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STUDENT´S GUIDE TO STATE EXAMINATION QUESTIONS FROM THE SURGERY

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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:

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CONTENT

SPECIAL SURGERY

NEUROSURGERY

NEURAL TUBE DEFECTS (10)

Barák Martin, Brichtová Eva

Congenital nervous system defects can involve various abnormalities in the brain, spinal cord, spine, or skull. These defects often occur in combination or may be part of complex genetic syndromes. Their development is associated with various stages of embryonic development and can be influenced by external and internal factors, with some having a hereditary basis. The most severe brain development disorders often result in miscarriage or death of the child shortly after birth. Some of these defects can be diagnosed prenatally, allowing the mother to make informed decisions regarding the continuation of the pregnancy.

The most common congenital nervous system defects requiring neurosurgical intervention include:

- **Spinal dysraphism**
- **Craniosynostosis**

Spinal dysraphisms encompass a wide spectrum of congenital disorders characterised by incomplete neural tube closure, from which the spinal cord later develops. They most commonly manifest as an absence of the vertebral arch or other tissues surrounding the spinal cord. These defects are called “spina bifida” and can lead to partial or complete neurological impairments below the lesion level. They most frequently occur in the lumbosacral region and develop between the 14th and 40th day of gestation. The causes of these defects are not fully understood, but risk factors include folic acid deficiency, exposure to teratogens during pregnancy (e.g., antiepileptic drugs), substance abuse, obesity, and maternal diabetes.

Classification of spinal dysraphism

Open Spina Bifida (Spina Bifida Aperta, Cystica): Defects in which the skin covering is very thin or absent, allowing direct contact of the spinal cord with the external environment. This category includes:

- **Myelomeningocele:** A defect of the vertebral arch with herniation of the spinal cord and nerve roots. Neurological impairment is common, often associated with hydrocephalus and Chiari II malformation—high risk of infection and cerebrospinal fluid hypotension.
- **Meningocele:** A defect of the vertebral arch with protrusion of the meninges without neural structures. Neurological impairment is milder with a better prognosis.

Hidden Spina Bifida (Spina Bifida Occulta): Defects of the vertebral arch where the skin covering is intact, but cutaneous stigmata are often present at the lesion site. These defects are often diagnosed later in life. This category includes:

- **Dermal Sinus (skin dimpling with communication to nervous tissue)**

- **Tethered Cord Syndrome**
- **Diastematomyelia (spinal cord duplication)**
- **Lipomyelomeningocele**

Prenatal diagnostic techniques include amniocentesis, serum alpha-fetoprotein level measurement, ultrasound examination, and prenatal MRI (contraindicated in the first trimester). **Postnatal diagnostics** involve clinical examination, ultrasound through the fontanelle (to rule out associated hydrocephalus), spinal MRI, and electrophysiological testing.

Treatment:

- **Open Spina Bifida (Spina Bifida Aperta):** Treatment is primarily surgical, aiming to close the defect by reconstructing the spinal meninges and skin. In cases of extensive defects, collaboration with a plastic surgeon is required. Surgery should be performed within 24 hours after birth.
- **Hidden Spina Bifida (Spina Bifida Occulta):** Surgical intervention is indicated in clinical symptoms or as a preventive measure. The type of surgery depends on the specific defect, and intraoperative monitoring of spinal cord and nerve root function using electrophysiological methods is necessary.

Craniosynostosis

The cranial vault consists of flat bones joined by cranial sutures, which prevent premature skull fusion. These sutures usually fuse in adulthood, except for the metopic suture, which ossifies around 18 months. Craniosynostosis arises from the premature fusion of one or more cranial sutures, leading to abnormal head shape and potential impact on brain development.

Classification according to the affected cranial suture:

- **Sagittal suture** – scaphocephaly, dolichocephaly (the most common type)
- **Coronal suture** – brachycephaly, anterior plagiocephaly
- **Metopic suture** – trigonocephaly
- **Lambdoid suture** – posterior plagiocephaly (rare)
- **Multiple craniosynostosis** – oxycephaly
- **Syndromic craniosynostosis** – associated with other congenital anomalies (e.g., syndactyly, polydactyly), such as Apert syndrome or Crouzon syndrome

The diagnosis is based on clinical examination (typical shape deformity, palpable bony ridge along the course of the ossified suture, and possibly facial deformities such as hypertelorism or maxillary hypoplasia). The anthropometric examination is primarily used to compare preoperative and

postoperative conditions. It is performed either with a craniometer or an optical 3D scanner. Prematurely fused sutures are well visualised by X-ray and CT scans, which, however, expose the patient to radiation; therefore, the necessity of such imaging must be carefully considered.

Surgical treatment focuses on correcting the skull shape and preventing any secondary damage to brain development. The optimal timing for surgery is at 6 months of age for simple craniosynostosis, whereas syndromic forms require later and often repeated interventions. Currently, two surgical techniques are used: calvarial remodelling (a comprehensive procedure involving resection of the affected part of the skull, reshaping it, and fixing it with absorbable plates) or endoscopic surgery (endoscopically assisted resection of the affected suture) followed by treatment with a remodelling head orthosis. The greatest risk of surgery is blood loss, which necessitates preoperative haematological preparation, meticulous anaesthetic management, and subsequent intensive care.

BRAIN VASCULAR MALFORMATION (13)

Fadrus Pavel, Smrčka Martin

Cerebral vascular malformations include:
1/ Arteriovenous malformations (AVM)
2/ Cavernous haemangiomas (cavernomas)
3/ Telangiectasias and venous anomalies.
However, only the first two diagnoses are of clinical significance.

Arteriovenous Malformations (AVM)

Pathophysiology: AVMs arise due to disordered embryonic development of the brain's vascular network**. A pathological communication forms between the arterial and venous systems** without a normal capillary bed, allowing arterial blood to flow rapidly through a pathological tangle of thin-walled vessels (referred to as the nidus of the malformation) directly into the veins. Due to the high flow, the feeding arteries and draining veins are usually markedly dilated and tortuous, and arterialised bright red blood is often seen in the veins. In some patients, AVMs have been shown to develop later in life.

Clinical symptoms: AVMs typically present with haemorrhage (in 70% of cases), usually originating from the nidus or the venous component of the malformation, causing spontaneous intracerebral haemorrhage, less commonly subarachnoid haemorrhage (SAH). **The cumulative risk** of AVM bleeding is 2-3% per year. Spontaneous intracerebral haemorrhage from an AVM, unlike hypertensive haemorrhage, typically occurs in the brain's lobes and affects younger individuals with normal blood pressure. Other possible presentations include hemiparesis, aphasia, or epilepsy, more commonly seen in larger malformations due to the so-called **"steal phenomenon."** As most of the blood flows through the malformation, the surrounding brain tissue is poorly perfused, leading to cellular dysfunction at the ion channel level, manifesting as deficit-related (hemiparesis) or irritative (epilepsy) symptoms.

Diagnosis: Diagnosis is established via CT, MRI, and mainly with cerebral panangiography. A pathognomonic feature is a visualisation of the draining veins during the arterial phase on angiography alongside the nidus of the malformation. Based on angiographic and MRI findings, **the Spetzler-Martin grading system (Grades I-V)** assesses AVM size, venous drainage, and eloquence of the nidus's location.

Treatment: The Spetzler-Martin grading influences treatment choice. It is essential to weigh the risks of the natural disease course against those of the selected therapeutic approach. AVM grades I

and II are suitable for **surgical resection**. Surgery involves interrupting the feeding arteries near the nidus and excising it. This procedure is often complex, with difficulty increasing with higher AVM grades. For grades III-V, a combination of surgery and endovascular methods is preferable.
Endovascular therapy involves selectively catheterising feeding arteries close to the nidus and performing embolisation, usually with glue. While eliminating an AVM solely via embolisation is challenging, this method can significantly reduce blood flow through the malformation, facilitating subsequent surgery. Small (up to 3 cm), deep, and eloquently located AVMs with high surgical risk may be better treated with **gamma knife radiosurgery**. Obliteration of the malformation occurs after irradiation through endothelial proliferation and thrombosis. However, in cases where the AVM has caused significant bleeding, time is of the essence, as complete obliteration following gamma knife therapy takes approximately two years, during which the patient remains at risk of further bleeding.

Cavernous Haemangioma (Cavernoma)

Pathophysiology: A cavernoma is a well-demarcated vascular lesion composed of thin-walled caverns through which blood flows very slowly. The lesion is supplied by small arterioles and drained by small venules. Due to the very slow blood flow through the cavernoma, this malformation is not visible on cerebral angiography. Cavernomas are typically located deep within the brain parenchyma, often near the ventricles, with a predilection for the brainstem. Cavernomas may enlarge over time and can be multiple. Multiple cavernomas may have a genetic basis and can be hereditary.
Clinical symptoms: Cavernomas typically present with recurrent small bleeding, which irritates the surrounding brain tissue and leads to epileptic episodes. In some cases, cavernomas may manifest as worsening focal neurological signs (hemiparesis, aphasia) or with bulbar symptoms and cranial nerve involvement (when located in the brainstem).
Diagnosis: Diagnosis is primarily via MRI, where the lesion is particularly well visualised on T2-weighted images. On CT, cavernomas can often be missed.
Treatment: Options include **observation**, particularly if the cavernoma is located in a highly eloquent area with high surgical risk, or if it is an incidental finding. **Surgery** is required for symptomatic cavernomas. In the brainstem or other eloquent locations, surgery is warranted after repeated haemorrhagic episodes with progressive clinical deterioration. Cavernoma excision is relatively straightforward. The surrounding brain tissue is typically stained rust-coloured by haemosiderin from previous haemorrhages. Stereotactic navigation is often beneficial for better surgical orientation. Gamma knife irradiation of cavernomas is considered ineffective.

BRAIN ISCHEMIA (14)

Fadrus Pavel, Smrčka Martin

Ischaemic stroke is a considerable health problem, being the third most common cause of death in developed countries and the main cause of disability. The primary risk factor for ischaemic stroke is hypertension, followed by coronary artery disease, atrial fibrillation, diabetes mellitus, hyperlipidaemia, obesity, and smoking.

Pathophysiology: Cerebral ischaemia occurs when cerebral blood flow (CBF) is reduced to a level that cannot maintain adequate oxygen delivery for cellular functions. The physiological value of CBF is 50 ml/100 g/min. When it drops to 30 ml/100 g/min, somnolence occurs. The ischaemic threshold is crossed below 20 ml/100 g/min, leading to the "penumbra" stage, where electrical activity in the neuron's ceases; however, their anatomical integrity remains intact. This stage is reversible. Acute therapeutic intervention focuses on the brain tissue within the penumbra. Necrotic cells are irreversibly lost. CBF below 10 ml/100 g/min results in irreversible brain damage.

- **Ischaemic stroke**: A permanent neurological deficit resulting from inadequate perfusion in the corresponding brain region.
- **Reversible ischaemic neurological deficit (RIND)**: Lasts more than 24 hours but less than one week.
- **Transient ischaemic attack (TIA)**: A transient focal neurological deficit lasting less than 24 hours, typically a few minutes.

The most common cause of ischaemic stroke is atherosclerosis, though a cardiogenic origin is also significant. Arteritis or fibromuscular dysplasia are rare. The causes of ischaemia include:

- **Embolism**: The source is often an unstable substance from a stenotic internal carotid plaque or thrombi forming in the heart in atrial fibrillation and bacterial endocarditis.
- **Thrombosis**: Occurs in hypertensive patients at sites of tight stenosis in large intracranial arteries.
- **Lacunar infarcts** affect small penetrating arteries in the basal ganglia.
- **Haemodynamic infarcts**: Result from a drop in mean arterial pressure exceeding the limits of autoregulation. These occur with extracranial internal carotid artery occlusion and an insufficient Circle of Willis. Global ischaemia, known as "brain hypoxia", due to prolonged hypotension, also falls into this category.

Diagnosis: 1/ **Imaging the extent of infarction**:

- **CT**: The basic method for determining the extent and location of the infarct. Its advantages include availability, the ability to differentiate haemorrhage, and speed. However, it is less sensitive than MRI. In the acute phase, within a few hours of the event, CT changes may be minimal or subtle, such as a loss of grey-white matter differentiation, disappearance of gyri, and mild expansion. CT perfusion improves sensitivity in early infarct detection and can distinguish between penumbra and necrosis. The brain tissue in the penumbra is the target of acute therapeutic intervention. CT angiography can identify the location of major arterial occlusions.
- **MRI**: Diffusion – and perfusion-weighted MRI can detect ischaemia in the acute phase. It is more precise than CT in identifying the penumbra. Additionally, MRI can detect small-volume lesions, such as lacunar infarcts, which are not seen on CT. FLAIR MRI visualises subacute and chronic ischaemic lesions.

2/ **Imaging vascular pathology**:

- **Carotid ultrasound**: The basic screening test for extracranial vessels. In the hands of an experienced sonographer, it has high sensitivity and specificity, and it is minimally invasive.
- **Angiography (DSA)**: Provides detailed and reproducible imaging of vascular pathology. Unlike ultrasound, it also visualises intracranial vessels and provides the surgeon with an excellent anatomical overview. Its disadvantages include invasiveness and associated risks, such as stroke and inguinal haematoma.
- **CT or MR angiography**: CT angiography has largely replaced DSA in determining the degree of carotid stenosis. In cases where CT angiography is contraindicated (e.g., contrast allergy), MR angiography can be used.
- **Transcranial Doppler (TCD)**: Visualises intracranial vessels and measures blood flow speed and direction.

3/ Functional diagnostics:

- **Perfusion CT under stress, SPECT, or TCD under stress**: These tests identify patients at risk of haemodynamic stroke. In response to reduced cerebral perfusion pressure (CPP), patients compensate by dilating cerebral arterioles to maintain adequate cerebral blood flow (CBF). This is the basis of CBF autoregulation.

Therapy: 1/ **Surgical treatment of cerebral ischaemia** Most strokes are treated conservatively. Surgical treatment can be divided into elective and acute procedures.

a/ **Elective procedures**: After the resolution of stroke, transient attack, or ocular symptoms, surgery aims to prevent the recurrence of clinical symptoms.

- **Stenosis of the internal carotid artery**
- **Carotid endarterectomy**: Patients who have experienced a cerebral TIA (transient hemiparesis contralateral to the carotid lesion) or ocular TIA, known as amaurosis fugax, where there is transient blindness in the ipsilateral eye due to embolism in the central retinal artery, undergo evaluation of the carotid arteries. Confirmation of more than 50% stenosis of the internal carotid artery is an indication for surgery. The procedure should be performed within 14 days of the event to prevent the recurrence of the stroke. Asymptomatic carotid stenosis (incidental findings on ultrasound) is rarely the cause of stroke over time with modern medical treatment (antiplatelet therapy, statins). Therefore, the decision for surgery is strictly individualised. Patients with stenosis over 60% are indicated for surgery if other risk factors for stroke are present (e.g., unstable carotid plaque, atypical Circle of Willis, male gender, etc.). Carotid surgery is preventive, aimed at preventing a threatened stroke. Patients who are non-ambulatory with severe residual neurological deficits are unlikely to benefit from surgery.
- **2/ Carotid artery stenting**: The method involves disrupting the plaque by dilating the stenosis with an angioplasty balloon and implanting a stent into the vessel wall. Indications include restenosis after previous carotid surgery (a difficult and risky procedure for both the surgeon and patient), a high bifurcation of the carotid artery that is surgically difficult to access, and polymorbid patients for whom open surgery would pose a greater risk.
- **3/ Kinking (bending) of the carotid artery**: If it causes ischaemic symptoms, it requires resection of the redundant vessel wall and reimplantation into the common carotid artery. The cause of kinking, like stenosis, is atherosclerosis.
- **4/ Coiling of the carotid artery**: This is usually not the cause of stroke and, therefore, does not typically require surgical intervention. Resection of the coil is justified if recurrent strokes occur despite maximum medical therapy (dual antiplatelet therapy).
- **5/ Extra-intracranial bypass**: This is indicated in patients with chronic occlusion of the carotid artery and recurrent minor strokes or TIAs despite maximal medical therapy. The indication for surgery is the depletion of cerebrovascular reserve capacity. In this procedure, one of the branches of the superficial temporal artery is dissected and anastomosed end-to-side to a recipient branch of the middle cerebral artery (MCA).

b/ **Acute procedures**:

- **Acute carotid thrombendarterectomy**: This is performed relatively rarely and is most often done in already hospitalised patients with minor stroke or TIA who experience a sudden deterioration in clinical status due to occlusion of a previously stenotic internal carotid artery.

- **Intravenous thrombolysis**: Intravenous administration of recombinant tissue plasminogen activator (rtPA) is recognised as the treatment of choice for severe strokes, based on positive results from European and American studies, provided there is no haemorrhage on CT and the time interval between stroke onset and treatment is no more than 4.5 hours. This method is highly effective.
- **Mechanical thrombectomy**: In cases where stroke is caused by occlusion of a large arterial trunk (e.g., internal carotid artery, MCA), intravenous thrombolysis may not be successful. In such cases, prompt intervention with mechanical thrombectomy is performed. The method involves introducing a stent retriever through the arterial system beyond the occlusion site. The stent dilates, allowing the embolus or thrombus to be captured and then removed retrogradely from the circulation via the femoral artery. The time window for this procedure is up to 6 hours from stroke onset, but there is no strict time limitation in the case of basilar artery occlusion. Stroke care and comprehensive cerebrovascular centres are concentrated in stroke centres, where intravenous thrombolysis can also be administered. Efficient logistics and cooperation are crucial, following the principle that "time is brain." Stroke care in the Czech Republic is currently well organised.
- **Decompressive craniectomy for expansively behaving ischaemia**: This is performed when the ischaemic volume involves the entire MCA territory and behaves expansively, compressing the brainstem. Clinical deterioration typically occurs 48–72 hours after the stroke, primarily due to the development of oedema. The patient becomes somnolent and eventually falls into a coma, with signs of temporal herniation. Removing the bone flap and dura mater reconstruction at this stage is life-saving. Decompressive craniectomy reduces intracranial pressure and ensures adequate cerebral perfusion. The residual neurological deficit is usually significant.

INTRACRANIA HYPERTENSION, BRAIN EDEMA (9)

Fadrus Pavel, Smrčka Martin

Intracranial and perfusion pressure

An adult lying down has normal intracranial pressure (ICP) values between 7-15 mmHg. In the upright position, ICP may drop below atmospheric pressure values. In children, normal ICP is 3-7 mmHg. During sneezing, coughing or other types of Valsalva manoeuvre, ICP rises physiologically up to values of around 60 mmHg. ICP values higher than 20 mmHg are generally accepted as pathological (**intracranial hypertension**).

In 1783, Monro was the first to describe the relationship between volume and pressure inside the cranial cavity. Later, this theory was supplemented by Kellie (1823) and Burrows (1848) and **the Monro-Kellie hypothesis** was created. According to this theory, the cranial cavity is a rigid box of fixed volume containing three incompressible compartments: brain tissue, blood, and cerebrospinal fluid. If the volume of one of these compartments increases, another must decrease if the pressure inside the skull is to remain stationary. This ability is used under physiological circumstances during cardiac activity. With each heartbeat, about 15 ml of new blood flows into the brain, the increase in volume being quickly compensated by the movement of cerebrospinal fluid and venous blood out of the cranial cavity. The most common cause of increased intracranial pressure is an intracranial expansion lesion (tumour, hematoma, cyst, abscess), cerebral oedema or cerebrospinal fluid passage disorder (hydrocephalus).

The effect of volume changes inside the cranial cavity on intracranial pressure is dependent on the state of compensatory mechanisms. It depends on how much cerebrospinal fluid can still be moved from the cranial cavity to the spinal canal, how much the cerebral blood volume (CBV) can be reduced in the cerebral vessels (especially the venous system) and what is the state of elasticity of the brain tissue. After compensatory mechanisms have been exhausted, the rapidly increasing intracranial pressure can cause displacement of the brain mass and the formation of **brain herniation**. If the expansive lesion acts unilaterally, there is a midline shift of the brain mass. A **subfalcine (cingulate) herniation** can occur when the brain tissue of the frontal or parietal lobe is hidden under the free edge of the falx, referred to as the signum falcis.

This may result in ischemia in the basin of pericallosal arteries (aa. pericallosae). Herniation of the temporal lobe into **the tentorial notch (tentorial herniation, conus temporalis)** is common. It was first described by Meyer in 1920. In its frequent form, the medial part of the temporal lobe is pushed into the tentorial notch. At the same time, the brainstem and the oculomotor nerve on the ipsilateral side are compressed. The posterior cerebral artery is often closed by direct pressure against the edge

of the tentorium, and an ipsilateral infarction occurs in its basin. Clinically, the temporal cone syndrome is manifested by severe impairment of consciousness (usually GCS 3–5), ipsilateral mydriasis, contralateral hemiparesis and often also disorders of vital functions. In the pathophysiological mechanism, however, the pressure of the opposite edge of the tentorium on the brainstem and the contralateral oculomotor nerve can also be applied. This syndrome must be understood as an expression of decompensated intracranial hypertension. **A tonsillar herniation (occipital cone)** may occur during expansions in the posterior cranial fossa or as a continuation of mass transfer from the supratentorial compartment. The cerebellar tonsils reach below the level of the foramen magnum, obliteration of the large cistern occurs, and mainly direct pressure on the elongated spinal cord. This mechanism can very quickly manifest as sudden respiratory arrest and death. Tonsillar herniation can be provoked if we perform a lumbar puncture in a patient with intracranial hypertension. For this reason, lumbar puncture is contraindicated in these patients.
In addition to intracranial pressure, **Cerebral Perfusion Pressure (CPP)** is also important in the pathophysiology of neurosurgical diseases. CPP is calculated as the difference between arterial pressure and venous pressure. In practice, it is difficult to determine the venous pressure in the cortical veins, but it is only slightly higher than the intracranial pressure value. **CPP is therefore defined as MABP - ICP (MABP, Mean Arterial Blood Pressure)**. In all patients, we try to keep CPP above 60 mmHg (if we monitor ICP and MABP). CPP is important because it relates to cerebral blood flow (CBF). The relationship between CBF and CPP is reflected in **the autoregulation curve**, when between about CPP 40-160 mmHg CBF remains unchanged, below 40 mmHg CBF decreases dramatically.
The quantity that mediates the relationship between CBF and CPP is cerebrovascular resistance. According to Poiseuille's law, it is inversely proportional to the fourth power of the diameter of the vessel. Changes in cerebrovascular resistance are the essence of autoregulation of cerebral blood flow. In practice, this means that as CPP is reduced, vasodilation occurs gradually to maintain CBF. Vasodilatation is maximal at the lower end of the plateau phase of the autoregulation curve, when the compensatory capacity of the vascular wall is exhausted. In many cerebral disorders (injuries, cerebral ischemia), the autoregulatory ability of the cerebral vessels is impaired.
Clinically, intracranial hypertension may not show any symptoms at first. In the later stages, headaches, nausea or vomiting appear, and gradually there is a disturbance of consciousness. One of the late symptoms of intracranial hypertension is the Cushing's reflex - hypertension and bradycardia. This is a reflex effort of the organism to maintain sufficient CPP. During an ophthalmological examination, after a certain duration of intracranial hypertension (usually at least

two days), we find the prominence of the papilla of the optic nerve (congestion on the fundus), which is indicated in diopters.

Cerebral oedema

The most important types of cerebral oedema are vasogenic and cytotoxic oedema (Klatzo, 1967). Cerebral oedema contributes to increased intracranial pressure by increasing the volume of the brain mass and reducing the viscoelastic properties of the parenchyma (compliance).

Vasogenic oedema appears primarily in the white matter of the brain. It is common, for example, after an injury, but it is typical for tumours and infections. Mechanical trauma to the brain tissue, tumour growth or infectious influences will disrupt the integrity of the blood-brain barrier. This results in extravasation of fluid and plasma proteins into the extracellular space. These proteins further exacerbate oedema based on an altered oncotic gradient. The cause of the disruption of the blood-brain barrier is not only mechanical, but substances released from damaged brain tissue such as bradykinin, arachidonic acid, histamine and free radicals are also involved in the increase in vascular permeability. Type IV collagenase is also apparently involved in this type of oedema by dissolving the basal lamina of the capillary bed. On CT, it appears as a hypodense area in the brain's white matter. Corticoids are effective for this type of oedema, especially in tumours or abscesses. Post-traumatic oedema, on the other hand, does not respond to corticoids, apparently because it is a combination of vasogenic and cytotoxic oedema.

Cytotoxic oedema is most pronounced in the grey matter of the brain. It is sometimes called ischemic because a disorder of cerebral blood flow causes it. This leads to deterioration of cellular metabolism and malfunction of membrane ion channels. Together with sodium, water enters the intracellular space, and oedema occurs. Another possibility is that cytotoxic oedema occurs secondary to vasogenic oedema, which causes microcirculation to collapse. We demonstrate cytotoxic oedema on CT as hypodensity in the grey and white matter of the brain.

Cerebral hyperemia (swelling) can be another cause of brain swelling. It is more common in children but can also occur in adult patients. Pathophysiologically, it is a different entity from brain oedema. There is an idea that the cause is direct damage to the hypothalamus and brainstem with vasoregulatory centres. This will result in vasoparalysis with a subsequent increase in cerebral blood flow and cerebral blood volume, to the extent that it manifests as an increase in intracranial pressure and obstruction of venous outflow. Eventually, decompensation, a decrease in cerebral perfusion pressure, and cerebral ischemia occur. On CT, the hyperemic brain appears as a homogeneous isodense or hyperdense mass with lost gyrification and obliterated basal cisterns.

Intracranial pressure monitoring

The only way to determine intracranial pressure values is by directly measuring it using a sensor inserted into the intracranium. Previously used epidural sensors are not very accurate; subdural sensors have slightly better results. We record the most accurate ICP measurements using fibre-optic intraparenchymal or intraventricular sensors. In the case of intraventricular administration, derivation of cerebrospinal fluid is also possible. If possible, both intraparenchymal and intraventricular leads are inserted from the Kocher point, usually in the non-dominant hemisphere. This point is located about 3 cm lateral to the midline and 2 cm ventral to the coronary suture. This is the site that is commonly used for ventricular puncture or ventriculostomy (Fig):

Current monitors used in the ICU for invasive monitoring already continuously record the ICP curve in mmHg without difficulty. If the patient also has invasive BP monitoring installed (e.g. in the radial artery), the monitor continuously calculates the mean arterial blood pressure and relates it to the ICP. This also gives us a continuous CPP curve. Based on the knowledge of the current values of ICP and CPP, we can carry out treatment measures.

HYDROCEPHALUS – PATHOPHYSIOLOGY, CLINICAL SIGNS, TREATMENT MODALITIES (11)

Barák Martin, Brichtová Eva

The term "hydrocephalus" originates from the Greek words "hydro" meaning water, and "kephale" meaning head. It refers to an abnormal accumulation of cerebrospinal fluid (CSF) within the cranial cavity, either in the brain's ventricles or in the external CSF spaces.
Cerebrospinal fluid is primarily produced in the lateral ventricles by the choroid plexus. It then flows from the lateral ventricles through the foramen of Monro (intraventricular foramen) into the third ventricle, continues via the aqueduct of Sylvius (cerebral aqueduct) to the fourth ventricle, and passes through the foramina of Luschka (laterally) and foramen of Magendie (medially) into the subarachnoid spaces (cerebral cisterns). From there, it is absorbed predominantly into the venous system through the arachnoid granulations (Pacchionian granulations) into the cerebral venous sinuses. The total amount of CSF in the ventricles and subarachnoid spaces of an adult is approximately 150 mL and it is renewed three times a day.

Types and basic classification of hydrocephalus:

By etiology - **Congenital**
- **Acquired**

By pathogenesis

- **Obstructive hydrocephalus (non-communicating) –** a blockage within the ventricular system leading to the obliteration of the normal flow of cerebrospinal fluid (causes include congenital malformations, tumors, hemorrhage with clot formation).
- **Communicating hydrocephalus –** further divided into two subtypes:
    - **Hyporesorptive** – impaired absorption of CSF due to fibrotic changes in the subarachnoid spaces (following subarachnoid hemorrhage and/or inflammation, such as meningitis and meningoencephalitis).
    - **Hypersecretory** – increased production of CSF (e.g., tumors of the choroid plexus).
    - A specific type is termed **normal pressure hydrocephalus** (NPH).

The clinical manifestation of hydrocephalus is associated with symptoms of intracranial hypertension (depends on the patient's age and the progression of hydrocephalus). Symptoms include headache, vomiting, visual disturbances, behavioral changes, cognitive decline, psychiatric

symptoms, and, in severe cases, loss of consciousness. In neonates and infants with an open anterior fontanelle, there could be head enlargement (macrocephaly), a bulging and tense fontanelle, and prominent scalp veins. Infants can be irritable or lethargic, sleepy, and exhibit delayed psychomotor development. Decompensated hydrocephalus may present with the “setting sun” sign (eyes deviated downward with the lower part of the iris covered by the lower eyelid) and Parinaud's syndrome (impaired upward gaze).

Diagnosis is based on clinical examination and imaging methods: MRI of the brain (to identify obstruction, especially at the aqueduct of Sylvius, and dynamic sequences for CSF flow), CT of the brain (quick and accessible but with radiation exposure), and cranial ultrasound through the anterior fontanelle in children (non-invasive and allows repeated assessments to monitor hydrocephalus dynamics). Fundoscopic examination to assess for papilledema is often performed.

Therapeutic management - symptomatic (active) hydrocephalus requires neurosurgical intervention for CSF drainage. This can be either temporary (external ventricular drainage, external lumbar drainage) or permanent (internal drainage - shunt, where the CSF is diverted to another body cavity, most commonly the peritoneal cavity or the circulatory system).

A permanent internal drainage system (shunt) consists of three parts: a proximal catheter (inserted into the brain ventricle, cyst, external CSF spaces, or dural sac in the lumbar region – known as a lumboperitoneal shunt), a one-way valve (fixed or programmable), and a distal catheter (leading to the peritoneal cavity – VP shunt or to the right atrium – ventriculoatrial (VA) shunt). **Complications** of internal shunt systems are mechanical (catheter or valve obstruction due to scarring or blood clots, or disruption of shunt continuity) or infectious (dehiscence with shunt exposure, ventriculitis, sepsis). Endoscopic surgery, aimed at creating a new drainage pathway for CSF, is an alternative treatment for obstructive hydrocephalus. The most common procedure is endoscopic third ventriculostomy (ETV), which involves perforation of the floor of the third ventricle to drain into the prepontine cistern. If a tumor obstructs CSF flow, the treatment involves surgical excision of the tumor.

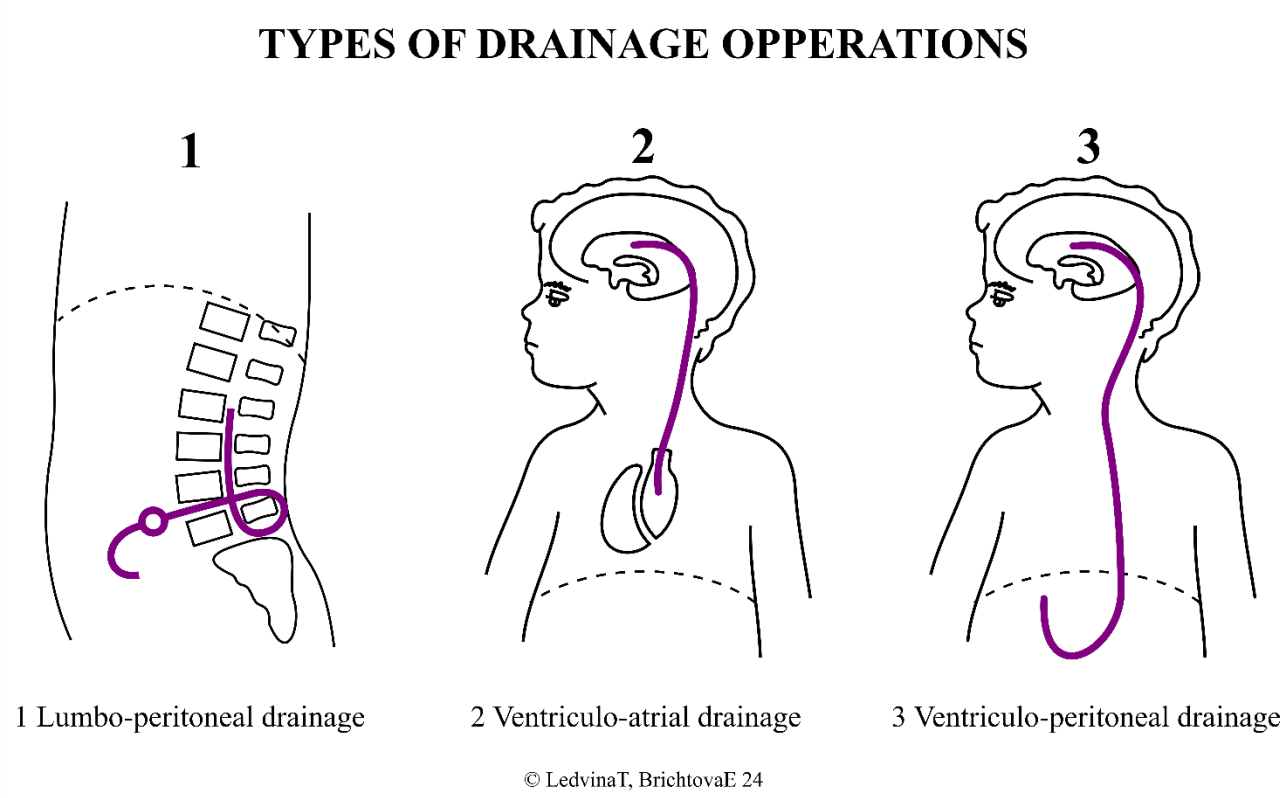
A specific subtype of hydrocephalus is **normal pressure hydrocephalus (NPH)**, a form of communicating hydrocephalus characterized by ventricular enlargement with abnormal CSF dynamics (intracranial pressure is normal or only episodically elevated), accompanied by a characteristic triad of clinical symptoms. It primarily occurs in older patients (over 60 years) and its exact pathogenesis is unknown (a risk factor includes a history of elevated intracranial pressure due to trauma, inflammation, or hemorrhage).

Clinical symptoms of NPH: Hakim's triad – dementia (memory impairment and cognitive decline), gait disturbances (short steps, wide base, instability), and urinary incontinence.

Diagnosis: based on clinical symptomatology, imaging studies (brain MRI, CT), and functional CSF assessment:

- CSF resistance tests (LIT – increased resistance is typical for NPH).
- Lumbar puncture (normal initial pressure) and the so-called TAP test (symptomatic improvement after the removal of 30-50 mL of CSF during lumbar puncture; an alternative is a multi-day lumbar drainage).

A typical clinical presentation and positive functional CSF tests are indications for VP shunt implantation.



SCULL FRACTURES (15)

Fadrus Pavel, Smrčka Martin

A plain skull X-ray, typically performed in two projections, can establish the diagnosis. However, this is now rarely used in practice. The most reliable imaging for craniofacial skeletal and intracranial structure injuries is a CT scan.

Skull fractures are divided into **linear (fissures)** and **depressed fractures**. Depressed fractures are often **comminuted (fragmented)**.

1/ **Linear fractures** do not require surgery and will heal spontaneously. However, they require monitoring as they can lead to an epidural haematoma. This may arise from injury to the meningeal artery by the bone edge or, more slowly, due to bleeding from the diploe.

2/ **Some depressed fractures** are also treated conservatively. This is usually the case when the bone fragment depression is less than the thickness of the bone or when the depression occurs at the site of venous sinuses, where elevation of the fragments could result in difficult-to-control bleeding. Surgical treatment is necessary if the depression is greater than the thickness of the skull if it causes focal neurological symptoms or epilepsy, or if it presents a cosmetically unfavourable appearance. Surgery is also recommended for a growing fracture, which occurs in young children. In this condition, the dura mater becomes entrapped at the fracture site, and due to the pulsation of intracranial contents, the defect enlarges as the bone edges progressively separate — a "ping-pong ball" fracture that normally heals spontaneously. A craniotomy is typically performed around the depressed bone plate. The bone is then fixed with bone stitches or mini-plates. In significant, comminuted, or infected fractures, bone fragments are removed. Large bone defects are typically reconstructed using custom-made 3D implants fabricated from various materials, such as titanium, Medpor, or PEEK.

Basilar Skull Fractures

Clinically, basilar skull fractures may present with raccoon eyes (periorbital haematoma), retroauricular haematoma (Battle's sign), haemotympanum, or cerebrospinal fluid (CSF) leakage (nasal or otorrhoea). Fractures involving a tear in the dura mater and CSF leakage carry a high risk of meningitis and brain abscess. CT is the most reliable diagnostic tool. The presence of pneumocephalus suggests a breach in the paranasal sinus walls and typically indicates a tear in the dura mater.

Conservative management is appropriate for basilar skull fractures without CSF leakage or with resolving leakage. Conservative therapy for CSF leakage includes fluid restriction, bed rest with head elevation, avoiding activities that increase abdominal pressure, administration of Diluran (to

reduce CSF production), and Framycetin nasal drops to prevent infection. Healing can be supported by inserting a spinal drain to divert a certain amount of CSF daily (typically around 100–200 ml over 24 hours).

Fractures with persistent CSF leakage require surgery. Surgery is also recommended for patients with a history of head trauma who develop recurrent meningitis, even if the injury occurred years ago and there is no evidence of CSF leakage. Other indications include pneumocephalus (which may also be valvular), orbital roof fractures associated with impaired ocular movement, enophthalmos, or exophthalmos.

The procedure involves the reconstruction of the anterior cranial fossa. A bifrontal craniotomy is performed, with the scalp reflected from the hairline to the supraorbital margins. Only loose bone fragments are removed from the base of the skull. If a dural tear is identified, it is sutured with non-absorbable material. Complications of basilar skull fractures can be severe and include recurrent CSF leakage, meningoencephalitis, and epilepsy. Transverse fractures of the petrous bone can cause injury to cranial nerves V, VI, VII, and VIII, while longitudinal fractures can result in conductive hearing loss. Injuries to cranial nerves I and II can occur with fractures of the anterior cranial fossa, and injuries to cranial nerves IX-XII can occur with condylar fractures. Less common vascular complications include traumatic carotid-cavernous fistula, false aneurysm of the petrous or cavernous segment of the carotid artery, and carotid artery occlusion.

CONCUSSION, CONTUSION AND DIFFUSE AXONAL INJURY (16)

Barák Martin, Brichtová Eva

Craniocerebral injuries (or TBI – traumatic brain injury) occur due to the impact of blunt force or as a result of penetrating head injuries, leading to structural damage to the CNS or CNS dysfunction. TBIs are often associated with injuries to the soft tissues of the head or skull, and cervical spine injuries are also frequently concurrent. These injuries are of significant epidemiological concern—TBI is the leading cause of death among children and young adults in all types of trauma (10% of TBIs are fatal; 50% of patients are initially unconscious), and survivors often suffer lifelong consequences.

Classification of TBIs:

By severity (most commonly assessed using neurological status/Glasgow Coma Scale GCS – not a linear scale):

- **Mild** (GCS 14-15; loss of consciousness/amnesia)
- **Moderate** (GCS 9-13; or focal neurological deficit)
- **Severe** (GCS 3-8; coma)

By pathophysiology:

- **Primary injury –** brain injury occurring at the time of trauma; the primary cause cannot be influenced.
- **Secondary injury –** indirect brain injury resulting from pathophysiological changes caused by the acute CNS insult; clinical management aims to minimize the development of secondary injury (e.g., brain edema, hypoxia of brain tissue)

By extent of brain damage:

- **Focal injuries –** brain contusion, intracranial hemorrhage (intracerebral hematoma, traumatic subarachnoid hemorrhage, subdural hemorrhage, epidural hemorrhage)
- **Diffuse injuries –** concussion, diffuse axonal injury (DAI)

Typical causes of TBI include falls (including those during intoxication), car accidents, and acts of violence. The specific injuries depend on the force exerted and the mechanism of trauma. Risk factors include older age, use of anticoagulant/antiplatelet medications, internal comorbidities, and intoxication.

Clinical presentation depends on the type and severity of the TBI. Classic symptoms may include headaches, nausea and vomiting, focal neurological symptoms (e.g., limb paresis, speech disturbances), seizures (with the risk of developing late epilepsy), and, in the most severe cases, impaired consciousness.

Common complications include bleeding, which typically manifests within 24 hours of the initial injury, and the risk of developing brain oedema with subsequent intracranial hypertension syndrome, epilepsy, hydrocephalus, and infectious complications.

Diagnosis relies heavily on a history of the injury and includes a neurological examination to stratify the severity of the condition and topically localize the injury. Imaging techniques—particularly CT (trauma protocol)—play a crucial role. CT is the method of choice in acute conditions and can clearly identify the most common types of brain injuries that require urgent neurosurgical intervention. In cases of TBI, CT of the cervical spine is always necessary (5% of head traumas are associated with severe cervical spine injuries); otherwise, the patient must be managed as if they have a cervical spine injury (semi-rigid cervical collar).

Brain concussion (commotio cerebri) represents a reversible functional disorder characterized by a brief loss of consciousness without evident traumatic anatomical changes (negative CT findings). Loss of consciousness may last only a few seconds and rarely exceeds 30 minutes. Clinical signs include headaches and memory impairment (typically retrograde amnesia—patients do not remember the injury), as well as nausea/vomiting. Some patients exhibit late sequelae of concussion (so-called post-concussion syndrome), with headaches, concentration difficulties, and sleep disturbances persisting for several months post-trauma. Diagnosis is based on injury history and normal neurological findings, with the need to exclude other traumas on brain CT that would require neurosurgical intervention. Treatment is fully conservative; patients are usually hospitalized for 24 hours for observation and analgesic therapy.

Brain contusion due to bruising of the brain tissue, ranging from minor lesions to extensive, multiple contusions. It typically occurs on the convexity of the brain, most commonly in the temporal and frontal lobes (coup—where the brain impacts the hard skull during sudden movement), and contusions may also develop on the opposite side of the impact site (contre-coup). Less commonly, contusions result from depressed skull fractures. Clinical symptoms depend on injury severity—minor contusions may be entirely asymptomatic. Typical manifestations include focal neurological symptoms, such as motor and speech disorders (depending on the affected eloquent area), headaches, behavioral disturbances, and impaired consciousness. Structural damage to the cerebral cortex is also a risk factor for seizure development. Larger contusions can lead to brain edema and subsequent intracranial hypertension. Diagnosis is based on CT findings—typically showing signs of bleeding within brain tissue on the convexity (hyperdensity on brain CT); however, contusion lesions may not be visible on initial examination and can develop over several hours, warranting additional CT scan within 24 hours. Anamnesis of the injury and neurological symptoms corresponding to the affected brain region are also crucial. Treatment is predominantly conservative,

requiring clinical monitoring and prevention of secondary damage (especially blood pressure correction; in the case of anticoagulant or antiplatelet medication use, reversing their effects). Surgical intervention is indicated in specific cases of developing intracranial hypertension from expansively behaving contusions —decompressive craniectomy is indicated in severe TBIs (supratentorial) with signs of intracranial hypertension unresponsive to antiedema therapy. In cases of expansive contusion in the posterior cranial fossa (risk of brainstem compression), decompression of the posterior cranial fossa with surgical treatment of the hemorrhage/contusion is required.

Diffuse Axonal Injury (DAI) is characterized by multiple axonal damages resulting from trauma. DAI typically follows high-energy trauma, involving shear forces and mechanical damage to brain pathways. The clinical picture is marked by post-traumatic unconsciousness, which may persist for months, depending on the injury's severity. Neurological findings also depend on specific neural pathway damage (focal lesions, decortication or decerebration symptoms, and brainstem involvement). Consciousness impairment (vegetative coma) or severe neurological deficits can persist long-term, representing a highly unfavorable prognosis. DAI diagnosis is based on trauma history and clinical status (persistent unconsciousness after TBI with exclusion of other causes). DAI patients may present minimal or negative findings on initial brain CT (necessary to exclude other urgent conditions requiring surgical intervention, such as SDH/EDH), which do not explain the neurological deficit. Specific DAI changes can be observed only on brain MRI (typically in the corpus callosum and basal ganglia). The initial therapeutic goal is to ensure vital function stability (mechanical ventilation and circulatory support) and overall clinical stabilization. Due to the risk of developing intracranial hypertension, intracranial pressure monitoring and appropriate treatment of intracranial hypertension are recommended. Patients often require subsequent intensive care with an uncertain neurological outcome.

INTRACRANIAL BLEEDING (17)

Barák Martin, Brichtová Eva

Intracranial hemorrhage can be classified by etiology into traumatic and non-traumatic (spontaneous). Based on location, intracranial hemorrhage is divided into extracerebral (EDH, SDH, SAH) and intracerebral (ICH, IVH). The most common cause of extracerebral hemorrhage is trauma.

Epidural Hematoma (EDH) – An epidural hematoma occurs due to bleeding into the space between the dura mater and the skull. Under normal conditions, this space does not exist because the dura mater firmly adheres to the skull. Epidural hematomas occur in approximately 1–6% of all head injuries and are most commonly caused by bleeding from the meningeal artery, which is damaged by a skull fracture. EDH can also result from bleeding from a damaged skull or an injury to a venous sinus. The most typical localization is the temporal region, where the source of bleeding is usually the middle meningeal artery, or its branches damaged by a fracture. However, a skull fracture is not a necessary condition for the development of EDH (up to 40% of cases have no fracture). The clinical presentation may resemble that of acute subdural hematoma (aSDH), but EDH often includes a so-called lucid interval (seen in 10–27% of patients). This interval is characterized by a brief loss of consciousness immediately following the injury, followed by a period of full consciousness, and then a subsequent loss of consciousness due to EDH. In the temporal region, EDH is most dangerous because even minor pressure on the temporal lobe can lead to its displacement and compression of the brainstem, resulting in severe neurological symptoms. If symptomatic EDH is not promptly and adequately treated, it usually results in death. Treatment involves evacuation of the hematoma via craniotomy, cessation of bleeding, and suturing the dura to the bone edges to prevent recurrence.

Acute Subdural Hemorrhage (aSDH) – This type of hemorrhage occurs between the dura mater and arachnoid. It is usually caused by bleeding from a bridging vein or from a cerebral contusion. On CT imaging, it appears as a hyperdense crescent of blood that follows the brain surface. Smaller aSDH may be asymptomatic, while larger ones typically lead to impaired consciousness, contralateral hemiparesis, and ipsilateral pupillary dilation (mydriasis). The development of ipsilateral mydriasis is a poor prognostic sign, indicating brain shift and compression of the third cranial nerve (oculomotor nerve). Brain compression caused by aSDH can also trigger epileptic seizures. Symptomatic hematomas must be surgically removed via craniotomy and the source of bleeding surgically managed, sometimes requiring decompressive craniectomy. Smaller aSDH may resolve spontaneously or gradually evolve into chronic subdural hematomas.

Chronic Subdural Hematoma (chSDH) – A chronic subdural hematoma is defined as a hematoma persisting for more than three weeks after injury. In some cases, trauma may not be evident in the patient's history. This type of hematoma is relatively common in neurosurgical settings and frequently occurs in patients with brain atrophy, where even minor head trauma can cause bleeding, usually from a bridging vein. This condition mainly affects elderly individuals, alcoholics, and patients with coagulopathies. Chronic coughing, such as in asthmatics, can also contribute to the development of chSDH. These hematomas do not undergo spontaneous resorption; instead, they gradually become encapsulated, with the capsule being richly vascularized due to neovascularization. This process can lead to gradual hematoma enlargement due to recurrent minor bleeding and osmotic activity drawing fluid from the surroundings. Clinical symptoms can be subtle for a long time and are often mistaken for dementia, presenting typically as progressive headaches, psychological changes, gait disturbances, and falls, leading to decompensation of chSDH and ultimately neurological deficits that prompt diagnosis. On CT, chSDH appears as an iso- or hypodense crescent-shaped collection around the hemisphere, often with components of various ages and signs of recent bleeding. Membranes and septations within the hematoma, visible on CT, may complicate treatment. Despite subtle clinical signs, chSDH is a severe condition that, without treatment, can be fatal. Decompensation, such as further bleeding into the hematoma, can quickly worsen the clinical picture. Treatment involves evacuating the hematoma through burr holes, washing the subdural space, and inserting drainage, which remains in the subdural space for 24–72 hours. In cases of septated hematomas, repeat surgery or more radical craniotomy may be required.

Traumatic Subarachnoid Hemorrhage (traumSAH) – Trauma can also lead to traumatic subarachnoid hemorrhage, involving bleeding between the pia mater and arachnoid. It is crucial to exclude other causes of subarachnoid hemorrhage, such as ruptured cerebral aneurysms, especially when trauma history is unclear or when SAH is located in the cisterns or Sylvian fissure, as purely traumatic SAH is usually found on the convexity. It should be recognized that falls or trauma might occur as a result of aneurysm rupture or other causes. Traumatic SAH is treated conservatively.

Intraventricular Hemorrhage (IVH) – Intraventricular hemorrhage involves bleeding into the cerebral ventricles. It can be caused by head trauma or, in the case of spontaneous IVH, by rupture of an intracerebral hemorrhage into the ventricles. Massive hemorrhage may lead to obstructive hydrocephalus, necessitating the insertion of a temporary external ventricular drain to divert cerebrospinal fluid.

Intracerebral Hemorrhage (ICH) – Intracerebral hemorrhage accounts for 15-30% of all cerebrovascular accidents (the remainder are ischemic). ICH refers to bleeding directly into brain

parenchyma and has significantly higher mortality than ischemic strokes. ICH can be classified etiologically into: a) primary (predominantly due to uncontrolled arterial hypertension) and b) secondary (due to vascular malformations – aneurysms, arteriovenous malformations, CNS tumors, coagulopathies including iatrogenic from anticoagulants, or complications of neurosurgical procedures). Primary (spontaneous) ICH typically occurs in the basal ganglia, thalamus, or brainstem and is associated with significant neurological deficits, including impaired consciousness. Clinically, hemorrhagic stroke is challenging to distinguish from ischemic stroke, though ICH usually shows clinical progression over minutes to hours, with headache and altered consciousness (in contrast to ischemic stroke, which typically has sudden onset with maximum initial deficit). Diagnosis relies on non-contrast CT of the brain showing hyperdensity in the parenchyma; CT angiography may be required if a vascular cause is suspected, particularly in younger patients without hypertension history. Primary ICH is usually managed conservatively, with ICU admission, stabilization of vital functions, prevention of secondary brain injury, and control of hypertension. Anticoagulant reversal is necessary if applicable. Surgical intervention is indicated for cerebellar hemorrhages with brainstem compression or atypical superficial hematomas to relieve pressure on surrounding brain tissue.

BRAIN ABSCESS, SUBDURAL EMPYEMA (18)

Barák Martin, Brichtová Eva

Inflammatory diseases of the brain and spine constitute a group of severe infectious conditions that require prompt diagnosis and treatment due to their potentially fatal course. The most common inflammatory diseases of the brain include meningitis, brain abscess, cranial osteomyelitis, and subdural empyema.

Central nervous system (CNS) infections can be categorized based on their cause. In neurosurgery, the most frequently encountered infections are a) **Spontaneous infections** (typically brain abscess), b) **Postoperative complications** (commonly meningitis), c) **Post-traumatic complications** (typically meningitis). These mechanisms are associated with different infection sources: a) **Continuous spread of infection** (ENT area - otogenic origin such as otitis media, paranasal sinuses, or dental complications), b) **Infections caused by direct trauma** (presence of cerebrospinal fluid leak or open injuries), and c) **Hematogenous infections** (chronic inflammation such as urinary tract infection or endocarditis).

Clinical manifestations of CNS infections include general symptoms of infectious disease (fever, malaise and fatigue). Depending on the infection site, focal neurological symptoms may develop, such as hemiparesis, speech disturbances, epileptic seizures, or meningeal signs (positive upper and lower meningeal signs, such as neck stiffness, Lasegue's, Kernig's, and Brudzinski's signs). Progressive disease can lead to consciousness impairment, signs of intracranial hypertension, cerebral edema, and death. The proximity of anatomical structures increases the risk of irreversible neurological deficits, intracranial vessel thrombosis, and systemic infectious complications, such as sepsis.

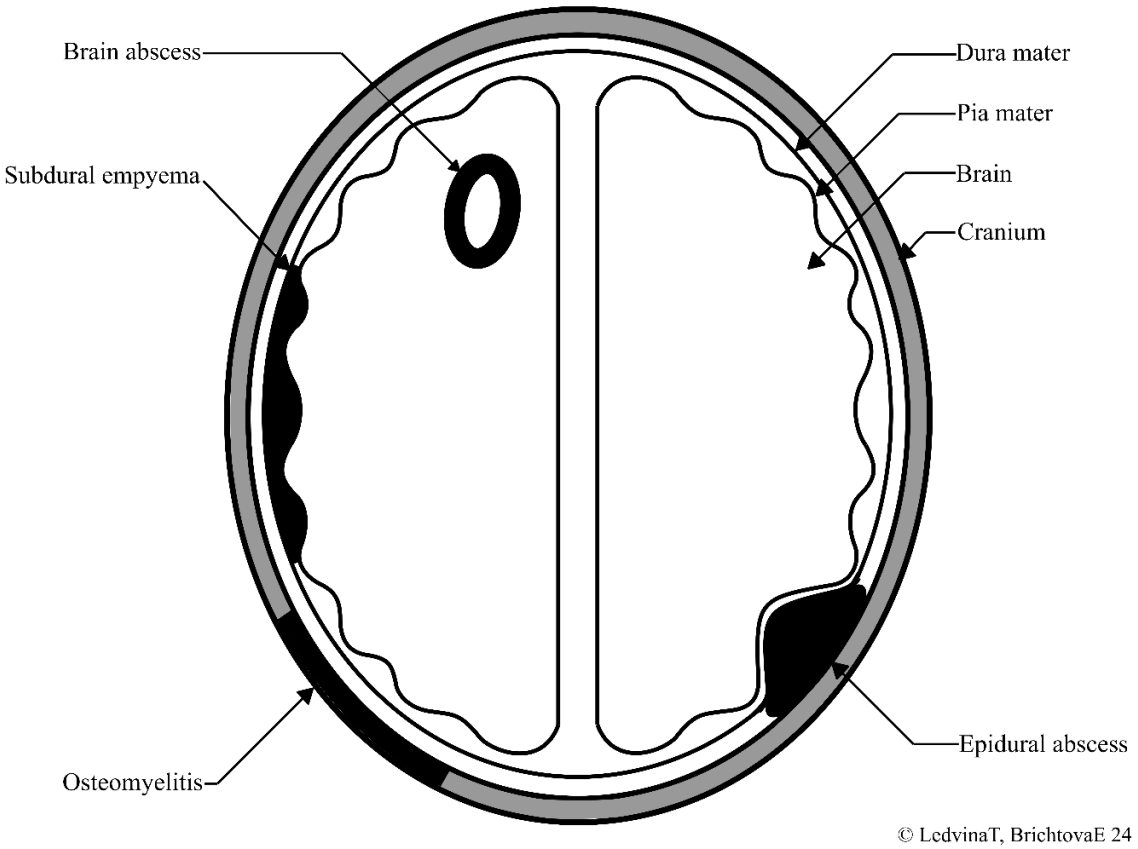
Diagnosis is based on a combination of clinical, paraclinical, and imaging methods. A comprehensive neurological examination is essential, with imaging techniques such as CT and MRI (including contrast enhancement) playing a central role in the initial localization of lesions and their monitoring over time. Laboratory findings typically include elevated inflammatory markers (leukocytosis, C-reactive protein) and an inflammatory profile in lumbar puncture (presence of polymorphonuclear cells, elevated lactate, decreased glucose levels). In many cases, biopsy sampling for microbiological examination is necessary to confirm the diagnosis, allowing targeted antibiotic therapy.

Treatment strategies differ among the specific nosological units and usually involve a combination of neurosurgical intervention (drainage of the abscess via craniotomy or stereotactic puncture; drainage of subdural empyema from craniotomy; surgical revision in postoperative inflammatory complications; extraction of osteomyelitic bone plates; duraplasty in traumatic cerebrospinal fluid leaks) followed by antibiotic therapy (empirically using Ceftriaxone + Metronidazole, with potential adjustments based on microbiological results). Epilepsy prophylaxis with antiepileptic drugs (e.g., Levetiracetam) is also recommended.

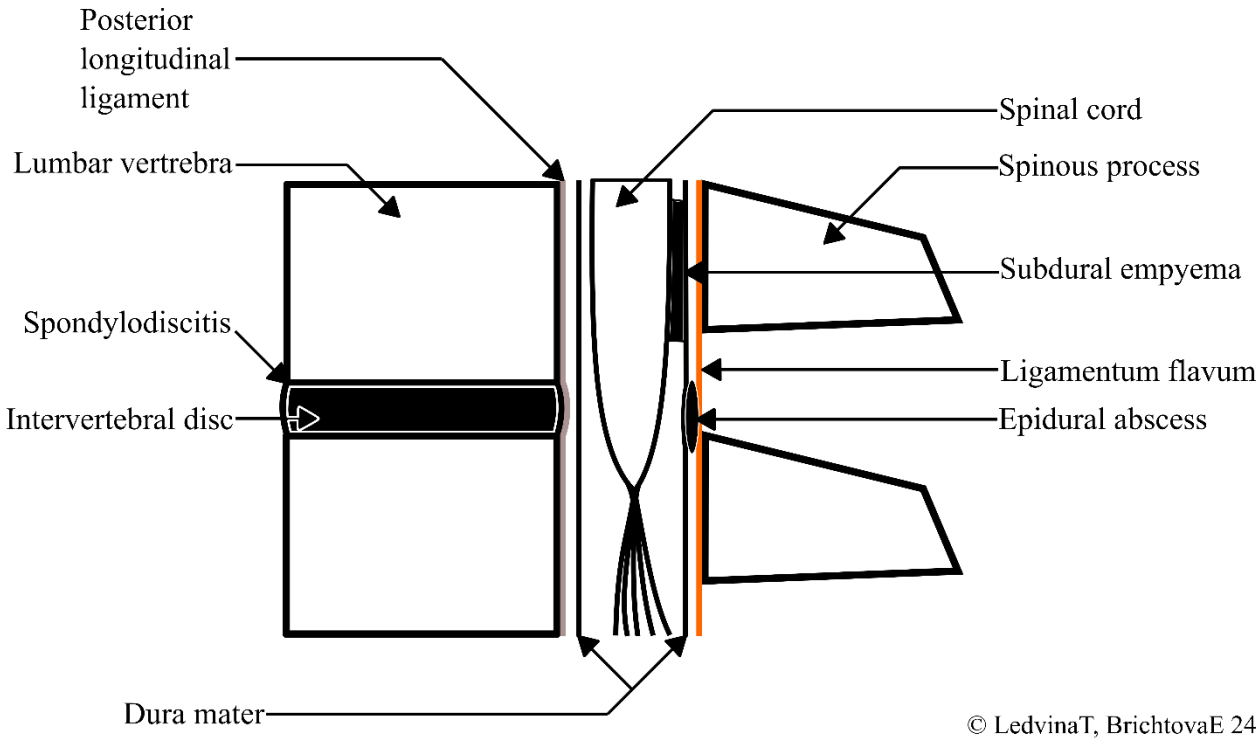
Brain Abscess represents a purulent focal inflammation of brain tissue with direct tissue destruction. The clinical picture depends on the anatomical location and size of the abscess. A distinguishing feature of brain abscesses is the absence of meningeal signs, such as meningeal irritation. Treatment consists of surgical drainage of the abscess (stereotactic puncture, evacuation, and drainage) followed by antibiotic therapy. There is a significant risk of irreversible neurological deficits and septic complications.

Subdural Empyema is a rare infection characterized by the accumulation of pus in the subdural space. Due to the absence of anatomical barriers in the subdural space, there is a high risk of rapid progression. The clinical presentation reflects this risk, often progressing quickly to septic shock. The primary goal of treatment is emergent surgical evacuation and drainage of the subdural space, followed by antibiotic therapy.

BRAIN SUPPURATIVE LESIONS



SPINAL SUPPURATIVE LESIONS



CENTRAL NERVOUS SYSTEM TUMOURS (12)

Barák Martin, Brichtová Eva

Central nervous system (CNS) tumours include those arising from brain tissue (**intra-axial tumours**) and those outside the brain parenchyma (**extra-axial tumours**, e.g., meningeal tumours). These tumours are classified based on their origin into **a) primary CNS tumours** and **b) secondary CNS tumours (metastases)** and by their biological behaviour into **a) benign tumours** and **b) malignant tumours**. A unique aspect of neuro-oncology is that even biologically benign tumours can clinically present similarly to malignant tumours, exhibiting significant clinical symptoms and progressive clinical deterioration over time (limited intracranial space).

CNS tumours constitute approximately **3% of all adult tumours**. In adults, secondary tumours are more common, with CNS metastases observed in 15-30% of patients in the late stages of other systemic cancers. In the **pediatric population**, CNS tumours are **the most common solid malignancy** (20% of all tumours) and the second most common cancer after hematologic malignancies. Unlike adults, CNS tumours in children are typically infratentorial (in the posterior fossa below the tentorium cerebelli), compared to the predominantly supratentorial location seen in adults.

The clinical presentation of CNS tumours is influenced by the pathophysiological principles of an expanding process within the intracranial space. Due to the constraints of the skull, the tumour's growth leads to increased intracranial pressure, resulting in symptoms of intracranial hypertension such as headache, nausea and vomiting, and altered consciousness. Local destruction or compression of surrounding brain tissue by the tumour or infiltration results in specific focal neurological symptoms corresponding to the location of eloquent brain areas.

MRI with contrast is the gold standard for **diagnosis**, while CT with contrast may be useful in acute situations. Histological examination, incorporating both histopathological and molecular-genetic analyses, is crucial for diagnosis and therapeutic planning, as it allows for tumour classification into specific groups and subtypes.

Treatment decisions are based on imaging findings, tumor location (resectable vs. non-resectable due to anatomical constraints), patient history (e.g., prior oncological disease suggesting metastasis), and overall patient status. Surgical resection is generally the cornerstone of treatment for most tumours, aiming for disease remission. In specific cases, an initial diagnostic biopsy may be performed to determine the tumour’s nature. Comprehensive oncological care, including CHT and RT, is provided in the Czech Republic in specialized comprehensive cancer centres.

Pediatric CNS Tumors

Pilocytic Astrocytoma – This is the most common benign CNS tumour in children. It typically grows slowly and is commonly located in the cerebellum. Clinical symptoms arise from its anatomical location, including cerebellar symptoms, hydrocephalus, signs of intracranial hypertension, behavioural disturbances, and failure to thrive in children. The primary goal is radical surgical resection, usually without the need for subsequent oncological treatment. The prognosis for patients is generally favourable.

Pediatric Diffuse Gliomas – This heterogeneous group of tumours varies in biological behaviour. The more aggressive subtypes are often unfavourably located in the brainstem, making them largely unresectable from a surgical perspective. Despite CHRT, the prognosis remains extremely poor.

Ependymal tumours (Ependymomas) are typically benign and occur in the region of the fourth ventricle. Symptoms arise from this typical location, including obstructive hydrocephalus, signs of intracranial hypertension, and cerebellar symptoms. The goal is maximal radical surgical resection, with subsequent oncological therapy considered based on histological findings.

Embryonal tumours include the most common malignant tumour in children - **medulloblastoma**. Medulloblastoma is found in the vermis on the roof of the fourth ventricle. It is an aggressive tumour with often poor prognosis, especially in cases of large tumours, late diagnosis, and in younger patients. There is also a risk of secondary metastasis spreading along the entire craniospinal axis (so-called drop metastases). The clinical course is determined by its anatomical location, manifesting as obstructive hydrocephalus, intracranial hypertension, cerebellar symptoms (ataxia), and in younger children, behavioural changes, irritability, lethargy, and macrocephaly. In the case of metastatic spinal involvement, paraparesis or paraplegia may develop (requires MRI of the entire craniospinal axis). The therapeutic aim is maximal radical resection; however, resection must be limited in cases of brainstem invasion to prevent neurological damage. Treatment also includes comprehensive oncological management, including CHRT, and targeted therapy depending on specific molecular subtypes.

Benign CNS Tumors in adults

Meningiomas are the most common benign brain tumours in adults. These are slow-growing, well-circumscribed tumours that can occur anywhere along the meninges (originating from arachnoid cells). Their incidence increases with age and is more common in women; they are also more frequently seen in patients with a history of head and neck radiotherapy. Clinically, they are usually asymptomatic (an incidental finding on MRI), but further progression may lead to headaches, focal neurological symptoms (depending on the tumour's location), and epileptic seizures. Diagnosis is based on brain MRI, where a direct connection of the tumour to the dura mater is observed.

