Review

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Coma Caused by Severe Traumatic Brain Injury – Biomechanical Aspects

Key Words
Coma
Severe Brain Injury
Biomechanical aspects
Pathomorphology
Cranio-cerebral traumatism

Abstract

For a better understanding of the occurred brain damages the knowledge of biomechanical consequences in the cranium followed by force impact is necessary. Localization and severity of a brain injury can be euroanatomically derived. Diagnosis and prognosis may be predicted better more exact which again helps to impement an individual rehabilitation program.

The important investigations regarding the biomechanics of brain injuries were performed by SELLIER AND UNTERHARNSCHEIDT decades ago. The results of this investigations implicate that not, as previously assumed, a high pressure is caused by the force impact at the site of the impact, and that the high pressure causes the localized damages, but that through physical forces a under pressure occurs that lead to the main damages in the brain.

A precise diagnostic schema is introduced that meets the modern standards made by the results of sophisticated additional investigations in cooperation with an exact neurological examination, especially when taking the neuropathological findings into consideration. A precise diagnostic scheme should also ease the logistic and forensic difficulties after a brain injury.

In context with the impacting force on the cranium GRĆEVIĆ introduced the term linear brain trauma and proved it by neuropathological investigationnal results, like the results of the magnetic resonance imaging. Through his investigations, GRĆEVIĆ made the effect of the force impact been thought over. Better investigations than these done by SELLIER UND UNTERHARNSCHEIDT have not been performed yet.

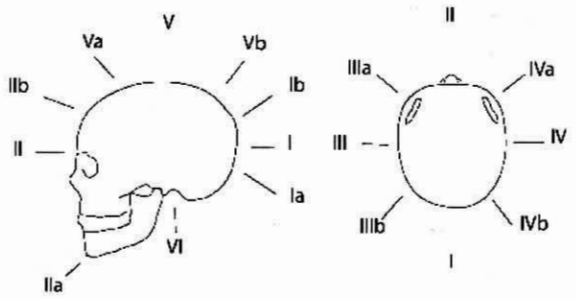
According to the introduced classification scheme the linear outer brain injury must be distinguished from the inner linear brain trauma, which is associated with severe inner injuries in the inner part of the brain. Lower linear inner and upper linear inner brain traumas are distinguished. The lower linear inner brain trauma must be considered as the most dangerous injury of the brain with damages in the midbrain. In the upper linear inner brain trauma the main physical factor is represented by the pressure gradient leading to a deformation of the skull. The findings of PUDENZ and SHELDEN explain the brain damages caused by rotational trauma quite considerably. In contrast to the linear brain trauma with supposed under pressure mechanisms rotational traumas with its shear forces which is the main cause for damages at the brain and at the vascular system in the cranium.

To estimate a brain trauma a exact classification of the effects is important. Primary brain injuries are distinguished from secondary brain injuries as well as tertiary traumatic brain injuries that often occur in severe and very severe brain damage, especially in patients traumatic apallic syndrome, that is a heavy burden for the patient. Finally, the quartiar brain damages and various complications must be considered.

Introduction

Traumatic Brain Injuries (TBI) are a major cause of death and disability in European countries. The incidence of TBI varies between 229 and 1967 per 100,000 inhabitants. The most common causes include vehicle accidents, falls and violence. The highest incidence occurs in men between 15 and 24 years of age. In fact, TBI is the leading cause of death for persons under 45 years of age (1, 2).

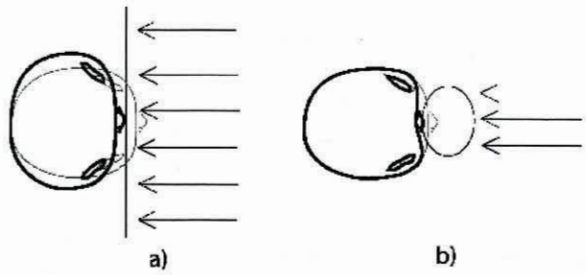
As early as 1936 SPATZ contributed significantly the scientific knowledge on commotio cerebri (3, 4). He focused on the reconstruction of the impact of the physical forces. 1934 SPATZ suggested a scheme, that is up to now as modified Innsbruck-Scheme well established and that is easy to use (5). TBI are classified into type I to type VI. In the Innsbruck-Scheme there are additional subclassifications, according to the lateral and vertical direction of the force impact to the head. Type I is referred to as a force impact to the back of the head, type II from the front (face, forehead), type III and type IV from the left and the right side respectively, type V from the top and type VI from below. The Innsbruck-modification of the SPATZ Scheme is shown in figure 1.



