthrosis from a knee; pus from an empyema or abscess

Exarticulation - removal of a limb or part of a limb at its joint

Excision - cutting out diseased tissue or a sample from a tumour. If it describes an operation with removal of an organ then it is formed by the suffix **-ectomy**, e.g. appendectomy, mastectomy, gastrectomy.

Excochleation - scraping with a surgical spoon, most often of a cavity, abscess, fistula.

Extirpation - complete removal of a pathological formation that is clearly circumscribed, e.g. atheroma – take care to perform adequate lancet-like excision of the skin to prevent complications.

Extraction - removal of tissue or foreign material, e.g. teeth, chip, gauze.

Incision - surgical opening, cutting

Procedures involving surgical cutting or opening of an organ or tissue end with the suffix **-tomy** according to its localisation, e.g. laparotomy, thoracotomy, or fasciotomy.

Injection - injection of a substance into tissues or blood vessels with a needle

Osteosynthesis - surgical treatment of fractures, fixation of bone fragments with fixation material

Puncture - impaling of a vascular lumen, body cavity, lesion or organ, e.g. venepuncture, abdominocentesis, thoracocentesis, transbronchial puncture.

Repositioning - returning organs or their parts to their correct anatomical position, e.g. repositioning of bone fragments.

Resection - removal of part of a tissue or organ

Trepanation - opening, drilling a hole into the skull, medullary cavity of a bone, or draining a subungual haematoma

Transplantation - transfer of taken tissue or organ from a donor site to a recipient site (recipient), e.g. Thiersch or dermal-epidermal skin graft, kidney transplantation.

Replantation - a procedure in which a separate body part is sewn back together, e.g. finger replantation
the suffix -ostomy is bringing the lumen of a hollow organ out through the skin or abdominal wall, e.g. gastrostomy, colostomy, tracheostomy.

How the names of the operations are formed

The name of the surgical procedures are formed by putting the operated organ or area first, e.g. gastro- (stomach), chole- (bile duct), cholecysto- (gall bladder), colon- (large intestine), masto/mammo- (breast), laparo- (abdomen), hystero- (womb), arthro- (joint), nephro- (kidney), angio- (blood vessels), thoraco- (chest), hepato- (liver), and others. The suffix of the compound word is then used to describe the actual procedure performed on the organ or in the area, i.e. -ectomy, -stomy, -tomy (see above for definitions); there are also suffixes used, such as **-scopy** (viewing with an instrument or device), **-plasty** (reshaping, resizing or strengthening), **-pexy** (attaching, fixing to a specific place), **-raphy** or **plication** (strengthening with sutures), **-section** (incision), **-centesis** (surgical puncture).

Examples of surgical procedures:

Laparoscopic appendectomy - minimally invasive removal of the vermiform appendix;
Hemicolectomy - removal of half of the colon; Ileostomy – bringing the lumen of the ileum out through the abdominal wall; Abdominocentesis - peritoneal cavity puncture; Thoracocentesis - thoracic cavity puncture; Prostatectomy - removal of the prostate gland; Gastropexy - attachment (sewing) of the stomach to the abdominal wall in a hiatal hernia; Choledochotomy - longitudinal incision of common bile duct during bile duct revision; Herniorrhaphy - hernia repair, suturing the hernia gate; Angioplasty - widening of a narrowed artery with a balloon or surgically; Nephrostomy - insertion of a thin catheter through the skin into the renal pelvis, it is used to derive urine from the hollow renal system into a

collecting bag; Segmentectomy - removal of the part (segment) of organ, most commonly of the liver or lungs; pancreaticoduodenectomy - removal of part of the pancreas and duodenum.

COMMENTS AND CONTROVERSIES

Routine areas for abdominocentesis are located at the interface of the right meso- and hypogastrium or at the centre of the umbilicus-symphysis junction. It is performed under local anaesthesia (LA). It is necessary to perform fractionated evacuation, especially in massive ascites. The abdominal drain drains into *a collection bag*.

Chest drainage – provides a method of removing air and fluid substances from pleural cavity. Thoracocentesis is routinely performed in patients with **pleural effusion (fluidothorax)** diagnosed on imaging, (Chest X-ray, ultrasound, CT scan, or on clinical examination by auscultation or percussion. Chest drains insertion is performed at the upper end of the lower rib in the corresponding intercostal space. It is performed under LA. “Lege Artis” is the fractional evacuation of effusion. The chest drainage - flows underwater sealed drain.

LAPAROSCOPY, THORACOSCOPY, ARTHROSCOPY (7)

Ledvina Jan, Plánka Ladislav

Modern methods of minimally invasive surgery (MIS). Compared to classical, open surgeries, they are performed from small incisions using optics and endoscopic instruments. This fact brings with it the following advantages and disadvantages.

Advantages: reduced healing time (\approx reduced hospitalization and disability), reduced pain, less blood loss, lower risk of postoperative adhesions and incisional herniation

Disadvantages: visceral injury during trocar insertion, lack of tactile sensation and visual depth perception (compensated by 3D imaging), higher demand on technique and surgeon's skill, limited possibility of safe surgery of advanced diseases, risks associated with capnoperitoneum.

Basic division of MIS: diagnostic - in case of diagnostic hesitation to clarify the diagnosis
therapeutic - with the aim of eliminating the pathological condition

Indications for conversion to classical open surgery: massive bleeding or organ injury without appropriate endoscopic treatment, large resection not removable by small incision, high risk of complications when proceeding by endoscopic method, e.g. due to anatomical predispositions

LAPAROSCOPY

A minimally invasive surgical technique allowing view and intervention in the abdominal cavity.

Surgical procedure: Once the patient is anesthetized, the operation begins with the establishment of a capnoperitoneum (to improve visibility and create a manipulation space) - a trocar is placed into the abdominal cavity from the supra or infraumbilical incision either open way (Hasson) under visual control and CO₂ is insufflated (especially suitable for patients after previous operations, in patients where adhesions are anticipated or in obese patients) or CO₂ insufflation with a Veress needle (puncture of the abdominal cavity with a safety tip needle) followed by trocar insertion for optics (higher risk of organ/vascular injury). Next, after visually inspecting the abdominal cavity with a laparoscope (most often with a 30° optic), we introduce working ports under visual control (in locations according to the expected procedure - see figure below). After the surgical procedure (often involving drainage through one of the trocar wounds), the ports are extracted under visual control and the abdominal wall is sutured in layers to prevent the formation of a incisional hernia.

Most common indications: appendectomy, cholecystectomy, intestinal resection, hernia repairs (e.g. inguinal, hiatal hernia), nephrectomy, hysterectomy

Contraindications: hemodynamic instability, intestinal obstruction with significant dilatation, severe comorbidities (coagulopathy, cardiopulmonary failure), septic shock, lymphomas

Complications: poor tolerance of capnoperitoneum (increased intra-abdominal pressure adversely affects cardiac output, respiration, and physiology affected by increased CO₂ tension), subcutaneous emphysema, organ (bladder, bowel) or vascular (inferior epigastric artery) injury, wound infection, deep vein thrombosis (DVT), scar hernia

THORACOSCOPY

A minimally invasive surgical technique allowing view and intervention in the pleural cavity.

Pre-operative preparation: pre-resection spirometry (to verify potential extent of resection), pre-operative imaging (to determine exact extent of disease), laboratory tests (not to forget coagulation)

Anesthesia: biluminal intubation or cannula intubation with obturator (before lung procedures requiring a collapsed lung)

Surgical procedure: after induction of anesthesia, the patient is most often positioned on his/her side so that the intercostal spaces are distracted (on the other side the chest is supported by a bridge or positioned in lateroflexion by the operating table). When draping, the need for conversion should be considered. Ports are introduced at the upper edge of the ribs (to prevent injury to the intercostal neurovascular bundle) according to the rule of triangulation (first port by blunt preparation, the others under optical control to form a triangle). In the case of thoracoscopy, CO₂ insufflation is not performed to increase the manipulation space (the chest does not collapse), but sometimes to promote the collapse of the non-ventilated lung. At the end of the operation, the port wounds can be used to insert the chest drainage.

Most common indications: lung resection (non-anatomical - wedge resection, anatomical - segmentectomy, lobectomy, pneumonectomy), bullectomy, pleurodesis, esophageal surgery, empyema treatment, sympathectomy, thymectomy

Contraindications: firm adhesions in the pleural cavity, hemodynamic instability, inability to perform or poor tolerance of selective ventilation (e.g. intraluminal tumor obstruction)

Complications: pneumothorax, atelectasis, pneumonia, organ and vascular injury, cardiac rhythm alterations

An extended variant of thoracoscopy is VATS (video assisted thoracic surgery). This technique uses a combination of the sufficient number of ports and a single working incision to allow a wider range of surgical instruments to be used and to extract the resectate.

Thoracoscopy in a broader sense includes mediastinoscopy. It can be performed by the jugular, parasternal or subxiphoid route.

ARTHROSCOPY

A minimally invasive surgical technique allowing intervention and a view inside the joint.

Arthroscopy can be performed under either regional or general anesthesia, depending on the expected extent of the procedure, localization and patient tolerance.

The principle is the introduction of instruments and endoscope from small incisions directly into the joint cavity, which is continuously filled with sterile solution during the procedure (providing distension of the joint to increase visibility and create a manipulation space).

Most used in the diagnosis and treatment of knee, shoulder, ankle, elbow, and hip disorders.

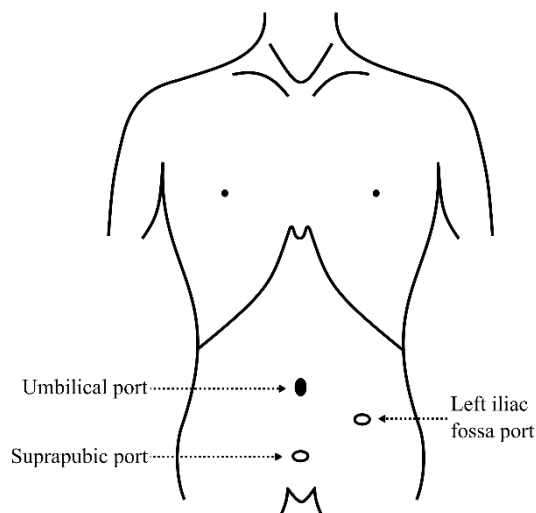
Spectrum of procedures on the knee joint: meniscectomy (often partial), meniscus suture, reconstruction of cruciate ligaments, treatment of damaged articular cartilage, extraction of intra-articular fragments, stabilization of the patella, removal of Baker's cyst.

Contraindications: local infections, advanced arthritic changes and deformities of the joint

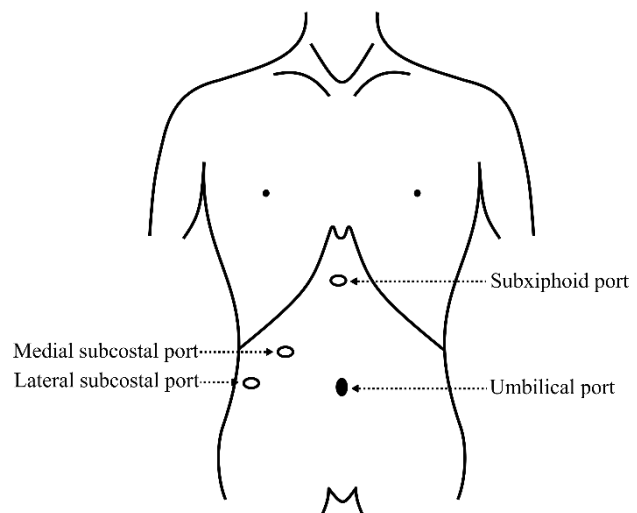
Complications: infection, bleeding, deep vein thrombosis and pulmonary embolism

PORT PLACEMENT AND PATIENT'S POSITION

Laparoscopic appendectomy (LAPPE)
Left side tilt with head down



Laparoscopic cholecystectomy (LCHCE)
Left side tilt with head up



INVASIVE RADIOLOGY AND ENDOSCOPY (8)

Invasive radiology

Andrašina Tomáš, Rohan Tomáš, Zavadil Jan, Matkulčík Peter, Hanžlová Barbora, Bárta Radek

Interventional radiology aims to perform diagnostic and therapeutic procedures under image guidance, using minimally invasive techniques associated with lower complication rates, reduced patient burden, and shorter recovery times than open surgery. Interventional radiology procedures can be classified based on the imaging modality used for guidance (DSA, fluoroscopy, CT, MRI, ultrasound-guided procedures), into vascular and non-vascular methods, or by organ system.

Fundamental vascular interventional radiology procedures include *recanalisation*, which is aimed at restoring blood flow and improving perfusion, and *embolisation*, which is aimed at occluding a vessel at the injury site.

In cases of **peripheral arterial disease**, recanalisation is the first-line treatment for patients with stenoses and short occlusions in the lower limb arteries who are symptomatic (typically claudication under 200 metres or lifestyle-limiting, rest pain, or tissue loss). Even in the case of long occlusions of the superficial femoral artery exceeding 20 cm, endovascular treatment can be attempted, with surgical bypass as the final solution in case of failure. Radiologically, occlusions or stenoses of the external and common iliac arteries can be addressed using retrograde puncture techniques. In contrast, lesions in the superficial femoral artery and distally can be treated with antegrade puncture techniques (with and against the direction of blood flow). Advanced interventions combining open surgical and endovascular procedures in a single setting are called *hybrid procedures*. Patients requiring surgical access for percutaneous puncture (e.g., common femoral artery) followed by endovascular treatment of the iliac or peripheral lower limb arteries benefit the most from this approach.

Neurointerventional procedures represent a delicate subset of vascular interventional radiology. A hyperacute procedure for ischemic stroke patients with large vessel occlusion (internal carotid artery, middle cerebral artery up to M2 segment, vertebral, and basilar arteries) is recanalised within the therapeutic window (6-48 hours). The thrombus is extracted using aspiration, a specialised retriever device, or a combination. Endovascular treatment of aneurysms (either ruptured to prevent rebleeding or incidentally detected with a high risk of rupture) can be performed in acute or elective settings. Currently, the vast majority of aneurysms can be treated endovascularly. Depending on the aneurysm's shape coils alone, coils with stents, or in patients with unfavourable anatomy, flow diverters or stents can reduce pressure within the aneurysm and its risk of rupture. Arteriovenous malformations (AVMs) can also be treated endovascularly, with their nidus typically filled with embolic glue (e.g., Onyx). Endovascular AVM treatment may be followed by neurosurgical resection of the now nearly avascular

lesion. Dural arteriovenous fistulas, carotid-cavernous fistulas, and, rarely, stenosis within dural sinuses can also be addressed endovascularly.

Non-vascular interventions predominate in procedures involving the **gastrointestinal and genitourinary tracts**. In acute settings, these include *percutaneous drainage* of intra-abdominal collections under CT or ultrasound guidance. While sample aspiration is technically feasible from almost any intra-abdominal space, drainage is typically reserved for collections with inflammatory content exceeding 3 cm in size. Using either a direct puncture technique with a *trocár* or, for collections located near critical structures or requiring an indirect approach, the *Seldinger technique*, a plastic drain ranging from 6-24F in size can be placed. A similar approach can be used to drain confined thoracic collections. Percutaneous drainage methods are also employed for biliary drainage - *percutaneous transhepatic cholangiography* (PTC) - in patients unsuitable for surgical anastomosis, those in whom endoscopic drainage is ineffective, or those eligible for palliative treatment (e.g., brachytherapy).

Embolisation refers to a group of procedures aimed at occluding an artery. In acute settings, this primarily involves controlling active bleeding (e.g., from visceral or renal arteries) in the context of trauma or following interventions. Various types of coils, adhesives, or particles can be utilised depending on the nature of the arterial injury. Percutaneous biopsies are noteworthy among non-acute interventions, allowing for precise and targeted tissue sampling for diagnostic purposes. Key indications include the diagnosis of focal liver lesions. *Percutaneous biopsies* are also standard for other parenchymal organs outside the gastrointestinal tract, including the kidneys, lungs, prostate, and breasts. Increasingly, biopsies are performed for advanced grading or tumour mutation assessment to guide targeted oncological therapy. For patients unsuitable for surgical resection (due to limited functional reserve, comorbidities, or refusal of surgery), *thermal and non-thermal ablation* of parenchymal organs offer an alternative. Primary indications include the treatment of small solitary or multifocal tumours up to 3 cm in size. The liver is the most common organ treated with this short procedure, performed without the need for general anaesthesia, followed by the kidneys and lungs (excluding the gastrointestinal tract).

Among the available techniques, *microwave ablation*, which utilises electromagnetic energy with higher temperatures and greater efficiency than *radiofrequency ablation*, is most frequently employed. In the case of primary renal tumours, *cryoablation* (temperatures as low as -140°C), with its excellent visualisation of the ablation zone as an "ice ball," is often preferred due to its less invasive nature. Outcomes of minimally invasive treatment for tumours up to 2 cm in size are now comparable to those with surgical resection. Synergistic effects in multimodal treatment, particularly for hypervascularised tumours (HCC, ICC), are achieved with *transarterial chemoembolisation* (TACE). TACE combines a

high local concentration of chemotherapeutic agents with the embolic effect of particles delivered through the hepatic artery branches. In contrast, *portal vein embolisation* aims to induce atrophy of the affected liver segment and hypertrophy of the future remnant liver parenchyma before planned liver resection. The main indication is extended hemihepatectomy, where, without increasing the volume of the remnant liver lobe, there is a risk of liver failure (25-40%).

Venous interventions can be categorised as preventive (implantation of vena cava filters) or therapeutic (venous thrombolysis), in which a thrombolytic catheter is placed directly into the thrombus to dissolve it through the local administration of fibrinolytic agents with limited systemic effects. Modern mechanical catheters that actively fragment and aspirate the thrombus are also available. The goal of implanting a *vena cava filter* in the inferior vena cava is to reduce the incidence of fatal pulmonary embolism. The indication for this procedure is primarily in patients with thromboembolic disease for whom conventional anticoagulation therapy is contraindicated, complications of anticoagulation arise requiring treatment interruption, anticoagulant therapy fails, or if there is progression of deep vein thrombosis (DVT) during therapy.

Digestive endoscopy

Kroupa Radek, Dastych Milan, Skutil Tomáš

The development of advanced methods of digestive endoscopy allows the treatment of many conditions that were previously indicated for surgery. In most cases, endoscopic treatment is associated with a lower risk of complications and comparable efficacy to surgery.

Based on the indication, therapeutic endoscopic interventions can be divided into

- Endoscopic removal of precancerous mucosal lesions and early T1 cancer.
- Treatment of stenoses and obstructions of the alimentary tract
- Endoscopic treatment of biliary and pancreatic diseases
- Drainage of fluid collections
- Endoscopic treatment of gastrointestinal bleeding
- Others (drainage of insufficient anastomosis, foreign body removal, etc.).

The basic technique that allows the **removal of early neoplasia** (most commonly polyps in the large intestine) is *endoscopic polypectomy* and *endoscopic mucosal resection*, where the pathological tissue is entrapped into a snare and mechanically and thermally removed. During the mucosal resection, a solution is injected into the submucosa, leading to the elevation of the mucosa and the possibility of its removal. *Endoscopic submucosal dissection* cuts through the submucosa just above the muscle.

Dysplasia and early cancer of the oesophagus and stomach can be curatively treated endoscopically. A special method is *radiofrequency ablation* for dysplastic Barrett's oesophagus. Benign oesophageal **strictures** are indicated for endoscopic dilation using balloon or bougie dilators; a similar technique is used to dilate stenotic anastomosis or short strictures in Crohn's disease. Malignant oesophageal strictures can be treated in a palliative setting *with a self-expandable metallic stent* (SEMS).

Endoscopic Retrograde Cholangiopancreatography (ERCP) enables an intervention on the biliary tree or pancreatic duct with fluoroscopic control. CT and MR, including MR cholangiopancreatography and endoscopic ultrasonography, are used for diagnostic imaging of the biliary tract and pancreas. The main indications for ERCP are treating choledocholithiasis and managing benign and malignant biliary stenosis, including biopsy. After cannulation and visualisation of the duct, an *endoscopic papilla sphincterotomy (EPST)* and extraction of the lithiasis with different instruments such as baskets or balloons are usually performed. Benign stenoses are usually bridged with a plastic stent, also known as a *duodenal biliary drain* and metallic stents are used in the palliative treatment of malignant stenosis. If drainage by ERCP fails, an alternative approach is to perform percutaneous transhepatic drainage or endoscopic ultrasonography-guided drainage. In *Cholangio/pancreaticoscopy*, another thin endoscope is used to pass through the working channel to perform targeted biopsy or *electrohydraulic lithotripsy* of large stones. Interventions on the pancreatic duct are less frequent. They mainly deal with complications of chronic (less acute) pancreatitis (symptomatic stenosis or drainage of the disrupted duct) by inserting a pancreatic drain. ERCP carries a 10% risk of severe complications (acute pancreatitis, bleeding, infection, perforation) and thus should be indicated judiciously.

Endoscopic ultrasonography (EUS) combines endoscopy with an ultrasound probe, allowing imaging of organs adjacent to the digestive tube and intramural formations. The main indication is the examination of the pancreas with the possibility of a targeted *biopsy*.

EUS-guided drainage should be considered preferentially in infected *pancreatic collections* in acute pancreatitis. It is advisable to wait at least 4 weeks from the onset of acute pancreatitis to perform the procedure safely, as it allows the collections to become circumscribed and to form the so-called WON (walled-off necrosis). Plastic double pigtail stents or lumen-apposing metal stents (LAMS) are used for transgastric or transduodenal drainage. These stents have a "barbell" shape with both ends widened and pull the walls of the organs together, enabling safe passage into the cavity to allow endoscopic debridement and necrectomy. EUS-guided drainage of the gallbladder enables overcoming acute conditions in patients at high risk for surgery.

Endoscopy is an essential method for **the diagnosis and treatment of gastrointestinal bleeding**. A combination of epinephrine injection followed by inserting a haemostatic clip or thermal coagulation

probe is recommended to treat *non-variceal bleeding*. Endoscopic ligation is the method of choice for active *variceal bleeding* and its prevention. Its principle is to suck the varix into a special attachment at the end of the endoscope and strangulate it with a rubber band resulting in subsequent thrombosis, necrosis and scarring of the wall.

Digestive endoscopy includes an increasing number of **new procedures** to address diseases that were previously treated surgically. Endoscopic treatment of achalasia includes dilation of the cardia with special large-size balloons or endoscopically performed myotomy through a long submucosal tunnel in the oesophagus called POEM (peroral endoscopic myotomy). Endoscopic bariatric procedures complement treatment options for severe obesity.