Symptomatic meningiomas often require surgical resection aimed at tumour removal, including treatment of the adjacent dura mater with the tumour origin.

Vestibular schwannoma is a benign tumour originating from Schwann cells of the vestibulocochlear nerve located in the cerebellopontine angle. Its incidence increases with age. It may occur sporadically, while bilateral vestibular schwannomas are associated with genetic syndromes (neurofibromatosis). Clinical symptoms result from damage to anatomical structures in the cerebellopontine angle—primarily gradually progressive hearing loss and vertigo (both related to the CN VIII), CN VII palsy, and, less frequently, CN V impairment. Large tumours can compress the brainstem, cerebellum, or cerebrospinal fluid pathways, posing a risk of hydrocephalus development. Diagnosis is based on MRI, and audiometry is necessary to assess hearing impairment objectively. The primary treatment for larger tumours is surgical resection, with intraoperative monitoring of cranial nerve VII and VIII functions to prevent neurological damage. Stereotactic radiosurgery is an alternative for smaller tumours.

A pituitary adenoma is a benign tumour located in the sella turcica. These tumours are classified as microadenomas (less than 1 cm) or macroadenomas (greater than 1 cm) and are further categorized based on hormonal activity (most commonly prolactin, ACTH, GH) or inactivity. Clinical symptoms arise from local effects—compression of the optic chiasm (visual disturbances), damage to cranial nerves, headaches, and hormonal manifestations (hyperprolactinemia, acromegaly, panhypopituitarism). Diagnostic evaluation includes imaging studies, endocrine assessment, and visual field testing. Therapeutic management is multimodal, combining pharmacological (dopamine agonists for prolactinomas) and neurosurgical approaches. Progressive or symptomatic tumours are indicated for neurosurgical intervention, which can be approached either classically via craniotomy or via endoscopic transsphenoidal access. Patients require endocrinological follow-up with potential initiation of hormone replacement therapy as needed.

Malignant tumours in adults and secondary tumours

Adult Gliomas (glioblastoma, astrocytoma, oligodendroglioma) comprise a heterogeneous group of the most common primary CNS tumours in adults. They are generally characterized by diffusely infiltrating growth into the surrounding brain tissue.

Glioblastoma is the most common malignant primary CNS tumour, accounting for 50% of malignant primary tumours in adults. Its incidence increases with age. Glioblastoma is known for its rapid progression and extensive infiltration of surrounding brain tissue. Clinically, it presents with headaches, focal neurological deficits, signs of intracranial hypertension, epilepsy, behavioural changes, and impaired consciousness. Diagnosis is based on MRI with contrast. The goal is maximal radical surgical resection followed by CHRT. The extent of resection may be limited by the

proximity to critical anatomical structures and the risk of neurological deficits. If the tumour is inoperable, a diagnostic biopsy is performed. The prognosis remains extremely poor despite comprehensive treatment.

Astrocytomas include subtypes with varying biological behaviour. Unlike glioblastomas, astrocytomas tend to occur in younger patients, typically under the age of 40, and can gradually progress to more aggressive variants. Clinical presentation, diagnosis and treatment are like those for glioblastoma. The prognosis is more favourable compared to glioblastoma.

Oligodendroglioma is a specific subtype arising from oligodendrocytes. It is a slowly growing tumour with a risk of progression to more aggressive forms. Typical clinical manifestations include epilepsy and focal neurological symptoms. The aim of surgical therapy is maximal radical resection followed by oncological treatment.

CNS Lymphomas are a group of haematological malignancies occurring within CNS tissue. Clinical symptoms depend on the tumour's location, often presenting with seizures. The goal of surgical intervention is biopsy confirmation, followed by comprehensive oncological treatment, including CHRT.

Metastases (secondary CNS tumours) are the most common type of brain tumours, accounting for approximately 50% of all brain tumours. There are often multiple cases at the time of diagnosis. Incidence increases with age and with improved diagnostics (MRI) and oncological care. Metastatic spread typically occurs haematogenously to the brain parenchyma or meninges, less frequently via local extension or cerebrospinal fluid pathways. Lung carcinoma is the most common primary site, followed by breast and prostate cancer, melanoma, renal cell carcinoma, and colorectal cancer. Clinical symptoms progress gradually, including headaches, focal neurological deficits, and signs of intracranial hypertension. MRI is crucial in the diagnostic process, as CT lacks sufficient resolution to assess metastatic involvement. The therapeutic management depends on the patient’s overall condition, primary cancer type, and the extent of metastatic spread. Surgical intervention is limited to solitary metastases accessible through a single craniotomy or in cases of multiple metastases where one lesion significantly impacts patient survival (e.g., brainstem compression). In the absence of a known primary malignancy, solitary lesions may be biopsied to establish the primary cancer diagnosis.

LUMBAR DISC HERNIATION, COMPLICATIONS, FAILED BACK SURGERY SYNDROME (19)

Fadrus Pavel, Smrčka Martin

Pathophysiology: From the second decade of life, the vascular supply to the intervertebral disc becomes impaired, first affecting the nucleus pulposus and later the annulus fibrosus. Gradually, degeneration of the annulus fibrosus occurs to the point where a portion of the disc may herniate into the spinal canal. If herniation does not occur, the height of the disc continues to decrease. Eventually, osteochondrosis, uncovertebral neoarthrosis, osteophyte formation, and spondyloarthrosis develop. These processes ultimately lead to the compression of neural structures within the spinal canal. The neurosurgeon's role in treating these patients is primarily to relieve the compression of the neural structures (decompression).

A/ Degenerative Disease of the Cervical Spine

Disc herniations in the cervical spine most commonly occur at the C5/6 and C6/7 levels. Once the neural structures in the cervical spine are compressed, a radicular (nerve root) syndrome or myelopathy develops. These syndromes may occur either in isolation or concurrently.

Radicular syndrome is characterised by irritation—pain radiating into a specific dermatome of the upper limb (cervicobrachial syndrome). It may also present with motor and sensory deficits (weakness, atrophy, reduced reflexes in certain muscles, sensory disturbances). Radicular syndrome develops particularly when the compression is located laterally within the spinal canal.

Myelopathy arises from medial spinal cord compression. The greatest pressure is exerted on the pyramidal tracts, leading to spasticity and pyramidal irritative signs. In advanced myelopathy, there is progressive gait disturbance.

Diagnosis: Plain X-rays may reveal osteophytes on the vertebral bodies and, in some cases, narrowing of the intervertebral foramina. CT scans are even more sensitive to these changes and can demonstrate the relationship between osteoproductive degenerative changes in the cervical spine and the contents of the spinal canal. MRI is essential for diagnosing degenerative disease of the cervical spine. It can reveal disc herniations, the degree of spinal canal stenosis, and changes within the spinal cord (e.g., hyperintense areas on T2-weighted images), indicative of advanced myelopathy. If there is any doubt, electrophysiological testing (e.g., electromyography, evoked potentials) is recommended.

Treatment: Surgery is usually performed through an anterior approach. This allows for decompression of both the dural sac and exiting nerve roots. A cage made of polymer or metal filled with osteoconductive material is inserted into the space of the removed disc, and the segment can be stabilised with a metal plate. The goal of the surgery is to fuse two adjacent vertebrae. Some centres

insert mobile artificial disc replacements. With appropriate indications, surgery is successful in 90% of patients.

In certain cases, surgery is indicated through a posterior approach, especially for lateral disc herniations causing monoradicular involvement of a specific nerve root.

B/ Degenerative Disease of the Lumbosacral Spine

The disease typically begins with pain in the lower lumbar spine, commonly called lumbago. When the integrity of the annulus fibrosus is disrupted, part of the disc herniates into the spinal canal. The disc herniation may remain beneath the intact posterior longitudinal ligament (subligamentous herniation), or a disc fragment may migrate into the spinal canal (epidural sequestration). Disc herniations most commonly occur at the L5/S1 (50%) and L4/5 (45%) levels, with a smaller percentage at L3/4 and even fewer at L2/3.

Clinically, the lumboischiadic syndrome (LIS) is characterised by pain radiating into the corresponding dermatome (irritative symptoms), sensory disturbances in that dermatome, reduced or absent reflexes, and paresis (loss of function symptoms).

Failed Back Surgery Syndrome (FBSS) refers to persistent back or leg pain in patients who have undergone surgery for degenerative spinal disease. One serious complication is **cauda equina syndrome**, which presents with perianogenital hypoaesthesia or anaesthesia and sphincter disturbances, manifesting acutely as urinary retention. This syndrome requires immediate surgery.

Diagnosis of disc herniation is primarily based on MRI, which provides a detailed view of the entire lumbosacral spine. MRI reveals not only disc herniations but also lumbar stenosis or various instabilities of the lumbosacral spine. It is particularly useful in recurrent disc herniations, as it can distinguish scar tissue from a true recurrent herniation (especially after contrast administration).

Treatment of lumboischiadic syndrome is initially conservative, which is effective in up to 70% of patients.

If there is no significant improvement after several weeks of conservative treatment, surgery is indicated. Most disc herniations are operated on through a transligamentous approach (through the yellow ligament). Surgery usually involves a single level, as a herniation of a single disc causes 95% of symptoms. In recent years, endoscopic techniques have been increasingly used, whereby an endoscope is introduced transcutaneously to the herniated disc and the herniated portion is removed through the working channel of the endoscope. This avoids the need for extensive dissection of paravertebral muscles and the removal of larger portions of the lamina and yellow ligament. Two main approaches are used—transforaminal and translaminar, which mirror the traditional microdiscectomy approach.

In strictly indicated cases, minimally invasive, often CT-guided, percutaneous methods may be used to treat lumboischiadic syndrome. These methods are reserved for patients with disc protrusion, causing leg pain primarily in the seated position when disc pressure is highest. The condition requires an intact, non-fragmented disc and no annulus fibrosus rupture. This is confirmed by discography (injecting contrast directly into the disc under CT guidance), where no contrast should escape outside the disc. These methods involve the percutaneous introduction of a needle or electrode into the disc (under CT guidance) and removing the nucleus pulposus through electrical thermocoagulation, laser vaporisation, or chemonucleolysis (chymopapain).

80-90% of patients experience significant relief from lumboischiadic syndrome following disc herniation surgery due to decompression of the nerve root. However, back pain (lumbago) may persist, as surgery does not address the underlying cause of this pain.

In a small number of patients (less than 1%), postoperative discitis (infection of the operated intervertebral space) may occur. Symptoms typically develop 5-7 days postoperatively and include severe lumbar pain without radiation to the legs, low-grade fever, and significantly elevated ESR. MRI confirms the diagnosis. Treatment involves extended bed rest (several weeks) and prolonged administration of anti-staphylococcal antibiotics. The prognosis is usually good.

Lumbar stenosis is a more serious degenerative condition of the lumbosacral spine than simple disc herniation. It primarily affects older patients over 60 years old. Stenosis in these patients develops from long-term degeneration across multiple lumbar segments. Osteoproductive changes (spondylosis, spondyloarthrosis) and often multilevel disc protrusions narrow the canal to just a few millimetres.

Clinically, the condition may present as back pain and radicular involvement of certain nerve roots, but the most common symptom is **neurogenic claudication**. Patients can only walk short distances before stopping and usually find relief in a flexed position. Diagnosis relies on MRI, which shows the degree of canal stenosis (in millimetres) and its extent (number of segments involved). Like lumbar stenosis, treatment is initially conservative. If symptoms worsen, surgery is indicated, focusing on decompression of the neural structures. Surgery is performed with the patient prone under general anaesthesia and involves complete dorsal exposure of the dural sac and exiting nerve roots—laminectomy (removal of vertebral arches and spinous processes). In cases of severe stenosis or laminectomy spanning more than two segments, lumbar stabilisation with transpedicular screw instrumentation and intervertebral disc replacement is often considered. Most of these procedures are performed through a posterior approach, using techniques such as PLIF (posterior lumbar interbody fusion), TLIF (transforaminal lumbar interbody fusion), and XLIF (extreme lateral interbody fusion).

An alternative is the anterior retroperitoneal approach, ALIF (anterior lumbar interbody fusion). These operations typically result in significant pain relief and improved walking ability.

Some patients with degenerative lumbosacral spine disease develop significant spondyloarthritis with pronounced instability between the vertebrae of the lower lumbar spine—**spondylolisthesis**. These patients primarily experience back pain, which is often worse when walking. Typical radicular symptoms may also be present. Diagnosis involves functional X-rays, CT, and MRI. If conservative treatment and rehabilitation fail, surgery is indicated. This involves decompression of the dural sac, disc replacement with a cage, reduction of vertebral slippage, and transpedicular fixation of the adjacent vertebrae in the affected spine segment.

PEPIPHERAL NERVE INJURY (20)

Fadrus Pavel, Smrčka Martin

Anatomical Notes: A peripheral nerve's basic morphological and functional unit is the neuron. It consists of the nerve cell body, peripheral nerve fibre (axon), and terminal branching, which ends at an effector (in the case of motor fibres) or begins at a receptor (in the case of sensory fibres). The bodies of the neurons forming peripheral nerves are located either in the spinal cord or in the spinal ganglia. The axons form the basic functional unit of the peripheral nerve. Axons are covered by a myelin sheath, which is produced by Schwann cells in the peripheral nervous system. The interruptions in the myelin sheath are called Nodes of Ranvier. Individual nerve fibres are covered by a layer of reticular and collagen fibres called the endoneurium. Multiple nerve fibres form bundles called fascicles, which are covered by the perineurium. Several fascicles combine to form the peripheral nerve, and its outer covering is called the epineurium.

Pathophysiology of Nerve Injuries: The neuron in the anterior horn of the spinal cord or in the spinal ganglia, together with its peripheral fibres and the motor end plate or receptor, forms a functional unit. After the axon is severed, distal to the site of injury, it undergoes degradation known as Wallerian degeneration.

Proximally to the site of axonal transection (the so-called central or proximal stump), Wallerian degeneration begins. From the end of the Node of Ranvier, fibrils grow from the axon and are directed into the Büngner bands. Suppose the nerve ends do not reconnect; an amputation neuroma forms at the proximal stump. This occurs through the proliferation of connective tissue from the epineurium, into which the proximal axons grow, eventually encased by fibrous tissue. Axons regenerate at a rate of 1 mm per day, with a delay of 4-6 weeks at the suture site.

Classification of Nerve Injuries

The simplest classification is the Seddon classification, which categorises injuries from least to most severe:

1. **Neurapraxia**: A reversible injury where the axons are not severed. The impairment is functional, comparable to a concussion in the central nervous system. Recovery typically occurs within six weeks, resulting in full restoration of nerve function.
2. **Axonotmesis**: An injury where the axons are severed, but the endoneurial tubes remain intact. Wallerian degeneration occurs distally, but the macroscopic structure of the nerve is preserved. Regeneration occurs spontaneously within six months.
3. **Neurotmesis**: A complete anatomical severing of the nerve. Wallerian degeneration ensues, and spontaneous regeneration is not possible without surgical intervention.

Diagnosis: Diagnosing a peripheral nerve injury involves taking a thorough history (mechanism of injury) and performing a detailed clinical examination. Electrophysiological testing is not indicated in the acute phase and is essential in delayed surgeries. Electromyography (EMG) helps monitor the progress of regeneration. Evidence of distal nerve conduction supports a conservative approach (e.g., external neurolysis, the release of scar tissue around the nerve). At the same time, the absence of potentials necessitates aggressive resection of the neuroma with defect repair using grafts.

Surgical Technique: Successful surgery requires microsurgical techniques. Fine, atraumatic sutures such as Ethilon 8-10/0 are used, and tension-free suturing is crucial. If a nerve defect is present, it is bridged using autografts, typically the sensory sural nerve. The graft acts as a conduit for neurofibril growth during Wallerian degeneration.

Brachial Plexus Injury (C5-Th1)

Brachial plexus injuries are among the most severe peripheral nerve injuries. They are classified as supraclavicular and infraclavicular injuries.

EMG, nerve action potentials (NAP), CT myelography, and MRI are useful in diagnosing brachial plexus injuries.

Conservative management is indicated if clinical and laboratory evidence (EMG) shows improvement. Surgery is indicated for open injuries and closed injuries where there is no improvement. Infraclavicular injuries have a better prognosis than supraclavicular ones.

Radial Nerve Injury (C5-8)

This is the largest nerve in the upper limb, responsible for motor control of the elbow, wrist, finger extension, thumb extension, and abduction.

Surgery: In relatively rare open injuries, acute revision is clearly indicated. Indications are more challenging in radial nerve palsy associated with humeral fractures. It is often difficult to determine whether paralysis occurred immediately after the injury (due to traction and nerve contusion) or as a result of iatrogenic injury during surgery. The first scenario typically resolves spontaneously (axonotmesis), whereas the second requires revision. Delayed surgery should be supported by intraoperative electrophysiological testing.

Ulnar Nerve Injury (C8-Th1)

The ulnar nerve is a mixed nerve, essential for fine finger movements.

Treatment: Open injuries require urgent revision. Injuries to the nerve at the wrist are often associated with ulnar artery transection, and it is advisable to treat the nerve while performing an arterial anastomosis. Ligating the artery can impair the regeneration of the ulnar nerve due to ischemia.

Median Nerve Injury (C5-Th1)

The median nerve is a mixed nerve that branches in the forearm. Median nerve injuries are typically lacerations at the wrist or forearm. Motor impairment includes difficulty with volar abduction of the thumb, and a positive "grinding test" may occur when the thumb cannot rotate around the other fingers. Patients often experience sensory disturbances, causalgia, and trophic changes in the skin, which become cyanotic and atrophic. Neuroma pain is intense, and patients tend to avoid using the affected hand.

Treatment: In open injuries (typically at the wrist), surgical treatment is required. In closed injuries, treatment is individualised.

Compression Syndromes

These syndromes occur when there is a mismatch between the volume of the peripheral nerve and the anatomical space it occupies. Many compression syndromes are described, but only the most clinically significant are mentioned here due to their high prevalence.

Carpal Tunnel Syndrome (CTS)

The carpal tunnel is bordered on the volar site (vertically) by the transverse carpal ligament and dorsolaterally by the carpal bones.

Treatment: For mild symptoms, conservative treatment may be attempted. This involves addressing the underlying condition, reducing stress on the limb, immobilisation (night splints), physiotherapy (mobilisation of carpal bones), and anti-inflammatory medication, either orally or through local injections.

Surgical treatment involves dividing the transverse carpal ligament under direct vision or endoscopically. This procedure is usually performed on an outpatient basis under local anaesthesia. Nerve decompression is achieved by dissecting the entire length of the transverse carpal ligament. The most common postoperative complication is insufficient symptom relief, usually due to incomplete division of the ligament, which results in residual syndrome rather than a recurrence. Occasionally, injury to the palmar cutaneous branch occurs, causing pain in the base of the thenar area. Anomalous courses of the recurrent branch of the median nerve must be considered. Direct injury to the median nerve is rare.

A rare but serious postoperative complication is complex regional pain syndrome (CRPS), characterised by motor, sensory, and autonomic disturbances that can lead to severely impaired mobility and necrosis of the skin in the affected area, accompanied by intense pain.

Cubital Tunnel Syndrome

The ulnar nerve is compressed at the elbow as it passes behind the medial epicondyle, where it lies superficially and is vulnerable to trauma. The condition initially presents with paraesthesias in the fourth and fifth fingers, which worsen with elbow flexion.

Treatment: Conservative management involves immobilisation in a splint and limiting activity. Surgical options include simple nerve decompression (sometimes with resection of part of the medial epicondyle of the humerus) or transposition of the ulnar nerve (less commonly). For post-traumatic deformities, medial epicondylectomy may be added. Long-term studies have not shown a significant benefit of transposition over simple decompression. Intramuscular transposition is indicated in cases of reoperation for progressive neurological symptoms.

Overall, surgical outcomes for cubital tunnel syndrome are somewhat less favourable than for carpal tunnel syndrome.

Compression of the Ulnar Nerve in Guyon's Canal

Guyon's canal is bordered volarly by the aponeurosis and dorsolaterally by the pisiform and hamate bones. The ulnar nerve divides here into motor and sensory branches. Differentiation from more proximal compression is confirmed by the preserved function of the flexor digitorum profundus and normal sensation on the dorsal ulnar side of the hand. The site of compression is confirmed by electrophysiological testing (EMG).

Treatment: After conservative options are exhausted, surgical decompression involves dissecting the aponeurosis.

CARDIOTHORACIC SURGERY AND TRANSPLANTATION

CONGENITAL ANOMALIES OF THE HEART AND GREAT VESSELS (21)

Fila Petr, Němec Petr

Congenital heart disease (CHD) affects approximately 0.6-0.8% of newborns.

Basic classification (only the most common defects are listed)

1. defects without shunt (congenital aortic valve stenosis, coarctation of the aorta), defects with left to right shunt (patent ductus arteriosus, ventricular septal defect, atrial septal defect) and defects with right to left shunt (tetralogy of Fallot, transposition of the great arteries).
2. Cyanotic and non-cyanotic defects
3. Critical and other defects

Defects without shunt

Congenital stenosis of the aortic valve:

Depending on the location, this may be subvalvular, valvar (most commonly bicuspid) or supravalvar aortic stenosis (AS). The narrowing causes pressure overload in the left ventricle.

The defect may be clinically silent for a long time, but may later present with dyspnoea on exertion or arrhythmias. Less commonly, it may be a critical stenosis requiring urgent treatment.

Treatment: Critical stenosis requires urgent treatment (balloon dilatation), where a balloon is inserted through the inside of the artery into the narrowed valve and inflated to dilate the valve. The planned surgical solution consists of incision of the fused valve cusps, reconstruction of the valve (valvular AS), resection of the connective membrane or muscular part of the septum (subvalvular AS), or dilatation of the ascending aorta with a patch (supravalvular AS). Definitive treatment of aortic stenosis can be achieved by replacing the aortic valve with an artificial valve.

Coarctation of the aorta

Coarctation of the aorta is the presence of a significant narrowing at the end of the arch, most commonly behind the left subclavian artery. It may be found in isolation or in combination with various CHD´s.

A small coarctation may not be clinically manifest and is sometimes an incidental finding in adulthood. In front of the narrowing there is high blood pressure in the aorta (increased blood pressure in the vessels leading to the head increases the risk of haemorrhagic stroke, epistaxis is typical). In contrast, the pressure behind the stenosis is low. There is a weak to non-palpable pulse in the lower extremities.

An echocardiogram of the heart may show congestion of the left half of the heart, enlargement of the heart, and sometimes the narrowing of the aorta itself. CT angiography or MRI angiography of the aorta is useful for confirmation.

Treatment is predominantly surgical (resection of the affected segment via a left-sided thoracotomy and end-to-end suture (Fig. 1). In neonates or older patients, catheter-based treatment is also possible - balloon angioplasty or stent or stent graft implantation.

Left-to-right shunt diseases

Patent ductus arteriosus (PDA)

During fetal development, this connection between the aorta and the pulmonary artery allows blood to bypass the lungs. In healthy newborns, it closes spontaneously during the first 24 to 96 hours of life. However, in some children, especially those born prematurely, it remains open and causes failure to thrive in a milder form and signs of heart failure in a more severe form. Treatment is mainly conservative (indomethacin, ibuprofen). However, if this is not successful, the PDA must be closed surgically, either by ligation or resection.

Atrial septal defect (ASD)

In ASD, the septum that separates the atria is not fully formed. This causes shunting from left to right and can lead to congestion of the right side of the heart.

Increased blood flow through the pulmonary circulation can result in increased pulmonary artery pressure and even the development of pulmonary fibrosis. In the terminal phase, Eisenmenger's syndrome may occur when the peripheral resistance of the pulmonary circulation is already so high that the pressures in both atria equalise and the shunt changes to left-to-right. In such patients, repair of the atrial septal defect is no longer contraindicated because of the risk of right ventricular failure.

Depending on the localisation of the defect, there are two basic types:

1. Ostium primum defect - localised above the atrioventricular valve and often associated with a mitral valve defect.

2. Ostium secundum defect - is more common and can be located anywhere at the level of the atrial septum.

Small openings in the atrial septum may be asymptomatic (detected by auscultation due to the presence of a murmur) and manifest with increased dyspnoea or arrhythmias in adolescence or adulthood.

The first manifestation may also be what is known as paradoxical embolism. This occurs when a thrombus from the deep venous system of the lower limbs passes through the orifice into the left atrium

and from there into the left ventricle and the systemic circulation as a result of increased pressure in the right-sided compartments (Valsalva manoeuvre).

Treatment of the defect consists of closing the defect with a suture (in the case of a small defect) or with a patch of autologous or bovine pericardium. Recently, if the localisation is favourable, the defect can be closed by catheter with an occluder (Fig. 2).

The atrial septal defect of the primum type is divided into incomplete and complete, with several intermediate stages in between. In a complete defect, the adjacent part of the ventricular septum is missing and the mitral or even the tricuspid valve is developmentally damaged. This type of defect is diagnosed in infancy and treatment is surgical and consists of complex reconstruction - closure of the defect between the atria and ventricles and reconstruction of the damaged mitral and tricuspid valves.

Ventricular septal defect (VSD)

VSD is the most common congenital heart disease in which there is a defect in the septum that separates the chambers of the heart, causing a shunt from left-to-right. It can be an isolated or a part of a complex heart malformation. It can be located in the area between the membranous and muscular parts of the septum (perimembranous defect) or in the muscular part of the septum (muscular defect).

VSD may be asymptomatic and is diagnosed by a systolic murmur on auscultation, or it may cause decreased exercise capacity or dyspnoea on exertion. The diagnosis is confirmed by echocardiography.

Treatment: for very small defects without significant clinical signs, the child can be followed regularly as the defect may close spontaneously, especially in the muscular region. Larger defects that cause clinical symptoms need to be closed surgically, usually with a pericardial or dacron patch. As the edge of the defect is crossed by the conduction system, there is a risk of grade III a-v block. Exceptionally, the defect can be closed by catheterisation with an occluder.

Right-to-left shunt diseases

Tetralogy of Fallot (F4)

F4 is an example of a complex cyanotic heart defect involving a VSD, aortic seeding over the defect and stenosis of the pulmonary artery causing right ventricular hypertrophy.

Symptoms: The presence of a high proportion of deoxygenated blood in the systemic circulation causes cyanosis, particularly of the face, lips, mucous membranes and fingers (club fingers). There may also be fatigue and mild dyspnoea, and sudden attacks of shortness of breath with pallor and loss of consciousness.

This needs to be treated by surgery. In the past, a shunt was created between the pulmonary and systemic circulations to increase the flow through the lungs in the critical state, thereby increasing the

amount of oxygenated blood. The current approach is to perform what is called a radical correction, i.e. a procedure that leads to normalisation of the anatomical and functional conditions in the heart. The VSD is closed and the stenosis of the pulmonary artery is removed either by a direct procedure on the pulmonary artery or by a patch on the outflow tract of the right ventricle. This significantly improves the prognosis. However, patients need to be followed up by a cardiologist after surgery, as about 25% of patients need reoperation in adulthood. The most common reason is pulmonary insufficiency, in which case the pulmonary valve must be a biological prosthesis or homograft. In appropriate morphological conditions, transcatheter valve implantation is also possible.

Transposition of great arteries - uncorrected:

Transposition of the great arteries is another serious cyanotic congenital heart defect where the branches of the great arteries from the ventricles are switched. The aorta is transposed from the right ventricle and the pulmonary artery is transposed from the left ventricle. In this defect, blood collects from the body into the superior and inferior vena cava, goes through the right atrium into the right ventricle, into the aorta and throughout the body. From the lungs, blood flows through the pulmonary veins into the left atrium, through the mitral valve into the left ventricle, into the pulmonary artery, and again into the lungs. This creates two independent circuits. This would not be a condition compatible with life, but usually other shunt defects are present that allow at least partial mixing of the blood (ASD, VSD or PDA). If none of these defects are present, the shunt must be artificially created immediately (a catheter is used to create a defect at the level of the atria).

Surgical treatment previously consisted of correction at the atrial level, i.e. the right ventricle became a systemic ventricle. However, this caused problems in the long term, so now the correction is performed at the level of the great arteries, i.e. the aorta (including the coronary arteries) is repositioned over the left ventricle and the pulmonary artery over the right. This creates a physiological circulation.

Transposition of the great arteries - corrected:

This type of defect is relatively rare and both the great arteries and the ventricles are transposed. Blood from the left atrium flows into the right ventricle and aorta, and blood from the right atrium flows into the left ventricle and pulmonary artery. In this case, we are not talking about the right and left ventricles, but the systemic and pulmonary ventricles. The right ventricle does not have an evolved wall to cope with the work of the systemic ventricle in the long term, so dilatation, atrioventricular valve insufficiency and heart failure gradually develop. Surgical treatment consists of replacement of the insufficient valve or, in later stages, heart transplantation.

Treatment: transposition of the great arteries is corrected as early as possible in the neonatal period by complex cardiac surgery aimed at normalising the anatomical conditions.

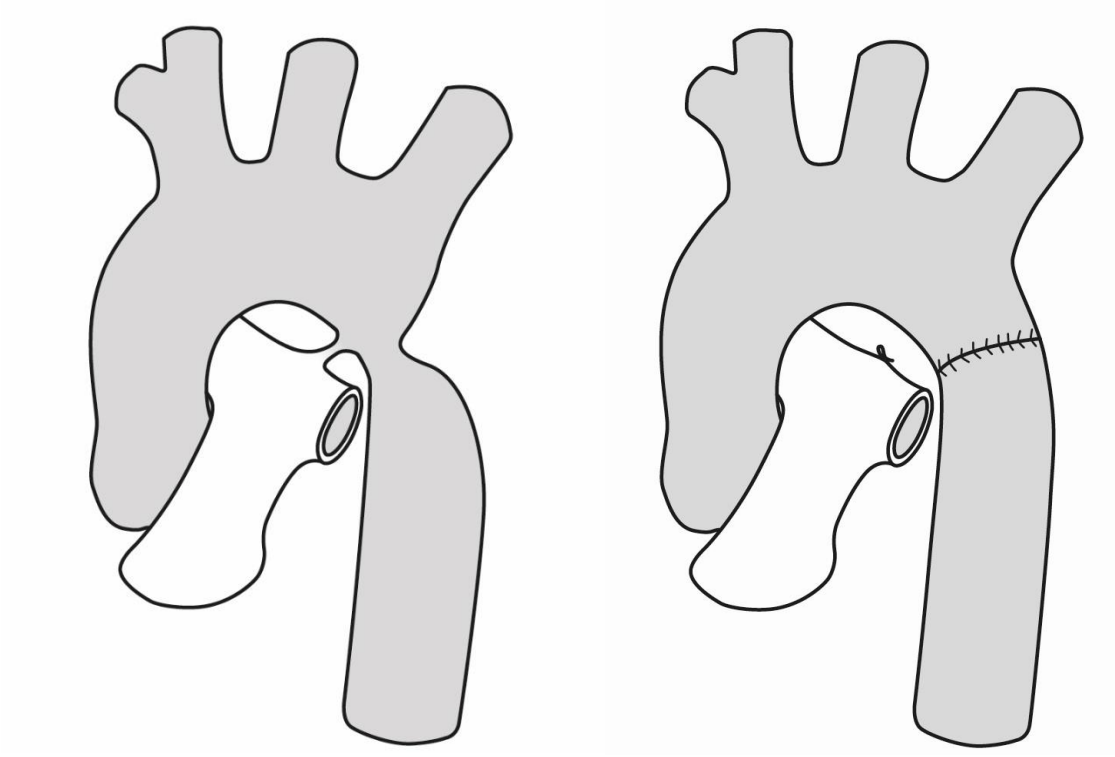


Fig. 1 Coarctation of the aorta and end-to-end anastomosis after correction

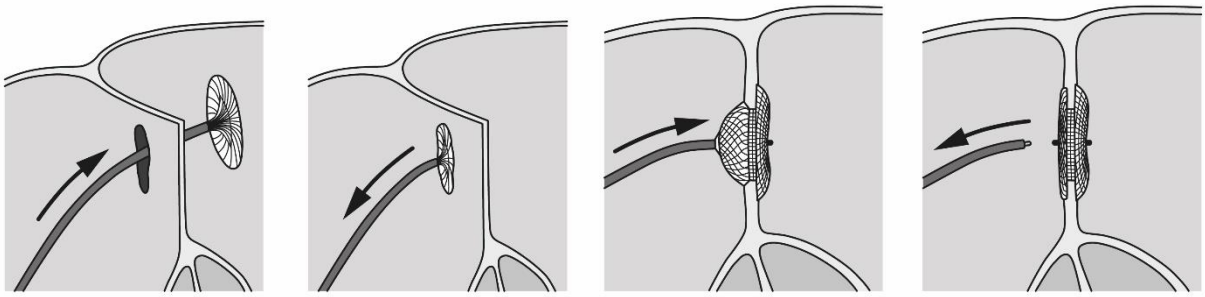


Fig. 2 Closing of the ASD by catheter with an occluder

SURGICAL TREATMENT OF HEART VALVE DISEASES (22)

Fila Petr, Němec Petr

Heart valves play a key role in maintaining unidirectional blood flow during the cardiac cycle. Their dysfunction leads to impaired haemodynamics, which can result in heart failure (HF), atrial fibrillation, pulmonary hypertension and other complications.

The valve is not only made up of leaflets from a functional point of view. In the aortic valve, the functional parts are the cusps, the anulus, the aortic root and the sinotubular junction (where the aortic root joins the tubular part of the ascending aorta). In the mitral valve, the cusps attach to the anulus, and the cusps are connected by chordae tendinae that branch from the papillary muscles, which attach to the wall of the left ventricle (LV). Pathology in any of the functional parts of the valve can cause valvular regurgitation, and an understanding of the functional parts is also important for the correct surgical management of valvular regurgitation, especially when the goal is to repair the valve.

The prevalence of valvular heart disease is estimated at 2-3% of the population and increases with age.

Pathophysiology of valvular disease

Valvular disease can be divided into two main categories: stenosis and insufficiency.

- *stenosis* is characterised by a narrowing of the valve orifice, resulting in an increased pressure gradient and restricted blood flow.
- *insufficiency* is a condition in which the valve does not close sufficiently, causing blood to flow backwards. In the atrioventricular valves (mitral and tricuspid), a distinction is made between primary insufficiency (involvement of the tips, tendons, papillary muscles) and secondary insufficiency (dilatation of the anulus and left or right ventricle).

Often, however, these defects are combined (stenoinsufficiency) with a possible predominance of one or the other component.

Heart valve replacement

The primary goal of surgical treatment is to repair the valve. If reconstruction (valve repair) is not possible, the solution is valve replacement. There are two basic types of valve replacement:

- *mechanical prosthesis* - require lifelong anticoagulation, have unlimited durability but higher rates of thromboembolic and haemorrhagic complications, more suitable for younger patients
- *biological prosthesis* - do not require lifelong anticoagulation, have limited durability, preferable for older patients

Aortic valve stenosis

The narrowing of the opening of the aortic valve restricts blood flow from the LV to the aorta and then to the systemic circulation, increasing the workload on the LV, which must exert more pressure to overcome the resistance of the valve. This can lead to LV hypertrophy and eventually LV dysfunction. It affects 3-5% of people over the age of 65. The incidence increases with age, with degenerative calcification of the aortic valve being the dominant cause. Severe stenosis is defined by an orifice area of less than 1.0 cm^2 and a maximum gradient above 40 mmHg.

Etiology

- *degenerative calcification* - in older patients, calcium deposits accumulate on the valve, reducing its mobility and functionality.
- *congenital* - bicuspid or unicuspid aortic valve (presence of two/one cusps instead of three) leads to early development of stenosis.
- *postrheumatic fever* - streptococcal infection that damages the valves and causes thickening and adhesions of the valve cusps, rare due to availability of ATB treatment.

Symptoms: shortness of breath, angina, syncope

Diagnosis: auscultatory systolic murmur over the aorta, ECG with signs of LV hypertrophy, echocardiography, catheterisation to evaluate the gradient between the LV and the aorta.

Treatment

Surgical valve replacement: standard for symptomatic patients or patients with severe stenosis, mechanical or biological valve. Selected patients may be offered the Ross procedure, in which the patient's pulmonary valve is used to replace the aortic valve. The pulmonary valve is then replaced with an allograft from a tissue bank (Fig 1). This procedure is mainly indicated in young patients with aortic valve dysfunction, where the long-term durability of the valve and the absence of the need for anticoagulation are an advantage.

Transcatheter aortic valve replacement (TAVI): catheter-based valve implantation (on beating heart, without extracorporeal circulation) for patients over 75 years of age or for polymorbid patients at high surgical risk.

Aortic valve insufficiency

Aortic insufficiency (AI), or aortic regurgitation, blood volume flows backwards into the LV leads to increased LV volume and congestion. Over time, dilation and hypertrophy of the LV may occur, initially maintaining cardiac output but later leading to decompensation and HF.

Etiology: congenital bicuspid/unicuspid valves, degenerative changes of the leaflets, dilatation of the annulus, root or sinotubular junction, infective endocarditis or, rarely post-rheumatic involvement.

Symptoms: in the early stages the patient may be asymptomatic. Gradually patients may experience shortness of breath on exertion, fatigue, palpitations and signs of HF. Sometimes acute AI can occur, often associated with severe dyspnoea, hypotension and signs of cardiogenic shock. Acute AI often follows aortic dissection or endocarditis and requires urgent surgical intervention.

Diagnosis: auscultation of the heart may reveal a diastolic murmur in the third or fourth intercostal space to the left of the sternum. Echocardiography is the basic diagnostic method. Magnetic resonance imaging may be used to assess regurgitant volumes.

Surgical treatment: in severe symptomatic regurgitation or in patients with left ventricular dilatation, surgical reconstruction (aortic valve repair) of the aortic valve is method of choice. In the case of dilatation of the cusp, root or ascending aorta, aortic valve replacement surgery is performed, including the David and Yacoub operations (aortic valve-sparing operations). Surgical replacement is the solution if the valve cannot be preserved (repaired).

Mitral stenosis

is mainly a consequence of rheumatic fever, when the mitral orifice is narrowed by calcification and fibrosis. The non-rheumatic cause of mitral stenosis is degenerative calcification of the valve. Clinically severe mitral stenosis has a valve area of less than 1.5 cm^2, with symptoms such as dyspnoea on exertion, pulmonary hypertension and HF occurring at values below 1.0 cm^2. Mitral stenosis increases left atrial pressure, leading to subsequent atrial fibrillation and thromboembolic complications.

The most common surgical treatment for mitral stenosis is replacement with a mechanical or biological valve.

Mitral insufficiency

also known as mitral regurgitation (MR), resulting in a backward flow of blood from the LV to the left atrium during systole. This backward blood flow causes congestion in the left atrium and LV, which can lead to the development of HF, pulmonary hypertension and other complications.

Etiology: prolapse of the mitral valve cusp (degenerative changes), chordal rupture, post-infarction papillary muscle rupture, infective endocarditis, post-rheumatic involvement - primary. LV dilatation with anulus dilatation or even a change in LV geometry (dilatation) and subsequent traction on the papillary muscles leads to MR - secondary.

Correct determination of the cause of MR in relation to the functional parts of the valve is a prerequisite for correct treatment (repair) of the valve.

Symptoms: shortness of breath, fatigue, palpitations or progressive signs of HF. In acute MR - cardiogenic shock, pulmonary oedema

Diagnosis: Auscultation of a holosystolic murmur over the apex, echocardiography (to assess the severity and cause of regurgitation, and to assess the size and function of the left atrium and LV).
Treatment: the principle of surgical treatment is primarily reconstruction (repair) of the valve. The prolapsed part of the tip can be resected, the ruptured/elongated chord can be replaced with a neochord. An annuloplasty with implantation of a mitral ring is almost always performed. A mitral valve that cannot be repaired is replaced with a mechanical or biological valve.
For patients at high surgical risk, less invasive mitral valve procedures (transapical implantation of the valve in the mitral position, on a beating heart) or the more common catheter-based implantation of a clip that anchors the anterior and posterior cusps (i.e. MitraClip), creating a double orifice mitral valve, have recently emerged.

Tricuspid stenosis

This is a rare disease of the tricuspid valve. It is caused by post-rheumatic disease, endocarditis, tumours or carcinoid syndrome. The solution is usually replacement with a bioprosthesis.

Tricuspid insufficiency

Primary tricuspid regurgitation (TR) results from involvement of the cusps, chords or papillary muscles. Secondary TR is more common with dilatation of the tricuspid annulus. Clinically significant TR leads to the development of right-sided HF, oedema and ascites. Surgical treatment consists of implantation of an annuloplasty tricuspid ring to correct the dilatation of the annulus.
Pulmonary valve defects are rare. They are mostly associated with congenital heart disease.

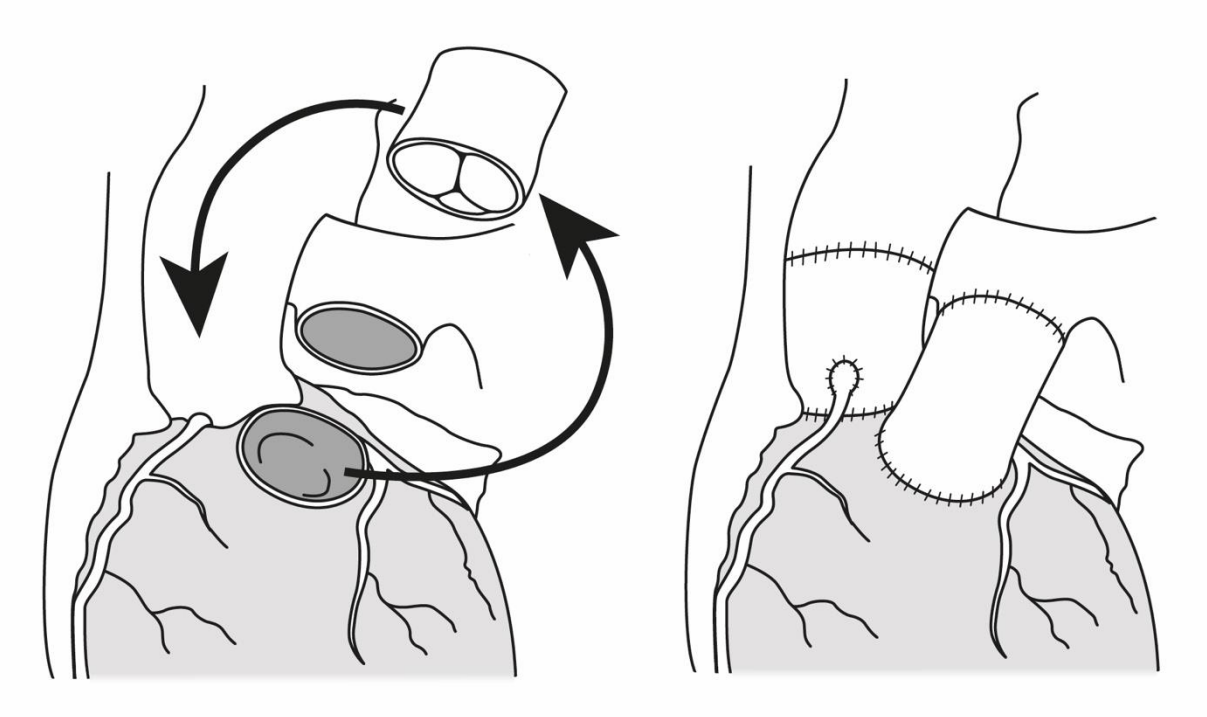


Fig. 1 Ross procedure - diseased aortic valve is replaced with the patient's own pulmonary valve. This is followed by the replacement of the pulmonary valve with a pulmonary allograft from tissue bank.

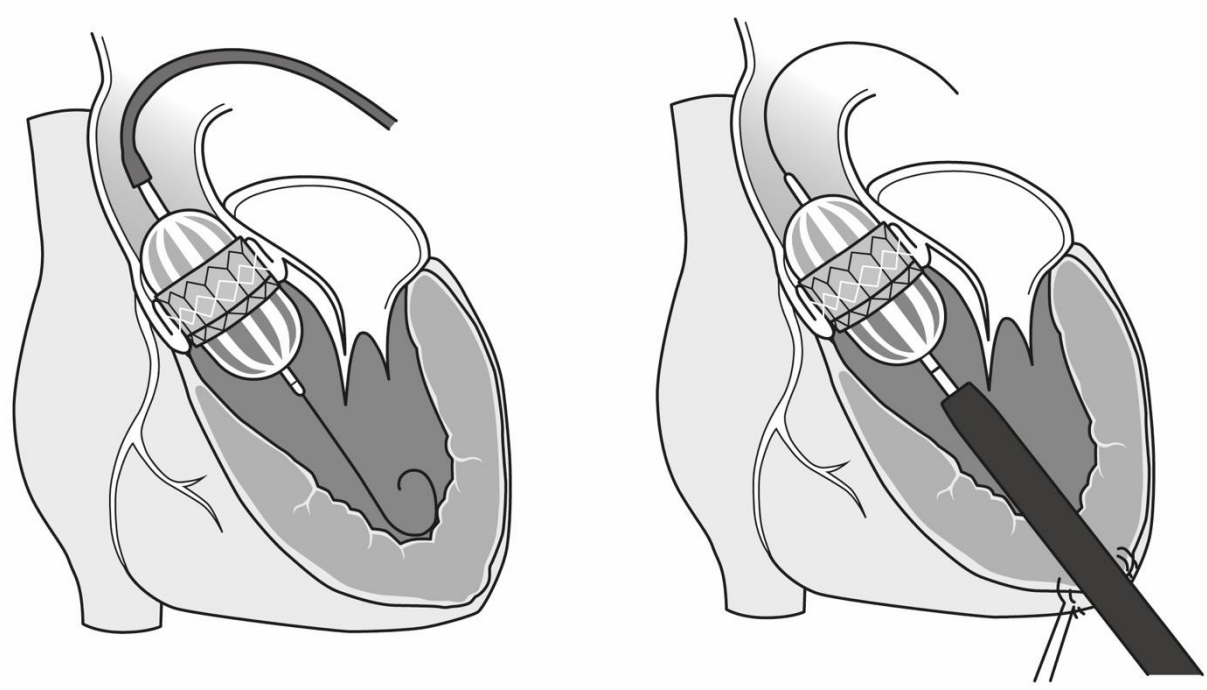


Fig. 2 Transcatheter aortic valve replacement (TAVI) – transfemoral or transapical access

SURGICAL TREATMENT FOR CORONARY ARTERY DISEASE (23)

Fila Petr, Němec Petr

Introduction

Ischaemic heart disease (IHD) is a disorder in which there is an imbalance between the demand for oxygen and the supply to the myocardium. The most common cause of IHD is atherosclerosis. Acute and chronic forms of IHD are distinguished.

Besides preventing IHD in patients, we use conservative medical treatment, percutaneous coronary intervention (PCI) and surgery.

One or more coronary artery bypass graft (CABG) are used in the surgical treatment of IHD. The principle of coronary artery bypass grafting is to bridge the affected coronary artery using an arterial or venous graft. The peripheral anastomosis of the bypass graft is placed on the coronary artery distal to the affected coronary artery (stenosis, occlusion). For venous graft, the central anastomosis is placed on the ascending aorta. In arterial bypasses, the central end is left in situ or anastomosed to the ascending aorta or connected to another bypass.

Indication for CABG

Patients with severe coronary artery disease are referred for surgical revascularization. Most commonly:

- left main disease
- two-vessel disease with stenosis or occlusion of the proximal segment of the left anterior descending artery (LAD)
- three-vessel disease (i.e. involvement of the LAD, circumflex artery and right coronary artery)

In patients with acute coronary syndrome, surgical revascularisation is preffered when percutaneous coronary intervention (PCI) was not technically feasible or in the presence of mechanical complications of myocardial infarction. In addition to coronary involvement, the general condition of the patient (age, comorbidities) must be taken under consideration in the decision-making process.

Grafts (conduits) for revascularisation

Arterial grafts: internal thoracic artery (ITA), radial artery (RA), gastroepiploic artery, inferior epigastric artery.

Venous grafts: great saphenous vein (GSV), small saphenous vein.

Arterial conduits have better long-term patency due to their wall structure (more than 90% of bypasses remain patent after 10 years). In venous conduits, neointimal hyperplasia develops after a

few years and can lead to stenosis or occlusion of the bypass (about 50% of bypasses remain patent after 10 years).

Harvesting conduits for revascularisation

Arterial conduits are harvested either as free grafts (e.g. right-sided ITA, RA) or left in-situ. The most commonly used left in-situ arterial graft is the left-sided ITA. Centrally, its origin from the subclavian artery is preserved, while the peripheral end of the artery is cut at the lower edge of the sternum and sutured to the coronary artery, usually the LAD.

The radial artery is harvested as a free graft, usually from the non-dominant limb. Prior to harvesting the RA, a thorough assessment of the arteries of the forearm (Allen's test, ultrasound examination) should be performed to ensure a good arterial supply to the hand via ulnar artery.

The right gastroepiploic and inferior epigastric arteries are rarely used as grafts.

Lower extremity vein grafts can be harvested openly from one or more skin incisions or endoscopically. Endoscopic harvesting of GSVs is the minimally invasive technique, which has the advantage of less postoperative pain, faster recovery and better cosmetic effect. Patients with obesity, diabetes or chronic lower limb ischaemia may benefit from this technique.

Types of procedures in the surgical revascularisation of the heart

CABG using extracorporeal circulation (ECC): during surgery, the patient is connected to an ECC and cardioplegic cardiac arrest is induced. Distal vascular graft anastomoses are constructed on the arrested heart and central anastomoses are sutured to the ascending aorta once the aortic cross-clamp is released and hearts starts to beat. After weaning from the ECC, the cannulas through which the ECC was connected are removed. The advantage of this technique is a stable and bloodless surgical field. The disadvantages are an increased risk of embolism due to manipulation of the aorta and a higher inflammatory response of the body to blood contact with the artificial surfaces of the ECC.

CABG without the use of ECC (off-pump CABG) is performed on a beating heart. During suturing of distal anastomoses, this area of the heart must be partially stabilised with various devices. Once the artery is opened, an intracoronary shunt is placed in the lumen to control bleeding while maintaining peripheral blood supply to the myocardial tissue. This type of operation is particularly important in high-risk patients after a stroke or in patients with renal failure. The disadvantage is that it is more technically challenging.

Minimally invasive CABG: This is a gentle surgical approach that is used to reduce the stress of the surgery and allow a faster recovery. This includes a procedure known as MIDCAB (Minimally Invasive Direct Coronary Artery Bypass Grafting) - a procedure in which the anterior wall of the

heart (usually the LAD or diagonal artery) is revascularised using a left-sided ITA. The surgical approach is an anterior left-sided thoracotomy in the 4th intercostal space. ITA is harvested thoracoscopically or directly through the thoracotomy. The anastomosis is performed on the beating heart as described above. In some units, the same type of operation is being done using a robotic system.

Complications of acute myocardial infarction:

1/ Acute mechanical complications of acute myocardial infarction are rare but very serious complications with a high mortality rate.

- **left ventricular free wall rupture** - a large rupture results in death due to massive cardiac tamponade. Pericardiocentesis is indicated for small tears with gradual leakage of blood into the pericardial cavity. The definitive surgical solution is to close the left ventricle with pledgeted sutures or to close the defect with a patch. If the tear in the left ventricular wall (and blood leakage) occurs gradually over several days or in case of pericardial adhesions, encapsulation of the site may occur with subsequent development of a false aneurysm (pseudoaneurysm).
- **post-infarction ventricular septal defect** - occurs as a complication of a large myocardial infarction in the territory of the LAD or the right coronary artery. A left-to-right shunt occurs at the site of the defect, resulting in volume overload of the right ventricle and low cardiac output of the left ventricle. Patients in cardiogenic shock require immediate surgical intervention to close the defect with a patch. Currently, the most common alternative to this procedure is to connect the patient to mechanical heart support (extracorporeal membrane oxygenation - ECMO) and postpone surgery. This has the advantage of allowing some healing and strengthening of the defect area, which makes the surgery safer for defect closure. In patients with a small septal defect in whom surgery can be delayed, we administer inotropic support or introduce an intra-aortic balloon counter-pulsation device.
- **acute mitral valve regurgitation** - occurs as a complication of post-infarction rupture of the papillary muscle. Acute mitral regurgitation leads to postcapillary pulmonary hypertension and pulmonary oedema with dyspnoea and hypoxia. As blood returns to the left atrium in systole, forward cardiac output decreases and organ hypoperfusion occurs. Emergent surgical management is indicated, consisting of mitral valve replacement with potential myocardial revascularisation.

2/ Left ventricular aneurysm is a late complication of transmural myocardial infarction. It is caused by the remodelling of the heart muscle into scar tissue. This leads to thinning of the left ventricular

wall and paradoxical bulging during systole. The main clinical symptoms are low cardiac output (some blood remains in the aneurysm and is not ejected during systole), systemic embolism from mural thrombosis in the aneurysm and ventricular arrhythmias. Symptomatic left ventricular aneurysm is referred for surgery - resection of the aneurysm with or without patch closure of the defect.

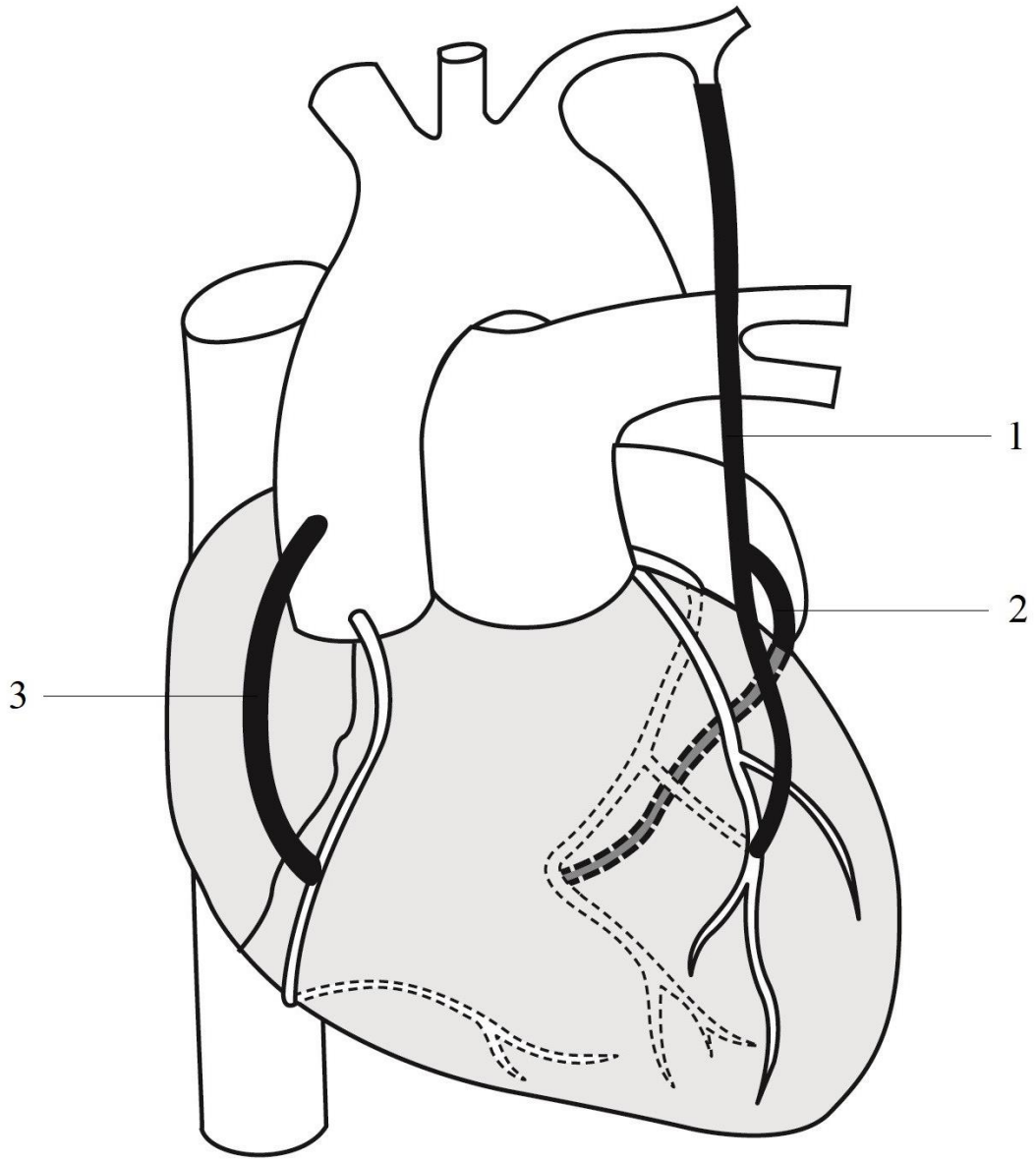


Fig. 1: Basic principles of surgical myocardial revascularisation: 1 - left ITA to the LAD, 2 - right ITA as Y-graft to the circumflex artery, 3-vein graft (or radial artery) from ascending aorta to the right coronary artery (ITA-internal thoracic artery, LAD-left anterior descending artery)

SURGICAL TREATMENT OF AORTIC DISEASE (24)

Fila Petr, Němec Petr

The most common types of acquired aortic disease encountered by cardiac surgeons are aortic aneurysm and aortic dissection. These diseases have different etiologies, clinical manifestations, and, most importantly, prognosis.

1. Aortic aneurysm

Aortic aneurysm is a localized enlargement of the aorta. Most aneurysms of the thoracic aorta are true, meaning they involve all aortic wall layers. The etiology is usually idiopathic (degenerative). Genetic predisposition (Marfan syndrome, Loeys-Dietz syndrome, bicuspid aortopathy…) is associated with aneurysms, especially in younger people. Inflammatory causes of aneurysm (infectious or non-infectious) are rare.

Thoracic aneurysms are usually asymptomatic and are diagnosed during routine physical examinations for other reasons. It is less common for them to be manifested by pain or signs of compression of the surrounding structures. The aneurysm may be filled with thrombi that can embolize.

The aneurysm puts the patient at risk of rupture, with the development of massive bleeding, dissection due to weakening of the aortic wall, embolization of mural thrombi, and, in the region of the aortic bulb, development of aortic valve regurgitation. Diagnosis is usually based on echocardiography, CT, or MRI.

The risk of aneurysm rupture increases with the size of the aorta. The indication for surgery is mainly determined by the diameter of the aneurysm - over 50 mm in the thoracic aorta and 40 mm in the abdominal aorta.

Resection of the aneurysmal aorta and its replacement with a prosthesis is the main principle of surgical treatment of aortic aneurysms. This principle is common to all surgical procedures. However, due to the specific nature of the individual parts, we divide the aorta into three parts: - the aortic root with ascending aorta, the aortic arch, and the descending aorta (thoracic and abdominal). In clinical practice, it is widespread for the aneurysm to affect more than one of these sections and the procedure performed will be a combination of the procedures described below.

1.1 Aortic root and ascending aorta

The most common procedure in this area involves replacing the aortic valve and ascending aorta and reimplantation of the coronary arteries into a prosthesis (Bentall operation).

This procedure uses a composite graft consisting of a mechanical or biological valve and an attached vascular prosthesis. After aortic valve excision, this graft is implanted like a simple aortic valve replacement. The coronary arteries are then reimplanted into the prosthesis. Patients with normal aortic valves can undergo a valve-sparing operation in which the aortic valve is preserved, and only the aortic wall is replaced.
These operations are more beneficial for the patient in the long term but are technically more challenging.
In the region of the ascending aorta, there are no branches from the sinotubular junction to the brachiocephalic trunk. The primary surgical procedure is to resection the aneurysm and replace it with a graft with end-to-end anastomosis at both ends.
PEARS (personalized external aortic root support) is the modern method for preventing further aortic dilatation. This custom-made mesh (based on the patient's aortic CT scan) is implanted externally to the aorta to cover the root and ascending aorta. This method is suitable if the aorta is not too dilated and the aortic valve is without haemodynamic pathology.

1.2. Aortic arch

The principle of surgical treatment of aortic arch aneurysms is resection of the aorta and its replacement with a vascular prosthesis, with reimplantation of the branch arteries.
These surgical procedures are complex because arteries supplying the brain branch off from this region of the aorta. This requires adequate protection of the brain during surgery. There are basic ways to protect the brain during aortic arch surgery: deep hypothermia (18°C) with circulatory arrest and orthograde cerebral perfusion. Currently, the most commonly used method is orthograde cerebral perfusion, in which the brain is perfused with oxygenated blood from the extracorporeal circulation (ECC) through cannulae inserted into the supra-aortic branches (brachiocephalic trunk, left common carotid artery and eventually the left subclavian artery) during circulatory arrest. This ensures adequate oxygenation of the brain. We combine orthograde cerebral perfusion with varying degrees of hypothermia. This has a protective effect on the lower half of the body, which is not perfused during circulatory arrest, and creates space for a brief interruption of orthograde cerebral perfusion, for example, during cannula insertion or suture finishing. Hypothermia in the range of 26 to 28°C is most used.

1.2 Descending aorta

The principle of surgical treatment of descending aortic aneurysms is still the resection of the dilated part and its replacement with a prosthesis. These procedures are performed through a left

thoracotomy, with or without the use of ECC. A specific feature of operations on the descending thoracic aorta is the presence of the arteries supplying the spinal cord and the associated risk of spinal ischaemia with the subsequent development of paraplegia. If a larger part of the thoracic aorta is replaced, the branches supplying the spinal cord must be reimplanted into the prosthesis. The risk of paraplegia can also be reduced by drainage of the cerebrospinal fluid. For descending aortic aneurysms, endovascular treatment is strongly advocated, in which a stent graft (coated stent) is introduced into the descending aorta by way of the femoral artery, deployed there, and anchored at both ends in the non-expanded part of the aorta, thereby excluding the aneurysm from the flow and preventing its rupture.

In the case of aneurysms (thoracoabdominal), we must also reimplant the visceral arteries (celiac trunk, superior mesenteric artery, renal arteries) and ensure adequate perfusion of these outlying branches during surgery. Thoracoabdominal aortic replacement is a technically demanding procedure that also requires a sizeable surgical approach (thoraco-freno-laparotomy). Therefore, interventional endovascular treatment with stent grafts or the so-called hybrid treatment (combination of open surgical and endovascular treatment) is beginning to be promoted in the abdominal aorta.

2. Aortic dissection

Acute aortic dissection is a life-threatening disorder in which a tear in the intima ('entry') and the blood flow causes a longitudinal 'split' of the layers of the vessel wall in the tunica media. The tunica adventitia remains intact. This creates a 'true' and a 'false' aortic lumen, which rapidly fills with blood and the pressure of which pushes the layers of the thoracic aortic wall apart, causing the dissection to spread proximally and distally. Proximally, the dissection may extend to the aortic valve annulus and distally to the femoral bifurcation. Distally, another intimal tear (re-entry) usually connects the true and false lumens. The true lumen is often significantly compressed by the false lumen.

Risk factors for dissection include inadequately treated hypertension, aortopathy causing aortic dilatation, and atherosclerosis. The disease usually begins with a sharp pain behind the sternum or in the back between the shoulder blades, often associated with physical exertion or a rise in blood pressure.

The patient's life is threatened by the rupture of the thin tunica adventitia and subsequent bleeding into the surrounding areas (pericardial cavity, pleural cavity, retroperitoneal space). Ischaemic involvement of organs whose supplying artery branches off the dissected aortic segment and is obliterated by a false lumen may also occur (Fig 1). The dissection thus manifests as ischaemia from the basin of these arteries, i.e. myocardial infarction, neurological symptoms (stroke, lower limb paraplegia), signs of splanchnic ischaemia, anuria or lower limb ischaemia. If the dissection extends

proximally to the aortic bulb, it may cause a commissural tear of the aortic valve, resulting in acute aortic regurgitation. Dissection is diagnosed by CT and echocardiography.

According to the location of the intimal tear, the Standford classification (Fig. 2) divides dissections into type A, when the ascending aorta is involved in the dissection, and type B, when the ascending aorta is not involved in the dissection. This classification is crucial for treatment. Patients with type A dissection are indicated for emergent surgery, as 50% of patients die in the first 48 hours without surgery.

The principle of surgical treatment is resection and replacement with a vascular graft of the part of the thoracic aorta where the primary intimal tear (entry) is located. If the tear is in the ascending aorta, we replace the ascending aorta during. The aortic arch is replaced if the tear is in the intimal region of the arch. The dissected distal part of the aorta (descending), which we do not replace, can be left to thrombotomise the false lumen. In patients with an intimal tear in the aortic arch and a dissection extending into the descending aorta, we can use the "frozen elephant trunk" method. In this technique, we place a composite stent graft into the descending aorta during circulatory arrest after the aortic arch is excised and position it so that the stent-reinforced portion is dissected into the descending aorta, and then use the other portion with the prosthesis to perform the aortic arch replacement. This method has the advantage of being more radical, with a greater likelihood of thrombosis of the false sac and healing of the descending aorta.