Scheme 1: Innsbruck modification of the Spatz- scheme

Biomechanic of severe Brain Injuries

According to the type of the force impact a deceleration trauma is distinguished from acceleration trauma. Deceleration trauma occurs when the moving head is decelerated by an impact, acceleration trauma occurs when the resting head is abruptly accelerated. The higher the mass and the impact area, the more energy the head absorbs. A flat impact leads to a general deformation of the scull (figure 2a), a narrow impact leads to a localized deformation, a localized impression trauma (figure 2b). A massive general deformation of the skull may lead to a diastatic skull fracture, an intensive local deformation may lead to a depressed skull fracture.



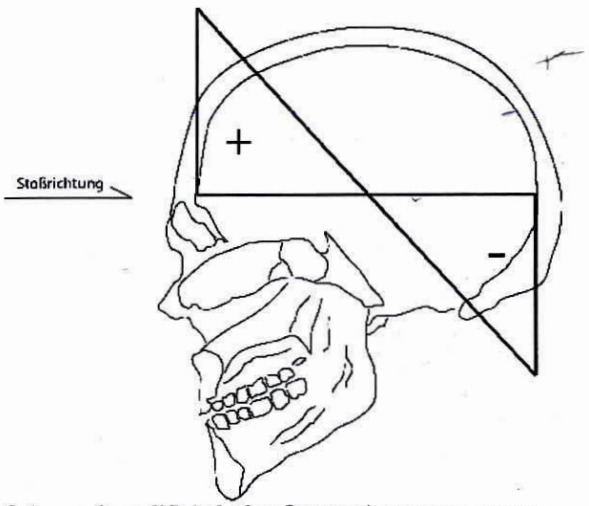
Scheme 2: a) large area force impact leads to a deformation oft he entire skull, b) a small impact area to a localized deformation

To understand the mechanisms, that lead to brain injuries due to force impact, the knowledge of physical basic principals are necessary. These were worked out by SELLIER and UNTERHARNSCHEIDT and published 1962 and 1963 (6-8). Two physical parameters play an important role in the blunt force impact on the head, this are the velocity ***v*** and the acceleration ***a.*** The simplified formula

$$a = v^2 / 2s \quad (s = \text{deceleration distance}).$$

With the same velocity ***v*** the acceleration ***a*** may vary strongly, when the deceleration distance ***s*** is very short. With higher acceleration ***a***, the risk for a brain injury next to an injury of the skull increases.

In their investigations SELLIER und UNTERHARNSCHEIDT indicated that except for the rotation trauma lesions in the brain cannot be explained by different movement directions and velocities of the brain and the skull. Due to acceleration ***a*** induced acceleration forces a pressure difference in the inside of the cranium. This pressure difference does not occur consistently in all points of the inside of the cranium. The pressure process during the impact is positive at the area of the impact (Coup-Pole), on the side opposite the area that was impacted (Countre-Coup-Pole) the pressure is negative.

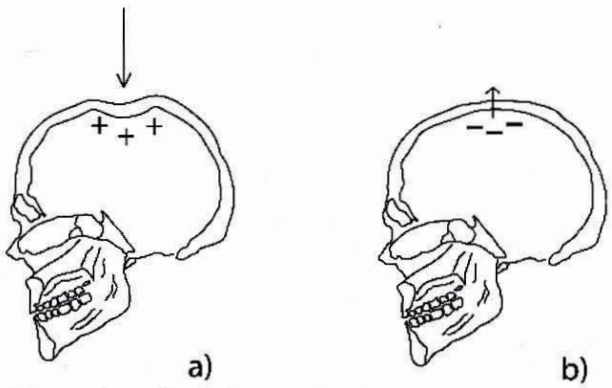


Scheme 4 modifizied after SELLIER) pressure curve within the skull at the very moment of force impact