Methods of digestive endoscopy, preparation, most common assessment

Method	Brief description	Preparation	Interventions
Gastroscopy	Examination of the upper part of the digestive tract, including the oesophagus, stomach and proximal part of the duodenum.	At least 4 hours of fasting	Haemostasis Foreign body extraction Percutaneous endoscopic gastrostomy Dilation
Colonoscopy	Examination of the lower part of the digestive tract including the entire large intestine and usually the terminal ileum.	Orthograde preparation of intestine with a special solution. Split regiment preferred.	Polypectomy Dilation Haemostasis
Enteroscopy (push, balloon)	Examination of the proximal part of the small intestine	At least 4 hours of fasting	Haemostasis Biopsy, Polypectomy Dilation
Video capsule enteroscopy	Examination of the entire small intestine by swallowed imaging capsule	Fasting, reduced amount of laxative solution	Imaging only, no possibility of any intervention
ERCP	A combined examination using endoscopically applied contrast agent and radiographic imaging of the biliary tree and/or pancreatic duct. Almost always supplemented by follow-up intervention	At least 4 hours of fasting NSAID suppository 30 minutes before the procedure to prevent pancreatitis	Extraction of lithiasis Biopsy Drainage, stenting
EUS	A combined examination using ultrasound probe, enabling accurate imaging of the organs adjacent to the alimentary tract	At least 4 hours of fasting	Biopsy EUS drainage

BASICS OF SURGICAL ONCOLOGY, ROLE OF ADJUVANT AND NEO-ADJUVANT THERAPY (17)

Žaloudík Jan

Surgical oncology, also known as oncosurgery, concerns the surgical management of tumours. This field extends beyond traditional surgical domains—such as abdominal, thoracic, breast, and skin malignancies—to include other surgical specialities, including urology, gynaecology, orthopaedics, neurosurgery, head and neck surgery, and paediatric surgery.

The multimodal management of solid malignant tumours is based **on four primary modalities: diagnosis, surgery, radiotherapy, and systemic treatment**. Effective treatment involves coordinating and integrating these approaches for each case, ideally facilitated by multidisciplinary teams (MDTs). **Early diagnosis of solid malignant tumours** significantly improves the efficacy of treatment. Early detection and targeted secondary prevention play a crucial role in surgical oncology, emphasising that reliance on emerging anticancer drugs alone is insufficient.

The significance of surgical treatment is emphasised by the fact that over 80,000 solid malignant tumours are diagnosed annually. Depending on the clinical stage of the tumour, surgery is the primary treatment modality in 60-70% of cases, either as a **surgery-alone approach** or in combination with other treatment modalities. For the remaining third of advanced tumours, **palliative and other surgical interventions** are often necessary. The role of surgical treatment is expected to grow with improved early-stage detection through screening and prevention programmes, as the treatment of early-stage tumours is predominantly managed through open or minimally invasive surgical techniques. Comprehensive data on cancer incidence, mortality, and stages in the Czech Republic are available in the System for Visualisation of Oncological Data (www.svod.cz).

In general, solid tumours are restricted to their organ of origin (clinical stages I and II) and are **primarily managed through surgery**. Tumours with evidence of regional spread (clinical stages II and III) often require **a combined approach** involving surgery, radiotherapy, and systemic treatment. For metastatic tumours (clinical stage IV), systemic treatments such as chemotherapy or targeted therapy are predominant, while surgery may be employed with **a palliative aim** to relieve symptoms, achieve cytoreduction, and address complications associated with tumour progression.

Radical oncosurgery aims to achieve three main objectives: **complete tumour resection, enhancement of diagnostic accuracy** through tissue sampling and intraoperative staging, and **reconstruction of defects** resulting from the surgery, both anatomical and functional. For instance, in colorectal surgery, bowel resection is typically straightforward. However, cosmetic or functional reconstructions are less commonly addressed during the initial surgical procedure, often necessitating subsequent interventions by plastic surgeons. Surgical oncology also includes practices such as

oncoplastic surgery—where plastic surgeons collaborate directly with surgical oncologist—and **pelvic surgery**, which involves coordinated efforts between surgeons, urologists, and gynaecologists for advanced pelvic tumours, with approximately 15,000 such cases annually. Optimal patient care requires collaboration among various specialists, emphasising the importance of organised and efficient care delivery.

Histopathologically, epithelial tumours are prevalent, with **a primary propensity to metastasise to regional lymph nodes**. Consequently, oncosurgery focuses on regional lymphatics and nodal metastases. **Lymphadenectomy** is a critical component of radical operations for solid epithelial tumours, with **sentinel lymph node biopsy (SLNB)** used to identify and assess potentially affected lymph nodes. **Regional lymphadenectomy** includes one or more types of dissections, such as axillary, cervical, ilioinguinal, or retroperitoneal. Additionally, dissection is always part of surgical resection of the gastrointestinal tract as it follows radical resections of the intestine, stomach, pancreas, oesophagus, or lungs.

In cases of solitary or limited **organ metastases** (clinical stage IV), particularly in the liver or lungs, anatomical resection may be considered in conjunction with systemic drug treatments. The biological behaviour of metastases, influenced by factors beyond mere mechanical circulation, necessitates a nuanced understanding of **the biology of metastatic processes** (e.g., the "seed and soil" hypothesis; Paget 1889, Fiedler 2008). At the same time, it should be noted that in many other aspects, oncosurgical reasoning must be not only anatomical but also biological. **Anatomical radicality alone** does not guarantee long-term cancer control, as factors such as immunosuppression and perioperative care also play significant roles.

Adjuvant (post-operative) **and neoadjuvant** (preoperative) **therapies** are critical components of cancer management. A small tumour may contain billions of cells, with each cell potentially capable of metastasising. Consequently, even radical surgeries for clinically localised tumours (stages I-III) are associated with a risk of subsequent **metachronous metastases**. **Adjuvant chemotherapy** targets circulating tumour cells and subclinical micrometastases, while post-operative **adjuvant radiotherapy** reduces the risk of local recurrence. **Neoadjuvant therapies**, including **chemotherapy or chemoradiotherapy**, aim to achieve tumour regression, eliminate aggressive cell clones, and guide the selection of post-operative treatments.

Despite potential risks, such as tumour progression during ineffective **neoadjuvant therapy**, adherence to monitoring protocols for pre-and post-operative treatments enhances overall outcomes. These outcomes are measured by overall survival (OS), five- or ten-year survival rates, and progression-free survival (PFS). The efficacy of treatment strategies, including their impact on adverse effects and patient satisfaction, requires continuous evaluation.

Advanced techniques in surgical oncology, including hyperthermic intraperitoneal chemotherapy (HIPEC), intra-arterial or extracorporeal chemotherapy for extremity sarcomas and melanomas, and post-resection interstitial brachytherapy, are employed for more complex cases.

Technological advancements in **endoscopic, laparoscopic, and robotic surgery**, along with improved **3D imaging and planning**, contribute to better surgical outcomes. Ultimately, the **future success of oncosurgery** hinges on early tumour detection and effective secondary and tertiary prevention, rather than solely on technological and surgical advancements.

PAIN MANAGEMENT OF SURGICAL PATIENT (20)

Koloděj Daniel, Štourač Petr

Pain management primarily involves pharmacotherapy (as well as physical therapy and others) for surgical patients – patients undergoing perioperative trauma.

The causes of pain in the perioperative period may include:

- a) condition associated with illness (e.g., femoral neck fracture),
- b) condition associated with surgery – surgical trauma,
- c) condition not caused by primary illness or surgical trauma (e.g., back pain from hospital bed).

The pathogenesis of pain is straightforward in practice – tissue injury leads to the activation and sensitisation of peripheral nociceptors, mediator release, and lactate production. The clinical presentation of the patient may vary! Subjective perception of pain is individual; patients often visit clinics specifically for pain, as it is the most common symptom and the hardest to assess objectively.

For pain intensity assessment, we may use:

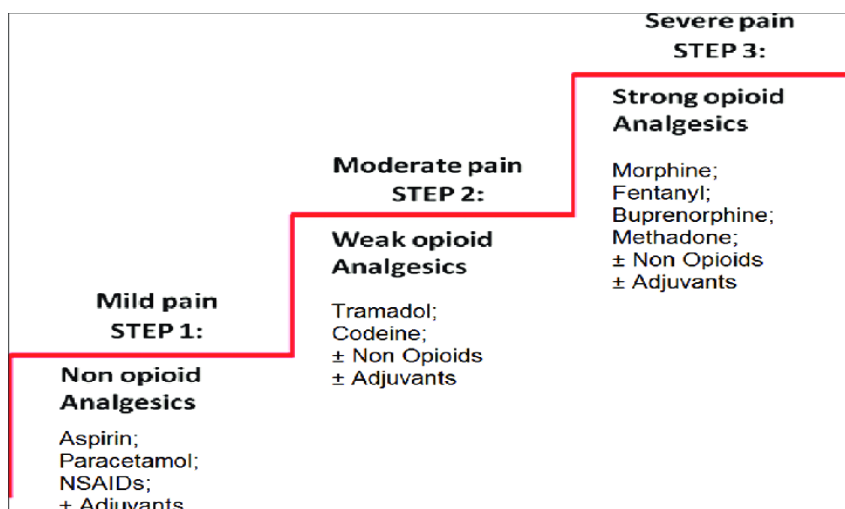
- a) VAS (Visual Analogue Scale)** – a score ranging from no pain to the worst possible pain, assessed on a numerical scale (commonly 1-10 or 1-5 depending on the clinic, etc.),
- b) VRS (Verbal Response Score)** – **pain assessed by number** (e.g., 4 out of 10) or by word (e.g., moderate, severe, unbearable), objective changes in autonomic functions – tachycardia, hypertension, sweating, inability to perform a dynamic command due to pain – inability to cough, move, or take a deep breath, etc.

Why do we do this? Pain pharmacotherapy has several benefits – wound healing, increased patient mobility, patient satisfaction, earlier discharge, and reduced risk of immobilisation syndrome and thromboembolic disease.

Complications of pharmacotherapy:

- a) inadequate pharmacotherapy, see above
- b) side effects of pharmacotherapy – PUD (peptic ulcer disease) with *NSAIDs* (non-steroidal anti-inflammatory drugs), appropriate prophylactic use of Proton Pump inhibitors (PPIs), allergic reactions, *opioids* – constipation, nausea, altered consciousness, **CAVE** in obese patients, apnoea, relative contraindications – *metamizole* in thrombocytopenia, *paracetamol* in hepatopathy, reduced dosage of ALL analgesics in patients with nephropathy. ***Do not administer metamizole to asthmatics! It may cause anaphylactic reactions.***

Pain pharmacotherapy – the most important section of this chapter! Muster recommends the WHO Analgesic ladder:



Before surgery, we may determine the anticipated intensity of postoperative pain. (For further information, see the Recommended Procedure – Management of Acute Postoperative Pain from ČSARIM)

1. Procedures with anticipated low-intensity postoperative pain (e.g., arthroscopy, endoscopy, minor skin procedures) – use Class I and II analgesics; if analgesia is insufficient, then Class III analgesics. Early transition to oral form upon patient tolerance is recommended.
2. Procedures with anticipated moderate-intensity postoperative pain (e.g., laparoscopy, hernioplasty, breast surgery) – primarily use Class II analgesics; weak opioids can be replaced with strong ones if necessary.
3. Procedures with anticipated high-intensity postoperative pain (e.g., thoracotomy, total endoprosthesis, diaphragmatic surgery) – primarily use regional anaesthesia via catheter; if analgesia is insufficient, administer parenteral analgesics.

Basic classes of analgesics and their dosing:

Class I: a) analgesics – antipyretics (AA): paracetamol is the cornerstone of pain treatment, administered orally and intravenously, with a maximum daily dose of 4 grams (10-20 mg/kg, maximum 40 mg/kg) administered every six hours. It is the most advantageous analgesic; contraindications include hepatopathy, and the dose is reduced in nephropathy. It is not administered in cases of known allergy. **Metamizole** is another representative of this category, administered orally and intravenously; there is a reported risk of agranulocytosis and relative contraindications in patients with thrombocytopenia. Combined preparations of metamizole and antispasmodics are advantageous

for patients with spasmodic pain. **b) NSAIDs – ibuprofen, diclofenac, indomethacin, aceclofenac, nimesulide** – administered orally, intravenously, intramuscularly, rectally, used in combination with AA or alone; advantageous for musculoskeletal injuries, they have anti-inflammatory properties, beneficial in oedema treatment (excluding cerebral oedema), effusion, etc. Dosage is individualised based on the medicinal product.

Class I analgesic combinations: AA + AA – advantageous, AA + NSAID – advantageous, AA + AA + NSAID – maximum possible Class I analgesic combination, NSAID + NSAID – never!

Class II: weak opioids – tramadol is the main representative, administered orally, intravenously, intramuscularly, and used in combination preparations, particularly with paracetamol. It almost always causes constipation and may induce nausea and altered consciousness. The co-analgesic effect of paracetamol + tramadol is very beneficial. Another representative is dihydrocodeine – administered orally. Class II is always combined with Class I analgesics.

Class III: strong opioids – morphine, buprenorphine, fentanyl, oxycodone, piritramide – administered orally, intravenously, intramuscularly, subcutaneously, rectally, transdermally, with dosage tailored to the patient's response. In the perioperative period, they are used in planned procedures with expected moderate and severe pain, as well as for severe breakthrough pain unresponsive to maximum doses of Class I and II analgesics. CAVE in patients with acute pancreatitis – the risk of Oddi's sphincter closure; it is better to consult the ICU and consider an epidural catheter.

Non-WHO Analgesic Ladder Analgesic Options:

I. Physical therapy – a foundation of trauma care. This includes immobilisation – plaster splints, orthoses, elastic bandages, offloading the injured limb – crutches, slings, and limb elevation. Cryotherapy – cold therapy is also included. Always apply through a dry cloth, such as a towel, to avoid frostbite.

II. Gabapentin – used in the perioperative period, particularly for patients with neuropathic pain following lower limb amputations, administered orally with a necessary loading dose. Risk of quantitative consciousness disorders.

III. Regional anaesthesia/analgesia – depending on the nature of the condition and pain, often requiring collaboration with the ICU. **Mucosal/infiltration/nerve block anaesthesia/analgesia** – administered by the surgeon (in the case of a nerve block, only in indicated cases; otherwise, it is the ICU's domain), with agents such as **mesocaine, bupivacaine** – used mainly in outpatient treatment (e.g., infiltrative administration of mesocaine at the fracture site before reduction) or intraoperatively in the subcutaneous tissue topically at the incision site – postoperatively reduces acute postoperative pain. **Central/peripheral nerve block anaesthesia/analgesia** – usually the ICU's domain, can be

administered as a single dose or via catheter for repeated administration. Peripheral blocks can be secured almost anywhere. A significant advantage is the reduction in doses of other analgesics.

Author's postscript for practice on a standard surgical ward (not required for examination):

I. Start with paracetamol, unless contraindicated. Saturate with the maximum dose, starting with the intravenous form every six hours. If insufficient, add metamizole every six hours if not contraindicated. Can you add NSAIDs? Go for it! But remember to add PPIs to regular medication. Monitor the patient; still, suffering? Is the nurse complaining about the additional orders? Listen to the patient; that's why you're in the right place.

II. If the maximum combination of AA + AA isn't enough, try adding tramadol; there are beneficial paracetamol + tramadol combination products available on the market, such as paracetamol + tramadol 325/37.5 mg every six hours, two tablets. Continue with metamizole and possibly NSAIDs. Always weigh the risk/benefit for your patient.

III. Use strong opioids cautiously for breakthrough pain in the perioperative period! A single administration may be beneficial, but repeated administration should lead you to reflect on whether you've done everything right. If so, consider consulting the ICU and arranging nerve block analgesia for the patient.

ASSESSMENT AND MANAGEMENT OF AN UNCONSCIOUS PATIENT (19)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Definition of Consciousness
- Types of disorders of consciousness
 - Quantitative
 - Qualitative
- Assessment of level of consciousness
- Differential diagnosis of disorders of consciousness (examples)
 - Intracranial causes
 - Extracranial causes
- Initial approach to the unconscious patient - ABCDE approach according to the European Resuscitation Council (ERC)
 - A (airway) - assessment of airway patency
 - B (breathing) - clinical assessment of breathing and ventilation
 - C (circulation)
 - D (disability - neurological status)
 - E (exposure - clinical examination)
- Therapy (cranial trauma, therapy for intracranial hypertension, status epilepticus)

HOMEOSTASIS, ACID-BASE BALANCE DISORDERS (4)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Homeostasis, acid-base balance
 - Definition of both terms
 - Definition of pH, acid, base
 - Range of physiological pH, pO₂, pCO₂
 - Definition of acidosis, alkalosis, acidemia, alkalemia
 - Buffer systems - especially bicarbonate, protein, phosphate
- Acid-base balance disorders
 - Definition of ABG disorders - metabolic/respiratory, alkalosis/acidosis, combined disorders
 - Buffer base, base excess - definition and relevance to ABG disorders
 - Compensatory mechanisms – the role of lungs, kidneys, liver
 - Anion Gap (AG), corrected anion gap - definition, practical significance
- Metabolic acidosis
 - Division - high AG (HAGMA) / normal AG – hyperchloremic
 - The direction of variation in pH, pCO₂, HCO₃⁻, BE
 - Compensatory mechanisms
 - Most common causes, examples
 - Therapy
- Metabolic alkalosis
 - Loss of anions/ hypernatremic/ from excess of other cations
 - Direction of variation in pH, pCO₂, HCO₃⁻, BE
 - Compensatory mechanisms
 - Most common causes, examples
 - Therapy
- Respiratory acidosis
 - Classification - acute/chronic
 - Direction of variation in pH, pCO₂, HCO₃⁻
 - Compensatory mechanisms
 - Most common causes, examples
 - Therapy

- Respiratory alkalosis
 - Direction of variation in pH, pCO₂, HCO₃⁻
 - Compensatory mechanisms
 - Most common causes, examples
 - Therapy
- Water regulation
 - Total body water distribution (intracellular fluid, extracellular fluid)
 - Definition of euvolemia, hypovolemia, hypervolemia
- Electrolytes and electrolyte disorders
 - The most important electrolytes for maintaining homeostasis - Na, K
 - Physiological values, functions
 - Na disorders - hypernatremia, hyponatremia
 - Causes, clinical presentation, therapy
 - K disorders - hyperkalemia, hypokalemia
 - Causes, clinical presentation, therapy

CARDIOPULMONARY RESUSCITATION (9)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Definition of respiratory and circulatory arrest
- Chain of Survival
- Causes of sudden circulatory arrest (primary/secondary causes)
- Approach to the critically ill patient (SSS ABC algorithm)
- Adult basic and advanced cardiopulmonary resuscitation (BLS – Basic Life Support, ALS – Advanced Life Support), according to the European Resuscitation Council (ERC)
- Paediatric basic and advanced cardiopulmonary resuscitation (BLS – Basic Life Support, PALS - Paediatric Advanced Life Support)
- Cardiopulmonary resuscitation technique (CPR; correct ratio of ventilation and chest compressions, location, frequency and depth of chest compressions, ventilation, securing the airway)
- Defibrillated rhythm – ERC recommended practice
- Non-defibrillated rhythm – ERC recommended practice
- Safe defibrillation technique, automatic external defibrillator
- Pharmaceuticals used in cardiopulmonary resuscitation
- Securing intravenous access
- Reversible causes of circulatory arrest
- Initiation of CPR, termination of CPR
- Restoration of cardiac function, post-resuscitation care

SHOCK (10)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Definition of shock
- Pathophysiology of shock
 - Initial phase of compensation
 - Decompensation phase
 - Irreversible phase
- Classification (including pathophysiology, examples)
 - Hypovolemic shock
 - Cardiogenic shock
 - Distributional shock
 - Obstructive shock
- Clinical symptoms and signs of shock
- Initial approach and treatment of the patient in shock – ABCDE approach according to the European Resuscitation Council (ERC)
 - A (airway) - assessment of airway patency
 - Methods of airway clearance in patients without risk of cervical spine injury/with risk of cervical spine injury
 - B (breathing) - clinical assessment of breathing and ventilation
 - C (circulation)
 - Macrohemodynamic assessment
 - Microhemodynamic assessment
 - Securing the intravenous access
 - D (disability – neurological condition)
 - E (exposure – clinical examination)
- Differential diagnosis
- Therapy (pharmacological/non-pharmacological)

GENERAL ANAESTHESIA (1)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Definition of general anaesthesia (GA)
- Components of GA
- Indications for GA
- Contraindications to GA
- Patient preparation
- Complications of GA (during general anaesthesia, postoperative complications, examples)
- Phases of GA - induction, maintenance phase, emergence
- Stages of GA
- Types of GA (inhalational, intravenous, supplemented)
- Inhalational GA
 - Inhalational anaesthetics - gaseous/liquid, examples
 - Indications, examples of use, complications
- Intravenous GA
 - Intravenous anaesthetics - examples (Propofol, Thiopental, Etomidate, Ketamine, Benzodiazepines)
 - Indications, examples of use (induction to anaesthesia, total intravenous anaesthesia), complications
- Combined GA
 - Indications, examples of use
- Other drugs used in CA
 - Analgesics (opioids/non-opioid analgesics)
 - Muscle relaxants

LOCAL ANAESTHESIA (2)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Definition of local anaesthesia
- Indications
- Contraindications
- Methods of local anaesthesia and a brief description
 - Topical anaesthesia
 - Infiltration anaesthesia
 - Peripheral nerve blocks
 - Neuraxial blockades
- Method of application (single, repeated - catheter techniques)
- Patient preparation
- Local anaesthetic agents
- Peripheral nerve blocks - definition, examples, indications, contraindications, complications
- Neuraxial blockades
 - Epidural anaesthesia
 - Subarachnoid anaesthesia
 - Explanation of methods
 - Anatomy of the spinal canal, including identification of the site of local anaesthetic application
 - Indications, contraindications, complications (e.g. post-puncture cephalgia)

COMPLICATIONS OF ANAESTHESIA (16)

Kořínková Bianka, Šoltysová Jana, Klabusayová Eva, Štourač Petr

- Types of complications
 - According to the occurrence: during induction of anaesthesia, during anaesthesia, during emergence from anaesthesia, postoperative complications
 - Early, late
- Unexpected perioperative bleeding
 - Definition, classification
 - Risk factors
 - Pathophysiological mechanisms in the development of bleeding
 - Diagnostic and therapeutic approach
 - Early recognition of blood loss
 - Hemorrhage arrest
 - Maintaining oxygen supply
 - Stabilizing circulation
 - Correction of anaemia and coagulopathy
 - Laboratory testing
 - Follow-up care
- Anaphylaxis
 - Definitions
 - Pathophysiology
 - Aetiology
 - Clinical picture
 - Therapeutic management, according to the European Resuscitation Council (ERC)
 - Follow-up care, monitoring
- Malignant hyperthermia
 - Definition and aetiology
 - Risk factors and triggers
 - Clinical presentation of the malignant hyperthermia (MH) crisis
 - Differential diagnosis, eradication
- Acute postoperative pain
 - Definition of pain
 - Division – acute/chronic, visceral/somatic

- Basic principles of perioperative analgesia
- Pain assessment
- Therapy (WHO analgesic ladder)
- Acute pain service
- Principles of multimodal analgesia

PRE-OPERATIVE PATIENT PREPARATION (14)

Urbanek Libor

Preoperative procedures can be divided into:

- managing the patient for an acute (urgent) procedure
- managing the patient for the planned procedure

When managing a patient for surgery, both general principles, valid for all surgeries without distinction, and also special principles, focused on the peculiarities of operations on certain organs (e.g. digestive tract, lungs), or in patients with certain chronic diseases are applied. Full procedure can only be done for planned procedures, In the case of acute conditions requiring immediate (urgent) surgery, the necessary procedure is carried out. Therefore, the management is limited to the most necessary examination.