In patients with type B dissection, conservative treatment or stent graft implantation is preferred. Only rupture or organ or limb ischaemia is an indication for surgical treatment.

The operative mortality of acute thoracic aortic dissection is high, at 10-20%. Post-operative morbidity is also relatively high, mainly because we are leaving the dissected aorta in the descending part, the wall of which has already been damaged by the dissection.

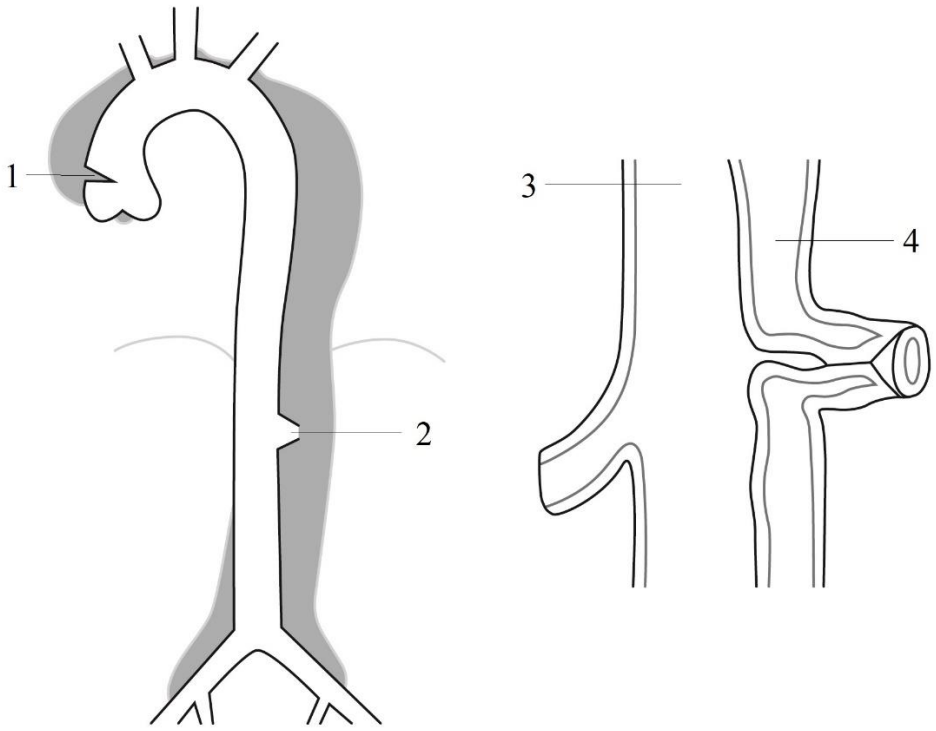


Fig. 1. Aortic disection with entry (1), reentry (2), true (3) and false (4) lumen impairing blood flow to the branches

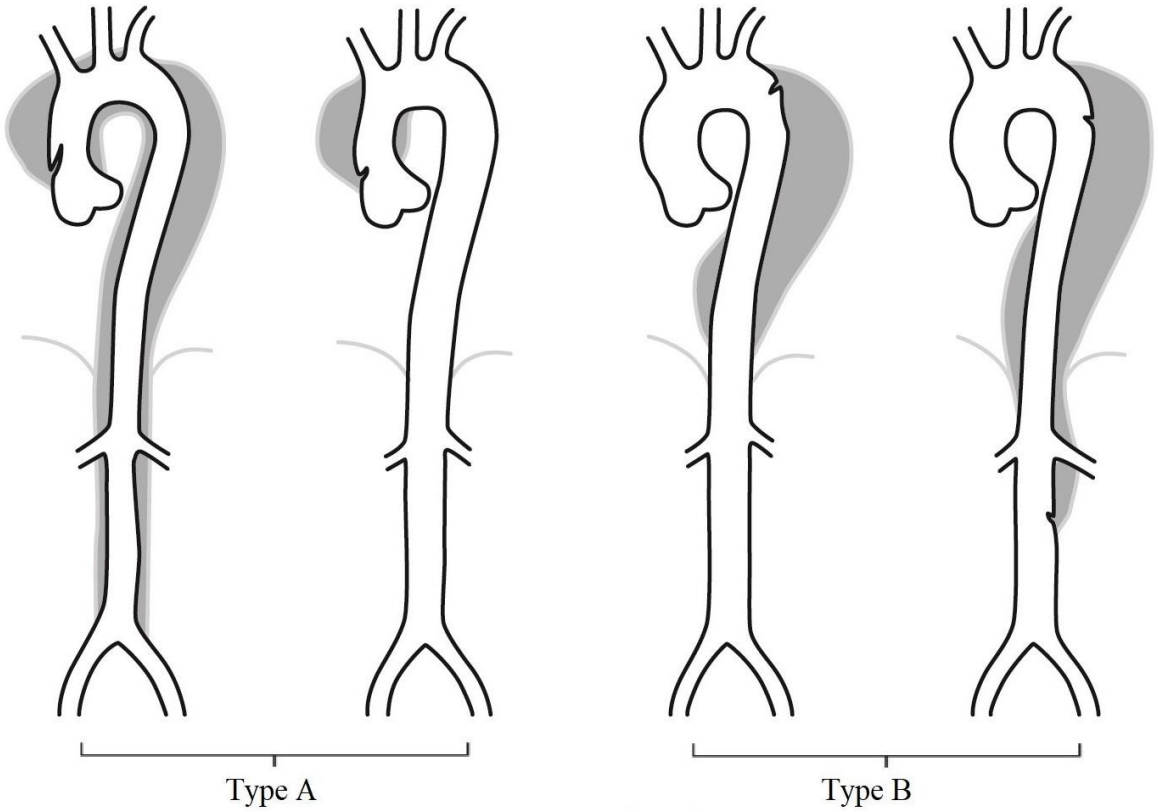


Fig. 3. Aortic dissection classification

CARDIOPULMONARY BYPASS, MECHANICAL CARDIAC SUPPORT (28)

Fila Petr, Němec Petr

1. Cardiopulmonary bypass (CPB)

CPB is a complex method that temporarily replaces the function of the heart and lungs by directly connecting the patient's circulatory system to an external device. The principle of CPB during cardiac surgery is to divert blood outside the patient's heart (body), oxygenate it, remove carbon dioxide, and return it to the systemic circulation. The heat exchanger, a part of the CPB, allows regulation of the patient's body temperature.

Components of CPB (Fig. 1):

Venous (drainage) cannula/cannulae – inserted into the venous system (right atrial appendage, superior and inferior vena cava, or femoral vein).

Venous line – tubing set connecting the venous cannula to the venous reservoir.

Reservoir – acts as a blood reservoir for the CPB system.

Pumps – CPB usually consists of one main pump and several auxiliary pumps. The main pump provides the flow of blood from the venous reservoir through the oxygenator and heat exchanger into the patient's arterial system. Auxiliary pumps are used for suction blood from the surgical field.

Oxygenator and heat exchanger

Arterial line – tubing set connecting the oxygenator to the arterial cannula.

Arterial cannula – inserted into the ascending aorta or femoral artery.

Additional devices – include filters, hemoconcentrators, blood cardioplegia system

Anticoagulation.

The activation of the coagulation cascade upon contact with CPB components usually leads to blood clotting and system thrombosis. Therefore, heparin is administered to the patient at the start of surgery at a dose of 3-4 mg/kg.

2. Myocardial protection during cardiac surgery

Under normal circumstances, the heart is supplied with blood through the coronary arteries that arise from the aortic root. To stop the heart, it is necessary to halt blood flow into the coronary arteries, which is achieved by placing a cross-clamp on the ascending aorta. From this moment, when the coronary arteries are not perfused with warm oxygenated blood, cardioprotective measures must be taken to prevent irreversible myocardial damage.

Myocardial protection is mainly ensured by administering cardioplegic solutions.

The basic principles of myocardial protection are:

- Electromechanical arrest.
- Prevention of cellular oedema and stabilization of cell membranes.
- Adequate cooling of the myocardium (thus slowing metabolism) or ensuring sufficient energy supply while maintaining normothermia.

Cardioplegic solutions can be categorized based on:

Composition:

Intracellular: Low sodium concentration and absence of calcium reduce action potential conduction and contractile force.

Extracellular: Higher potassium concentration (up to 40mmol/L) leads to depolarization of the cell membrane.

Solutions are mildly alkalic to compensate the acidosis, which occurs due to lactate accumulation in muscle during anaerobic metabolism. Cellular oedema results from ischemia, so these solutions must be slightly hyperosmolar to ensure fluid transport out of the cell, with glucose or mannitol added for this purpose. Procaine may be added to stabilize cell membranes.

Blood-based: A blood-based cardioplegic solution is created by mixing the patient's blood with a crystalloid solution high in potassium in a predetermined ratio (commonly 1:4). This combination reduces hemodilution, provides better buffering capacity, protects against oxygen radicals through endogenous antioxidants, and maintains physiological pH.

In most cardioplegic solutions, local hypothermia is used in addition to their composition.

Administration methods:

-Antegrade: Cardioplegic solution is delivered through a specialized cannula into the ascending aorta (between the aortic root and the clamp) or, after opening the aorta, directly into the coronary ostia.

Retrograde: A cannula is inserted into the coronary sinus, and cardioplegia is administered retrogradely through the heart's venous system, with pressure not exceeding 50mmHg.

Combined: A combination of antegrade and retrograde administration ensures the best distribution of the cardioplegic solution in the myocardium.

The effect of cardioplegic solution is time-limited, so its administration must be repeated based on the duration of the surgery.

3. Mechanical circulatory supports (MCS)

MCS encompasses a range of devices used when the heart cannot provide sufficient perfusion to the body's organs and tissues. The general indication is medically unmanageable heart failure, often resulting from myocardial damage.

Indications for MCS use:

1. Cardiogenic shock after heart surgery or cardiogenic shock of another origin (e.g., post-myocardial infarction, unsuccessful PCI, myocarditis).
2. Hemodynamic deterioration in patients on the heart transplant waiting list or those selected for destination therapy.
3. Acute rejection or heart failure immediately after transplantation.

MCS Classification (Table 1):

- Based on duration of the use, they are divided into short-term supports (for acute heart failure, where heart function is expected to recover) and long-term supports (for patients awaiting transplantation for months/years). Permanent devices are for patients contraindicated for transplantation.MCS can support the function of the left ventricle (left-side MCS), the right ventricle (right-side MCS), or both (biventricular support).

A specific type of MCS is the total artificial heart, where both ventricles and valves are removed and replaced by the device.

Purpose-based MCS classification:

1. Bridge to Decision (BTD) – for patients with acute heart failure pending evaluation of further therapeutic options, neurological status, and organ functions.
2. Bridge to Recovery (BTR) – for acute but reversible heart failure cases. Recovery of heart function leads to MCS removal.
3. Bridge to Transplantation (BTT) – to support patients on the waiting list for heart transplantation until a suitable donor heart is available.
4. Destination Therapy (DT) – permanent MCS use for patients in the terminal phase of heart failure who are not candidates for transplantation.

Most commonly used MCS:

-Short-term: Intra-aortic balloon pump (IABP), extracorporeal membrane oxygenation (ECMO), left-side MCS with a centrifugal pump.

Long-term: Left-side MCS with an intracorporal centrifugal pump (LVAD), where the patient is connected to an external power supply by a cable.

Future of MHS: This field is rapidly evolving due to the growing number of patients with heart failure who could benefit from this treatment. If the problem of transcutaneous energy transfer can be solved, long-term MHS may become an alternative

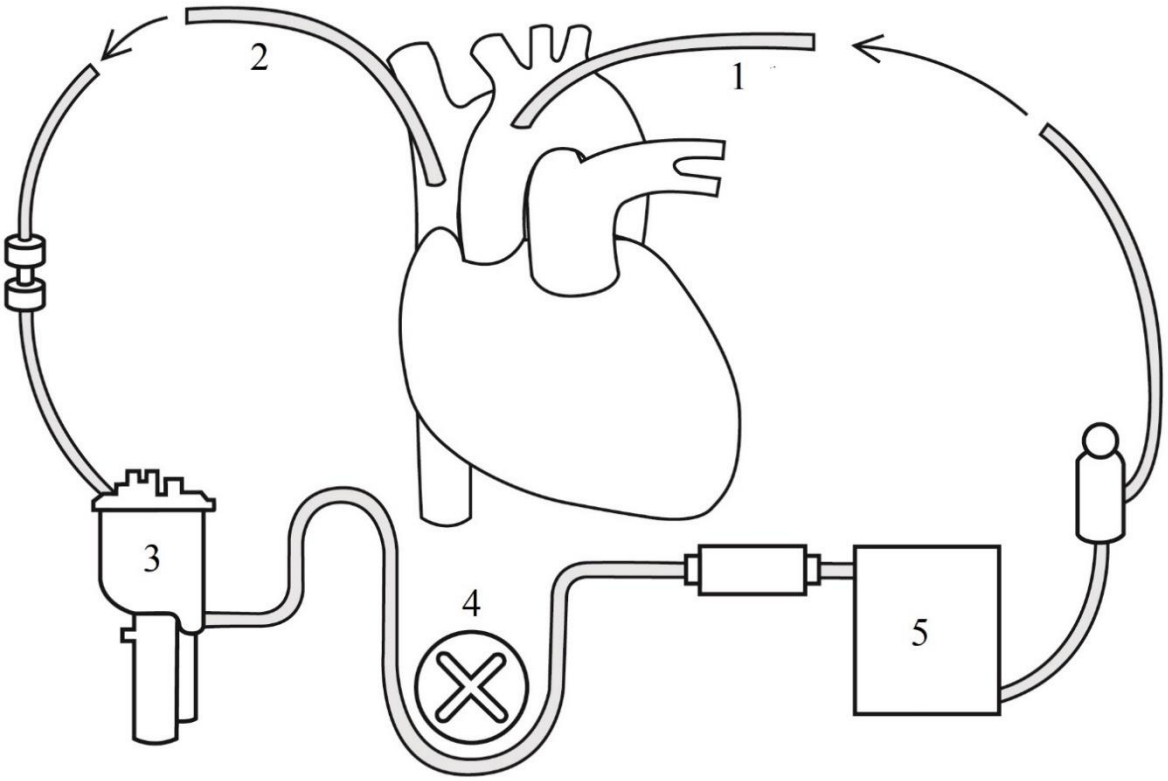


Fig. 1 Schema of Extracorporeal Circulation Systém

1 – arterial cannula, 2 – venous cannula, 3 – reservoir, 4 – pump, 5 -oxygenator and heat exchanger

Duration	**Support**	**Localisation**	**Character of the flow**
Short-term - Reversible failure - Possibility of recovery Long-term - Bridge to transplant Destination - Not suitable for heart transplant	Left-side Right-side Biventricular	Paracorporal Implantable	Pulse flow Continuous flow

Table 1 Mechanical circulatory support classification

CARDIAC AND GREAT VESSEL INJURIES (25)

Fila Petr, Němec Petr

Heart injuries can be divided into penetrating and blunt injuries according to the mechanism of injury. The consequences of these injuries can range from clinically silent arrhythmias to immediate death at the site of injury.

Penetrating injuries

These are caused directly (stabbing, gunshot) or indirectly (by sharp fragments of ribs or sternum in chest trauma). Due to anatomical relationships, the right ventricle is most commonly injured. The left ventricle, atria and great vessels may also be affected. Of the coronary arteries, the left anterior descending artery is most commonly injured. The patient is at risk of haemorrhagic shock or cardiac tamponade. Possible sequelae of penetrating cardiac injury include haemothorax/pneumothorax, myocardial infarction, arrhythmias, valvular injury or projectile embolism from a gunshot. Diagnosis is based on clinical examination, noting the open chest wall and epigastric wound, signs of severe skeletal chest injury, attenuation of cardiac echoes, jugular venous filling, respiratory impairment and absence of peripheral pulsations. Tachycardia and hypotension are present. Of the imaging modalities, transthoracic echocardiography (TTE) is of great diagnostic importance. The detection of pericardial effusion may be an indicator of cardiac injury. CT angiography is part of the protocol for the investigation of severe chest trauma and reliably detects associated injuries. An ECG helps to detect arrhythmias.

In patients with proven tamponade, pericardiocentesis can be performed under TTE control. As a last resort, emergent left-sided thoracotomy is performed to allow evacuation of the tamponade, temporary hemostasis (digital compression, clamp loading, Foley catheter), clamp loading of the descending aorta to centralise circulation and direct cardiac massage. The definitive treatment is surgical from a mid-longitudinal sternotomy, which allows access to all cardiac compartments and possible connection to the extracorporeal circulation. Cardiac injury is treated with polypropylene mattress sutures over pericardial or Teflon felts (Fig 1). Coronary artery injuries require ligation of the arteries and bypass grafting. The affected valve may be repaired or replaced. Any foreign bodies in the chambers of the heart will need to be removed.

Blunt injuries

The mechanism of occurrence includes direct impact on the precordium, deceleration, compression of the heart between the sternum and the spine, and rapid filling of the heart with venous blood as the lower half of the body is compressed. They occur as a result of chest trauma.

They are particularly likely to occur in cases of sternal fracture or avulsion of the anterior segment of the ribs with the sternum.

Myocardial coma is manifested by malignant ventricular arrhythmias with no morphological basis, followed by sudden death. Myocardial contusion is the most common form of blunt cardiac injury. Diagnosis is based on the description of the mechanism of injury, abnormal ECG findings (non-specific ST segment changes, negative T wave, supraventricular or ventricular extrasystoles, new-onset block, ischaemic changes) and/or elevation of cardiac-specific enzymes (troponin). The patient is at risk of developing malignant arrhythmias or heart failure with more extensive bruising; therefore, continuous monitoring of vital signs and ECG is necessary.

Therapy should focus on treating arrhythmias (pharmacological or electrical cardioversion, pacing for high-grade block) and haemodynamic stabilisation. In severe myocardial contusion, blunt injury to the coronary artery with its thrombosis may occur, leading to myocardial infarction with all its consequences and possible complications. Treatment depends on the coronary findings. Other blunt trauma modalities are rare.

Traumatic rupture of the ventricular wall is often lethal; survivors present with cardiac tamponade and require urgent surgical management.

Traumatic septal defects often present with a time latency and treatment is then deferred (depending on the size of the defect and the significance of the left-to-right shunt) - conservative, transcatheter occlusion or surgical closure.

Rupture of the pericardium can lead to herniation of the heart into the pleural or abdominal cavity and torsion of the great vessels. Clinical manifestations include haemodynamic instability and cardiac arrest. Treatment involves repositioning of the heart and suturing of the defect.

Blunt valve injuries most commonly affect the aortic and mitral valves with the development of acute regurgitation, which in severe cases can lead to left heart failure, cardiogenic shock and pulmonary oedema. Depending on the perioperative findings, treatment is either surgical (reconstruction) or prosthetic (replacement).

Large vessel injury

The major intrathoracic vessels include the aorta and the branches of the aortic arch, the superior vena cava and its tributaries, the inferior vena cava, the pulmonary artery and its branches, and the pulmonary veins.

Penetrating injuries may be direct or indirect, as in the heart. They often result in exsanguination at the site of injury. Surviving patients in haemorrhagic shock or after successful initial resuscitation are transferred directly to the operating theatre. Only haemodynamically stable patients with large

vessel injuries are referred to the operating theatre for a chest CT with CT angiography as part of the trauma protocol. The principle of treatment is direct suturing of the injured vessel, interposition of a vascular prosthesis, reconstruction of the vessel with a patch or bypass surgery. In patients with a prolonged course, endovascular therapy - stentgraft implantation - may be considered.

Blunt aortic injury (car accidents, fall-related injuries) is caused by shearing forces at the junction of the mobile aortic arch and the fixed descending aorta - behind the left subclavian artery. Complete transection is a life-threatening injury with massive haemorrhage ending in exsanguination. In incomplete subadventitial transection, therapy consists of controlled hypotension with systolic blood pressure levels below 100 mmHg and sten graft placement to bridge the defect in the aortic wall. Cardiac surgery is required for arch injury at the site of the great vessel outflow.

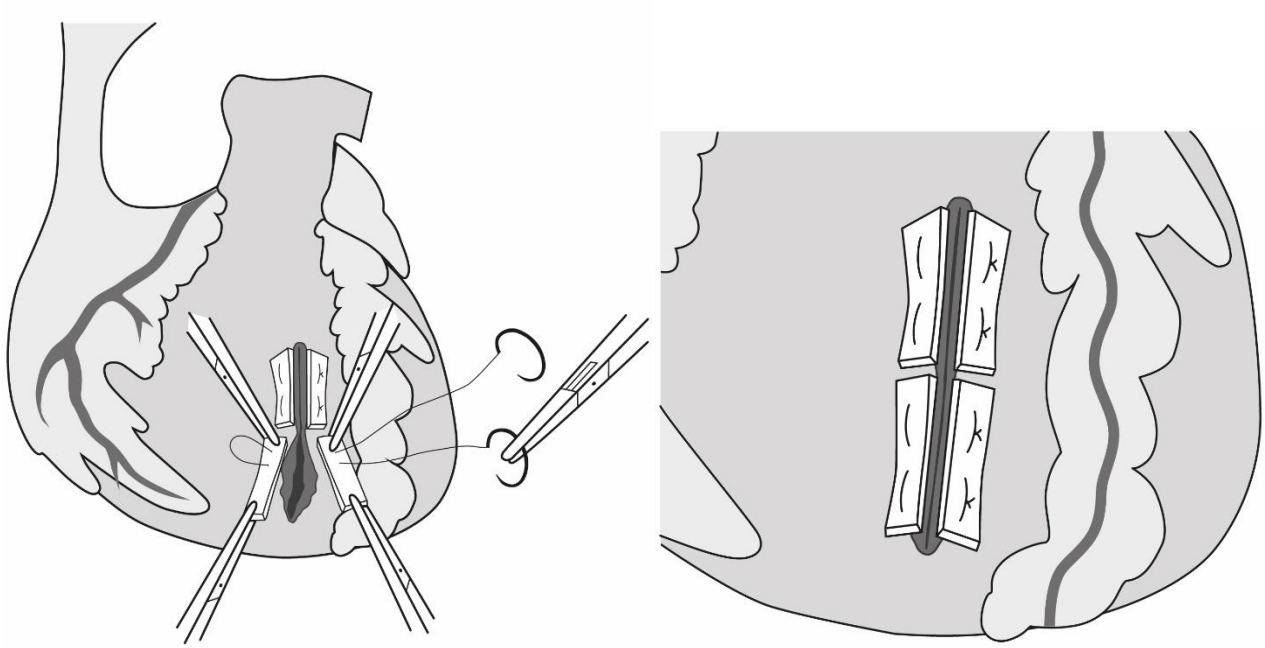


Fig. 1. Ventricle suture using U-stitches using of teflon felt supporting the suture

TRANSPLANTATION OF HEART, LUNG AND LIVER (26)

Fila Petr, Němec Petr

Currently heart, liver, lung, kidney and pancreas transplants are routinely performed. It is also possible to transplant the uterus or intestine. In case of multiple organ failure so-called multi-organ transplant can be performed most commonly kidney + pancreas or liver + kidney combination. In certain specific situations a multivisceral transplant may be carried out which involves the transplant of the liver, stomach, small intestine and pancreas.

Patients in the terminal stage of a disease which cannot be treated otherwise and who have no contraindications are indicated for organ transplantation.

General contraindications for transplantation:

Malignant disease

Chronic infection

Active infection or sepsis

Poor patient compliance

Patient's age

Nowadays most contraindications are considered relative with the overall biological condition of the patient being crucial. The patient must undergo comprehensive examination be assessed by a multidisciplinary committee and if no contraindications are found they are placed on the waiting list.

Heart Transplant (HT)

The first successful orthotopic heart transplant (HT) was performed in 1967 by Ch. Barnard in South Africa. The results of early transplants were poor due to the lack of adequate immunosuppressive therapy. Since the 1980s, with the introduction of new immunosuppressants, heart transplantation has gradually become an established treatment method for patients in the terminal stage of heart failure (classified as NYHA III or IV) refractory to medical treatment and where no other surgical options exist.

Indications for heart transplantation:

Patients most commonly indicated for transplantation have:

Ischemic heart disease with surgically untreatable coronary artery damage and low ejection fraction (usually below 20%)

Dilated cardiomyopathy with a low ejection fraction

Less common indications include other types of cardiomyopathy, surgically untreatable valvular or congenital heart defects and patients with malignant arrhythmias.

Specific contraindications for heart transplantation:

Increased pulmonary vascular resistance

Severe extracardiac disease (CNS, liver, lungs etc.)

HT procedure:

Heart transplantation is performed using cardiopulmonary bypass and cannulation of ascending aorta and both vena cava. After clamping the ascending aorta and stopping the blood flow to the coronary arteries the diseased heart is excised leaving long stumps of the aorta, pulmonary artery and both vena cava. The posterior part of the left atrium is also preserved (Fig 1). The new graft is then transplanted with sutures done in the following order: left atrium, inferior and superior vena cava, pulmonary artery and aorta. This so-called bicaval technique replaced the original method where the entire atria were preserved, which led to frequent arrhythmias.

Surgical outcomes are excellent given the patients' condition with early mortality under 10%. One-year survival is between 80-90% and about 70% of recipients survive five years. After a successful transplant patients experience dramatic improvements in health and mostly are able to fully return to social and work life.

Liver Transplant (LT)

The first liver transplant was performed in 1967 in Denver, and the first LT in the Czech Republic was carried out in 1982 at the 2nd Surgical Clinic at university hospital St. Anna in Brno.

Indications for liver transplantation:

Patients with terminal liver failure, most commonly caused by:

Liver cirrhosis due to cholestatic diseases, viral hepatitis, excessive alcohol consumption, etc.

Metabolic diseases (e.g., Wilson's disease, hemochromatosis)

Polycystic liver disease, Budd-Chiari syndrome, or liver failure of unknown etiology

Other indications include liver tumours (unresectable primary hepatocellular carcinoma or carcinoma in cirrhosis).

LT procedure:

Access to the abdominal cavity is carried out via a subcostal incision extending across the midline. The structures in the hepatoduodenal ligament (hepatic artery, portal vein and bile duct) are isolated and cut. The liver is then detached from the inferior vena cava (IVC), which is left in situ. A new liver is implanted by suturing the anastomoses in the following order: IVC of the graft end-to-side to the IVC of recipient, portal vein, hepatic artery, and finally a bile duct end-to-end anastomosis is created. Most patients with liver cirrhosis have coagulation disorders, requiring precise surgical technique and careful control of bleeding.

Surgical outcomes are excellent with early mortality ranging from 5-10%. Five-year survival rates are 80% and approximately 60% of patients survive ten years. Most patients experience a significant improvement in quality of life and return to normal daily activities.

Lung Transplant (LuT)

The first successful lung transplant was performed in 1983 in Toronto.

Indications for lung transplantation:

Restrictive lung disease (e.g., cystic fibrosis)

Obstructive lung disease (primary or secondary pulmonary emphysema)

Pulmonary vascular disease (e.g., primary pulmonary hypertension)

Specific contraindications include smoking, extreme cachexia, or obesity.

LuT procedure:

A transplant can involve one or both lungs (bilateral transplant is now preferred). Lung transplantation is performed via thoracotomy (bilateral thoracotomy for both lungs). After dissection of pulmonary hilum the pulmonary artery, pulmonary veins, and main bronchus are cut, and the lung is removed. End-to-end anastomoses of the pulmonary artery, pulmonary veins (directly to the left atrium), and bronchus are created. For bilateral lung transplants, extracorporeal membrane oxygenation (ECMO) is used to ensure stable circulation and oxygenation of the patient.

The results of lung transplants are also very good, with one-year survival rates between 70-80% and five-year survival rates around 50%.

Kidney Transplant (KT)

Kidney transplantation is not a life-saving procedure because patients with terminal kidney failure can be treated with regular dialysis.

Indications for kidney transplantation: Chronic kidney failure of various aetiologies. Patients can be transplanted after being included in a dialysis program or even before (preemptive transplantation).

KT procedure:

A single kidney is transplanted which is functionally sufficient. The transplant is performed heterotopically (in a different location than the original kidneys which are usually left in place). After incision above inquinale ligament the external iliac artery and vein are isolated. The kidney is placed in the pelvic region and an anastomosis is created to connect the graft's vessels end-to-side with the recipient's pelvic vessels. The ureter is implanted directly into the urine bladder.

Early results of kidney transplants are excellent and it is proven that successful kidney transplants significantly improve quality of life long-term survival and reduce healthcare costs compared to dialysis treatment.

Long-term Post-Transplant Care

After all organ transplants patients must take lifelong immunosuppressive therapy to prevent graft rejection. However, this therapy carries certain complications such as hypertension, diabetes, impaired kidney function, obesity and hypercholesterolemia. Therefore, it is essential to regularly monitor the levels of immunosuppressive drugs and combine different medications to minimize their side effects.

For early diagnosis of rejection biopsies are necessary (tacking of several small pieces of the tissue). In the long-term follow-up the patients are in risks of infection, chronic rejection (manifesting differently in each organ) a higher incidence of tumours and the side effects of chronic immunosuppressive therapy.

Specific postoperative monitoring:

For heart transplants, regular endomyocardial biopsies are performed, taking samples from the right ventricle. Coronary angiography is done annually.

For lung transplants, regular transbronchial biopsies are performed.

For liver transplants, biopsies are only done if acute rejection is suspected. The most common postoperative complications involve bile ducts, including anastomotic leaks and strictures.

For kidney transplants, biopsies are also only done when rejection is suspected. The most common complication is an anastomotic leak between the ureter and the urine bladder.

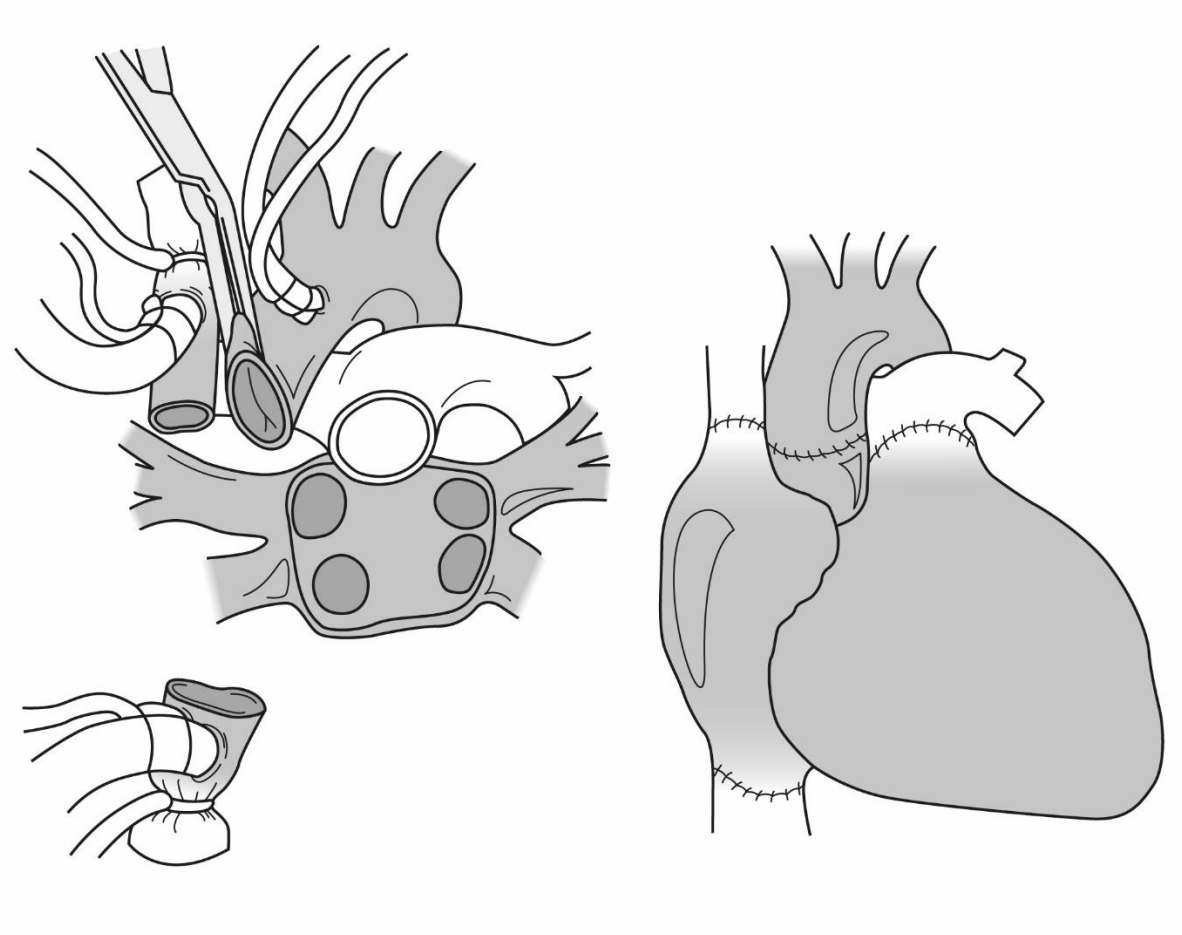


Fig. 1 After excision of the heart with preservation of long stumps of the aorta, pulmonary artery and both vena cava. The posterior part of the left atrium is also preserved. The new heart after the bicaval technique of transplantation

ORGAN DONORS, SURGICAL TECHNIQUE OF ORGAN PROCUREMENT (27)

Fila Petr, Němec Petr

The issue of organ donation in the Czech Republic is governed by Act No. 285/2022 Sb. According to this law, every person can be considered an organ donor unless they have expressed objection to organ donation during their lifetime (presumed consent principle). Objection can be made by for example registering in the National Registry of Persons Objecting to Postmortem Tissue and Organ Donation.

The number of donors in all countries worldwide is lower than the number of transplants needed which is why there is an effort to maximize the use of all patients with a hopeless prognosis who meet the criteria for donation. The process of donation and transplantation is concentrated in transplantation centres. There are seven in the Czech Republic. Each centre has its designated region where it conducts these activities.

Organ donors can be divided into two groups:

Living Donors

From a living donor, either a paired organ (kidney) or part of another organ with regenerative ability (liver segments or lobe) can be taken. The donor must undergo a thorough examination to ensure that their health will not be harmed. The donor must give a consent to the procedure.

The main advantages of transplantation from a living donor include that both the procurement and transplant can be planned as elective procedure, with both the donor and recipient in optimal condition and minimal cold ischemia. This results in better long-term transplant outcomes.

Deceased Donors

Organs can be procured from a deceased donor if their death has been confirmed. Death is determined by two independent physicians based on the irreversible cessation of circulation or the irreversible cessation of brain function, including the brainstem.

Donors after Circulatory Death (DCD)

Donation follows cardiopulmonary failure under defined conditions. Patients with a hopeless prognosis receive palliative care. If cardiac arrest occurs (two of the three criteria must be met: asystole on ECG, flat arterial curve or a stopped heart on echocardiographic examination) a five-minute no-touch interval follows. Afterward the time of death is recorded and organ procurement can begin.

Donors after Brain Death (DBD)

A donor is a patient who has suffered irreversible brain injury, resulting in coma and loss of brainstem reflexes.
Before inclusion in the donor program, potentially reversible causes (intoxication, drug effects, and metabolic imbalances) must be ruled out.
This irreversible loss of consciousness is considered as brain death which is regarded as the biological death of the person.

Brain death must be confirmed through:
Clinical examination – Neurological examination of central nerves (demonstrating areflexia above C1), apnoea test
One of imaging methods – Cerebral angiography, perfusion scintigraphy, evoked potentials, or transcranial Doppler ultrasound.
Once all these conditions are met, the patient can be considered brain-dead and included in the donor program. Brain death presents challenges such as hemodynamic instability, thermoregulatory issues, metabolic and endocrine changes, which need to be managed during intensive care.

Transplant Coordination Centre (KST)
KST is an independent body responsible for coordinating donation and transplantation, maintaining legally mandated registries, identifying the most suitable recipients for procured organs, and ensuring international cooperation and coordination.

Contraindications for Donation
Medical: Sepsis, HIV-positive, systemic viral infection, most cancers
Non-medical: Patient objection, family objection (even though this type of objection is not legally recognized, it is generally respected)

Types of Donors:
Ideal Donor: Under 60 years of age, with no organ diseases.
Marginal Donor: Over 60 years old and/or with mild clinical or laboratory abnormalities. This type of donor has become more common due to the general population's health status. The risks and benefits of transplantation from such donors must be carefully weighed.

Organ Procurement

Due to the larger number of patients on waiting lists than available donors there is a push to always perform multi-organ procurement where all viable organs from the donor are collected.
Multi-organ procurement is logistically complex procedure as it involves several surgical teams from different hospitals. Close cooperation with the anaesthesiologist is essential to ensure optimal organ quality.

Surgical Technique:

In multi-organ procurement, an incision is made from the jugular to the symphysis. The abdominal and thoracic cavities are assessed macroscopically to exclude any unexpected pathological findings (e.g., tumour). All the organs being removed are partially isolated and their macroscopic appearance is assessed. After administering heparin, large vessels (abdominal aorta for abdominal organs, ascending aorta for the heart, pulmonary artery for the lungs) are cannulated. Clamps are applied to these vessels to stop blood flow to the organs which are then flushed with cold preservation solution and externally cooled with ice slush. Once adequately flushed the organs are sequentially explanted in this order: heart, lungs, liver, pancreas and kidneys.

Between Procurement and Transplantation, Organs Are Stored:

- In a cold solution
- Connected to a perfusion device (hypothermic or normothermic perfusion). The advantage of this method is better organ quality, though it is logistically and economically demanding.

Time Intervals:

For successful transplantation, it is crucial to minimize the time between organ procurement and transplantation. Two key intervals are monitored:

Cold Ischemic time: The time from the start of organ perfusion with preservation solution to the restoration of blood flow in the organ (maximum tolerated time varies by organ—shortest for the heart at 2-4 hours, longest for the kidney at 18-24 hours).

Warm Ischemic time: The time from cessation of blood flow in the organ to the start of perfusion with preservation solution. Ideally, this is zero; in living donor procedures and DCD, it is acceptable up to 30-60 minutes.

Waiting List

Each patient waiting for a transplant must be placed on a waiting list in one of two categories:

Elective: A patient in a stable condition waiting at home

Urgent: A patient in critical condition requiring hospitalization. Criteria for urgent status vary by organ. For example, a patient waiting for a heart transplant must be on intravenous therapy (inotropes, diuretics) or have mechanical heart support.

Organ Allocation (Matching Donor and Recipient)

Organ allocation is a process that considers many factors and must be entirely transparent, based solely on medical criteria. Allocation criteria differ for each organ.

Factors considered include blood type (identical or at least compatible), body weight (for example, donor and recipient should be of similar build for heart or lung transplants), recipient's clinical condition (urgent or elective status; urgent status always takes precedence), and waiting time on the list. For kidneys, HLA matching is also considered.

Once an organ is allocated to a specific patient, the transplantation procedure begins. According to law, transplantation centres must respect the anonymity between the donor and recipient.

UROLOGY

CONGENITAL DEFECTS OF THE UROGENITAL TRACT (29)

Husár Matej

Congenital defects of the genitourinary system are most often the result of a defect in the interaction between the developing ureteric bud and the metanephrogenic blastema or a defective ascent of the kidneys. A distinction is made between upper and lower urinary tract anomalies.

Upper urinary tract anomalies

- Numerical abnormalities: supernumerary kidney, unilateral renal agenesis, bilateral renal agenesis (incompatible with life)
- Disorders of kidney migration: ectopic kidney (pelvic/lumbar dystopia), crossed ectopia
- Fusion anomalies of the kidney: most commonly horseshoe kidney (ren arcuatus)
- Rotational renal anomalies (malrotation)

Many upper urinary tract anomalies are asymptomatic and their detection is incidental. In symptomatic defects, the defect can results is obstructive or reflux uropathy. The severity of renal involvement depends on the function, obstruction of urinary outflow, risk of infection, vesicoureteral reflux and formation of stones. Ultrasonography is used in the diagnosis, which can detect some congenital renal defects prenatally. Ultrasonography is used not only for diagnosis but also for monitoring the dynamics the obstruction. In case of diagnostic discrepancy, MRI or CT can be used. Dynamic scintigraphy is used to determine renal functions in obstructive uropathies and static scintigraphy in reflux uropathies.

The most common obstructive uropathy in childhood is hydronephrosis that is an obstruction in the pyeloureteral junction. If the obstruction is urodynamically significant, pyeloplasty is performed - resection of the pyeloureteral junction and anastomosis of the ureter to the renal pelvis.

Hydronephrosis may manifest clinically in children as pyelonephritis, usually detected prenatally on ultrasonography. If hydronephrosis arises suddenly, it manifests as renal colic, which is most commonly seen in nephrolithiasis.

The second common nephropathic defect is vesicoureteral reflux, It is the return of urine from the bladder to the ureters and the hollow system of the kidneys. We distinguish between primary and secondary reflux. Primary reflux occurs on the basis of short course of the ureter through the bladder wall and ectopic location of the ureteral orifice, which is usually gaping. Secondary reflux is caused by bladder dysfunction or subvesical obstruction (posterior urethral valve, urethral stricture...). In children, the reflux is most often detected after the first pyelonephritis or as an incidental finding on ultrasound as dilatation of the ureters and the hollow renal system. Currently, the only reliable method to confirm vesicoureteral reflux is micturition cystourethrography. Treatment is initiated

with antibiotic prophylaxis medication. Another option is minimally invasive endoscopic antireflux injection. In limited indications, ureteral reimplantation is performed, where the ureters are submucosally reimplanted to ensure antireflux positioning of the ureters in the bladder wall. The indication for each therapy depends on several factors, namely the function of the kidney, the degree of reflux, the effect of antibiotic prophylaxis and the cooperation of the family in the treatment of the child.

MALE INFERTILITY AND SEXUAL DYSFUNCTION (32)

Kašík Marek

Infertility

Infertility is defined by the inability of a sexually active, non-contraceptive couple to achieve spontaneous pregnancy within twelve months. It affects 12-15**%** of couples worldwide, the male factor is responsible in half of the cases either alone, or associated with infertility issues of woman as well. In majority of the cases it is connected to pathologic semen analysis. Physiologic parameters are shown in table 1. Asthenozoospermia refers to low sperm motility or an insufficient amount of sperm with overall progressive movement. Teratozoospermia is defined as less than 4% of sperm with normal morphology. Oligospermia refers to low sperm count and cryptozoospermia is a condition where spermatozoa are detected only after centrifugation of semen. Azoospermia is defined as the absence of sperm in ejaculate. These pathological conditions are sometimes occurring at the same time and termed OAT syndrome (oligoasthenoteratozoospermia). Etiology can be congenital, acquired or idiopathic. The last one constitutes around 30-40% of cases. Hypogonadism refers to a condition that is characterized by the diminished levels of testosterone and is one of the causes of infertility. Hypogonadism can be divided into primary, in which testicles are affected, and secondary, which is caused by a disorder of the hypothalamus or pituitary gland. Causes of primary and secondary hypogonadism are summarized in tables 1 and 2, respectively.

Varicocele

Varicocele refers to dilation of veins of the pampiniform venous plexus accompanied by some degree of reflux of blood in the scrotum. It is not an uncommon finding, as much as 20% of men are affected. Varicocele occurs typically on the left side, less commonly on the both sides. Isolated right-sided varicocele is rare – the incidence is around 2%. This condition is caused by venous insufficiency and so called nutcracker phenomenon. Secondary varicocele forms as a result of compression of veins that are responsible for venous drainage from the scrotum or vein blockage due to thrombus associated with kidney tumour. This type of varicocele occurs on the both side with the same incidence rate. Diagnostic work-up includes physical examination, the patient must be examined in the standing position. We can differentiate 3 grades of varicocele. Varicocele is usually asymptomatic, although it may cause discomfort and pain in the scrotum propagating to lower abdomen. Scrotal ultrasound examines size of the testes, dilated veins and presence of reflux during Valsalva maneuver. Furthermore, semen analysis is done, as varicocele can be associated with infertility.

Cryptorchidism

Cryptorchidism is the most common congenital anomaly of male genitalia; the incidence is around 1% at the age of 1. Approximately 50% of patients with bilateral cryptorchidism are infertile.

Azoospermia

Azoospermia, defined as the absence of sperm in ejaculate. It is be divided into obstructive and non-obstructive. Obstructive azoospermia is less common (15-20% of cases), laboratory results show normal levels of FSH, LH and testosterone. Testes are a normal size and the epididymis is usually enlarged. The site of obstruction is within the testes or epididymis, ductus deferens can also be obstructed (post-vasectomy, cystic fibrosis or iatrogenic injury during surgery of inguinal hernia) as well as ejaculatory ducts. Non-obstructive azoospermia is a complex pathological condition with multifactorial etiology involving genetic disorders, hormonal imbalances or scrotal injury.

Diagnostics and therapy

Examination includes inspection of the patient's body (absence of body hair, gynecomastia etc.) and external genitalia. We evaluate the size of the testes and epididymis and assess any pathological conditions by palpation (varicocele, absence of ductus deferens). Doppler ultrasonography is used to examine the scrotum, additionally, we perform a transrectal ultrasonography of the prostate and the seminal vesicles. Laboratory examination includes hormone tests and a semen culture test. Antibiotic therapy is warranted in case of proven infection. In selected cases hormonal therapy is indicated. In patients with hypergonadotropic hypogonadism we use human chorionic gonadotropin usually in combination with recombinant FSH. Surgical therapy includes reconstructive surgery and sperm retrieval procedures. MESA (microsurgical epididymal sperm aspiration) is indicated in case of obstruction of ductus deferens or epididymis, TESE (testicular sperm extraction) is performed in case of intratesticular obstruction. Acquired sperm is subsequently used for techniques of assisted reproduction. Reconstructive procedures include vasovasostomy, vasoepididymostomy and transurethral resection of ejaculatory ducts (TURED). Varicocele is usually managed conservatively. Surgery is recommended only in selected cases - clear indications for varicocelectomy are infertility, testicular hypotrophy or symptomatic varicocele. Dilated veins of the pampiniform plexus or the internal spermatic vein are ligated during the surgery. Inguinal or subinguinal microsurgical lymphatic and artery sparing varicocelectomy is considered 'gold standard' treatment. Suprainguinal open or laparoscopic surgery is also feasible. Sparing the lymphatic vessels lowers the risk of postoperative

hydrocele. Other treatment option is sclero-embolisation of the internal spermatic vein performed by an interventional radiologist.

Sexual dysfunction

Erectile dysfunction

Erectile dysfunction (ED) is defined as an inability to attain or sustain an erection long enough for satisfactory sexual intercourse. It occurs in as much as 50% of men above 50 years old, although it can affect younger men as well. Erectile dysfunction can develop gradually or all of a sudden. Recognized risk factors are stress, depression, obesity, lack of physical activity, smoking, drug abuse, atherosclerosis, diabetes, liver disease and pelvic surgery (radical prostatectomy). Oftentimes, ED can be an adverse effect of certain medications. Loss of erectile function can be a symptom of other illnesses, particularly cardiovascular diseases.

Diagnostic work-up relies on the patient´s history, inquiring about nighttime and morning erections, rigidity and duration of erections, issues with ejaculation or orgasm. Furthermore, it is necessary to assess signs of hypotestosteronism and the mental health of a patient. The presence of morning erections and normal rigidity during masturbation may help to distinguish organic from psychogenic etiology. We examine external genitalia – testes size, presence of pubic hair, pathological conditions such as Peyronie´s disease etc. A crucial laboratory examination is testing the testosterone level, lipid panel and fasting glycaemia. Moreover, the standardized IIEF questionnaire is used (international index of erectile function).

The first stage of treating erectile dysfunction should involve having a healthy active lifestyle and a healthy diet. Testosterone replacement therapy is indicated if hypogonadism is detected. Untreated prostate cancer is a contraindication to testosterone supplementation. Patients with mental health issues can be referred to a psychologist or sex therapist. Inhibitors of phosphodiesterase 5 (PDE5-I) are used as first line treatment of erectile dysfunction. They induce relaxation of smooth muscle in the corpora cavernosa, resulting in increased penile arterial blood flow. Different PDE5-I agents, namely sildenafil (first one discovered), tadalafil, vardenafil and avanafil, differ in onset and duration of action. They are taken on-demand, before planned sexual intercourse. Alternatively, tadalafil can be taken daily in lower dose. Common side effects include headaches, flushing and nasal congestion. Vacuum erector device is another treatment option for men with erectile dysfunction. After using the device, constriction ring can be used to maintain the erection. The ring should not be placed for longer than 30 minutes. Intracavernosal alprostadil injection is a second-line treatment

after failure of PDE5-I. Erection occurs 5-15 minutes after administration and duration depends on the dose. Complications include pain, hematoma, fibrosis of corpora cavernosa, prolonged erection and even priapism. Penile implants represent last-line treatment of ED, they can be classified into semi-rigid and inflatable prosthesis.

Peyronie´s disease

Peyronie´s disease, also called induratio penis plastica, is a disorder that may cause difficult or impossible sexual intercourse. It is characterized by occurrence of fibrous calcified plaques in the tunica albuginea, leading to curvature, shortening of the penis and erectile dysfunction. Precise underlying cause in unknown. Repeated microtrauma of tunica albuginea is thought to play role in the pathogenesis. There is an association with other fibromatous disorder such as Dupuytren's contracture. Peak incidence is around 55-60 years of age. Disease is divided into two stages – acute and chronic. . The first is the active (acute) stage characterized by painful erections and changing of the deformity. The second stage is the stable (chronic) phase, in which the deformity is stabilized and pain during erection disappears. Diagnosis is determined by patient's history (onset of symptoms, painful erection, bending or shortening of the penis) and physical examination. Penile examination is done to evaluate the presence of palpable nodule or plaque. An objective assessment of penile curvature during erection is necessary, this is obtained by a home (self) photography in two planes. Doppler ultrasonography is used to assess localization, size and number of plaques. Conservative management is feasible in patients who are able to have sexual intercourse. Use of oral treatment (vitamin E, colchicine, potassium para-aminobenzoate) is controversial as there is no convincing evidence about the efficacy. Intralesional treatment allows a localised delivery of a particular agent directly into the plaque and leads to improvement of the curvature. Steroids, calcium channel antagonists, clostridium collagenase and interferon α-2b are used. Surgical treatment is considered if conservative treatment fails.

There are two types of surgical repair: penile shortening and penile lengthening procedures. Penile shortening procedures include the resection or plication techniques performed on the convex side of the penis. Penile lengthening procedures are performed on the concave side of the penis using a graft. Patients with a complex deformity are offered implantation of penile prosthesis. Shock wave treatment is a novel therapeutic modality for Peyronie´s disease.

Table 1: Normal sperm analysis values

Sperm concentration (mil/ml)	**≥15**
Semen volume (ml)	**≥1,5**
Total sperm count (mil)	**≥39**
pH	**7,2**
Progressive motility (%)	**≥32**
Total motility (%)	**≥40**
Normal morphology (%)	**≥ 4**

Table 2: Causes of primary hypogonadism

Factors	Cause
Congenital	**Anorchia** **Cryptorchidism** **Genetic abnormalities**
Acquired	**Trauma** **Torsion** **Varicocele** **Tumor of testes** **Inflammation, mumps orchitis** **Exogenous factors (environment, drug, radiation)** **Systemic diseases (cirrhosis, renal failure)** **Surgery that may impair vascular supply of testes (inguinal hernia repair)**
Idiopathic	

Table 3: Causes of secondary hypogonadism

Factors	Cause
Congenital	**Idiopathic hypogonadotropic hypogonadism** **Kallmann syndrom**
Acquired	**Tumors of hypothalamus or pituitary gland** **Basilar skull fracture** **Hyperprolactinemia** **Drugs, anabolic steroid, exogenous testosterone**

VESICOURETERAL REFLUX, HYDRONEPHROSIS (34)

Kašik Marek, Fedorko Michal

Vesicoureteral reflux

We define vesicoureteral reflux (VUR) as the backflow of urine from the bladder into the ureter, or up to the collecting system of the kidney. Prevalence of VUR in a healthy pediatric population is not known, estimates range from 0.4% to 1.8%. In children with a proven urinary tract infection (UTI), VUR occurs quite often, especially in children with acute pyelonephritis, where it is present in 29-50% of those affected and its occurrence is inversely proportional to the age of the child. Reflux is more often diagnosed in girls, on the contrary, prenatal diagnosis of VUR is more common in newborn boys. In the pathogenesis of VUR, genetic predisposition plays a role, its familial occurrence is proven in many children, however the mechanism of transmission is not known. VUR does not show specific symptoms, it is characterized by recurrent UTIs, through reflux of infected urine, bacteria reach the kidney. Increased backpressure during VUR leads to intrarenal reflux (IRR), which is the propulsion of urine from the apex of the renal papilla in the pelvis towards the collecting ducts and kidney parenchyma.

The presence of triads – VUR, IRR and UTI is a high risk for early onset parenchymatous scars in the kidney and the development of so-called reflux nephropathy. The result of the harmful effect of reflux on the kidney is the slowing down of its growth with the formation of a "small shrivelled kidney", in then bilateral impairment with the possibility of developing chronic renal insufficiency. VUR without UTI usually does not lead to scarring. The first symptom of kidney damage from reflux can also be hypertension, which is suffered by up to 15% of children with VUR. One of the dominant signs in infants may be failure to thrive. A rare symptom of reflux can also be bedwetting. According to the pathophysiology, we distinguish between primary VUR (it arises in case of congenital insufficiency of the ureterovesical connection) and secondary (occurs during other pathological changes of the urinary tract, e.g. with subvesical obstruction, UTI, neurogenic micturition disorders, after trauma or iatrogenic damage, etc.).

The diagnostic method for detecting VUR is classic X-ray voiding cystourethrography (VCUG), which alone can determine the degree of VUR and should help in differentiation of primary VUR from secondary. According to the international classification, we recognize 5 grades: VUR (Fig. 1):

1. Reflux into the lower part of the undilated ureter
2. Reflux into the renal pelvis, which is not dilated.
3. Reflux with mild dilation of ureter and pelvis, the contour of the calyces is preserved

4. Reflux with significant dilatation of the ureter and pelvis, blunting of the calyces, but they are still concave
5. Reflux with massive dilatation of the collecting system of the kidney, the calyces are convex

Reflux can be passive, when retrograde urine flow to the kidney occurs when filling the bladder with contrast material, or active, when VUR appears only during micturition.

Other investigative methods include urethrocystoscopy in boys when subvesical obstruction is suspected or to evaluate the shape of the ureteral orifices. Urodynamic evaluation is performed in case of suspicion of more serious dysfunction of the lower urinary tract.

The goal of VUR treatment is to prevent morphological and functional kidney damage, to secure their normal development and growth, adjust the function of the ureterovesical junction and eliminate infection.

Today, conservative therapy is mostly recommended - long-term application of prophylactic dosage of antibacterial drugs. After eradication of the infection, low doses of furantoin or co-trimoxazole are most often administered prophylactically. This approach is based on the theory of maturation of the intravesical ureter, which anticipates that maturation of antireflux mechanisms make take up to 12 years. Therefore, in cases of VUR grades I – III spontaneous resolution can be expected, but higher grades of VUR require surgical treatment. Surgery includes transurethral operations (submucosal injections, Fig. 2), open operations (antireflux reconstructions) and laparoscopic operations.

Hydronephrosis

Hydronephrosis means dilatation of the excretory system of the kidney, i.e. calyces and pelvis, due to urinary retention. The extent of dilatation of the pelvicalyceal system (PCS) does not always correspond to the severity of the obstruction in the upper urinary tract. The etiology may be primary (congenital) or secondary. Individual causes are listed in Table 1.

Most cases of primary hydronephrosis are asymptomatic and diagnosed during prenatal or postnatal newborn screening. Less often, the first symptom is acute pyelonephritis, abdominal pain, palpable resistance in the abdomen or failure to thrive.

The symptomatology of secondary hydronephrosis depends on the cause. As a rule, these symptoms are nephralgia, hematuria, renal colic, acute pyelonephritis. Recurrent urinary tract infections with fever, abdominal pain, hematuria and pyuria may also occur. In the case of a solitary kidney, the symptom may be oliguria or anuria if the obstruction is absolute, as well as in bilateral hydronephrosis.

Ultrasonography (USG) is the most widely used for diagnosis of hydronephrosis, both prenatal and postnatal. According to the ultrasound findings, we distinguish 4 grades of hydronephrosis from a

slightly enlarged pelvis up to significant dilatation of the collecting system with reduction of renal parenchyma.

Newborns are recommended to be examined at the earliest after 48 hours of life, preferably on the 3rd or 4th day, due to lower hydration and physiological oliguria in the first days of life.

Another examination method is dynamic scintigraphy of the kidneys - mutual comparison of the functional capacity of the kidneys. It shows the kinetics of an intravenously administered radiopharmaceutical (MAG3) and its transport through the urinary tract. In case of unclear findings, obstruction can be confirmed or ruled out by administration of a diuretic (furosemide). Excretory urography is currently rarely used.

Most primary hydronephrosis tend to resolve spontaneously. Patients undergo repeated USG and isotope examinations. Surgical treatment is indicated in cases with low relative function (< 35%) of affected kidneys detected at the first isotope examination, kidneys with extreme dilatation of the PCS (≥ 50 mm in the anteroposterior dimension) or if during the follow-up the dilatation progresses or the relative function of the kidney decreases. Furthermore, patients with symptomatic congenital hydronephrosis (pain, recurrent pyelonephritis, etc.) are also indicated for surgery. The most common the procedure is pyeloplasty due to obstruction of the ureteropelvic junction. Part of the renal pelvis, ureteropelvic junction and part of the proximal ureter are removed. The ureter is then pulled upwards, spatulated and sutured to the remaining part of the renal pelvis. In adulthood, the method of choice is a laparoscopic or robotic approach. Endoscopic treatment consists of incising the stenosis with a knife or laser either by antegrade or retrograde approach. The aperistaltic section of the ureter is managed with reconstructive surgery in childhood. In secondary hydronephrosis, the management involves eliminating the cause. Acute drainage of the upper urinary tract due to hydronephrosis can be achieved by introducing a JJ stent or nephrostomy.

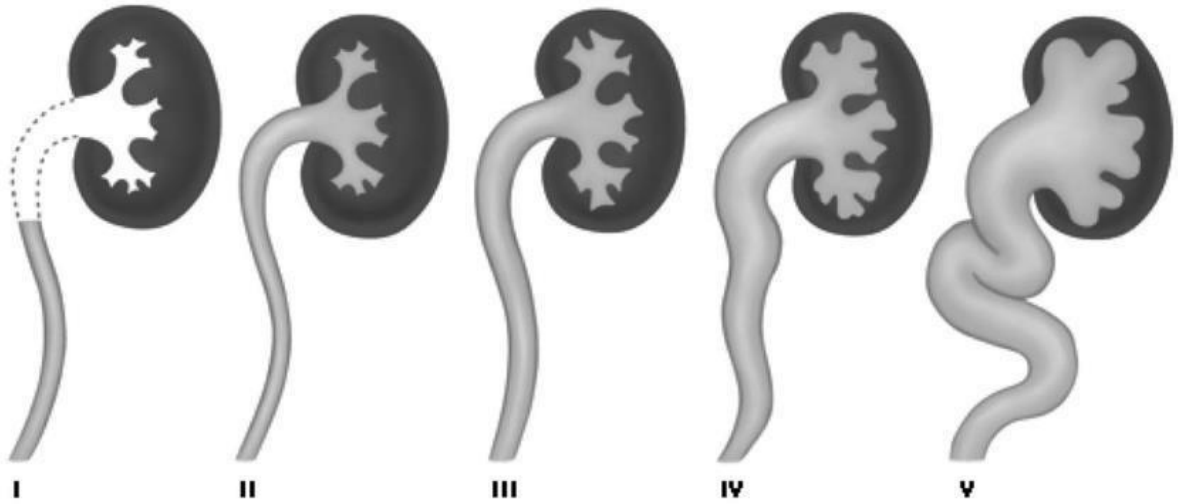


Figure 1 Stages of VUR, source: Kočvara R., Drlík M.: Pediatric urology. Maxdorf. 2023

Primary hydronephrosis	Obstruction of the ureteropelvic junction • Stenosis • compression by an aberrant vessel supplying the lower pole of the kidney • high insertion of ureter into renal pelvis • persistent and fixed fetal folds of the ureter
	Vesicoureteral junction obstruction • aperistaltic juxtavesical section of the ureter • ureterocele
	Vesicoureteral reflux
Secundary hydronephrosis	Ureterolithiasis
	Ureteral stricture and stenosis
	Extramural compression of the ureter, e.g. by a tumour
	Subvesical obstruction

Table 1

UROLITHIASIS, RENAL COLIC (33)

Trinh Tuah, Fedorko Michal

1. Epidemiology and risk factors

The lifetime prevalence of urolithiasis ranges from 1% to 15%, varying by age, race, sex and geographical location (prevalence in the Czech Republic is about 5%). The causes of the formation and growth of urinary stones are multifactorial and can be divided into internal and external factors. Internal factors include age, sex, the presence of obesity and genetic influences. Urinary calculi occur most commonly in male patients of working age however, the occurrence of urolithiasis in childhood or old age is not rare. External factors may include local climate, season (high prevalence in summer), low fluid intake, dietary habits and also the work environment (typically in workers in hot environments).

2. Pathophysiology of stone formation and composition

2.1 Pathophysiology of stone formation

The basic prerequisite for the formation of stones is hypersaturation of urine with lithogenic substances. Free stone-forming crystals can be detected in most urine samples, and yet the majority individuals do not form stones. In patients with urolithiasis, there are more crystals of lithogenic substances in their urine and they have a higher risk of aggregation. The crystal theory of the formation of urolithiasis has several stages.

- **supersaturation** is a state where a substance is present in a solution several times more than its own solubility
- **crystal nucleation** from supersaturated urine (crystallization)
- **crystal growth** - increase in volume
- **crystal aggregation**

Urinary stones consist of a crystalline and a non-crystalline component (the so-called matrix). The Matrix is composed of organic matter - a combination of mucoproteins, proteins, carbohydrates and urine inhibitors.

Citrate	inhibits the formation of calcium oxalate and calcium phosphate stones, especially by forming complexes with calcium, thereby reducing its availability
Phosphate, pyrophosphate, magnesium	bind to oxalates, reducing aggregation and crystal growth
Nephrocalcin and Tamm-Horsfall glycoprotein, osteopontin (uropontin)	inhibit the aggregation of calcium oxalate crystals

Tab. 1: examples of crystallization inhibitors

2.2 **Composition of urolithiasis**

Analysis of the composition of urinary stones is important for further diagnosis and treatment. It is performed in specialized laboratories, mainly infrared microscopy and polarization spectroscopy are used. The stones are often of a mixed nature. The most common component of urinary of stones is calcium. The most common compositions of urinary stones include:

- calcium oxalate – approx. 80%
- calcium phosphate – approx. 22%
- urate – 15-20%
- struvite – 7-10%

2.3 **Metabolic disorders related to stone formation**

2.3.1 **Calcium oxalate and calcium phosphate stones**

Hypercalciuria – the most common disorder, we distinguish 3 types

- absorptive (excessive absorption from the intestines)
- renal (excessive losses of calcium through the kidneys)
- resorptive (excessive mobilization of calcium from bones)
- examples of diseases: primary hyperparathyroidism, immobilization, sarcoidosis, hypervitaminosis D, Cushing's syndrome, treatment with corticoids, hyperthyroidism, multiple myeloma, malignancies

Hyperoxaluria - causes an increase in urine saturation with calcium oxalate

- primary hyperoxaluria – AR hereditary disease
- enteric hyperoxaluria - e.g. Crohn's disease, chronic pancreatitis, ileal resection
- dietary hyperoxaluria - increased absorption from food rich in oxalates (nuts, chocolate, spinach, rhubarb)

Hypocitraturia - citrates reduce the amount of ionized calcium in the urine, thus preventing the formation of calcium-oxalate complex, occurs in 15-63% of patients with nephrolithiasis

• disease example: distal renal tubular acidosis

Renal tubular acidosis – a disorder of urine acidification, a higher pH causes urine hypersaturation calcium and phosphate

2.3.2 Urate stones

Hyperuricemia, hyperuricosuria – excess uric acid, low urine pH (5.5 ± 0.4) and low diuresis increases the formation of urolithiasis

• example of a disease: gout (50% of patients with urate stones have gout), myeloproliferative disease

2.3.3 Rare types of urolithiasis

• **xanthine** - the cause is a xanthine oxidase defect, AR hereditary disorder

• **cystine** - AR hereditary disorder, hypercystinuria

• **infectious** – magnesium ammonium phosphate (struvite) stones, formed by the action of urease produced by bacteria, e.g. Proteus mirabilis strain, etc.