The pressure decreases towards the center until a zero point (figure 3). The elastic human skull retreats so that an unsymmetrical pressure amplitude occurs. The damages, which occur in the brain tissue due to the pressure differences, are, according to SELLIER und UNTERHARNSCHEIDT, caused by a localized depression, because biological tissue is extraordinary resistant towards constant high pressure. Local depression leads to a dilation of the dissolved gases in the body tissue that leads to a severe dislocation in the brain tissue with damages of the brain tissue and small hemorrhages. The tissue damage at the area of the impact is, according to SELLIER and UNTERHARNSCHEIDT also caused by depression, that does not occur at the exact moment of the impact. They assume that an immediate impression is caused by the force impact (figure 4), this is succeeded by a rebound of the elastic skull bones into normal position. In the impression area after a short immediate overpressure a decompression occurs (figure 4 b). Small area impression traumas lead to localized impressions of the skull to an impact of the brain onto the skull. In this case, according to SELLIER und UNTERHARNSCHEIDT , the brain injury is also caused by depression, that occurs by the rebound of the skull.

Besides to the tissue damages caused by depression, in case of a force impact from frontal (type II), and also but less intense from the back (type I), another biomechanical factor was described. In the moment of the force impact, a deformation of the skull leads to a lateral expansion of the ventricular system. This leads to a depression on the lateral ventricular areas, which can cause tissue damages in the thalamus and in the corpus callosum with localized hemorrhages and tissue damages. At the poles of the ventricles however, due to the non deformed liquor, a high pressure occurs.

The third biomechanical component is the so-called rotation trauma. It was described by PUDENZ and SHELDEN in 1946 (9). When the skull is accelerated by a force from lateral, a rotation of the head occurs. Due to the inertia of the brain, it does not immediately follow the rotational movement of the skull. This leads to shear stress that can cause a tearing of the brain tissue and brain vessels. Lacerations in the brain and to intracerebral hemorrhages, but also to extracerebral hemorrhages may be the consequence (acute and chronic subdural hematoma, epidural hematoma).



Schema 3: small area impact first induces an increased localized intracranial pressure; the elastic skull then snaps back causing local negative pressure

In penetrating head injury the brain damage is also caused by pressure, if the brain tissue is not affected directly, although the opening should theoretically prevent it, as air flows into the skull. According to SELLIER and UNTERHARNSCHEIDT the opening of the skull occurs after the mechanical high pressure and decompression has already occurred. In addition of the local injuries caused by the force impact, dependent from the direction and intensity of the impact, also cortical contusion injuries and lacerational injuries occur.

When missiles penetrate the skull, a high pressure in the cerebral cavity occurs (according to SELLIER and UNTERHARNSCHEIDT 20-40 Atm) leading to a compression trauma. As result of the high pressure a destruction of the skull can

occur. Due to the high resistance of the brain tissue towards high pressure in many cases there is no further damage to the brain tissue except for the primary ballistic injuries.

Pathomorphologic features of distribution in severe Brain Injuries

Because of neuropathological findings, especially by GRĆEVIĆ in 1965 and in 1988 (10, 11), LINDENBERG in1960 and in 1964 (12, 13) and JELLINGER in 1967 (14), confirmed by MRI (Magnetresonance-Imaging) Investigations and by biomechanical findings, different features of cerebral injuries are distinguished. In principal the linear outer brain trauma with injuries on the brain surface must be distinguishes from the linear brain trauma with injuries in the deep parts of the brain and the rotational trauma.

