Craniocerebral and spinal trauma

Craniocerebral trauma

Craniocerebral trauma (CT) is the most serious group of all injuries (e.g. in the USA 2 million people suffer brain injuries every year, 20 % of injuries are fatal and the cost of treatment reaches 25 billion dollars per year)

Classification of craniocerebral trauma

CT can be divided into groups according to the mechanism of injury to **direct' *(*closed** or **penetrating**) and **indirect**, according to the time of onset into **acute** and **chronic**, and according to the severity of the clinical condition into **mild**, **moderate** and **severe** disability (*Tab. 1.*, *Fig. 1.*).

The **direct acute injuries'** include skull fractures, brain envelope injuries, cranial nerve injuries, vascular injuries, and especially coma, concussion, laceration, and diffuse axonal injury.

In association with these, but also independently, **indirect acute injuries** may occur, including acute epidural haematoma, acute subdural haematoma, traumatic subarachnoid haemorrhage, intracerebral haematoma, swelling, brain oedema, pneumocephalus and acute cerebral hygroma, as appropriate. In acute lesions, both direct and indirect, **symptoms** appear **immediately or within a few hours**.

An important group is also **indirect chronic damage**, manifesting days, weeks or years after the trauma. These include delayed intraparenchymatous hemorrhage, late posttraumatic hygroma, chronic subdural hematoma, hydrocephalus, epilepsy, and several other clinical entities.

Table 1. - Traumatological groups in craniocerebral trauma'

Trauma classification'	**Clinical status of the patient**	
	state of consciousness	***local neurological symptoms***
mild CT	conscious or rapidly regaining consciousness	absent
moderate KT	conscious	present

Direct acute brain injury

Coma (commotion) of the brain

Reversible functional impairment, in which a **loss of consciousness** of varying duration (from seconds to minutes) is most often induced by a **direct** or **more rarely by a transmitted blow** to the head. After regaining consciousness, the **normal clinical findings**, dominated by **headache'** and **amnesia** for the period of loss of consciousness, and depending on the severity of the coma, usually a variable length of retrograde amnesia (for the period preceding the injury) and possibly also anterograde (for the following period). Long-term complications may include **non-specific post-ictal difficulties** (cephalea, photophobia, sleep disturbance, anxiety, depression, etc.) - these difficulties may be a manifestation of functional axonal impairment.

Concussion, brain contusion

Together with the contusion of the pia mater, it is most often caused **at the site of the applied force** *(par coup)* or **on the opposite side**, by a counterpunch against the skull *(par contrecoup)*. Impairment of consciousness may not always be present, but **focal symptoms** are usually evident, depending on the location of the lesion. Complications include cerebral edema of varying extent.

Brain Laceration (Rupture)

It is characterized by **the extensive brain and vascular involvement** with frequent subsequent intraparenchymal haemorrhage and subarachnoid haemorrhage.

Diffuse Axonal Palsy (DAP)

A very severe multiple axon disability is severed by **rotational and translational forces** acting on the head, most commonly in the corpus callosum and brainstem. The severity of the subsequent condition is compounded by the fact that **axon disruption leads to the washout of potassium**, which has a toxic effect on the surrounding brain tissue.

In DAP, the diagnosis is based on **a history of head injury**, **clinical status** (severe impairment of consciousness) and **MRI imaging** (CT is unhelpful, changes are usually below its resolution). It is the most common cause of subsequent vegetative state in trauma patients.

Indirect acute brain injury

Mechanism of diffuse axonal palsy (DAP)

Acute epidural haematoma (AEDH)

It usually arises as a consequence of Cranial vault fracture|calva fracture at the site of the course of the *a. meningea media*, which is associated with the rupture of this vessel. The bleeding is **rapid'** and forms an expanding mass between the dura mater and the calva (see figure). Increasing intracranial pressure results in **a displacement of central structures** (uncal, tentorial or occipital herniation) with **the oppression of the brainstem**. The **clinical picture'** sometimes includes an anamnestic **lucid interval**, in which a post-traumatic loss of consciousness (cerebral coma) is followed by a progressive impairment of consciousness (hematoma expansion, brainstem involvement) within minutes to tens of minutes after awakening. The **diagnosis** is made by the finding of **topical signs of herniation** (e.g. Griesinger's sign, **unilateral areactive mydriasis** from the oppression of the n. oculomotorius in incisura tentorii in temporal herniation) and the finding of **lens-shaped hyperdensity under the calva** on head CT.