3. Renal colic

3.1 Symptoms

Small kidney stones usually asymptomatic, larger stones can cause indeterminate symptoms dull pain (nephralgia). A renal colic occurs when kidney stones are released into the pelvis and travel to the ureter. Renal parenchyma do not have pain receptors, kidney pain originates from of the fibrous capsule of the kidney, which responds to distension and local irritation. The pain is localized to the area of the ipsilateral costovertebral angle below the 12th rib near the spine. Renal colic is characterized by sudden, sharp, spasmodic pain in the lumbar region of fluctuating intensity, pulsating in nature and shooting into the surroundings, with propagation up to the labia or testicles. Accompanying symptoms may be hematuria (microscopic or macroscopic), dysuria or GIT problems (nausea, vomiting). Infectious stone occurs in severe complications such as pyonephrosis, septicemia or perinephritic abscess.

3.2 Diagnosis

Taking a patient history, physical and laboratory examination (urine, blood serum) is crucial. Upon first contact with the patient, we perform an **ultrasound of the kidneys and bladder** (or the entire abdomen). Obstruction of the excretory urinary tract manifests itself as dilatation of the calico-pelvic

system (hydronephrosis). A **non-contrast CT scan** of the abdomen and pelvis is the **gold standard** for confirming or ruling out urolithiasis. In case of suspected infectious complications associated with obstruction of the urinary system, abdominal CT with contrast material is indicated.

3.3 Therapy of renal colic and urolithiasis

The basis is the administration of spasmo-analgesics, which relieves the patient of pain. First-line analgesic agents are non-steroidal anti-inflammatory drugs: metamizole, diclofenac, indomethacin or ibuprofen. Second-line analgesic agents: opiates (hydromorphine, pentazocine, tramadol). Conservative (expulsive) treatment can be chosen for small ureterolithiasis (usually up to 5 mm), the treatment aims to expel the stone by administering alpha-blockers (e.g. tamsulosin) and anti-edematous drugs, it is important to increase fluid intake and exercise. In case of refractory pain, signs of incipient sepsis, renal failure, obstruction of a solitary kidney or general alteration of the patient's condition it is necessary to relief the obstruction (introduction of a nephrostomy or double J stent). Active stone removal means performing an endoscopic procedure, or extracorporeal shock wave lithotripsy (ESWL). Commonly performed endoscopic procedures are ureteroscopy (flexible or semi-rigid) and percutaneous nephrolithotripsy. Laparoscopic and open surgery for urolithiasis are currently rarely performed.

3.4 Prevention of urolithiasis

Primary prevention (**prophylaxis**) consists in sufficient fluid intake (around 2.5 litres daily), movement, adherence to the principles of healthy nutrition and lifestyle with an emphasis on limiting intake of simple sugars, animal proteins and saturated fats. Adequate consumption of dairy products and foods containing calcium are beneficial (restricted calcium intake leads to an increase in available intestinal oxalate and subsequent increase in oxalate absorption). Specific pharmacological prevention (**metaphylaxis**) is based on the knowledge of the stone composition and the detected metabolic disorder after assessment. The goal is to prevent recurrence of stones. It is necessary to monitor metabolic parameters in urine and blood.

HYDROCELE, EPIDIDYMITIS, ORCHITIS, TESTICULAR TORSION (35)

Morančíková Mária, Fedorko Michal

Hydrocele – fluid collection between parietal and visceral layer of tunica vaginalis.

The typical signs are the enlargement of the scrotum and a feeling of weight or pressure. The enlargment is gradual and there is no palpable change of the testicle. Hydrocele can be congenital, caused by patent processus vaginalis leading to a communication between the scrotal and peritoneal cavity. It usually spontaneously resolves itself within 18 months after birth. Primary hydrocele is idiopathic and secondary type is associated with other pathological conditions such as epididymitis or testicular tumour. Hydrocele can be diagnosed by palpation and ultrasonography, it is important to rule out the presence of a testicular tumor or intestines, therefore ruling out indirect scrotal hernia. Surgery is a mainstay of therapy. Hydrocelectomy is done under general or spinal anesthesia through a scrotal incision. The sac is opened, drained, and partially excised and the edges of are sewn together behind the spermatic cord so that the hydrocele does not reoccur.

Epididymitis – inflammation of epididymis

Clinical hallmarks of epididymitis are pain, swelling, and higher temperature of the epididymis, but the condition can spread to the testicle and affect the whole hemiscrotum. It is usually not possible to differentiate the testis and epididymis during palpation if the whole hemiscrotum is inflamed. Laboratory results show CRP and white blood cell count elevation, there may be a finding of leukocytes in urinary sediment and positive urine culture. The route of infection is the ascent of pathogens from the lower urinary tract. The most common pathogens are Chlamydia trachomatis (accounts for sexual transmission in younger men) and E. coli (in older men related to incomplete voiding due to prostate enlargement and the spread of infection from the urinary bladder). Excluding testicular torsion or tumor is of utmost importance. The use of Doppler ultrasonography is essential. The patient has to be inquired about risky sexual encounters. This condition can be preceded by urethritis, which manifests as dysuria, a burning sensation in the urethra (even between voidings), and urethral discharge. The main focus in elderly men is on voiding symptoms, which usually uncovers some lower urinary tract symptoms or dysuria before the epididymitis. Antimicrobial therapy should be chosen according to the most anticipated pathogen. If there is suspicion of Ch. trachomatis or Mycoplasma genitalium infection, doxycycline is the antibiotic of the first choice. In the case of gonorrhea, parenteral application of third-generation cephalosporine is an effective treatment. If inpatient treatment is necessary, parenteral administration of aminopenicillin with aminoglycoside is the most appropriate therapy. Treatment includes placement of a suprapubic

catheter if high post-void residual urine is detected. Sexual abstinence is necessary until the completion of treatment.

Orchitis – inflammation of the testicle

Isolated orchitis is relatively rare. In most cases, it is accompanied by a concurrent infection of the epididymis. This condition is referred to as orchiepidymitis. It is typically bacterial in origin, and its signs, symptoms, and management are the same as epididymitis. Isolated infection of the testicles is a complication of a viral mumps infection and it affects 20-30% of men with mumps after puberty. In this case, the inflammation is usually bilateral, associated with malaise, fever, preauricular tenderness, and palpable swelling of the scrotum. Ultrasonography of the scrotum is necessary in all cases to rule out a testicular tumor or torsion. Orchitis is managed conservatively, treatment is symptomatic and includes bed rest, local cooling, and scrotal support.

Testicular torsion

Testicular torsion along the axis of a spermatic cord leads to venous and arterial occlusion, with subsequent ischemia and infarction. It occurs mainly in the neonatal period and during puberty. It is caused by the contraction of the cremasteric muscle, which is bound to the spermatic cord. Its spasm can occur during sports, other physical activity, even getting up from a bed, or during sleep. The affected testis is usually twisted medially. Predisposing conditions are cryptorchidism, proximal attachment of tunica vaginalis on a spermatic cord, long mesorchium, or insufficient gubernacular fixation of testis in a scrotum. It is crucial to take action within 6 hours of the onset of symptoms. Irreversible changes in the testicle start to develop after 6 hours, which strongly reduces the probability of saving the affected testicle. The sudden onset of strong testicular pain and swelling are typical symptoms. Pain can propagate to the groin or lower abdomen, and is usually accompanied by nausea and vomiting. It is therefore necessary to examine the genitals in male patients with lower abdominal pain. There is no dysuria or fever. Upon physical inspection, there is visible asymmetric swelling of the scrotum, with or without skin erythema. Palpation of the affected testicle is extremely painful and the testicle itself lays higher in the scrotum (Brunzel sign). Depending on the degree of torsion, the testicle can change its original vertical axis to horizontal. The cremasteric reflex is almost always absent. Elevation of the testis does not relieve the pain as in the case of epididymitis (Prehn sign). Laboratory findings in urine sediment are normal. The absence of blood perfusion of the affected testicle on the scrotal Doppler ultrasonography test is the most important finding in diagnostic work-up. It is useful to compare perfusion on both sides. Besides this, an ultrasound scan can detect other pathology, e.g. hydrocele, scrotal hernia, tumor, or torsion of the

appendix testis. Immediate scrotal explorative surgery is required to untwist the testicle and assess its vitality. Before surgery, manual detorsion may be attempted (with mediolateral rotation) and, if successful, it leads to immediate pain relief. However, even in this case surgery is mandatory. The highest chance for testicle salvage is when the surgery is performed within 6 hours after onset of the symptoms. During surgery, we evaluate the perfusion and vitality of the testicle, in case of torsion it is of livid or even black color. After making sure the perfusion is normal again (it appears as a colour change), we perform orchidopexy, which fixates the testicle to the inner scrotal layer to prevent a recurrence. The contralateral testicle is fixated as well in the same time or as a secondary surgery later. In a case of necrosis, the colour of a testicle after detorsion remains black, orchiectomy is performed as well as orchidopexy of a contralateral testicle.

Torsion of an appendix testis

Testicular and epididymal appendices are small rudimental residues of a Müllerian duct. They usually measure just a few millimeters, but in the case of longer peduncles, there is a possibility of their torsion as well. Clinically there is not much difference from testicular torsion, as there is also acute onset of pain. The swollen appendix is usually palpable by the upper pole of a testicle and its ischemia can be visible through the skin as a „blue-dot sign“. This condition, although it does not pose a risk for testicular vitality, is often hard to differentiate from a testicular torsion. Doppler ultrasonography shows normal testicular perfusion with hypervascularization of an appendix, which is usually clearly visible due to edema. Ischemia of appendix leads to atrophy over time. Because of frequent uncertainty about torsion of the testicle, exploration surgery is often inevitable. The necrotic appendix is then excised. If the diagnosis is clear and the pain is moderate, management can be conservative with scrotal cooling and pain relief with NSAID administration.

BENIGN PROSTATIC HYPERPLASIA (30)

Wasserbauer Roman, Fedorko, Michal

Benign prostatic hyperplasia (BPH) is the most common benign condition that affects men as they age. Hyperplasia of predominantly stromal cells of the prostate leads to the diverse set of symptoms called LUTS (lower urinary tract symptoms). These symptoms are associated with a worsened quality of life. The incidence is increasing with age. At the age of 65, about 30% of men have a worse quality of life and this amount doubles every decade. This progressive disease is characterized by decreased flow of urine, increased post-void residual urine, enlarged prostate and LUTS.

Terminology:

- benign prostatic enlargement (BPE) – increased size of the prostate due to BPH
- bladder outlet obstruction (BOO) – decreased flow of urine without specific reason
- benign prostatic obstruction (BPO) – obstruction caused by BPH

Etiology:

Increasing age and the presence of androgens (testosterone) definitely plays a role, but etiology seems to be multifactorial. Other presumed causes include an increased amount of prostatic stem cells, clonal expansion of proliferating cells, growth reactivation trough embryonic induction and the effect of growth factors.

Pathophysiology:

BPH is a histologic diagnosis defined as an increase in the total number of stromal and glandular epithelial cells within the transition zone of the prostate gland. Hyperplasia is induced by the presence of androgens during puberty and then later in life. Men with defective androgen production or boys castrated before puberty never develop BPH. Growth occurs in the periurethral transition zone of the prostate and leads to compression of the adjacent zone towards the capsule. Prostatic hyperplasia leads not only to BPE, BOO and LUTS, but also to the development of bladder dysfunction and urinary retention (acute or chronic). The mechanism of subvesical obstruction due to BPH involves two different components: mechanic (compression of the urethra by enlarged prostate) and dynamic (increased smooth muscle tone). There are secondary changes of detrusor muscle due to subvesical obstruction and this, alongside with age and altered nervous system function, leads to worse LUTS and complications associated with BPH.

Diagnostics:

Symptomatology - there are various lower urinary track symptoms (LUTS):

- **obstructive symptoms** – weak stream, straining, hesitancy, intermittent stream, terminal dribbling, incomplete voiding, urinary retention, overflow incontinence
- **irritative symptoms** - urgency, frequency, nocturia, urge incontinence

Urinalysis

IPSS (international prostate symptom score): questionnaire consisting of 7 questions evaluating how bothersome are urinary symptoms, 8th question enquires about quality of life

DRV (digital rectal examination): assessment of size, shape, symmetry, tenderness, consistency, palpable nodules and possibly extracapsular extension

PSA: diagnostics and follow-up of patients with prostate cancer

Post-void residual urine (PVR): the amount of urine retained in the bladder after voiding, measured by catheterization or ultrasonography

Uroflowmetry (UFM): measurement of urine flow during urination ,evaluating maximum flow rate and other parameters, voided volume of at least 150ml is required for valid results

Pressure-flow studies: urodynamic invasive examination allowing more detailed assessment of bladder function and evaluating bladder outlet obstruction

Transabdominal ultrasound: measurement of prostate size, configuration, post-void residual urine, examination of upper urinary tract. **Transrectal ultrasound:** more accurate measurement of prostate size. It may reveal suspicious prostatic lesions.

Endoscopy: diagnostics of other causes of subvesical obstruction or hematuria (possibility of a bladder tumor)

Therapy:

Watchful waiting (WW): it can be offered to men with mild or moderate LUTS, that are minimally troubled by their symptoms. In 85% of cases, men are clinically stable for one year, however, progression occurs in 65% of cases over a period of five years. WW includes education, calming down the patient, follow-up and drinking enough fluids.

Pharmacotherapy:

- **alpha-1 blockers**: by antagonizing the effect of endogenous catecholamine on smooth muscle of the prostate and bladder neck they induce relaxation of prostatic muscle tone and therefore effect dynamic component of subvesical obstruction. They are offered to men with moderate to severe symptoms. These medications may cause retrograde ejaculation as an adverse effect.

- **5-alfa reductase inhibitors:** inhibition of testosterone to dihydrotestosterone leads to apoptosis and results in reduction of prostate size as well as lowered PSA levels.
They are suitable for men with moderate to severe symptoms and prostate size at least 40ml. Full effect can be observed after 6 months from the onset of treatment. Low libido and erectile dysfunction are the most notable adverse effects.
- **antimuscarinic drugs:** block the effect of acetylcholine on the muscarinic receptors, thereby reducing detrusor hyperactivity
- **combination therapy:** used for patients who complain of persisting LUTS despite monotherapy, overall effect of pharmacotherapy is increased in combination therapy
- **phytotherapy:** use of herbal extracts, that have anti-inflammatory, anti-androgenic or estrogenic effect, effectivity was not usually proven in vivo and mechanism of action is unclear, nevertheless, some patients benefit from this therapy

Surgical therapy: indications are LUTS refractory to pharmacotherapy, urinary retention, recurrent urinary tract infections, increased post-void urinary volume, recurrent hematuria, bladder stones, renal insufficiency caused by BPH

- **TURP (transurethral prostate resection):** first described in 1932, using monopolar or bipolar resectoscope prostatic tissue of transitional zone is resected. Long-term efficacy and safety of this surgery is evidence-based and it is considered ‘gold standard’ surgical treatment of BHP in patients with prostate up to 70g. Possible complications include bleeding, TUR syndrome (caused by absorption of irrigation fluid through blood vessels, does not occur when bipolar resectoscope is used), urinary retention or urinary tract infection.
- **TUIP (transurethral prostate incision):** this procedure is used in patients with prostate size smaller than 30g, bladder neck is incised using incision knife, removing obstruction at bladder outlet obstruction is removed. Figure 1
- **Open prostatectomy:** the oldest surgical method for treatment of BPH, ideal for patients with prostate larger than 80g. It starts with suprapubic midline incision, after dissection, the bladder wall is revealed and incised, subsequently using index finger prostatic adenomas are separated from the capsule and removed. This method is proven to have long term effectiveness. Figure 2
- **HoLEP:** transurethral procedure that uses holmium laser fiber (wavelength 2140nm), enabling precise incision, dissection and resection. Adenomas are removed as a whole, then moved to bladder cavity and evacuated using morcellator. It is an alternative to open prostatectomy. Other type of laser, such as a thulium laser, can be used.
- **Mini-invasive methods:** procedures done in local anesthesia indicated in patients with smaller prostate size, high-risk patients or young patients who wish to avoid possible complications

such as erectile dysfunction or incontinence. Another currently used technique is called Rezum, it works by inserting water vapor into the prostate and causing coagulation necrosis with subsequent absorption of adenomatous tissue. Furthermore, a method called Urolift (prostatic urethral lift) uses an implant that is permanently inserted in prostatic urethra and pushes the prostate lobes towards capsule. HIFU (high-intensity focused ultrasound), TUMT (transurethral microwave thermotherapy), or aquablation are other surgical treatment options worth mentioning.

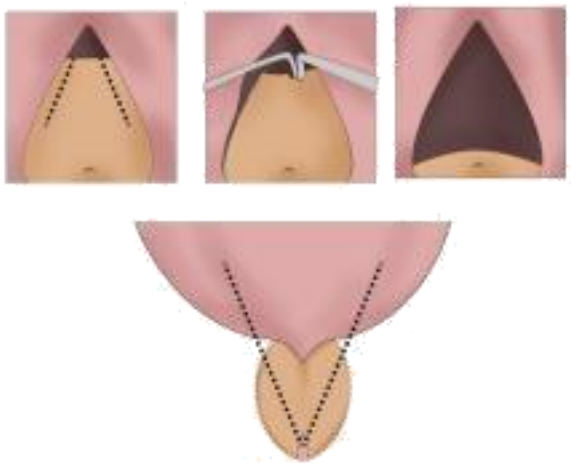


Figure 1: transurethral incision of the prostate (source: Fedorko, 2020)

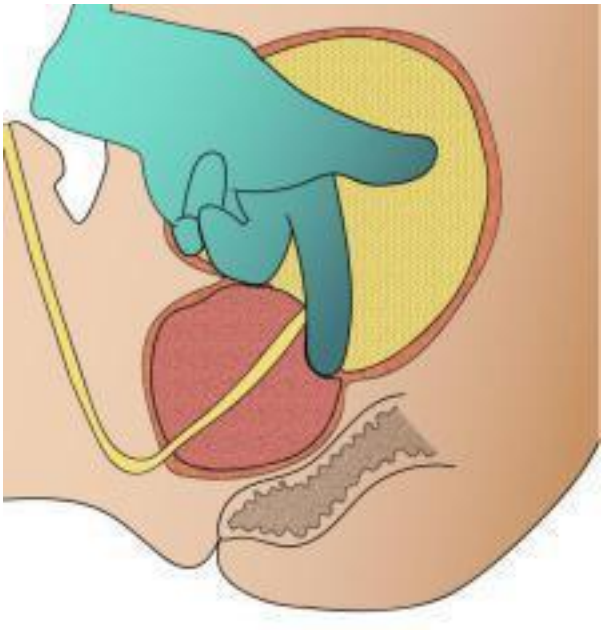


Figure 2: Open prostatectomy (source: Fedorko, 2020)

UROGENITAL TUMORS (31)

Staník Michal

I. PROSTATE CANCER

Prostate cancer (PCa) is the most common solid tumour in men and the second leading cause of cancer-related death, following lung cancer. Risk factors include the presence of male sex hormones and a family history of PCa. The incidence of PCa increases with age, being relatively rare in men under 50.

Pathology

The most common malignant tumour of the prostate is adenocarcinoma (90%). Less common tumours include neuroendocrine carcinoma, which has a significantly worse prognosis and is often resistant to hormone therapy. Unlike other solid tumours where tumour grading is used, PCa is assessed based on the architectural disruption of the prostate glands, using a 1-5 scale known as the Gleason grade. The two most prevalent Gleason grades observed in a tumour are combined to create a Gleason score ranging from 6 to 10, which reflects the tumour's aggressiveness (see Table 1). For simplification, the Gleason score is expressed as the ISUP (International Society of Urological Pathology) grade, which ranges from 1 to 5. ISUP grade 1 indicates an indolent, slow-growing tumour, while ISUP grades 4-5 represent the most aggressive forms of the disease.

Gleason score	ISUP grade	Risk groups
2-6	1	Low-risk
7 (3+4)	2	Intermediate-risk
7 (4+3)	3	
8	4	High-risk
9-10	5	

Table 1 Comparison of Gleason score classification, ISUP grading, and risk group classification for prostate cancer

Clinical manifestation

Early stages of PCa are most often asymptomatic. Urinary symptoms such as dysuria, urgency, and obstructive voiding are nonspecific and can be mistaken for benign prostatic hyperplasia. In locally advanced stages, symptoms may arise from tumour infiltration into surrounding tissues, causing lower urinary tract symptoms, pelvic pain, or hematuria. In metastatic disease, bone pain and pathological fractures are common due to bone metastases. Spinal cord compression can lead to neurological symptoms that require urgent intervention. Other nonspecific signs include weight loss and cachexia.

Diagnosis

The main diagnostic tools include digital rectal examination (DRE), prostate-specific antigen (PSA), prostate MRI, and subsequent prostate biopsy. DRE is performed on every patient, although it only detects around 20% of PCa cases. With a threshold of 3 ng/mL, PSA testing has a high sensitivity for identifying men at risk for PCa. In men with elevated PSA, pre-biopsy MRI reduces unnecessary biopsies and enhances the detection of high-grade tumours while minimising the identification of indolent cancer. Prostate biopsy remains necessary for histological confirmation of PCa. Biopsy methods include transrectal or transperineal ultrasound-guided biopsies and MRI-targeted biopsies, with the latter improving accuracy by focusing on MRI-detected lesions. A CT scan of the abdomen and pelvis and bone scintigraphy are performed to assess for metastatic spread (the lymph nodes and bones).

Early detection and screening

PCa screening based on PSA testing can reduce mortality by identifying cancer at an early stage when treatment is more effective. However, it also poses risks of overdiagnosis, detecting cancers that may not cause harm (indolent cancer), and overtreatment.

Treatment

For localised prostate cancer, treatment options include:

1. **Active surveillance** - recommended for men with low-risk, indolent tumours. It involves regular monitoring with PSA, DRE, and repeat MRI and biopsies.
2. **Radical prostatectomy** – most often performed robotically, involving complete removal of the prostate gland and seminal vesicles (in men with intermediate- to high-risk PCa).
3. **Radiation therapy -** external beam radiation or brachytherapy (implanting radioactive seeds). It can be used alone or in combination with hormone therapy for higher-risk cases.

Metastatic PCa often initially responds to hormone deprivation therapies that lower androgen levels. However, over time, the disease can progress from a **hormone-sensitive** state to **castration-resistant** prostate cancer (CRPC), marked by continued cancer progression despite low levels of circulating androgens. The treatment approaches include:

1. **Androgen Deprivation Therapy** (ADT): It can be achieved through surgical castration (orchiectomy) or medical options, including luteinising hormone-releasing hormone (LHRH) agonists/antagonists and anti-androgens.
2. **Chemotherapy**: Chemotherapy such as docetaxel or cabazitaxel may be used for aggressive disease.
3. **Next-Generation Hormonal Therapies:** Drugs like enzalutamide and abiraterone are used to further suppress androgen signalling in combination with ADT.

II. KIDNEY CANCER

Kidney cancer accounts for about 2-3% of adult cancers worldwide. It is more common in men than women, typically diagnosed between ages 50-70. Risk factors include smoking, obesity, hypertension, and family history.

Pathology

The most frequent benign renal tumours are papillary adenomas, renal oncocytomas and angiomyolipomas. Renal cell carcinoma (RCC) is the most common malignant tumour, comprising subtypes such as clear cell (70%), papillary (15%), and chromophobe (5%).

Clinical manifestation

In the early stages, RCC is often asymptomatic and discovered incidentally during imaging for other conditions. The classic triad in advanced cases includes hematuria, flank pain, and a palpable abdominal mass. Other symptoms can include unexplained weight loss, fever, and fatigue. Paraneoplastic syndromes include hypercalcemia or anaemia.

Diagnosis

Suspicion for RCC usually arises from an ultrasound examination. To confirm the diagnosis, the standard imaging tests are a CT scan of the abdomen or an MRI. A biopsy is rarely done, typically only if thermal ablation is considered or in metastatic settings before systemic treatment. Additionally, a CT scan of the thorax is used to rule out pulmonary metastases.

Treatment

In localised renal cancer, the primary treatment is the surgical removal of the tumour. This can involve a partial nephrectomy, where only the tumour is removed, or radical nephrectomy. In some cases, thermal ablation (radiofrequency or cryoablation) or active surveillance may be used for smaller tumours or patients who are not surgical candidates. In metastatic renal cancer, treatment typically involves targeted therapies (tyrosine kinase inhibitors) or immunotherapy (immune checkpoint inhibitors). Cytoreductive nephrectomy may be considered in select cases, followed by systemic treatment to manage distant metastases and prolong survival.

III. BLADDER CANCER

Bladder cancer (BC) is the 10th most common cancer worldwide, with a higher incidence in men than women. It is most prevalent in older adults after age 65. Smoking and occupational exposure to chemicals like aromatic amines are the leading risk factors.

Pathology

Benign bladder tumours include urothelial papillomas and leiomyomas. The most frequent types of BC are urothelial carcinoma (90%), squamous cell carcinoma (5%), and adenocarcinoma (2%). BC is classified into three stages: non-muscle invasive (NMIBC), muscle-invasive (MIBC), and metastatic disease. NMIBC remains confined to the bladder's mucosa and submucosa, while MIBC invades deeper into the muscle layer.

Clinical manifestation

Painless gross hematuria is the most common symptom, which always warrants thorough examination to rule out cancer. Other symptoms may include frequent urination, urgency, dysuria, and pelvic pain. Signs like lower back pain (due to ureteral obstruction), weight loss, and fatigue may develop in advanced stages.

Diagnosis

Cystoscopy is the primary test, allowing direct visualisation and biopsy of bladder lesions. Urinalysis and cytology help detect blood or cancer cells. Imaging tests like CT urography or MRI evaluate the tumour's extent and assess the upper urinary tract. CT scans of the abdomen and thorax are performed to rule out metastases (the lymph nodes and lungs).

Treatment

Generally, the first step involves transurethral resection of the bladder tumour (TURBT) to obtain histology. In NMIBC, this is followed by intravesical therapy with chemotherapy to prevent recurrence in selected patients. MIBC is usually treated by radical cystectomy combined with cisplatin-based neoadjuvant chemotherapy. A bladder-sparing approach with chemoradiation is an option. After cystectomy, urinary diversion options include an ileal conduit (ureteroileostomy) or an ileal neobladder, which allows normal urination through the urethra. Metastatic BC treatment includes cisplatin-based chemotherapy and immune checkpoint inhibitors.

IV. TESTICULAR CANCER

Testicular cancer (TC) is a rare malignancy, constituting about 1% of all male cancers. It primarily affects younger men, typically between 15 and 35 years old. Risk factors include family history and finding of undescended testis.

Pathology

Testicular cancer is primarily classified into germ-cell tumours and non-germ-cell tumours. Germ cell tumours are further divided into seminomas, which are the most frequent type of TC, and non-seminomas, which include embryonal carcinoma, yolk sac tumour, choriocarcinoma, and teratoma. Non-germ cell tumours, like Leydig and Sertoli cell tumours, are rare.

Clinical manifestation

Testicular cancer often presents as a painless lump or swelling in one testicle, which may be accompanied by discomfort or a feeling of heaviness. Advanced cases might present with back pain, cough, gynecomastia or other systemic symptoms due to metastasis.

Diagnosis

The diagnosis of TC is based on a physical examination, scrotal ultrasound, and serum tumour markers [e.g., alpha-fetoprotein (AFP), human chorionic gonadotropin (hCG), and lactate dehydrogenase (LDH)]. If TC is suspected, a radical orchiectomy via an inguinal approach is performed. CT of the thorax and abdomen is used to assess for metastasis, with the most frequent sites being lymph nodes, pulmonary, and hepatic metastases. Stage I is confined to the testicle, Stage II involves regional lymph nodes, and Stage III indicates distant metastases.

Treatment

Treatment for Stage I involves a radical orchiectomy. Afterward, patients may undergo active surveillance or chemotherapy to prevent recurrence. In metastatic testicular cancer (Stage II and III), treatment involves a radical orchiectomy followed by chemotherapy. In non-seminoma cases, residual disease in the retroperitoneum is often surgically removed through retroperitoneal lymphadenectomy.

UROLOGICAL TRAUMA (36)

Varga Gabriel, Fedorko Michal

Kidney trauma

Etiology: Renal trauma presents around 1-5% of all trauma cases. It results most commonly from road traffic accidents, falls, sporting injuries. Motor vehicle accidents can lead to deceleration injuries causing contusion, laceration of renal parenchyma or renal pedicle injury. Stabbing or gunshot trauma is the most common cause of penetrating injury. Iatrogenic trauma most frequently results from percutaneous nephrostomy, renal biopsy, percutaneous stone extraction, endopyelotomy and endovascular interventions (vascular injury)

Diagnostics: Medical history (flank pain, mechanism of injury, presence of hematuria), physical examination can show abrasions, penetrating gunshot wounds, stabbing or blunt force trauma of the upper abdomen or flank area, injured thorax or broken ribs. Vital signs monitoring is essential. (possible hemodynamic instability)

Laboratory examination: Urinalysis, complete blood count (hematocrit), creatinine (kidney function before the injury)

Imaging tests: abdominal ultrasound (subcapsular hematoma, perirenal hematoma, hemoperitoneum), CT urography (grading of renal trauma, evaluating extravasation of contract dye as an evidence of active bleeding, assessing function of contralateral kidney), single shot intraoperative excretory urography in unstable patients. MRI is as effective in diagnostics of renal trauma as CT, however, it is not routinely used in diagnostic algorithm. The severity of renal injury is classified into 5 grades depending on presence of different characteristics such as perirenal hematoma, laceration of parenchyma, collecting system rupture, injury of the main vessels or shattered kidney.

Conservative management: Blunt renal trauma

Hemodynamic stability is crucial criterion for the management of all renal injuries. Non-operative management has become the treatment of choice for most cases (Grade I-IV). In stable patients, this means a period of bed rest, observation and close monitoring in the ICU unit. Renal injuries grade IV-V in polytrauma patients usually require exploration surgery and nephrectomy. Conservative approach is recommended in case of traumatic unilateralcomplete renal artery thrombosis. Angiography with selective embolization is a minimally invasive treatment used especially in iatrogenic injuries.

Surgical management: Penetrating injuries

Penetrating wounds have traditionally been managed surgically. The goals of exploration following renal trauma are vascular control and renal salvage. Feasibility of renal reconstruction should be judged during the operation. Repair should be attempted in patients with a solitary kidney or bilateral injuries, even in grade V vascular injuries. A nephrectomy is performed if reconstructive procedure is not possible.

Ureteral trauma

Etiology: Trauma to the ureters is relatively rare. Iatrogenic injury during open, laparoscopic and endoscopic surgery is responsible for the majority of cases. The injury is often missed intra-operatively. Penetrating external ureteral trauma is predominantly caused by gunshot wounds. About one-third of cases of external trauma to the ureters are caused by blunt trauma, mostly road traffic accidents. Common mechanisms of iatrogenic ureteral trauma include ligation with a suture, crushing by instruments, partial or complete transection, thermal injury, or ischemia due to devascularization. Gynecological operations are the most common cause of significant iatrogenic ureteral injury. Risk factors for iatrogenic trauma include malignancy, prior surgery or irradiation, diverticulitis, endometriosis, anatomical abnormalities, and hemorrhage.

Clinical picture and diagnostics: In penetrating injuries, the diagnosis is made intra-operatively, while it is often delayed in blunt and iatrogenic cases. The patient may present with flank pain, vaginal or drain urinary leakage, hematuria, fever and uremia. Extravasation of contrast dye on CT scan is the hallmark sign of ureteral trauma. Furthermore, urinoma, hydronephrosis or ascites may be present. In unclear cases, a retrograde or antegrade ureterography can confirm the injury.

Therapy: Immediate intraoperative diagnosis can be managed by a repair and stent placement. A partial transection is repaired by suture and insertion of a JJ stent. Stenting decreases the risk of stricture. Injuries that are diagnosed late are treated first with a nephrostomy tube and reconstruction is performed in 6-12 weeks. The type of reconstructive surgery depends on the extent and localization of the injury.

Bladder trauma

Etiology: Majority of cases (around 75%) are blunt trauma occurring when the bladder is full. Motor vehicle accidents are the commonest cause of blunt bladder injury, followed by falls, industrial pelvic crush injuries and blows to the lower abdomen. 60-90% of patients have associated pelvic fractures. Children are particularly at risk of bladder trauma due to higher position of bladder (above pubic symphysis).

Classification: According to the location, bladder injuries are classified into two distinct types. Intraperitoneal injury usually occurs at the apex of the bladder and leads to leak of urine into the peritoneal cavity. Extraperitoneal injury is almost always associated with pelvic fractures and usually caused by distortion of the pelvic ring or direct rupture due to bone fragments. Iatrogenic injury results from urologic (TUR - transurethral resection) or gynecologic (cesarean section, sling surgery) procedures.

Clinical picture and diagnostics: Gross hematuria is present in 80-95% of cases. Other clinical signs include suprapubic pain, inability to void, edema or hematoma of perineum, scrotum or thighs. Iatrogenic injury is recognized as low return of irrigation fluid during TUR and abdominal distention. During a laparoscopy, bladder injuries may be identified as filling of a urine drainage bag with gas. The key examination in the diagnostic algorithm (following taking medical history and physical examination) is retrograde cystography. CT scan is used as well because of the common association with pelvic fractures, and in fact, CT imaging is routinely used in polytrauma cases. In intraperitoneal injuries, free contrast medium is visible in the abdomen, outlining bowel loops and abdominal viscera.

Therapy: Extraperitoneal injuries are managed conservatively with continuous bladder drainage (7-14 days) using a transurethral or suprapubic catheter and antibiotic prophylaxis. In case of an extraperitoneal injury with pelvic fractures treated by internal fixation, repair of the bladder is recommended. Intraperitoneal ruptures and penetrating bladder injuries are always managed by surgical repair as well. Iatrogenic injuries recognized intra-operatively are immediately repaired.

Urethral trauma

Etiology: Anterior urethral injuries in males are caused by blunt trauma (fall or kick to the perineum). Penetrating injuries are rare (gunshot trauma). Trauma by inserting foreign bodies or penile fractures are also uncommon. Furthermore, transurethral catheterization or endoscopic surgery can lead to iatrogenic urethral injury. Posterior urethral injury in males is associated with pelvis fracture. Urethral injuries in females are extremely rare and the main cause is pelvis fracture.

Classification: Urethral trauma can be divided into injury of anterior urethra (penile and bulbar) and posterior urethra (membranous and prostatic). From a surgical aspect, there can be a complete or partial rupture. In case of complete urethral rupture there is a gap between the ends of the urethra. Partial rupture is associated with urethral elongation, mucosal injury and partial tear in urethral wall.

Complications: Late complications include stricture, incontinence and erectile dysfunction.

Diagnostics: Major symptoms are blood at the meatus, inability to pass urine, palpable overfilled bladder, hematuria, painful urination, ecchymosis, potentially edema in scrotal and perineal region, or difficult catheterization. Upon digital rectal examination there may be finding of so-called high riding prostate. Retrograde urethrography is a standard diagnostic examination to evaluate urethral injury. It can assess extent and localization of injury. Flexible cysto-urethroscopy may evaluate urethral injury as well and additionally, it enables placement of urethral catheter if necessary.

Management of anterior urethral injuries: Suprapubic urinary diversion or early endoscopic re-alignment with transurethral catheter placement are treatment options in case of anterior urethral trauma. Partial or complete ruptures require urinary diversion for at least 2-3 weeks. Penetrating injuries are managed with immediate repair. Small lacerations and stabbing injuries can be repaired by simple closure. Short defects of bulbar (<2-3cm) and penile (<1,5cm) urethra can be managed with spatulation and primary anastomotic repair. In the case of longer defects or apparent infection, a deferred repair is needed.

Management of posterior urethral injuries: It can be divided into immediate repair (< 48 hours after injury), delayed surgery (2 day to 2 weeks after injury) and deferred treatment (>3 months after injury). Immediate management is carried out by ultrasound-guided suprapubic cystostomy. Partial ruptures of posterior urethra can be managed with insertion of suprapubic or transurethral catheter. Retrograde uretrography is used after 2 weeks, until injury is not healed up. Residual or consequent stricture is managed either with direct vision internal uretrotomy or anastomotic urethroplasty, depending on the length of the stricture. Complete rupture of posterior urethra is treated with early endoscopic realignment (approximation of urethral ends over catheter) or early urethroplasty (suturing ends of the urethra). Early urethroplasty is difficult because of poor visualization and the inability to accurately assess the degree of urethral disruption, due to extensive swelling. Standard treatment is deferred one-stage urethroplasty (at least 3 month after injury) - perineal anastomotic repair represents the surgical technique of choice.

ORTHOPAEDICS

CONGENITAL DEFORMITIES OF MUSCULOSKELETAL SYSTEM (2)

Urbášek Karel

Congenital malformations of the musculoskeletal system refer to a diverse range of conditions characterized by abnormal development of the bones, muscles, joints, and connective tissues present at birth. These malformations can affect various parts of the body, including the limbs, spine, skull, and pelvis. The severity and specific manifestations of these malformations can vary widely, ranging from minor single limb abnormalities to more complex systemic and debilitating conditions. The causes of congenital musculoskeletal malformations are multifactorial and can involve a combination of genetic, environmental, and developmental factors. Here we present examples of the most common pathologies.

Pes Equinovarus Congenitus (Clubfoot)

Is most common musculoskeletal birth defect. Overall incidence is 1:1.000 with male:female ratio approximately 2:1. Half of the cases are bilateral. In 80%, clubfoot is an isolated deformity. Genetic component is strongly suggested, unaffected parents with affected child have 2.5% - 6.5% chance of having another child with a clubfoot. In 20% is associated with conditions such as arthrogryposis, diastrophic dysplasia, myelodysplasia, tibial hemimelia, amniotic band syndrome (Streeter dysplasia) etc. And it is called syndromic clubfoot.

Diagnosis is made clinically. It is a complex deformity consisting of Cavus (tight intrinsics, FHL, FDL), Adductus of forefoot (tight tibialis posterior), Varus (tight tendoachilles, tibialis posterior, tibialis anterior) and Equinus (tight tendoachilles). The gold standard treatment is Ponseti method casting (casts changing at weekly intervals with gradual correction of deformity) followed by achillotomy to correct residual equinus. Subsequently, it is necessary to use a special brace until the age of 5 years to maintain the correction. Supplemental surgical procedures such tibialis anterior tendon transfer may be required during treatment to correct residual deformity. The overall success rate of the Ponseti method is over 90%.

Congenital Femoral Deficiency (CFD)

Congenital femoral deficiency (CFD) is a rare birth longitudinal postaxial defect that is characterized by a short femur, associated with hip and knee deformity, deficiency, or instability. Leg length difference can reach 40-50 cm. The more severe types were previously known as proximal femoral focal deficiency (PFFD). The incidence is 1:40.000. The cause of an isolated single-limb abnormality is generally unknown and is usually not associated with a genetic abnormality. CFD is often associated with Fibular Hemimelia. The treatment strategies for CFD depend on the severity. In all

but the most extreme cases, deformity correction surgery and limb lengthening are the preferred treatment method. The use of orthotics is also an option.

Fibular Hemimelia (FH)

Fibular hemimelia is a longitudinal postaxial defect where part or all of the fibula is missing. The Incidence is 1:40.000. Bilateral fibular hemimelia is even rarer. It is assumed that genes guiding the formation of the limb are activated in an abnormal order. FH is not heritable.

Major problems associated with FH are foot and ankle deformities, limb length discrepancy and knee deformities. The treatment strategies FH depend on the severity. Deformity correction surgery (reorientation of ankle joint, talo-calcaneal osteotomy, knee stabilization) and limb lengthening are the preferred treatment method. The use of orthotics is also an option.

Tibial Hemimelia (TH).

Is the least common longitudinal bone defect. The incidence is1:250.000-1.000.000. Almost all TH will need surgery to help children stand and walk. The type of surgery needed depends mainly on how much of the tibia is present and the condition of the knee and ankle joints.

Congenital Pseudarthrosis of the Tibia (CPT)

Pseudarthrosis usually develops prenatally or within the first two years of life. The incidence of CPT is 1:250.000 births. The cause of CPT is currently unknown; however, there is a strong association with neurofibromatosis in 50% of cases and an association with fibrous dysplasia in 10% of cases. The difficulty of treatment lies in the weak healing power at the fracture site, a tendency to refracture after treatment, and the difficulty of stabilizing small osteoporotic bone fragments in small children. The most promising treatment is the "Cross-union" method which combines surgical and pharmacological approaches. With the aim of fusing the tibia and fibula with a bone graft from the iliac crest and fixing the tibia with a telescopic nail and plate.

Radial Club Hand (RCH)

Is a longitudinal deficiency of the radius. It includes a spectrum of deficiencies involving the radius, the radial side of the carpus, and the thumb. The incidence is 1:30.000–100.000. Fifty percent are bilateral and are usually associated with other upper extremity deformities and deficiencies, including absent or hypoplastic thumb, TAR syndrome, Fancomi’s anemia etc. RCH is an aesthetically unappealing deformity that also results in significant functional impairment (due to absent or hypoplastic thumb) and instability and restricted motion in the elbow, wrist, and finger joints. The aim of the surgery is to correct the position of the hand and stabilize it and reconstruct the gripping ability of the thumb.

Achondroplasia

Accounts for about 75% of all dwarfisms, affecting 1 out of 10.000–50.0000 live births. It is caused by a mutation in the fibroblast growth factor receptor 3 (FGFR3) gene that results in impaired endochondral bone growth. Most cases (80%) are spontaneous mutations.

The average adult height for men is 131 cm and for women is 125 cm. Limb deformities, frontal bossing, midface hypoplasia, hyper-lordosis, spinal stenosis, and normal intellect are typical.

For diagnosis physical exam. and X-rays are sufficient, genetic testing confirms the diagnosis.

Treatment: new drug is available, applied daily subcutaneously, it antagonizes downstream FGFR3 signaling and thus alleviates the symptoms of achondroplasia.

Traditionally treatment focuses on any problems or complications and includes guided growth, osteotomy, limb lengthening, spinal cord decompression, etc.

Osteogenesis Imperfecta (OI)

Is an inherited (genetic) bone disorder that is present at birth. It is also known as Brittle bone disease. The incidence is 1:10.000-20.000. Mutations in the COL1A1 and COL1A2 genes cause approximately 90% of all cases. These genetic changes reduce the amount of type I collagen. Patients have soft bones that are not formed normally and fracture easily, and other problems. Signs and symptoms may range from mild to progressively deforming to perinatally lethal. The spine is commonly affected.

The main goal of treatment is to prevent deformities and fractures. Bisphosphonates are used in the symptomatic treatment of OI to reduce the number of fractures. Of the surgical interventions, the most common are deformity corrections secured by pediatric telescopic intra-medullary nails.

Arthrogryposis Multiplex Congenita (AMC)

AMC is a descriptive term for the development of nonprogressive contractures affecting two or more different areas of the body. The incidence is approximately 1:3000. A contracture completely or partially restricts the movement of the affected joint. Specific symptoms and physical findings can differ greatly in range and severity. The legs are affected more often than the arms and the jaws and back may also be affected. Variants in over 400 genes have been identified as responsible for different types of arthrogryposis. One-third of individuals with AMC may have structural or functional abnormalities of the central nervous system.

Treatment: A multidisciplinary approach is best. Standard physical therapy, removable splints and serial casting to mobilize stiff joints is helpful. In some patients, surgery may be necessary to improve joint motion and avoid muscle atrophy.

Congenital Scoliosis (CS)

It occurs in 1 in 10.000 newborns and is much less common than juvenile idiopathic scoliosis. There are 2 main causes of CS: *Incomplete Formation of Vertebrae:* part of one vertebra (or more) may not form completely and hemivertebra developed. More than one hemivertebra can sometimes balance each other out and make the spine more stable.

Failure of Separation of Vertebrae: the spine forms first as a single column of tissue that later separates into segments. If this separation is not complete, the result may be a partial fusion (boney bar) joining two or more vertebrae together, preventing spine from growing on one side.

Combination of Bars and Hemivertebrae: causes the most severe growth problem. These cases can require surgery at an early age to stop the increased deformity.

In rare cases there may be a problem with the spinal cord or nerves that produces weakness, numbness, or a loss of coordination. CT scans and MRI are mandatory in treatment planning.

Nonsurgical Treatment: Braces are not effective in treating the curvature caused by the congenitally abnormal vertebrae, but they are sometimes used to control compensatory curves.

Surgical Treatment: The important goal of surgery is to allow the spine and chest to grow as much as possible. Surgical options include hemivertebra removal and spinal fusion etc.

Myelomeningocele (MMC)

Is the most common central nervous system congenital condition. It is a type of spina bifida. The incidence is 1:1.500. The cause of MMC is still unknown, but it's a complex combination of genetic, nutritional and environmental factors that are being considered. MMC most commonly occurs in lumbar and sacral areas. Hydrocephalus is also a problem.

Treatment involves surgery to repair the opening in spine. (fetal or postnatal surgery). MMC causes moderate to severe disabilities, such as muscle weakness, loss of bladder or bowel control, and/or paralysis. Most common orthopedic complications are hip dislocation, contractures and spine deformities. Most patients are wheelchair-bound, but there are some who are able to walk independently.

SCOLIOSIS AND OTHER SPINE DEFORMITIES (3)

Chaloupka Richard, Ryba Luděk, Parížek Dominik

Scoliosis is a three-dimensional spinal deformity (in frontal, sagittal and transverse planes). Curvature in the frontal plane is called scoliosis, in the sagittal plane lordosis and kyphosis and in the transverse plane it´s rotation of vertebrae. In the forward bend – flexion test, scoliosis is manifested by a prominence (hump), so-called gibbus. It occurs in the population with a frequency of about 3%. The most commonly used classification of scoliosis is based on etiopathogenesis: 1/ Idiopathic (the most common) 2/ Congenital 3/ Neuromuscular (CP - cerebral palsy, muscular dystrophies, myopathies).

Diagnosis is based on anamnesis, clinical examination and imaging. When taking a medical history, we focus on the family anamnesis, in the personal history we focus on the patient's period of psychomotor development. We ask about the patient's current difficulties (pain, respiratory insufficiency, fatigue), previous kinds of treatment and date of the first occurrence of a problem. During the clinical examination we focus mainly on the limbs and spine. We perform a forward bending test (Adams test) and side bends. During the examination we focus mainly on symmetry (Is the spine misaligned? Are the shoulder blades/glutes/shoulders at the same height? Is there a difference in limb length? Is the pelvis oblique?), the condition of the musculature, not forgetting the orientation neurological examination. If a prominence is present, measure it in cm. From the imaging methods we choose X-ray examination, using long formats capturing the whole spine, images are taken standing in anteroposterior and lateral projection, the size of the deformity is determined by the Cobb angle measurement (see figure).

Treatment is either 1/ conservative or 2/ surgical.

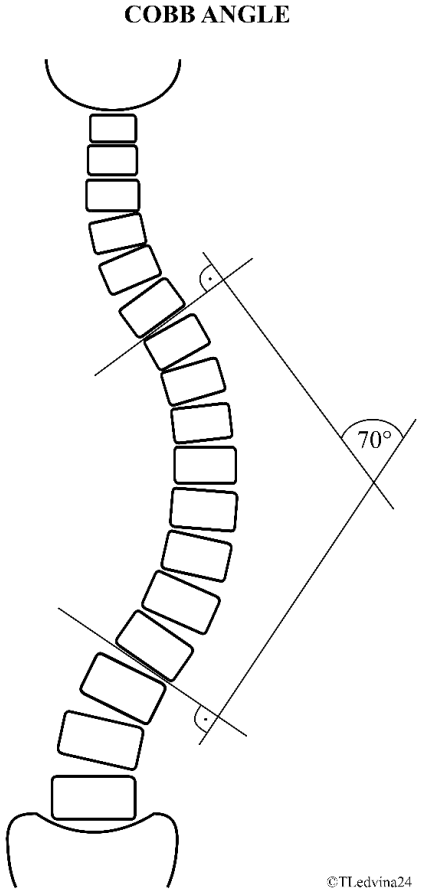
Conservative treatment is chosen for less severe deformities. It mainly includes physiotherapy, continuous daily exercise, change of habits and posture, sparing of the spine, alternating regime. We use bracing during the growth period at curve values of 20-40°, the minimum duration of brace use is 16 hours per day, physiotherapy and daily exercises are necessary. We choose surgical treatment for severe deformities, it serves as a prevention of oppression of internal organs (abdominal, lungs - restriction of breathing functions), prevention of development of spine degenerative changes. The operation is indicated in growing children with a curve above 40°. In children under 10-12 years of age we use GGS (Guided Growth Systems, instrumentation without vertebral fusion – transpedicular screws and rods in majority of cases). For patients over 10-12 years of age, we perform definitive treatment from a posterior approach with transpedicular fixation and vertebral fusion. For idiopathic and congenital scoliosis we make sure to keep instrumentation and fusion as short as possible.

For neuromuscular scoliosis, we choose long construct/intrumentation with the possibility of fixation also to the sacrum and iliac bone (in case of an oblique pelvis or instability of the sitting position, pelvic fixation improves the stability of the sitting position).

Wrong posture is a common cause of spinal deformity. It manifests itself as a flexible deformity with muscular imbalance. The deformity disappears when lying down and when muscles are activated (correction occurs). Treatment consists of rehabilitation, stretching of shortened muscles (especially pectoral and knee flexors), strengthening of abdominal and back muscles. It is important to pay attention to correct posture, continuous daily exercises, correct movement regime, regular changing of activities and positions.

Morbus Scheuermann (kyphosis dorsalis juvenilis) is genetically determined enlargement of the thoracic kyphosis due to incorrect ossification. X-ray imaging plays a key role in the diagnosis; we use long formats in two standing projections. On the X-ray we can observe wedge-shaped vertebrae, disc reduction, and irregularity of the end plates of vertebrae.

For flexible kyphosis (we perform lateral X-ray in lying position with reclination with the wedge at the apex of the kyphosis) in range of 50-70 degrees is the method of choice bracing and physiotherapy during the growth period of the patient. Surgical treatment is chosen when the kyphosis is above 70 degrees.



ORTHOPAEDIC TREATMENT OF DEGENERATIVE AND RHEUMATIC DISEASES (8)

Liskay Jakub, Tomáš Tomáš

Degenerative joint disease (osteoarthritis) is a non-inflammatory degenerative joint disease characterized by degradation of articular cartilage, subchondral sclerosis, osteophyte formation and soft tissue changes involving the synovial membrane, joint capsule, joint ligaments and muscles. It is a very common disease that increases with age, affecting more women with characteristic changes in the small joints of the hands, weight-bearing joints and spine. Risk factors include age, gender, genetic predisposition, biomechanical disorders, trauma, obesity, as well as ethnic and geographical influences. The disease is uncommon in those under 40 years of age but is the most common chronic disease in older patients. More than 80% of people over 75 years of age are affected.
Osteoarthritis is divided into **primary** (cause unknown - affects the small proximal and distal interphalangeal joints of the hands, 1. Carpometacarpal joint of the thumb, cervical C5-6 and lumbar L5-S1 spine, hip and knee joints) and **secondary** (cause is known - metabolic diseases (gout, hemochromatosis), hormonal conditions (acromegaly, diabetes) , recurrent bleeding in the joint, inflammatory process, mechanical factors (intra-articular fractures, coxa vara congenita, conditions after developmental dislocation of the hips, conditions after aseptic necrosis of the femoral head, overweight, after total menisectomy)

Clinical manifestations - at the beginning there is a slight pain in the joint, which increases with exertion and weather changes, morning stiffness, with progression the pain is higher, limping, the patient cannot stand, the muscles react with a protective spasm, the joint stiffens, joint contractures are formed, the joint swells and during movement there are audible jerks.

Diagnosis - the basic imaging method is X-ray, it divides osteoarthritis into I- IV grades according to Kellgren- Lawrence classification, MRI examination.

Treatment - conservative therapy is usually the method of first choice of treatment of osteoarthritis. Conservative treatment is divided into non-pharmacological and pharmacological treatment. Non-pharmacological treatment is based on the modification of the life and work regime, reduction of the vertical load on the weight-bearing joints (weight reduction, canes, crutches), regular exercise, prevention of contractures, appropriate sports (cycling, swimming, pool exercises), physical treatment (magnetotherapy, radiation). Pharmacological treatment has an anti-inflammatory and analgesic effect (Paracetamol, Metamizol). To suppress the inflammatory component, the application of non-steroidal anti-inflammatory drugs (NSAIDs) is appropriate. NSAIDs are divided according to their mechanism of action into cyclooxygenase 1 inhibitors - ibuprofen, indomethacin, diclofenac, and cyclooxygenase 2 inhibitors (preferential - meloxicam, nimesulid, selective - celecoxib).

The use of opioid analgesics (Tramadol, DHC, Durogesis) is possible. Opioid analgesics (Tramadol, DHC, Durogesis) may be used in high grades of osteoarthritis. Pharmacological therapy must be used with caution in view of side effects, drug interactions and associated diseases. Another group of drugs are SYSADOA (symptomatic slow acting drugs in osteoarthritis). These include glucosamine, chondroitin sulphate and diacerein. Corticosteroids have a strong anti-inflammatory effect. Intra-articular administration of depot corticosteroids (e.g. methylprednisone) may provide temporary pain relief. The strong anti-inflammatory effect of corticosteroids is useful in acute synovialitis. Hyaluronic acid provides lubrication of the joint, shock absorption and is an important component of the extracellular matrix of cartilage. Hyaluronic acid derivatives are injected into the joint and are obtained from various sources (cock's comb, bacterial fermentation, etc.).

Surgical treatment - indicated in case of failure of conservative treatment, divided into primary preventive procedures, secondary preventive procedures, palliative procedures and definitive surgical solution.

Preventive primary procedures are procedures to prevent the development of arthrosis. They include the correct treatment of intra-articular fractures, the correct treatment of ligament ruptures and luxations, ligament stabilisation and resurfacing for joint instability, treatment of meniscal ruptures, treatment of chondromalacia deposits, removal of loose bodies, early procedures for congenital and developmental defects ensuring an anatomically and functionally normal joint (especially early surgical treatment of developmental dysplasia of the hip - VDKK , Morbus Perthes, coxa vara adolescentium)

Preventive secondary surgery is surgery in the field of arthritic changes or changes that will clearly lead to arthrosis, with the aim of slowing or stopping the progression of these changes. These include surgical correction of the axis (valgus osteotomy of the proximal tibia at the genu varum, varus osteotomy of the distal femur at the genu valgus, osteotomy of the proximal femur, pelvis.), late procedures in the context of congenital and developmental defects providing correction in order to delay the onset of secondary degenerative changes (e.g. Chiari osteotomy or tectum repairs in developmental hip dysplasia), synovectomy in rheumatoid arthritis.

Palliative surgeries are procedures for advanced arthritic changes that are symptomatic, do not affect the progression of the disease, but delay the need for definitive resolution. These procedures include arthroscopic debridement of articular cartilage and menisci, synovectomy for osteoarthritis, styloidectomy and denervation of the carpus or e.g. Man surgery for hallux rigidus.

The definitive treatment of osteoarthritis is resection arthroplasty (e.g. Keller's operation for arthrosis of the metatarsophalangeal joint of the thumb, Girdlestone operation), arthrodesis, partial joint

replacement (hemiarthroplasty) and total joint replacement, which has the best clinical results especially in the hip and knee joints.

Arthrodesis is the artificial induction of joint ossification between two bones by surgery. The surgical principle is the removal of cartilage debris, the modification of articular surfaces with the revival of bone surfaces, the possible use of bone grafts, the correct axial and rotational positioning with respect to limb function and the provision of resting to solid bone healing by means of stable osteosynthesis (LCP, compression plates, compression screws, staples, possibly biological autograft using a fibula in ankle arthrodesis). Arthrodesis is the method of choice as a definitive solution in the wrist (arthrosis of the RC joint and carpus), the tibiofemoral joint, the subtalar joint as a triple arthrodesis and the metatarsophalangeal joint of the big toe. Arthrodesis is also used as final surgery especially in joint infections in resistant cases.

Rheumatic diseases

Rheumatoid arthritis is an inflammatory disease of the joints of the extremities in which the infectious agents causing the disease have not been identified or cultured. For the actual diagnosis, the following criteria must be fulfilled: morning stiffness for at least 1 hour for at least 6 weeks, swelling of at least three joints for at least 6 weeks, symmetrical swelling for at least 6 weeks, rheumatic nodules, evidence of rheumatic factors, radiographic changes in the hands.

Clinical manifestations - joints have all the signs of inflammation except redness in the acute phase. Subsequently, swelling, subluxation, deformity and ankylosis occur. Some deformities are typical, e.g. "swan neck" in the fingers, ulnar deviation in the metacarpophalangeal joints, palmar flexion in the wrist, flexion contractures in the IP and dorsal in the MTP joints of the foot, semiflexion contractures in the knees and elbows or flexion/adduction in the hips. Life-threatening is instability of the atlantoaxial joint.

Therapy - NSAIDs are the first-choice drugs, DMARDs (disease-modifying antirheumatic drugs) - patients with active synovialitis (Methotrexate, Sulfasalazine, glucocorticoids) are designated for therapy with so-called basal drugs. Surgical therapy - synovectomy is the extirpation of the hypertrophic inflammatory lining of the affected joint(carpometacarpal joints, knee joint) in order to reduce the volume of inflammatory tissue as much as possible and prevent joint destruction , reconstruction of the forefoot and hindfoot (e.g. procedures to correct hallux valgus - arthrodesis of the metatarsophalangeal joint, resection of the metatarsal heads at the flatfoot, arthrodesis of the subtalar joint and ankle), implantation of total replacement of large joints and small joints of the hand (silicone implants), osteotomy, tenotomy.

Ankylosing spondylitis (Bechterew’s disease) is an autoimmune disease with the production of antibodies against its own tissue, where the target tissues are the ligament attachments of the spine

and the sacroiliac joints (SI joints). The result is ankylosis of the spine. Males are predominantly affected, and the disease begins in the 2nd to 3rd decade of life. The SI joints and spine are the main structures affected, but extraskeletal manifestations such as uveitis are also part of the disease; pulmonary restriction is a secondary affliction of reduced thoracic capacity. The underlying process is inflammatory involvement of the intervertebral joints and progressive calcification of the spinal ligaments with the development of spondylophytes. Gradually, with bridging of the intervertebral spaces, calcification of the anulus fibrosus of the intervertebral disc occurs, the final picture of which may be ankylosis of the spine (“bamboo stick"). The disease progresses most commonly in a caudocranial direction.

Therapy - Surgical therapy of spinal deformities in ankylosing spondylarthritis is very challenging. It consists of osteotomy (OT) of the spine with eventual deliberation and is burdened with a high incidence of neurological complications.

NEURO-ORTHOPAEDIC SURGERY (7)

Chaloupka Richard, Ryba Luděk, Parížek Dominik

Neuro-orthopedics is a field that merges neurology and orthopedics, focusing on conditions that involve both the nervous system and the musculoskeletal system. Disorders in this area often require multidisciplinary care to improve motor function, alleviate pain, and enhance overall quality of life. Among the most significant conditions in this area are cerebral palsy, entrapment syndromes, and birth-related brachial plexus injuries like Erb's palsy and Klumpke's palsy.

1. Cerebral Palsy

Overview:

Cerebral palsy (CP) is one of the most common neuro-orthopedic conditions. It refers to a group of permanent movement disorders that appear in early childhood, primarily affecting motor function due to brain injury or malformation during, or shortly after birth. The injury typically occurs either before, during, or shortly after birth. The condition is non-progressive, but its effects can lead to progressive musculoskeletal deformities.

Diagnosis:

The diagnosis of cerebral palsy is often made based on clinical observation of motor development and neurological functions. Symptoms include poor coordination, stiff or weak muscles, and exaggerated reflexes. MRI and CT scans are often used to detect brain abnormalities. Diagnosis usually occurs in early childhood when motor delays become evident. Additional diagnostic tests, such as EEG, may be used to rule out other related conditions.

Treatment:

Cerebral palsy cannot be cured, but treatment focuses on improving the quality of life and mobility of the affected individual. A combination of physical therapy, which is essential and must be long-term (such as the Vojta method), occupational therapy, speech therapy, and medical interventions are used to improve motor function and coordination. Orthopedic surgeries, such as tendon lengthening or correction of bone deformities, may be necessary to manage spasticity and improve mobility. Injections of botulinum toxin (Botox) and intrathecal baclofen therapy are often used to relax spastic muscles. In severe cases, selective dorsal rhizotomy (SDR) surgery, which involves cutting the nerve fibers causing spasticity, may be performed.

Orthopedic Care:

Children with cerebral palsy often suffer from musculoskeletal problems such as scoliosis, hip dislocations, or foot deformities. Regular monitoring by orthopedic specialists is essential for

preventing or correcting deformities, with orthopedic interventions, such as braces or surgeries, used to maintain mobility and function. For lower limb contractures, surgeries such as adductor tenotomy, hamstring lengthening, and Achilles tendon lengthening are indicated by the age of 6, followed by 3-6 weeks of plaster immobilization and subsequent rehabilitation to enable walking.

2. Entrapment Syndromes

Overview:

Entrapment neuropathies are conditions where nerve becomes compressed or restricted, leading to pain, numbness, or muscle weakness. These syndromes can occur in various parts of the body and affect both children and adults. Examples include carpal tunnel syndrome, cubital tunnel syndrome, and tarsal tunnel syndrome. While these conditions are more commonly associated with adults, they can also occur in children, often due to trauma or congenital deformities.

Diagnosis:

The diagnosis of entrapment syndromes is based on a combination of physical examination, medical history, and nerve conduction studies. Electromyography (EMG) and nerve conduction velocity (NCV) tests are often used to determine the extent of nerve damage. Imaging studies, such as ultrasound or MRI, can help identify the precise location of nerve compression.

Treatment:

The treatment of entrapment syndromes varies depending on the severity and location of the nerve compression. Non-surgical options include physical therapy, splinting, anti-inflammatory medications, and corticosteroid injections. In more severe cases, surgery may be necessary to relieve the compressed nerve and alleviate symptoms. Common surgical procedures include carpal tunnel release and cubital tunnel release.

3. Brachial Plexus Injuries

Erb's Palsy

Overview:

Erb's palsy is a type of brachial plexus injury that affects the upper arm and shoulder. It is usually a result of birth trauma, occurring when the baby's neck is stretched to one side during a difficult delivery. This injury primarily affects the upper part of the brachial plexus (nerve roots C5-C6), which control the movement and sensation of the arm and shoulder.

Diagnosis:

Diagnosis is usually made at birth based on the infant's inability to move the affected arm. EMG and nerve conduction studies may be performed to determine the extent of nerve damage.

Treatment:

Treatment for Erb's palsy begins with physical therapy to encourage movement and prevent muscle atrophy. If there is no significant improvement within the first few months, surgical options such as nerve grafts or nerve transfers may be considered to restore function. Many children with Erb's palsy regain partial or complete function with early rehabilitation.

Klumpke's Palsy

Overview:

Klumpke's palsy is a rarer form of brachial plexus injury that affects the lower nerves (C8-T1). It results in weakness or paralysis of the forearm and hand muscles. Like Erb's palsy, Klumpke's palsy is often associated with birth trauma but can also occur in older children due to trauma or stretching of the lower brachial plexus.

Diagnosis:

The clinical presentation includes a "claw hand" deformity, where the forearm and hand muscles are weak or paralyzed. Diagnostic tests are the same as for Erb's palsy.

Treatment:

Physical therapy is the mainstay of treatment to maintain joint mobility and muscle strength. In cases where function does not improve within the first year, surgical intervention may be necessary, such as nerve or tendon transfers to improve hand function.

4. Other Neuro-Orthopedic Conditions

Spina Bifida and Myelomeningocele:

Spina bifida is a congenital condition where the vertebral arch fails to close. Mild forms do not require treatment. If the defect is larger, leading to a herniation of the dural sac and spinal cord, surgical intervention after birth is required. Children with myelomeningocele may suffer from leg paralysis or weakness, bladder and bowel dysfunction, and orthopedic deformities such as congenital clubfoot (pes equinovarus) or scoliosis. Early diagnosis through prenatal screening or at birth allows for early treatment, often involving a combination of surgery, physical therapy, and orthopedic procedures to manage musculoskeletal deformities.