The purpose of preparing the patient for surgery is to prevent possible intraoperative and postoperative complications. The goal is to identify pathological conditions and try to eliminate them or at least mitigate them

We divide the preoperative procedure of the patient into general and local. LOcally is prepared field for surgery

Managing the patient for the planned procedure

In terms of time, we divide the preparation into long-term, short-term (24 hours before the procedure) and immediate (2 hours before the operation).

Distribution:

- Long-term preoperative preparation – about 14 days before the planned surgery
- Short-term preoperative preparation – 24 hours before the surgery.
- Immediate pre-operative preparation – 2 hours before the surgery
-

Long-term preoperative procedure

It contains internal as well as other medical examinations, which should not be later than 2 weeks before surgery is planned

Basic examination: Blood sampling – biochemical (liver tests, ions, urea, creatinine), hematological (blood count.),paramethers of blood clotting (QUICK, APTT...), blood group and Rh factor. Urine examination – urine + sediment. EKG. Chest X-ray

Anesthesiology assesment– ASA classification

Expert examinations reflects individuals comorbidities

- Cardiological.
- Diabetological.
- Neurological.
- Nephrological.
- Nutritional
- Prehabilitation

The preparation of patients with diabetes mellitus is pointed at adjusting blood glucose levels. Patients are usually switched to insulin in the perioperative period. Patients with lung diseases must be operated on in the resting stage of their disease.

Patients with heart disease require a cardiac evaluation before surgery. These patients often use drugs that affect blood clotting. These drugs must always be discontinued in a sufficient time interval before the surgery.

Short-term preoperative management

- Instructing the patient about the planned procedure and the informed consent to the surgery is signed.
- Prevention of TEN – miniheparinization, DK bandages.
- Consultation with an anesthesiologist
- Administration of evening premedication
- Application of maltodextrin solution up to 2 hours before the start of anesthesia (if there is no other contraindication)
- Emptying of the GIT, ATB

In most patients, prevention of thromboembolic disease is carried out - administration of low-molecular-weight heparins and bandaging of the lower limbs. The risk of thromboembolic disease is significantly increased in major orthopedic operations and in cancer patients In surgical procedures with an increased risk of infection, preventive antibiotic prophylaxis is administered, usually shortly before the procedure or at the beginning of anesthesia.

Immediate preoperative preparation:

- Control of general hygiene, removal of make-up and nails.
- Jewellery, and dental prostheses getting off.
- Checking or redoing DK bandages.
- Measurement of physiological functions
- Administration of morning premedication
- Emptying the bladder

The anesthetic risk is expressed by a code, e.g. the code according to the American Society of Anesthesiologists (ASA):

The ASA Physical Status Classification System is a system for assessing the fitness of patients before surgery.

- ASA 1 – patient without complicating disease
- ASA 2 – mild disease without limitation of performance
- ASA 3 – serious disease limiting performance
- ASA 4 – a serious illness that threatens the patient's life in connection with the operation and without this connection
- ASA 5 – a terminal condition with an extremely unfavorable (infaust) prognosis without dependence on surgery

The current ASA classification in Great Britain and the USA sometimes adds an ASA-VI category: a patient who has been declared *lege artis* brain dead and who is an organ donor.

Management the patient for acute (urgent) surgery

It includes only the absolutely necessary procedures that are needed so that the patient can be operated on. In some (urgent) situations – e.g. serious injuries, the patient is immediately transported from the emergency department to the operating room. Urgent surgery increases the risk of complications due to lack of time for examination and sufficient preparation. As standard in acute operations, at least a laboratory examination is performed and access to the venous system is ensured, or a urinary catheter and nasogastric tube are introduced, and an acute internal and anesthetic examination is performed.

POSTOPERATIVE CARE AND COMPLICATIONS (3)

Kysela Petr

Introduction: Surgery is a controlled trauma that activates the hypothalamus–hypophysis–adrenal axis. It results in catabolism, fluid retention, cardiac and respiratory functions, depression and increased insulin resistance. Surgery induces Systemic Inflammatory Response Syndrome (**SIRS**). It may develop into a Multiple Organ Dysfunction Syndrome (**MODS**) when the apparent depression of organ systems (circulatory, respiratory, urinary, gastrointestinal, neural) does not require substitution (artificial ventilation, circulatory support, elimination, nutrition) or Multiple Organ System Failure (**MOSF**). For these reasons, it is imperative that the patient undergo surgery in the best possible condition.

Minimisation of further trauma, substituting substrates and step-by-step loading of organ systems **according to individual tolerance** are crucial measures in postoperative care. The Early Recovery After Surgery (**ERAS**) approach has worked in all these aspects (www.erassociety.org, www.ftsurgery.com). Today, widely accepted guidelines exist in pancreatic, colorectal, pelvic surgery or cystectomy.

Physiologic postoperative course: It follows the model of physiological wound healing. The inflammatory (exudative) phase lasts 3 – 5 days, the following proliferative phase takes three days to weeks, and the last phase - remodelling - may last for months. The inflammatory phase corresponds to catabolism and is ALWAYS unwelcomed. Other phases are anabolic. This is reflected by energy and protein demands. The length of the latter two phases depends on the extent of surgery and the postoperative care. **Undesired prolongation of the catabolic phase arises as a result of a postoperative complication!** It is suspected when a patient is still unwell on the 3rd to 5th day.

Postoperative complications: a) Medical (internal) complications – a **result of the surgical trauma** (metabolic, cardiac, respiratory, neural), b) Anaesthesiologic – a **result of anaesthesia** including adverse effects of medication, c) Surgical – **consequences related to the surgery itself**. The surgical complications can be divided into procedure-specific (pancreatic fistula or diabetes after pancreatic surgery) and non-specific (bleeding, SSI). According to timing, one recognises 1.) very early, up to three hours (surgical – bleeding, linked to general anaesthesia – aspiration, bronchospasm, laryngospasm, apnea), 2.) early, up to 3 – 5 days (comorbidities decompensation, technical issue with anastomotic leakages) and 3.) late (anastomotic leakages, fistulas, delayed bleeding often with fatal consequences)

Complications prediction: **CPET** (CardioPulmonary Exercise Test) and **sarcopenia** are the most sensitive before surgery, whereas positive cumulative fluid balance post-operatively.

Complications prevention: Thorough preoperative preparation, risk evaluation, and long-term compensation of all comorbidities are checked through **internal preoperative assessment** and recommendations. **Anaesthesiologic assessment** should rethink the type of anaesthesia and postoperative analgesia regarding the scheduled surgery and comorbidities. Invasive monitoring and therapy access (NG tube, urinary catheter, CVC, arterial line, epidural analgesia) should be considered. Postoperative nausea and vomiting (PONV) prevention should be addressed as anaesthetics and painkillers cause it most often. **Surgical assessment** must consider the timing, extent, and type of surgery. Patients must be fully informed about the surgery, possible perioperative complications, postoperative course and recovery, and **what is usually expected from them after surgery**.

Postoperative care: **Thromboembolic disease prevention** (TED) comprises LMWH, compressive stockings and early rehabilitation. **Stress ulcer prevention** – impaired blood supply to the gastric mucosa is the most significant risk factor, and the main goals are circulation optimisation and pain fighting. PPIs are usually added. **Postoperative nausea and vomiting (PONV) prevention** relies on effective pain management. Prokinetics can be added in case of bowel paralysis. Antiemetics can be helpful if PONV is medication-induced. Postoperative goal-directed intervention is to keep patients comfortable (adverse effects). Sound sleep at night and the ability to get some relief during the day mean **adequate analgesia**.

Medicaments administration: The oral or rectal route is preferred. It is not possible during the first hours after surgery until full recovery of reflexes and if a patient suffers some bowel malfunction.

Nutrition: A patient in good condition with expected adequate oral food intake within 5 to 7 days does not need any nutritional support. All others do. If a central venous line has already been introduced, one must not hesitate about parenteral nutrition. Total parenteral nutrition (TNP), including an all-in-one nutrient solution, can be used for both central and peripheral venous access. Oral or enteral feeding is restored gradually according to individual tolerance. Even in well-nourished patients, it is necessary to supply glucose from the same day of surgery at least 1.5 – 2.0 g/kg/d (Krebs cycle maintenance, prevention of excessive protein loss of 0.5 to 1.0 kg of muscle mass daily). The stress ratio of nutrients favours protein. We should avoid ω -3 unsaturated fatty acids fortified formulas in critical care. The ideal energy ratio of protein: fat: carbohydrates seems to be 20:30:50. The energy expenditure rises to 150 kJ/kg daily after simple laparoscopic cholecystectomy.

Glycaemic control: It has been a big topic. Guidelines mandate to keep it between 6.0 and 8.0 mmol/l. If the daily glucose intake is not above 4 g/kg/d (1.5 – 2.0 g/kg/d is the minimum), the hyperglycemia must be treated by INSULIN, not by withdrawing glucose (the stress insulin resistance keeps glucose out of cells). People with diabetes are in greater danger due to higher resistance and sugar-free drinks. They must get everything parenterally.

Fluids: The need for fluids can be estimated to be 1.5 – 2.0 ml/kg/hour. It is modified according to wastes (stomas, drains, sweat, diuresis). The volume administered should maintain diuresis of 0.5 ml/kg/hour, consciousness and circulatory stability. If this is not possible despite a positive fluid balance of 1 – 2 L/day, it is better to add some small catecholamines. Balanced crystalloid formulas are advantageous not just because of a more appropriate amount of Na and Cl but also because of Krebs cycle boosters (lactate, acetate, maleate, fumarate).

Ventilation: Ventilation variation can be tolerated with pCO₂ of 4.5 - 6.5 kPa. O₂ saturation of 90 % and pO₂ 9.0 kPa is also acceptable, namely if we can thus avoid artificial ventilation.

Physiotherapy: Spare the patient's energy for isometric exercises (muscle preservation) and respiratory gymnastics (pneumonia prevention). Any extra power can be used for the patient's verticalisation, which **does not affect peristalsis** onset but prevents DVT.

Wound care: We do not redress clean wounds within the first 48 hours.

Postoperative monitoring: The goal is to reveal imminent complications in time. Its **intensity depends on a patient's overall status and the extent of surgery**, including issues during the procedure. The issues alone mandate at least a short observation at the ICU. Means of monitoring include drains introduced during surgery to reveal early complications (bleeding, leakages), central venous catheters – CVT – hydration status, heart failure, arterial lines – continual blood pressure monitoring, blood chemistry, Nasogastric tube – functionality of GIT. The urinary catheter can be used for the intra-abdominal pressure measurement.

A) **Subjective complaints.** Pain and analgesic consumption should keep decreasing over time. Breathing problems may point to the diaphragm elevation caused by inflated bowels, intrathoracic complications, overhydration or heart failure. The patient may lose stamina within the first 3-5 days. Maintaining the physiologic sleep pattern is very important.

B) **Physical examination.** We check the general condition of a patient, capillary filling, and heart rate (biliary peritonitis lacks peritoneal symptoms and tachycardia, looks like n. X irritation – bradycardia, hypotension). We look at wounds, the operating field, and peripheral blood supply. The appearance of stomas (gathering, ischaemia) gives the best information on circulation and hydration.

C) **Circulation.** ECG monitor – pacing, silent ischaemia (patient is on analgesia). The mean blood pressure of 65 mmHg or even less is enough if the diuresis and consciousness are preserved, and blood pressure correction would need excessive catecholamines. Keeping the systolic pressure at 110-130 mmHg is better if a patient suffers from stenotic arteries. We should not correct hypertension below 130 – 150 mmHg. The pressure is taken periodically on the arm or continuously via an arterial catheter (for three days only).

D) Fluid balance and wastes from GIT, stomas, and drains. Character and amount of waste are equally important – blood, enteral, pancreatic juices and bile in drains. High output from NGT means a malfunction of the GIT. Drains should be removed within three days after surgery if there is physiologic output. If not, they increase the risk of surgical site infections. The patient gathers fluid in the first 3- 5 days. People lose body mass in this catabolic phase. The weight gain caused by swelling of more than 1 kg or fluid retention of more than 3 l significantly increases the risk of complications. Good diuresis is the most critical factor.

E) Blood chemistry and count. Urea, kreatinin, essential minerals and glucose (glycaemic profile) should be monitored and reassessed daily until full recovery of enteral nutrition to ensure stable homeostasis. Resistant hypoglycemia is the first sign of imminent fulminant liver failure. Blood count may reveal silent bleeding. Serum amylase will be checked the day any instrumentation is carried out on the pancreas. We also check the amylase level in abdominal drains after pancreatic surgery before their extraction. An extended set of ions is taken on the third day after surgery or after the nutrition support has started to exclude "refeeding-like" hypophosphatemia in nutritionally intervened patients. Liver enzymes, cholesterol and triglycerides must be monitored there too. Wastes in 24-hour urine can also be helpful (nitrogen metabolism, ion depletion/retention). We can use the following possible **markers of prolonged catabolism and suspected hidden complication:** CRP 48-72 hours after surgery should be lower than 80 – 100 mg/l. IL-6 24 – 48 hours after surgery must fall below 400 ng/l. To differentiate between SIRS and sepsis as an early complication, the Intensive Care Infection Score using blood count (ICIS) might be the best marker, with the cut-off being 5. However, presepsin (cut-off 327 ng/l) or procalcitonin (cut-off 2.0 ng/l) are used more often.

ADMINISTRATION OF BLOOD, BLOOD DERIVATIVES, COMPLICATIONS (11)

Urbánek Libor

A blood transfusion is the process during which blood or blood derivatives are injected into the recipient's bloodstream.

Transfusion can be differentiated according to the origin of the blood derivative:

- Allogeneic transfusion – use of other people's blood products
- Autologous transfusion – (autotransfusion) application of the patient's own blood, collected before the planned operation. This procedure is accompanied by a lower risk of complications.

Preparations for transfusion (prepared at the transfusion station):

- whole blood – blood from which none of its components have been removed. It contains erythrocytes, leukocytes, platelets and blood plasma. Its use is, for example, in cases of serious trauma.
- erythrocyte concentrate
- plasma (fresh frozen plasma)
- thrombocyte concentrate (platelet concentrate)
- *Blood derivatives (supplied by pharmaceutical companies from processed plasma)*
- albumin
- concentrates of clotting factors
- fibrinogen concentrate
- immunoglobulins

Blood derivatives and blood are a risk for the transmission of infectious diseases. Therefore, screening of potential risk factors and laboratory testing of the donor for some infectious diseases is carried out.

Application of erythrocyte concentrate:

Pre-transfusion examinations performed in the laboratory:

Examination of blood groups AB0 and Rh system in both the donor and the patient. Screening of the recipient's serum for the presence of irregular antibodies.

The big cross-examination.

The donor's red blood cells are tested against the recipient's plasma. If agglutination occurs, it means the presence of antibodies in the serum against antigens on the surface of red blood cells. The

presence of agglutination is a sign of incompatibility between the blood bank and the patient in question. The absence of an agglutination reaction is a prerequisite for transfusion.

Testing at the patient's bedside

We check recipient's temperature, also blood pressure and pulse are measured. Documentation is checked at the recipient's bedside. We check whether the data on the application form and the blood container match

Checking the blood groups of the donor and the recipient at the patient's bedside:

It is performed using ABO diagnostic bed-side kits. The kit contains anti-A and anti-B sera, pre-printed cards and plastic sticks for mixing the blood sample with the antiserum. Drops of the recipient's blood and samples from the blood bank are applied to the red circles. Anti-A and anti-B sera are dropped into the blue and yellow circles (the antisera should be in excess compared to the amount of blood). The blood is mixed and agglutination is read after 1 minute by carefully tilting the card.

Possible post-transfusion complications:

- Acute hemolytic reaction – occurs when there is incompatibility in the ABO system, especially when the recipient's blood contains antibodies against the donor's erythrocytes,
- Delayed hemolytic reaction
- Febrile non-hemolytic post-transfusion reaction
- Anaphylactic reaction
- Infection – HBV, HCV, HIV, Treponema pallidum, CMV, parvovirus B19,
- Overloading the organism with iron during repeated transfusions,
- Post-transfusion acute lung injury (Transfusion Related Acute Lung Injury, TRALI) caused by leukocytes present in the transfusion
- Volume overload
- Occurrence of cardiac arrhythmia – transfusion of untempered fluid via a central venous catheter near the right atrium
- Transfusion-associated graft vs. host disease (GvHD) – T-lymphocytes of the donor react with the HLA antigens of the recipient; when transfusing blood to immunocompromised recipients or when transfusing from a blood relative



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VENOUS THROMBOSIS, PULMONARY EMBOLISM, VENOUS THROMBEMBOLISM PREVENTION (27)

Kurečková Petra, Vysloužil Pavel

Deep Vein Thrombosis (DVT) and **pulmonary embolism (PE)**, i.e. thromboembolic disease, are one of the most serious, life-threatening complications of surgery.

Etiopathogenesis:

Thrombus formation is closely related to endothelial damage, hypotension and increased blood clotting (Virchow's triad) due to various risk factors, including surgical intervention, which are mentioned below. DVT usually starts distally in the veins of the leg and deep calf muscles, from where it spreads proximally. Initially, a fluttering thrombus is dangerous, as it adheres to the vein wall only partially, later organising by adherence to the wall and leading to an inflammatory reaction in the vein wall. In front of the vein closure, venous congestion occurs, which leads to swelling of the surrounding tissue. In thromboembolism, venous thrombi are released, most commonly from the femoral, iliac and pelvic veins, but also from the subclavian and jugular veins. Localisation of thrombosis in the superior vena cava basin is usually associated with central catheterisation.

Pulmonary embolisation (PE) is caused by the closure of the small circulation by an embolic plug. The level of the pulmonary artery (a. pulmonalis) basin at which the obstruction occurs determines the subsequent clinical picture and severity of the condition. A distinction is made between small, submassive and massive pulmonary embolism. The embolus is most commonly a loose thrombus, but occlusion can also occur due to fatty parts, air bubbles, amniotic fluid, tumour mass, or even a foreign body.

When closure occurs, reflex spasm occurs in the pulmonary circulation but can also occur in the coronary and bronchial circulation. The occlusion of the pulmonary trunk results in right-sided heart failure (acute cor pulmonale). Pulmonary resistance is so high, and overpressure in the right atrium causes dilatation of the right ventricle and its dysfunction.

Fat embolism occurs in polytrauma when the shock state contributes to the instability of the released lipids in the serum, which aggregate. It can be pulmonary or cerebral when fat emboli move in the large circulation.

Air (gas) pulmonary embolism may occur during operations on the neck, arm, head and chest; air may fill the right ventricle or, mixed with blood, obstruct pulmonary capillaries. The underlying cause is a negative pressure in the venous system that draws in a bubble of air or a direct iatrogenic injection of air into the vein. Cerebral and coronary embolism can also occur when air enters through an injury to the pulmonary veins.

Additionally, we need to mention a paradoxical embolism, which occurs when the foramen ovale is open, and the emboli originate from the great circulation.

Risk factors:

The resultant development of thrombosis and, subsequently, pulmonary embolism is usually an interplay of multiple factors.

Congenital thrombophilic conditions include Leiden mutation, prothrombin gene mutation, deficiency of antithrombin, protein C, protein S and others.

By acquired thrombophilic conditions, we mean mainly surgery and trauma, prolonged immobilisation, age over 60 years, cancer, long-standing central venous catheter, history of thromboembolic events, pregnancy and postpartum six weeks, hormonal contraception, sepsis, Covid infection, obesity, smoking, hyperhomocysteinemia, heparin-induced thrombocytopenia and others.

Clinical picture:

Small pulmonary embolisation is often clinically mute, but otherwise dyspnea, chest pain, cough, hemoptysis, tachycardia, syncope, hypotension or shock. Auditory accentuation of the second heart echo and pleural murmur may be present. Signs of central circulation and deep vein thrombosis may be also evident.

Alarming signs of deep vein thrombosis include unilateral oedema. It may be from the periareolar to the whole limb swelling with tissue leakage in Scarpa's triangle, depending on the level of involvement. In deep vein thrombosis of the tibia, the deep muscles of the calf are painful, and the subcutaneous veins on the anterior surface of the tibia, the so-called Pratt's warning veins, may also be accentuated by pressure. The basic physical examination of the lower extremities includes the Homans and plantar sign, where we equip the calf pain with passive dorsiflexion of the foot and compression on the plantar surface of the leg. The most serious picture is ileofemoral thrombosis, when an alteration of the general condition is additionally present, called phlegmasia alba dolens. If the pale limb begins to take on blue tints and marbling, there is a danger of losing the limb as the artery closes reflexively, we call it Phlegmasia Coerulea Solens (PCD).

Differential diagnosis of pulmonary embolism: acute myocardial infarction, aneurysm rupture, aortic dissection, spontaneous pneumothorax, psychogenic hyperventilation and other acute pulmonary complications.

Diagnosis of pulmonary embolism:

The ECG examination may show a cor pulmonale pattern (I S beat, III Q beat, T wave inversion in III, V1-V3, blockade of the right arm of Tawar). ECHOCardiography is sensitive and demonstrates right ventricular dysfunction and increased pulmonary artery pressure. CT angiography of the chest and scintigraphy of the lungs will confirm the diagnosis, but it is not so available among the investigations. Serum fibrin degradation products D-dimers, then troponin and BNP, which indicate the degree of myocardial damage, will be elevated in thromboembolism.

Deep venous thrombosis can be confirmed by ultrasound Doppler examination.

Therapy of pulmonary embolism:

Depending on the severity of the condition, we range from treatment of pain and anxiety to full resuscitation. In thromboembolic disease, anticoagulation therapy is essential. In unstable patients, thrombolysis is indicated. Embolectomy by catheterisation or surgery (Trendelenburg operation) is a causal therapy, but a high mortality rate burdens them. In indicated cases, extra-corporeal membrane oxygenation (ECMO) can be used in some institutions for cardiac support during systemic thrombolysis.

Prognosis of pulmonary embolism:

Massive embolism is very serious with high lethality. Sequelae after pulmonary embolisation include the development of chronic cor pulmonale and pulmonary hypertension. Prevention is important.

Prevention of thromboembolic disease

Especially before surgery, if there is a risk of thrombophilia, prophylactic measures are recommended. It includes breathing training, lower limb exercises, and modification of the internal environment. TED stockings are commonly provided to patients undergoing surgery. After surgery, early mobilisation, breathing and limb exercises are recommended. In bed, the lower limbs are preferably elevated legs with TEDs stoking or Intermittent Pneumatic Compression (IPC) in immobilised patients.

It is important to assess patients' risk of thromboembolic disease. The different scoring systems of surgery societies and centres are very similar. The basic distinction is between low, intermediate and high-risk patients. The prophylactic anticoagulation therapy is set, and Low Molecular Weight Heparins (LMWH) are commonly used. In high-risk patients who need a longer course of DVT prophylaxis, there is a switch to an oral form of anticoagulation or antiplatelet therapy, according to the recommendation of internal medicine physicians and haematologists.