In the linear outer brain trauma a impact force to the occipital area leads to a Coup- injury in the occipital lobe and the cerebellum. The Countre-Coup-Injury effects the frontal lobe and also often the temporal lobe. The Countre-Coup-injuries are mainly more severe than the Coup-injuries. In the deeper parts of the brain there will be no injuries. In the linear outer brain trauma with a force impact towards the face (type II), a good part of the energy is absorbed by the facial skull and the fractures caused by the impact, so the cortical injuries in the Coup-pole are not as severe and the lesions are mainly localized in the frontal lobe, mainly fronto-convex. At the Countre-Coup-Pole injuries in the occipital lobe and the cerebellum are found. When a diagonal impact occurs (type IIa and b) a more severe damage results on one hemisphere. In the linear outer brain trauma due to an impact force from the side (type III and IV) the results of the injury is mainly less severe as in cerebral traumas after a force impact type I and II. The cortical injuries are also more severe in the Countre-Coup- area than in the Coup area and often laterally shifted.

The linear inner brain trauma is subdivided into the linear upper inner brain trauma (GRĆEVIĆ) and the lower linear brain trauma (LINDENBERG). Both forms cause injuries in the deeper parts of the brain with tissue damages and hemorrhages.

After an impact force to the upper frontal parts of the skull (type IIb) and also from the front directly (type II) deformation of the skull occurs, which leads to a decompression in the areas surrounding the lateral ventricles and a high pressure in the frontal and occipital pole areas, as mentioned above, that causes the linear inner upper brain trauma. Due to the depression symmetrical tissue ruptures in the cella media occur (Butterfly-Pattern described by GRĆEVIĆ), that can reach to the basal ganglia including the thalamus and cause a rupture of the cerebral commisura.

After force impact from the top (type V) and from the upper front (type Va) the pressure wave continues to the inner part of the inner parts of the brain and causes the linear inner lower brain injury (LINDENBERG). The linear inner lower brain injury leads to injuries in the upper brainstem area, the surrounding brain parts, the medial temporal lobe, the cerebellum and occasionally also the diencephalon. The linear inner lower brain injury may cause an acute mid-brain syndrome and may occasionally end lethal in a bulbar brain syndrome. Mostly the acute mid-brain syndrome leads due to injuries of the upper brainstem to a traumatic apallic syndrome with a bad prognosis

The rotational trauma described by PUDENZ and SHELDEN is characterized by lacerations of the inner part of the brain mainly in the area of the internal capsule, as well as intracerebral hemorrhages due to a tearing of the perforating arteries.. Pathomechanism is a rotational trauma of the head around the cervical vertebral column (15,16). Due to injuries of the veins it may come to an extra cerebral hematoma (acute subdural hematoma, and after a temporal interval chronical subdural hematoma), caused by an injury of the medial meningeal artery an acute epidural hematoma can occur.

Force impact type VI leads to a more or less distinctive combination of a linear brain trauma with a rotational trauma, depending of the direction of the impacting force. An impact force type VIa and VIb leads to a rotation of the head that moves freely backwards and forwards on the vertebral column with counter movement of the brain around a fictive horizontal axis, in which various variants of the axis alignment are possible depending of the direction of the force impact and the posture of the head. The spinal column absorbs a part of the impact and is often also injured. Because these accidents are mainly due to

car and plane accidents, there is also an absorption of the force by the cushioning and the head cushioning, so the head only rarely suffers from high acceleration forces. Due to the collision itself an indirect force impact on the head occurs, leading to a secondary linear outer brain injury type I or type II. A force impact type VIa can occur in boxing sports by rotational trauma that leads to a tearing of the perforating arteries or the veins. In some cases intracerebral lacerations and intracerebral hematomas are possible, when additionally a lateral friction of the impact occurs.

After direct impact on the back or the legs, the impact force gets transduced upwards via the vertebral column causing a direct force impact type VI and hereby to a direct force impact compression trauma. According to this model the force hits the cranial base and leads to a linear lower brain injury with a predilection site of the lesion on the brainstem. Detailed corresponding neuropathological data and MRI investigations are still missing. In a type VI force impact additional injuries of the cervical spinal column must be considered.

Multiple force impacts to the cranium lead to multiple forms and to a multiple degrees of severity. Cortical contusions and at the same time occurring injuries in the inner parts of the brain can lead to intra- and extracerebral hemorrhages. The effect of the brain injury and its progression are associated with the degree of severity and the localization of the destroyed brain tissue as well as complications caused by an expanding hemorrhage and a concomitant localized and/or diffuse brain edema.