Duchenne Muscular Dystrophy:

Duchenne muscular dystrophy (DMD) is a genetic disorder characterized by progressive muscle degeneration and weakness, primarily affecting boys. DMD requires a multidisciplinary approach that includes physical therapy, corticosteroids to slow muscle loss, and orthopedic surgery to correct contractures or scoliosis.

Spinal Muscular Atrophy (SMA):

Spinal muscular atrophy (SMA) is a genetic disorder affecting motor neurons, leading to progressive muscle atrophy. Orthopedic complications, such as scoliosis and joint contractures, often arise as muscles weaken. Recent advances in gene therapy, such as Spinraza and Zolgensma, have significantly improved the prognosis for children with SMA, but orthopedic care remains crucial to ensure mobility and prevent deformities.

Conclusion

Neuro-orthopedics plays a vital role in treating a wide range of conditions that affect both the nervous system and the musculoskeletal system. Disorders such as cerebral palsy, Duchenne muscular dystrophy, and spinal muscular atrophy require a multidisciplinary approach that involves neurologists, orthopedic surgeons, and rehabilitation specialists. Early diagnosis and intervention are key to optimizing outcomes and improving the quality of life for affected individuals. Effective treatment often involves a combination of physical therapy and surgical procedures tailored to the specific needs of the patient.

ASEPTIC BONE NECROSIS, ENTHESOPATHY AND TENDINITIS (5)

Lukáš Martinek, Luboš Nachtnebl

Aseptic bone necrosis

Aseptic bone necrosis (osteonecrosis) is a degenerative bone disease characterized by the death of cellular components of bone due to interruption of the subchondral blood supply. Synonyms are avascular necrosis or ischemic bone necrosis. It affects predilectively the epiphysis of the long bones of the weight-bearing joints. In the most severe cases, bone destruction or collapse of the entire joint occurs. The most common sites for avascular bone necrosis are the femoral head, the knee joint (femoral condyles), the talus, the wrist region and the humeral head.

Risk factors includes: chemo-, radiotherapy, smoking, intraarticular fractures, arterial and venous abnormalities, intraarticular bleeding, use of high doses of corticosteroids, metabolic diseases (Gaucher disease), dyslipidemia, bleeding disorders, sickle cell anemia, autoimmune conditions, repetitive microtrauma during overuse activities, etc. In a small percentage of cases, mutations in the COL2A1 gene, which affects the production of type 2 collagen, have been shown to have autosomal dominant inheritance. However, in a large percentage of cases the cause is not clear.

Pathophysiologically, a decrease of subchondral blood circulation causes a state of hypoxia in the bone, leading to loss of cell membrane integrity, cell necrosis and an increase in neutrophiles and macrophages. Macroscopically, subchondral bone collapse occurs with subsequent degenerative joint involvement. Macroscopic changes visible on X-ray and CT imaging usually detect more advanced stage of the disease. On MRI, signal changes due to edema and ischemia of fat cells in the bone marrow detect disease earlier.

The clinical state is dominated by pain in the affected area of subjectively varying intensity and often difficult localisation. The early stages of the disease are usually difficult to diagnose by clinical examination, that‘s why there is often a delay in establishing the correct diagnosis. For this reason, it is extremely important to take a correct history of the patient, focusing on the occurrence of the above-mentioned diseases in the past or present and to identify previous traumatic conditions or sports and work stress on the musculoskeletal system.

The most common aseptic bone necrosis:

Aseptic necrosis of the femoral head - often asymptomatic in the early stages. In case of atraumatic etiology, up to 70% of cases are bilateral. Pain localized to the hip and groin already indicates a progressive stage of the disease. Irradiation of pain to the thighs, buttocks and rest pain, signs of stiffness and changes in gait stereotype are often present.

Aseptic necrosis of the knee (condyles of the femur) - characterized by pain in the knee under load and at rest at night. On examination, the pain in the vast majority of cases is localized in the area of the medial femoral condyle (morbus Ahlbäck), imitating meniscal rupture with possible palpation of the painful area during knee flexion with movement deficit. The most common types are spontaneous and post-traumatic. Iatrogenic after an arthoscopic meniscectomy is relatively rare. Treatment depends on the affected area. In general, the strategy for conservative treatment or salvage procedures is to try to revascularise the affected site. However, these procedures have numerous limitations depending on the losalisation.

- ***For the hip joint***, lightweighting and limitation of motion are indicated as part of conservative treatment. Treatment with a combination of bisphosphonates remains controversial, although some papers have shown benefit. Among surgical treatments in the early stages, when the affected area is small, revascularization procedures with decompression of the affected head or in combination with autologous or allogeneic grafts or biologic agents may be used. For larger lesions, if diversion of the supporting surface is possible, rotational osteotomies in the intertrochanteric area can be used. In irreversible conditions with head collapse or failure of previous methods, total joint replacement is the only solution.
- ***For the knee joint (medial femoral condyle)*** in case of spontaneous necrosis, some cases are treated with lightening and physiotherapy with a focus on quadriceps strengthening. In the case of elderly patients in whom spontaneous necrosis is more frequent, unicompartmental joint replacement provides good results. For larger lesions with more degenerative destruction of the entire joint, total arthroplasty is the solution. In younger ones at the precollapse stage, arthroscopy and decompression using an osteochondral autograft may be helpfull. When the mechanical axis is impaired, the procedure often needs to be supplemented with a high tibial osteotomy.

Enthesopathy and tendon inflammations

Tendons and tendon attachments are anatomical structures of the human body providing leveraged transmission of muscle force to the skeleton, thus enabling locomotion. Another function is to absorb shock and limit muscle damage by overloading.

Etiologically, one of the most important factors in the development of the disease is age-related degeneration and frequent excessive mechanical stress. Most often, the development of these diseases occurs with a change in the style or intensity of physical activity.

Pathological-anatomical changes include microcracks, dystrophic changes, mechanical damage of Sharpey fibers, periostitis, ossifications, and metaplastic changes.
Diagnosis consists in taking a proper history of the patient. We are interested in trauma, cyclic mechanical overload with regard to jobs, sports activities. With age-related degeneration, patients may not experience any changes in movement habits or activities. On clinical examination, we can see palpatory tenderness, swelling, crepitus, and positivity of tension and resistance tests.
On imaging, we are interested in changes in the skeleton, ossification or calcification in the tendon or tendon attachment, or changes in the tendon-to-bone transition and the formation of traction spurs. In the ultrasound image, inflammatory changes are visible by hypoechogenicity of the image, we can observe thinning, echogenicity changes in degenerative diseases, hyperechogenic calcifications, fluid rim in tenosynovialitis, or neovascularization in the ultrasound image. MRI examination has significantly improved the diagnosis of tendon pathologies. It allows to differentiate tendinosis, peritendinitis or partial tendon rupture.
Treatment options depend on the affected area and the patient should always be warned of the very likely possibility of a long healing process. When treatment is initiated, it is ideal to eliminate processes causing the difficulty. Calmness and elimination of congestion relieves symptoms and starts the reparative processes. Modification of workload, training activities, sports equipment or exercise regimen at work is necessary. Long-term fixation is inappropriate due to the possibility of atrophy. Targeted rehabilitation is aimed at increasing tensile strength and developing adaptation to the activity performed. Stretching, eccentric exercise with mechanical cooling after the performed exercise is important. Exercising in cycles of increasing load will gradually eliminate symptoms as the patient is exposed to the load. Among physical methods, therapeutic ultrasound, electrotherapy, magnetotherapy, cryotherapy in the initial stages and thermotherapy in the later stages of the disease are used as an adjunct. Non-steroidal antiinflammatory drugs are generally recommended in the initial stages of the disease. Local application of corticosteroids may be used if initial treatment fails. Application should be limited to 2 to 3 doses to an identical area with a minimum interval of 6 weeks. After their application, intense exertion should be avoided for some time. Some areas - Achilles tendon, ligamentum patellae are contraindicated for corticosteroid application due to the possibility of tendon rupture. If conservative therapy is ineffective and symptoms persist beyond 6-12 months, surgical treatment is indicated.
The most common enthesopathies and tendonitis and their treatment:

Radial and ulnar epicondylitis of the humerus (tennis or golfers elbow) - in addition to racket sports players, it affects also patients performing repetitive unilateral movements in the workplace

(assemblers, plumbers, etc.). The pathological process mainly affects m. carpi radialis brevis in the case of tennis elbow, m. pronator teres and m. flexor carpi radialis in the case of golfers elbow. It is characterised by collagen fibre disruption, neovascularisation and mucoid degeneration. It affects the outer respectively inner side of the elbow with the possibility of irradiation proximally and distally. Typical is pain of the epicondyle and positivity of resistance tests - chair test, painful extension or flexion of the wrist against resistance. Therapy consists in the acute phase in pain relief - NSAIDs, icing, resting the limb, short-term use of a brace is usually appropriate. If the pain persists, local application of corticosteroids is indicated. After relief of acute manifestations, the rehabilitation process is followed by physical therapy (ultrasound, electro-magnetic therapy). If conservative treatment fails, surgical treatment may be indicated, consisting in the shift of the origo with débridement and distalization of the tendon, usually accompanied by resection of part of the ligamentum anulare in the case of tennis elbow.

Peritendinitis of the Achilles tendon - the maximum painfulness is localized 3-5 cm above the tendon, the tendon on the calcaneus is also painful. A single overload leads to the development of acute peritendinitis, diffuse seepage and swelling. The tendon is painful to pressure, crepitations may be palpable. In chronic state, the pain is less intense, swelling may be absent, spindle-shaped swelling may be present. Therapy consists in interruption of activity, general or local using of NSAIDs. Local application of corticosteroids is contraindicated. Treatment of the chronic form is more difficult, sometimes rigid fixation is necessary. Physical therapy is also helpfull. When conservative treatment fails, peritenonectomy is indicated. However, the results of surgical treatment are far from 100%.

PAEDIATRIC ORTHOPAEDIC CONDITIONS OF HIP JOINT (6)

Václav Ondrej, Urbášek Karel

A limping child is one of the most common reasons for visiting a pediatric orthopedist. In addition to the injury, we always think about hip disease. The trouble may be caused by benign transient synovitis, or it may be a serious problem like SCFE that can affect future quality of life, all the way up to life-threatening septic coxitis.

Dysplasia of the Hip (DDH)

DDH is a disorder of hip joint development occurring both prenatally and postnatally, with an annual incidence of 10-20 per 1000 newborns. It represents a group of various forms – may manifest as **acetabular dysplasia** (disorder of the acetabular development where the "steep" bony roof does not properly cover the femoral head) which can be associated with a certain degree of hip joint decentration – **subluxation** (partial dislocation) or **luxation** (complete dislocation) of the femoral head, with the need to further differentiate between reducible dislocations (so-called unstable hip) and irreducible dislocations.

The disease typically exhibits a dynamic evolution, sometimes pathology identified at birth spontaneously corrects, while at other times, the decentration may worsen.

The etiopathogenesis of DDH remains unclear, favoring multifactorial theories involving genetic, mechanical, hormonal, and constitutional factors.

In Czech Republic, the diagnostic process is part of the mandatory newborn screening. Early detection shortens the treatment duration and reduces the need for surgical intervention, thereby decreasing the risk of early degenerative changes in the hip joint (pre-arthrosis) in the future. The process involves clinical and sonographic examination, known as the **triple stage examination**. The initial examination takes place at birth, the second at 6-8 weeks of age, and the third at 3-4 months. In medical history, we explore the occurrence of disease in the family and the course of pregnancy. During the clinical examination, we observed the positioning and movement of the lower limbs and muscle tone, an indirect sign may be asymmetry of skin folds. The fundamental diagnostic maneuver is the Barlow sign – during flexion of the hip and knee joints, the knee on the dislocated side is lower; the Barlow test (dislocate) – applying pressure along the longitudinal axis of the thigh and adducting the hip leads to the femoral head slipping out of the acetabulum; and the Ortolani test (reduce) – moving the hip from adduction to abduction leads to reduction of the femoral head into the acetabulum with an accompanying palpable phenomenon in the case of a reducible dislocation.

The goal of ultrasonographic examination is to elucidate morphological changes in the hip joint, with findings categorized according to Graf's classification, from Type I (physiological) to Type IV (dislocation).

After introduction of national screening program, the radiographic examination is indicated only in cases of diagnostic uncertainty or for treatment monitoring, due to radiation exposure and the predominance of the non-ossified skeleton.

The aim of therapy is to achieve a stable and properly centered femoral head in the acetabulum.

Conservative treatment should be initiated as soon as possible after birth, utilizing so-called abduction devices, keeping femur correctly centered in the socket until proper development is completed. In early stages (from birth to 3 months of age), the **Frejka pillow** is used, and in later stages (3 months to 1 year of age), **Pavlik harness** is used. In cases of irreducible dislocation or persistent hip joint instability, **vertical (overhead) traction** is indicated, which involves continuous pulling of the lower limb using weighted loads to release juxtaarticular contractures, followed by repositioning of the femoral head into the acetabulum. The traction regime is conducted during hospitalization and followed by arthrography (contrast X-ray examination of the hip joint) under general anesthesia, to reveal non-ossified articular structures. If successful repositioning is achieved, the limb is fixed with a plaster cast for approximately 6 weeks.

Surgical therapy, an open hip reduction, is reserved for cases where reduction cannot be achieved or maintained conservatively. It involves joint revision, removal of reduction obstacles, and reduction of the femoral head, with possible additional procedures such as osteotomy of the proximal femur and/or pelvis.

Complications of DDH depend on the extent of joint damage. Generally, DDH is considered a pre-arthritic condition with the risk of early degenerative changes in the hip joint and development of coxarthrosis. A feared complication of DDH therapy is avascular necrosis of the femoral head.

Legg-Calvé-Perthes Disease (LCPD)

This is an idiopathic aseptic (avascular) necrosis of the femoral head, with an annual incidence of 1-25/100 000. It affects children aged 4 to 12, with the peak occurrence between the ages of 5 and 8. Boys are more commonly affected, and approximately 10% of cases are bilateral.

The **etiopathogenesis** remains unknown, hypothesis involves the role of coagulation factors.

The first subjective symptom is usually load-related joint pain, often initially referred to more distal areas (thigh, knee). **Clinically** we can explore restriction in the range of motion of the hip joint and a characteristic "Perthes limp," which involves limping on the affected limb due to a combination of antalgic and Trendelenburg gait caused by hip abductor muscles insufficiency.

Paraclinical examination is based on X-ray imaging in 2 projections – anteroposterior and the so-called Lauenstein or "frog-leg" view. In the early stages of the disease, skeletal changes may not be visible on standard X-rays. MRI or bone scintigraphy, which allow for early diagnosis and prompt treatment, have higher sensitivity. Ultrasound examination often reveals synovitis with intra-articular effusion.

The **course** of the disease is individual, typically lasting several months and progressing through four stages - the stage of necrosis and fragmentation, during which the femoral head becomes mechanically vulnerable and may deform or collapse, followed by the stages of re-ossification and remodeling, during which the bone structure is reconstituted.

In the **differential diagnosis**, transient synovitis, SCFE, infectious conditions such as septic arthritis and osteomyelitis of the proximal femur, and rare forms of skeletal dysplasia (e.g., spondyloepiphyseal dysplasia) must be ruled out.

Treatment is based on the "containment" principle, which involves maintaining full coverage of the femoral head by the acetabulum to prevent deformity and its associated consequences. This can be achieved either conservatively or surgically, with the treatment method chosen based on the severity of the disease and the patient's age.

Generally, for children under 6 years of age, conservative treatment predominates. This includes relieving weight from the affected limb using crutches, physical rehabilitation, and possibly orthotic devices (e.g., the Atlanta brace). For children over 6 years old and/or with significant involvement of the femoral head, surgical treatment may be necessary. This typically involves relatively complex re-directional osteotomy of the proximal femur and/or pelvis.

Complications and consequences of the disease include femoroacetabular impingement due to joint surface incongruence from bone deformity, and the early development of secondary coxarthrosis.

Slipped Capital Femoral Epiphysis (SCFE, Coxa Vara Adolescentium, CVA)

SCFE is an acquired hip joint condition characterized by idiopathic non-traumatic slip of the femoral head (epiphysis). It typically occurs in obese individuals (more frequently in boys) during the period of growth acceleration (ages 13-15 for boys and 11-13 for girls) with an incidence of 1-10 per 100 000 children. Approximately 30% of cases are bilateral. The etiology is unknown, but hormonal influences are evident. SCFE is more frequently observed in individuals with adiposogenital dysmorphia. The pathogenesis involves transient subcapital growth plate weakening, resulting in slippage of the femoral head into retroversion and varus (backward, downward and medial) during period of rapid growth, often associated with significant overweight (high BMI).

Typically, the condition is categorized as:

Chronic: symptoms develop over weeks to months (groin pain, often referred to the thigh and knee, restricted hip joint movement), a hallmark is so-called Drehman sign (flexion of the hip joint is only possible with external rotation)
Acute: prodromal symptoms develop for 2-3 weeks, often followed by sudden deterioration accompanied by severe pain like that of a fracture (fracture-like pain)
Acute on a chronic basis: long-term issues with sudden worsening, with radiographic evidence of remodeling changes in the growth cartilage
Diagnosis involves **clinical** examination and **radiographic** imaging of both hips (to compare sides and assess the risk of bilateral occurrence) in two basic projections – anteroposterior and axial (abduction). From both projections, the Southwick angle (HSA, head-shaft angle, between the axis of the femoral diaphysis and epiphysis) can be calculated. The degree of slippage determines the severity of the condition, classified as Grade I (mild, 0-30°), Grade II (moderate, 30-60°) and Grade III (severe, >60°). The term "pre-slip" is used to describe changes in the growth cartilage without measurable slip.
Differential diagnosis includes physeal trauma (epiphyseolysis), as well as Legg-Calvé-Perthes disease or infectious hip joint conditions such as osteomyelitis or septic arthritis. A thorough medical history is essential, considering any potential trauma and the patient’s age.
The principle of therapy is to restore the position of the femoral head and stabilize the growth plate. For stable Grade I slippage, fixation with Kirschner wires or screws ("pinning in situ") is used. For more severe slips, corrective osteotomy of the proximal femur is necessary. Only in unstable (acute) slips can a closed reduction and pinning with Kirschner wires be performer.
Complications include **chondrolysis** – necrosis of the articular cartilage, which is often spontaneously reversible, and the feared **avascular necrosis** of the femoral head due to damage to the retinacular vessels supplying the epiphysis. This can lead to secondary deformity resulting in femoroacetabular impingement and the development of early degenerative changes in the articular cartilage (pre-arthrosis).

BONE TUMOURS – TYPES, DIAGNOSTIC, TREATMENT MODALITIES (1)

Marian Kubíček, Lukáš Pazourek

According to the WHO classification of 2020, primary bone tumours are divided into 9 groups according to the type of cell population that the tumour generates and more than 20 subtypes are described.

Detailed knowledge of the WHO classification is beyond the scope of this paper.

Basic subdivision of bone tumours including an overview of the most common types:

A. BENIGN

- ***chondroma (osteochondroma/enchondroma), osteoma, osteoid osteoma, …***
- SEMIMALIGNANT (locally aggressive) - benign histological picture of the tumour, aggressive growth, high tendency to local recurrence when intralesional resection is performed, rarely can form pulmonary metastases - ***giant cell tumour of bone, chondroblastoma, osteoblastoma, ...***

B. MALIGNANT

PRIMARY

- ***osteosarcoma (OSA)*** - the most common bone sarcoma in children and adolescents (usually 2nd decade), typically affects the metaphyses of the long bones in the knee (distal femur, proximal tibia)

  chondrosarcoma (CHoSA) - the most common sarcoma in adults, affecting mostly the flat bones and proximal parts of the limb skeleton (typically pelvis, scapula, femur and proximal humerus)

  Ewing's sarcoma (ES) - the most common primary malignant bone tumour under 10 years of age (usually 5-25 years), typically affecting flat bones (pelvis, scapula) and the metadiaphyses of long bones
- haematological malignancies - they fall within the competence of haematooncology, the basis of their therapy is systemic treatment, in some cases radiotherapy is also used, the surgeon is mainly involved in the management of pathological or impending pathological fractures - ***multiple myeloma*** (the most common primary malignant bone tumour representing up to 1% of all malignancies), ***primary bone lymphoma, ...***
- - other very rare ones - ***chordoma, adamantinoma, angiosarcoma, ...***

SECONDARY (metastatic bone disease)

- typically ***breast*** *cancer* (osteolytic/mixed lesions), ***prostate*** cancer (osteoplastic lesions - low risk of pathological fracture), ***lung*** *cancer* (osteolytic/mixed lesions), ***kidney*** and ***thyroid*** *cancer* (osteolytic lesions, high vascularity)

Bone tumours can be lined up according to their incidence as follows: benign > secondary malignant > primary haematological malignancies > primary sarcomas (rare, representing only 0.2-0.3 % of all malignant tumours, up to 100 cases diagnosed annually in the Czech Republic)
The basis of diagnosis is a well taken medical history, clinical and paraclinical examination. **Pain** is one of the most common symptoms, in malignant tumours it is often nocturnal, intermittent, localized in depth and gradually worsening. Later, swelling, redness, palpable pain resistance or impaired limb function may occur. Malignant tumours are usually accompanied by general non-specific symptoms (fever, loss of appetite, weight loss, leukocytosis, increased sedimentation, elevation of CRP, ...). Bone tumours may be clinically silent for a long time and the first manifestation may be a pathological fracture or, in the case of malignant forms, distant metastases. Especially in benign and slow-growing lesions, they are also rarely captured as incidental findings on imaging modality. The basis of non-invasive paraclinical examinations is **X-ray** or supplemented by MRI/CT examination. Malignant tumours are characterized by an aggressive growth, periosteal reaction, erosion of cortex and endosteal irregularities (scalloping). As part of staging, lung X-ray/CT, skeletal scintigraphy, and abdominal ultrasonography are supplemented. The collection of material for histological examination can be performed by needle biopsy or excisional biopsy. Bone tumours are a rare and often neglected diagnosis. It is necessary to think of this disease in differential diagnosis - i.e. to have an oncological reflex. In particular, it is important to avoid prolonged ineffective conservative therapy without having an X-ray or other imaging modality done.
Treatment should take place in specialized centers under the direction of an experienced multidisciplinary team. The treatment strategy is chosen individually for each patient, often based on the decision of a multidisciplinary tumour board.
Local treatment options include mainly surgery, radiotherapy and methods such as embolization or radiofrequency ablation. According to the oncological radicality, surgical resection can be radical, wide, marginal or intralesional and according to the degree of mutilation we distinguish:
A, ablative procedures (amputation, exarticulation)
B, limb-preserving procedures (curettage with cemento- or spongioplasty, resection without replacement or with replacement using bone graft or endoprosthesis).
In **systemic therapy**, chemotherapy, hormone therapy, bisphosphonates and biological therapy are

mainly used. In general, the principles of therapy vary according to the biological nature and type of tumour.

OSA is a radioresistant tumour. The method of choice for low malignant OSA is radical surgery. Treatment of highly malignant OSA is multimodal in the sequence: neoadjuvant chemotherapy - surgery (wide resection) - adjuvant chemotherapy.

CHoSA is a chemo-/radioresistant tumour. The method of choice is radical surgery.

ES is characterized by high chemo/radiosensitivity. After combined neoadjuvant chemotherapy, local treatment takes place. Surgery is preferred over radiotherapy. Consolidation treatment is followed by adjuvant chemotherapy/radiotherapy.

Bone metastases - indication for surgical treatment is most often pathological or impending pathological fracture, less often resection of solitary or oligometastatic lesions in some types of tumours. Bone metastases are surgically treated according to the extent of the disease and the prognosis of the patient. In the case of bone solitary metastases and good prognosis of the underlying disease, care is taken to ensure radicality of surgical therapy. Patients with multiple metastases and a less favorable prognosis undergo the simplest possible procedure with early mobilization. The basis of treatment is of course systemic oncological treatment of the underlying disease. Radiotherapy is also often used.

Benign affections can be treated by intralesional procedures, most often by curettage of the lesion. To reduce the number of local recurrences, it is advisable to combine curettage with the use of local adjuvants (cryosurgery, application of phenol, bone cement, argon beam, ...).

A large proportion of benign tumours do not need surgical treatment or histological verification and are often only observed.

In the case of localized forms of highly malignant OSA and ES, approximately 70-75% of patients survive 5 years. If metastases are already present, approximately 30% of patients survive 3 years. The prognosis for low malignant CHoSA is favorable if wide resection is possible. 90% of patients survive 10 years. In highly malignant forms with a higher risk of dissemination and no possibility of systemic treatment, the prognosis is significantly worse. Survival time in secondary bone tumours depends on the extent of the underlying disease and the treatment options. With advances in systemic cancer treatment, survival of patients with disseminated oncological disease is generally prolonged. The goal of the orthopaedic surgeon is to ensure the quality of life of these patients.

Bone tumours in the spine form a separate chapter. The spine is a typical site of bone metastases and multiple myeloma, on the other hand, the occurrence of primary bone sarcomas is very rare. The main difference from other sites is mainly in the principles of surgical therapy.

Patients **without nerve findings** undergo a multidisciplinary tumour board meeting where further management is recommended. If a **progressive, severe nerve finding** is present, surgery is performed within 24 hours of the onset of plegia or severe paresis. Surgery is performed by an anterior approach (resection of the body, adjacent discs, replacement with special cages), posterior (Th and L spine, transpedicular fixation) or combined approaches. Of course, systemic oncological treatment and radiotherapy are also an integral part of the care and are indicated based on histological examination and extent of the disease.

BACK PAIN – DIFFERENTIAL DIAGNOSIS (4)

Chaloupka Richard, Ryba Luděk, Parížek Dominik

The basic step in the differential diagnosis of back pain is a thorough examination. It includes medical history, physical examination, imaging methods and laboratory tests.
Medical history: Was there an injury? Is the pain at rest, at night (tumors, inflammations)? Assess pain intensity (e.g.: VAS - visual analogue scale 0-10) and character of pain.
Physical examination: we evaluate the shape of the spine and its deformities (scoliosis?), the condition of the muscles (symmetry, amount of muscle mass, spasm), mobility of the spine, limbs (difference in length?), orientation neurological examination (is it a root irritation? is the sensitivity (a)symmetrical? weakening of the limbs?)
Imaging methods: X-ray examination in standing position in 2 projections (anteroposterior and lateral), especially for the lumbar spine. In trauma cases we need X-ray and CT examination, in some cases MRI. For nerve involvement, inflammation, tumors we indicate MRI scanning. Bone densitometry is useful for the diagnosis of osteoporosis.
Laboratory tests: used mainly to exclude inflammatory etiology, i.e. KO and CRP - blood count and C reactive protein. In osteology, these are bone remodeling markers (BTM - Bone Turnover Markers).
Pain is divided according to its duration into acute (up to 3 months duration) and chronic (over 3 months).
According to the etiology, we divide pain into nociceptive, neuropathic and mixed. Nociceptive pain is caused by irritation of pain receptors by trauma, inflammation or tumor. Neuropathic pain is caused directly by nervous system involvement (damaged nerve). Back pain may be isolated, without irradiation (cervicalgia, thoracalgia, lumbalgia), or may radiate to varying degrees to the extremities (pseudoradicular, radicular pain). Pseudoradicular pains do not follow a root distribution, they are only in the proximal parts of the limbs. Radicular irritation has a typical localization (according to the dermatome), in the same place it can be accompanied by tingling, burning. With severe nerve oppression, there may neurological deficiency as hypesthesia or paresis.
The impairment may be functional, structural or combined. In functional impairment, we assess mobility, range of motion and muscle status (symmetry, muscle strength). Structural impairment is mainly revealed by imaging (X-ray, CT, MRI, densitometry).
Causes of back pain according to the affected tissue: vertebrogenic, discogenic (disc prolapse), myogenic (most common, muscle imbalance), neurogenic, vascular, pain transmitted from internal organs (e.g.: renal colic), psychosomatic, psychogenic (rare).

Causes of back pain according to the pathological-anatomical division: congenital defects (e.g.: scoliosis), degenerative disorders, tumors (most commonly metastases), injuries, metabolic diseases, inflammatory diseases. Inflammatory diseases are divided into rheumatic inflammation (rheumatoid arthritis, m. Bechterew's), non-specific inflammation (caused most often by staphylococci) and specific inflammation (most often TB - tuberculosis).

We always treat back pain according to the etiology. In the differential diagnosis of back pain, we must first exclude severe causes - trauma, inflammation, tumor or pathological fractures in osteoporosis and tumors. The basis of treatment is rehabilitation, paying attention to correct posture, regular stretching and strengthening of the abdominal and back muscles. It is important to exercise daily. Rest only for the necessary time (few days), then active rehabilitation and return to normal activities as soon as possible. Regimen measures such as sparing of the spine and changing positions are important. Medication and braces are used only temporarily.

We indicate surgery when there is a clear cause of pain - injuries, inflammation, tumors, deformities, spinal instability, worsening of neurological findings. In the cervical spine, treatment from the anterior approach with vertebral fusion predominates. In the thoracic and lumbar spine, the posterior approach with transpedicular fixation and vertebral fusion predominates.

TRAUMATOLOGY

NECK INJURIES (54)

Urbanek Libor

In patients with a neck injury, only soft tissues or also deeper structures can be affected - vascular injuries and injuries to the proximal GIT and airway may be life-threatening

The main causes of neck injuries include:

- Traffic accidents
- Professional injuries
- Sports and leisure activities
- Hobbies
- Crimes
- Suicides

We divide neck injuries into 2 main groups:

- Blunt trauma – skin surface without discontinuity
- Sharp trauma – characterized by open wounds affecting the skin and underlying tissues in various depth

Blunt neck injury

- No open wounds
- Soft tissues are damaged by external pressure against underlying bony or cartilaginous structures of the larynx and spine
- Simple contusions are very common
- Small or weak external signs may not correlate with the severity of the injury (minimal skin changes, no visible hematomas, swelling, etc.)
- We must rule out lesions of deep neck structures (main vessels, larynx, trachea, spine, proximal GIT) always in patients with a history of blunt neck trauma or the possibility of it
- Acute suffocation can occur as a result of edema, hematoma, or traumatic structural changes

Injuries to the larynx

These injuries can be both closed and open. A frequent etiology is suicide or as a result of interpersonal conflicts. The thyroid cartilage is most often damaged. Damage to the larynx can result in severe breathing problems, up to apnea, and even death. Blunt trauma to the larynx may be followed by reflex cardiac arrest. Trauma can also cause edema or hematoma, which will lead to narrowing of the airway. In severe neck injuries, destruction of the larynx structure, mucosal

disruption associated with severe trauma and bleeding to the airways, and cartilage fractures may occur.

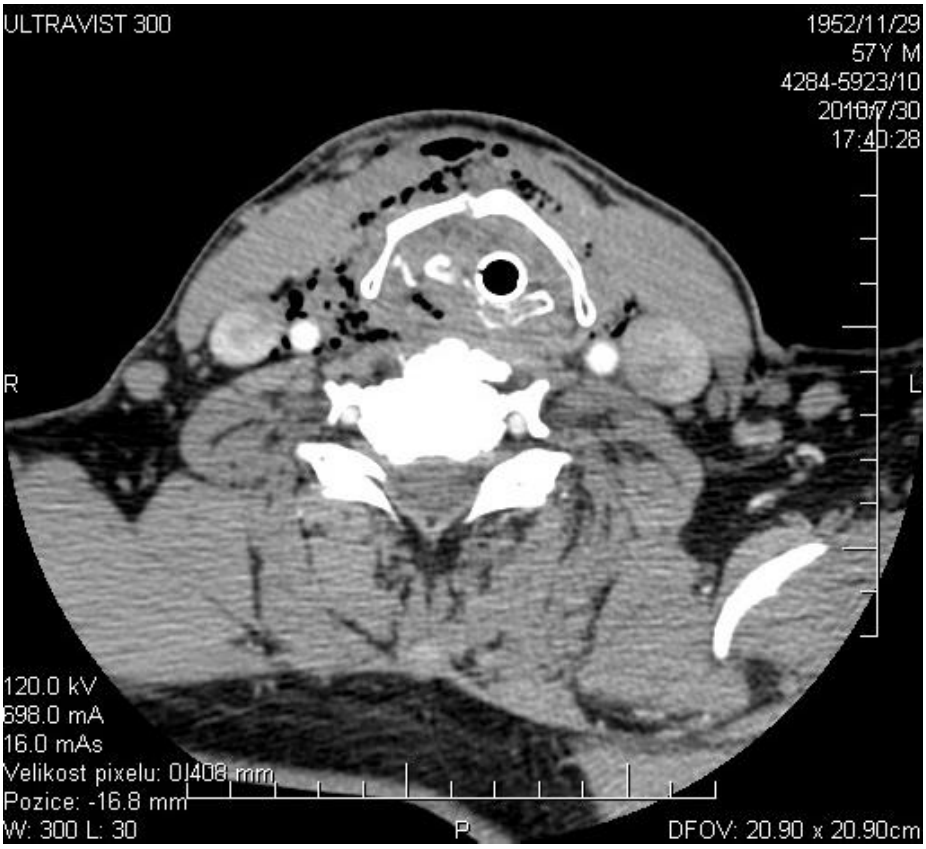
Sharp neck injury

Sharp trauma causes open wounds. These can be of variable depth and direction and can therefore affect different structures and organs of the neck. Heavy bleeding can occur especially when large vessels are involved (opening of the ACC leads to death within 30 seconds without emergency first aid). Various types of wounds such as vulnus lacerum, scissum, sectum, contusolacerum, etc. may occur according to the traumatic mechanism.

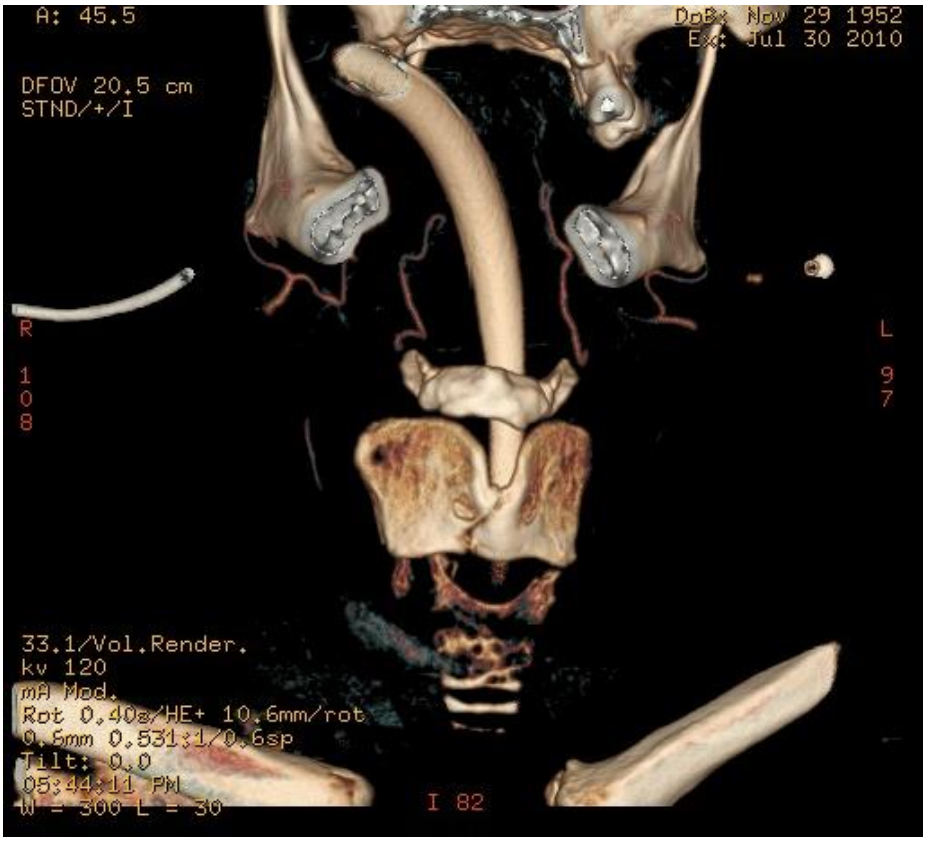
Neck injury management

As part of the treatment, it is primarily necessary to control bleeding, which can be life-threatening. Furthermore, possible suffocation must be urgently managed. It is mostly difficult to secure the airways in the case of primary or secondary obstruction (edema, hetatoma, lacerations, injury and deformation of the larynx cartilages). Orotracheal intubation is often impossible, coniotomy and/or emergent tracheostomy must be performed. After basic stabilization (respiratory tract, bleeding, blood pressure), it is necessary to rule out further injuries and treat open wounds. Revision to exclude deeper and structural injury, removal of tissue debris and damaged tissue, lavage to clean the wound and suturing. Heavily contaminated with severe tissue destruction should not be closed immediately. Antitetanus prophylaxis should be a matter of course.

As part of the long-term consequences, post-traumatic stenosis of the larynx and trachea may develope, and permanent tracheostomy can be long-term consequence



CT scan -laryngeal trauma-fracture of the thyroid cartillage, destruction of arytenoids, laryngeal obstruction due to oedema and haematoma, emphysema in soft tissues



3D reconstruction - oblique fracture of the thyroid cartillage

SPINAL CORD AND SPINE INJURIES, INCLUDING VERTEBRAL FRACTURES (55)

Veselý Radek, Kelbl Martin, Wendsche Peter

Spine and spinal cord injuries

Spine injuries are serious disabilities that can lead to permanent consequences such as chronic pain, deformities, motor and sensory impairments of varying degrees. Epidemiologically, there are 85 cases of spinal fractures per 100,000 inhabitants per year, 2/3 of the injured are men. The thoracic and lumbar spine is injured in 75 % of cases, of which 50 % are Th11-L2 fractures.

1. Mechanisms of injury

- **Compression:** forces applied in the craniocaudal axis of the spine - typical mechanisms include falling on the buttocks or extended lower limbs
- **Hyperflexion and hyperextension:** extreme anteroposterior or lateral bending of the spine can lead to ligament injuries, more complex vertebral fractures or spinal cord compression. A typical mechanism is a fall from a height onto the back
- **Rotation:** violent rotation of the spine can cause vertebral dislocation or fracture. Most commonly high-energy injuries e.g. in road traffic accidents.
- **Penetrating trauma:** gunshot or stab wounds with direct damage to the spinal cord and vertebrae

2. Classification of spinal fractures

The AO classification is the most used.

Due to the complex and unique anatomical conditions, upper cervical spine injuries are separately classified (C0-C2). An example of an injury is the fracture of a dens axis in low-energy head injuries in the elderly population.

The lower cervical (C3-7), thoracic and lumbar spine have a classification divided into 3 types:

- **Type A - Compression fractures:** these fractures primarily affect the anterior portion of the vertebral body, leaving the posterior elements (vertebral arches and spinal processes) intact.
    - A0: Isolated vertebral process fractures
    - A1: Fractures of one end plate without injury to the posterior wall of the vertebra
    - A2: Pincer fractures of the vertebral body
    - A3: Fractures of a single end plate with posterior wall involvement (incomplete burst)
    - A4: Cominuted fractures involving both end plates and posterior wall (complete burst)

- **Type B - Distraction Injuries:** These include damage to the spine resulting from excessive stretching (extension) or bending (flexion) of the spine. These fractures can damage both the anterior and posterior structures of the spine, and often result in a disruption of the stabilizing ligaments.
    - B1: Purely bony injury to one vertebra (Chance fracture)
    - B2: Bone and/or ligamentous injury to posterior structures
    - B3: Extension injuries (typical of m. Bechterev)
- **Type C - Dislocated injuries with translation or rotation:** these are the most severe injuries that are caused by high-energy violence with a combination of rotational forces and other mechanisms (compression, distraction). These fractures are accompanied by neurological deficits in up to 90%.

3. Stability of the injury

An unstable injury is a case where the physiological range of motion results in abnormal mobility of the injured segment and a threat to the spinal cord or other neural structures. Unstable injuries are usually indicated for surgical treatment.

4. Clinical manifestations

Clinical manifestations depend on the site and severity of the injury. The main manifestations include:

Soreness at the site of injury, deformity (gibus), local haematoma, palpable defect in the area of the posterior structures of the spine. The most serious are neurological symptoms:

- **Paraplegia:** transverse spinal cord lesions below the C8 level, complete sensory and motor deficits below the injured segment of the thoracic or lumbar spine.
- **Tetraplegia:** transversal spinal cord lesions above Th1 level, complete sensory and motor deficits of trunk and lower limbs, varying severity of upper limb involvement depending on the level of injury
- **Paraparesis and tetraparesis:** incomplete spinal cord lesions (some function preserved)
- **Root syndromes:** irritation or paresis of the injured nerve root according to the level of injury (pain in the relevant dermatoma, loss of sensation or motor function of the relevant root).
- **Caudae equinae syndrome**: multiple nerve roots below the L2 level are affected, characterized by impaired sensation, motor skills, impaired urinary and anal sphincters.

5. Diagnostics

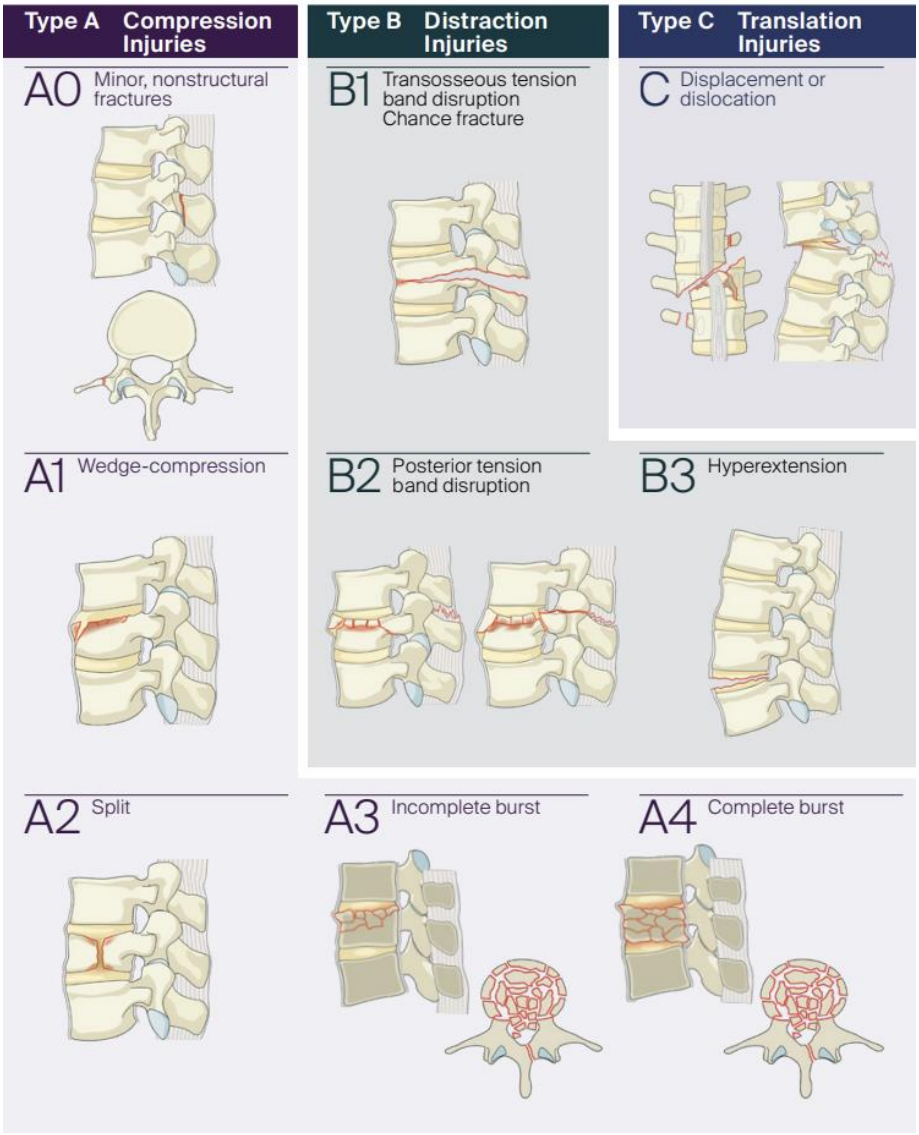
- **Clinical examination:** based on the history and mechanism of injury, the physician assesses local findings, pain, motor and sensory functions, autonomic functions and general neurological status
- **Radiological examination:**
    - X-ray in two projections - basic overview of fractures and dislocations
    - CT including 2D and 3D reconstructions to show bone injuries in more detail, show sites difficult to access on X-ray (e.g. C-Th level).
    - **Until a fracture is clearly ruled out, treat the patient after major mechanisms of trauma as if they have a spinal injury (immobilization, hard collar)**
    - MRI provides the best view of soft tissues such as the spinal cord, intervertebral discs and ligamentous structures. In severe acute conditions, the usefulness is limited due to lower availability and the length of the examination
- **Other examination** - neurological, EMG, motor and sensory evoked potentials (MEP, SEP)

6. Treatment

- **Conservative management** - wherever stability is not significantly compromised, no risk of deformity, no neurological symptoms, contraindications to surgery (internal, age, non-cooperation).
    - **Hard cervical collar (Philadelphia)** - stable C spine injury
    - **Halo-apparatus** - more stable fixation of the C spine - 4 screws fixing the halo ring to the skull and then connected to the halo-vest on the trunk
    - **SOMI** - **s**ternal-**o**ccipital-**m**andibular-**i**mmobilization - a brace consisting of a cervical collar and thoracic stabilizer, used to immobilize the upper thoracic spine (above Th6)
    - **Jewett brace** - orthosis for fixation of Th and L spine fractures
- **Surgical stabilization**
    - **Isolated anterior approach** - the gold standard for cervical spine injuries and some types of Th/L spine injuries. It consists of repositioning, removal of the vertebral body or intervertebral disc, replacement with a bone graft or other implant and stabilization with a bridging plate. The aim is to create a fusion of the damaged segment of the spine.
    - **Isolated posterior approach** - a typical procedure in thoracic and lumbar spine injuries, consists of inserting screws into the pedicles above and below the injured segment, repositioning the axis and height of the vertebral body and connecting the screws with rods.

- **Combined approach** - in severe injuries with significant comminution, a posterior approach is followed by the addition of an anterior approach.

- **Rehabilitation:** an important part of the treatment is physiotherapy and rehabilitation aimed at restoring function, preventing muscle atrophy and improving the patient's quality of life.



Obr.: AO Spine Thoracolumbar Injury Classification System

CHEST INJURIES (52)

Peštál Adam

Nosology a

Concept - A chest injury is a trauma affecting the thorax or intrathoracic organs.

Etiology - Mechanical impairment of the chest is caused by contusion, compression or deceleration.

Pathogenesis - chest trauma may result in severe conditions associated with impaired ventilation and circulation. Causes of ventilation impairment include airway injury, lung compression, lung contusion, and inadequate breathing mechanics. Circulatory failure is caused by hypovolemia in hemorrhage or cardiac injury, cardiac tamponade. Subsequent complications of injury can result in very serious conditions such as ARDS, SIRS, MOF.

Severe chest injuries are divided into critical: massive intrathoracic haemorrhage, tension pneumothorax, airway obstruction, cardiac tamponade, chest wall instability. And potentially life-threatening conditions: aortic injury, diaphragmatic rupture with herniation of abdominal organs, tracheobronchial injury, oesophageal injury.

Nosology b

Clinic - the clinical manifestations of the injury are very varied, but conditions associated with impaired ventilation or circulation are in the foreground. Manifestations are dyspnoea, hyposaturation, hypercapnia, hypoxaemia, chest or tachycardia, hypotension. On examination, we monitor respiratory excursions, jugular venous filling, assess chest stability, crepitations, subcutaneous emphysema by palpation, and investigate auscultatory findings. The mechanism of chest injury should always be considered. Great caution is needed in the initial diagnosis of penetrating chest injury in the area medial to the medioclavicular line. The injury is often associated with cardiac or large vessel involvement. In fractures of the caudal ribs, injury to intra-abdominal organs - liver or spleen - should be thought of.

Laboratory examination - standard basic laboratory examination is extended by Astrup blood test and determination of cardioenzymes.

Imaging methods - in the care of patients with chest trauma requiring admission to the high threshold UP, FAST or direct CT is the method of choice - the so-called trauma protocol, e.g. triphasic hybrid protocol. In other cases, chest X-ray and chest ultrasound are used. Chest X-ray is also performed in lateral projection, to exclude fracture of sternum, thoracic vertebrae.

Treatment - in the case of a mild injury, treatment is conservative, i.e. physical examination, relief in a semi-sitting position, sufficient analgesic therapy (pleural pain is very intense due to the

sensitive innervation of the pleura), conservative treatment is also possible in the case of serial rib fractures and so-called door fractures (double serial fractures). In the situation of an unstable chest with significant impairment of respiratory mechanics with hypoventilation or extensive reduction of pleural volume, surgical stabilization of the ribs with osteosynthetic splints is indicated. In case of minor PNO or haemothorax, conservative therapy is possible provided very careful observation of the patient. As a rule, however, these findings require thoracic drainage, ideally modo Elander, even in the case of PNO! Drainage in the V intercostal space in the anterior or middle axillary line. Before drainage, the position of the diaphragm should be known (to exclude diaphragmatic rupture or the presence of diaphragmatic hernia). It is necessary to master the correct technique of chest drain insertion. In PNO, one should always think of a form of so-called pressure overload PNO with mediastinal compression, this situation can also occur gradually over time! It is necessary to monitor the patency of the drain, the nature of the secretion, its quantity and the general condition of the patient - shock curve, anaemia, ventilation parameters. In case of continued bleeding, it is necessary to search for the cause of bleeding we perform control angio CT to identify active bleeding from the injured intercostal artery, lung... Often a surgical revision is necessary. Surgical revision is always indicated for penetrating chest trauma. In the case of a stable patient, revision is also possible by VATS. Treatment of tracheal and main bronchial injuries is conservative (observation, lesion gluing, stenting) or operative. It depends on the extent of the injury and symptomatology. If surgical treatment is necessary, right-sided thoracotomy is the most common approach. Urgent thoracotomy is indicated in case of suspected cardiac tamponade (clinical picture - Beck's triad, ultrasound examination). The approach is complete longitudinal sternotomy or anterolateral thoracotomy in the Vth intercostal space. Separate chapters of the scripts are devoted to the treatment of oesophageal and spinal injuries. Patients with severe chest trauma are often treated in the ICU or the ARC according to their respiratory status. Targeted ATB therapy, respiratory care, adequate nutrition and rehabilitation are an integral part of treatment.

Nosology c)

Complications - a potentially very diverse spectrum resulting from the nature of individual injuries, generally the risk of rebleeding, the development of infectious complications in the sense of pneumonia, pleural empyema, infections in the wound.

Prognosis - some types of chest injuries are fatal, especially avulsion injuries of the aorta or VCS with deceleration mechanism. Cardiac injury is often fatal. In the case of combined trauma or polytrauma, chest injury significantly increases both morbidity and mortality. In view of the advances in medical care, especially in the field of ARO (ECMO, UPV, elimination methods...) and

in the field of microbiology, the prognosis of patients with severe chest trauma is incomparably more favourable than in earlier times. Active surgical treatment also contributes to the success of successful cure of patients.

Prevention - specific prevention consists in the use of available protective equipment both at work and in sport. In traffic, the use of seat belts. When working at heights, correctly stabilized so-called approach aids (ladder, scaffolding...) or the use of "climbing" belay.

ABDOMINAL INJURIES (51)

Konečná Drahomíra, Mihalčin Matúš, Horváth Teodor

Abdominal trauma includes penetrating and blunt injuries of the abdominal wall, injuries to the parenchymatous abdominal organs, gastrointestinal (GI) tract and its vascular supply, starting with the cardia and ending with the rectum and anus.

A subgroup of abdominal trauma is retroperitoneal (RP) organ injuries, with the magistral vessels running in the RP and pelvic organs. The hemoperitoneum is a life-threatening internal bleeding with an accumulation of blood in the abdominal cavity. It arises from traumas of the spleen, liver or the vessels of the GI tract. The clinical signs of massive bleeding are abdominal pain, pale-coloured skin, tachycardia, and arterial hypotension, with physical findings corresponding to the mechanism of injury. The laboratory methods and imaging confirm this diagnosis.

The multidetector computed tomography (MDCT) is employed in modern diagnostics, especially in polytrauma patients. The extent of the injury is shown, and its classification is enabled – creating basic conditions for the exact decision-making process of appropriate treatment options 1/conservative, i.e. nonoperative 2/ damage control surgery (DCS) 3/surgical anatomical reparation.

Careful follow-up is included in the choice of conservative treatment.

Damage control surgery (DCS) is represented by the life-saving arrangement of actions in patients with critical polytrauma. *It aims to restore the physiological functions before anatomical restoration* in the following order:

Hemostasis, contamination control by lavage and drainage, prevention of hypothermia, renewal of homeostasis, including coagulation, preventing abdominal compartment syndrome in the case of the need for a temporary closure of the abdominal wall. An integral part of the treatment is the prevention of Deep Vein Thrombosis (DVT).

LIVER

Conservative treatment can be undertaken in hemodynamically stable patients with blunt liver injury of Grade I and II (see Fig Fix1) with small hemoperitoneum (no circulatory response). Follow-up is required.

For patients with continuing bleeding, surgical intervention is indicated with surgical revision of all particular anatomical structures. Performed through the upper midline or transversal incision, the surgery starts with hemostasis by manually compressing the liver against the posterior abdominal wall. The operating surgeon then searches for other injuries and sources of the bleeding.

If, in liver wounds, there is extensive bleeding or the source of the bleeding cannot be found, **the Pringle manoeuvre** is a solution. This is the insertion of a vascular clamp across the porta hepatis en bloc, followed by management of the vascular structures and the branches of the bile duct. Small vessels can be cauterised or ligated. Managing greater liver veins requires a vascular suture on the vascular clamp. Then, an anatomical reconstruction of the parenchyma is performed using absorbable adaptation mattress sutures. If the Pringle manoeuvre does
not stop the bleeding but only reduces it, this indicates retrohepatic venous trauma.
In the case of greater liver resection, there is a need to avoid 1/ the interlobar plane being endangered by injury of the middle hepatic vein and 2/ the plane of the falciform ligament where there is a danger of damage to the portal vein.

The liver packing using gause pacs or abdominal towels is indicated in the circumstances of 1/diffuse nonsurgical oozing, 2/developed coagulopathy, 3/if a less experienced surgeon needs to stabilise the patient who is waiting for a specialist or before the transfer to a more specialised hospital, e.g. university hospital.

The omentoplasty: After significant bleeding has been controlled, oozing can be stopped with the omentum used
as a living pack and obliteration of dead space (bolster sutures).

Debridement: Small fragments of necrotic tissue should be removed from the wound.

Nonsurgical hemostasis can utilise (porcine) gelatine foam, oxidised cellulose, microfibrillar collagen, human thrombin, fibrin glue, and autologous concentrated fibrin platelet enriched fibrin sealants and medical technologies such as monopolar or bipolar electrocautery using high-frequency electric current or Argon beam generator - the procedure destroying tissue with high-frequency electric current passed through a stream of argon gas to the tissue.

Liver drainage: Every Grade III or greater liver laceration should be put in the drain to monitor postoperative bleeding (PB) and biliary secretion. Early PB usually comes from an undiscovered injury of the hepatic artery. Late PB can be a consequence of arterial pseudoaneurysm manifested by hemobilia. In such cases, invasive radiology with catheter
embolisation of the right or left hepatic artery branches is considered.

SPLEEN

Conservative treatment is indicated in hemodynamic stable patients after blunt abdominal trauma with no or minimal bleeding into the peritoneal cavity without continuation (Fig 2, Grade I).

Surgical treatment: The access to the spleen is enabled by upper midline or left subcostal incision.

Except the hilar branching of the splenic artery is not constant, keep surgeon in mind the vessels of gastrosplenic, splenocolic, phrenicosplenic, and splenorenal ligaments and the presence of pancreatic tail near the hilus of the spleen. For the assessment of the splenic injury, mobilisation is needed by cutting all upper mentioned ligaments, removing the clots from injured areas, and ligating injured vessels.

According to features of the injury (Fig 2, Grade II, III, IV), the following treatments options may be used:

A. Splenic salvage

Splenorrhaphy by individual mattress sutures with viable omental plug and Teflon pledges. Its success is limited by the fragility of splenic parenchyma.

Splenic capping with absorbable polyglycolic acid mesh or nonabsorbable teflon wrapped around the spleen to tamponade the injury. Drainage is needed after splenic salvage operations.

To stop the nonsurgical bleeding, the same pharmaceutical products and technologies are mentioned under the subsection liver.

Partial or Hemisplenectomy using an ultrasonic surgical aspirator or laser is represented by segmental vessel ligation, resection, and mattress sutures. The omentum, splenic capping and wrapping can be utilised.

B. Splenectomy

The splenectomy is clearly indicated in the case of a completely shattered or avulsed spleen or hilar laceration (Fig 2, Grade V). This procedure is performed as one fastest, especially in higher grade spleen injury in patients with current serious trauma of the neurocranium, thorax, and pelvis, and in patients with coagulopathy.

The risk of **Overwhelming PostSplenectomy Infection (OPSI)** is lifelong. Immediate postoperative prevention is represented by wide spectrum antibiotics (ATB); prospective protection means routine vaccination against *Streptococcus pneumoniae, Haemophilus influenzae, and Neisseria meningitidis* and annually against influenza and COVID-19, both with a predisposition to bacterial superinfections. Before the discharge, the patient should 1/ get notified of the necessity of seeking medical attention at the elevation of body temperature to 38°C (100°F) and to be equipped with the prescription of emergency reserve of ATB; these will be used no later than 2 hours after the onset of the fever.

As emergency antibiotics (ABx) are chosen, amoxicillin/clavulanate 1 gram tablet to be taken every 6-8 hours or cefuroxime/axetil 500mg tablet to be taken every 6-8 hours Whereas, in patients with penicillin allergy, moxifloxacin 400mg tablet per day.

VESSELS

The vascular trauma is manifested by hematoma or intraabdominal bleeding with obvious signs of acutely damaged abdomen and hypovolemia. It is not usually isolated. Midline (central) supramesocolic hematomas, including haemorrhage into the lesser sac and inframesocolic hematomas, should be opened and explored by midline incision from the xiphoid process to symphysis.

Periduodenal, periportal, and right-sided pericolic hematomas are clarified by Kocher manoeuvre – i.e. the releasing and medial rotation of the duodenum and the head of the pancreas. Cutting of parietal peritoneum from hepatic flexure laterally longitudinally alongside right colon down to caecum uncovers inferior vena cava. The operation continues with tamponade, followed by revision. A vascular suture or venous patch treats the solitary lesion. In a dramatically hemodynamic unstable patient with more injuries of the inferior vena cava, there is no other way except its ligation. Collateral vessels will develop.

The source of the hematoma or haemorrhage in the region of the right upper abdominal quadrant is the superior mesenteric artery or vein. For the overview, digital compression is needed between the index and middle finger placed into foramen Winslow behind the head of the pancreas and thumb on its anterior surface. Discovered injury can be tamponaded with a finger or controlled with a Satinsky clamp and sutured.

The simple suture of the vein (venorrhaphy) or venous patch is indicated in a circulatory stable patient. The ligature of the portal vein is possible in the circulatory unstable patient.

Central inframesocolic hematomas are accessible after retracting the transversal colon cephalad and small bowel to the right. They may be caused by injury of visceral vessels or the aorta. If the vitality of the bowel is damaged, resection is needed.

The assessment of bowel vitality is facilitated by the fluorescence of the indocyanine green (ICG) dye administered into the circulation through the peripheral vein. The fluorescence is generated by irradiating the operating field with near-infrared electromagnetic waves (806 nm). ICG absorbs the waves, and its energy is emitted in the visible part of the light spectrum. The intense green colour of the bowel confirms physiological blood perfusion.

COMMENTS AND CONTROVERSIES

There are different classifications of liver and spleen injury. The illustrative examples of their complexity were chosen here.

Quite small subcapsular hematoma of the spleen or liver can break too in a time period of some hours to some weeks, giving rise to bleeding or even two-stroke rupture.

Figure 1: Liver Injury Scale by Arthur J. Donovan

GRADE		
I	Hematoma	Subcapsular, nonexpanding, <10% surface area (SA)
	Laceration	Capsular tear, nonbleeding, <1 cm parenchymal depth
II	Hematoma	Subcapsular, nonexpanding, 10% to 50% SA
	Laceration	Capsular tear, active bleeding
		1 to 3 cm parenchymal depth, < 10 cm in length
III	Hematoma	Subcapsular, >50% surface area or expanding
		Ruptured subcapsular hematoma with active bleeding
		Intraparenchymal hematoma >2cm or expanding
	Laceration	>3 cm parenchymal depth
IV	Hematoma	Ruptured intraparenchymal hematoma with active bleeding
	Laceration	Parenchymal disruption involving 25% to 50% of hepatic lobe
V	Laceration	Parenchymal disruption involving > 50% of hepatic lobe
	Vascular	Juxtahepatic venous injuries (i.e. retrohepatic vena cava, major hepatic veins)
VI	Vascular	Hepatic avulsion

Figure 2: Splenic Organ Injury Scale by American Association for the Surgery of Trauma

GRADE	
I	Nonexpanding subcapsular hematoma involving <10% of surface area (SA)
	Nonbleeding capsular laceration with <1 cm deep parenchymal involvement
II	Nonexpanding subcapsular hematoma involving 10% to 50% of SA
	Nonexpanding intraparenchymal hematoma <2 cm in diameter
	Bleeding capsular tear or parenchymal laceration 1 to 3cm deep without of trabecular vessel involvement
III	Expanding subcapsular (subcaps.) or intraparenchymal hematoma
	Bleeding subcaps. hematoma or subcaps. hematoma involving > 50% of SA
	Intraparenchymal hematoma > 2 cm in diameter
	Parenchymal laceration >3 cm deep or involving trabecular vessels
IV	Ruptured intraparenchymal hematoma with active bleeding
	Laceration involving segmental or hilar vessels producing major devasculari sation (> 25% splenic volume)
V	Completely shattered or avulsed spleen

Hilar laceration that devascularizes entire spleen

GASTROINTESTINAL TRACT INJURIES (53)

Penka Igor

Injury to the gastrointestinal tract can affect any part of the digestive apparatus, whether it is the parenchymatous organs (liver, spleen, pancreas) or the intestinal tract at any level. It can be open – penetrating injuries from stabbing, cutting, cutting or gunshot wounds, but also blunt injuries from contusions of the abdominal wall, where the intensity of the injury mechanism plays a crucial role. The most serious are high-energy injuries from traffic accidents and falls from heights, which is a very important anamnestic data in itself. In these cases, gastrointestinal tract (GIT) injury is usually part of a serious associated injury or part of polytrauma. Iatrogenic injuries during instrumental diagnosis and therapy (endoscopic and radiointerventional procedures and invasive procedures in any field) also form a significant group. Of the GIT injuries, the most serious are those that lead to leakage of the contents of the gastrointestinal tract into the environment – mediastinum, free abdominal cavity, retroperitoneum. All of these conditions are extremely serious and are immediately life-threatening. Symptomatology and diagnosis. The difficult fact is that the symptomatology of these events can be relatively poor at the beginning and the only data on which the surgeon can rely is the anamnestic data of the insult and almost always pain of varying intensity in the immediate period after the injury. A slightly later symptom is usually fever, sometimes associated with shivering. Also, during clinical examination, the picture is initially poor and non-specific. Peritoneal symptoms also appear with a certain delay. In laboratory examination, an increase in inflammatory indicators – CRP and leu – is characteristic. However, the greatest support for diagnosis can be found in imaging methods – especially in contrast CT scans. Of the native X-ray methods, X-ray of the lungs with pathognomonic enlargement of the mediastinum in mediastinitis will help. In perforation of the GIT, native X-ray subphrenis in a standing position, which demonstrates a more massive pneumoperitoneum. In cases where we suspect a possible injury of the gastrointestinal tract supported by clear apathic data even with poor symptomatology and almost negative clinical examination, we do not hesitate to request a contrast CT scan on the suspicious area (chest, abdomen, retroperitoneum). If the patient is circulatory stable and there is no danger of delay, there may be room for diagnostic laparoscopy (thoracoscopy), which also carries significant therapeutic potential in the form of laparoscopic treatment. In the case of severe concomitant injuries or polytraumas, often associated with circulatory instability or unconsciousness, the requirement for contrast whole-body CT examination is usually implemented in emergency departments with concomitant circulatory restitution of the patient.

Surgical treatment. It has already been said that GIT perforation events are among the most serious conditions in surgery and are indicated for urgent surgical treatment.