Rarely, a caval filter to prevent embolism may be indicated in patients with presenting deep vein thrombosis, e.g. in case of intolerance to anticoagulation therapy and frequent recurrence of embolism.

HEMATURIA, URINARY RETENTION, ANURIA (24)

Varga Gabriel, Fedorko Michal

Acute urinary retention

Definition:

Acute urinary retention is the sudden inability to empty the bladder.

Pathophysiology:

The most common cause is subvesical obstruction (benign prostatic hyperplasia, urethral stricture, presence of a foreign body) or an impaired lower urinary tract nerve supply. Urinary retention most commonly occurs in men over 60 years old, and it is associated with benign prostatic hyperplasia. This condition is relatively rare in women.

Clinical picture and diagnosis:

It manifests with an abrupt inability to void, an intense urge to urinate and pain in the suprapubic region. It is usually preceded by long-term lower urinary tract symptoms. During a physical examination, we can notice overflow incontinence, also called ischuria paradox, resulting from an exceeded capacity of the bladder. Furthermore, in slender patients, we can observe a distended lower abdomen, painful to palpation and percussion. An ultrasound scan would confirm the diagnosis and reveal an overfilled bladder. Additionally, ultrasound imaging could indicate the aetiology of retention (enlarged prostate, cystolithiasis).

Therapy:

Urinary catheterisation or suprapubic cystostomy (epicystostomy) relieves the retention. Preferably, a transurethral indwelling catheter is placed. In adults, we opt for a catheter of the size CH16-CH18. Further treatment depends on the underlying aetiology of retention.

Hematuria

Definition:

Hematuria is the presence of red blood cells in the urine.

Terminology:

Microscopic hematuria refers to having blood in the urine that can only be confirmed by a microscopic examination of the urinary sediment. When the urine is visible to the naked eye, it is termed **macroscopic hematuria**.

In clinically significant hematuria, there might be a blood clot formation, resulting in the inability to

void. This condition is called bladder tamponade, and the symptomatology resembles acute urinary retention.

Pathophysiology:

Spontaneous hematuria occurs in older people with benign prostatic hyperplasia, prostate cancer, radiation-induced hemorrhagic cystitis, urolithiasis, infection, carcinoma of the bladder, upper urinary tract or kidney. Hematuria could be present in patients on anticoagulation or anti-aggregation therapy. Traumatic hematuria happens as a result of penetrating injury and high-energy trauma associated with kidney laceration, upper urinary tract injury, and urethral or bladder rupture. Different aetiology of hematuria include postoperative bleeding originating from the upper or lower urinary tract after transurethral, percutaneous or open urologic procedures.

Clinical picture:

Blood in the urine, pain above pubic symphysis, and inability to void are the main symptoms. Hemorrhagic shock can occur in case of massive blood loss.

Diagnosis:

Diagnostic work-up is based on taking the patient's history and a physical examination. Hypotension and tachycardia are concerning signs that imply incipient hemorrhagic shock. Crucial laboratory markers are haemoglobin and thrombocyte levels. Examination of clotting times could reveal coagulation disorder. Blood type testing is essential in case of necessity to give the patient a blood transfusion. Ultrasound scan detects a distended bladder and the presence of blood clots surrounded by fluid collection. The diagnostic algorithm of posttraumatic hematuria usually includes a CT scan. This imaging is also advisable in case of suspected bleeding from the upper urinary tract that would require surgical or radiological intervention. If a conservative approach fails, endoscopic examinations of the lower urinary tract can detect the source of bleeding and allow subsequent coagulation.

Therapy:

Clinically significant hematuria requires acute hospitalisation. Assessment of hemodynamic stability is mandatory when bladder tamponade is suspected. A three-port catheter is inserted, followed by repeated bladder irrigation with normal saline and blood clot aspiration. Afterwards, a catheter is used for continuous bladder irrigation. Hemostatic agents should be administered, and coagulopathy should be treated if necessary. The patient must be admitted to an intensive care unit in case of hemodynamic instability and, in such a case, requires hemorrhagic shock treatment. Refractory hematuria, recurrent catheter blockage or persistent worsening of blood count values are reasons for urgent endoscopy. The aim is to extract all the blood clots and treat bleeding sources in the lower urinary tract (coagulation, prostate or tumour resection). In the case of bleeding originating from a lacerated kidney, we indicate selective embolisation if the patient is hemodynamically stable. This

procedure is performed by an interventional radiologist and can be done repeatedly. We can avoid nephrectomy in 67 % of cases. Traumatic bladder rupture requires open surgical repair and bladder suture.

Anuria

Definition and terminology:

Anuria is a decreased urine output or complete cessation of urine production. It often follows oliguria. Anuria is usually a sign of kidney failure – also termed acute renal insufficiency. Long-term or chronic anuria is a manifestation of chronic renal insufficiency. In young children, this condition is defined as urinary output less than 0–0,5 ml/kg/h and 0–50 ml/den in adults. Oliguria is urinary output less than 400 ml/24h or <20 ml/h. It is crucial to differentiate between anuria and urinary retention. In the latter case, urine is retained in the bladder, and the patient cannot pass urine. Kidney failure is when the kidney can no longer adequately filter waste products from the blood and excrete them in the urine.

Aetiology and pathophysiology:

Causes of acute renal insufficiency can be divided into pre-renal, renal and post-renal. Pre-renal causes lead to decreased kidney perfusion, which results in decreased urine production. When reduced perfusion reaches the lower limit of kidney autoregulation (approximately 70 mmHg MAP), renal vasoconstriction increases, and glomerular filtration decreases. This leads to (reversible) elevation of serum creatinine and urea. Renal parenchyma is damaged (ischemic acute tubular necrosis) unless the precipitating factor is eliminated. It is possible to prevent acute tubular necrosis when pre-renal causes are treated promptly. Pre-renal aetiology includes hypovolemia, hypotension, low cardiac output, as well as drugs altering renal autoregulation (especially non-steroidal anti-inflammatory drugs, inhibitors of angiotensin-converting enzyme, angiotensin II receptor blockers). Intrinsic causes encompass a plethora of disorders affecting different renal structures (glomeruli, tubules, blood vessels). Post-renal causes are predominantly diseases that lead to subvesical obstruction (BPH, prostate cancer, urethral stricture), and abdominal and pelvic malignancies causing obstruction of the upper urinary tract. Obstructive uropathy can often have a favourable outcome if it's recognised in an early stage. The aetiology of chronic renal insufficiency involves primarily diabetic nephropathy, chronic glomerulonephritis, hypertension and diseases affecting renal vasculature.

Clinical picture and diagnostics:

There are other clinical signs of kidney failure apart from anuria. Anuria is usually associated with fluid overload, manifested as eyelid swelling, lower extremity oedema, ascites, pleural effusion and pericardial effusion. Non-specific signs of kidney failure include fatigue, nausea or confusion. Loss of appetite, dyspepsia and vomiting are also common. Furthermore, deep breathing associated with metabolic acidosis and „foetor ex ore“ – bad breath as a sign of uremia could be present. Signs of fluid overload are the most prominent when we perform a physical examination. Pale skin, petechiae, purpura or hematomas could be noticed as well. Blood pressure is usually elevated. Laboratory values show elevation of creatinine and urea, hyperkalemia, hyperuricemia, hypophosphatemia, hypocalcemia and signs of metabolic acidosis. Classic signs of chronic renal insufficiency are severe anaemia, hypocalcemia, hypophosphatemia, and hyperparathyroidism, as well as morphological changes as a result of degeneration and atrophy of renal parenchyma. Additional clinical signs of anuria are associated with a distinct type or specific kidney disease that leads to anuria and kidney failure.

Therapy:

Pre-renal and renal anuria is treated with causal therapy and hemodialysis.

Post-renal anuria: relieving obstruction of upper/lower urinary tract (double J-stent placement or percutaneous nephrostomy in case of upper urinary tract obstruction, suprapubic cystostomy or transurethral catheterisation in case of lower urinary tract obstruction).

After restoring renal function, the polyuric phase of renal failure can develop. It is necessary to expect and treat electrolyte imbalances during this phase of renal failure. The final treatment of post-renal anuria is the management of the underlying reason.

INFECTIONS IN UROLOGY (34)

Moravčíková Mária, Fedorko Michal

Urinary tract infections (UTIs) affect the kidney, its surrounding tissue and the urinary tract. The urinary tract is divided into the upper (kidney and ureter) and lower (urinary bladder and urethra). Urological infections also include infections of the male genital organs. It is the second most common infection after respiratory infections, more common in women at a young age, the frequency evens out in older age in both sexes due to prostate hyperplasia. UTIs are the most common hospital-acquired infections.

Most urinary tract infections are caused by *E. coli* bacteria. Other common pathogens include *Proteus mirabilis*, *Enterobacter cloacae* and *Klebsiella pneumoniae*. The most common nosocomial pathogens include *E. coli*, *Pseudomonas aeruginosa* and *Staphylococcus aureus*.

The pathogenesis of most UTIs is an ascending spread. In women, bacteria from the genital area can easily penetrate and ascend up the short urethra into the bladder and then eventually to the kidney. Less common is the spread of infection by haematogenous or per continuitatem. The basic defence mechanism against UTIs is the free passage of urine, its specific properties (pH, osmolality, concentration) and factors inhibiting the adhesion of bacteria to the epithelium.

Acute cystitis – inflammation of the bladder

Typical urinary symptoms are strangury (burning and cutting during urination), pollakisuria (frequent urge to urinate), urgency (uncontrollable urge to urinate), and cystalgia (urinary bladder pain), sometimes associated with macroscopic hematuria. The condition is not usually associated with fevers or alteration of the general state of the patient. For diagnosis, we perform a urinalysis - typically with a finding of leukocyturia or erythrocyturia. In case of repeated infections and atypical symptoms, we also perform a urine culture. A finding of more than 10^5 CFU of bacteria is significant. A suitable therapy is the short-term use of antibiotics (ABX) such as nitrofurantoin, pivmecillinam, fosfomycin, cotrimoxazole or amoxicillin. ABX therapy should be supplemented with increased fluid intake, restriction of heavy physical activity and necessary symptomatic treatment. Since some patients have recurrent infections, prevention is also important - immunoprophylaxis (probiotics, cranberry preparations), personal hygiene, postcoital micturition and treatment of gynecological infections.

Acute urethritis – inflammation of the urethra

It is typically manifested by discharge from the urethra, strangury, redness of the external urethral opening, urgency, pressure pains in the perineum or dyspareunia.

In young patients, it is typically a sexually transmitted infection, the most common pathogens being *Chlamydia trachomatis*, *Ureaplasma genitalium*, *Ureaplasma urealyticum* and *Neisseria gonorrhoeae*. All patients require a urethral swab (the patient must not urinate for at least 2 hours beforehand) and a urine culture examination. The basis of therapy is targeted ABX treatment, most often tetracyclines or azithromycin. It is also necessary to treat all sexual partners.

Acute pyelonephritis - inflammation of the kidney

Kidney inflammation usually precedes the symptoms of acute cystitis. Dull pressure pain in the lumbar region (lumbargia), positive tapotement, painful bimanual palpation of the kidney and pain in the mesogastric area are typical. Patients are febrile, with tremors or chills, sometimes there is also an alteration of the general condition. The para-clinical tests may reveal positive findings in the urinalysis (pyuria), significant bacteriuria in the urine culture, and blood tests may show an elevation of CRP and leukocytosis. It is important to rule out urinary tract obstruction using an ultrasound examination. Oral ATB therapy is sufficient for mild pyelonephritis, for example, with co-trimoxazole, amoxiclav or cefuroxime. It is important to rest, increase fluid intake and adjust therapy according to the sensitivity of the uropathogen to ABX. Patients with altered state, vomiting, and fevers require hospitalisation for rehydration, insertion of a urinary catheter, and administration of IV ABX. We most often administer aminopenicillins in combination with aminoglycosides or 3rd generation cephalosporins.

Urosepsis

It is a life-threatening organ dysfunction caused by the body's deregulated response to the presence of an infection. The most common activators are bacterial endotoxins in the walls of G-bacteria, which activate the release of cytokines. Endothelial and vascular permeability are impaired, procoagulants and coagulation cascade are activated, and fibrinolysis is inhibited, which can lead to disseminated intravascular coagulation (DIC). As a result of the centralisation of blood circulation, tissue hypoperfusion occurs with subsequent tissue hypoxia and the development of metabolic acidosis. In severe cases, septic shock develops with circulatory failure with a high mortality rate. Clinically, tachycardia, tachypnea, fever, disturbances of consciousness and local symptomatology are present. It is essential to rule out urinary tract obstruction at any level. Therapy must be timely with quick identification of the source of infection and provision of urine drainage (urinary catheter, ureteral stent insertion, nephrostomy, epicycstostomy). Doctors should immediately use empirically

broad-spectrum bactericidal ABX parenterally like carbapenems or 3rd generation cephalosporins, possibly potentiated penicillins with aminoglycosides. Hospitalisation in the intensive care unit is inevitable with monitoring, vasopressors and oxygen.

Prostatitis - inflammation of the prostate

We distinguish between acute and chronic prostatitis. The infection most often spreads ascending from infected urine. In addition, catheterisation and procedures in the urinary tract (cystoscopy, prostate biopsy) carry a significant risk of infection. Symptoms include pressure or pain in the anus with propagation to the scrotum, pain in the perineum, painful urination, weak stream, urinary retention, pollakisuria and urgency. Acute prostatitis is usually associated with fevers or alteration of condition, elevation of inflammatory parameters. Diagnostically, a tender and boggy prostate is typical upon digital rectal examination. Prostate-specific antigen (PSA) serum values are usually elevated in prostatitis; therefore, PSA sampling during the period of inflammation is not valid; it can be tested earliest one month after healing. Depending on how severe the infection is, patients with sepsis might be admitted to the hospital for intravenous antibiotics (IV ABX). Moreover, urine drainage should always be ensured. Acute prostatitis can be complicated by the formation of an abscess, which requires puncture and evacuation by the transrectal or transperineal route. Chronic prostatitis is a difficult-to-diagnose and hard-to-treat disease that could be of inflammatory origin, but it could also be part of the so-called chronic pelvic pain syndrome. Treatment that is symptomatic includes non-steroidal anti-inflammatory drugs, analgesics, prostate massages, or alpha-blockers.

Fournier's gangrene

It is an aggressive and often fatal polymicrobial infection of the soft tissues of the perineum, perianal area and external genitalia. This condition is relatively rare, with an estimated incidence of 1.6/100,000 males. In most cases, the source of infection is a skin infection of the genitals or perineum. In most cases, older men are affected, with additional risk factors including diabetes and obesity.

Symptoms come on quickly, and their presentation is quite dramatic. Painful swelling of the scrotum and perineum is typical, followed by the development of sepsis. Necrotic areas of the skin on the scrotum or perineum with surrounding erythema and oedema, as well as blisters, bullae, swelling, foul-smelling exudate and crepitations, are evident. The infection can spread to the anterior abdominal wall, gluteal area and genitals (penis, scrotum and labia). Therapy requires a

multidisciplinary approach, immediate initiation of broad-spectrum ABX therapy and surgical intervention – necrectomy and drainage. Mortality is still quite high nowadays, 20-40%.

Fig. 1: Fournier's gangrene, after debridement and right orchiectomy



URINARY INCONTINENCE (35)

Wasserbauer Roman, Fedorko Michal

Urinary incontinence is defined as spontaneous leakage of urine that poses a social and hygienic problem. It is part of the storage symptoms of LUTS (lower urinary tract symptoms). It occurs in both sexes, twice as often in women. With increasing age, the frequency gradually equalizes in both sexes. The ability to hold urine is determined by the interplay of sufficient urethral sphincter function and proper functioning of the bladder.

Terminology:

We distinguish several types of incontinence. Stress incontinence is a condition caused by insufficient function (contraction) of the sphincter when abdominal pressure increases. Urgent incontinence refers to a condition where the sphincter cannot hold urine due to a sudden increase in vesical pressure due to detrusor hyperactivity. Furthermore, incontinence can be mixed, when both previous types apply simultaneously. Postural incontinence refers to the leakage of urine when the body position is changed, nocturnal enuresis refers to bed-wetting, permanent incontinence is a condition where urine leaks constantly, and coital incontinence refers to the leakage of urine during coitus.

Pathophysiology:

- **Stress incontinence** is the leakage of urine during physical activity, coughing or laughing caused by insufficient activity of the urinary sphincter, when intra-abdominal pressure overcomes intra-urethral pressure. It is more common in women, where it is the result of birth trauma to the muscles of the lesser pelvis, or in men after prostate surgery. In women, the urethra is supported by the levator ani muscle, the endopelvic fascia and the anterior vaginal wall, damage to the interplay of this support or its innervation leads to the development of incontinence. Another essential role is played by the bladder neck, where the internal urethral sphincter is located.
- **Urge incontinence** is the leakage of urine associated with the presence of urgency as part of the syndrome of overactive bladder (OAB) on the basis of hyperactivity (neurological disease, inflammation, BOO, idiopathic, detrusor hypertrophy) or hypersensitivity (inflammation, neurological disease, psychologically determined, idiopathic) of the bladder. It is usually caused by an involuntary contraction, reduced compliance, or a combination of both.
- **Enuresis nocturna**, or bed-wetting, is a condition where involuntary leakage occurs during sleep, is common in toddlers and improves with age, persists in about 10% of seven-year-olds and in 2% of adolescents.

Risk factors:

- **Women:** increasing age, number of pregnancies, child's birth weight, mode of delivery, obesity, smoking, drinking and diet
- **Men:** increasing age, radical surgeries in the lesser for malignancy

Diagnostics:

- **Patient history** should consist of basic anamnestic data with a focus on neurological disease, surgery, trauma or radiotherapy in the area of the small pelvis, in women it is necessary thorough gynecological anamnesis, including ascertaining the number of births, the method of delivery and birth weights of children. Of the medicines, it is necessary to look for those that can potentially influence functioning of the bladder or sphincter (sympathomimetics/sympatholytics, anticholinergics, diuretics, opiates).
- **The physical examination** consists of a basic examination of the patient, a careful external examination of the genitals, examination of pathologies in the area of the urethra, assessment of estrogen insufficiency, position and mobility of the urethra, exclusion of prolapse of the pelvic organs. Basic neurological tests are essential, examination of the bulbocavernous reflex and digital rectal examination to assess the findings on the prostate and evaluation of the tone of the anal sphincter, which has a similar tone to the urethral sphincter.
- **Symptom evaluation** – evaluation of symptoms by interviews and questionnaires. The most frequently used questionnaire is OAB V8 (evaluation of subjective symptoms) and then a voiding diary (patient records fluid intake and output per 24 hours for two to seven days). It is also possible to perform a simple PAD test or weighted diaper test, with this test it is possible to objectify the intensity of urine leakage, the standard test measures the pad after 24 hours and the shortened after 1 hour. The patient puts on a pre-weighed pad and weighs it after the test, and the difference in weight is the amount of urine leaked. Leakage of more than 1.4g in 1-hour and 4.4g for 24-hour confirms a positive test.
- **Urodynamic examination** is an invasive examination of bladder and sphincter function urethras, which is performed using special equipment at the urology clinic. A small catheter with sensors is introduced into the urethra and another into the rectum. The bladder is filled with a sterile solution and bladder and abdominal pressures are measured. After filling the bladder to full capacity if stress incontinence is suspected, a profilometry examination can be performed, which serves to assess the sphincter tone, if profilometry is not indicated, the patient urinates into a prepared device for measuring urine flow, even with sensors inserted. The result is a urodynamic record

that serves to determine the cause and type of incontinence or to determine the cause of the problem.

- **Cystoscopy** is an invasive examination of the urethra and bladder, which serves a detailed visual aspect of the lower urinary tract and the exclusion of possible macropathology.

Treatment of stress incontinence

In the treatment of stress incontinence, surgical treatment dominates, which has significantly better results than conservative treatment, although it is recommended to start treatment with conservative methods. Conservative methods of treating stress incontinence include behavioural therapy, pelvic floor muscle rehabilitation and/or pharmacotherapy (alpha-adrenergic agonists, estrogens).

- **Behavioral treatment** uses bladder training and modification of the drinking and dietary regimen
- **Pelvic floor exercises** or also Kegel exercises strengthen the muscles of the pelvic floor, thereby reducing the frequency and intensity of leaks when performing this exercise regularly
- **Surgical treatment**
 - **Tape operations:** The basic types of tape operations are TVT (tension-free vaginal tape) and TOT (transobturator tape). There are many different types of tapes but the basic principle is the same for all of them, which is to provide a suspension and support for the urethrovesical connection between the bladder and the urethra. This aims to ensure outflow resistance of the urethra and improve continence. Fig.1
 - **Artificial sphincter implantation** (AUS) is used for moderate to severe incontinence that does not respond to conservative therapy. It is mainly performed on men. Tape operations can also be used in men, but they tend to have worse outcomes than AUS. The artificial sphincter consists of 3 parts - a cuff that is inserted around the urethra, a fluid reservoir located in the lower abdomen and a pump located in the scrotum, which is used to pump fluid and thus fill or empty the cuff around the urethra, thus ensuring continence and preserving the possibility free urination. Fig.2
 - **Intraurethral injection:** a special substance is applied endoscopically to the bladder neck into the submucosal layer (collagen, autologous fat, silicone, Teflon), which narrows the diameter of the urethra and thus increases the resistance in the urethra tube.
 - **Retropubic operations:** the most common type of operation is the so-called colposuspension. Patients with moderately severe stress incontinence without the response to conservative treatment are indicated for this procedure. The current trend is to perform this procedure laparoscopically.

Treatment of urge incontinence

Again, conservative and operative methods are used to treat urgent incontinence. Compared to stress incontinence, conservative methods have a much higher success rate and are the dominant method of treatment.

- **Conservative methods:** weight loss, stop smoking, diet and drinking regimen, practice micturition at regular intervals, bladder drill, pelvic floor exercises
- **Pharmacotherapy** is the basic method of treatment, it uses drugs to suppress the activity of the bladder detrusor. The most widely used drugs are anticholinergics (darifenacin, fesoterodine, oxybutynin, solifenacin, trospium). Another option is mirabegron, which is a beta3agonist acting via smooth muscle cell beta receptors and inducing detrusor relaxation.
- Surgical treatment is not the dominant type of treatment, but is used in strictly indicated cases when medical treatment fails.
- o **Intravesical therapy** is the more commonly preferred surgical treatment. Botulotoxin is applied endoscopically into the wall of the bladder, the procedure consists of applying 20-30 injections of diluted Botulotoxin, the effect lasts 6-9 months and then the procedure must be repeated.
- o **Sacral neurostimulation and neuromodulation** using electric current after prior implantation of the needle to the relevant nerve, thereby stimulating the inhibitory nerves leading to the detrusor and thus inhibiting contractions.
- o **Neurostimulation of the posterior tibial nerve** uses a special device that uses a thin needle to stimulate this nerve in the medial ankle area.
- o **Augmentation procedures** on the bladder are almost no longer used, they are used to create a diverticulum of the mucosa, which will allow an "escape space" for urine in case of hyperactivity, so that it does not leak. The last treatment option is to perform a derivation procedure, when a replacement from the intestine is created instead of the bladder and the bladder itself is actually removed.