Classification of the brain damages

For the therapy of a brain injury in the acute phase next to the location at the in the moment of the impact occurring damage, the degree of severity of the primary morphological changes is of substantional relevance. Additionally, to the primary irreversible damages in the post acute stadium secondary damages occur, also during remission tertiary and quaternary damages can occur (17) leading to complications, especially in severe and most severe brain injuries.

The primary brain injury occurs at the moment of the blunt force impact to the cranium or respectively to penetrating brain injuries. Cortical contusions at the surface of the brain must be distinguished from traumatic injuries of the inner parts of the brain as well as lacerations of the brain tissue with intracerebral hemorrhages caused by shear forces. Primary traumatic brain damage are also found in the surroundings of intracerebral hemorrhages. These primary traumatic brain damages are irreversible.

TERTIARY CNS-DAMAGES AFTER TRAUMATIC BRAIN INJURY
Caused by Malnutrition, Hypovitaminosis, elektrolyte dysbalance, Bed Rest Syndrome
enzephalopathy
Pontine Myelinolysis
Myelopathy
Polyneuropathy

Table 1: Causes and Consequences of tertiar damages after brain injuries

Secondary traumatic brain damages originate in many different reasons. Secondary traumatic brain injuries occur in the penumbra, the peri-epicenter of the zonular area surrounding the primary lesion with a partly damaged brain tissue. The severity of the damage decreases towards the periphery. The penumbra represents the "battle field" for the therapy. Secondary traumatic tissue damages of the brain can be caused by a global hypoxemia of the entire brain, due to hypotension of the blood pressure, blood loss or a state of shock. Secondary disturbed blood flow in a localized part of the brain can be caused by a compression (of the posterior cerebral artery) due to uncal herniation and can cause secondary brain damages in the accordant irrigation area (pseudolaminar necrosis of the occipital lobe, infarction of the Thalamus, pallidum, sub- and hypothalamus, etc.) (10, 18). Secondary cerebellar damages might occure by compression of the cerebellar arteries.. A diffuse or local brain edema leads to a global or regional brain damage (20). A localized pressure in tentorial herniation or foraminal herniation causes secondary traumatic damages in the brainstem (18,21). Due to tentorial herniation, cranial nerves (esp. oculomotorius nerve) are endangered. All secondary traumatic brain injuries are treatable.

Tertiary damages occur in the post-acute phase and often during rehabilitation of severe brain traumas. Multiple causes like malnutrition,

QUARTIAR DAMAGES AFTER TRAUMATIC BRAIN INJURY
Hydrocephalus occlusus
Hygrome
Meningoencephalitis per continuitatem or embolisch bedingt
Brain abscess

Table 2: Quartiar damages after brain injury

malresorption, avitaminosis and the bed rest-syndrome (partial microgravity) etc. are responsible for it. In highly severe brain injuries the critical care neuropathy may also occur. Tertiary consequences of brain injuries can occur as encephalopathy, pontine myelinolysis, myelopathy and polyneuropathy (Tab. 1). Polyneuropathy in patients with the bed rest syndrome show a very severe muscular atrophy and is the typical syndrome of a badly treated apalic syndrome. Tertiary brain damages are considered as iatrogen and can be avoided by optimal therapy.

Quartial brain trauma damages include hydrocephalus occlusus, hydrocephalus aresorptivus, menigo-encephalitis and brain abszess. These quartial damages can be recognised very soon, when the patient is monitored, and can be controlled with a rapid treatment.

Complications may occur quite early but often occur during the rehabilitation process after severe brain injuries. In addition to the above mentioned CNS damages, contractions of the large and medium-sized joints, periarticular ossifications, pressure sores and damages have to be prevented.

BED REST SYNDROME
Polyneuropathy
Primary muscular atrophy with change of the muscular enzymes and structural lesions.
Proprioceptive damages (Spinal ataxia, Hinterstrang Symptome)
Spastic Symptoms (mild)
Cerebellar Symptoms (mild)
Reduced Vigilance
Confusion
Vegetative Instability
Osteporosis

Table 3: Symptoms of the Bed Rest Syndrome

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