The extent of perforation is absolutely essential – from small perforations in the range of a few mm to extensive devastation of long sections of the GIT with destruction, bruising and devitalization of the wall of the GIT organ in the immediate vicinity of the perforation hole. A timely surgical revision allows suture to be performed – with a single or continuing suture, or with the stapler technique. The later the surgical revision is performed, the more extensive changes in the vitality of the perforation area can be expected, in addition to the progressive development of infection in the immediate vicinity of the defect. After 24 hours, the hope of the suture being successful is small and later it is not even advisable to attempt suture. In cases of a time delay, there are only the options of surgical drainage as a definitive treatment, or, on the contrary, complex extensive resection procedures, usually with the establishment of a derivation of the more oral sections of the GIT - a stoma with the prospect of temporariness, which is in many cases relative. It is always necessary to keep in mind the invaluable assistance of interventional radiology and endoscopy with the therapeutic potential of stent insertion (mainly in cases of esophageal injury). In cases of severe concomitant injuries and polytraumas, perforation injuries of the gastrointestinal tract are evaluated as rather secondary concomitant injuries, which must be treated surgically during urgent surgical revisions – of course, according to the principles of Damage Control Surgery. This represents the fastest possible closure of the GIT perforation (stapler resection of the intestine), without any reconstruction, which is performed in the second period after stabilization of the patient. Oesophageal injury: not a common injury, but not an isolated one. Examples are burns of the esophagus by acid or lye or swallowing of foreign bodies. Both either by mistake or as part of self-harm. Serious perforation injuries occur most often during endoscopic procedures. From the perforations of the esophagus, it is necessary to mention the not quite exceptionally occurring so-called Oesophagus perforation. Boerhaave syndrome – spontaneous rupture of the distal esophagus due to excessive vomiting, mostly postprandial in allocoholics. Chemical burns – acid causing coagulation necrosis, alkalis causing colic necrosis. The mucous membrane of the esophagus is affected first, but if a larger amount is used and prolonged exposure to noxa, necrosis of the entire esophageal wall can occur. It is always necessary to rule out perforation of the esophagus – p.o. contrast CT scan or X-ray contrast examination with aqueous solution, careful gastrofibroscopic examination to assess the extent of oesophageal involvement, including the stomach. In non-perforating cases, it is possible to proceed conservatively and carefully monitor whether perforation develops. Therapy consists of administering analgesics, treatment of the shock state. Immediately after the burn, chemical neutralization can be attempted. For acids, it is best to use water or soda (sodium bicarbonate), for

alkalis, significantly diluted with vinegar water. It is necessary to ensure nutrition - in mild cases it is possible to take fluids after the disease, in more severe cases it is necessary to provide enteral nutrition by nasojejunal tube.

In case of suspicion of perforation, it is necessary to administer antibiotics and surgical solution, which is very difficult in the most severe cases with extirpation of the esophagus and establishment of a derivative cervical esophagostomy and a nourishing gastrostomy (jejunostomy) with the prospect of reconstructive surgery in the second period. Injury to the duodenum. It is similar to oesophageal injury in incidence, diagnostic and therapeutic severity, including high mortality. It can occur with blunt and penetrating abdominal trauma and can be associated with injury to surrounding organs, including major vessels. It also occurs following instrumental endoscopic procedures of the duodenum, biliary tract and pancreas (ERCP and papillotomy, extraction of choledocholithiasis and placement of stents in the biliary and pancreatic ducts). The most serious are, of course, perforating injuries of the duodenum with extraluminal leakage of duodenal contents - with development of peritonitis in the case of leakage into the free abdominal cavity, or - development of retroperitoneal phlegmon in the case of leakage of duodenal contents into the retroperitoneum. Surgical treatment, as with oesophageal treatment, depends on the extent and location of the perforation and the vitality of the duodenal wall around the perforation. Suturing of the duodenal wall can be performed in the mildest cases, while more extensive, devastating injuries may require complex resection and reconstruction procedures (anastomosis with the small bowel). Gastric injuries are considered for penetrating injuries and blunt abdominal injuries – blows to the front abdominal wall, injuries caused by car belts in traffic accidents. The most serious are certainly perforations with leakage of gastric contents and the development of acute peritonitis. All of the above applies to the diagnosis and is rather based on the detection of pneumoperitoneum, which is in itself an indication for urgent surgery. The stomach is usually easily accessible and suture of even larger defects is usually well done. In more severe cases, it is necessary to perform a resection with a connection of the stomach with a thin loop. The issue of thin loop injuries is practically identical. However, in the case of colon injuries, in the case of more extensive perforations, often with nine-fold perforation and insufficient vitality of the intestinal wall around the perforation, we cannot avoid a resection procedure and, above all, the need for a colostomy. Anorectal injury is relatively rare. It most often comes as a foreign body injury and as an iatrogenic injury during diagnostic-therapeutic colonoscopy. Perforation injury of the intraperitoneal part of the rectum leads to the development of stercoral peritonitis. Injury to the extraperitoneal part of the rectum leads to infection in the area of the small pelvis with the formation of pararectal, periproctal abscesses. In the diagnosis, anamnesis and clinical examination play a crucial role, with an emphasis on per rectum examination, which often

shows a palpable rectal defect. It is beneficial to perform ano-rectoscopy as soon as possible. Contrast CT imaging is also a standard diagnostic method – in case of uncertainty, magnetic resonance imaging (MRI) may be important. The aim of surgical treatment is to treat the rectal defect, prevent infection, maintain continence and evacuation function of the rectum.
A crucial role, as in the case of the oesophagus and duodenum, is played by the extent and location of the affliction. Minor perforations of the intraperitoneal rectum can be treated by simple surgical suture, extensive devastation by difficult resection procedures, in the vast majority of cases with simultaneous establishment of a derivation stoma. Extraperitoneal rectal injury with the development of abscesses is treated by drainage (CT-guided or transanal), in more extensive injuries it is necessary to establish a derivation stoma. Very serious injuries with relatively high morbidity include almost exclusively iatrogenic injuries of the biliary tract – most often in laparoscopic cholecystectomy. Quite exceptionally, hepatocholedochus injury also occurs during biliary tract instrumentation during ERCP or difficult extractions of bulky choledocholithiasis. Now in the era of laparoscopic cholecystectomy, the frequency of biliary tract injuries has practically increased – 0.7% of LSK cholecystectomy. Diagnosis is based on the postoperative course - abnormal abdominal drain waste, increase in obstructive liver enzymes, elevation of serum bilirubin values. An acute ERCP examination and contrast CT examination with an assessment of arterial supply to the liver play a crucial role in the diagnosis. If it is a bile duct injury with preserved continuity, then the method of treatment is endoscopic treatment or interventional radiology options (ERCP, stent insertion or PTD drainage under CT navigation). In the case of complete rupture of the bile duct or even loss injury, the method of choice is a surgical solution with anastomosis of the severed ends of the bile duct end to end on a T drain, or the establishment of a biliodigestive shunt. Injury to the omentum and suspension apparatus of the intestine. It occurs in penetrating and blunt rather high-energy injuries. In most cases, it is associated with injury to surrounding organs. It is manifested by bleeding from injured blood vessels – hemoperitoneal syndrome. In cases of mesentery and mesocolon injuries, the main vessels may be affected, resulting in necrosis of the intestinal wall with perforation and leakage of GIT contents into the abdominal cavity – perforation peritonitis syndrome. Diagnosis and treatment are governed by the principles of the affected organ – see above.

TENDON INJURIES, DUPUYTREN'S CONTRACTURE (56)

Menoušek Jan, Kubát Martin

Dupuytren's contracture is a **benign, slowly progressive fibroproliferative disease** of the **palmar aponeurosis**, event. other localities (plantar aponeurosis – **Lederhose's disease**, penis - **Peyron's disease**). It is characterized by the formation of retracted skin in the palm, as well as the formation of nodules and bands in the palmar and digital fascia, which shorten and lead to finger contracture and thus limitation of hand function. The flexor tendons are not involved in the contracture and are intact. The disease is named after the French surgeon **Guillaume Dupuytren**.

Dupuytren's contracture is an **autosomal dominant disease** with variable penetrance. **Negative influences promoting** the onset of the disease are heavy manual labor, smoking, diabetes, epilepsy, and repetitive trauma to the hand. Rheumatoid arthritis is considered to be a **protective influence**. The disease is referred to as **Viking disease and the highest prevalence is in Scandinavia and Western Europe**. The disease has a **median age of onset of 50 years**, with the incidence of the disease increasing with advancing age. The **earlier the disease** manifests, the **more aggressive** the disease behaves and the risk of recurrence increases. The incidence **in males is 10-15 times higher** than in females and the **white race has the highest incidence**, unlike other races where the incidence is more sporadic. The disease progresses in three stages. **Proliferative** - fibroblastic proliferation and nodule formation. **Involutional** - fibroblasts are not regulated and transform into myofibroblasts. **Residual stage** - type III collagen begins to be deposited and this connective tissue begins to organize into bands that the fingers of the hand subsequently contract.

Classification of Dupuytren's contracture according **Karfík** (it is not very accurate and does not allow to evaluate the postoperative outcome): This classification consists of three types of hand involvement: palmar, simple and complex. The **Tubiana** complex classification seems to be the most appropriate.

Indications for surgery: a) a positive **"table top test"** (the patient does not touch the table with the entire palm surface when placing the hand on the table), b) **painful nodules** in the palm - painful gripping when the neurovascular bundle is compressed, c) **30st flexion contracture** in the MP, or any other contracture in the PIP, d) as soon as the patient **starts to be bothered** by the disease.

Treatment of the disease can be conservative, minimally invasive, surgical and enzymatic (collagenase application). Follow-up rehabilitation is an integral part of the treatment.

Fasciectomy (on the finger) and aponeurectomy (in the palm of the hand) are the most commonly used invasive surgical procedures in which surgical removal of the pathological contractile band

from the finger ray is performed. According to the extent, they are divided into limited, regional (usually ulnar half of the palm) or total.
The disadvantage of this method is a long convalescence, the advantage is the immediate and long-term effect of the operation. Dermofasciectomy is a surgical solution in which the contracting bands and nodes are removed with the skin and the defect is covered with a skin graft. Percutaneous aponeurotomy is a minivascular method in which the pathological contracting bands are cut through and released from the skin by means of a needle through tiny punctures in the skin. The advantage of the method is a very short recovery time and a limited amount of scarring. The disadvantage is early recurrence. It can be complemented by lipografting, in which a small amount of fat is additionally removed from the abdomen or buttocks using the lipoaspiration technique, the fat is centrifuged or sedimented and then infiltrated into the operated area using a special application cannula.
Rehabilitation: varies from one surgical procedure to another and according to the surgeon's habits. After fasciectomy/aponeurectomy, a splint is applied immediately after surgery volarly with compression, where the fingers are flexed as if holding a ball in the hand. Flexion and compression is important because of the attachment of the skin flaps to the underlying skin and because of the lack of skin cover (in extension, it would be necessary to cover the volar defects with a skin graft). After all sutures are removed (3 weeks after surgery), we begin full finger rehabilitation with intermittent splinting. Patients actively warm up the fingers during the day and leave the hand in an extension splint overnight.
Recurrence: postoperative recurrence rates have been reported to range from 10 to 77%, with no uniform criteria for recurrence. Some authors consider recurrence to be secondary to a flexion contracture or knot, but this does not affect the functionality of the hand. Furthermore, the recurrence rate varies from year to year after surgery

Tendon injuries

Flexors

The flexors are divided into extrinsic and intrinsic muscles, depending on the location of the origin. Extrinsic muscles originates on the humerus, radius, ulna, and interosseous membrane and are responsible for the overall flexion of the fingers, especially at the IP joints. Intrinsic muscles have orrigin and attachment to the hand. We divide them into **lumbrical** and **interosseous** muscles, which both help flexion in the MP joints and extension in the IP joints. An important part of the flexor apparatus are **pulleys (annular and crossed)**, which serve to keep the tendon close to the bone and thus prevent the formation of a "bowstring". ¨

Flexor tendon injuries by location. Three phalangeal fingers: zone 1 – distal to the PIP (contains only the FDP), zone 2 – from the PIP joint to the distal palmar groove, the so-called "no man's land", zone 3 – between the distal palmar groove of the palm and the distal edge of the carpal tunnel., zone 4 – area carpal tunnel. Injury in this area usually also includes injury to the median nerve, zone 5 – forearm proximal to the carpal tunnel.

Thumb: zone T1 - distal to the IP joint, zone T2 - area above the proximal phalanx from the A1 loop to the IP joint, zone T3 - thenar groove - distal edge of the carpal tunnel, zone T4 and T5 are the same as the finger zones.

Diagnosis and surgical treatment of flexor injuries. In an FDP injury, there is a failure of flexion of the distal joint in the DIP. When the FDS is injured, there is a loss of flexion in PIP. In the case of a combined injury, there is a loss of flexion in the DIP and in the PIP while flexion in the MP is preserved (flexion using intrinsic muscles). We always treat flexor injuries with a 4strand suture, which we divide into primary (within 24 hours), delayed primary (1st to 14th day) and secondary, which requires tendon replacement using a tendon transplant.

Extensors. The muscles originates at the humerus, radius, ulna and interosseous membrane and pass through the dorsum of the hand to the fingers. The thumb has 3 extensors attach at different levels. The abductor attaches at the base of the 1st metacarpal, the EPB at the base of the basal joint and the EPL at the base of the distal joint. The index and little fingers have 2 extensors. II. -V. extensors have junctions between them on the back of the hand and therefore there may not be a complete loss of extension upon interruption. Extensors II.-V. of the finger form distally from the MP joints together with the attachments of the intrinsic muscles, the so-called dorsal aponeurosis.

Extensor tendon injuries by location – 8 zones of injury. Three-jointed fingers. zone 1 - DIP, zone 2 - middle joint, zone 3 - PIP, zone 4 - base joint, zone 5 - MCP, zone 6 - metacarpals, zone 7 - retinaculum, zone 8 - distal forearm. Inch. zone T 1 - IP joint, zone T 2 - base joint, zone T 3 - MCP joint, zone T 4 - metacarpal area, zone T 5 - CMC (carpo-metacarpal joint), zones 6 to 8 are identical to the zones of three-jointed fingers

Treatment of extensor injuries can be treated closed (splitting, Kirschner wires). Avulsion extensor injury of the distal joint in zone I is called **mallet finger**. Most often, it is a closed injury, tearing of the dorsal aponeurosis with semiflexion in the DIP. Treatment consists of immobilizing the middle and distal joint in DIP hyperextension for 6 - 8 weeks, followed by night splinting for 4 weeks. An open injury in zone II of the middle strip of the dorsal aponeurosis, when the lateral strips remain in continuity, which slide palmarly on the sides of the PIP joint, causing its flexion and hyperextension in the DIP, the so-called **buttonhole (boutonnière)**. **Swan neck deformity** is characterized by PIP hyperextension with DIP semiflexion. It arises on FDS imbalance, dorsal aponeurosis and

lengthening of the PIP volar plate, when the shortened deep flexor pulls the distal joint. The therapy is conservative(splinting) and surgical.

Rehabilitation of the hand is an integral part of the treatment of tendon injuries. We distinguish between early (within 5 days from the suture) and delayed (after 3 weeks). Further to active and passive, when the tendons slide in the channels on the base of the extension. In early rehabilitation, one must think about the strength of the tendon suture, which is least strong between the 9th and 15th POD.

Rehabilitation after flexor suture: 1. strict 4-week immobilization (after replantation, in children under 10 years, patient noncompliance 2. passive mobilization according to Duran-Hauser 3. passive mobilization according to Kleinert 4. active mobilization according to Strickland, "place and hold" technique.

Rehabilitation of extensors: 6 weeks of hyperextension immobilization followed by very gentle active flexion for zone I. In zone III. active DIP flexion at zero PIP extension with a static splint by week 6. Followed by active exercises of the DIP and MP joints with active extension in the PIP joint with continued splinting of the PIP joint until the end of the 12th week. In zone V., VI., and VII. volar extension plate. From the 4th week, dynamic splint, static at night. From the 6th to the 12th week, active exercise of the MP joints with active flexion and passive extension is performed.

CLAVICLE, SCAPULA, AND SHOULDER INJURIES (57)

Dobiášek Miroslav

Injuries to the clavicle, scapula and shoulder can be collectively referred to as shouder girdle injuries. The latter is formed by the junction of these 3 parts and the adjacent chest wall. Injuries range can by from contusions and soft tissue injuries, to fractures (closed, open), luxation fractures and traumatic amputations.

Clavicular injuries

Anatomy: The clavicle connects the brachial girdle to the trunk. It provides abduction of the arm, mobility of the limb, stability of the shoulder and protects the neurovascular structures.

Epidemiology and aetiology: Fractures are common, accounting 5% of all fractures. Injuries are most often caused by direct violence or by a figurative fall on an outstretched limb.

Clinical picture and diagnosis: Clavicle contusion is manifested by swelling, pain, haematoma and more or less restriction of movement. In fractures, there is also defiguration, crepitations and pathological movement in the clavicle area. They may be associated with a breach of the skin covering, intrathoracic injury or injury to the subclavian vessels or brachial plexus. They are divided into 3 types. The most common type I is injury of the middle part (diaphysis). Type II is injury of the lateral part and the least common medial type III. Examination of the periphery is an integral part. The imaging modality of choice is radiography in two projections.

Therapy and complications: Treatment of the contusion consists of temporary wearing sling until the pain subsides and local physical therapy. In the past, fracture therapy has been only conservative. Today, it has a place in non-dislocated or minimally dislocated and stable fractures. It consists in fixation of the limb with an anti-rotation sling, Gilchrist or Delbet rings fixation, depending on the localization of the fracture. The immobilization period is 3-4 weeks with subsequent rehabilitation. In children, fixation for 2-3 weeks is sufficient. Open fractures, fractures with threatening injury to the skin cover, injuries to the neurovascular bundle and dislocated fractures (shortening of more than 2 cm and dislocation ad latus by the width of the bone) are indicated for surgical treatment. Osteosynthesis is performed either by intramedullary or splint osteosynthesis. Complications may include skin cover injury, neurovascular injury, infection, pseudoartrosis and osteosynthesis failure.

AC joint injuries

Anatomy: The acromioclavicular joint is secured by the acromioclavicular (AC) and coracoclavicular (CC) ligaments.

Epidemiology and aetiology: The injury is most often caused by a fall on the shoulder or on an outstretched limb. These are common sports injuries. We use the Tossy or Rockwood classification. Tossy's type I: distortion-injury of the joint capsule of the AC joint, AC and CC ligament intact, type II: subluxation-tearing of the joint capsule and AC ligament, CC ligament intact, type III: luxation-tearing of all 3 structures including the CC ligament.

Clinical picture and diagnosis: Swelling, pain, prominence of the lateral clavicle and piano key symptom. X-rays are performed, possibly with stress images or comparison with the healthy limb. MRI may also be added.

Therapy and complications: Fix in a sling or shoulder brace for 3 weeks with subsequent rehabilitation. Surgical treatment is reserved for type III. We stabilize the AC joint using tension cerclage, "hook" splint or minimally invasive anchors.

A rare injury in the clavicular region is luxation in the sternoclavicular joint. It is caused either by direct force on the clavicle or by a fall on the shoulder. Anterior (presternal) and posterior (retrosternal) types are distinguished. The anterior type can be closed repaired, the posterior type is treated by open repositioning and at the same time stabilization with a splint.

Scapula injury

Anatomy: The scapula connects the chest to the upper limb and is essential for free movement at the shoulder joint. It is protected by a large number of muscles.

Epidemiology and aetiology: Fractures are rare and are caused by a direct impact on the scapula from behind or by a fall on the shoulder. Often associated with intrathoracic injury.

Clinical picture and diagnosis. Imaging methods include X-ray and more detailed CT scanning. Fractures of the scapula are divided into 5 groups: fractures of the process, body, neck, glenoid and combined fractures. An interesting type of injury is the "floating shoulder" when the bony and ligamentous apparatus of the upper limb is separated from the axial skeleton (in double injury of the upper suspensory apparatus or simultaneous fracture of the clavicle and glenoid).

Therapy and complications. We indicate for surgery dislocated glenoid fractures with deficit on the articular surface of more than 2 mm and double injury of the superior suspensory complex. We use splints and tension screws using 3D printing to treat scaphoid fractures. Postoperative fixation tends to be for 3-4 weeks with subsequent rehabilitation. Complications can include pseudoartrosis, implant failure, infections or post-traumatic arthrosis.

Shoulder injury

Due to the complexity of the area and the variability of injuries, we will select the 4 most common ones:

1. Rotator cuff injury:

Anatomy: The rotator cuff is composed of the muscles m. subscapularis, m. supraspinatus, m. infraspinatus and teres minor, which provide rotational movements and elevation of the limb.

Epidemiology and aetiology: Acute traumatic rupture with high load during limb elevation is rare. Chronic irritation of the subacromial bursa and progressive fibrotization with impaired rotator cuff function is more common.

Clinical picture and diagnosis: Long-term shoulder pain with progressive deterioration of function is typical. We investigate with resistance tests, drop test, Cyriax pain arc helps to differentiate. X-ray, ultrasound and MRI are performed.

Therapy and complications: On the basis of the examination, we decide between conservative treatment, which is suitable only for partial ruptures, and surgical treatment, which is arthroscopic suture.

2. Injury to the m. biceps brachii

We divide it into injuries in the proximal part, the muscular middle part and the distal part. Proximally, the long head of the biceps brachii is injured, which is manifested by a typical "bump" deformity in the medial part of the arm. Therapy is predominantly conservative with short-term immobilization of the limb. Surgical treatment is possible by refixation. Injury to the medial part of the muscle is most often caused by a direct mechanism. Swelling and haematoma may be present. Therapy is conservative. Injury to the distal biceps tendon most often occurs when lifting heavy loads. Clinically, there is a significant limitation of muscle strength, hematoma in the distal part of the arm and an impalpable tendon. Therapy is surgical with refixation of the tendon to the tuberositas radii. In addition to the typical clinical picture, ultrasound examination is used in the diagnosis.

3. Luxation of the shoulder joint:

Anatomy. Static (joint capsule, labrum, ligaments) and dynamic stabilisers (muscles) ensure the stability of the joint.

Epidemiology and aetiology: Etiologically, the most common is a fall on an outstretched limb, but luxation can occur without an injury mechanism (habitual luxations).

Clinical picture and diagnosis: Pain, shoulder defiguration and palpable empty joint socket are present. There are 4 types of luxations (anterior, posterior, superior and inferior). Anterior luxation is the most common. It is diagnosed by X-ray.

Therapy and complications. Surgical treatment is necessary for irreversible luxations and for concomitant vascular injury. Injury to the brachial complex or vascular injury may be the most common complication. Recurrent luxations are also not rare.

4. Fractures of the proximal humerus

Epidemiology and aetiology. They represent a relatively large number of fractures (5-6%). The cause is most often a trivial fall on the shoulder, predominantly in elderly patients.

Clinical picture and diagnosis: They present with pain, limitation of mobility, haematoma, and in luxation fractures an empty joint socket can be palpated. It is important to examine the periphery to exclude nerve or vascular lesions. The basic imaging modalities are X-ray and CT to verify and plan the management of the fracture. The classifications used are AO and more commonly Neer's (types I-VI). They use the division of the proximal humerus into head, diaphysis, greater and lesser tuberosity.

Therapy and Complications: Evaluation and treatment must be approached on a strictly individual basis. Conservative treatment is the appropriate treatment for up to 75% of fractures. It is indicated for all non-dislocated fractures, dislocated fractures in elderly patients with lower expectations of a good functional outcome and polymorbid patients. Surgical treatment is most commonly performed from a deltoideopectoral approach. The choice is between splint osteosynthesis, which is suitable for all types of dislocated fractures in younger patients (up to 65-75 years). Hemiarthroplasty, which provides good pain relief, but the functional outcome is limited - suitable for patients with multifractured fractures without the possibility of osteosynthesis. The last option is reverse total endoprosthesis, which has a better functional outcome. They are indicated in elderly patients (65-75 years) with the requirement for the best possible functional outcome, in failed osteosyntheses and in luxating four fragments fractures. The recommended immobilization with a sling or shoulder brace for conservative therapy is for 2-4 weeks, with passive exercise initiated after 1-2 weeks, followed by active rehabilitation. For postoperative conditions, fixation and rehabilitation are individually selected. Early complications include vascular and nerve injuries. Late ones include the development of avascular necrosis, healing in malposition, pseudoartrosis or failure of osteosynthesis.

Comments and controversy: When treating injuries, it is important to make an individual assessment and weigh the benefits and risks to the patient of the proposed therapy (especially surgery). We should ascertain the patient's expectations for the future and respect the patient's wishes.

HUMERUS AND ELBOW INJURIES (58)

Reška Michal

Humerus injury

1. *Fractures of the proximal humerus*

Diagnostics:

- Clinically, pain localized to the shoulder or proximal part of the arm with limitation of range of motion. Swelling may be present, and the hematoma usually resolves after a few days. It is always important to examine the neurocirculation of the limb up to the periphery.
- X-ray: the minimum is two projections perpendicular to each other - anteroposterior + transthoracic , but three perpendicular images are recommended - anteroposterior, lateral transscapular and axial.
- CT with 3D reconstruction will clarify the findings, help the therapist. Balance sheet
- USG functional to examine the rotator cuff
- MR before reconstructive procedures, assessment of rota. cuffs and head vitality.
- epi- or electrocution patients

Classification: AO classification: 11 A, B, C see. www.aosurgery.org, Neer classification: Neer I-VI

Therapy: The aim is to restore the function of the shoulder joint as best as possible.

- Functionally conservative treatment

Immobilization with a Desault bandage event. three-point shoulder brace (Gilchrist) usually for 3 weeks. Abduction splint. Hanging plaster (hanging cast) especially in neck fractures with shortening - it has no significance in lying patients.

- Operative treatment

The basic principle of surgical treatment is the reposition of fragments, including bumps with muscle attachments, and their adequate fixation. Choice of commonly known implants – K. wires, screws, plates incl. angularly stable, special intramedullary nails. In case of non-reconstructable fractures, especially of the head, replacement of the head or frame is necessary . joint.

Rehabilitation:

After conservative therapy, we start passive rehabilitation first, gradually depending on the condition and X-ray checks with the addition of a muscle component. After surgical treatment, it is necessary to approach individually and take into account the procedure performed - for example, with adaptive osteosynthesis, you must first start with passive rehabilitation, on the contrary, with stable osteosynthesis, you can rehabilitate early and actively.

Complication:

Early: - vascular injury, nerve injury

Late: - avascular necrosis of the head, healing in dislocation, impingement you are subacromial space, hip joint, failure of osteosynthesis, adhesive capsulitis

2. Fractures of the diaphysis of the humerus

Diagnostics:

Clinically, crepitation, pain, deformity, pathol . momentum, X-ray in two projections. Examination of the neurocirculation of the limb periphery!

Classification: AO: 12- A, B, C - see. www.aosurgery.org.

Therapy:

Conservative therapy requires patient cooperation and more frequent X-ray checks due to the possibility of redislocation.

- high plaster fixation with epaulet over the shoulder, functional treatment according to Sarmiento - orthosis (Brace)

Operative treatment - technique : Open reposition with radial nerve revision and plate osteosynthesis, intramedullary anterograde or retrograde nailing , external fixator mostly single-plane , possibility of extension to the forearm in case of combined injury with the elbow joint.

Complication:

radial nerve paresis , complications during implant insertion

Rehabilitation:

Early after surgery – after the surgery has healed. wounds pac. passively rehabilitates the shoulder and elbow joint. After splinting, full weight bearing until the fracture heals. After nailing, you can exercise actively right away, only the rotation of the arm is limited until the bone heals.

3. Distal humerus injury

Diagnostics:

- Injury history
- Swelling, hematoma, deformation, dysfunction
- X-ray image in two projections for basic orientation
- CT examination for closer verification of intra-articular involvement

Classification: AO classification 13- A, B, C - see. www.aosurgery.org

Therapy:

Conservative therapy is rarely possible for non-dislocated extra-articular fractures. But the vast majority of these fractures require surgical treatment.

Note: treatment of type C fractures in the area of dist . humerus and elbow is a problem of special traumatology.

4. Proximal forearm injury

Radial head injury

Diagnostics:

- Clinically, pain, limitation of function, typical pain targeted above the head of the radius and in rotation
- X-ray in two projections, it is advisable to perform an X-ray of the elbow and forearm - watch out for the Essex- Lopresti fracture (fracture of the head of the radius with rupture of the interosseous membrane, typ. widening of the radio -ulnar distance on the X-ray)

Classification: according to AO.

Therapy:

- Conservative therapy: for non-dislocated fractures without instability, short-term immobilization for 14 days with early rehabilitation, initially excluding rotational movements of the forearm.
- Operative treatment for comminuted or significantly displaced fractures:

Pronatio dolorosa

Typical for children, popping out of the head of the radius from the ligamentum annulare , especially during traction and rotation behind the extentoxed forearm. Clinically, pain is manifested by rotation of the forearm and pain above the head. The therapy is head repositioning and short-term immobilization for 7-14 days.

Fractures of the proximal ulna

Process fractures coronoid

Diagnostics: X-ray, CT

Therapy: reposition and osteosynthesis, most often with a tension screw, in the case of a more massive fragment, a splint synthesis can also be chosen

Olecranon fractures ulnae

Most often, they enter by falling directly on the elbow. Dislocation by traction of the triceps muscle proximally is typical .

Diagnostics:

- Clinical swelling, hematoma, palp . defect, crepitation
- X-ray in two projections

Therapy:

Conservative therapy with plaster fixation exceptionally, in the vast majority of cases of surgery.

Dislocation of the elbow joint

Diagnostics:

- Typical clinical picture with deformity corresponding to anterior or posterior dislocation
- X-ray elbow and dist . humerus in two projections
- Considering the possibility of injury to the median nerve and ulnaris nerve , it is always necessary to examine the periphery of the limb as well
- In old patients, the combination with a fracture of the dist . humerus

Therapy: Urgent reduction under general anesthesia

Complications: Vascular injury (necessary angiographic examination), paresis of the ulnar nerve , compartment syndrome on the forearm especially after prolonged dislocation, paraarticular ossification and stiffness of the elbow joint

FOREARM AND HAND INJURIES (59)

Pavlacký Tomáš, Zukal Radek, Veselý Radek

PROXIMAL FOREARM INJURY

Fractures of the proximal radius

The radius head is an important stabilizer of the elbow and forearm. These fractures are caused by the application of force either directly to the elbow or more commonly indirectly through the forearm. Therefore, they are often associated with other injuries of upper limb.

Diagnostics: Clinical examination with pressure on the radial head during forearm rotations. X-ray of the elbow, forearm and wrist simultaneously with respect to associated injuries.

Therapy: A) Conservative - for non-displaced fractures with joint incongruence up to 2 mm, without ligamentous instability of the elbow. Immobilization for 7-10 days and early functional treatment otherwise stiffness occurs. Relieving aspiration remains controversial.

B) Surgical - for dislocated or comminuted fractures. Isolated screws and LCP plates (2.7 mm or 2.0 mm). In severe comminution extirpation and replacement of the head.

Cave: 1) *Essex-Lopresti* – complex injury of the radial head, interosseous membrane and radioulnar junction, which always requires surgical treatment. 2) After resection of the head, proximalization of radius, valgus deformity and limitation of forearm function occur.

Fractures of the proximal ulna

Coronoid fractures

They are most often part of a luxating elbow injury. Their therapy depends on the extent and localization of the fractured fragment and the degree of dislocation.

Diagnostics: Clinical examination, including instability. In case of therapeutic uncertainty, it is advisable to supplement the X-ray with CT reconstruction.

Therapy: A) Conservative - non-dislocated, up to 5mm. POP for 2 weeks

B) Surgical – plate fixation, isolated screws or anchor.

Olecranon fractures

Almost always intra-articular fractures, that arise from a direct impact on the elbow.

Diagnostics: Clinical findings are significant: swelling, hematoma, sometimes palpable defect and disturbance of extension. X-ray in precise projections is sufficient for isolated injuries.

Therapy: A) Conservative - very limited, only for non-displaced fractures or patients with contraindications to surgery. Plaster fixation in semiflexion for 3 weeks and subsequent functional treatment. Healing is often associated with pseudoarthrosis and associated limitation of movement, but it is surprisingly well tolerated by elderly patients.

B) Surgical – preferred approach. The aim is to restore the joint congruence. For simple fractures, the so-called *tension band wire* (Fig.1) is used. It consists of two parallel K-wires and a cerclage wire. In this way, the distraction on the outer cortices is converted to compression on the inner cortices during flexion, thus exploiting the principle of absolute stability on the articular surface. For comminuted fractures, angle-stable splints, alternatively, a nail fusion, isolated screws or an external fixator are used.

Elbow dislocations

It is the third most common dislocation after shoulder and finger dislocation. It usually occurs in younger patients because the distal humerus rather fractures than dislocates in the older age group with the same mechanism of injury. The most common mechanism is a fall on the extended forearm, when the anterior joint capsule loosens and the olecranon, which is encased in the olecranon fossa, travels dorsally. Less commonly, a fall on the olecranon with the elbow joint flexed results in anterior luxation. Isolated luxation is rather rare, more often there are associated fractures of the proximal forearm.

Diagnostics: Clinical examination reveals obvious deformity and limitation of movement. Stability, neurovascular state and X-ray examinations need to be repeated after the reposition.

Therapy: A) Conservative - in patients without residual instability, rigid fixation for 10-14 days, then brace with limited range of motion

B) Surgical – suture or reinsertion of the collateral ligaments in acute elbow instability, using an anchor. Anatomical reconstruction of associated fractures is important. Chronic instability is resolved by the substitution of associated ligament with a graft or tape.

Cave: Associated median and ulnar nerve injuries, possible compartment syndrome.

FOREARM INJURIES

The forearm is a complex unit consisting not only of the forearm bones, but also their proximal and distal articulation and the interosseous membrane. Therefore, its injuries should be perceived as similarly complex. Thus, an indirect mechanism, although less common, may result in a complex osteoligamentous injury in several stages. (Fig.2) In contrast, fractures resulting from the more common direct violence to the forearm, usually cause isolated fractures of the diaphysis of the radius and ulna.

Diagnostics: On clinical examination, marked defiguration, swelling and crepitations are often present. Radiographs in two perpendicular projections with a consistent view of the elbow, forearm and wrist are necessary.

Therapy: A) Conservative - only for non-dislocated fractures, plaster fixation in neutral position from the metacarpophalangeal joints to above the elbow to prevent rotation for 4-6 weeks with early realignment and release of the elbow in the vertical axis.

B) Surgical - the aim is to restore axis, length and rotation. Plate osteosynthesis is used to achieve absolute or relative stability depending on the nature of the fracture.

Cave: 1) Forearm fractures have the highest incidence of open injuries in the upper limb

2) *Galeazzi's fracture* - fracture of the diaphysis of the radius with simultaneous luxation of the ulna head, solved exclusively by surgical osteosynthesis of the radius and transfixation of the distal radioulnar joint in the correct position. 3) *Monteggio fracture* - fracture of the proximal ulna with simultaneous luxation of the radial head. We also first perform a stable osteosynthesis of the ulna. It is subsequently supplemented by revision and possible suture of the ligamentum anulare in case of persistent luxation of the radial head.

FRACTURES OF THE DISTAL RADIUS

Distal radial fracture is the most common skeletal injury of the upper limb with a bimodal distribution. It occurs less frequently in younger patients as part of a high-energy injury. More often it is a classic osteoporotic fracture, typical of older patients with a female predominance.

Diagnostics: Clinical examination, X-ray in two perpendicular projections clearly defines the stability criteria and possible indications for surgery. CT scan in intra-articular fractures.

Therapy: Before the final decision, the fracture is reduced in LA with 1% Mesocaine injected into the hematoma. Subsequently, traction and counter traction are used to reposition the fracture using the ligamentotaxis. Finaly, repositioning is completed by direct manipulation.

A) Conservative - for minimally dislocated, stable fractures. The limb is fixed for 4-6 weeks in plaster fixation extending from the distal palmar crease below the elbow. Initially it is cut because of the swelling onset.

B) Surgical - for dislocated, unstable fractures. Most often by volar approach using a pre-shaped, LCP plate. For open and grossly comminuted fractures, treatment with an external fixator inserted into the diaphysis of the radius and second metacarpal bone is possible.

Cave: Fractures in this location are often characterized by eponyms. (Fig.3) 1) Colles' fracture– extraarticular fracture with bayonet-like deformity and dislocation of the periphery dorsally and radially. 2) Smith's fracture– inverse to Colles' fracture - it is an extraarticular fracture with volar dislocation. It is more often unstable.

HAND INJURY

Scaphoid fractures

The scaphoid is the most injured bone of the carpus. Majority of the fractures pass through the middle third and are caused by a fall on the wrist in extension. The clinical presentation is associated with tenderness over the *fossa la tabatière* and may be relatively subtle in the early stages. The standard of care for imaging is radiographs in 3 projections supplemented by CT scanning whenthe fracture is suspected and to facilitate classification. Conservative treatment is reserved for non-displaced fractures. Cast fixation immobilizes the thumb and elbow joints in addition to the wrist and is usually left in place for 6 weeks. Surgical treatment belongs to all the remaining categories, with the so-called Herbert screw used for fixation, allowing compression of the fracture due to two threads with unequal lengths and steepness (Fig.4).

Luxation of the wrist

These are severe and complex osteoligamentous injuries. They are divided into *perilunate luxations* in which the lunate remains in contact with the radius and the rest of the carpus is dislocated dorsally, and *lunate luxations* in which the lunate is dislocated ventrally, and the rest of the carpus remains in place. The cornerstone of therapy is early diagnosis, which from plain radiographs can be relatively difficult especially in the AP plane. This is followed by acute closed reposition and then early operative revision of the injured ligaments.

Metacarpal fractures

It is one of the most common hand injuries with typical dislocation. The eponyms describe the most frequent units such as Boxer's fracture in the region of the fifth metacarpal and Bennett's or Roland's fractures in the region of the base of the first metacarpal. Therapy is frequently conservative with plaster fixation in "safe position" (Fig.5) for 4-5 weeks. For dislocated fractures, we use a combination of K-wires, small screws or plates for osteosynthesis.

Fractures and dislocations of phalanges

They are similar in character to metacarpal fractures. The more common conservative therapy is fixation for 3 weeks with an aluminium splint at the level of interphalangeal joints. In the aMCP joint a plaster fixation in "safe position" is mechanically preferable. Given the minimal soft tissue cover and the high risk of adhesions, the surgical treatment is done using K-wires or small screws. It is most often indicated for rotational deviation or significant intra-articular inequality. In this location, there is a low correlation between clinical and radiographic signs of healing. Luxation of interphalangeal joint is one of the most common injuries. Reposition is usually easily accomplished with traction and counter traction followed by fixation for 3 weeks with an aluminium splint or brace.

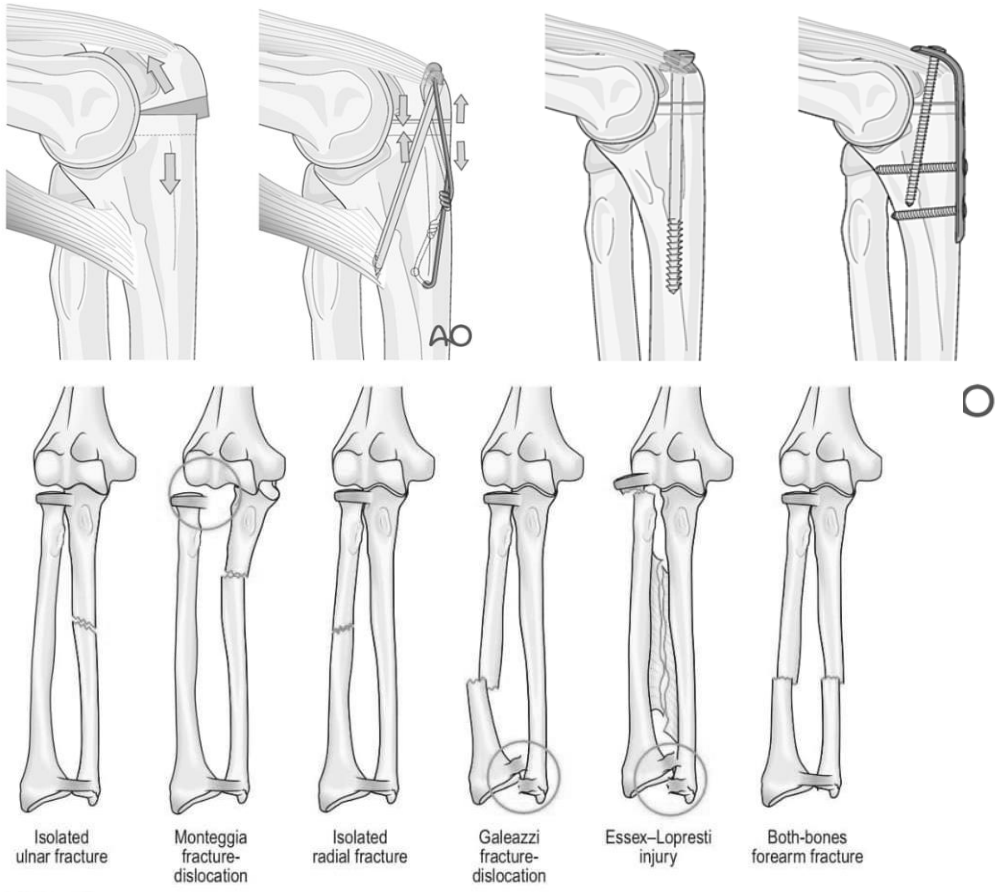


Fig. 2 Types of forearm fractures

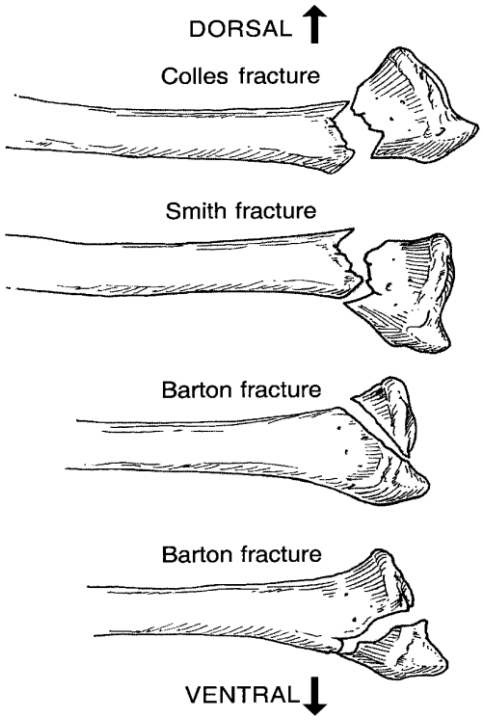


Fig. 3 Types of distal radial fractures

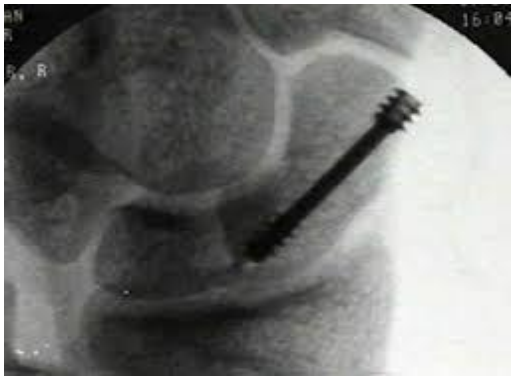


Fig. 4 Herbert screw used in scaphoid fracture

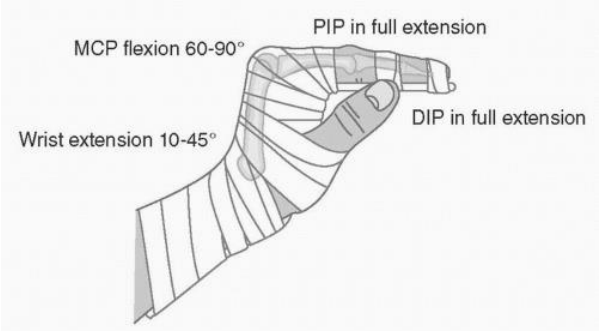


Fig. 5 Safe position for fixation of hand injuries

PELVIC AND HIP INJURIES (60)

Rak Václav

Fractures of the pelvis

Anatomy

The pelvis is made up of two pelvic bones (os ilium, os ischii and os pubis) and the sacrum (os sacrum), forming a confined space containing some abdominal organs.

Epidemiology and etiology

Pelvic fractures account for about 2% of all fractures, but occur in 20% of polytrauma cases. They are most commonly caused by road traffic accidents or falls from height, where high energy trauma is the cause.

Classification

According to the stability of the fracture, there are three categories - stable A, whereby individual bones are injured, - partially stable B (this includes open book fractures), - unstable C, which have a mortality rate of over 25%.

Clinical picture and diagnosis

In stable fractures, pelvic injury is manifested by pain and haematoma, and the patient usually cannot walk due to pain. The diagnosis is confirmed by X-ray (anteroposterior, inlet and outlet projections) and possibly by a refining CT scan. In types B and C, the patient is often in a state of shock from massive haemorrhage, with pain in the sacral region or in the symphysis and lower abdomen if conscious. Pelvic instability is determined by clinical examination, evident by pressure on the iliac crests or symphyseal defect, which is confirmed by radiological methods.

Therapy and complications

Conservative treatment with bed rest is chosen for type A fractures. Displaced fractures can be treated with repositioning and osteosynthesis. Type B and C already require anti-shock treatment, stopping bleeding (angioembolization or extraperitoneal tamponade) and stabilization of the pelvic ring with an external fixator. With a delay after stabilization of the condition, splint osteosynthesis follows, where type C is solved by stabilizing the anterior segment with a splint, the posterior segment with screws, splint or screws. The diagnostic and therapeutic procedure is illustrated in the diagram below. In pelvic surgery, we choose, for example, from an anterior or posterior approach. Due to the proximity of organs of the urogenital and gastrointestinal systems, blood vessels and nerves, pelvic injuries are often accompanied by damage to these structures, which can lead to serious complications, most commonly e.g. injury to the urinary bladder, rectum, blood vessels threatening acute bleeding, thrombosis or embolization.

Fractures of the acetabulum

Epidemiology and etiology

As intra-articular fractures, acetabular fractures usually occur by an indirect mechanism when the femoral head strikes the acetabular socket (typically in car accidents). It accompanies hip luxation in 50%. When the femoral head breaks through the acetabulum, we speak of the so-called central luxation.

Classification

According to clinical anatomy, we distinguish between the anterior and posterior pillar of the acetabulum, which is also used in the classification. There are 3 categories - partial intra-articular fractures affecting one pillar, - transverse fractures passing through both pillars, - intra-articular fractures of both pillars (so-called floating acetabulum).

Clinical picture and diagnosis

The patient suffers from sharp pain in the hip and cannot step on the foot. The limb is in a position of relief (abduction, external rotation). When the acetabulum breaks through, a shortening of the limb is seen. X-ray focus on the anterior (oblique iliac) or posterior (oblique obturator) edge of the acetabulum. CT and 3D reconstruction are used for better clarity.

Therapy and complications

Non-displaced fractures can be treated conservatively - bed rest and rehabilitation. Dislocated fractures are indicated for surgery, when we choose osteosynthetic splints or screws. Because of the localization, post-traumatic arthrosis often occurs, often resolved by total endoprosthesis.

Hip injury

The hip joint (articulatio coxae) is a spherical synovial joint connecting the lower limb and pelvis. It allows movement and provides stability to the body. It consists of the head of the femur (caput femoris) and the socket (acetabulum).

Anatomy of the proximal femur

The proximal femur consists of the femoral head, neck and body. The head forms the articular surface on which the socket for the ligamentum capitis femoris is located. The vascular supply comes from the arteria femoralis through the arteria circumflexa femoris medialis and lateralis. Their preservation during injury plays a role in the viability of the femoral head and the potential risk of avascular necrosis. The neck makes an angle of 125-135° with the body of the femur (the collodiaphyseal angle). An important structure is the Adams' arch, the strongest trabecular part of the bone on the medial side, which plays a role in stabilizing osteosynthesis. On the body of the bone,

we distinguish the trochanter major and trochanter minor humps, which are important for fracture classification and influence fracture stability.

Epidemiology and etiology

Fractures of the proximal femur are common, especially in older women (7th-8th decade). In younger patients, they are the result of high-energy injuries (car accidents, falls). In older people, on the other hand, they often occur in less severe falls, usually as a result of osteoporosis. Head fractures are the rarest. Trochanteric fractures account for over half of proximal femur fractures and heal very well.

Classification

According to Pipkin, head fractures are categorized into four types. According to Pauwels and Garden, we classify cervical fractures, and we further divide them into intra- and extracapsular. According to the course of fracture through the trochanteric mass we divide trochanteric fractures. For a more detailed classification with therapeutic management see the diagram below.

Clinical picture and diagnosis

Typical symptoms are limb shortening, external rotation and semiflexion. The patient cannot step on the limb. Diagnosis includes X-ray (AP and axial projection) including the other side for comparison and possibly CT scan for more accurate determination.

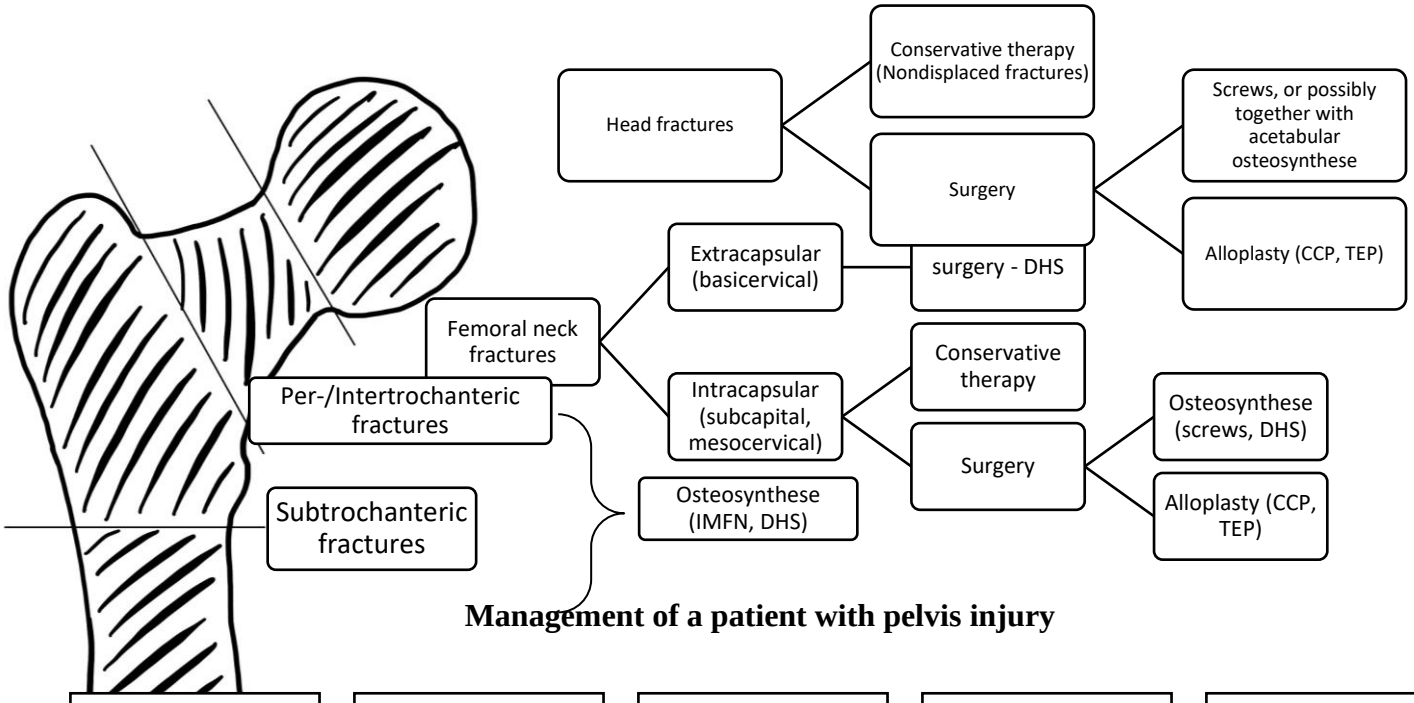
Therapy and complications

Conservative treatment is chosen for stable wedged fractures, but it is associated with a high risk of complications and is nowadays chosen only minimally. Surgical treatment is indicated for all dislocated fractures and includes osteosynthese or alloplasty. Osteosynthese is performed with dynamic hip screws (DHS) or intramedullary femoral nails (IMFN). Alloplasty (CCP - cervicocapital prosthesis, TEP - total endoprosthesis) is the method of choice for dislocated intracapsular neck fractures, for proximal femur fractures with the presence of more advanced arthrosis and for some pathological fractures. Complications also include the development of avascular necrosis of the head in case of injury to the vascular supply, but here it is more rare. Head fractures are often associated with hip luxation or acetabular injury. The healing of fractures may be complicated by the formation of a pseudoarthrosis, or the fracture may heal in a varus position and cause long-term complications even after healing, e.g. a shortened limb.

Hip luxation is a separate chapter. It is a rare injury. It occurs in younger patients in high-energy trauma. It is divided into simple or complex occurring with fractures of the acetabulum or proximal femur. We further divide it into posterior and anterior luxation, with the posterior occurring much more frequently. It is manifested by severe and sudden pain, the patient is unable to walk, the limb is shortened and in pathological rotation. For diagnosis we use X-ray or CT scan for clarification. For

therapy we choose emergent repositioning under general anaesthesia, failing which we choose open repositioning followed by stabilisation.

Fracture of the proximal femur – classification and therapy



Management of a patient with pelvis injury

Pelvic injury	Emergency department Primary survey	Emergency department Secondary survey	Operating theatre Acute treatment	Operating theatre Definitive treatment
•Polytrauma •Unstable patient	•A - Airways •B - Breathing •C - Circulation •D - Disability •E - Exposure	•Physical examination •Pelvic belt loading •Imaging (X-ray, CT scan)	•Bleeding stopping, angioembolisation, extraperitoneal tamponade •Stabilisation, external fixator, clamp	•Osteosynthese: Splint, screws, possibly combination with external fixator

FEMUR, THIGH, AND KNEE INJURIES (61)

Konečný Jan

I. Femur injuries

a) Proximal femur

1. Head fractures - rare, usually with hip luxation. Mechanism - most commonly axial violence in high-energy trauma, 95% of patients have other injuries requiring hospitalization. DG - x-ray - only a rough guide, CT scan is required. Treatment - according to fragment size, fragment location, breakage site, age and general condition. Repositioning of the luxated hip is necessary, the sides of the fracture can be treated conservatively and surgically. Complications - osteonecrosis of the fragment or head, development of arthrosis

2. Femoral neck fractures - common in the elderly, average age between 70 and 80 years, more common in women. Mechanism - low-energy trauma in the elderly, high-energy trauma in the younger. Investigation - history - may not indicate major fall, sometimes associated with impaired consciousness, chest pain etc. Ascertain patient's pre-injury condition and mobility. Clinically - typical position of the injured limb - external rotation, anteflexion at the hip and shortening of the limb, reported pain from hip to knee, worsening on movement. Always examine pelvic stability, blood supply and innervation of both hips. DG - X-ray - injured hip, clear image of the whole pelvis (exclusion of pelvic fracture and assessment of arthrosis of the secondary hip), according to the clinic X-ray of the whole femur and knee. CT scan - only when unclear. Classification - basic division - according to the fracture site in relation to the joint capsule - a/intracapsular - subcapital or mediocervical - risk of head necrosis, b/extracapsular - basicervical or laterocervical - no risk of head necrosis. Further classification - according to the direction of fracture and stability of the fracture - according to Pauwels - I.st. - the fracture has an angle with the horizontal up to 30°, II.st. 30-70°, III.st. more than 70°. According to dislocation - according to Gardena - risk of compromising the vitality of the head. Garden I - incomplete fracture - medial cortical fracture, valgus angulation - laterally the neck is impaction into the head, Garden II - complete fracture without dislocation. Garden III - fracture with dislocation - the neck remains in contact with the head, Garden IV - dislocated fracture - the neck loses contact with the head. Treatment -intracapsular fractures - risk of subsequent necrosis of the head with dislocation, treatment according to the Garden classification. G I - conservative treatment (in case of secondary dislocation implantation of TEP or CKP), G II - osteosynthesis. G III and IV - risk of subsequent necrosis of the head - procedure according to the age, general condition of the patient, time since the injury and arthrosis - osteosynthesis, implantation of TEP - total hip replacement, implantation of CKP - replacement of the head and neck

(lifetime of the implant about 5 years). Extracapsular fractures - without a greater risk of disruption of the nutrition of the head - osteosynthesis, as pertrochanteric fractures.
3. Pertrochanteric fractures - fractures of the area between the trochanters. About 50% of prox. femur fractures - like neck fractures, average age slightly lower - around 70 years. Mechanism - predominantly fall from standing to the ground with direct impact on the region of the greater trochanter in the elderly, in young high-energy. Examination and X-ray diagnosis as for cervical fractures. Treatment- surgical - osteosynthesis with the use of special implants - most often PFN (Proximal Femoral Nail), less often DHS (Dynamic Hip Screw) splints.
Without osteosynthesis, the elderly, often polymorbid patient is immobile for a long time - high risk of complications with a relatively high mortality rate (bronchopneumonia, TEN, decubitus, uroinfections, etc.). Surgery for "vital indication" in the broader sense. Temporal aspect - the least complications are in surgery within 3 days of injury. Do not postpone surgery unless follow-up and preparation of the patient will clearly reduce the risks of surgery and postoperative course. This applies to all fractures of the neck and trochanteric region of the femur.
4.Isolated trochanteric fractures - rare - breakage of the greater trochanter by direct impact - femur stability not compromised, isolated fracture of the lesser trochanter - avulsion - muscle pull. Conservative procedure.
5. Subtrochanteric fractures - up to 5cm distal to the lesser trochanter - cortical bone with little blood supply is highly stressed by a combination of compression and tensile forces, usually dislocated by strong muscle pull. Mechanism - older patients - low energy - "minimal" falls, young - high energy injuries, pathological fractures - more common than elsewhere. Examination - often significant deformity, possibly significant blood loss - hypovolemic shock. X-ray of pelvis, hip and femur including knee. Treatment - under first aid and point C in ABCDE procedure - acute, temporary stabilisation of limb in traction. Non-operative - skeletal traction - in adults rarely. Operative - in adults mostly - intra-articular ridging - aim for closed repositioning. Early surgery reduces blood loss. Complications - relatively common - failure to heal/healing with deformity - high demands on stability of osteosynthesis.

(b) Femoral diaphysis

Fractures - most commonly high energy trauma, less commonly pathological fractures - especially in the elderly after 'small falls', usually dislocated and unstable. Examination - due to the mechanism of careful overall - frequent simultaneous injury to multiple organ systems, There is considerable blood loss even in closed fractures - 1000-2000ml - average 1200ml. Often the same knee is also injured. DG - X-ray - whole femur with both joints in 2 projections. Treatment - first aid - repositioning and

stabilising the fracture with traction - significantly reduces blood loss (point C from ABC algorithm), risk of secondary soft tissue injuries, pain etc.. Definitive treatment - operative - most commonly intramedullary nailing.

c) Distal femur

Fractures - extraarticular - supracondylar , intraarticular . Large muscle forces - typical dislocation of fragments by traction - m.gastrocnemius - dorsal displacement and angulation, m.quadriceps femoris and hamstrings - traction proximally - shortening. Examination - possibility of vascular injury in typical dislocation of dist. fragment. DG - X-ray - baseline, CT - relationship to articular surface. When vascular injury is suspected angiography. MRI only subsequently - diagnosis of soft knee injury. Treatment - conservative - incomplete or undislocated stable fractures - immobilization, for dislocated fractures - skeletal traction for 6-12 weeks - high risk of local and overall complications - therefore rare in adults. Surgical - mostly, type of osteosynthesis - special implants - splints (LCP), spikes, for smaller fragments screws, or a combination of methods. External fixator - in vascular reconstructions and in polytrauma or open fractures - usually temporary, after stabilisation conversion to internal osteosynthesis.

II. Thigh injuries

Muscle injuries - mostly incomplete muscle ruptures, direct mechanism - impact, or indirect - excessive muscle contraction. Treatment mostly conservative - icing, immobilization, gradual mobilization. **Quadriceps tendon rupture** - most common in over 40 years of age, 2cm proximal to the patella. Examination - palpable defect, complete rupture - loss of active extension in the knee. X-ray - to rule out fractures, ultrasound (MRI) may help, but clinical examination is most important. Treatment - complete rupture - surgical - suture or reinsertion, incomplete rupture - conservative - immobilization of the knee with a brace and gradual mobilization (about 6 weeks). **Ligamentum Patellae** - much less common, approach similar.

III. Knee injuries

Fractures of the patella - indirect mechanism - muscle traction - most common - dislocation, direct mechanism - impact, often incomplete fractures, combined mechanism. Examination - extensor apparatus - managing active extension in the knee, possibility of injury to the same hip, tibia, leg in high energy injuries. DG - X-ray - 2 projections. Treatment - conservative - non-displaced fractures without violation of extensor apparatus - fixation - brace for 6 weeks with gradual mobilization. Operative - dislocated fractures with violated extensor apparatus - open repositioning and osteosynthesis - tension cerclage, screws.

Soft knee injuries

Luxation of the knee - not common but threatens the limb - close to blood vessels and nerves, often spontaneous repositioning. Often with fractures of prox. tibia and fibula. Mechanism - high-energy injuries, or sports. Examination - reposition as soon as possible, examination of blood supply to limb - in 20-60% of a.popliteal injuries - reconstruction required within 8h, only then examination of ligamentous apparatus, examination of innervation - n.peroneus - not rushed - most often neurapraxia, complete lesions rare - usually cons. procedure. DG - X-ray - 2 projections, when fracture sides are unclear CT, when vascular lesion is suspected angiography, MRI diagnosis of ligamentous injury only at second time. Treatment - urgent closed repositioning - during investigation, fixation with brace, in case of instability external fixator, vascular reconstruction as needed, open repositioning - if not possible closed - exceptionally. Treatment of ligamentous injury only afterwards.

Patella luxation - more common in women - hypermobility, anatomical predisposition, muscle pull - lateral, medial by direct force, risk of osteochondral fractures of the patella or femur, often spontaneous reposition. Examination - clinic, X-ray. Treatment - repositioning in knee extension, fixation with brace. In recurrent - surgical solution - arthroscopy, correction of patello-femoral instability.

Injuries - of the menisci, ligaments and cartilage of the knee joint

Mechanism - from high-energy - luxation, to sports injuries - ligament and meniscus injuries, to minimal trauma - meniscus injuries. Sports injuries - about 70%. Most often indirect mechanism. Injuries according to the direction and magnitude of the force applied and the position of the knee joint. Examination - history - description of the injury event, difficulties - pain during a certain movement, swelling after loading, etc. Objective - Special manoeuvres on the menisci (if 2 manoeuvres are positive, the probability of injury is over 90%). Imaging - X-ray - skeletal trauma and bone position, MRI - shows ligaments, cartilage and menisci - over 90% yield. Ultrasound - not beneficial. Arthroscopy - the pinnacle of diagnosis and resolution at the same time. Treatment of meniscal injuries - operative - arthroscopy - procedure according to type of rupture, age and comorbidities - attempt salvage surgery (sutures), resection procedures - removal of loose part of meniscus. Treatment of ligament injuries: lateral ligaments - LCM and LCL - mostly conservatively - immobilization with a brace with gradual mobilization - 6 weeks in total, surgically rarely. Crossed ligaments LCA and LCP - acute, mostly conservative procedure, followed by ligament replacement with an interval of several months - most often with the use of tendons m.semitendinosus, m.quadriceps femoris, or lig. patellae - indication according to age, comorbidities and degree of instability.

TIBIA, ANKLE, AND FOOT INJURIES (62)

Zukal Radek, Pavlacký Tomáš, Veselý Radek

Fractures of the proximal tibia are most often caused by falls and side impacts on the knee joint, and there is often a rotational force present. Skeletal injuries are often accompanied by ligament and meniscal injuries. A distinction is made between extra-articular and partially or completely intra-articular fractures. A significant proportion of fractures are comminuted fractures.

Diagnosis is based on clinical examination, when we find an altered contour of the knee joint, limited movement, impossibility of weight-bearing, pain and swelling, or hemarthrosis. The state of innervation and vascular supply at the periphery of the limb should always be carefully examined. The standard radiological examination includes X-ray in two projections, followed by CT scanning when intra-articular fracture is confirmed.