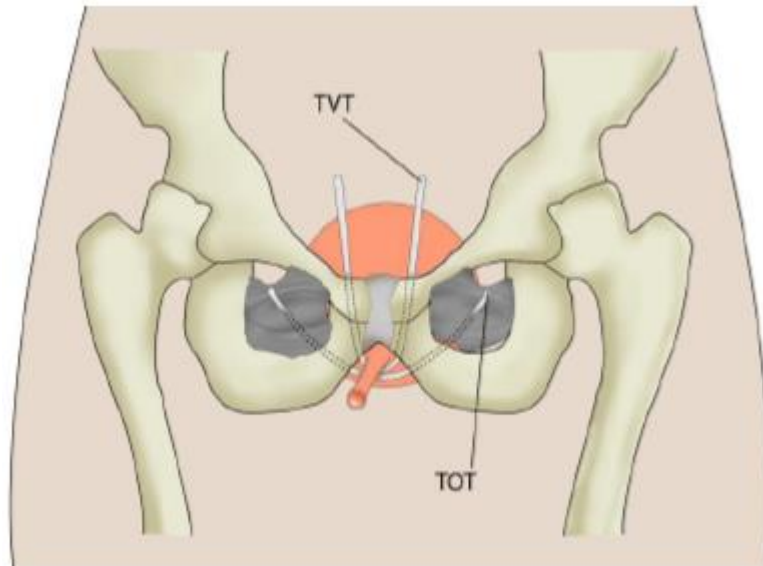


Fig. 1: tape operations, (source: Fedorko, 2020)

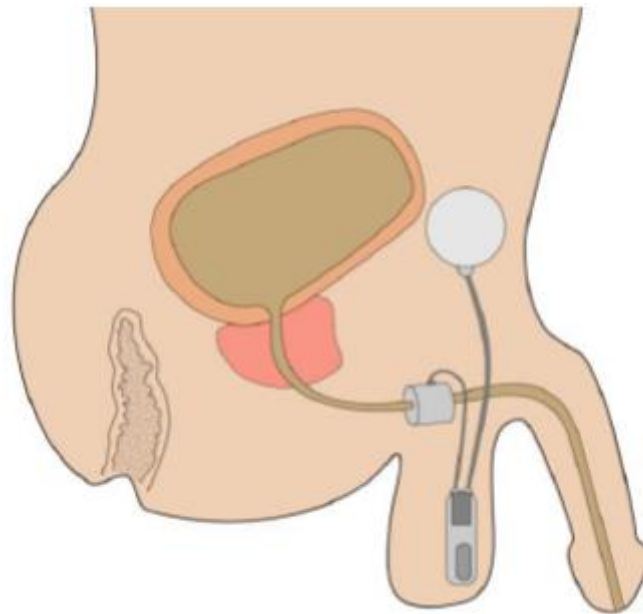


Fig. 2: artificial urinary sphincter (source: Fedorko, 2020)

SIGNS AND SYMPTOMS OF ACUTE ABDOMEN (23)

Hemmelová Beáta

Acute Abdominal Emergency (AAE) is acute abdominal disease that demands urgent attention and treatment. With proper diagnosis and treatment follows a relatively quick recovery; otherwise, the patient is at risk for serious complications, potentially resulting in death. The symptoms of AAE can be very acute and severe, or mild and nonspecific, requiring a thorough diagnostic procedure, detailed clinical examination, obtaining patient history, laboratory tests, and complementary imaging methods, abdominal ultrasound or CT. The development of clinical symptoms over time plays an important role, making it essential to conduct repeated clinical examinations alongside paraclinical evaluations.

Primary Symptoms of AAE: Pain

Pain from the abdominal cavity is transmitted to the central nervous system through nerve pathways involving both somatic and autonomic nerves. Somatic nerves transmit pain from somatic structures (such as skin, muscles, or damaged abdominal walls), typically relaying pain via spinal nerves.

Spinal nerves C3-C5 (via the phrenic nerve) are responsible for pain from diaphragm irritation, while Th6-Th12 spinal segments innervate most abdominal organs like the stomach, liver, and intestines, where this pain is experienced as visceral pain, making it difficult to localize.

Visceral pain is conveyed by autonomic nerves. Sympathetic nerves originate from the thoracic and lumbar spinal cord. Although visceral pain is typically less sharp and less well localized than somatic pain, it may induce autonomic reactions such as vomiting or changes in heart rate. Parasympathetic pathways (including the vagus nerve) play a role in transmitting painful signals from the lower abdominal organs, such as the intestines and bladder. Additionally, pain from internal organs may be "referred," meaning it can be felt in remote areas. For instance, appendicitis can cause pain around the navel that later shifts to the right lower quadrant. **Character of Pain:** Initially, it can provide diagnostic clues. Continuous, escalating pain often indicates inflammatory AAE (e.g., appendicitis, cholecystitis, peritonitis). Colicky pain may indicate biliary colic, renal colic, or mechanical ileus. Pain from intestinal ischemia or perforation of the gastroduodenal ulcer may present with sudden onset, transient improvement, and ultimately progress to peritonitis. **Radiation of Pain:** For instance, hydronephrosis may cause referred pain beneath the right scapula, while renal colic may lead to pain radiating to the groin, back, and under the left rib cage in instances of pancreatitis or rupture of the abdominal aorta, with pain localized beneath the right rib cage from gallbladder issues, duodenal ulcers, or liver enlargement.

Nausea and Vomiting accompany almost every AAE and can have various causes: Irritation of nerve pathways that innervate the affected abdominal organ during distension or inflammation. Inflammation e.g., gastroenteritis, colitis, or cholecystitis may irritate the mucosa, activating receptors in the stomach and leading to vomiting. Obstruction due to intestinal volvulus — leads to a disruption of passage, distension of intestinal loops and the stomach, causing nausea and vomiting. A systemic response during certain AAE, leads to the release of inflammatory mediators into the bloodstream, which can affect the vomiting center.

Loss of Appetite: Anorexia is a common bodily response to inflammation or pain; it may also be accompanied by altered habits in smoking or alcohol consumption, serving as a diagnostic indicator of disease severity. It is also essential to inquire about dietary indiscretion or excess preceding the onset of AAE in the patient's history.

Fever: Fever levels provide insight regarding potential causes and severity of the condition.

Subfebrilis (up to 38°C) suggest mild inflammatory processes, such as early-stage appendicitis or gastroenteritis. *Febrilis* (up to 39°C) may indicate conditions like cholecystitis, appendicitis, or diverticulitis, both locally inflamed. *Febrilis* over 39°C are associated with systemic inflammatory responses due to diffuse peritonitis. Chills followed by high fevers are characteristic of renal colic, biliary obstruction with cholangitis, leading to the onset of septicemia. Febrile continuums occur in severe infectious states, such as perforated appendicitis with diffuse peritonitis, perforated ulcers, sepsis, or severe pancreatitis.

Afebrile AAE: Conversely, no organismal response producing increased temperature are often due to passage disorders in the early clinical stage, intestinal obstruction, ischemic colitis, or forms of choledocholithiasis. The absence of fever may occur if no significant inflammatory response develops or if the patient's body is exhausted, reflecting a lack of defense mechanisms, typical for geriatric or polymorbid patients. The dynamics of febrile states provide significant information: rapid onset of high fever with quick deterioration may suggest acute inflammation. A vital diagnostic step in the presence of fever is acquiring biological material for potential infectious agents before starting antibiotic treatment. This includes urine samples, swabs, or pus from the infection site, or blood cultures in cases of septicemia.

Changes in Gastrointestinal Passage (Diarrhea or Constipation): Changes in stool characteristics may reflect various abdominal conditions. For instance, digestive distress caused by infectious gastroenteritis can lead to diarrhea, while intestinal obstruction or diverticulitis can cause constipation. Paradoxical diarrhea may occur due to stool retention above an obstruction and its passage in a liquefied form during dilation of the bowel or above retained fecal matter. The stool's

characteristics, color, odor, and presence of blood, hematin, or mucus also provide important diagnostic information.

Abdominal Distension – Swollen Abdominal Walls, Meteorism : Accumulation of gas or fluid in the abdominal cavity may lead to distension and swelling of the abdominal wall, potentially causing intra-abdominal hypertension. This can be caused by intestinal obstruction or inflammatory processes that provoke fluid retention, or through digestive disorders. Abdominal distension may manifest as elevation of the abdominal wall, a characteristic "drum-like" sound upon percussion, and diffuse tenderness upon palpation. Measurement of intra-abdominal pressure can objectify this value, serving as a guide, especially in monitoring disease progression over time and the risk of multi-organ failure, e.g., in acute pancreatitis or paralytic ileus. Normal intra-abdominal pressure (IAP) typically ranges from 0 to 5 mm Hg (–17 to +5 mm Hg during expiration); pressure above 12 mm Hg is considered intra-abdominal hypertension, and pressure above 20 mm Hg may indicate abdominal compartment syndrome, necessitating immediate diagnostic and therapeutic measures to prevent organ failure. (Intra-abdominal pressure is usually measured by placing a catheter in the bladder.) The first step involves decompressing the digestive tract via the insertion of a nasogastric tube, performing endoscopic decompression of the colon, and, as a last resort for therapeutically unmanageable hypertension, establishing an open abdomen through laparostomy and provisional closure using non-adherent materials.

Symptoms of Peritoneal Irritation, Reflex Contraction of the Abdominal Wall

In cases of localized inflammation within the abdominal cavity, for instance, appendicitis or cholecystitis, the abdominal wall muscles may contract reflexively in response to pain and inflammation in the affected region. This defensive mechanism causes significant tenderness upon touch in the affected area, which is categorized as a symptom of localized peritonitis. In instances of diffuse peritoneal inflammation (diffuse peritonitis), the abdomen presents as rigid, and the patient may resist movements, with respiratory waves also restricted. Based on the localization of peritoneal irritation, diagnostics can be directed accordingly. **Hemorrhage** can be into the abdominal cavity from trauma, injury to parenchymal organs, mesentery, or rupture of an abdominal aortic aneurysm, where signs of hypovolemia and peritoneal irritation from trapped blood manifest along with the development of hypovolemic shock. In cases of injury to parenchymal organs, initial symptoms may include pain in the region of the liver, spleen, or kidneys due to tension on their capsules, followed by later rupture of a hematoma, presenting with hypovolemia and signs of peritoneal irritation. Gastrointestinal hemorrhage may present as vomiting blood or the passage of digested blood (melena), or alternatively enterorrhagia or proctorrhagia, depending on the source within the digestive tract. It is crucial to objectively identify blood in vomit by checking for discoloration from

previously ingested liquids or foods that may mimic blood. Similarly, identifying melena (digested blood) during rectal examination and in cases of enterorrhagia should be accompanied by at least preliminary examination via rectoscopy to assess the location of the source in the anorectal area or more proximal gastrointestinal tract, following which upper GI endoscopy and, in cases of enterorrhagia, colonoscopy may be indicated. If the source of hemorrhage cannot be localized, further steps include CT angiography to demonstrate its extravasation.

Basic Vital Functions

Monitoring basic vital functions (consciousness, blood pressure, pulse, temperature, respiration, oxygen saturation) is an indispensable part of the diagnostic and therapeutic process for acute abdominal emergencies, and any changes should always be documented in medical records. Changes in these parameters indicate the severity of AAE progression. Physical examination involves palpation, percussion, auscultation, and rectal examination and is focused on the above-mentioned symptoms. All types of acute abdominal emergencies are associated with pain, nausea and vomiting, and disturbances in the passage of gas and stool (functional or mechanical). Inflammatory AAEs tend to exhibit more pronounced symptoms in terms of increased respiratory and heart rates, as well as localized signs related to specific organ and peritoneal involvement, especially in the early stages of the disease; in later stages, mechanical and inflammatory symptoms may overlap. Inflammation within the abdominal cavity can lead to complete intestinal paralysis with all the signs typical of a developed ileus state. When establishing a diagnosis, it is crucial to consider **extra-abdominal causes of abdominal pain** related to thoracic diseases like pneumonia, as well as acute heart failure, myocardial infarction, dissection or ruptured abdominal aortic aneurysm, renal conditions such as pyelitis, perirenal abscess, hydronephrosis, urolithiasis, and also retroperitoneal hematoma or vertebrogenic pain syndrom. **Pseudoperitonitis** can be accompanied by metabolic disorders arising from decompensated diabetes, uremia, or porphyria but may also be part of leukemia or hemophilia. **AAE** are a daily practice in surgery, making it essential to observe their symptoms in patients from the first contact. However, in pregnant women, geriatric patients, individuals with impaired mental health, or with intellectual disabilities, it may be challenging to recognize symptoms. Thus, in cases of uncertainty, it is recommended to admit the patient for observation. While pain may be exhausting, it is important not to suppress it before a diagnosis is established to avoid obscuring the expressed symptoms.

ACUTE ABDOMEN – CLASSIFICATION, PRACTICAL MANAGEMENT (22)

Kučera Adam, Horváth Teodor

Acute abdominal conditions (AAC), also known as acute abdominal emergencies, are not a term for one specific disease but a collective term for a group of conditions with etiologically and clinically heterogeneous origins requiring rapid diagnosis and timely treatment. The basic division from an aetiological perspective categorises AAC into 1) traumatic and 2) non-traumatic. Traumatic AAC are divided based on the integrity of the abdominal wall into A) closed and B) penetrating injuries. Non-traumatic AAC are classified by aetiology into A) inflammatory, B) ileus-related, and C) haemorrhagic.

Diagnosis of AAC:

To make a correct diagnosis, it is essential to follow a diagnostic and treatment algorithm that can be universally applied to all suspected AAC cases.

1/ **Thorough Taking of a Patient's History** is a fundamental part of the clinical examination and should always be performed, provided that time, the patient's consciousness, and cooperation allow. We inquire about the pain, its character, location, intensity, radiation, migration, time dynamics, and whether there is a relieving position. Abdominal pain is classified based on the nerve fibre pathways into A) visceral, which is typically dull and difficult to localise, and B) parietal, arising from peritoneal irritation, which is typically sharp and well-localised. Both types of pain may combine. In some AAC, the pain presents as colic, with rhythmic weakening and intensification (intestinal, renal, biliary colic, or colic due to gastroduodenal ulcers). We also ask about anorexia, nausea, vomiting, the nature of the vomitus and pathological admixtures (blood, mucus, bile), the time of the last bowel movement, and the presence of pathological admixtures. Unintentional weight loss should be noted, particularly to exclude oncological conditions. Finally, we focus on the patient's history, including chronic illnesses. We ask about medications that modulate pain: strong opioids may mask the pain entirely, NSAIDs can cause gastroduodenal ulcers, antipsychotics and opioids may affect GI motility and anticoagulant and antiplatelet therapy may exacerbate haemorrhagic symptoms.

2/ **Physical examination** is an indispensable part of the diagnostic algorithm for every suspected AAC case and cannot be replaced by any paraclinical examination. We use all five examination modalities.

A/ **Inspection** assesses the patient's overall condition, painful posture, relieving position, habitus, the state of the abdominal wall, external deformities, scars, abdominal distension compared to the chest (niveau), breathing pattern, and pathological distension of venous patterns like "caput medusae" in portal hypertension. Additionally, we inspect the skin, noting pathological efflorescence, jaundice, cyanosis, the state of the adnexa, and hygiene.

B/ **Palpation** starts from the point farthest from the maximum tenderness. Premature manual stimulation of the painful area can lead to peritoneal irritation, preventing an objective assessment of the abdominal findings. We begin with light superficial palpation to assess the tension of the abdominal wall and reflex contraction of the abdominal muscles (**defense musculaire**), the presence of resistance, and the painful response. We proceed towards the centre of the pain, evaluating its intensity and radiation. We assess for signs of peritoneal irritation (/Blumberg's sign, Rovsing's sign, Plénies' sign, Murphy's sign/). After superficial palpation, we perform deep palpation, not only assessing the painful area but also evaluating the condition of other abdominal organs, palpating the liver and its extension beyond the right costal margin, and performing bimanual palpation of the spleen and kidneys.

C/ **Percussion** of the abdomen should typically yield tympanic sounds. A dull or shortened percussion note is found in ascites, fluid collections, abscesses, or tumours. On the other hand, an accentuated or hyper-resonant percussion note is present in excessive meteorism, intestinal pneumatosis, or pneumoperitoneum. Diffuse tenderness on percussion is a sign of peritoneal irritation, known as Plénies' sign.

D/ **Auscultation** is used to assess bowel sounds. Absent or diminished bowel sounds with high-pitched tones may be a finding in ileus-related AAC or advanced peritonitis. In mechanical bowel obstruction, vigorous peristalsis may be above the obstruction, while peristalsis below the obstruction may be weakened.

E/ **Rectal examination** must not be omitted. Omitting it in the diagnostic algorithm of AAC is considered "non-lege artis". We inspect the area around the anus, checking for external haemorrhoids, fissures, or external openings of perianal fistulas. Through digital rectal examination, we check the tone of the sphincters, which may be weakened in peritonitis or low bowel obstruction, and palpate to exclude the presence of haemorrhoids. We assess the rectal ampulla's filling and the consistency of the stool, identify low-lying rectal tumours, and detect bulging of the Douglas/Proust

space, which, as the anatomically lowest point, can accumulate fluid. In women, we assess the adnexa; in men, we assess the prostate felt through the anterior rectal wall. Stool admixtures can help identify GI bleeding (enterorrhagia, melena).

3/ **Paraclinical investigations**, in terms of laboratory and imaging methods, represent another essential modality in the comprehensive diagnostic algorithm.

A/ **Laboratory tests** provide insight into the patient's internal environment. Elevated inflammatory markers (leukocytosis or, conversely, leukopenia, CRP, PCT, IL-6), blood count, electrolyte panel, acid-base balance (ABR), creatinine, and urea, serum bilirubin, liver panel, amylase, and lipase. In women, pregnancy (as a cause of abdominal pain) must be ruled out, with hCG elevation in laboratory tests.

B/ **Imaging methods** are an inseparable part of AAC diagnostics. A plain abdominal radiograph (PAR) is a quick and accessible method indicated in suspected perforation (pneumoperitoneum) or ileus (pathological fluid levels). Another method is **ultrasonography (USG)**, which better assesses soft tissue conditions and free intra-abdominal fluid. It can detect fluid collections, abscesses, and inflammatory infiltrates and identify free fluid without reliably determining its nature (blood, transudate, ascites, intestinal contents). **CT** is now commonly used as a diagnostic tool and represents the next step in the algorithm in cases of unclear diagnosis. Due to its time-consuming nature, **MRI** is not commonly used in AAC diagnostics.

C/ **Endoscopic methods**, including fibroscopy and colonoscopy, are irreplaceable in diagnosing and treating many GI conditions. In the context of AAC, they are most commonly used in diagnosing and treating bleeding.

Traumatic AAC

From a diagnostic and therapeutic perspective, it is essential to assess the integrity of the abdominal wall and differentiate between A) closed and B) penetrating injuries. Parenchymal organs (liver, spleen, kidneys), large vessels, or the digestive tract can be damaged. In gastrointestinal perforation, we can expect signs of pneumoperitoneum; whereas in parenchymal organ laceration, hemoperitoneum and free fluid. The foundation is systemic management of potential haemorrhagic or septic shock, fluid resuscitation, timely diagnosis (including the FAST protocol), CT, and early surgical intervention.

Non-traumatic AAC:

A/ **Inflammatory AAC:** Acute appendicitis, acute cholecystitis, acute pancreatitis, acute diverticulitis, acute peritonitis, acute adnexitis, tubo-ovarian abscess.

B/ Ileus-related AAC: 1/ Mechanical ileus, 2/ Neurogenic ileus, 3/ Vascular ileus.

C/ Haemorrhagic AAC: Visceral thrombosis/embolism, GI bleeding, aortic aneurysm dissection, ectopic pregnancy.

HEAD INJURY (26)

Staňa Martin

Head injury is the most common cause of trauma-related deaths (mortality approximately 50%). It is the leading cause of long-term disability. Injury to one structure is often associated with injuries to others. Therefore, it is necessary to consider possible concomitant injury to the cervical spine.

SCALPATION means the separation of the soft tissues of the scalp. It can occur, for example, by pulling the hair into a machine or in traffic accidents. With open head injuries, severe bleeding can be expected. Careful haemostasis is required. In the case of total scalp surgery, a complex and time-consuming replantation of the scalp, including restoration of the vascular supply and revision of the exits of the affected nerves, is offered. In cases of subtotal or partial scalp avulsion, wound revision and suturing, often accompanied by drainage, may suffice. However, it is sometimes necessary to employ plastic surgery techniques, such as the use of tissue expanders, dermo-epidermal grafts, and, less commonly, flap surgery.

CONCUSSION represents one of the less severe forms of brain injury, often referred to as mild "traumatic brain injury (MTBI) or minimal trauma brain injury. It is characterised by the absence of focal neurological findings, with objective changes being non-specific. Concussion involves a transient disruption of brain function resulting from axonal stretching and reversible membrane dysfunction. Its symptoms are initially similar to those of more serious brain injuries, making early differentiation challenging. These symptoms commonly include a temporary impairment of consciousness, lasting from several minutes to an hour. Dizziness, nausea, and vomiting may also be present.

Initial **treatment management** involves close monitoring vital signs. On admission, a computed tomography (CT) scan of the brain is typically performed based on established criteria, such as the Glasgow Coma Scale (GCS), to exclude the possibility of intracranial haemorrhage. If concussion symptoms persist, the patient should be admitted for observation for a minimum of 24 hours. During the first 24 hours of admission, it is essential to monitor blood pressure (BP), heart rate (HR), level of consciousness, alertness, mobility, and pupillary status every hour. Specific warning signs include: sudden onset of unconsciousness, behavioural changes following the trauma, vomiting, periorbital bruising ("raccoon eyes"), clear fluid discharge from the nose or ears (suspected cerebrospinal fluid), bleeding from the ears, unequal pupil size (anisocoria), and nystagmus.

A repeat acute CT scan is mandatory in case of sudden clinical deterioration. Even after the acute symptoms resolve, memory deficits—such as anterograde and retrograde amnesia—may persist. Similarly, other longer-term symptoms, also known as post-concussion syndrome, may include headache (cephalea), fainting, dizziness during walking or movement, orthostatic tachycardia and hypotension, impaired concentration and memory, as well as sleep disturbances. This syndrome can persist for several days to months.