Acute subdural hematoma (ASDH)

It is the **most common** type of traumatic intracranial haemorrhage, occurring even **after negligible**, often almost unnoticed trauma. The accumulation of blood is between the **dura mater** and the *arachnoidea* (see figure), as a consequence of **rupture of the bridging veins** or **pial vessels**. The most frequent localisation is in the **frontal** and **parietal** regions, not infrequently (15-20%) bilaterally. The classic course is characterized by the development of **focal symptoms** (from the direct pressure of the hematoma or herniation) and **alteration of consciousness**. In contrast to AEDH, the **development of symptoms is slower**, within hours after the injury. It is semilunar in shape on a CT scan. The elderly and patients with increased bleeding are particularly **at risk** of developing ASDH.

Traumatic subarachnoid hemorrhage (SAK)

It is characterized by **bleeding into the liquor ducts and subarachnoid space** (very often associated with cerebral contusion or laceration). Clinical symptoms are dominated by **headache**, and meningeal symptoms and in massive haemorrhages, **impaired consciousness** may be present. A subsequent complication is the development of **spasms of the cerebral arteries**.

Intracerebral hematoma (ICH)

It is caused by **injury to brain tissue and blood vessels**. It may be an isolated finding, but is often associated with **contusion and laceration** of the brain, in predilection areas **frontally and temporally**.

Clinical status varies from normal findings to progressive impairment of consciousness with an expansion of oedema around the haematoma and subsequent herniation. With the progression of clinical findings, **status monitoring** and **follow-up brain CT** (12-24 hours apart, possibly later) are necessary for the possibility of delayed intraparenchymatous hemorrhage.

Cerebral edema

It is a regular **subacute complication** (within tens of hours) of almost all Direct acute brain injuries and non-direct brain injuries. Due to **increased intracranial pressure**, blood flow is impaired even in unaffected areas, thus exacerbating brain damage. Further deterioration occurs with the development of **brain herniation**. Regular follow-up with CT scan**,** neurological examination **or Intracranial pressure (ICP)-monitoring is necessary.**

Swelling (edema) of the brain

It results from **vasomotor centre involvement**, leading to vasoparalysis and **cerebral congestion**. Increased vascular compartment volume leads to **increased intracranial pressure** and **impaired blood outflow** due to compression of venous structures. As a consequence, **diffuse swelling** and the development of **herniation of the brain** are common. The term **swelling** is not used uniformly, and sometimes no distinction is made between edema and swelling.

Pneumocephalus

The presence of **air in the intracranial space**. It is always the result of penetrating head injuries and may coexist with or without a complicating infection. Diagnosis is based on a CT scan.

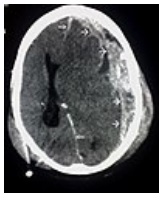
Acute cerebral hygroma

It is caused by **rupture of the arachnoidea** and permeation of cerebrospinal fluid into the subdural space. The acute course is caused by **the expansive behaviour** of the hygroma, which is explained by the valve mechanism of the arachnoid tear. The clinical course and diagnosis are identical to those of subdural hematoma.

Indirect chronic brain injury

Chronic subdural hematoma (CHSDH)

CHSDH is bleeding that occurs **even without an apparent history** and thus may be an incidental finding on imaging. However, it usually has a clinical correlate where the dominant finding is **headache**, **psychological alteration**, or **focal neurological symptoms**. A similar clinical finding is found in **late posttraumatic subdural hygroma**, arising after arachnoidea rupture, when the expansive process consists only of cerebrospinal fluid without significant blood admixture. Chronic subdural hematoma **is not a developmental stage of acute subdural hematoma** but a distinct nosological entity.