The main aim of therapy is to restore the smooth articular surface, physiological axis and stability of the knee joint, which leads to the preservation of the best possible knee function and allows early rehabilitation. The conservative approach is chosen only when surgery is contraindicated, e.g. due to severe comorbidities of the patient or in case of a non-displaced fracture (infraction). The preferred solution is surgical treatment with osteosynthesis with condylar buttress plates, separate screws or their combination and treatment of the soft knee with special suture material. Preferably, the surgery is arthroscopically assisted. In the case of extensive soft tissue injury or open fractures, a two-stage approach is necessary, whereby a thorough debridement is performed in the first stage and the knee is fixed with a bridging external fixator. Complications include paresis of the n. fibularis, injury to the popliteal vessels (especially in luxation fractures) and compartment syndrome.

Fractures of the diaphyseal part of the tibia are among the most common diaphyseal fractures in general. Due to the poor soft tissue cover in the ventromedial part of the shank, they are largely open fractures. Usually both bones are fractured simultaneously; isolated injury to one bone occurs mainly due to direct violence. The tibia is the predominant injury; the diaphyseal part of the fibula is of little clinical significance as it is not a weight-bearing bone.

Clinical diagnosis is usually not difficult; pain, crepitations, contour changes and pathological movement are present. X-rays are useful to evaluate the type of fracture and to choose the therapeutic approach, and it is advisable to capture the entire fibula. We choose surgical treatment for almost all tibial fractures, because only this way we avoid complications from long-term immobilization and it allows early mobilization of adjacent joints. Temporary cast fixation must be applied over the knee and the ankle with thorough padding at the malleoli, heel and fibula head. For osteosynthesis, closed reduction and the use of an intra-medullar secured nail is the most common

method of choice; open reduction and plate osteosynthesis are also possible in some cases. In the case of open fractures or closed fractures with a greater degree of soft tissue contusion, usage of an external fixator is indicated.

Increased attention in tibial injuries should be paid to the so-called **compartment syndrome**, where, as a result of soft tissue swelling in closed osteofascial spaces, muscle ischemia and oppression of vascular and nerve structures occur. Late or insufficiently radical intervention may result in permanent functional damage to the limb, in extreme cases even amputation. Diagnosis is mainly clinical, with symptoms dominated by swelling and severe pain unresponsive to analgesic therapy. Sensory and motor disturbances are present only at an advanced stage of this condition when there is already developed mostly unrepairable damage to nerve structures. Beware, pulsations in the periphery may be preserved. Treatment is surgical, consisting of dermofasciotomy and pressure relief in all four compartments of the shank.

Fractures of the distal tibia (pilon) are often caused by a high-energy mechanism of injury with soft tissue damage and the presence of comminuted zones and articular surface defects; in most cases, a fibula fracture is also present.

Similarly to the previous types, we find the presence of clear deformity and crepitations; we perform X-ray imaging, and in case of more complex multifragmentary or intra-articular fractures, we also perform CT scanning. Often, swelling and damage to the skin cover is caused directly by the mechanism of injury or secondarily by the pressure of dislocated fragments; early reduction and fixation can prevent skin bullae or necrosis of entire skin regions.

Conservative therapy in plaster cast fixation is acceptable only in non-dislocated extraarticular fractures; its disadvantage is the long period of immobilization of the ankle with subsequent difficult rehabilitation. The surgical solution most often consists of open reduction and stabilization with screws and various types of plate. Initial usage of an external fixator is indicated in similar situations as for other parts of the shank.

Injury to the ankle is one of the most common injuries to the lower limb. Usually, it is caused by distortion mechanism during a misstep or fall even from a small height. Depending on the mechanism of injury, distension or partial rupture of the ligamentous structures of the ankle occurs, most often the anterior fibulotalar ligament in supination injuries and the superficial portion of the deltoid ligament in pronation injuries. Distension of the ligament from a partial rupture can be distinguished clinically by the degree of swelling and the local presence of a haematoma in partial rupture, whereas ankle instability may be present in complete rupture. To determine the severity of ligamentous injury, sonography and MRI are useful imaging modalities. In ankle injuries, we should

always perform an X-ray to rule out possible skeletal injury. In the case of soft tissue injury, we most often indicate fixation in a brace with crutches for two to three weeks.

Fractures in the upper ankle joint have similar mechanisms of formation as the above-mentioned soft tissue injuries, often they are luxation open fractures, which is due to the thin skin cover in this area. In closed fractures, the vitality of the skin is compromised by the pressure of the dislocated fragments; therefore, early, at least approximate, reduction is indicated.

The classification of fractures is based on the Danis-Weber categorization according to the type of distal fibula fracture: below the tibiofibular syndesmosis (type A), at the height of the syndesmosis (B), above the syndesmosis (C).

In bimalleolar fractures, in addition to the fibula fracture, there is also a fracture of the medial malleolus (or rupture of the deltoid ligament) present, in trimalleolar fractures, a fracture of the dorsal edge of the articular surface of the distal tibia is also present. These fractures are considered intra-articular fractures and CT scanning is often necessary to determine the extent of damage.

Conservative treatment in cast fixation is indicated only for isolated type A fractures or for nondisplaced type B fractures without medial side injury and without asymmetry of the ankle mortise. Surgical management consists of open reduction and stabilization with plates, separate screws and Kirschner wires.

A special type of fracture of the ankle is the **Maissonneuve fracture**, i.e. a high type C fracture. Clinically, we find pain in the medial ankle area, on the X-ray we find a fracture of the medial ankle with asymmetry of the ankle mortise, or the X-ray is without evidence of a fracture (only a rupture of the deltoid ligament is present on the medial side). In this fracture, the interosseous membrane between the tibia and fibula is torn and the fibula is fractured in its proximal third. Therefore, during the ankle examination, we do not forget to investigate any pain in the area of the proximal fibula, and in case of an isolated medial ankle injury, we carry out X-ray of the entire shank. A Maissonneuve fracture requires operative treatment, otherwise it results in ankle instability.

In most cases, **Achilles tendon injuries** occur when there are degenerative changes to the tendon present (e.g. local or systemic corticosteroid therapy); open injuries are also possible.

The diagnosis is based on the anamnestic data of the circumstances of the injury, clinically we find palpation pain and a palpable defect, Thompson's test is positive (there is no passive plantar flexion when the calf is compressed). Active plantar flexion is usually preserved to a limited extent by m.plantaris activity. If in doubt, we perform sonography.

The scope of therapy ranges from conservative treatment in plaster or special vacuum fixation (with gradual conversion from full plantar flexion to full ankle movement) to surgical solution with open suture or minimally invasive solution from mini-incision or percutaneous suture.

Talus fractures are usually the result of high-energy trauma, typically a fall from a height or in a car accident. Most common talus fractures occur in the cervical region. For an accurate diagnosis, a CT scan is necessary, treatment consists of osteosynthesis with individual screws.

Fractures of the calcaneus also occur most often as a result of car accidents or falls from heights. Clinically, we find swelling and pain in the heel area, on the lateral X-ray we usually find a straightening of Böhler's angle, for a precise diagnosis a CT scan is necessary. Conservative therapy is reserved for minimally dislocated fractures and, conversely, highly comminuted fractures where operative treatment does not usually lead to better functional results. Other types of fractures are indications for surgical treatment, which is usually complex and involves a range of osteosynthetic material and is associated with the risk of problematic wound healing due to inferior vascular supply to the skin in this area. Fractures of the calcaneus, despite all efforts and care, are not infrequently burdened with permanent consequences.

Luxations in the tarsal area (talus luxation, Chopart joint luxation) and **luxations and fractures in the Lisfranc joint area** are usually the result of low-energy (sports injuries) or high-energy trauma, depending on the severity of the injury. X-ray and CT scan will provide a diagnosis after a thorough clinical examination. Conservative treatment is chosen only for stable and non-displaced injuries, otherwise surgical transfixation with Kirschner wires or screws is indicated.

Fractures of the metatarsal bones are most often caused by direct force, dropping of heavy objects on the foot and in this area we also encounter so-called stress fractures resulting from long-term fatigue. Diagnostics is provided by X-rays, conservative treatment with plaster cast fixation or special walking braces is reserved only for fractures without major dislocation, otherwise surgical stabilization is recommended.

Toe fractures or luxations are caused by direct force, often by tripping during sporting activity. For the proper function of the foot, the big toe is the most important, therefore we pay the most attention to its injuries; in some more serious injuries, surgical treatment is indicated for the big toe, while for most injuries and for other toes we are satisfied with conservative treatment. Instead of fixation with a plaster cast, we prefer adhesive bandage fixation, where the adjacent healthy toe serves as a support for the injured toe. Keep in mind that toe luxations should be reduced as soon as possible (by pulling in the physiological axis of the toe) because of the risk of ischemic damage.

NON-SPECIFIC BONE INFLAMMATION, SPECIFIC INFLAMMATIONS OF BONES AND JOINTS (63)

Jindra Jakub, Mahdal Michal

Osteomyelitis is an inflammatory disease of the bone; if the inflammation penetrates into the relevant joint, it causes its inflammation - arthritis. Inflammation of the spine with involvement of the intervertebral disc is called spondylodiscitis.

Depending on the mechanism of origin, osteomyelitis is divided into haematogenous and exogenous (occurring after trauma to the skeleton - e.g. open fractures).

The course of osteomyelitis can be divided into:

1. acute - the patient's symptoms manifest in the first 14 days to 12 weeks of the disease
2. chronic - often an extension of the acute phase, become chronic (lasting more than 6 months)
3. sub-acute - the time of manifestation is given as 12 weeks from the onset of the disease.

The causative agent of non-specific diseases is most often a bacterium (Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus pyogenes), the causative agent of a less common group of specific inflammations is e.g. Mycobacterium tuberculosis.

The mechanism of infection is the colonisation of bone tissue by pathogenic organisms. Leukocytes are attracted to the bone and enzymes are produced at the site to destroy the bacteria. There are produced cytokines. As a result, blood flow to the areas is reduced and devitalised necrotic bone forms, known as sequestra (infected necrotic bone). Antibiotics have little penetration into the avascular bone and their effect is limited (hence the surgical approach to treatment). The body's defence against infection is to seal the infection by forming new bone around the sequestrum. This new bone is called the involucral bone. If the sequestrum drains through the sinus to the surface, a fistula is formed.

Symptoms of OM	
General	febrillia, inappetence, general alteration of the organism, weight loss in chronic patients, night sweats, hepatorenal insufficiency, MODS
Local	pain, redness, oedema, fluctuating resistance with abscess formation, synovialitis and joint filling with restriction of movement, possibly with contracture formation

Diagnosis of OM	
Laboratory	detect and monitor the overall inflammatory activity of the organism (e.g.: CRP, leukocytosis, procalcitonin, erythrocyte sedimentation rate)
Imaging	X-ray, CT, MRI, scintigraphic imaging
Microbiological examination	culture, PCR

The treatment of osteomyelitis is based on **combination of antibiotic therapy and surgical intervention**. In addition to antibiotic treatment, surgical intervention may be necessary to address the necrotic masses (debridement) and introduce a lavage (a supply and drainage drain into the cavity or joint space, followed by lavage with a dilute disinfectant solution to eliminate the bacterial load). In case a bone defect is present, it can be filled (bone grafts or a bioscaffold). Antibiotics are only effective until a pus-filled deposit is formed; they do not penetrate avascular tissue. Such deposits require surgical removal.

Antibiotics can be applied locally in carriers to the site of infection during surgery. It is recommended that antibiotic administration be continued after surgery.

Acute haematogenous osteomyelitis

Acute haematogenous osteomyelitis occurs mainly in paediatric patients. The course of acute haematogenous OM varies with age due to the different blood supply to the bones (neonates, children, adults):

In neonates, the purulent process penetrates the cortical bone intracapsularly or along the periphery of the epiphysis into the epiphysis and perforates the joint (pyarthrosis). The joint is severely affected by the infection and, despite appropriate treatment, the prognosis is usually poor. Joint deformities and limb length discrepancy occur during growth.

Prematurity is a risk factor for the development of the disease. Prenatal, perinatal and postnatal infections (most commonly umbilical sepsis, purulent skin disease, enterocolitis or pneumonia) can often be identified in the medical history. Treatment consists of immobilisation of the limb and administration of ATB. If pyarthrosis is suspected, an acute puncture of the joint is performed and if pus is aspirated, an acute revision of the joint with lavage and introduction of lavage is indicated. Early surgical intervention minimises joint damage.

Osteomyelitis in childhood predominantly affects the metaphysis. The growth plate presents an insurmountable barrier to the advancing infection, which enters the joint only where the joint capsule

reaches the metaphysis. The focus of infection may be a skin, ENT or genitourinary infection. Chills, high fever, redness and inflammatory oedema over the affected area are present. The site is palpable and painful. Haemoculture is positive in about 50% of cases. If acute haematogenous OM is suspected, puncture of the lesion - subperiosteal abscess or bone marrow - is indicated. Surgical treatment (debridement, lavage) is necessary in sequestration.

The clinical presentation and radiological findings may appear identical to those of Ewing's sarcoma, and only a biopsy and histological examination can provide definitive differentiation.

Acute haematogenous OM is a rare occurrence **in adults**. The infectious focus may be located in the ENT region, the genitourinary tract, the dental area, or in a cutaneous inflammatory condition. The condition predominantly affects the bones of the appendicular skeleton, with the vertebrae being the most commonly affected bones. Infection of long bones is an uncommon occurrence. Those at increased risk of developing this condition include patients with diabetes mellitus, lower limb ischaemic disease, liver disease, immunosuppression or HIV infection. The process initiates at the peridiskal portion of the vertebra, where blood flow is reduced in the broad venous plexuses. The process then progresses to the penetration of the vertebral body, resulting in the destruction of the disc. It may subsequently extend along the disc periphery to the adjacent vertebral body. The process is frequently referred to as spondylodiscitis, given that there is always concomitant involvement of the disc and the adjacent vertebral body. Localised abscesses may arise in the pre-vertebral or epidural region, which may result in neurological symptoms. Magnetic resonance imaging (MRI) is the most effective method for diagnosis. The standard treatment is conservative, comprising corset application (Jewett corset, TLS brace) and long-term (3-6 months) administration of antibiotics (ATB). Surgical revision is indicated when conservative treatment has failed, signs of dural sac compression are present, or when kyphotic progression is evident.

Acute exogenous osteomyelitis is a disease that occurs following soft tissue injuries, open fractures and surgical procedures on bones. It is caused by direct contamination of the affected area with bacteria or their penetration from a nearby site. The most common causative agents are Staphylococcus aureus and S. epidermidis, although there is also a significant proportion of nosocomial infections with resistance, including enterococci, E. coli, Acinetobacter, Pseudomonas, and Klebsiella. In open fractures, infectious complications occur in up to 30% of cases, with a particularly high incidence observed in open fractures of grades II and III according to Tscherne's classification. Infections manifest between three and 14 days following surgical or traumatic procedures. The patient reports a sensation of heat, burning, and twitching in the wound, accompanied by a feeling of tension. Clinically, the presence of swelling, redness, fluctuation and possibly secretion from the wound can be observed. Concurrently, the presence of febrile symptoms,

elevation of laboratory inflammatory parameters and alteration of general condition can be documented. In order to facilitate optimal therapeutic outcomes, the following measures should be undertaken: the release of sutures, performance of an incision, evacuation of the hematoma or pus, debridement and flushing of the wound with a disinfectant solution. The administration of antibiotic therapy represents an essential component of the therapeutic approach.

Secondary chronic osteomyelitis arises from the transition from acute haematogenous or, more commonly, exogenous osteomyelitis (OM) after open fractures or infected osteosyntheses. The underlying cause is the delayed recognition or inadequate treatment of acute osteomyelitis (OM) or the inappropriate management of open fractures and infected osteosyntheses. It occurs in three distinct forms:

1) The inflammatory process subsides, but recurs with an interval of months to years, and the fistula either does not develop or appears intermittently.

2. The inflammation is at an inactive state, yet persistent in its formation of fistulae.

3) The fistula persists, and recurrent exacerbations occur. The disease is long-lasting, frequently lasting a lifetime, and often resulting in the loss of a limb. We are not talking about the cure of chronic OM, but about its transformation into a resting stage. Surgical intervention is indicated when the outcome of conservative treatment is inadequate, when there is pus retention, when exacerbations are more frequent and prolonged, or when sequestrum is clearly evident.

Specific inflammations of bones and joints

The main agent of specific inflammation is Mycobacterium tuberculosis, another agent is Treponema pallidum. Tuberculous inflammation most frequently manifests as spinal disease (spondylitis anterior profunda) when the primary focus is situated in the peridiskal region of the vertebral body. However, in its early stages, the inflammatory process perforates the annulus fibrosus and spreads to the disc, resulting in TBC spondylodiscitis. The infection subsequently spreads to other vertebral bodies. The infected vertebrae are filled with necrotic material and undergo gradual collapse, predominantly in the ventral region. Cold abscesses are formed, which spread prevertebrally and paravertebrally. Due to the action of gravity, these abscesses converge from the cervical spine to the retropharyngeal space, from the thoracic spine to the mediastinum, and through the diaphragm to the retroperitoneum. The infection may also spread from the lumbar spine along the m. psoas through the trigonum femorale to the thigh, or it may perforate the lig. longitudinale posterior and pass into the spinal canal, where it causes dural sac oppression with neurological symptoms. The classic course of this affliction may result in the so-called Pott triad, which comprises an abscess, kyphosis, and paraplegia. The second most common site for tuberculosis of the bone is the knee joint. The

process is initiated by primary involvement of the synovial membrane and develops into exudative synovialitis with hydrops.

PLASTIC SURGERY

CONGENITAL NEVUS, MALIGNANT MELANOMA, SKIN TUMOURS (47)

Rothová Veronika, Fait Vuk, Zapletal Ondřej, Krsička Petr

MALIGNANT MELANOMA

Malignant melanoma (MM) is one of the most malignant tumours. It mainly affects the skin, but it can also affect the eye, ear, leptomeninges, gastrointestinal tract and mucous membrane of the mouth or genitals. Malignant melanoma develops through (by) the proliferation of melanocytes. It can be malignisation of an already present melanocytic nevus or, more often, de novo in more than 70% of cases. Melanoma cells most often spread through the lymphatic vessels from the primary tumour. Initially, it is creating regional nodal metastases. Later, it also spreads through the bloodstream and forms (creates) distant metastases in other organs (lungs, liver, brain).

Development and spread of cancer: Genetic factors and environmental factors contribute to its development. Genetic factors include a family history of malignant melanoma (FAM syndrome – familial MM syndrome), a proven mutation in the CDKN2A gene, and a BRCA 2 mutation. The most significant external factor is exposure to sunlight (UVA and UVB). This is further related to the increased sensitivity of the skin to sunlight (skin phototype I and II), increased exposure to sunlight during childhood, skin burns, increased number of dysplastic nevi or large congenital nevus, immunosuppression, phototherapy (treatment of psoriasis PUVA - psoralens + UVA radiation), overusing a solarium.

Epidemiology: The incidence of MM is increasing dramatically and mainly affects the Caucasian population. In 2021, the incidence was about 24 cases/100,000 inhabitants in the Czech Republic. Mortality was 4 cases/100,000 inhabitants. However, the highest incidence of MM is in Australia and New Zealand. In patients of African, African American and Asian descent, melanoma is mostly found on the soles of the feet, palms and under the nail plates **(Acral lentiginous type),** which is, in contrast, rare in the Caucasian population.

The most common clinical variants of melanoma:

- **Superficial spreading melanoma (SSM)** – is the most common and accounts for about 70% of all melanomas. It has the classic ABCDE warning signs (see below).
- **Nodular melanoma (NMM)** – is the second most common variant of melanoma, accounting for about 15-30%. It is characterized by rapid vertical growth and the formation of nodules that enlarge rapidly.
- **Lentigo malignant melanoma (LMM)** mainly affects older individuals over 65 years of age. It appears mainly in the head and neck, rarely on the backs of the hands and feet. It arises from

lentigo maligna, which is carcinoma in situ; the transition to invasion takes (in) an average of 10-15 years.

- **Acral lentiginous melanoma (ALM)** is a melanoma that mainly affects the soles, palms, and subungual areas. Diagnosis is often challenging and delayed, as ALM is frequently misdiagnosed as subungual haematoma, bloody verruca, or clavus. **Subungual melanoma** is manifested by a diffuse change of colour under the nail plate. The spread of pigment to the proximal or lateral nail plate is referred to as *Hutchinson's sign*. It is very important to distinguish subungual melanoma from subungual hematoma. The basic difference is that the subungual hematoma deposit gradually grows, shrinks and disappears, whereas subungual melanoma does not.

Differential diagnosis: Melanoma needs to be distinguished from pigmented basocellular carcinoma, spinocellular carcinoma, seborrheic keratosis, dysplastic nevus, hemangioma, and subungual hematoma.

Diagnosis: This includes clinical and dermatoscopic examination using a dermatoscope, which focuses on examining the skin of the entire body, including the palms, soles (feet), and scalp.

An **ABCDE rule** was created that evaluates the warning signs of melanoma:

- **A - Asymmetry**
- **B - Border** (edges) - irregular edges,
- **C - Colour** - spotted colouring,
- **D - Diameter** - diameter greater than 6 mm,
- **E – Evolution** (development) – permanent increase in time.

If 3-4 of the above parameters are met, the suspicion of melanoma is high.

Excisional biopsy and histological examination are necessary in case of unclear clinical findings. It is performed by complete excision of a suspicious skin lesion with a margin of 1-3 mm under local anaesthesia and closure of the defect with a linear suture. When this is not possible due to the size or unfavourable location of the skin lesion, an excisional biopsy - a smaller biopsy from the most suspicious part of the lesion - is recommended. If subungual acral lentiginous melanoma is suspected, a biopsy is taken after fenestration of the nail.

Histopathological examination must include one of the most important prognostic factors, which is:

- The thickness of the tumour in millimetres according to the **Breslow thickness**.
- The **histological subtype** of the tumour.
- The presence of **ulceration** on the tumour surface.
- The depth of invasion according to the **Clark scale**.
- The **mitotic index** (the number of mitoses per 1 mm2).

- The presence of microscopic **satellites**.
- The presence of **regression**.

Imaging methods for staging to diagnose distant metastases are X-ray, ultrasound, CT, PET/CT, and MR; however, in advanced disease, PET/CT is most appropriate. The indication is considered individually.

Melanoma stages: The international TNM classification for melanoma is determined as follows: T (tumour) is determined by the tumour's thickness (Breslow thickness), N is the status of the draining lymph nodes, and M describes whether the cancer cells have spread and made as distant metastases. After completing the TNM classification, patients are, based on the findings, divided into Stages 1-4. The stage provides information on how big the tumour is and how far it has spread. Treatment management is then determined based on the stage and clinical condition of the patient.

Treatment: The basis is **surgical treatment**:

a) Surgery to remove primary tumour - wide local excisions / re-excision of margins. This procedure aims for the complete removal of the tumour with a sufficient margin of healthy tissue or re-excision of the scar at the site of the primary melanoma and subsequent closure of the skin defect. The ideal option is a primary linear suture; in case of larger defects, plastic surgery methods, such as skin graft or flap reconstruction, must be used. The recommended clinical margin is between 1-2cm, determined by the thickness of the melanoma, and can be modified considering anatomical site and functional needs. According to many statistics, a larger hem is not of fundamental importance for the occurrence of further recurrences and the prognosis of the disease. A specific situation is subungual acral lentiginous melanoma when exarticulation of the distal limb of the finger is recommended.

b) Surgery to remove lymph nodes:

– *Sentinel Lymph Node Biopsy (SLNB)* - is primarily a diagnostic method. The goal is to identify or rule out metastatic involvement in the regional lymphatic basin. Sentinel lymph node biopsy (SLNB) is recommended for melanomas with nodularity, ulceration or regression and in the case of histologically verified melanomas with a thickness (Breslow) of more than 0.8 mm. Several methods can be used to identify the sentinel lymph node (SLN). In the 1990s, Morton et al. developed the first method for identifying SLB, which was based on using lymphatic mapping with dye (patent blue or methylene blue). Subsequently, another option, using a radio-colloid labelled with the isotope technetium (Tc-99), was developed. For the most accurate identification of SLNB, numerous specialists use a combination of both methods. Other techniques, such as the ICG fluorescence or Semtimag technique, are not commonly used in routine practice. The localization substance

infiltrates the vicinity of the tumour/scar, from where it reaches the first lymph node via the lymphatic channels. Subsequently, SN is detected and extirpated using a radiocolloid using a gamma probe.

Among the most common locations are the axilla, groin, head and neck area, the iliac area, or, less commonly, the popliteal area. If metastasis in SN has not been confirmed, there is a high probability that other lymph nodes are not affected. Initially, SN involvement used to be an indication for complete lymphadenectomy. However, nowadays, those patients more frequently receive adjuvant systemic therapy - immunotherapy rather than for further radical surgery.

– *regional dissection (lymphadenectomy)* - therapeutic performance is indicated in case of evidence of recurrence metastases in the regional lymphatic region. Evidence of metastases can be clinical or sonographic with biopsy verification. According to the location, the following are most often performed: Axillary Lymph Node Dissection (ALND), ilio-inguinal dissection (ILD) and cervical dissection, rarely popliteal dissection. Undesirable consequences include limb lymphedema, neuropathy, and limitations in movement.

c) Managing locoregional relapses - Melanomas tend to relapse often, even after several years. Patients might develop local recurrences, regional nodal relapses, in-transit metastases, multiple limb relapses, and distant metastases that can occur anywhere (predilection lungs, liver, brain). Primarily, the solution is always surgical. If surgical resection is no longer possible, local radiotherapy is recommended. In exceptional cases, the hyperthermic isolated perfusion method can be used for limb relapses. In the case of solitary metachronous distant metastases, the finding can be solved by resection procedures.

Systemic oncological treatment: In recent years, targeted treatment and modern immunotherapy have been used for advanced disease (stage III and IV). The indication for targeted treatment is linked to the presence of a BRAF oncogene mutation (positivity is present in about 40% of patients). In the case of immunotherapy, these are monoclonal antibodies. If these treatment options cannot be indicated, palliative chemotherapy and the use of cytostatics can still be offered. Irradiation of, for example, brain metastases, skin, bone or nodal metastases can also be indicated as a palliative treatment.

Prognosis: The prognosis corresponds to the clinical stage, or melanoma thickness - for a tumour with a thickness < 1 mm, the probability of 10-year survival is 92%. On the contrary, for a melanoma > 4 mm, it is approx. 50%. Patients with affected lymph nodes have a worse prognosis. The prognosis with disseminated melanoma is very poor, and approximately 10% of patients survive 5 years. Recently, with the advent of modern oncological treatment, survival has improved significantly.

NON-MELANOMA SKIN TUMOURS

Among the most common are basocellular carcinoma (basaliomas) and squamous cell carcinomas (spinalioma). The main risk factor is UV radiation, which mostly affects elderly patients. Basaliomas grow slowly (within years) - locally invasive and destructive growth is rare, and they do not produce distant metastases. Spinal tumours can grow exophytically and have a greater disposition to metastasize. The basis of diagnosis is clinical and histological examination. In advanced spinal tumours, an ultrasound examination of the lymph nodes is performed for the risk of lymphogenic dissemination. The basic treatment is surgical removal of the skin tumour with a border to healthy tissue. If surgical treatment is not possible, cryotherapy or curative radiotherapy can be used. Systemic targeted treatment can be used for locally advanced and metastatic basal cell carcinomas. In the case of a disseminated spinal tumour, palliative systemic chemotherapy is recommended. The prognosis for both skin tumours is excellent. 90-95% of patients are cured by radical excision without affecting the nodes.

CONGENITAL NEVI

These are benign melanocytic skin tumours. If they do not change or enlarge, it is not necessary to remove them. However, their excision is usually performed for cosmetic reasons. In excision, a minimal margin into healthy tissue is sufficient. Dysplastic nevi (DN) is a separate group - a clinical unit between melanocytic nevus and melanoma. Their clinical appearance resembles melanoma in situ. DN carriers have a higher risk of developing melanoma than the general population. Excision is indicated in case of clinical and dermatoscopic uncertainty. Correct histological evaluation by an experienced pathologist is important.

SKIN GRAFTS AND LOCAL SKIN FLAPS (46)

Hokynková Alica, Streit Libor, Šín Petr, Matysková Dominika

Skin grafting and flap plasty are used in the reconstruction of skin and soft tissue defects of various origins, extents, and locations with the aim of restoring the damaged skin coverage. In reconstructive surgery, there are a number of reconstructive techniques, and their selection is guided by the reconstruction ladder, meaning from the simplest technique to the most complex (Fig. 1). Defects of limited extent can be closed with direct suturing. Larger defects can be reconstructed by autotransplantation of a skin graft without a vascular supply, or by a skin flap, which has a vascular supply, and due to the included subcutaneus tissue the reconstruction is more complex than in the case of a skin graft (Fig. 2).

Skin Grafting

Skin grafting is a surgical procedure aimed at restoring the integrity of the skin by transferring it from a healthy area to a damaged area. A skin graft always consists of a full layer of the epidermis and varying layers of the dermis. The transfer of skin graft is facilitated by the fact that skin without subcutaneous tissue remains temporarily viable until capillaries grow into the graft from the base of the wound, ensuring its nourishment. Unlike flap plasty, skin grafts do not have their own blood supply. Skin grafting is indicated for reconstructing skin defects of various etiologies, such as burn injuries, traumatic defects, infectious complications, venous ulcers, etc. It is not applied to defects exposing deeper structures, such as tendons, nerves, blood vessels, and bones. Harvesting is performed using an air- or electro-dermatome, Watanabe knife, or Humby knife, or scalpel for smaller defects. Skin grafts are fixed itno the wound with stitches or metal clips and are slightly compressed against the base of the defect. Uncomplicated healing typically takes about 7–10 days. Skin grafts are classified according to the type of donor into: **1. Autotransplantation (autograft):** This is the most commonly used type of transplantation, where skin is taken from one part of the patient's body and transferred to the skin defect, making the skin donor also the recipient; **2. Allotransplantation (allograft):** In this type of transplantation, skin graft is taken from the body of another person; in the case of identical twins, the graft is referred to as an isograft. Allografts are applied only temporarily (until autotransplantation can be performed) to prevent rejection; **3. Xenotransplantation (xenograft):** This type of transplantation uses skin from another species, most frequently from pigs, and is applied only temporarily.

Types of Autografts by Thickness and Contained Tissue:

Dermoepidermal Skin Graft (Split-thickness skin graft - STSG): This type includes only the epidermis and part of the dermis (thin 0.15–0.3 mm, moderately thick 0.23–0.45 mm, thick 0.45–0.6 mm). For extensive defects, the harvested graft is expanded using meshing with a mesh dermatome, allowing for the graft to spread over a larger area through multiple small incisions (mesh), that enable fluid drainage and quicker adherence of the graft to the base. These grafts heal faster but are less mechanically durable than full-thickness grafts. They are suitable for covering large surface defects. The donor area (usually the ventral side of the thighs) is typically healed by reepithelialization in about 14 days.

Full-thickness Skin Graft (Full-thickness graft - FTSG): The full-thickness skin graft includes the entire epidermis and dermis (0.6 mm and more). The graft is only perforated with the point of a scalpel to allow fluid drainage from the base of the defect. These skin grafts are primarily used in the reconstruction of defects in the head area, showing better aesthetic results and greater durability. They are suitable for covering smaller defects. The donor area (retroauricular, supraclavicular area, groin, etc.) is sutured.

Flap plasty

Flap plasty utilize the transfer of tissue (skin and subcutaneous fat, sometimes also fascia, muscle, etc.) from a donor site to the defect. A flap always has its own blood supply (fasciocutaneous, myocutaneous, direct cutaneous), which is crucial during its planning and preparation. Flaps are categorized according to various criteria, including distance (local, distant), tissue content (skin, fasciocutaneous, myocutaneous, osteomusculocutaneous-composite, visceral, etc.), blood supply (random, axial, myocutaneous, fasciocutaneous), method of transfer (rotational, transpositional, free, etc.), function (sensory, innervated muscle, transfer of a toe from the foot to the hand, etc.), and the time interval from the injury, etc. **Local flaps** utilize surrounding tissue for reconstructing defects. They can have various shapes and methods of transfer, such as island (unidirectional or bidirectional), rotational, transpositional, sliding, and island "keystone flaps," etc. Local flaps can also be combined with one another to close defects. Local flaps are very frequently used to reconstruct defects after skin cancer removal, typically in facial area, but it can iclude larger volumes of transferred tissue, such as the rotational fasciocutaneous gluteal flap in the reconstruction of sacral pressure ulcers. An example of the use of a double transpositional flap is Z-plasty, which is used to address the distribution and elongation of contracted scars (Fig. 3b). **Distant flaps** are characterized by the use of tissue that is located away from the defect site. They are utilized in cases where local flaps cannot be used. Distant flaps can be direct, where the flap is directly connected to the defect

and a temporary pedicle remains preserved between the defect and the donor site, allowing for the growth of neocapillaries between the defect and the flap. Subsequently, after 3-5 weeks, the pedicle of the flap is severed. An example is the inguinal flap used for reconstructing defects on the hand. In this specific case, the flap is sewn onto the defect on the hand (Fig. 4).The most technically demanding are **free flaps**, where the composite tissue is dissected on a specific nutrient pedicle, transferred to the defect site, where it is connected to recipient vessels using an operating microscope. These operations are discussed in the chapter "Microsurgical Reconstruction: Free Flaps and Allogenic Transplantation", which is part of the Addendum to this book.

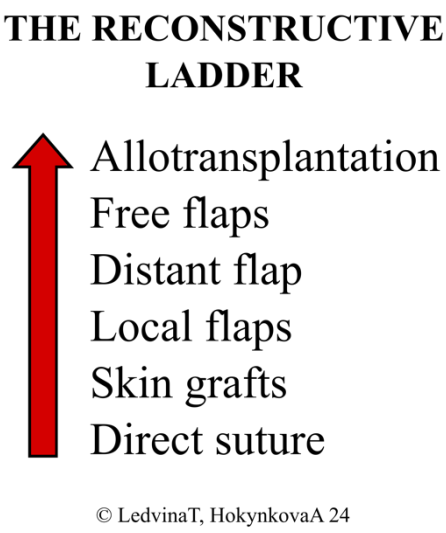


Fig. 1: Reconstructive ladder

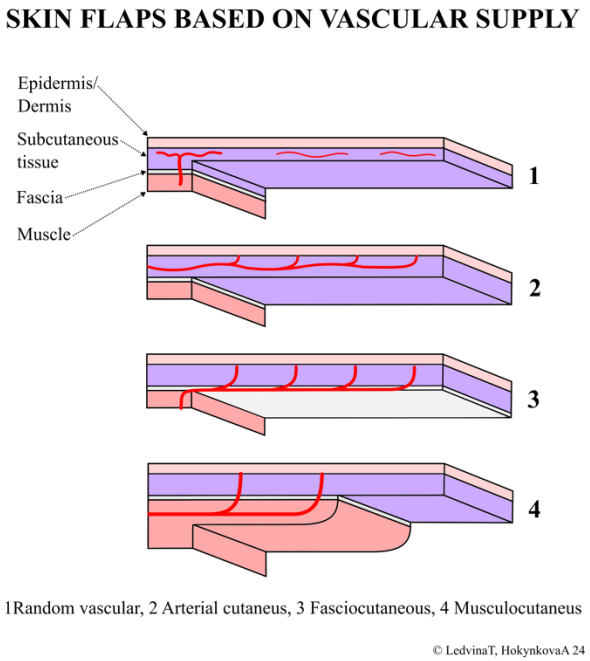


Fig. 2: Blood supply of a flap

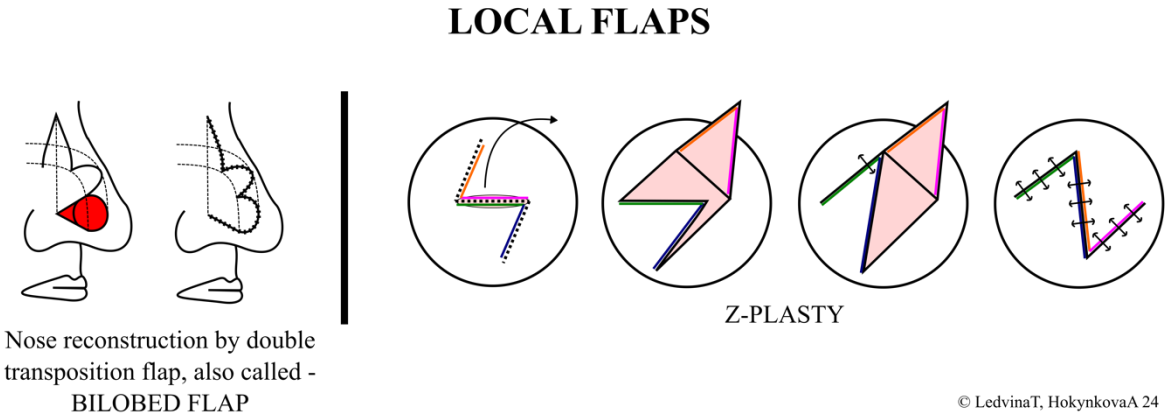


Fig. 3: Local flaps: a) Reconstruction of the nasal defect with double transpositional „bilobed“ flap b) Z-plasty

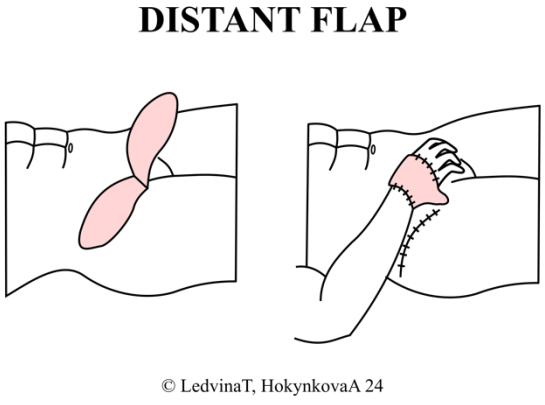


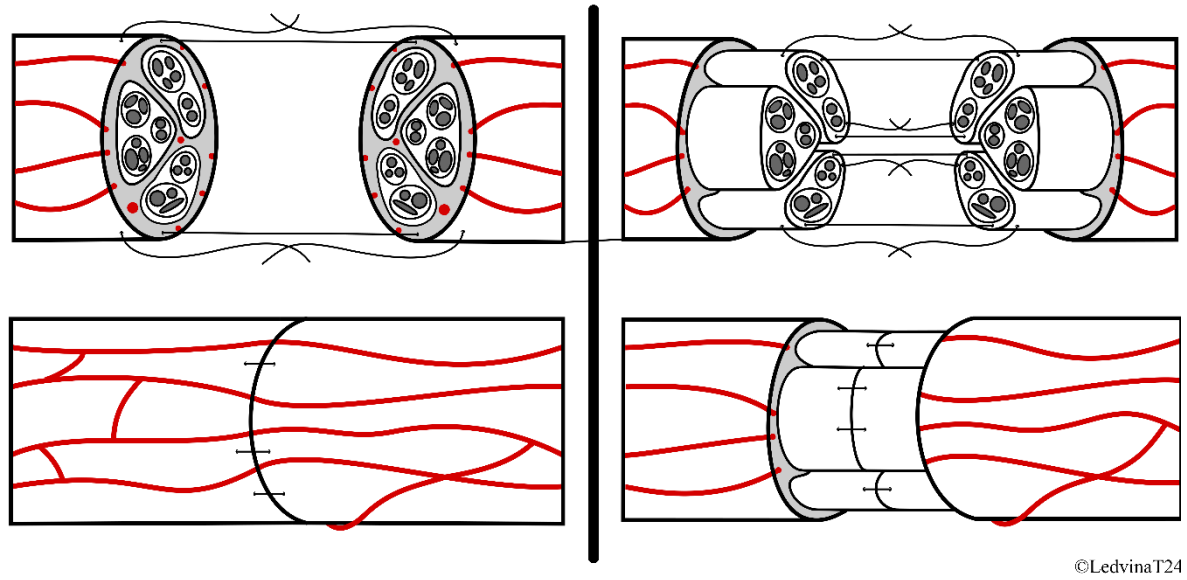
Fig.4: Distant flaps

NERVE REPAIR, GRAFTING AND TRANSFERS (50)

Janeček Pavel, Klabusay Filip, Dvořák Zdeněk, Streit Libor, Bayezid Can Kadir

The principle of repairing a damaged nerve

The basic prerequisite for successful peripheral nerve repair is the restoration of nerve continuity in case of nerve severance. The rule of thumb is the use of microsurgical technique with adequate magnification, the use of microinstrumentation and suture material of 8/0 - 10/0 thickness (0.2 mm thread thickness). A tension-free suture is essential for a successful suture of the nerve, and partial mobilization of the nerve can be performed to facilitate suturing. In the event that the loss of injury is so great that tension-free suture of the nerve cannot be achieved, it is preferable to proceed with the use of a nerve graft. Depending on the size of the nerve, we choose 2 types of suture. For smaller nerves where motor and sensory fascicles cannot be identified, we choose the epineural type of suture, in which we adapt the nerve ends to each other behind the epineurium with several (usually four) single sutures. For larger nerves, an interfascicular suture can be used, in which we suture the individual fascicles together.



epineural type interfascicular type

Timing of nerve reconstruction

The best therapeutic results are achieved with immediate nerve reconstruction. The actual shape and position of the fascicles and the vascular network on the nerve surface allow proper coaptation of the nerve stumps.

With early reparation, retraction of the nerve stumps and the formation of a neuroma does not occur. In addition, motor fascicles can be differentiated by stimulation within 72 hours after injury.

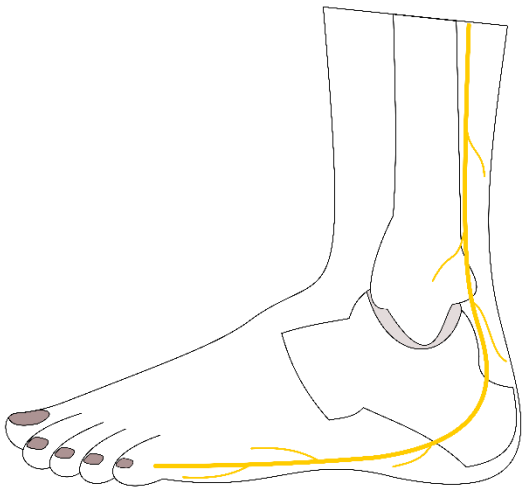
In situations where adequate primary treatment is not possible, nerve stumps should be approximated to prevent retraction and subsequent reparation delayed until the general condition of the patient or wound allows, preferably within 3 weeks of injury. On subsequent revision, the resulting neuroma must be resected down to healthy tissue. The mechanism of peripheral nerve injury also has a significant effect on the final outcome, with the best results being those in which there is a sharp break in the nerve and the worst results in crush or avulsion type injuries.

Nerve grafts

If the nerve stumps are too far apart and a tension-free suture cannot be achieved, nerve grafts should be used to reconstruct the nerve. The grafts are placed in the defect according to the position of the fascicles. To avoid loss of part of the axons before distal anastomosis by branching, it is advisable to rotate the inserted nerve graft 180°. Nerve grafts are divided into autografts, allografts and conduits.

Autographs

Nerve grafts taken from another part of the patient's body. For the purpose of a nerve graft, we usually use nerve-sensitive nerves, mainly because of the morbidity of the donor site (the loss of surface sensation is less severe for the patient than the loss of motor function of a particular muscle). The most commonly used donor nerve is the sural nerve, from which a graft 30-40 cm long can be obtained in an adult and can be removed by a special forceps from small incisions. After its removal, there is a sensory loss on the lateral ankle and lateral heel side, the extent of which diminishes with time. For smaller nerve defects, the lateral cutaneous antebrachial nerve from the forearm can be used. The usual length of the graft obtained is approximately 8 cm. Another variant is medial cutaneous antebrachial nerve (in the groove between triceps muscle and biceps muscle), in this way a graft of up to 20 cm length can be obtained. Of the motor nerves, the distal part of the interosseous antebrachial anterior nerve or intercostal nerve can be used for graft harvesting.



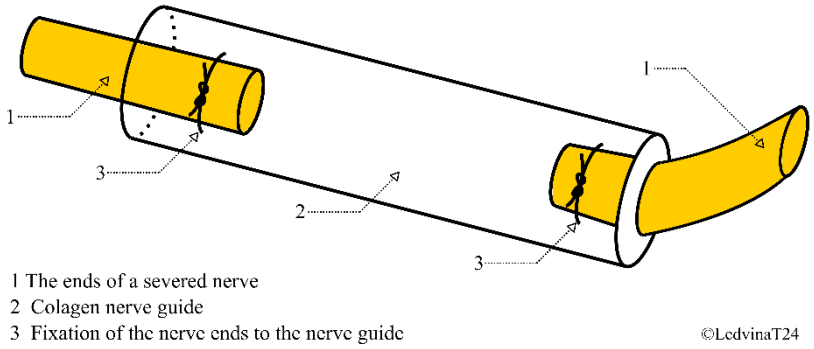
sural nerve

Allographs

Nerve grafts from cadaveric donors. Sterilized and decellularized, they essentially serve as a matrix into which the axons of the proximal end of the injured nerve grow. Due to the decellularization method, there is no need to worry about graft rejection or other immunogenic reaction. The use of allografts has similar results to autografts.

Conduits

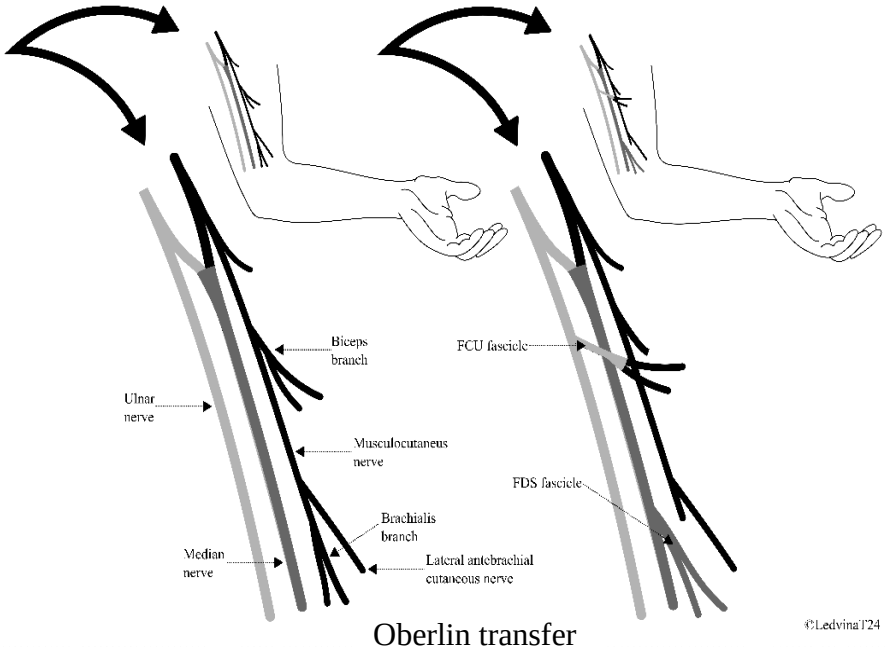
Nerve graft made of artificial material (most often collagen). Not immunogenic, it can be purchased in multiple diameters and lengths. Compared to autografts and allografts, they have poorer regenerative results according to available studies, and their other disadvantage is their high cost.



conduit

Nerve transfers

Nerve transfers (neurotization) are most often indicated for very proximal peripheral nerve injury or for avulsion of nerve roots from the spinal cord. In these cases, a nerve transfer will facilitate better reinnervation of the effector organ (muscle) than would be achieved with the use of a nerve graft.



Oberlin transfer

In motor nerve transfer, donor nerves are used, i.e., nerves that are detached from the functional muscle that is dispensable and, when connected to the distal stump of the injured nerve, will innervate the desired muscle unit. Because of the speed of regeneration, we try to perform the nerve coaptation as close as possible to the motor discs of the reinnervated muscle, and the use of a motor nerve of a synergistic muscle is also an advantage. One example is the Oberlin tranfer for elbow flexion recovery, which is used in brachial plexus injuries when the n. ulnaris is preserved. Thus, part of the motor fibers of the n. ulnaris are reconnected to the biceps muscle branch of the n. musculocutaneus.

Failure of peripheral nerve reconstruction

For motor nerves, it should be remembered that the lifetime of neuromuscular discs in skeletal muscle without innervation is a maximum of 2 years, and it also depends on the age of the patient. The earlier the reconstruction is performed, the better the chance of success. The electrostimulation of the affected muscle is indispensable and should be carried out until its full reinnervation. The time aspect must be taken into account, where we can approximate a nerve regeneration rate of 1mm/s and a delay in the anastomosis of approximately one month. If successful reinnervation of the muscle does not take place, it is effectively lost forever as a motor unit for the organism. In this case, reconstruction of the lost function can be accomplished by free microvascular transfer of the motor unit, i.e., the functional muscle, which can be sacrificed (e.g., m. gracilis) or the muscle function is replaced by tendon transfers. Their principle is to reconnect the tendon apparatus of the less important functional muscle to the tendon apparatus of the non-functional key muscle. In the case of sensory nerves, reconstruction can be performed at any time during life, and there are known cases of tissue regaining sensitivity even after 40 years. All these techniques belong to the field of highly specialized reconstructive surgery.

Facial Paralysis

The resting position and movement of a healthy face are determined by the actions of the muscles of facial expression, which are innervated by the facial nerve. A lesion of the facial nerve hampers the ability to generate facial expressions. Facial palsy is a distressing condition that causes both functional and aesthetic issues, posing significant challenges to the patient's quality of life. Facial palsy can be subclassified as central or peripheral, depending on the location of the lesion. Central paresis results from damage to the brain center or fibers above the nerve nucleus (lesion of the upper motor neurons). In the case of peripheral palsy, the facial nerve is damaged at the level of the lower motor neurons during its course after arising from the pons. Peripheral palsies are more common and manifest on the affected side. In complete peripheral palsy, we observe drooping of the eyebrow and modiolus, an effaced nasolabial fold and absent function of the orbicularis oculi. The eyelids do not close, blinking disappears, and the protective functions of the eyelids are impaired. The prognosis of paralysis can range from persistent complete (flaccid) paralysis to the full return of normal facial function. However, there is a wide range of clinical presentations, from hypoactivity to hyperactivity, between these two extremes in spastic facial palsy.

The aim of complex surgical treatment for **flaccid facial palsy** is to restore the resting position and symmetry of the face, recover the protective functions of the eyelids, and, ideally, also restore facial

movement and the ability to smile. To correct incomplete closure of the eyelids (lagophthalmos), oculoplastic operations are performed, most often using lateral tarsorrhaphy, passive suspension of the lower eyelid, and/or implantation of a platinum weight in the upper eyelid. To restore movement, facial reanimation surgery is performed within 2 years of the onset of paralysis, most commonly in the form of neurotization procedures. These procedures aim for tensionless nerve coaptation of the interrupted or missing section of the facial nerve, either through end-to-end anastomosis or other anastomotic approaches involving nerve grafts, enabling the connection of the distal end of the nerve stump to the healthy contralateral facial nerve. A different approach involves connecting the peripheral branches of the facial nerve to other cranial nerves on the ipsilateral side (e.g., the masseteric nerve). After 2 years from the onset of paralysis, irreversible changes occur in the motor end plates of the facial muscles, leading to permanent atrophy and fibrosis. A reconstructive procedure to replace the mimetic muscles is required. An example of such an operation is reanimation by so-called lenghtening temporalis myoplasty or a free muscle transfer (most often the gracilis muscle). **The spastic type of facial nerve palsy**, characterized by synkinesis, arises as a result of aberrant nerve regeneration, which most often occurs after viral palsy or Bell's palsy. The treatment of this type of paresis typically consists of neuromuscular training, the application of botulinum toxin, and, in resistant cases, selective neurectomy of the distal branches of the facial nerve.

HAND AND FINGER INJURIES AND AMPUTATIONS (48)

Hokynková Alica, Šín Petr, Matysková Dominika

Injuries of the hand may involve defects in the skin, connective tissues, tendons, intrinsic hand muscles, blood vessels, nerves, or the skeletal system. These injuries are typically caused by trauma, often resulting from occupational accidents, and can range from minor superficial wounds to severe, devastating injuries, including amputations (for more detailed information, refer to the chapter on Replantation). The cornerstone of preoperative evaluation is a thorough clinical examination of the injured hand. This assessment includes evaluating the size, depth, and location of the defect, as well as the functional status of key structures—such as mobility (e.g., flexion, extension, abduction, adduction, radial and ulnar deviation), blood circulation (assessed by skin color, temperature, and capillary refill), and sensory function (evaluating the distribution areas of the median, radial, and ulnar nerves). Scars from previous injuries or surgeries should also be carefully documented. For severe injuries in the hand area, it is necessary to think about the risk of compartment syndrome. In the indicated cases of severe hand injury, it is necessary to perform releasing fasciotomies of individual compartments in the forearm and hand area. The aim is to relieve the pressure in the fascial loci due to increasing swelling of the muscle groups, ev. also to release the carpal tunnel by dissection of the carpal ligament to prevent median nerve retraction.Additionally, a detailed history of the trauma is essential, including the mechanism of injury (e.g., avulsion, laceration by sharp or blunt objects, animal bites, or complex trauma involving mechanical, pressure, or thermal components such as burns from hot rollers), as well as the time of injury and, if relevant, the duration of tourniquet application in cases of severe hemorrhage. The dominant hand should be identified, and a complete personal history, including age, comorbidities, smoking habits, occupation, hobbies, and tetanus vaccination status, should be obtained. In cases where skeletal involvement is suspected, radiographic imaging in both anteroposterior and lateral views is performed, with additional projections or imaging modalities considered if indicated. The management of hand injuries is performed in an operating room, under antibiotic prophylaxis, in sterile conditions, and using appropriate anesthesia—whether local, regional (e.g., digital block, wrist block, axillary block), or general—depending on the extent of the injury and the planned reconstructive procedure. As a general principle, surgery is carried out under bloodless conditions, achieved using a tourniquet, and magnification (such as magnifying loupes) is often utilized. The surgical exploration involves extending the wound proximally and distally to properly visualize the extent of the injury and assess the nature and amount of tissue loss. This evaluation guides the choice of the reconstructive method, which may range from simple direct suturing to more complex procedures such as free flap

reconstruction. Whenever possible, the reconstructive procedure is performed immediately, provided the condition of the wound permits. However, in cases involving severe contamination, infection, or anticipated necrosis of the surrounding tissue, temporary wound management techniques are applied. These may include the use of synthetic wound coverings, moist wound healing methods, or negative pressure wound therapy (vacuum-assisted closure). Definitive reconstructive surgery is then delayed until a later stage, when the wound is more suitable for intervention.

Superficial Skin defects

Superficial skin defects of the hand refer to injuries where the skin layers (epidermis, dermis, or subcutaneous tissue) are absent, but crucial underlying structures—such as tendons, ligaments, joint capsules, blood vessels, nerves, and bone—remain intact. These injuries commonly result from sharp instruments such as glass, knives, or woodworking tools like planers. In cases of minor injuries, direct suturing of the defect may be feasible after mobilization of the surrounding tissue, although this is often limited in the palmar region. Small, superficial wounds—such as those on the fingertip—can be allowed to heal by secondary intention, typically managed with antiseptic ointments. For excoriation injuries limited to the epidermis, a conservative approach is preferred.For larger superficial defects that are not infected, skin grafting may be indicated. Either full-thickness or split-thickness skin grafts are applied and secured to the wound bed using metal clips or sutures, along with gauze compression. Skin grafts generally take 7 to 10 days to heal, with full-thickness grafts requiring more time. The final outcome typically results in a flat scar with some degree of contraction, which tends to be more pronounced with thinner grafts. Comprehensive scar management, including the use of silicone gels, compression sleeves, pressure massage, and even laser therapy, may be required. Rehabilitation plays a critical role in preventing contractures, which could otherwise limit the range of motion, particularly in the hand and fingers.

Deep Skin Defects

Deep skin defects are characterized by the absence of skin coverage, with exposed or damaged deeper structures. These injuries require reconstruction using flap techniques, transferring well-vascularized tissue to the site of the defect. Depending on the nature and extent of the injury, local, distant, or free flaps may be employed (see chapter on Skin Flaps and Transplantation).

Local Flaps

Local flaps utilize tissue from the surrounding area of the defect. Examples include the V-Y advancement flap (for fingertip reconstruction, either unilaterally or bilaterally), the Moberg flap (used to move volar thumb skin to cover a thumb tip defect), rotation flaps, Limberg flaps, transposition flaps, and reverse forearm flaps (based on radial, ulnar, or posterior interosseous arteries). Additional examples are the Kite flap, based on the second dorsal metacarpal artery, and

others.

Distant Flaps

Distant flaps involve tissue from more remote areas of the body that remain connected to both the donor and defect sites for a period, typically around three weeks, after which surgical detachment is performed. Examples include cross-finger flaps, tarsal flaps, and inguinal flaps, among others.

Free Flaps

Free flaps are often employed in the reconstruction of severe or complex hand injuries, especially in cases of large or combined defects. These flaps not only restore skin coverage but can also help reconstruct functional units, such as through free transfer of a toe to the hand. These procedures are among the most intricate reconstructive techniques, often requiring several hours. The vascular pedicle of the flap is meticulously connected to recipient vessels near the defect through microsurgical techniques. Examples include the SCIP (superficial circumflex iliac perforator) flap from the groin area, the ALT (anterolateral thigh) flap nourished by perforators from the descending branch of the lateral circumflex femoral artery, and the fascia of the serratus anterior or temporalis muscle.

Loss of Functional Structures

Hand injuries may involve the loss of functional structures, such as bone, tendons, or nerves. When bone is lost, bone grafting or shortening may be performed. In cases of tendon loss, tendon transposition surgeries can replace the missing tendon with another, such as using the extensor indicis or extensor digiti minimi, or with a tendon graft, such as the palmaris longus or plantaris tendon. This may be done either at the time of injury or as a staged procedure following the insertion of a temporary silicone replacement. Similarly, missing blood vessels may be replaced by a venous graft from the nearby area or from the lower limb. If part of a nerve is lost, a nerve graft, often harvested from the sural nerve, may be used.

REPLANTATION SURGERY (INCLUDING TRANSPORT OF THE PATIENT AND AMPUTATED BODY PARTS) (49)

Dvořák Zdeněk, Berkeš Andrej

Introduction and definition of terms

Replantation and revascularization of amputated limbs is a standard procedure for restoring hand grip. These procedures are performed in specialised replantation centres and the following terms need to be defined for communication with them:

Amputee - separated acral part of a limb (separated fingers, palm, whole hand, forearm…)

Total amputation - the amputate and stump are completely separated and not connected by any tissue. The procedure to restore the integrity of the body is called replantation.

Subtotal amputation - the amputate and stump are conected by tissue without sufficient nutrition (bone, tendon, nerve, skin bridge up to 1/ circumference of the finger) and blood vessels are interrupted. The amputate is not perfused, replantation is needed to restore integrity.

Severe combined injuries with interruption of blood vessels - severe injuries of the limb with disruption of functional anatomical structures and interruption of major blood vessels, but with blood supply to the semi-amputated part of the limb by way of collaterals. The procedure to restore blood supply is called revascularisation.

Replantation - includes all procedures to restore the continuity, function and nutrition of the replanted part, i.e. osteosynthesis of bone, suturing of tendons or muscles, anastomosis of blood vessels, microsuturing of nerves and reconstruction of the skin covering.

Revascularisation - a procedure to improve blood supply to the periphery in injuries where vascular supply is significantly restricted but other functionally important structures are fully or largely preserved.

Warm ischaemia - is a condition in which the amputee is uncooled, left at body temperature. The duration of warm ischaemia (during which the amputate can still be replanted) is approximately 10 hours for fingers and 6 hours for amputates containing muscle tissue.

Cold ischaemia is a condition in which the amputee is separated from the body and cooled to 4°C. The duration of cold ischaemia should be less than 24 hours for fingers and 10 hours for an amputee containing muscle tissue.

Replantation surgery, indications and contraindications for surgery

The good status of the structures of the amputated tissue is important for the success of replantation. Another factor is the maintenance of a period of warm or cold ischemia of the amputate. The general condition of the patient is also important (extent of injury, other injuries, associated diseases -

atherosclerosis, diabetes mellitus, hypertension, vascular disease, etc.). It is necessary to take into account the age of the patient, his ability to undergo the procedure. Replantation of a single finger takes approximately 2 - 4 hours, replantation of the hand in the palm of the hand 6 - 10 hours, higher amputation 6 - 8 hours, with thrombosed anastomoses the procedure is prolonged and there is a higher risk of subsequent revisions.

The suitability of replantation is related to the mechanism of injury, as there are variations in the success rate of the procedure ranging from smooth cut, sawing off, circular sawing to crushing injuries. The highest percentage of replant failures occurs with the avulsion injury mechanism.

The indications for replantation are categorized into absolute and relative. Absolute indications for replantation include thumb amputation, multi-finger amputation, palmar amputation, wrist amputation, distal forearm amputation, and all amputations in children. Relative indications for replantation are high amputations, amputation of one finger or part of it, and isolated amputation of the index finger across the PIP joint. Contraindications to replantation are psychiatric disease, advanced age, heavy smoking, severe stage diabetes, or severe polytrauma.

Management of the treatment

Most of the injured are primarily treated by an ambulance with a doctor. After administering of first aid, the patient is transferred to the nearest surgical outpatient clinic, where the surgeon assesses the severity of the injury, the condition of the amputated parts and contacts the replantation centre. He or she then communicates the following facts to the replantation centre by telephone. Nowadays, it is also possible to send electronically photodocumentation of the case. After considering all the circumstances, the surgeon of the replantation centre will decide whether to transfer the patient for replantation. Replantation centers with 24/7 service are:

q Department of Plastic and Aesthetic Surgery, St. Anne's Hospital, Brno

q Department of Plastic Surgery, FN Královské Vinohrady, Prague

The Department of Plastic Surgery of the Ostrava University Hospital and the Traumatology Department of the České Budějovice Hospital offer replantation on a regular basis. Due to the small size of the Czech Republic, transport by ambulance is suitable both for surgical treatment and subsequently transfer to the nearest replantation centre. Air transport of the patient is indicated only if the patient is in severe state or if the nearest replantation centre is busy treating another patient and transport by ambulance would be too long.

Principles of amputation and stump treatment

A completely amputated parts should be treated by wrapping it in sterile gauze moistened with saline, then it should be placed in a bag, which is placed in a second bag with water and ice. The ratio of water to ice should be approximately 2:1, aiming for a cooling temperature of 4°C. The

stump of the limb should be treated with a sterile gauze cover and compression bandage (see Fig. 1). When treating the stump, do not interrupt any skin or tissue that maintain continuity in subtotal amputation, do not apply disinfectant to the wound area, ensure good haemostasis (compression dressing), avoid the use of a tourniquet if possible (time of application should be indicated if used). The tourniquet is used only in high amputations with continued arterial bleeding and the risk of hypovolemic shock. Also, immobilization of the injured part is performed and elevation of the limb during transport is ensured.

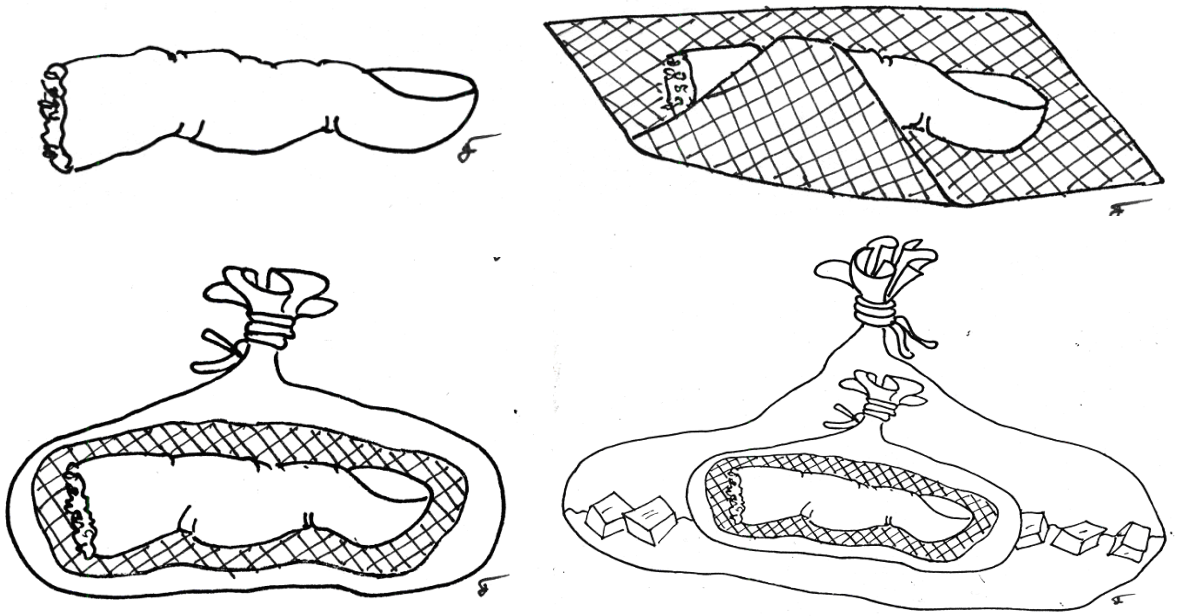


Fig. 1 - Correct treatment of amputees. The amputee is placed in gauze moistened with saline and then placed in a plastic bag. The bag containing the finger amputates is then placed in another bag with a 2:1 solution of water and ice.