INTRACRANIAL INJURIES include trauma to the skull and brain extending beneath the dura mater and may result in death or severe, permanent harm to the patient. These injuries may occur in isolation or as part of polytrauma. **Primary injuries** are directly linked to the trauma and include skull fractures, cerebral coma, cerebral contusions (bruising of brain tissue), lacerations of the brain parenchyma, and diffuse axonal injury (DAI). A **lucid interval** is a temporary improvement in a patient's condition after a traumatic brain injury, especially in epidural haemorrhage, after which the condition deteriorates. This phenomenon is highly dangerous if underestimated. It may be obscured by concomitant diffuse brain injury or overlooked in cases of concurrent alcohol intoxication. **Secondary injury** develops over time following the initial trauma and can be therapeutically influenced. These include intracranial haematomas (epidural, subdural, subarachnoid, and intracerebral) and cerebral oedema, which can lead to hypoxia and ischaemia. Additional serious complications include liquorrhoea and intracranial infections, such as meningitis, encephalitis, and brain abscesses.

In cases of rapid deterioration in consciousness and a Glasgow Coma Scale (GCS) score of ≤ 8 , intubation and artificial pulmonary ventilation should be initiated. Patients with pupillary dilation should receive a bolus of mannitol, which reduces intracranial pressure and subsequently improves cerebral blood flow. Once conservative therapeutic measures—such as the administration of mannitol and NaCl, sedation, and mild hyperventilation—have been exhausted, surgical treatment should be considered. This may involve craniectomy with the evacuation of the haematoma. An unconscious patient usually needs nutritional support, usually via a Nasogastric tube (NG tube).

Maintaining **intracranial Pressure (ICP)** below 20 mmHg is essential to preserving cerebral perfusion pressure. Hypovolaemia must be avoided, even if catecholamines are required. **An intracranial pressure sensor** is indicated in patients with a GCS score of 3–8 and evidence of pathology on a brain CT scan. CT imaging should be performed after surgical interventions, at regular intervals during the intensive care unit (ICU) stay, prior to discharge, and in response to any sudden changes in the patient's neurological status.

Further details will be provided in the chapters of the Neurosurgery in Special Surgery section.

CERVICAL SPINE INJURIES most commonly occur in traffic accidents, falls from height, and especially during head-first dives into shallow water or when axial force is applied to the head. Cervical spine injuries should always be considered in patients with head injuries. In unconscious patients with suspected cervical spine injuries, a rigid cervical collar should be applied, and it must not be removed until these injuries have been excluded using appropriate imaging methods, such as an X-ray or CT scan. Sprains and strains of the cervical spine are typically associated with pain and muscle stiffness. Management involves rest and the use of a soft cervical collar followed by physiotherapy until symptom relief. In cervical spine injuries with bony fragments protruding into the spinal canal or spinal oedema, neurological symptoms—ranging from paraesthesias (e.g., tingling of the fingertips) to paresis or plegia—may present below the affected segment, depending on the severity of the spinal cord injury. In the regions of C0 (occipital condyles), C1, and C2, ligamentous injuries may be lethal. Mild to moderate cervical sprain injuries, such as whiplash, are treated with a soft cervical collar. Stable skeletal injuries may be managed conservatively with a rigid cervical collar (e.g., Philadelphia or SOMI). However, in cases of unstable injuries or neurological deficits, surgical stabilisation is needed, which may involve a posterior, anterior, or combined approach. As part of a trauma protocol, immobilisation with a rigid cervical collar is essential to ensure airway patency. Nonetheless, the maintenance of vital signs takes precedence over the preservation of neurological function, particularly in upper cervical spine injuries (above C4).

FRACTURES OF THE SKULL include simple fissures that do not require treatment but indicate the severity of trauma with a risk of intracranial lesion and require hospitalisation of the patient. Fragmentary and impression fractures may be accompanied by more severe trauma requiring decompression of the brain and repositioning of the fragments. It is also necessary to revise and suture the dura mater. This procedure is a prevention of post-traumatic epilepsy. Radiography of the calva for trauma is an obsolete examination. If a fracture is suspected, it is better to perform a CT scan of the head and search for intracranial injury directly. A CT scan of the brain and skull will also show severe skull base fractures. A spectacle hematoma (a monacle is possible in single orbital injuries), an unconfined subconjunctival hematoma, and ear and nasal liquorrhoea (usually masked by blood content) may suggest their presence. The main danger in skull fractures is meningitis and sometimes an abscess. We administer prophylactic antibiotics and antiseptic drops locally. We forbid blowing the nose because of the risk of pneumocephalus. Liquorrhoea usually resolves itself within 2-3 weeks. Injuries to the cranial base and splanchnocranium may also result in cranial nerve dysfunctions.

THE CRANIOFACIAL SKELETON is related to the anterior cranial fossa, and its injuries may extend to the skull base. Facial injuries are often managed in collaboration with a maxillofacial surgeon, ENT, ophthalmologist, plastic surgeon, neurosurgeon and anaesthetist. Preservation of airway patency is essential. The occlusal plane of the teeth (bite), the ability to eat and articulate, eye movement and innervation by the cranial nerves may be impaired. A hydraulic (blow-out) fracture of the orbit may occur after a blunt impact on the eyeball (the bulbus). Multiple dislocated bone fragments must be repaired and stabilised to an anatomical position. Metal implants are used for this purpose: small plates and screws. In case of fractures of one of the jaws, temporary interdental fixation with a wire bond can be used. Fractures of the midface include Le Fort I-III fractures. Clinical examination is made difficult by rapidly forming swelling and hematoma. Surgery is performed in the vast majority after it has resolved. Nasal intubation is preferable during surgery.

NOSE, EYE, CAVITIES AND EAR examination include the points mentioned above. Discharge from the ears and nose should be observed, and eye movements and pupillary status should be noted, along with other signs of skull base injury. Isolated nasal bone fractures are repaired by the ENT physician or supplemented by nasal tamponade. He examines the ear, may introduce an ear strip and indicate antibiotics. Isolated eye and orbital injuries should be treated by an ophthalmologist, along with extraction of any foreign bodies. Dislocated fractures are indicated for acute surgery, as well as salvage ocular procedures. Therapy includes evacuating secretions from the respiratory tract (Airways), and the use of nasal drops with anticongestants. Adequate fluid intake and nutritional and oral hygiene are emphasised. The dentist simultaneously treats the injuries of the oral cavity.

COMMENTS AND CONTROVERSY: To speed up the diagnostic process and rationalise the use of ionising radiation, brain CT and cervical spine X-ray are becoming the gold standard for head injuries with the risk of cervical spine injury. In cases of radiographic suspicion of spinal injury, CT of the spinal segment is always performed. According to the guidelines, a CT of the brain and cervical spine is requested in the first instance in cases of symptomatology (neurological deficit) and thus an obvious need for spinal surgery due to pathologies of this area (M. Bechterev), previous injuries, and advanced age, which impairs the readability of a plain radiograph. When examining a patient with polytrauma in a whole-body protocol, the CT scan automatically includes the head and cervical spine, thoracic, abdominal, and pelvic cavities.

COMPARTMENT SYNDROME (25)

Melichar Jindřich, Ledvina Jan, Ledvina Tomáš

Compartment syndrome (CS) is a condition in which increased tissue pressure within a enclosed space (compartment) reduces perfusion to such an extent that the metabolic needs of tissues are not met and their neuromuscular functions are impaired.

Damage to tissue structures within the compartment can be caused by: reduction of the fascial bed volume (closure of the fascia defect with a tight suture, tight dressing, circular burns, prolonged immobilization with a pneumatic splint), increase of the compartment content (bleeding and edema after fractures, intense exercise, cramps or burns), reduction of the tolerance against tissue pressure (when arterial perfusion pressure is reduced, e.g. In the case of a decrease in the arterial perfusion of the limb or systemic hypotension).

There are three theories to explain the local circulatory disturbances in CS: the arterial spasm theory, the critical closing pressure theory, and the arterio-venous gradient theory. (The third theory is generally the most accepted one. According to it the rise in tissue pressure reduces local arterio-venous gradients and thus local blood flow.)

Division: 1. primary CS - further divided into a) imminent CS, b) manifested CS, 2. secondary CS

In imminent CS, there is no reduction in peripheral blood flow, the microcirculation is "still" sufficient. Symptoms of neurological disorders are absent or subtly expressed. The leading symptom is severe pain, unexplained by primary trauma. In patients with normal blood pressure, the subfascial tissue pressure threshold is between 30 and 40 mm Hg.

Manifested CS is characterized by perfusion shortage and increasing neurological deficit with loss of muscle function. Objective signs are swelling and palpation-detectable tissue tension of the affected compartment with severe pain.

Secondary CS can occur after removal of a strangulating limb dressing or after a revascularization procedure, when the volume of muscles increases because of postischemic swelling and presses on the borders of the fascial bed.

The anatomical division determines the CS according to the body parts - upper or lower limbs, possibly other parts of the human body (abdominal CS, retroperitoneal CS, intracranial CS).

Diagnosis of CS only from clinical examination in patients with associated head, spinal or peripheral nerve injuries is not possible and it is necessary to use other diagnostic techniques.

Subjective symptoms: spontaneous, movement- or pressure-induced pain with increasing tendency and disproportionately greater intensity than corresponds to local findings. Paresthesias, dysesthesias or even anesthesia.

Physical examination remains crucial for the diagnosis of KS in most cases. The leading early symptoms are sensory and motor impairment. In uncooperative patients, with impaired consciousness or neurological deficit, tissue pressure measurement is needed.

Measurement of subfascial tissue pressure is the most valuable method to make an objective diagnosis of CS. Measurements can be performed in a single session, but monitoring the dynamics of pressure values is preferable. It is measured using needle methods, catheters or piezoelectric sensors. Other examinations: electromyography, measurement of muscle blood flow, angiography, ultrasound examination of peripheral vessels, laboratory tests.

The treatment of patients with KS depends on whether the KS is imminent or manifest. In imminent CS, anti-edematous medication, vasodilators and sympathetic blockade can be applied. The most urgent measure when CS is suspected is to split the strangulation dressing wide open and place the limb no higher than the level of the heart atrium. In the severely injured, blood volume modification in the sense of increasing mean arterial pressure is necessary to improve arteriovenous pressure differential. The definitive and causative treatment is decompression by dermatofasciotomy providing effective pressure relief. During revision, we always check the vitality of the muscle tissue using the "4 C" rule - contractility, consistency, coloration, capillary blood supply.

The most common site of KS is the lower leg, where we use either bilateral or parafibular incision for decompression.

Abdominal CS is an emergency when intra-abdominal pressure (IAP) measured with a urinary catheter exceeds 20 mm Hg. The method of choice is decompressive laparotomy (open abdomen). A complication of CS in children with supracondylar humerus fracture may be Volkman's contracture resulting from fibrotic forearm muscles after insufficient arterial perfusion. Typically, wrist flexion and claw-like posturing of the fingers develop.

If all muscle is lost and sensation cannot be expected to return, amputation and appropriate prosthetic measures are the method of choice.

PNEUMOTHORAX, PNEUMOMEDIASTINUM, PLEURAL EFFUSION (21)

Ledvina Tomáš, Horváth Teodor

PNEUMOTHORAX (PNO) means the presence of the air in the interpleural space. Etiology 1/ spontaneous 2/ trauma 3/ artificial (iatrogenic). This represents physiologically the disposal of the negative pressure and lung collapse (of different extent). They are distinguished 1/ closed 2/open 3/tension 4/both-sided PNO from mechanical points of view. Closed PNO arises by one-shot air penetration classified according to the degree as a/ coat, b/ partial, c/ complete. In the open PNO persists communication between interpleural and outer space. This creates mediastinal flutter during the breathing (i.e. mediastinal shifting dependent on the changes of intrathoracic pressure) and worsening of the venous return. In the tension PNO the defect represents a valve - during breathing in it is open and the air is gathered, during expiration is closed up. This increases volume of the air trapped in the interpleural space, the pressure is raised, mediastinum is shifted and the heart and contralateral lung are pressurised. Both sided PNO is most dangerous, fortunately rare.

The clinical features are substantial – dominates pressure, later thoracic pain and dyspnea. The aspection finds out asymmetrical excursions of the left and right thorax, alternatively area of the injury. The hematoma, crunching or grinding sounds or crepitus of the subcutaneous emphysema are found by the palpation. The percussion is sonorous on the afflicted side, the breathing sounds are weakened up to absent, the bronchophony is weakened. Small coat PNO is usually physically undiagnosed therefore there is a need to conclude it by patient history. Front projection thorax X-ray is the basis of the non-clinical diagnostics. One can search the details by much more sophisticated imaging methods (CT) they do not influence the treatment significantly.

They are two basic directions of the treatment – conservative and operative. Small asymptomatic PNO requires observation, more extensive 1/ thoracic tube 2/ VATS procedure. Essential therapeutic procedure is a water sealed thoracic tube. Complete reinflation of the lung parenchyma usually appears; in the case of lasting collapse follows active suction; if reinflation does not come the bronchoscopy is indicated to exclude/prove of the mucous and/or coagulum plug / obturation and their aspiration. VATS or open thoracotomy are used to remove the PNO source e.g. ruptured bleb, area with pulmonary emphysema or injury.

Thoracic tube is inserted through second or third intercostal space in the midclavicular line (Fig.1), in the midaxillary line at the level of anterior end of third or fourth rib (Fig.2) paying attention on aesthetic; or by back upper suprascapular access through first intercostal area. The tubing failure represents the indication for the operation. This represents usually wedge resection or segmentectomy of the afflicted region, further bullectomy, pleating of the bullous region and its

stitching together as well scarification of the visceral and parietal pleura, or visceral and parietal pleurectomy. In the borderline cases talc pleurodesis. Prevalent complications are coming in pathologically changed pulmonary parenchyma (e.g. chronic obstructive pulmonary disease or pulmonary fibrosis), in patients with internal comorbidities, nutritional handicaps and less exact management of postoperative thoracic tubing (drains).

As PNEUMOMEDIASTINUM (PNM) or mediastinal emphysema is signified the presence of the air in the mediastinum. It occurs as a complication of the PNO, as the symptom of central airways injury or oesophageal perforation (warning! mediastinitis). An isolated PNM originates by rupture of a pulmonary bulla adhering (post)inflammatory to the mediastinal pleura. It arises sometimes from the hyperinflation of the obturation cuff onto cannula of orotracheal intubation.

PNM is linked in most cases with subcutaneous emphysema initially on the neck afterthought in the face where can lead up to closure of the eyelids. Massive PNM pervades subcutaneous space of the trunk, scrotum and foreskin / or vulva and thighs. This is accompanied with anxiety (“what’s matter with me? “), rhinolalia, dyspnoea, retrosternal pain; and with peculiar crepitation by palpation. The PNM is confirmed by X-ray thorax imaging. For symptomatic treatment of massive PNM involvement are used a) 5-10 centimetres long subclavicular skin incisions with drain of the subcutaneous tissue and sterile dressing of the wound; b) in extreme cases jugular mediastinotomy. c) PNM as a result of PNO is largely treated by thoracic tubing. Causal PNM treatment is represented with the elimination of its cause.

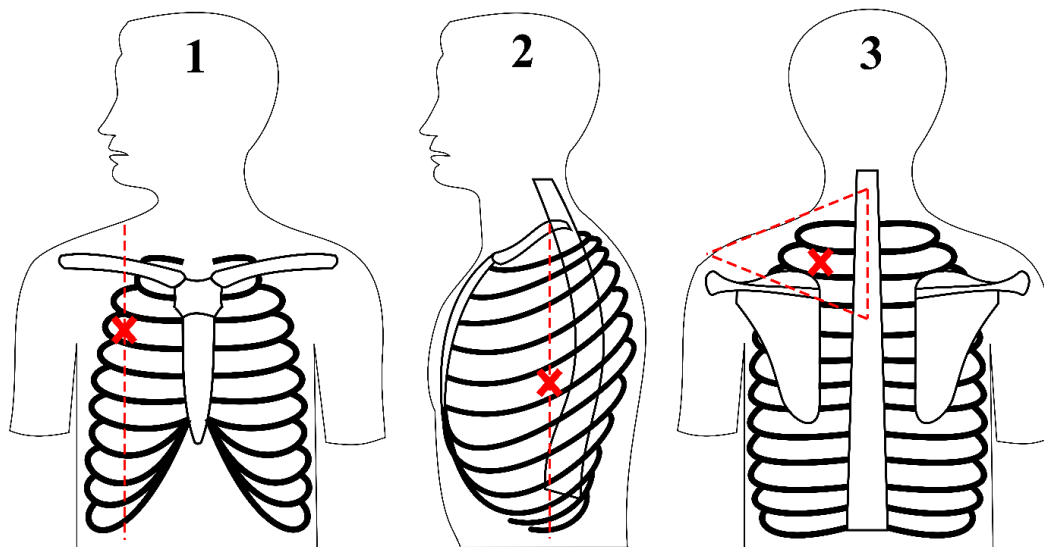
PLEURAL EFFUSION means the presence of the pathological fluid in the pleural cavity by its character 1/exsudate or 2/ transudate. The exsudate, abundant with protein, originates primarily from pleural damage – pleuritis, bronchopneumonia, malignancy (mesothelioma), by the induction with abdominal pathology, chylothorax, uraemia. The transudate, low in protein, arises from the imbalance of the secretion and the absorption of the pleural fluid from the extrapleural causes – congestion heart failure, liver cirrhosis, hypoproteinaemia, pulmonary embolism, nephrotic syndrome. They are clinically in the foreground dyspnea, cough, expectoration, pain, fever, shivering. The percussion is on the pleural effusion dull, the auscultation weakens up to absent, they could be present unstressed wet rhonchi, bronchophony is weaken.

Non-clinical accent consists onto 1/ LABORATORY examinations distinguishing inflammation (leucocytes, CRP), liver damage (ALT, AST), heart failure (NT-pro BNP), pulmonary embolization (D-dimers), hypoproteinaemia (total protein, albumin). There is a need of making a search first for all of these in the patient history and through physical examination. 2/ IMAGING The pleural

effusion, pneumonia, and congestive heart failure are commonly confirmed by Thorax X-ray, subphrenic abscess seldom, here is indicated ultrasonography. CT investigation of the thorax / trunk with contrast media most exactly defines amount of the effusion, status of the pulmonary parenchyma, intraabdominal pathology, and can estimate the character of the effusion. The kind of the effusion is defined by microbiological, cytological, and biochemical examination of the punctate. The symptomatic treatment is represented with evacuation pleural puncture or tubing. The causal treatment means to solve the primary disease. Extensive breadth of differential diagnosis and potential complications emerge from the nature of the underlying diseases.

COMMENTS AND CONTROVERSIES The thoracic tubing starts as a rule by underwater sealed drain; The injection needles inserted into the subcutaneous tissue of the pectoral area are not the solution of the PNM. In the case that pleural effusion is not evacuated in time, the patient is, besides other things, endangered with pleural empyema.

CHEST TUBE



- 1 Second or third intercostal space of the midclavicular line
- 2 The intercostal space of the midaxillary line located at the level of sternal attachment of the third rib
- 3 First intercostal space located in the middle of equilateral triangle formed by the spine of scapula, spinous processes of the vertebrae and anterior edge of trapezius muscle

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BURNS, ELECTRIC SHOCKS AND FROSTBITE (28)

Mager Radomír, Šín Petr, Dubovská Nikola, Hokynková Alica

Thermal injuries involve damage to the skin and subcutaneous structures due to exposure to extreme temperatures. These include burns, frostbite and electrical injuries.

Classification by mechanism of injury

1. **Contact burns:** caused by direct contact with a hot surface (e.g. solid body, hot liquids, hot air - flame, explosion).
2. **Electrical burns:** result from the passage of an electric current through the body, or indirectly from an electric arc (spark) or subsequent charring.
3. **Chemical burns (scalding):** caused by acids (coagulation necrosis), alkalis (liquefactive necrosis) or other chemicals.
4. **Radiation burns:** a specific type of burn caused by exposure to radiation.
5. **Frostbite:** cold-induced damage affecting the skin and subcutaneous tissues.

Classification by extent

The extent of burns is given as a percentage of the total body surface area (TBSA). For quick reference, **the nine percent rule (according to Wallace)** is used:

- Head and neck: 9 %.
- Front of torso: 18%
- Back of torso: 18 %
- Upper limbs: 9 % each
- Lower limbs: 18 % each
- Genitalia: 1 %

Breakdown by depth of disability

The burn has an evolution from the beginning of the healing process and the proper classification is important to choose the optimal treatment. Subsequently, burns can be classified into several groups:

- **First-degree burns:** cause redness (erythema) and pain, affecting only the epidermis.
- **Second-degree (II. a) burns:** superficial damage with blistering between the epidermis and dermis, usually heals spontaneously.
- **Second-degree (II. b) burns:** affecting the deeper layers of the dermis, creating multiple blisters and healing by epithelialization from hair follicles and sebaceous glands within 2-3 weeks. Surgical treatment may sometimes be required. Prolonged healing increases the risk of hypertrophic scars.

- **Third-degree burns:** cause full-thickness devitalization of the skin with necrosis, the skin is white or black in colour, without capillary refill. There is minimal or no pain, as nerve endings are destroyed. These burns do not heal spontaneously and require necrosis removal and skin grafting. Deeper tissues may be affected, which can lead to limited mobility, amputation or death.

The prognosis for a burn patient depends on the extent and depth of the burn and the quality of initial care. Maintaining an aseptic environment is crucial for healing. For **first and second-degree burns**, local cooling is indicated in the early phase, of greatest importance in the first 24-48 h with saline solution over oily tulle dressing or a cooling HydroTac cover. The optimal temperature is 4-8 °C with a maximum cooling surface of 5% TBSA (face, neck, hands, feet, genital area). In the presence of bullae, it is recommended to perforate them and leave them as a protective cover. Bullae larger than 6 cm should be removed with adequate pain management. For circumferential burns, particularly in the extremities, neck or chest, are at risk of neurovascular bundle compression requiring early skin relaxing incisions (escharotomy). **In third-degree burns**, surgical intervention is necessary in the form of necrectomy (sharp/chemical) followed by autotransplantation of a dermo-epidermal graft, usually from donor sites such as the thighs or buttocks.

Burn shock

Burn shock is a generalized response of the body to extensive burns. It results in fluid and protein leakage into the interstitial space, leading to hypovolaemia, oedema and activation of the adrenergic system, which causes vasoconstriction and reduces perfusion of peripheral tissues. Anaerobic metabolism in tissues can lead to multi-organ failure. Treatment includes fluid resuscitation, pain management, provision of breathing support and prevention of complications such as thromboembolism.

Electric shock (electrotrauma)

Electrical injuries occur either through direct passage of current through the body or indirectly via an electric arc. The point of entry and exit of the current plays an essential role in determining the extent of damage. Electric current can cause severe muscle contractions and ventricular fibrillation, which can lead to cardiac arrest. The electric arc, even without passing through the body, can cause deep burns that require surgical intervention (necrectomy, skin grafting).

Electrotrauma is monitored by ECG and determination of cardioenzymes, especially creatine phosphokinase (CK), to exclude myocardial necrosis. If muscle ischemia is suspected, fasciotomy and decompression incisions are necessary.