Subdural hematoma on CT

Other forms of chronic brain injury

- **Delayed intraparenchymatous hemorrhage** - see ICH.
- Hydrocephalus
- **Post-traumatic** epilepsy
- Organic psycho-syndrome

Diagnostic and therapeutic management of craniocerebral trauma

⚠Caution: the procedure starting with the history is given for didactic reasons only, the priority is always to examine and ensure the patient's vital functions!!!⚠

When examining the patient, it is necessary to anamnestic determine the **mechanism and intensity of injury**, **time since injury**, **duration and duration of unconsciousness**, and possible **risk factors** - medication, comorbidities and abuse *(Table 2., Fig. 7.)*.

Table 2 - Risk groups for craniocerebral trauma

Characteristics		***Typical mechanisms and complications of KT***
Age	over 65 years	subdural hematoma
Medication	warfarin, sedatives, analgesics	increased bleeding, distortion of clinical picture
Mechanism of injury	car accidents, falls from height, assault, epilepsy	fractures of the calva, skull base, mostly polytrauma, cervical spine trauma
Comorbidity	coagulation disorders	increased bleeding
Abusus	alcohol, drugs	subdural hematoma, increased bleeding in hepatopathy, fragility terrain

The principles of ABC must be followed in the **assessment and provision of vital signs**. We measure **blood pressure**, **heart rate**, and **saturation of O_2**. We assess the **level of consciousness** using the GCS, which we check at regular intervals. We apply a cervical collar whenever we suspect cervical spine involvement. We also assess **vegetative accompaniment** (nausea, vomiting) or the occurrence of **convulsions**. We look for other signs of trauma (hematomas and deformities on the head, face and body) and perform **body fluid sampling** for basic biochemical tests.

The neurological examination should assess **quantitative (GCS) and qualitative state of consciousness *and look for* *focal neurological signs* *(speech or behavioural disturbances, particularly anisocoria and abnormalities of stem reflexes on the cranial nerves, paresis on the limbs, reading disorders, coordination disorders). According to the findings, CTs are divided into severe, moderate and mild*** **(Table 1)**. The examination of **meningeal phenomena** (intracranial haemorrhage, possibly secondary infection) and the status of **mnestic functions** (retrograde, anterograde amnesia) are essential *(Table 3.)*.

Table 3. - Examination of a patient with craniocerebral trauma

Assessment of vital signs	breathing, pulse, blood pressure, O_2 saturation	ABC principles
Aspect	signs of trauma (*hematomas* on head and face), *otorea*, *rhinorea*, signs of trauma to the body	
Neurological examination	1. **Vigilance level'**	GCS
	2. **Stem symptoms**	cranial nerves, pupils - anisocoria!, mydriasis!
	3. **Other symptoms**	impairment of mobility, sensation, coordination, aphasia
	4. **Meningeal phenomena**	
	5. **Behavioural and mnestic disorders**	amnesia, frontal behavioural deliberation

Imaging methods

The main method in acute CT diagnosis is currently a CT scan. The role of **native X-rays** (bone structure injuries) continues to be important. MRI is more important at a later stage of diagnosis (method of choice in suspected DAP).

Diagnostic Conclusion

It is necessary to exclude other causes of impaired consciousness or coincidence with other pathologies (cardiac failure, syncope, pulmonary embolism, myocardial infarction, hypoglycaemia, epilepsy, etc.).

Therapy

For CT, the **surgical solution** should always be considered. Subsequent therapeutic measures must always be taken after **interdisciplinary collaboration** between, neurologists, neurosurgeons and intensivists. The **general condition** of the patient, the **time elapsed since the trauma**, the **other diseases** and the **prognosis** of the patient should always be taken into account. In some cases, **conservative management** consisting of intensive care with simultaneous monitoring of vital signs, intracranial pressure, and repeated imaging (CT) may be chosen in cases of **minor findings**.