Principles of replantation and specifics of amputation injuries

Most replantation procedures are performed under general anaesthesia; exceptionally, local or regional anaesthesia (axillary block) may be used. During replantation, the skeleton is first shortened and then osteosynthesized, most often using K-wires for fingers. Skeletal shortening is always necessary because of deficit of the soft tissue. A minimum 4-strand flexor tendon suture is performed to allow early rehabilitation. The volar neurovascular bundles are identified and a microsuture of usually one dominant (thicker) digital artery is performed. If the vessel is destroyed in a longer distance, then the damaged part of artery is replaced with a vein graft. The surgery continues with microsuturing of both digital nerves and suturing of the dorsal aponeurosis of the finger. To provide venous drainage, microsuturing of the veins on the dorsum of the finger is performed, most commonly at a ratio of 2 veins per supply artery. At the end of the operation, an skin cover suture is

performed and the blood supply to the replanted tissue is monitored. If the amputated part is containing muscle tissue, the time of ischemia must be respected. Rapid osteosynthesis of the skeleton is performed, then the procedure continues with reconstruction of the artery and suturing of the thick vein. This is followed by reconstruction of tendons, nerves and the remaining stuctures. Replantation procedure can be performed for all acral losses on the human body. Replantation of the lower limb, scalp, auricle, nose or even the penis could be performed.

Postoperative care and complications

Postoperative care after replantation is performed in specialized ICU, where, the blood supply to the replanted part is intensively monitored. The patient is heparinized and is using Acetylsalic acid 100 mg p.o. in one daily dose from the third postoperative day for 3 weeks after surgery. The blood supply to the replant is assessed by capillary reflow, color, temperature and skin turgor. The principle of assessing capillary reflow is to experss the blood from the capillaries by pressure and to monitor its re-entry, which is possible when the blood perfusion in the capillaries is sufficient. Its normal time is about 1 second. The prolongation of capillary reflow is observed in ischemia, while its shortening is observed in venostasis. Complications of replantation could be venous and arterial thrombosis, vasospasm, infection, bleeding or failure of replantation. Most of these states require urgent surgical revision.

The importance of replantations

Replantation is a standard reconstructive procedure for upper limb injuries. It contributes to the restoration of upper limb function by improving the gripping ability of the injured hand. These factors, together with a favourable appearance, contribute significantly to the psychological stabilisation of the patient and enable him to return to work.

PEDIATRIC SURGERY

CONGENITAL DIAPHRAGMATIC HERNIA, ABDOMINAL WALL DEFECTS IN CHILDREN (42)

Turek Jakub, Plánka Ladislav

Definition

A congenital diaphragmatic hernia (CDH) is a malformation in which the abdominal content herniates into the thoracic cavity due to incomplete development of the foetal diaphragm, leading to pulmonary hypoplasia with alteration of the development of pulmonary vascular structures. Bohdalek's hernia (about 70%) represents a herniation in the posterolateral part of the diaphragm (preferably on the left side), Morgagni's hernia arises based on a defect in the anteromedial part of the diaphragm.

Aetiology

The aetiology of the occurrence is probably multifactorial. It may occur as an isolated malformation or as part of chromosomal abnormalities (Turner syndrome, trisomy 13. and 18.), chromosomal aberrations (e.g. Fryns or Cornelia de Lange syndromes). Exposure to teratogens (lithium, allopurinol) during pregnancy may be associated with the development of this malformation.

Pathogenesis

The diaphragmatic hernia is caused by a defect in the continuity of the pleuroperitoneal membrane, which results in herniation of the abdominal contents into the thoracic cavity, compromising the normal development of the lung tissue and the pulmonary vessels, leading to thickening of vessel wall and resulting in persistent pulmonary hypertension.

Clinical manifestation

More than 50% of CDH is diagnosed during the prenatal screening between 18 and 24 weeks of gestation by ultrasound, which may be supplemented by foetal MRI if in doubt. Mothers may also present with low α-fetoprotein levels at 18th gestation or polyhydramnios.

Postnatally, CDH manifests as worsening respiratory insufficiency, cyanosis, disappearance of the auscultatory finding on the lungs on the affected side and dislocation of the cardiac echoes.

CDH is confirmed on a native chest X-ray, which shows intestinal villi in the thoracic cavity and mediastinal shift. In case of diagnostic confusion, CT or MRI may be added.

Course

Management depends on prenatal recognition of the malformation. If the diagnosis of CDH is confirmed at the screening examination, the option is to transfer the child to children surgery centres for foetal surgery - tracheal occlusion. After birth, the clinical manifestation is significant and as soon as the patient is stabilized, surgical management is considered. Patients are primarily transferred to centres where neonatal ECMO is available for postoperative care.

Complications

Long-term complications in patients include chronic lung disease, aspiration pneumonia, pulmonary hypertension, and obstructive lung disease. Gastrointestinal complications can include GERD or growth disturbances. Recurrence is another risk that can occur in months to years after surgery. Later, the orthopaedic problems such as chest deformities or scoliosis may occur after CDH repair.

Prognosis

Prognosis depends on many factors. Negative prognostic features include a low Apgar score, the presence of severe pulmonary hypertension, shock, and the need for ECMO.

Differential diagnosis

The differential diagnosis may include congenital cystic adenomatoid malformation, bronchopulmonary sequestration, bronchogenic cyst, bronchial atresia or teratomas.

Treatment

Surgical treatment is the only alternative for patients with CDH. Surgery is performed after control of pulmonary hypertension. The defect is approached from a subcostal incision, but recently minimally invasive - thoracoscopic approaches have come to the fore. For smaller defects, marginal suture is performed, while for larger defects, a patch must be implanted, both from artificial materials (Goretex) and biological materials.

Prevention

The only possible prevention is to avoid carcinogens in pregnancy and to undergo prenatal screening, which can detect most cases of CDH and thus time the delivery in perinatal centres.

Gastroschisis

Definition

This is a paraumbilical defect in the entire thickness of the abdominal wall with protrusion of the intestines, which are not covered by any membrane.

Aetiology

The exact aetiology is unknown. Gastroschisis is caused by a malformation of the ventral part of the abdominal wall. Risk factors for this diagnosis include smoking, environmental influences (exposure to certain substances such as nitrosamines), or the use of certain medications during pregnancy (aspirin, ibuprofen, decongestants).

Clinical manifestations

The clinical picture is typical - after delivery, a defect is observed in the abdominal wall area, usually paraumbilical to the right, the intestines are herniated extracorporeally, macerated and oozing, not covered by a membrane. In addition to the intestines, other organs of the abdominal cavity may be herniated. The picture of herniation is evident in prenatal screening.

Progress

After the diagnosis is made, the pregnant woman is monitored at the perinatology centre, delivery is planned based on the baby's condition, often the delivery is induced. Based on the study, with a standard course of pregnancy, there is no contraindication to vaginal delivery.

Prognosis

Compared to other abdominal wall defects, patients with gastroschisis have the best prognosis and more than 95% of patients survive.

Differential diagnosis

Gastroschisis must be differentiated from omphalocele. In the differential diagnosis, we must also think of umbilical hernia, bladder exstrophy or cloacal exstrophy.

Treatment

The therapy is the closure of the defect primarily or by means of a temporary cover - a silo, which gradually closes with the expansion of the volume of the abdominal cavity and the repositioning of the contents into the abdominal cavity.

Omphalocele

Definition

This is a defect of the abdominal wall in which prothorax of the intestinal villi from the base of the umbilical stump occurs and the herniated contents are covered by the amniotic membrane.

Aetiology

Unlike gastroschisis, omphalocele is associated with some genetic syndromes such as Beckwidt-Wiedemann or trisomy 13., 18. and 21. chromosomes. Omphalocele occurs when there is no rotation and repositioning of the physiologically herniated intestines after the 12^{th} week of gestation.

Clinical manifestation

It is the herniation of intestines (rarely by other organs of the abdominal cavity) through the umbilical stump, which are covered by the amniotic sac. This condition is recognizable in prenatal screening.

Prognosis

Depends on the size of the defect and the associated conditions within the syndromes.

Differential diagnosis

It is most important to differentiate omphalocele from gastroschisis. We must also think of other diagnoses such as umbilical hernias or umbilical cord anomalies.

Treatment

The therapy is similar to that of gastroschisis, i.e. closure of the defect primarily or in several steps with the help of a so-called silo.

GASTROINTESTINAL CONGENITAL DEFECTS (41)

Marek Ondřej, Plánka Ladislav

Definition of the term

Congenital malformations of the digestive tract include many separate diseases. Atresia and stenosis of the different parts of the GIT are most commonly encountered. These include atresia of the esophagus, duodenum and other parts of the small intestine. Another separate unit is anorectal malformations. Other congenital anomalies of the digestive tract include pylorostenosis, duplications of individual parts of the GIT. Malrotations - a failure of the physiological rotation of the small and large intestine, which includes nonrotation, compression of the duodenum, congenital volvulus of the small intestine, Ladd's syndrome and internal hernias are also included in this group. The last important congenital anomaly of the digestive tract is intestinal aganglionosis - Hirschsprung's disease.

Atresia of the oesophagus

Definition of the term

It is the most common congenital malformation of the esophagus. It is a failure of a differentiation during embryonic development. It occurs in approximately 1:5000 live births. We most commonly use the Vogt or Gross classification to classify this disease. We encounter 85% of esophageal atresia with lower tracheoesophageal fistula (Vogt IIIb). The second most common in about 10% is isolated esophageal atresia without fistula (Vogt II). Recently, prenatal diagnosis has advanced and most esophageal atresia is detected during prenatal ultrasound screening.

Associated congenital anomalies, including congenital heart defects, urogenital, skeletal and other gastrointestinal defects, may also be seen. When multiple anomalies occur together, we refer to VATER or VACTERL syndrome.

Clinical manifestation

Newborn infants do not tolerate the first dose of milk. Patients have increased salivation, coughing, tachypnea and desaturation can be observed. There is a risk of developing aspiration pneumonia in case of fistulas.

Prenatal screening reveals most esophageal atresia. After birth, the nasogastric probe encounters resistance or rolls back. In the most common atresia with lower tracheoesophageal fistula, a coiled probe in the oral esophageal stump and gas in the aboral parts of the GIT can be seen on plain chest and abdominal X-ray. In atresia without fistula, no gas is seen in other parts of the GIT. Only in case of diagnostic doubt, it is possible to use 0.5 ml of aqueous contrast fluid injected into the nasogastric probe and the X-ray reveals the diagnosis definitively.

Complications

The most common surgical complications include stricture at the anastomosis. And repeated endoscopic dilatations are necessary. We can also observe dehiscence of the anastomosis or recurrent tracheoesophageal fistula.

Prognosis

If it is an isolated anomaly, the prognosis depends on the type of atresia, the birth weight of the child and the distance between the esophageal stumps. With low birth weight and greater distance between stumps, the risk of anastomotic stricture and recurrent dilatation is greater. Patients are also more likely to experience dyskinetic movements of the esophagus and more likely to suffer from respiratory tract infections.

Differential diagnosis

Esophageal stenosis, esophageal diverticulum, esophageal duplication, laryngotracheoesophageal cleft.

Treatment

Treatment is exclusively surgical. In the most common type of atresia with lower tracheoesophageal fistula, an end-to-end anastomosis from the right-sided posterolateral thoracotomy or thoracoscopic approach is usually performed within 24-48 hours after birth. In long-distance atresia or without fistula, ligation of the tracheoesophageal fistula and creation of a nutritive gastrostomy is appropriate, and esophageal continuity is restored postponed. Most often, this involves replacement of the esophagus with the stomach. Increasingly, especially in long-gap or atresia without fistula, a thoracoscopic approach and gradual closure of both esophageal stumps using sliding knots is being used.

Atresia and stenosis of the duodenum

Definition of the term

A congenital disorder of patency in the duodenum that manifests early after birth as a sudden obstruction of the digestive tract. It is probably on the basis of incorrect vacuolization and recanalization of the GIT during embryonic development. Three types of duodenal atresia are encountered. Intraluminal mucosal membrane, two blind ends of the duodenum connected by a ligamentous strand or two blind ends of the duodenum that are not connected and there is a "V" shaped defect in the mesentery. Stenosis of the duodenum is most often caused by external compression.

Clinical manifestation

Vomiting in the first few hours after birth, most often with admixture of gall, because the obstruction is more often placed infrapapillary.

In later diagnosis and repeated vomiting, are observed signs of dehydration and hypochloremic alkalosis. The diagnosis begins by plain abdominal X-ray or laterogram. Two bubbles (double bubbles) are typical. Possible stenosis is differentiated by administration of a contrast fluid.

Complications

They occur in the form of dehiscence of the anastomosis or injury to the papilla. Duodenogastric reflux and other complications may manifest themselves later. Other complications may be associated with associated anomalies.

Differential diagnosis

Other types of atresia or stenosis in the GIT area, malrotation.

Treatment

Insertion of nasogastric probe and adjustment of the internal environment. Subsequently, reconstruction of GIT continuity, most commonly duodenoduodenal anastomosis from a transverse laparotomy in the right epigastrium. The membrane can be excised from the duodenotomy, but only if it is possible to locate the Vater's papilla quite safely.

Small intestine atresia

Definition of the term

Atresia of the small intestine occurs in 1:5000 newborns. Most often they result from intrauterine ischemia or after trauma (twisting, torsion...) and it is classified according to Grosfeld into types 1-4, see. Fig. 1.

Clinical manifestation

After birth, occurs persistent vomiting with a gall admixture and abdominal distention. In developed vomiting, signs of dehydration and hypochloremic alkalosis appear. A plain abdominal photograph shows widely dilated levels of small intestines.

Complications

Postoperative ileus, stricture or dehiscence of the anastomosis.

Prognosis

Generally good for isolated defects, for complex defects it depends on the most severe defect.

Differential diagnosis

Ladd's syndrome, Hirschsprung's disease, congenital volvulus of the small intestine, meconium ileus.

Treatment

Insertion of nasogastric probe, adjustment of internal environment. Reconstruction of continuity by intestinal anastomosis, usually with the necessity of modelling (tapering) of the oral and aboral part of the intestine.

Anorectal malformations

Definition of the term

It is a congenital disease of the rectum and anus resulting in the absence of an external anal opening and anomalous placement of the anorectum on the perineum or into the lower urinary or genital tract. The incidence is 1:5000 live births. Several classifications are encountered. Malformations can be divided into low, medium and high, according to the location of the atretic stump. In boys, perineal, rectourethral, rectoprostatic and rectovesical fistulae are distinguished; in girls, perineal, vestibular or vaginal fistulae.

Clinical manifestation

Depending on the type of malformation, stool may not pass after birth. A detailed clinical examination of the external genital area and perineum is necessary. In atresia where stool cannot be derived, we will add an X-ray and ultrasound examination to reveal the type of malformation. We can use MRI to assess the pelvic conditions.

Complications

Various types of incontinence, obstipation, wound dehiscence and ileus may occur with colostomy.

Prognosis

The incontinence depends on the type of anorectal malformation and associated defects.

Treatment

In atresia without the possibility of stool derivation, sigmoidostomy is necessary. Subsequently, around six months of age, the patient undergoes laparoscopically assisted or conventional posterosagittal anorectoplasty. Subsequently, a colostomy is closed after approximately 2 months. In patients with a fistula where stool derivation is possible, we can perform the reconstructive surgery directly, without a temporary colostomy, usually around three months of age.

Malrotation

A congenital abnormality of the physiological rotation of the small and large intestine, which may manifest as a sudden abdominal accident or ileus.

Nonrotation is the arrest of physiological rotation after the first phase. The entire small intestine is located in the right half of the abdominal cavity and the large intestine in the left half. This defect may not manifest clinically and may be an incidental finding during abdominal surgery.

Ladd's syndrome is a combination of congenital volvulus with compression of the duodenum by an underrotated cecum. It manifests clinically as an ileus. Treatment is surgical and often urgent because of possible ischemia of the small intestinal in the case of volvulus. Derotation of the bowel and dissection of the peritoneal strips leading from the cecum to the posterior abdominal wall in the

region of the liver and gallbladder are performed, and duodenal release is also performed. The duodenum is placed vertically, to the right of the spine, and the bowel are placed in a nonrotational position. The small intestine in the right half of the abdominal cavity and the large intestine on the left.

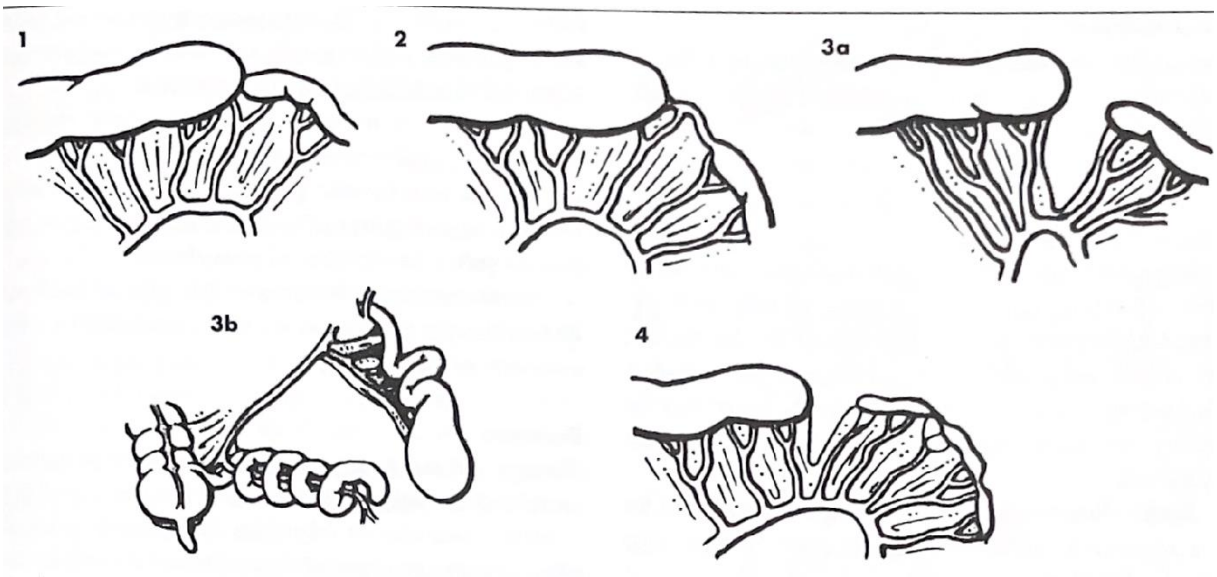


Fig. 1: Classification of small intestine atresia according to Grosfeld

ŠNAJDAUF, Jiří a ŠKÁBA, Richard. Dětská chirurgie. Praha: Galén, c2005. ISBN 80-7262-329-x.

HIRSCHSPRUNG'S DISEASE (44)

Turek Jakub, Tůma Jiří

Definition

Hirschsprung disease (HD) is a congenital intestinal aganglionosis in which ganglion cells of the myenteric Auerbach's and submucosal Meissner's plexus are absent. It always affects terminal rectum, i.e. from the linea dentata of the anal canal and extends proximally. The incidence of the disease is 1:5000 - 10,000 live births. Depending on the length of the affected segment of the intestine, several forms are distinguished: the ultra-short form (1-3 cm long aganglionic segment), the classical - rectosigmoideal form (rectum and distal part of the colon sigmoideum), the long form (up tp ½ colon transversum), aganglionosis of the whole colon or even the extensive aganglionosis of the NTBA type (nearly total bowel aganglionosis).

Aetiology

The aetiology is not fully understood. 70% of the disease occurs as an isolated disorder without any associated syndromes. Familial occurrence is 8-20% and in approximately 12% the disease is associated with chromosomal abnormalities, mainly trisomy of 21. chromosome. To date, several genes regulating neural tube and nerve cell development have been discovered, the most important of which is the RET proto-oncogene.

Pathogenesis

In HD, there is the failure of migration of neuroblasts from the neural crest or in the mis-differentiation of neuroblasts into ganglion cells leading to their accelerated apoptosis. The migration of neuroblasts occurs in a craniocaudal direction, which explains the different types of disease according to the length of the affected segment. The earlier the failure of migration occurs, the longer the segment of the intestine remains without intervention.

Clinical manifestation

The clinical picture is based on the nature of the disease, where the affected segment is permanently spastic, impossible to relax and pass stool. It acts as a functional barrier that can only be overcome by increased effort of the oral segment of the bowel, which gradually hypertrophies, and a megacolon can develop. The clinical picture depends on the age of the child and the length of the affected section. In the most common, the rectosigmoid form, the symptoms occur early after birth – late passage of meconium or the meconium does not leave in proper portions, the child is nauseous or vomits, the abdomen is markedly distended, and there are extensive peristaltic movement of bowel. Massive amounts of stool and gas are passed after insertion of a rectal tube or rectal thermometer. Eating disorders, chronic anaemia or intermittent vomiting are typical for older children.

Paraclinical findings

- Imaging methods

The primary imaging modality is a contrast study - irrigographic examination. The image of the classic form is typically a narrowed rectum funnelling into a dilated colon sigmoideum.

- Histological diagnosis

Biopsy may be performed as a full-thickness biopsy of the rectal wall, or biopsy of mucosa and submucosa as part of a colonoscopy examination, or by the method of suction biopsy. In the latter 2 cases, immunohistochemistry for AChE reaction (elevated levels in the absence of positive findings) or detection of calretinin (negative findings in the absence of an aganglionic segment) is used.

- Rectal manometry

This examination monitors the rectosphincteric inhibitory reflex, the presence of which rules out Hirschsprung's disease, whereas its absence does not confirm the diagnosis.

Course

In patients with suspected Hirschsprung's disease, surgical resection of the affected segment of bowel is the definitive method of treatment. On per rectum examination, gentle sphincter divulgence can temporarily improve symptoms but does not eliminate them. When patients are decompensated, they may even develop an ileus.

Complications

Complications are mainly related to enormous dilatation of the intestines, which can lead to ileus or HAEC (Hirschsprung's associated enterocolitis) with subsequent sepsis and shock. Other complications related to the surgical procedure include rectal stricture, dehiscence of the anastomosis, chronic constipation or, conversely, faecal incontinence in the long term follow up.

Prognosis

It is mainly determined by the length of the affected segment and the correct performance of the surgery. However, the long-term functional results are favourable and more than 50% of patients are completely free of difficulties.

Differential diagnosis

In the differential diagnosis, we must exclude other forms of neonatal intestinal obstruction, namely Ladd bands, meconium ileus, meconium plug syndrome, congenital intestinal hypoganglionosis and isolated intestinal neuronal dysplasia type B.

Therapy

Definitive therapy is surgery to remove the affected segment into the healthy tissue, remove the sphincteroachlasia of the internal sphincter by partial resection and suture the colon anastomosis. The most common techniques used are the techniques according to Swenson, Duhamel or Soave. The

surgery can be performed transrectally in certain circumstances, but often the bowel needs to be released in the abdominal cavity by laparotomy or laparoscopic approach.

Prevention

As this is a congenital disease, prevention of the disease is difficult to achieve.

PYLORIC STENOSIS, INTUSSUSCEPTION (43)

Marek Ondřej, Plánka Ladislav

Pylorostenosis

Definition of the term

Hypetrophy of the pyloric channel usually in newborns and infants most commonly between 4 and 6 weeks of age. It is the most common cause of vomiting in neonatal and early infancy.

Etiology and pathogenesis

The exact etiology is not yet clarified. One theory postulates that increased acidity of gastric juices leads to muscle spasm and thus muscle hypertrophy. A protective effect of breast milk is assumed. Pylorostenosis may be congenital or acquired. It occurs in about 1:5000 births and affects boys about four times more often. The muscle of the pylorus is thickened, edematous - the muscle fibers are hypertrophied.

Clinical manifestation

Milder vomiting may occur as early as around the 2nd week of life, but characteristic is bow vomiting of engorged milk without gall admixture between the 4th and 6th week of life. Vomiting occurs shortly after eating. Patients stop gaining weight and may show signs of dehydration. Sometimes a "pyloric tumor" can be palpated right below the rib arch.

In laboratory tests, hypochloremic alkalosis and hypokalemia are typical.

Of the imaging modalities, the first priority is the abdominal ultrasound, where the radiologist assesses the width and length of the muscle of the pyloric channel. We speak of mature pylorostenosis when the length of the pyloric canal muscle exceeds 17 mm and the width exceeds 4 mm. Another option is examination with a contrast fluid under X-rays.

Course

The patient typically develops recurrent bow vomiting between 4 and 6 weeks of life and stops gaining weight. On ultrasound examination, pylorostenosis is confirmed. The patient is admitted to the intensive care unit, the internal environment is modified with infusion therapy before surgery, and a nasogastric probe is inserted for recurrent vomiting. Surgical treatment is followed by laparotomy or laparoscopic pyloromyotomy. In 12 hours after surgery, if uncomplicated, gradual oral intake is started.

Complications

The most common complication of surgical treatment is injury to the pyloric mucosa, which is treated with a suture and pyloromyotomy at another location in the pylorus. Another complication

is inadequate pyloromyotomy, which manifests as continued vomiting with another surgery and a new pyloromyotomy out of the original scar.

Prognosis

The prognosis is good; recurrence or complications are rare.

Differential diagnosis

Overfeeding of the child, gastroesophageal reflux, or other obstructions in the aboral parts of the gastrointestinal tract.

Treatment

Definitive treatment is surgical. Before the procedure, it is necessary to stabilize the patient, infusion therapy, adjustment of the internal environment and, in case of repeated vomiting, insertion of a nasogastric probe. Surgical treatment is longitudinal pyloromyotomy of the pyloric hypertrophic muscle. The procedure can be performed by laparotomy or laparoscopically. With sufficient pyloromyotomy, the gastric mucosa is interposed between the muscle fibers along the entire length of the pylorus.

Prevention

Prevention of this disease is not yet known. A protective effect of breast milk is assumed.

Intussusception (Invagination)

Definition of the term

Intussusception is the telescopic insertion of the oral part of the intestine into the aboral part following the direction of peristalsis. It is a combination of mechanical ileus by invagination with strangulation of the vessels of the retracted mesentery. Most commonly encountered is ileocolic intussusception, less frequently with small or large bowel (ileoileal or colocolic) intussusception.

Etiology

Intussusception is typical in children under three years of age. The exact etiology is often unknown, so we speak of idiopathic intussusception. Among the causes of Intussusception we can include the intensification of peristalsis or the multiplication of mesenteric lymph nodes. In older children, the cause is the leading point - Meckel's diverticulum, polyp and others.

Pathogenesis

Retracted bowel - invaginate causes obstruction and retracted mesentery causes impaired blood outflow, venostasis and subsequent swelling of the bowel. In case of late diagnosis, ischemia and necrosis of the bowel may occur due to hypoperfusion.

Clinical manifestation

Intussusception is characterised by severe colicky pain at intervals. In between the pains, children are usually free of difficulties. With prolonged duration, vomiting also occurs, initially reflex, then more from a developed ileus. Another sign is "raspberry jelly" - hemorrhagic mucus with venostatic edema of the intestinal wall, which we detect on per rectum examination.

Laboratory tests may be unremarkable. When vomiting develops, hypochloremic alkalosis and hypokalemia are encountered. Of the imaging tests, the method of choice is abdominal ultrasound with a target image. On X-ray, we observe an ileus. Another option is irrigography, which is also a conservative treatment.

Course

Typically, the patient is between the sixth week to the second year of age with a picture of severe colicky pain. Ultrasound examination reveals ileocolic invagination. The patient undergoes hydrostatic desinvagination under general anaesthesia under ultrasound control. If it fails or if there are repeated episodes of recurrence, treatment is surgical.

Complications

With late diagnosis, ischemia and necrosis of the intestinal wall may occur. Invaginations may recur during a single hospitalization or with intervals.

Prognosis

The prognosis for idiopathic and early diagnosed invaginations is good, they may only recur usually within 10%. For invaginations with a leading point, the prognosis depends on the point itself. In late-diagnosed invaginations, ischemia and perforation of the intestine with all its consequences may occur.

Differential diagnosis

Enterocolitis.

Treatment

In stable children, hydrostatic desinvagination under general anesthesia and under ultrasound control is the treatment of choice. In children with a developed ileus, stabilization, insertion of a nasogastric probe, adjustment of the internal environment and infusion therapy are in place first. Hydrostatic desinvagination is retrograde filling of the colon with a contrast fluid up to 100 cm of water column. In addition to ultrasound, skiagraphy can be used for control. If hydrostatic desinvagination fails, surgery is indicated.

Prevention

There is no prevention in this disease.

NECROTIZING ENTEROKOLITIS IN CHILDREN (40)

Bibrová Štěpánka, Starý David

Definition: Necrotizing enterocolitis is one of the most common and serious abdominal episodes in children of neonatal age. It is a life-threatening disease, affecting 5-12% of preterm newborns with very low birth weight (below 1,500 g). It usually manifests in the 2nd -3rd week of life, worldwide it occurs in 0.3-2.4/1,000 live births, 70% of witch are babies born before 36 week gestation.
Aetiology: It is an inflammatory disease and at the same time an ischemic disability or necrosis of the intestinal wall cells which are caused by the penetration of bacteria into the wall of the affected part of the digestive tract. In the most severe cases even perforation of the intestinal wall, leakage of intestinal contents into the abdominal cavity and secondary peritonitis may occur. Especially in premature babies, immaturity of the digestive tract wall, including the immune system, plays a role. In term infants, it often occurs in the first days of life and a history of hypoxic episode is often given. The most commonly affected compartment is the terminal ileum and caecum but NEC can arise in any part of the gastrointestinal tract.
Pathogenesis: NEC arises on a multifactorial basis, when various noxes affected the immature gastrointestinal tract. Inadequate production of gastric acid and digestive enzymes prevents elimination of ingested pathogens and antigens, insufficient mucus production may lead to increased microbial adherence, and slower peristalsis makes it easier for bacteria to penetrate the intestinal wall.
The primary risks are low birth weight, low gestational age, artificial pulmonary ventilation and artificial nutrition (especially with high osmolality), chorioamnionitis. Other risk factors include intestinal immaturity, genetic predisposition, abnormal and delayed microbial colonization (e.g. after administration of broad-spectrum antibiotics, delayed enteral and artificial nutrition), abnormal changes in microvascularization during the transition to ex utero life (e.g. Perinatal asphyxia in non-progressive or premature labour). Maternal prenatal and perinatal risks include pre-eclampsia, premature amniotic fluid outflow, placental abruption, drug exposure (e.g. Indomethacin, corticosteroids), abuse (e.g. cocaine). Other risk factors include congenital and acquired abnormalities of the cardiopulmonary system (e.g. RDS, shock, hypotension, congenital hear disease, patent dictus arteriosus).
Clinical manifestation: Signs and symptoms of NEC are often nonspecific, variable and inconspicuous. Gastrointestinal symptoms include pain, abdominal distension, inappetence, transient food intolerance, delayed gastric emptying, nausea and vomiting, occult or even major bleeding in stools, change in stool pattern, diarrhoea. With the development of the disease, there is also a general

reaction of the organism, the development of sepsis and shock. Systemic symptoms include lethargy, respiratory failure, thermal instability, circulatory failure, cyanosis, unconsciousness and ultimately multiorgan failure.

Based on the clinical findings and imaging results, there are 3 grades of NEC (Bell´s criteria modified by Kliemann) – see the table below.

Physical examination of the abdomen shows abdominal distention, intestinal villi are visible through the skin, sometimes the abdominal wall is red coloured. Abdomen is painful on palpation, there may be palpable resistance. Bowel sounds may be weakened.

Laboratory tests tend to diagnose acidosis (metabolic or respiratory), hyperglycaemia, neutropenia, thrombocytopenia, also DIC or positive haemoculture may be detected.

Among imagine methods, X-ray and ultrasound examinations are used to diagnose NEC. The specific symptom on a plain abdominal radiograph is the finding of pneumatosis intestini (gas seen in the intestinal wall), a subileous or ileous state. Also a specific, adverse sign is the appearance of gas in the portal tract. Pneumoperitoneum is indicative of intestinal perforation. Gas in the portal tract can also be diagnosed by ultrasound examination.

Course: The course may be mild to peracute.

Therapy: Primary care is to ensure patient´s vital function, provide venous access, resuscitation therapy if necessary, fluid resuscitation in hypotension, endotracheal intubation and mechanical ventilation in respiratory failure.

When NEC is suspected and in mild forms, conservative therapy is indicated. In the first place, enteral nutrition must be discontinued, nasogastric tube should be inserted (to decompress the dilated bowel), broad-spectrum antibiotics are administered intravenously, usually in two or three combinations according to the patient´s general condition. Adequate paternal nutrition is essential. If conservative therapy is successful, enteral nutrition can be continued after resolution of infectious symptoms. In case of failure conservative therapy, deterioration of the intestine, surgical therapy is indicated. The aim of laparotomy is revision of the abdominal cavity, resection of the perforated or clearly necrotic part of the affected bowel and stitching of the stoma. Primary anastomosis is not recommended because of the high risks of ischemia at the anastomosis site and subsequent complications. When resecting the bowel, we try to take the gentlest approach and preserve as much of the bowel as possible.

In critically ill, unstable patients with pancolitis, laparotomy may be contraindicated. In these patients with proven bowel perforation or pneumoperitoneum, only peritoneal drainage is recommended, followed by laparotomy after stabilization of the condition.

Complications: Surgical therapy and the need to remove large parts of the affected bowel can lead to short bowel syndrome. Resections can lead to gastrointestinal dysmotility, bacterial overgrowth, digestive enzyme deficiency, gastric acid hypersecretion, overgrowth of intestinal bacteria and their translocation into the bloodstream, which can cause sepsis. Patients with NEC are at high risk of failure to thrive, fistulas, strictures and adhesions. In addition to gastrointestinal difficulties, there is a risk of delayed psychomotor development and cognitive changes are observed.
Differential diagnosis: If the differential diagnosis i tis necessary to exclude other congenital developmental defects of the digestive tract (e.g. malrotation, intestinal volvulus, atresia). In raticular, gastroenteritis, meningitis, urinary tract infection or sepsis of other aetiology should be excluded from infectious causes.
Prognosis: A 10-50% mortality rate is generally reported, which depends on the timeliness of diagnosis and therapy of NEC. In fulminant course the prognosis is very negative.
Prevention: Multidisciplinary collaboration is important for early diagnosis and treatment of NEC to minimalize complications. Early administration of colostrum and natura breast milk, which contain high levels of immune mediators that contribute to bacterial and anti-inflammatory protection of the gut wall, is a preventive factor for NEC. The growth factors contained in colostrum stimulate the development of the digestive tract. The administration of probiotics also helps to regulate the pro.inflammatory response.

Classification of NEC (Bell´s criteria modif. Kliemann)				
Grade		**General signs**	**Abdominal signs**	**X-ray signs**
I. grade – Suspicious NEC	**A**	temperature instability, lethargy, bradycardia, apnoe	gastric residue, vomiting, abdominal distension, occulte bleeding	normal findings or mild dilatation of intestinal villi, ileus
	B	as IA	IA + macroscopic bleeding in stool	as IA
II. grade - Cinfirmed NEC	**A**	as IA	inaudible intestinal peristalsis ± palpation sensitivity	as IA
	B	as IIA + trombocythopenia and mild metabolic acidosis	clear palpatory sensitivity ± redness, abdominal skin phlegmona ± palpable resistence in the right lower quadrant	gas in v. portae ± ascites
III. grade - Developed NEC	**A**	respiratory failure, hypotension, bradycardia, combinated respratory and metabolic acidosis, DIC, neutropenia	diffuse peritonitis, abdominal distension, marked panifulness	clear ascites
	B	as IIIA	as IIIA	pneumopertoneum

Table 1 – Clasiffication of NEC

UNDESCENDED TESTICLES, PHIMOSIS, PARAPHIMOSIS (38)

Bibrová Štěpánka

Undescended testicle

Definition: Undescended, retined testicle is one of the most common congenital defects of the male external genitalia and crytorchism. I tis divided into abdominal and inquinal retention according to the level of fit, and may occur unilaterally or bilaterally. I tis more often diagnosed in premature infrants or is part of another disability (e.g. Klinefelter´s syndome).

Etiology and pathogenesis: Embryonic gonadal development begins with the indiferent stage, followed by the stage of differentiation of the base into the testis or ovary. The initially high-based gonád descends into the genital mounds. At the beginning of the 4th intrauterine month, i tis located at the entrance to the canalis inquinalis, where it remains until the 7th month and i tis usually abbreviated at the end of the 8th to 9th month of intrauterine development. Mechanical influences, hormonal action of androgens anf progressive histological changes in the testis gubernaculum are involved in the descent of the testis. Due to a transient increase in hormonal levels after birth (usually by the 3rd month), testicular descent may continue after birth.

Clinical manifestation and course: Diagnosis is based on anamnestic data and careful physical examination by palpation. The patient is examined supine and in the Turkish sitting position (a decrease in the cremaster reflex can distinguish a retractile testis from a non-descended testis). Sometimes other sings of insufficient masculanisation may by present (e.g. hypospadias, micropenis). Among paraclinical methods, ultrasound examination can sometimes help in diagnosis. In the case of abdominal retention, laparoskopy is also used for dagnosis. The presence of functional Leydig cells can be demonstrated by the hCG stimulation test in the presence og bilateral nonpalpable testis.

Therapy: Treatment is mainly surgical – orchidopexy, fixation of the testicle in the scrotum. The goal is to correctly position the testicle in the scrotum until the child is 1-1,5 years old. The inquinal appoach is used for the palpable testicle during the inquinal canal, for the nonpalpable testicle a diagnostic laparoskopy is performed. Abdominal retention is usually treated with a two-stage operation. In the first time, the internal testicular vessels are interrupted laparoscopically, the testi sis inserted into the internal anulum of the inquinal canal and in the second time (usually after about 2 months) an orchidopexy is performed. In case of finding of hypotrophic or atrophied testis, orchiectomy is indicated. Hormone therapy, fromerly widely used, is nowadays hardly used anymore, but may have a positive effect on future fertility.

Complications: After surgical therapy, testicular atrophy, torsion of the spermatic funikulus (e.g. scarring, insufficient relaxation) may occur, and fertility may be impaired if the fallopian tube is injuried. In the case of untreated crytorchidism, there is an incerased risk of developing a mailgnant testucular tumor, a seminoma (especially after puberty).
Prognosis: In case of orchidopexy, the risk of malignancy of testicular tissue is reduced. In case of orchiectomies, testicular protheses can be implanted into the scrotum after puberty.
Differential diagnosis: Differentiation from other causes of cryptorchidism – ectopic testis, anorchia (testicular atrophy or agenesis), acquired cryptorchidism after inquinal surgery (e.g. hernioplasty, hydrocele surgery). Furthermore, i tis necessary to distinguish a retractable (bouncing) testis (by the action of increased cremaster reflex, a transient withdrawal of the testis into the inquinal canal). Bilateral retention may be part of a metabolic disease (e.g. congenital hypopituirtarism, CAH).
Prevention: Early detection and therapy by careful clinical examination of newborns and infants as part of preventive pediatric checkups.

Phimosis

Definition: Inability to pull the foreskin of the penis over the glans penis. It is a disproportion between the circumference of the foreskin of the penis and the glans.
Etiology: 2 form occur – primary, naurally in most newborn boys, and secondary due to scar tissue formation (e.g. in systemic disease, postinflammatory, forced pulling).
Pathogenesis: At birth, the foreskin is assiciated with the glans penis, encircling it. By multifactorial action (gradual detachment of the epithelial layers of the inner leaf and glans, formation of the smegma, nocturnal erection), the adhesions gradually relax spontaneously and the circumference of the foreskin expands. Secondary phimosis is induced by the pathological action of inflammatory processes, microtrauma and trauma to the fereskin and external genitalia.
Clinical manifestation: A careful, gentle physical examination of the patient is important. We detect the presence of coglutination, between the inner leaf of the feroeskin and the glans penis, elasticity of the foreskin orifice and formation of ragades. Durin urination, transient expansion of the foreskin sac may occur. In secondary phimosis, we observe rigid collagenous ligaments that prevent overstreting of the foreskin. A history of recurrent balanoposthitis may be found.
Therapy: In asymptomatic cases, observation only and natural development in monitored. In symptomatic patients (e.g. pain, recurrent balanoposthitis), treatment – conservative or surgical – is necessary. The aim of the conservative pharmacological therapy is to achieve relaxation of the constricted part of the foreskin, corticosteroids are applied locally for 6-8 weeks. Conglutination of the foreskin is mechanically released on an outpatient department, under local anaesthesia.

Surgery is performed by partial resection of the foreskin (removal of only the stenotic part of the foreskin and its plastics) or complete circumscission. Cpmplete circumscission is contraindicated in conditions where the foreskin can be used for reconstructive procedures (e.g. disorders of sex development, hypospadias, epispadias, recessed penis).

Complications: The most common complcation of phimosis is recurrent balanitis, difficulty urinating. Infectious complcations may occur after cirrcumcision, scarring with poor cosmetic outcome, skin bridges, urethral stenosis. More serious postoperative complications include hemorrhage, injury to the glans, necrosis of the penis, and formation of ureterocutaneous fistulas.

Prognosis: Depends on the duration, development of ischemic changes. In most cases, i tis good. In caase of repeated failure of conservative therapy or rcurence of phimosis within 2-6 months after its completion, surgicle treatment is the method of choice. Complete circumcision is necessary in balanitis xerotica obliterans.

Differential diagnosis: We must distinguish paraphimosis, hypospadias, epispadias, balanitis from phimosis.

Prevention: Observance of meticulous hygiene, non-voilent retraction, early detection and therapy of balanitis.

Paraphimosis

Definition: This is an acute affection of the penis, the foreskin is overstretched beyond the edge of the glans penis and cannot be repaired back.

Etiology: Paraphimosis can occur after prolonged overstretching of the foreskin (especially at the tighter orifices), when irritation of the foreskin (e.g. inflammation, intercourse, allergic reaction) causes swlling and repositioning cannot be performed.

Pathogenesis: The overstretched outh of the foreskin behind the edge of the glans acts as a stragulating ring, restricting lymphatic and venous drainage from the foreskin and glans penis. The condition may progress to massive swelling, ischemia of the affected tissues (including the gland and glandular urethra) and development of necrosis of the foreskin and penile skin.

Clinical manifestation and course: Massive, circular swelling of the foreskin, in stages, venostasis, discoloration of the penile foreskin and gland and superficial skin defects may occur.

Therapy: In paraphimosis, manual compression of the foreskin and glans penis and repositioning of the foreskin back as early as possible is neccessary. In case of impossibility of manual reposition, early surgical therapy is performed – dorsal dissection of the strangilating ring of the foreskin followed by complete circumscission at a second time.

Complications: postischemic changes of the glans penis with the appearance of deformities, necrosis of the penile skin, urethral stricrures.

Prognosis: Mostly good.

Differential diagnosis: It is necessary to distinguish balanitis, phimosis in particular.

PEDIATRIC UROLOGY (45)

Husár Matej

Almost 30% of all birth defects in boys are urological diagnoses. Some diseases can be detected prenatally by ultrasound, such as the more severe obstructive and reflux nephropathies, megavesica and bladder exstrophy. Postnatally, external genital examination is performed as a part of neonatal screening to detect congenital adrenal hyperplasia (CAH) in children, and testicular descensus in boys and exclusion of hypospadias. If CAH is found, or bilateral non-palpable testes with hypospadias, endocrinological and genetic testing should be added. Endocrinological examination in boys is best to be performed during the minipuberty period (around the 2nd-3rd month after birth) due to activation of the hypothalamic-pituitary-gonadal pathways. For more details on congenital defects in terms of obstructive uropathy and cryptorchidism, see the next questions.

Infancy and toddlerhood

In this age, febrile urinary tract inflammations of the character of pyelonephritis are quite common. According to the guidelines, it is currently recommended to perform micturating cystourethrography to diagnose vesicoureteral reflux (VUR) after the first attack. A child with VUR should be provided with a prophylactic dose of ATB to prevent another attack of pyelonephritis. Surgical management of VUR is performed at an older age, taking into account the degree of VUR, recurrence of inflammation and the clinical condition of the child. It is possible to perform minimally invasive submucosal antireflux injection into the ureteral orifice or to perform ureteral reimplantation. Severe obstructive uropathy resulting in hydronephrosis or megaureters is solved by acute urinary diversion - puncture nephrostomy or puncture epicystostomy, and the actual surgical solution is indicated after stabilization of the patient and according to the evolution of the severity of the obstruction.
In infancy and toddlerhood, correction of cleft defects of the external genitalia is performed, and at the end of the first year of life, cryptorchidism begins to be solved.
At this age, we must think about metabolic defects that can result in the formation of urolithiasis. The most common metabolic defect with concrement formation is cystinuria.

Preschool and school age

The most critical period for the development of Wilms tumour (nephroblastoma of the kidney) is the 4th-10th year of life. Most often a palpable bulky mass extruding into the abdominal wall is found. Current oncologic treatment protocol is: neoadjuvant chemotherapy, nephrectomy, followed by chemotherapy/radiotherapy. In addition to renal tumours, testicular and paratesticular tumours

(sarcomas) and bladder tumours are also found. For testicular and paratesticular tumors, a safe high orchiectomy from the inguinal approach is performed. For bladder tumours, a biopsy is performed first to typify the tumour and then surgical intervention.

Urinary tract infections in pre-school and school age are mainly suffered by girls, they are often associated with inadequate or improper hygiene, lower urinary tract dysfunction (most often overactive bladder) and also due to urethral stenosis or persistent synechiae of labia minora. Also, one already needs to think about sexually transmitted infections.

In boys, phimosis, whether physiological or due to inflammation of the foreskin or due to lichen planus (balanitis xerotica obliterans), is often addressed.

After the 5th year of life, bedwetting begins to be addressed in children. Clean bedwetting, without daytime micturition problems, can be significantly improved by regimen measures - no drinking in the evening, proper urination, or waking up at night and urinating. If there is no effect, it can be helped by putting on an adiuretine hormone.

At the age when children are already beginning to develop sports activities, trauma occurs. The most common are blunt impacts to the lumbar region with injury to the kidneys, or injuries to the external genital area associated with urethral injury. Most renal trauma is treated conservatively (strict bed rest, haemodynamic therapy, antibiotics), only when urinary extravasation and haemodynamically unstable bleeding could require surgical intervention. Urethral injuries are also treated conservatively, only in case of complete urethral rupture urethroplasty is needed or late complications in terms of post-traumatic ruptures need to be solved.

A final note for this age is urolithiasis, which is most often associated with low diuresis on the basis of low fluid intake (oxalate stones). Exceptionally, there is a metabolic basis for lithiasis or the formation of stones due to repeated infections (struvite stones).

PEDIATRIC TUMORS (39)

Doušek Robert, Tůma Jiří

Childhood tumours are a very heterogeneous group of diseases. Children with cancer show significant differences from adult patients - they have different tumour types with different biological behaviour, they are treated differently, they respond differently to treatment, they process experiences differently psychologically. The most common childhood tumours are of haematogenous origin, followed by tumours of central nervous origin, neuroblastoma, sarcomas, nephroblastoma and germ cell tumours.

The general paediatric surgeon treats paediatric patients mainly with tumours of the thoracic and abdominal cavities and some limb tumours. Patients with tumours in other locations are operated on by a neurosurgeon, orthopaedic surgeon, otorhinolaryngologist, urologist, gynaecologist and others.

Approximately one hundred years ago, surgery was the only treatment option for pediatric tumors; today, surgery is part of multimodal cancer therapy. This text deals mainly with the surgical aspects of the care of pediatric cancer patients.

The surgeon (as well as the physician of other specialties) can play an important role in the early detection of childhood cancer. The tumor typically manifests nonspecifically, either by a solitary symptom or a combination of symptoms. The most common symptoms include tactile resistance, pain, stagnation or weight loss, behavioural changes, fatigue, dry cough, changes in movement patterns, vomiting, and defecation disorders.

The clue to suspecting a correct diagnosis should be a correct reflection of the observation that symptoms attributed to a more trivial disease do not disappear after the usual period of treatment or recur.

Various imaging and laboratory tests are used in the diagnostic process. Key investigations (histology, molecular biology) leading to an accurate diagnosis usually require a sample of tumour tissue. Properly performed biopsy has its own rules - selection of the collection site with the least traumatization of the patient, sufficient sample size - typically 1 cm^3, collection of vital tumor tissue instead of necrosis, gentle handling of the sample to prevent its destruction, prevention of contamination of surrounding tissues, careful hemostasis and suture, delivery of the sample without fixation, correct labeling and, if necessary, orientation of the sample. In some situations, it may be appropriate to request a frozen section biopsy.

At important junctions in the cancer patient's journey, a panel of experts from different specialties (the oncology committee) meets, evaluates what has been learned so far and proposes a course of

action. The surgeon has the opportunity to request further necessary examinations, specify the risk level of the considered surgical solution, etc.

The main benefit of the oncology committee (MDT) for the paediatric patient is the multidisciplinary discussion with the search for consensus on the most appropriate treatment approach.

A common difficult decision for cancer patients is whether it is better to perform a biopsy or a definitive resection procedure. Generally, in smaller tumours, definitive resection is the first choice, whereas in advanced disease, non-surgical oncological treatment follows biopsy, and definitive surgical treatment is usually followed by oncological therapy once the tumour has reduced in size.

The actual surgical procedure with therapeutic intent has common general patterns, but in some aspects the principles differ significantly depending on the type of tumour. The general principles include the choice of the optimal surgical approach, early ligation of supply and drainage vessels, sufficient radicality with the least invasiveness, prevention of injury and contamination of surrounding organs, careful haemostasis, and correct transfer of the tumour for further examination.

The differences according to the type of tumour can be illustrated by the example of soft tissue sarcomas and neuroblastoma (the basic explanation for the different surgical approaches is the different sensitivity of tumours to non-surgical treatment).

For patients with sarcoma, a sufficiently radical removal of the pathological lesion en bloc with a rim of healthy tissue (R0 resection) is a vital principle. If part of the tumor tissue remains in the patient's body (microscopic (R1) or macroscopic (R2) residue), the risk of difficult-to-treat local recurrence increases substantially. After insufficiently radical surgery, we consider so-called primary radical reexcision. Furthermore, due to the biological nature of sarcomas, we are actively looking for lymphatic metastases.

On the other hand, in neuroblastoma we do not necessarily need to achieve maximum radicality; 95% resection is a frequent goal, with deliberate retention of small remnants of tumor tissue on vital structures. The emphasis is on preserving the integrity and function of the surrounding organs. It is not necessary to remove the tumour en bloc; resection is facilitated by the piecemeal technique, whereby removal of several accessible portions of the tumour allows the originally inaccessible part of the lesion to be safely resected. The main pitfall of neuroblastoma tends to be its intimate relationship to major blood vessels (aorta and inferior vena cava and their branches).

A special subchapter is represented by young patients who are born with the tumour. The treatment of these patients combines the principles of oncological and neonatal surgery (extremely gentle tissue manipulation, emphasis on minimal blood loss, special instruments, optimal timing of surgery). The most common neonatal tumour is sacrococcygeal teratoma.

The general hierarchy of surgical treatment goals (saving life takes precedence over saving health, and health takes precedence over cosmetic outcome) is also valid for paediatric cancer patients, yet only rarely are mutilation operations indicated (resulting in ablation or loss of function of an important organ), as a similar therapeutic effect can usually be achieved by a combination of less invasive surgery and non-surgical treatment.

In the 1990s, the indications of the newly developed minimally invasive surgery (e.g. thoracoscopy, laparoscopy) gradually expanded. Initially, the presence of malignant disease was taken as a contraindication to the minimally invasive approach; nowadays, minimally invasive surgery has a firm place in the treatment of paediatric patients with cancer.

Surgical intervention is necessary to ensure quality vascular access for the administration of cancer therapy. Most commonly, this involves the insertion of a central venous catheter, a tunneled central catheter or a venous port.

The surgeon also encounters pediatric patients treated in palliative care. Typical procedures include drainage of thoracic effusion or ascites, bypass surgery, construction of an ostomy for nutrition or, conversely, for derivation of intestinal contents, and arrest of bleeding.

Pediatric tumors carry the risk of many complications and acute conditions. We mention some requiring surgical intervention. The tumor may make itself known by rupture, violent hemorrhage, ileus, urinary retention, suppurative inflammation, torsion of an important organ, compression of the spinal cord. Serious complications may arise from the resection surgery itself. Complications of chemotherapy include, among others, hemorrhagic cystitis, acute pancreatitis, neutropenic colitis with the necessity of resection of the necrotic section of the intestine, invasive aspergillosis requiring radical surgical removal of all foci persisting after antifungal therapy.

The overall survival prognosis is highly variable, generally more favourable in a paediatric cancer patient than in an adult cancer patient. The variability in prognosis is illustrated by neuroblastoma tumors with two extremes: patients with an unfavorable biological profile of small localized neuroblastoma may have a poor prognosis, whereas other patients with neuroblastoma will be cured spontaneously without any therapeutic intervention. Along with efforts to further reduce mortality, there has been a recent emphasis on substantially reducing treatment morbidity, as surviving pediatric patients carry more long-term sequelae into their lives than their adult counterparts.

In the future, pediatric oncologic surgery can be expected to intensify its efforts to provide maximally effective surgical treatment with the least invasiveness and least morbidity. This goal could be achieved, for example, by refinement of navigation techniques including 3D imaging, further development of minimally invasive and robotic surgery, prophylaxis of later fertility

disorders and the use of precision medicine (based on molecular-biological specification of the disease) in direct combination with surgery.

Despite all the technical advances, the therapeutic relationship is based on fundamental medical principles, treating a unique human being at a fragile age.

PEDIATRIC FRACTURES (37)

Mareček Lukáš, Plánka Ladislav

Definition and introduction

The composition of a pediatric skeleton varies from an adult skeleton especially by a large content of cartilaginous tissue and the presence of growth cartilages, which ensure the bone growth. Bones are also more elastic in childhood. For these reasons, different types of fractures occur in childhood and require a specific therapeutic approach.

Specific types of child age fractures:

a) Subperiosteal fractures (Torus/Buckle fracture) - an incomplete fracture with cortical injury on one side of the bone,
b) Bowing fractures - no obvious fracture line, the angulation of a bone is greater than physiologically,
c) Greenstick fractures - one side of the cortical bone is broken, the other side is only angulated,
d) Epiphyseal separation and fractures - injuries in the region of the growth cartilage, according to Salter and Harris they are classified into five types,
e) Avulsion fractures - when a muscle or ligament is overpulled, a bone fragment is displaced instead of soft tissue being injured.

Fractures typically occur in children when they fall or by an object impaction, or during pathological movement, similarly to adult patients. Pathological fractures (e.g. in the field of a bone cyst) or stress fractures (from overload) are exceptions. Atypical fractures result from an intentional violence.

Diagnostics

Diagnosing fractures in children is similar to adult patients. However, we emphasize on physical examination and low radiation dose in imaging methods.

Medical history - we ask about the problem's development, it's circumstances and changes over the time.

Physical examination - we look for certain signs (obvious bone fragment, pathological mobility, change in shape) or uncertain signs of fracture (swelling, hematoma, palpation or tenderness).

X-ray examination - basic imaging examination method. The great advantage is a low dose of ionizing radiation, however obtaining only 2D images of bones and the absence of cartilaginous tissue imaging is the disadvantage.

Ultrasound examination – frequently it is the first diagnostic method in epiphyseal region imaging in a case of growing skeleton.

Other imaging methods - in case of complicated fractures, uncertain X-ray findings or in case of polytrauma, CT scan is the method of choice. MRI has its place in investigating pathologies and complications of pediatric fractures, such as bone bridge formation or fractures of the neonatal skeleton.

Pediatric fractures therapy

In the treatment of pediatric fractures, it is necessary to take an advantage of the propitious healing potential and remodelling abilities of the pediatric skeleton. Most of the fractures in the child age is treated conservatively. If surgery is necessary, it must be performed in a such way to minimize injury in the area of the active growth plate by the osteosynthetic material.

Conservative approach

The fixation time for healing pediatric fractures depends on the patient's age and the localization of the injury. The healing rate and required fixation time increases with age, with the distance of the fracture site from the growth cartilage and with a reduced vascular supply to the injured area.

Location	Standard fixation time
Metaphysis / Epiphysis	2,5–4 weeks
Diaphysis	5–7 weeks
Scaphoid bone / Talus	6–9 weeks

The fixation can be made of different materials. However, at the beginning of the treatment, a non-circular, ideally plaster splint is required because of the swelling and the risk of compartment syndrome development. Once the swelling has subsided, fixation with lighter materials can be used for fractures without risk of dislocation. A preformed splint can be used for uncomplicated finger injuries. Some traction techniques may be applied in the treatment of diaphyseal fractures of long bones in small patients (up to 15 kg).

Surgical approach

Unstable and potentially unstable fractures need to be fixed with osteosynthetic materials. If possible, the operation is perfomed from a small skin incision or percutaneously. A drilling thin Kirschner wire is the most frequently used material in the treatment of epiphyseal fractures. The ESIN method is used in the treatment of diaphyseal fractures in long bones (usually a Prévot nail is implanted into the intramedullary cavity of long bones). Another possible methods include cancellous, usually cannulated screws, or rigid intramedullary nails (if the growth plate is closed), and rarely applied are plating systems or external fixators.

Typical fractures of children age:

Birth injuries - during difficult fetus delivery, a fracture of the clavicle or the humerus diaphysis may occur. These fractures are treated by fixing the limb to the body for 2-3 weeks. **Supracondylar fracture of the humerus** - the most common fracture in the elbow region in children. A displaced fracture requires precise repositioning and osteosynthesis under general anaesthesia. This type of fracture is dangerous by a neurovascular injury and a risk of healing in residual dislocation (especially rotational), which may lead to permanent consequences (limitation of full mobility in the elbow joint). **Monteggia lesion** - fracture of the ulnar bone with injury to the humeroradial joint. Usually, a fracture of the ulnar bone is associated with luxation or subluxation of the radial head. **Avulsion fracture of SIAS/SIAI** - fracture typical for adolescent patients. Hypercontraction of the m. quadriceps femoris occurs during rapid acceleration or braking and its onset tears a bone fragment from the hip bone. **Impressive skull fracture (ping-pong fracture)** - a fracture of the neurocranium typically occurs when an infant or toddler falls from a greater height on an obstacle by the top of the head. It requires a head CT scan to rule out other intracranial injuries. Depending on the degree of impingement, operative therapy is indicated.

Complications of pediatric fractures

Acute complications include **injury to blood vessels, nerves and soft tissues** (partial or complete laceration by the mechanism of injury, injury caused by bone fragments or osteosynthetic material). Late complications include **bone bridge**, which occurs when the injured part of the growth plate undergoes necrosis. During healing, a conjunction of the epiphysis and metaphysis occurs and the growth stops in the injured region. Depending on its location, the bone bridge then causes impaired bone growth in length, angulation, or a combination of both, and thus shortening or curvature of one of the limbs. **A non-union** is the name given to a fracture that does not heal in six months or more and requires surgical treatment. Axial deformity, or **malunion**, is the result of a fracture healing in an inconvenient position in which the skeletal remodelling capabilities are insufficient. **Bone synostosis** is caused by transverse ossification of the long bones of the forearm or lower leg and it is caused by hematoma ossification connecting the bones with an injured periosteum.

Generally it can be said it is more difficult to diagnose and classify the children fractures than to operate them. The major mastery consists in the art of conservative treatment methods usage and especially some specific types of fractures should be treated in the pediatric skeletal traumatology centers.

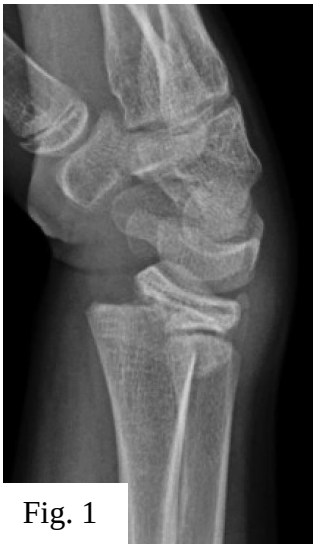


Fig. 1

Epiphyseal separation of distal radius SH II

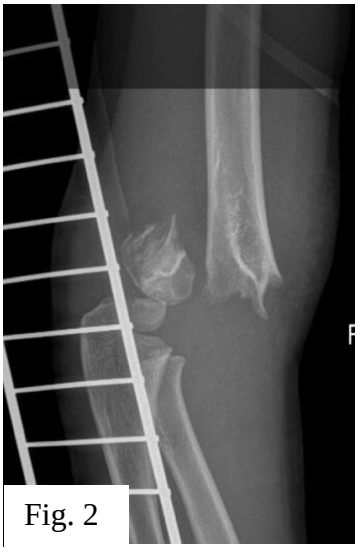


Fig. 2

Distal humerus supracondylar fracture

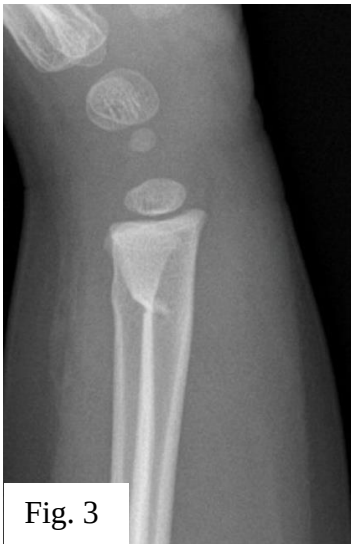


Fig. 3

Distal forearm subperiostal fracture

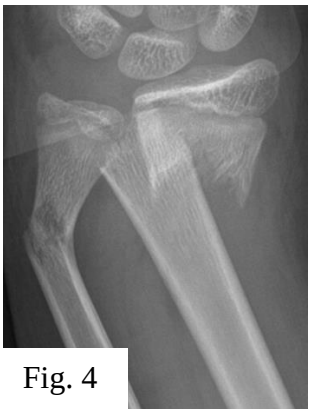


Fig. 4

Distal ulnar greenstick fracture with complete metaphyseal distal radius fracture

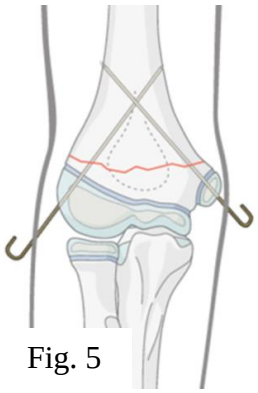


Fig. 5

Supracondylar facture fixation with Kirschner wires

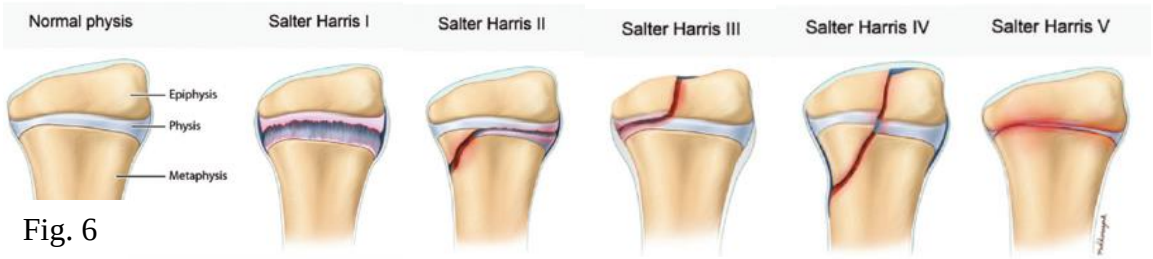


Fig. 6

Image references:

(Figures no. 1 – 4) - Archive of Department of Radiology and Nuclear Medicine, The University Hospital Brno

(Figure no. 5) - AO Surgery Reference website (https://media.aofoundation.org/-/jssmedia/surgery/p13/p13_p010_i1200.png?w=400)

(Figure no. 6) - Little JT, Klionsky NB, Chaturvedi A, Soral A, Chaturvedi A. Pediatric distal forearm and wrist injury: an imaging review. Radiographics. 2014 Mar-Apr;34(2):472-90. doi: 10.1148/rg.342135073. PMID: 24617692.