Frostbite

Frostbite is caused by exposure of tissues to low temperatures, often in combination with moisture. It primarily affects peripheral areas with poorer blood perfusion, such as the fingers, ears, nose and cheeks. The severity of frostbite is classified according to the depth of involvement:

- **First-degree frostbite:** manifested by redness, swelling and a tingling or burning sensation, without permanent sequelae.
- **Second-degree frostbite:** blisters form and the skin appears waxy white with reduced sensitivity.
- **Third-degree frostbite:** bluish-purple to grey skin with blood filled blistering and necrosis, treatment is prolonged and often associated with scarring.
- **Fourth-degree frostbite:** tissues become black, mummified and irreversibly damaged, usually requiring amputation.

Treatment of frostbite involves gradual warming but not rubbing, administration of analgesics and thrombolytics (e.g. heparin) to improve microcirculation. In severe cases, necrectomy and amputation are necessary.

A more detailed description of the comprehensive treatment of burns and burn shock can be found in Chapter ADDENDUM

FRACTURES – DIAGNOSIS, FIRST AID (6)

Hasara Roman

Fractures are defined as a disruption of the continuity and structural integrity of a bone due to trauma or disease. This disruption can be complete (a complete fracture) or, less frequently, incomplete (in fracture or subperiosteal fracture in children). It is important to note that a fracture is always associated with injury to the adjacent soft tissues and potentially the neurovascular structures and the extent of this injury dictates the treatment strategy. From an etiological perspective, we differentiate between traumatic fractures (the most common), pathological fractures (structural damage to bone tissue due to disease, typically fractures in the setting of bone metastases, multiple myeloma, osteogenesis imperfecta, etc., as well as fractures resulting from primary osteolytic bone tumours; this group also includes fractures in the context of bone infections, such as chronic osteomyelitis, and, rarely, tuberculosis or syphilis), and stress fractures (caused by chronic repetitive subthreshold mechanical overload).

From a morphological perspective, fractures are classified as diaphyseal fractures and fractures of the joint ends (proximal and distal), which are further subdivided into **extra-articular, partially articular, and complete intra-articular fractures**. Based on the relationship between the bone fragments, fractures can be classified as non-displaced and displaced, with a specific subtype being fracture-dislocations. The direction of displacement of bone fragments can be described as **ad latus** (to the side), **ad axim** (out of axis), **ad peripheriam** (rotational), or **ad longitudinem** (longitudinal) in length – shortening, **cum contractione**, or elongation, **cum distractione**). Based on the mechanism of injury, we classify fractures as compressive, distraction, impact, and bending fractures. Fractures are also categorised based on the fracture line, which can be transverse, oblique, spiral, with an interfragment, or comminuted. According to the integrity of the skin, fractures are classified as closed or open.

Diagnosis:

The foundation of any examination is the patient's history, with a focus on identifying the mechanism of the traumatic event. This is followed by a clinical examination, including careful palpation of the affected area and its surroundings. The leading symptom is pain, both spontaneous and provoked by pressure along the long axis of the bone or by attempting to move the injured limb. Other symptoms include swelling, hematoma, and limited passive and active mobility (**functio laesa**). These are the so-called "uncertain" signs of a fracture. The "certain" signs of a fracture include deformity, crepitus, abnormal movement, and visible bone in an open fracture wound.

A thorough clinical examination should also include a neurological assessment (sensory and motor function distal to the injury site), as well as an assessment of peripheral circulation (pulsation, capillary refill) to rule out injury to neurovascular bundles caused by the primary trauma or by secondary bone fragment movement during manipulation. It is essential to consider the possibility of additional associated injuries (e.g., pneumothorax or hemothorax with rib fractures, liver or spleen injury with caudal rib fractures, pelvic injuries with distal femoral fractures in traffic accidents, vertebral fractures following a fall onto extended legs, etc.).

The dominant imaging modality for diagnosing fractures is **X-ray** examination, which must include the entire bone from the proximal to the distal ends. The standard examination includes two orthogonal views (anteroposterior and lateral), with additional special projections (e.g., oblique views for the clavicle, Judet views for the acetabulum, and inlet-outlet views for the pelvis) in some cases. **CT scanning** with 2D and 3D reconstruction is particularly useful for intra-articular fractures, spinal and skull fractures, polytrauma protocols, or in cases of diagnostic uncertainty (e.g., suspected scaphoid fracture). Given its widespread availability, CT often replaces specialised projections and provides excellent information on fracture morphology, though its drawback is the higher radiation dose. **MRI** is not a standard modality for fracture diagnosis but provides valuable information about associated soft tissue injuries (e.g., rotator cuff injuries, knee ligament injuries, spinal cord injuries, or spinal ligament apparatus injuries). MRI is also used in the diagnosis of subchondral fractures, avascular necrosis, or osteomyelitis.

First Aid:

The basis of primary care for suspected fractures is immobilisation of the affected body part. This prevents pathological movement and secondary trauma to soft tissues, helps control bleeding, and provides analgesia. For grossly displaced fractures, approximate reduction is appropriate, usually performed by gently pulling the limb along its long axis, followed by immobilisation. Immobilisation should typically extend to include the proximal and distal joints adjacent to the suspected fracture.

Kramer splints, plastic splints, and especially vacuum splints are widely used in prehospital care.

For suspected diaphyseal femoral fractures, immobilisation with a special extension splint is possible, where continuous traction along the bone's axis leads to a partial reduction of the fracture, which is then retained by increased muscle tone in the surrounding muscle groups.

In suspected cervical spine injuries, it is essential to apply a cervical collar, ideally supplemented with **head blocks**. For suspected thoracolumbar spine fractures, the patient should be transported on a **spine board** or in a **vacuum mattress**, lying supine. In cases of suspected unstable pelvic fractures, the application of a **pelvic binder** should be considered. If splinting devices are unavailable, improvisation is possible, for example, by splinting the injured lower limb to the

uninjured limb. Splinting must be long enough, as comfortable as possible for the patient, and adequately padded to prevent pressure sores on soft tissues. Care must be taken to avoid overly tight splinting, which could compromise blood circulation. Gentle handling of the patient is imperative in all circumstances, minimising secondary trauma, reducing pain, and decreasing the risk of iatrogenic injury.

In cases of open fractures, sterile dressings should be applied even in the prehospital phase. If there is significant bleeding, haemostasis is required; however, the application of a tourniquet should be avoided in most cases, as compression dressings or pressure bandages are usually sufficient. Never attempt to push protruding bone back into the wound, as this would risk iatrogenic contamination of the wound and further soft tissue injury. Adequate analgesia, maintaining thermal comfort, and ensuring the patient is directed to the appropriate medical facility based on the type and extent of the suspected injury are essential.

FRACTURES CLASSIFICATION AND TYPES OF HEALING (32)

Tručka Robert

A fracture is defined as a disruption of bone continuity, complete or incomplete (infracture, subperiosteal fracture in children). Division by etiology: traumatic, fatigue, pathological.

Traumatic fractures

They arise by a direct or indirect mechanism. We distinguish torsional, bending, compressional, avulsion and shearing violence or their combinations.

Division by fracture line:

- Transverse
- Oblique
- Spiral
- Vertical (chiseled in cortical bone, impressive in spongy bone)
- Tangential (osteochondral)
- Avulsion - at the attachments of ligaments and tendons

According to the number of fragments, we divide fractures into two-, three-, four-fragmentary and comminuted. Sometimes, we encounter a segmental fracture in diaphyseal fractures, when the intermediate fragment is intact and longer than 6 cm.

Dislocation of fragments is evaluated according to position of peripheral fragment against the proximal one. Dislocations of proximal vertebra against the distal one are described only in luxation fractures of spine. We distinguish dislocations: ad latus (to side), ad longitudinem (to length, with distraction and contraction), ad axim (angular), ad peripheriim (rotational).

Stress fractures (fatigue fractures)

They are caused by overloading the skeleton during fatigue of muscle cuff, excessive and repeated straining of hypertrophic muscle cuff. A march fracture of 3rd metatarsus is typical, middle third of tibia or distal third of fibula may be affected in runners and dancers.

Pathological fractures

They arise on the ground of pathologically altered bones only by minor or atypical violence. The causes are osteoporosis in old people, bone cysts in younger people, however, the most common are metastatic processes in tumors of breast, thyroid gland, Grawitz tumor, prostate and others.

Classification of fractures

The classification is intended to inform the severity and prognosis of fracture and to guide the therapeutic strategy. Many different fracture classifications have been created. The most used

classification is that of AO Trauma (Arbeitsgemeinschaft für Osteosynthesefragen).

AO classification

It is based on X-ray and fractures are defined for common clinical practice by a four-digit code. The 5th number (subsegment) is supplementary and is intended for special evaluation.

The coding principle can be best demonstrated on long bone fractures.

The 1st code number determines anatomical area of fracture, the 2nd code number indicates injured segment of bone: 1. proximal part, 2. diaphysis, 3. distal part of bone.

The 3rd position of code indicates a character of fracture with letters A, B, C.

For diaphyseal fractures, these are:

A - simple two-fragment fractures,

B - three-fragment fractures with an interfragment,

C - multifragmentary (comminuted) fractures.

For intraarticular/metaphyseal fractures, these are:

A - extraarticular fractures (joint surface is not damaged),

B - partially intraarticular fractures (part of articular surface is related to diaphysis), called monocondylar fractures,

C - complete intraarticular fractures (articular fragments are not connected to diaphysis).

The 4th digit of code is the number 1-3, which expresses severity of bone injury.

For diaphyseal fractures, these are: A1 - spiral, A2 - oblique, A3 - transverse,

B1 spiral with interfragment, B2 bending with interfragment, B3 breaking of interfragment, C1 - spiral, C2 - segmental, C3 - comminuted.

The classification is more variable for epiphyseal fractures.

A more detailed division of type of fracture is in 5th place of code.

The skull, spine, pelvis, scapula, clavicle, patella and heel have specific divisions.

Open fractures (compound fractures)

Fracture lines directly communicate with skin cover injury, we solve healing of bone and soft tissues, as well as issue of bacterial contamination or manifest infection (antibiotics).

Classification of open fractures (Gustilo-Anderson, Tscherne)

Grade I: soft tissues, including skin, are perforated by sharp bone fragment from inside out, wound is less than 5 cm, smooth edges, mechanism of injury is mostly indirect rotational.

Grade II: skin and soft tissues are injured from outside in, violence is direct, wound is larger than 5 cm, surroundings usually bruised, muscles, larger vessels and nerves may be injured.

Grade III: high energy injuries with extensive devastating skin and soft tissues damage, often up to subtotal amputation, major vessels and nerve trunks may be interrupted, they have high degree of contamination, and the condition may be complicated by tissue ischemia distal to fracture. In the event that injured bone can be covered by soft tissues, we assess it as Grade IIIa, otherwise as Grade IIIb, if there is simultaneous damage to large vessels, it's Grade IIIc.

Types of bone healing

Primary bone healing (direct) represents healing "called without a gap", when osteons grow directly from one fragment to another, thanks to performed interfragmentary compression.

Secondary healing (indirect) is healing with the formation of a callus, and has 4 phases:

1. inflammatory - it lasts 3-5 days, hematoma is formed at the fracture site, which subsequently convert to richly vascularized fibrous tissue.
2. reparative - periosteal cells are transformed into chondroblasts forming cartilage, at the same time osteoclasts break down bone fragments, osteoblasts produce bone matrix and within 3 weeks a bone-cartilaginous soft callus is formed, bridging the fracture site.
3. formation of bony callus - osteoclasts resorb old bone, osteoblasts continue to produce new bone, which is subsequently mineralized.
4. remodeling - bone is adapted to ideal biomechanical load following the action of biomechanical forces associated with human movement.

Complications of bone healing:

Healing of a bone in a wrong position (malunion) - it occurs when fracture is not fully repositioned or as a result of unstable fixation.

Prolonged healing (delayed union) - healing time is longer than the expected healing of the given bone type (healing time extended up to twice).

Pseudoarthrosis - bone fusion does not occur even after twice as long.

- Vital pseudoarthrosis - arises as a result of insufficient immobilization (stabilization) of fracture, blood supply at fracture site is sufficient,
- Avital pseudoarthrosis - arises as a result of unfavorable biological conditions at fracture site (insufficient blood supply).

Necrosis of bone fragments.

Bone infection (Fracture Related Infection – FRI).

Infected pseudoarthrosis.

Refracture.

CONSERVATIVE TREATMENT OF FRACTURES, PLASTERING, BANDAGING AND TRACTION

TECHNIQUES (33)

Kašpar Michal

Conservative treatment of fractures, tendon and ligament injuries is one of the basic treatment methods performed in the surgical outpatient clinic.

The 3R principle

Repositioning - in dislocated fractures, using repositioning manoeuvres, return the bone position to anatomical conditions or to those close to them. **Retention** - immobilisation - stabilisation - using a plaster or cast bandage to allow the fracture to heal using ligamentous, then cartilaginous and then bony union for a sufficiently long period of time necessary to heal the fracture. **Rehabilitation** - after healing of the fracture, warming up of the adjacent joints, strengthening of weakened muscles, remodelling of the bone in the fracture area according to the load

Indications for conservative treatment:- non-dislocated fractures--fractures dislocated after repositioning but without tendency to redislocation or with tolerable dislocation—most pediatric fractures--patient in poor biological condition where the risk of surgery or anaesthesia significantly exceeds the benefit of surgery--vital indications—patient's disagreement with surgery--the patient is a partner in treatment - chooses the treatment procedure - must be thoroughly informed about his/her health status, recommended therapy and risks of treatment.

First R - Closed reposition

By tension and counter-tension, or subsequent bending, we try to compensate for dislocation ad axim (axial deviation), ad latus (lateral deviation), ad longitudinem (rotational deviation) or contraction (shortening). For each fracture location, we tolerate a certain threshold (degrees or millimetres) of dislocation at which functional changes have not yet occurred. In the area of intra-articular fractures, a displacement of up to 1 mm at the joint is tolerated, in the diaphyseal area up to a displacement of 2 cortices. For better cooperation and especially patient comfort, it is better to use local or short general anaesthesia. After repositioning, the fracture is stabilised.

Second R retention – immobilisation

Every fracture needs sufficient rest for a sufficiently long period of time for healing. Immobilisation allows secondary healing of the bone by the muscle. Immobilisation allows minimal movements at the fracture line, thus stimulating healing with the formation of first soft/fibrous and cartilaginous, then bony union until this is subsequently visible on X-ray. The rule is that both joints adjacent to the fracture should be immobilised - i.e. the joint above and below the fracture ...with a few exceptions:

isolated distal radius fracture (cast ends below the elbow), tibial fracture (cast ends below the knee), patella fracture. We immobilise the joints in the middle position when the tension on the capsule, ligaments and tendons is lowest. The following methods can be used for retention of fragments: plaster bandage x resin bandage x for healing and RHB orthosis.

Third R -Rehabilitation

It is an attempt to return to the functional state before the injury, to warm up stiff joints, to strengthen atrophied muscles, to heal broken bones by means of a regulated load. The basis of medical rehabilitation is kinesiotherapy, i.e. therapeutic physical education (LTV). The outpatient patient has the possibility to practice LTV on his/her own, if his/her physical condition allows it, or he/she is referred to outpatient controlled rehabilitation. The exercise starts from the first days of immobilisation: isometric muscle tensioning, practising non-mobile joints of the limb.

Physical therapy is used as an adjunct to kinesiotherapy. Water therapy, ultrasound, magnets, and IR radiation are used.

Notes on plastering

-is a basic surgical technique used for immobilisation of the musculoskeletal system – either as a conservative treatment of fractures, therapy of distension and partial ruptures of ligaments and joint capsule injuries or immobilisation in the treatment of inflammatory soft tissue affections or bone tumours. It is a medical procedure - either performed by the doctor himself or supervised by the doctor, who is forensically responsible for it.

Indications for plaster bandage

Immobilisation: soft tissue injuries, fractures, inflammations, tumours, plastics, healing of fused fractures, sutured tendons

Plastering rules

- * Maintain physiological position in joints and relaxed muscle tone.
- * When special repositioning positions are necessary - flexion, duction - to maintain the anatomical position on the fracture - these are repositioned to the physiological position within 14 days
- * Primary treatment- we assume swelling and hematoma on the fracture and surrounding area. There is a risk of compartment syndrome when clamped with a tight circular plaster bandage, therefore always only a plaster splint or cut plaster bandage during primary treatment
- * Secondary treatment- 7-10 days after the post-injury swelling has subsided: recasting of the splint, recasting to a complete plaster bandage
- * Strengthening in areas of greater stress *Modelling according to the shape of the limb
- * Let dry sufficiently

Practice – procedure

Always use a backing material - stocking on dry skin, cotton padding on it - areas of the ends of the plaster bandage, absence against the skin where decubitus ulcers are a risk, crepe paper to achieve uniformity of the backing materials, remove the plaster bandage from the original packaging, immerse it in water about 20°C warm for 3 seconds, then squeeze the excess water out of the bandage. Gradually unwrap the bandage and apply it to the desired part of the body. The turns overlap half the width of the bandage. Cover the edges of the plaster bandage with the overhanging part of the underlying dressing and smooth it so that it does not irritate or hurt the skin. The bandage is then immediately moulded into the desired shape. The maximum time for modelling is 2-4 minutes. Afterwards, allow to set for 4-7 minutes. Mechanical resistance of the bandage is after about 30 minutes. Total drying time approx. 24 hours.

Patient instruction

The patient must necessarily be informed about the principles and objectives of the treatment, and possible complications. Immediate follow-up is necessary. When there is significant pain and pressure in the fixed limb. Peripheral oedema, peripheral discoloration, Impaired mobility, paresthesia. When dealing with a patient with a cast, the ... The cast patient is always right.

Complications

Too loose fixation - loss of repositioning, restlessness at the fracture - impaired healing. Too tight fixation: - Pain, Decubitus, Peripheral nerve deficit, Compartment syndrome - Ischemic contracture. Complications are the responsibility of the attending physician - the plaster dressing must always be carefully handled and the patient instructed.

Traction

A special type of continuous repositioning is elastic or skeletal traction. Nowadays, it is only allowed to „bridge“the time period between the admission and surgery of a patient with a fracture of the diaphysis of the long bones - i.e. femur or tibia, if for some reason, the operation cannot be performed urgently / necessary internal preparation of the patient, there is no adequate team, other urgent operations are underway, no suitable osteosynthetic material is available /. It consists either in wrapping the traction behind the tibia with elastic bandages or in the percutaneous insertion of the K-wire into the distal femur in the area of the condyles, into the tuberosity of the tibia, or into the calcaneus. A weight (weighing 1/10 of the patient's weight) is tied to the wire by means of a metal horseshoe, hanging over a pulley, e.g., a Braune plate, thus causing continuous traction in the long axis

of the limb. This results in immobilisation, preventing shortening at the fracture by muscle tension, rotation of the limb, and traction, which has an analgesic effect. After subsequent preparation, an elective osteosynthesis is performed – with nail, plate, etc.

OPERATIVE TREATMENT OF FRACTURES, PRINCIPLES OF OSTEOSYNTHESIS (30)

Musilová Zuzana, Vojtaník Pavol

It complements conservative therapy and is indicated where the conservative approach would not suffice. Strict adherence to asepsis is essential. Osteosynthesis (OS) can be performed within 6-8 hours after the trauma, or 4-14 days post-trauma once the swelling has subsided or depending on the condition of the soft tissues – the "wrinkle test" (skin wrinkling upon swelling resolution).

The goal is to restore full function, which is why there is an effort to achieve stable fixation (with early rehabilitation and functional treatment) to avoid the need for further interventions. This is achieved through stable osteosynthesis, according to the fundamental principles of the AO group (Arbeitsgemeinschaft für Osteosynthesefragen) – Switzerland, Davos, 1958. This involves anatomical exact reduction (ORIF = open reduction internal fixation versus CRIF = closed reduction internal fixation), a gentle surgical technique (preserving soft tissues, vascular supply, and bone vitality), stable retention (fixation) of fragments, and early rehabilitation. The osteosynthetic material must be strong, non-allergenic, and shapeable (e.g., titanium, steel, etc.).

Indications:

- Absolute – associated with injuries to the neurovascular bundle, unstable and significantly displaced fractures, open fractures (Tscherne gr. II and III), fractures with interposition of soft tissues
- Relative – polytrauma, intra-articular fractures

The principles of bone healing are described in question no. 32, direct (primary, direct) and indirect (secondary, indirect).

- Direct healing requires interfragmentary compression of vital fragments. Surgical techniques that ensure compression and mechanical stability are referred to as methods of "absolute stability" (osteosynthesis with a lag screw, compression plate, or cerclage compression wiring, used for example in intra-articular fractures).
- Indirect healing through callus formation, similar to cast fixation, is primarily used for fractures of the metaphysis and diaphysis (intramedullary nailing, angular stable LCP plates, or external fixation).

Types of osteosynthesis (OS) based on the placement of OS material:

- Intraosseous placement, where nails, wires, or rods are introduced into the medullary cavity of the bone.

- Extraosseous placement, where the OS material is placed on the surface of the bone, beneath the soft tissues. However, in the case of external fixation (EF), the main metal component is left outside the soft tissues.

Types of osteosynthesis (OS) based on technique:

- Transfixation with wires (Kirschner wire = K-wire, KW)

Wires alone do not fulfill the principles of stable osteosynthesis (OS); additional fixation (internal fixation, external fixation) is necessary. Typically used in paediatric fractures (does not damage cartilage), and in adults for epiphyseal/metaphyseal fractures – distal humerus, wrist, and fractures of small bones – metacarpals, metatarsals, and phalanges. It is also used for intramedullary stabilization of diaphyseal fractures (not in comminuted fractures, where there is a risk of wire migration).

Complications may include damage to the neurovascular bundle during insertion, wire migration, or breakage.

- Cerclage compression wiring

Cerclage alone (compression) has low strength and needs to be supplemented with 2 parallel K-wires (prevents rotation and ensures anatomical alignment). For example, used in fractures of the patella, olecranon, lateral and medial malleolus, and acromioclavicular dislocations.

- Osteosynthesis with Screws

We use two basic types of screws. **Cortical screws** are used in (meta)diaphysis, anchored in the cortical layer, and have a denser thread. **Cancellous bone screws** are drilled into the metaphysis and epiphysis, have a coarser thread, and hold well in cancellous bone, often combined with a washer (for osteoporotic bone). Screws are often combined with other forms of osteosynthesis (plates, K-wires, etc.).

- Specific types of screws:

Lag screw provides interfragmentary compression and absolute stability and is placed through the fracture line.

Plate screw fixes the plate and does not pass through the fracture line.

Cannulated screw (hollow), e.g., Herbert screw, used for osteosynthesis of scaphoid fractures, is inserted over a K-wire.

Locking head screws have a fine thread on the head that tightly engages with the plate, maintaining the set angle.

- Osteosynthesis with a Plate

It requires good condition of the soft tissues. Based on the technique, we use four types of plates:

Compression plate increases the pressure between bone fragments. Compression is achieved using a compression device (compression tool) or special eccentrically designed oval holes (DCP – Dynamic

Compression Plate – the oval hole for the screw head and its inclination allows compression of the fracture during tightening). This ensures absolute stability, but the plate is placed directly on the bone (risk of periosteal compression). Used, for example, in oblique or transverse diaphyseal fractures.