Urgent surgical management is always necessary for **acute epidural hematoma**, when it is necessary to perform a craniotomy and **treat the source of bleeding**. **Removal of the hematoma from the trepanation ports** or the craniotomy is also the basic surgical procedure for **a subdural hematoma**. In **traumatic SAC**, in contrast to non-traumatic SAC, a **conservative procedure** is usually chosen. In other entities, especially ICH, DAP, cerebral edema, etc. the operative management is directed towards the **normalization and reduction of intracranial pressure (ICP)** and thus avoiding further involvement of vital brain areas for the time being, with **constant monitoring of the clinical status** (Intracranial sensor, transcranial Doppler, ventricular pressure, etc.). If necessary, a sufficiently **wide craniectomy** (unilateral, bilateral) can be performed. A therapeutic option also leading to a reduction of ICP is the introduction of **ventricular drainage** in case of the development of post-traumatic hydrocephalus.

Spinal cord trauma

Spinal cord (ST) trauma can be divided according to the **extent of involvement** into **complete**, i.e. acute transverse spinal cord lesion, where initially there is a loss of all spinal activity distal to the affected spinal segment (spinal shock), later on, primitive spinal activity is restored, but the involvement of the central pathways persists, and into **incomplete** involvement, i.e. posterior cord syndrome, lateral cord syndrome, or spinal cord hemisyndrome **(Brown-Séquard)**.

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The mechanism of spinal cord trauma is usually of the nature of shearing and rotational forces (whiplash injury, hyperflexion,
hyperextension) and, if the trauma is associated with fracture of bony structures, also compression' and injury by vertebral fragments or
haematoma (e.g. Secondary spinal cord lesions may also occur ischaemic spinal cord lesions through injury to a blood vessel.
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Thus, ST can be divided similarly to craniocerebral trauma into **direct** traumas (spinal cord compression, spinal cord contusion) and **indirect** traumas, which include haematomyelia (bleeding into the central parts of the spinal cord), secondary myelomalacia (spinal cord ischaemia with damage to vascular structures) and epidural haematoma.

Approach a patient with suspected ST

⚠ Attention: the examination starting with anamnesis is given for didactic reasons only, the priority is always to examine and ensure the patient's vital functions!!! ⚠

During the investigation of the patient, it is necessary to anamnestic **find out the mechanism and intensity of the injury**, the mechanism (slip, fall, jump, car accident - whiplash injury, etc.), the influence of **alcohol** (distortion of the clinical picture), **comorbidities** (osteoporosis, oncological history, etc.). The ABC principle of **(Airways, Breathing, and Circulation)** applies in the assessment and '**treatment of vital signs** - see also Acute Conditions in Neurology and Disorders of Consciousness/PGS. **Blood pressure**, **heart rate**, and **saturation of O_2** (80% of spinal cord injuries are associated with respiratory failure - see Table *Level of spinal cord lesion and respiratory failure*), *ECG*, etc. If cervical spine involvement is suspected, the head and cervical spine should be **fixed** immediately, at least by loading a cervical collar.

Level of spinal cord lesions and respiratory failure

level of disability	TLVC lung vital capacity	expectoration
C1 and C2	5-10 %	not
C3-C7	20 %	ineffective, weak
Th1-Th4	30-50 %	weak
Th5 onwards	minimal changes	normal

CAVE: Any patient with craniocerebral trauma or polytrauma must automatically be treated as having a possible spinal cord lesion.

As part of the physical examination, we look for other signs of trauma (limb deconfiguration, abdominal tenderness, etc.). In the neurological examination, we always look for possible signs of concomitant cranial trauma (up to 25 % of cases).

Examination and classification of spinal cord lesions

During the examination, the **motor function status** (monoparesis, paraparesis, quadriparesis/plegia) and **sensory function'** (*quantitative* - dysesthesia, hypesthesia, anaesthesia and **qualitative** - surface, deep sensation, dissociated disorders) are assessed - see Table 1. *Frankel classification of spinal traumata*, *Muscle strength assessment by muscle test* and image. We also detect **sphincter disorders** (mostly retention in the acute phase, more often incontinence later) and the status of **autonomic functions** - hypertension, pulsatile cephalea, bradycardia, profuse sweating, piloerection and flush (esp. (atrial fibrillation, supraventricular extrasystoles, atrioventricular conduction disturbances).