Neutralization plate (protection plate) prevents rotational, bending, and shearing forces and is supplemented by osteosynthesis with a tension screw. Used, for example, in lateral malleolus fractures.

Buttress plate (antiglide)– used in meta-epiphyseal fractures, where the plate provides mechanical support. For example, in proximal tibia or proximal humerus fractures.

Bridging plate is used to span comminuted zones, particularly in cases of complex fractures. LCP – Locking Compression Plate, ensures relative stability.

- Intramedullary Osteosynthesis

Antegrade nailing (nails are inserted intramedullary from the center to the periphery) or retrograde nailing. This technique is gentle on the periosteum and bone's vascular supply, as well as on soft tissues, providing relative stability, but complications such as pseudoarthrosis (bone nonunion, „false joint“) can occur. The nail is secured proximally and distally with at least two screws. For example, used in fractures of the meta/diaphysis of the femur, tibia, and humerus.

This group also includes other types of nails beyond the classic ones, such as:

Hackethail nails (used for fractures in the diaphysis of the humerus, with several strong K-wires inserted into the cavity),

Ender rods (several rods inserted into the cavity),

Kuntscher nail, or

Special nails used for pertrochanteric femur fractures (PFN – Proximal Femoral Nail).

- External Fixation

This involves stabilizing bone fragments with an external structure, either temporarily or permanently, providing relative stability. Fixation to the bone is most commonly achieved with Schanz screws (K-wires can also be used, among others). It is utilized in acute traumatology (for open fractures, soft tissue injuries, compartment syndrome, replantation, polytrauma, comminuted fractures, pelvic trauma), reconstructive traumatology (infected fractures, pseudoarthrosis, limb lengthening of lower extremities with shortening), and orthopaedic traumatology (leg length discrepancies, arthrodesis).

COMPLICATIONS OF FRACTURE TREATMENT AND RELATED CONSEQUENCES (31)

Musil Tomáš

1. Fat embolism
2. Early complications
3. Phlebothrombosis and thromboembolism
4. Technical errors and failure of osteosynthesis
5. Bone healing disorders
 - Nonunion - failure to heal at 8 months after surgery
 - Rhizome - fracture line filled with connective tissue
6. Healing in the wrong position - malunion
7. Post-traumatic arthrosis
8. Sudeck's algodystrophy
9. Paraarticular (central) ossification

1. Fat Embolism

Fat embolism is characterised by the penetration of **fat droplets** into the pulmonary circulation and the release of **free fatty acids** into the plasma. It is most commonly associated with **long bone fractures** and typically occurs within **48 hours** of the initial trauma.

Pathophysiology and Clinical Features:

- Fat embolism causes damage to the microcirculation, particularly affecting the **lungs, central nervous system (CNS), and skin**, resulting in a constellation of symptoms:
- **Pulmonary system:** Dyspnoea, tachypnoea, and haemoptysis.
- **CNS:** Neurological impairment ranging from confusion to coma.
- **Skin:** Petechial rash and exudative changes observed on the ocular fundus.

Diagnosis:

- **Imaging** - Chest X-ray: Demonstrates **bilateral pulmonary infiltrates**.
- **Laboratory Findings:** Full blood count (FBC): Indicates **thrombocytopenia**.

Treatment:

Treatment of fat embolism syndrome involves supportive care and pharmacological interventions:

- **Supportive measures:**
 - Intravenous fluid administration.
 - Oxygen therapy to address hypoxaemia.
 - Analgesics to manage pain.

- **Pharmacological therapy:**
 - **Low molecular weight heparin (LMWH)** to mitigate coagulation-related complications.
 - **Intravenous Lipostabil** to stabilise fat particles and reduce inflammatory responses.

2. Early Complications

1. Wound Infection

Superficial: Involves the skin and subcutaneous tissue.

Deep (subfascial): Extends to the muscle and fascia.

Superficial Infection

- **Definition:** Affects the skin and subcutaneous tissue, typically occurring within 30 days of surgery.
- **Clinical Features:**
 - Purulent discharge from the wound.
 - Local signs of infection, including redness and swelling.
 - Positive microbial culture.

Deep Infection

- **Definition:** Involves the muscle and fascia and may occur up to one year post-operatively, particularly in the presence of an implant.
- **Clinical Features:**
 - Purulent discharge from the wound.
 - Wound dehiscence.
 - Possible formation of an abscess.
- **Treatment:**
 - Antibiotic therapy (preferably targeted based on sensitivity testing).
 - Local treatment of the lesion, including drainage, lavage, and, if necessary, implant removal.

2. Wound Dehiscence

- **Superficial:** Involves only the skin and subcutaneous tissue, with the fascia remaining intact.
 - **Treatment:**

If no infection is present, resuture the wound.

If infection is present: manage as per wound infection guidelines.
- **Complete (Fascial):** Involves disruption of the fascia.
 - **Treatment:** Treat in the same manner as a deep infection.

3. Collection in the Wound

Types:

- **Seroma:** Accumulation of serous fluid.
- **Haematoma:** Collection of blood.

3. Phlebothrombosis and Thromboembolism

Phlebothrombosis refers to the **occlusion of the venous system** by a blood clot, most commonly affecting the **deep venous system of the tibia**. It frequently arises as a consequence of **immobilisation**, with potential complications involving the **iliac vein** or **pulmonary circulation**.

Clinical Presentation

- Tenderness and swelling of the tibia.
- Positive **Homans sign** and **plantar signs**.
- Evidence of collateral venous filling.

Diagnostics

- Elevated **D-dimer levels**.
- Imaging studies: **Doppler ultrasound**, **Contrast phlebography**, if necessary, **Magnetic resonance (MR) phlebography**.

Treatment

- Application of an **elastic bandage**.
- Administration of **Low Molecular Weight Heparin (LMWH)**.
- Implementation of **rehabilitation** measures.

4. Technical Errors and Osteosynthesis Failures

Causes

- **Inappropriate choice of osteosynthesis method:** For example, using a **dynamic hip screw (DHS)** instead of a **proximal femoral nail (PFN)** in an unstable pertrochanteric fracture.
- **Incorrect implant selection:** Choosing a **standard splint** rather than an **angle-stable (lockable) splint**.
- Misjudging the requirement for **absolute** versus **relative stability** depends on the fracture type and healing dynamics.
- **Failure to adhere to biomechanical principles:** Neglecting these principles can result in **osteosynthesis failure**, such as a loose implant or failure of fracture fixation.

5. Bone Healing Disorders

Healing Timeframes

- **Standard healing:** Typically occurs within **3–4 months** following osteosynthesis.
- **Prolonged healing:** Extends to **6–8 months** post-osteosynthesis, often referred to as **nonunion**.
- **Delayed union (rhizome):** Defined as the absence of clinical or radiographic evidence of bone healing after **8 months**.

- Types of Rhizome

- **Vital:** Capable of **spontaneous healing** following stable osteosynthesis.
- **Avital:** Incapable of spontaneous healing, requiring **stable osteosynthesis** combined with **spongioplasty**.

- Vital Nonunions

Hypertrophic (elephant foot): Can bear some degree of load.

Oligotrophic: Limited regenerative potential.

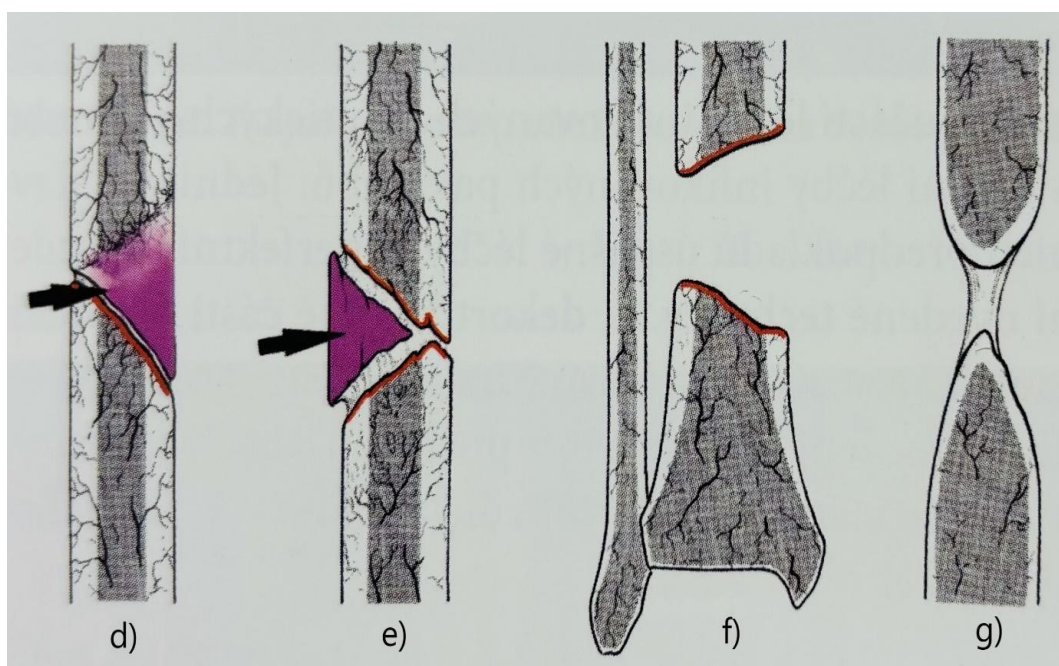
Atrophic: Lack of cellular activity with minimal or no callus formation.

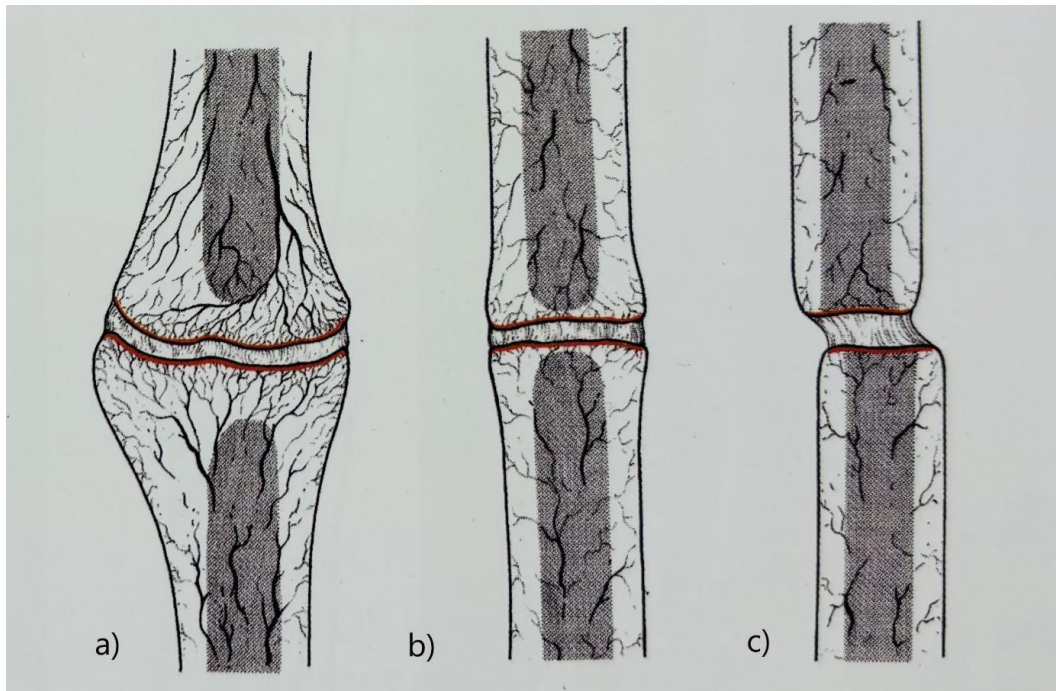
- Avital Nonunions

Devitalised butterfly fragment: Occurs when the intermediate fragment has lost vascularity.

Defective hilum: Results after removal of necrotic bone fragments.

Atrophic: Represents the final stage of all avital nonunions or subluxations, characterised by a complete absence of biological activity.





6. Healing in the Wrong Position – Malunion

Malunion refers to **post-traumatic deformity** or **misalignment (shunt)** that occurs during the healing process of a fracture.

Causes

- **Inadequate repositioning:** Occurs due to incomplete or incorrect alignment, particularly in cases of **multifragmentary** or **comminuted fractures**, where precise repositioning may be challenging or impossible.
- **Insufficient stability of osteosynthesis:** Results from failing to account for the **mechanical forces** acting at the fracture site, leading to instability and subsequent malalignment.
- **Inadequate follow-up or inappropriate rehabilitation:** Insufficient monitoring or poorly planned rehabilitation can contribute to suboptimal alignment during the healing process.

7. Post-Traumatic Arthrosis

Post-traumatic arthrosis arises due to **inadequate repositioning** and **insufficient fixation** of fracture fragments within the articular surface area.

Clinical Features

- **Progressive limitation of joint mobility.**
- **Chronic joint pain.**
- **Deformity** of the affected joint.

Treatment Principles

- Exact repositioning of fracture fragments is essential.

- Osteosynthesis should be performed adhering to the principle of **absolute stability** to prevent joint degeneration and preserve functionality.

8. Sudeck's Algodystrophy

Sudeck's algodystrophy is characterised by **patchy osteoporosis** in the acral parts of the affected limb, accompanied by **progressive impairment of limb function**.

Clinical Stages (The condition typically progresses through three stages.)

- **Inflammatory Stage** (0–3 months):
Skin: Warm, red, sweaty, and swollen.
Muscles: Evidence of atrophy, with pain on any movement.
Radiographic findings: Skeletal porosis with a "pencil-drawn" appearance of the bone contour.
- **Cyanotic Stage** (3–4 months):
Skin: Cyanotic and cold with associated hair loss.
Muscles and subcutaneous tissue: Ongoing atrophy.
Radiographic findings: Spotted porosity.
- **Contracture Stage** (beyond 3 months):
Development of **contractures** and **irreversible muscle atrophy**, resulting in significant functional limitations.
Pain typically subsides during this stage.

Prevention

- **Accurate repositioning** of fractures.
- **Proper immobilisation** techniques.
- **Appropriate rehabilitation** to ensure restoration of limb function.

9. Paraarticular Ossification

Paraarticular ossification refers to the abnormal formation of bone around a joint.

Causes

- It often occurs as a consequence of **severe trauma** or **repeated repositioning** of the joint.
- It may also result from **aggressive rehabilitation techniques**.

Common Sites - Most frequently localised around the **elbow** and **hip joints**.

Clinical Features - **Joint pain** and **Limitation of mobility** due to ossification obstructing normal joint movement.

POLYTRAUMA, POLYTRAUMA MANAGEMENT (29)

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Polytrauma is a simultaneous injury to at least two body systems, at least one of which compromises basic life functions. By body systems we mean the chest, abdomen, musculoskeletal system, head plus neck. This condition is the most common cause of death in patients under 45 years of age. Examples of the mechanism of polytrauma are typically car accidents, falls from heights, work accidents and acts of violence. The incidence of death associated with polytrauma is 60-80/100,000 population.

In polytrauma, we have observed a **trimodal distribution of deaths**, which can be divided into three phases. In the first immediate phase, 50% of patients die within one hour of the injury, death occurs mainly due to devastating injury to the brain, brain stem, spinal cord, heart and aorta. In the second, or early phase, up to 30% of patients die, with death occurring as a result of the development of shock or as a consequence of cavity injury or intracranial haemorrhage. The middle phase lasts between one and three hours after the onset of the injury. The last phase of death occurs at a latency of 3-4 weeks after the injury and is manifested by the general decompensation of the patient's condition, the development of infectious complications in the form of septic state and multi-organ failure.

In the case of injuries to two or more organ systems that do not directly threaten the patient's life, we speak of the so-called **associated injury**.

Pathophysiology

The hemorrhagic shock that develops in the acute loss of about 25% of the total blood volume, resulting in central circulation, hypotension, tachycardia, and oliguria, threatens the life of a polytraumatized patient immediately after trauma. This condition leads to hypoperfusion and organ hypoxia. There is also tissue lactate production and the development of metabolic acidosis, which reduces myocardial contractility and leads to a reduction in cardiac output. Furthermore, metabolic acidosis has a negative effect on the coagulation cascade, causing further blood loss and exacerbating organ hypoxia. The loss of circulating volume leads to hypothermia. When the core body temperature falls below 36°C, coagulopathy is exacerbated, thrombin synthesis is reduced and its degradation is accelerated. This phenomenon causes further blood loss and creates a '**vicious circle**', also known as the lethal triad, a condition in which hypothermia, coagulopathy and metabolic acidosis potentiate each other.

A large number of inflammatory mediators released from the injured tissue due to trauma, e.g. interleukins 1, 2, 6, TNF-alpha, proteases and PAF, activate thrombocytes and macrophages. The release of tissue factor activates the coagulation cascade with the formation of microthrombi, which causes reduced tissue perfusion due to microthrombotization. Cytokines and tissue factors cause vasodilatation, increasing capillary permeability, which ultimately leads to fluid transfer to the third

compartment and the development of edema. We use the name **Systemic Inflammatory Response Syndrome** (SIRS) to describe this process, which further adversely affects tissue blood flow and creates another barrier to oxygen transport to the tissues. This syndrome, together with haemorrhagic shock, can, depending on its severity, result in **Multiple Organ Dysfunction Syndrome** (MODS), a condition in which the body is unable to maintain homeostasis without therapeutic intervention and the function of internal organs is impaired. Typical examples are disseminated intravascular coagulopathy (DIC), respiratory failure (ARDS), acute renal failure, etc. When multiorgan dysfunction escalates, **multiple organ failure** (MOF) occurs.

If the patient survives the acute phase of SIRS, which develops within hours of the trauma, a compensatory counter-response to inflammatory mediators occurs within days, leading to activation of anti-inflammatory substances (e.g. interleukin 4, 10), cortisol and adenosine secretion. This phenomenon is called the **Compensatory Antiinflammatory Response Syndrome** (CARS). The goal of these responses is to control inflammatory processes. When SIRS predominates, the aforementioned multi-organ failure occurs. When CARS predominates, the body's immune responses are suppressed and sepsis occurs.

Diagnostics

The initial examination and determination of the condition of the polytrauma patient already takes place during pre-hospital treatment. When the paramedics perform the so-called triage of the patient, which consists in identifying the basic life-threatening conditions of the patient. In the case of triage positivity (the patient meets the positive criteria within the set triage system), the patient is primarily transported by the ambulance service for further care to the trauma centre and not to the nearest medical facility that is not equipped to provide comprehensive medical care for the polytrauma patient.

We use a number of scoring systems to assess the severity of the trauma and the patient's vital signs, such as the AIS (Abbreviated Injury Scale), TRISS (Trauma and Injury Severity Score), GCS (Glasgow Coma Scale), etc., which aim to simplify the management of the patient's care and decision-making about further therapy.

Once the patient is transported to the trauma centre, initial assessment and treatment in the emergency department takes place. As a standard, the patient is treated according to the ATLS (Advanced Trauma Life Support) protocol, a system of standardised procedures for the diagnosis and care of traumatised patients. ATLS treatment is carried out in 2 steps, i.e. primary and secondary assessment of the patient's condition.

The primary assessment follows the **ABCDE system**, as does first aid: (A) checking and securing the airway, (B) ensuring adequate ventilation of the patient, examining by palpation and listening, (C) assessing circulation, stopping bleeding and replacing blood loss, (D) assessing neurological status,

(E) fully exposing the patient, looking for hidden injuries. The primary assessment includes immediate treatment of identified life-threatening injuries. If these are not resolved during the primary assessment, then the patient is urgently transported to the operating room. Otherwise, a secondary assessment can be performed.

Secondary assessment includes medical history taking (allergies, chronic medication, illnesses monitored, last meal, events prior to injury), laboratory and detailed radiological examinations. Still as part of the primary evaluation, when the patient is circulatory unstable and abdominal trauma is suspected, we use FAST (Focused Assessment with Sonography for Trauma) ultrasound to look for the presence of free fluid primarily in the areas around the liver, kidneys, spleen, and small pelvis. In a state of circulatory stability, or if the patient responds positively to the volume challenge, then a CT scan is indicated, either targeted to a specific trauma area or in full-body mode. This provides comprehensive information about the cavity injury and axial skeletal fractures. Then, depending on the clinical condition, other targeted X-ray examinations are added, most often focused on the axial skeleton of the limbs.

Therapies

The secondary examination also includes the determination of a treatment plan, which must take into account the clinical condition of the patient. In patients with polytrauma, we follow damage control procedures as part of primary care.

Damage Control Resuscitation (DCR), are sequential steps that mitigate shock, stop non-surgical bleeding, optimize coagulation and reduce the effects of the lethal triad. Throughout the procedure, efforts are made to circulatory and ventilatory stabilize the patient immediately from admission to the emergency department, during the investigation, and before and after surgical procedures. In DCR, we aim to actively warm the patient, correct metabolic acidosis and hypothermia. The main goal is to maintain adequate organ perfusion and to maintain homeostatic potential.

In the surgical treatment of polytraumatized patients, we use the concept of **Damage Control Surgery (DCS)** in the first phase, which are urgent, life-saving operations to stop surgical bleeding, minimize contamination of the abdominal cavity from the perforated gastrointestinal tract, and prevent further damage. During the surgery, volume resuscitation is performed simultaneously.

Another protocol is **Damage Control Orthopaedics (DCO)**, by which we mean a minimally invasive surgical procedure to treat the injured skeletal system in the initial phase of treatment of a polytrauma patient. DCO involves repositioning and temporary stabilization of long bone and pelvic ring fractures, most commonly with an external fixator. The stabilization of fractures has the effect of stopping bleeding from the fractures, reducing the risk of fat embolism and also, last but not least, an analgesic

effect for the patient. In the case of a devastating limb injury, primary amputation procedures are not infrequently chosen.

Knowledge of the pathophysiological processes that take place during trauma is very important for therapeutic management.

In the first, urgent phase, which follows pre-hospital care, acute diagnostics and life-saving procedures such as volumotherapy, hypothermia prevention, etc. are performed. This phase lasts up to 3 hours from the onset of the injury. The second, stabilization phase, lasting from 3 hours to 3 days after the injury, is the time of limited operations in the DCS mode. After this stabilization phase, the intensive care phase occurs, during which the patient is monitored in the ICU, where vital signs continue to stabilize and symptoms of chronic diseases are compensated for. In the first 5 days after the injury, SIRS is going on massively, so every effort is made to minimize surgical procedures during this period. The recovery phase, the interval between days 5-10, is also referred to as the "window of opportunity". During this period, the ongoing SIRS balances with the concurrent CARS, so it is a good time to perform osteosyntheses. After this phase, CARS predominates between days 10 and 20, and the patient is immunosuppressed and therefore susceptible to infectious complications and sepsis. Therefore, operations are not recommended at this time. The last phase is a period of recovery and rehabilitation, during which passive and active physiotherapy is performed to prevent decubitus, hypostatic pneumonia and restore the patient's mobility.

Conclusion

The prognosis of the patient depends on the type of injury and his biological reserves. Rapid transport of a polytraumatized patient to a trauma centre and qualified, comprehensive medical care realistically improves his chances of survival.