CAVE: Spinal cord traumas are very often associated with others within multiple traumas and polytraumas. Because of autonomic dysfunction (especially when the involvement is above the Th6 segment), the patient is unable to have peripheral vasoconstriction and acceleration of the heart rate; therefore, hemorrhagic shock should always be thought of, even if clinical signs are absent.

Classification of spinal cord traumata according to Frankel

A. Complete neurological failure	no motor and sensory function below the level of the lesion
B. Incomplete neurological lesion	only sensory functions preserved
C. Incomplete neurological lesion	partial preservation of mobility, muscle strength below 3
D. Incomplete neurological lesion	preserved mobility, muscle strength above 3
E. Motor and sensory functions normal	

Muscle strength assessment by muscle test

grade 5	Patient holds against maximum resistance, full range of motion
grade 4	Holds against strong, moderate resistance and gravity, full range of motion
Grade 3	Contraction with the movement against gravity, full range of motion
Grade 2	Contraction, motion weak, unrestricted when gravity is excluded
Grade 1	Weak contraction, no movement (twitch, trace)
grade 0	No contractile activity

In the topical diagnosis of a spinal lesion, it is necessary to determine its location in two planes - vertical and horizontal lesions.

Vertical level of involvement is consensually defined as the last segment with normal sensory and motor function (i.e., limits of sensation, motor segments, extinct reflexes, see figures) - e.g., C5 quadriplegia is characterized by abnormal motor and sensation from segment C6 onwards.

CAVE: the height of the spinal segments does not match the height of the corresponding vertebrae (see image)

In determining the vertical level of the lesion, it is necessary to **distinguish root involvement** (contusion, avulsion of nerve roots), which is characterized by paresis with extinct reflexes, from the manifestations of suprasegmental spinal cord lesions, where paresis is accompanied by reflexes (but may be distorted by spinal shock in the initial phase).

Depending on the horizontal extent of the lesion, spinal cord involvement may manifest as transversal spinal cord lesion syndrome, posterior fascicle syndrome, lateral fascicle syndrome, or spinal cord hemisyndrome, see also Differential diagnostic reasoning in neurology/PGS).

Transverse spinal cord lesion syndrome is initially characterized by **spinal shock** when spinal functions (areflexia, urinary and faecal retention) are extinguished. Gradually, however, reflex spinal activity (hyperreflexia and automatic bladder) appears.

Hemispinal hemisyndrome (Brown-Séquard syndrome) is characterized by homolateral central paresis and loss of proprioception and contralateral loss of sensation to pain and heat below the level of the lesion. **Posterior cord syndrome** is characterized by impaired proprioception and spinal ataxia. In **Anterior cord syndrome**, bilateral central motor paresis is accompanied by simultaneous central spinal gray lesions, loss of sensation to pain and heat, and preserved proprioception.

Diagnosis

Imaging methods: 3 projection radiographs (anteroposterior, lateral, transoral projection to dens), CT scan (axial sections, other planes, 3D imaging), MRI when soft tissue injury is suspected, including spinal cord (direct sagittal sections possible).

Other diagnostic methods: electromyography, somatosensory evoked potentials, motor evoked potentials

Therapy

ABC principles (see above) apply, including management of any shock state, for the administration of Methylprednisolone at 30 mg/kg i.v. bolus over 45 minutes with a break of 15 minutes followed by continuous administration of 5.4 mg/kg/h i.v. for 23 hours, possibly for 48 hours if the initial dose is administered only three hours after injury (Bracken et al., 1997), there is currently insufficient relevant evidence to confirm its effectiveness in patients with complete spinal cord lesions. On the contrary, the associated complications associated with its application predominate and thus its administration is not recommended (Hurlbert et al., 2013). **Urethral catheter insertion** is necessary. Of course, **spinal fixation** and transport in a stabilizing position to a **spinal unit** or to a trauma, neurosurgical or orthopedic unit where **spinal stabilization and spinal cord decompression** are performed. In the subsequent course, **early intensive rehabilitation** and careful nursing care are necessary, especially important for the **prevention of decubitus**.