Traumatic brain injury, new aspects in classification, using biomechanical and neuropathological factors

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Traumatic Brain Injury (TBI) as an attribute to the modern way of life is the most frequent cause of death in the male population aged between 20 and 35 worldwide. Improved standards of living, modern traffic, industrialisation, development and advance of the society is responsible for this development.

Severity and localisation of the brain lesion in TBI patients depends on direction, focus and intensity of the impact. A great percentage of brain injury patients suffer from more then one impact. For documentation, archiving and for the biomechanical reconstruction of the impact force, the Innsbruck Impact Scheme (IIS) modified to SPATZ is essential. Type I to type VI are differentiated.

Based on clinical symptomatology, the neuropathological findings and modern neuroimaging methods (living pathology-Grcevic), there are three distinguished forms of TBI.

1. The linear outer brain trauma (type I, II, III, IV) with lesions of the surface of the brain (contre coup, coup region).
2. The linear inner brain trauma, divided in two forms.

   The linear inner upper brain trauma (type II b, I c) with periventricular lesions (butterfly defect-Grcevic).

   The linear inner lower brain trauma (type V a) with lesions in the upper brain stem and in the surrounding region (Lindberg).
3. The rotational brain trauma (type III a, III b, IV a, IV b) with intracerebral lesions (intracerebral haematoma, delaceration), extracerebral haematoma (Pudenz, Shelden).

Depending to the clinical symptoms, the course of a TBI and the changes of the brain tissue, mild, moderate, severe, severest TBI have to be classified.

Regarding the histological and the anatomical features after TBI there are four well-defined forms of traumatic brain lesions. The primary brain damages induced in the very moment of force impact to the brain tissue are irreversible. Secondary brain lesions as sequences of circulatory and oxygenous deficits in the Penumbra of primary defects causing local tissue lesions. Hypoxia and/or hypoxemia are responsible for diffuse and regional tissue lesions. Tertiary lesions developing mostly in a longer posttraumatic course are responsible for encephalopathia, pontine myolinolysis, myelopathia and polyneuropathia, originated by malnutrition, malabsorption, avitaminosis and the bed rest syndrome. Quartery lesions might emerge months to years after the TBI event in form of hydrocephalus occlusus, meningoencephalitis and brain abcess. As complications contractions of the bigger joints, periarticular ossification, decubitus and lesions of peripheral nerves have to be kept in mind.

In the acute state every patient with a TBI needs exact neurological controls with different additional examinations depending on the grade, but obligatory in severe and severest conditions. A neuromonitoring is necessary for every patient.

Acute complications and most endangering the outcome of TBI patients are brain oedema and intracranial haematoma. Both increase the intracranial volume resulting in a tentorial herniation, sometimes with foraminal herniation, accompanied by an acute mid brain syndrome and sometimes by an acute bulbar brain syndrome (Gerstenbrand, Lücking). In severest conditions a traumatic apallic syndrome (an unfortunate synonym for this condition is vegetative state) may develop (Gerstenbrand). It is important not to miss an accompanying cervical spine injury in cases of spinal cord trauma.

Regardless on the grade of TBI every patient needs acute therapy. The treatment programme has to be accompanied by a monitoring system, essentially including MTBI (Vos et al.). In severe and severest TBI a neurosurgical consultation is necessary. The treatment of TBI has to start already on the site of accident, initiated with measures to insure the vital functions. Severest TBI patients have to be transferred immediately to a up-to--date ICU.

Main symptoms of the apallic syndrome are coma vigile, no recognition of the surroundings, optomotoric disturbances, loss of all voluntary movements. flexed strechted position of the extremities with stretched position of trunk, primitive motor patterns and dysregulation of the vegetative system. 80% of apallic patients develop a remission course with eight different phases (Innsbruck Remission Scale).

In phase III to IV Klüver Bucy Symptoms are the leading features. Reaching these stages promises a good prognosis. On the other side the primary and secondary lesions of the brain can be noticed in detailed form. This local neurological lesions may deteriorate the whole condition and decisively decide the clinical outcome.

During the remission state the preannounced tertiary lesions have a great influence on the remission course in the same way the complications too.

Every patient with TBI – regardless to severity — needs a neurorehabilitation with an individual schedule. A special centre with trained personal under the responsibility of a neurologist is necessary. The neurorehabilitation has to start immediately, already in the admitting hospital. A consistent programme of modern neurorehabilitation can reduce not only distress and the independency of young patients, it reduces the expenses of the health system to a considerable rate.

1. Birbamer G., Gerstenbrand F., Grcevic N., 1999, Klassifikation d.schweren cerebralen Traumas. Acta. Chir. Austr., 31,20-22.
2. Gerstenbrand F., 1967, Das traumatische apallische Syndrom, Springer. Wien-New York.
3. Gerstenbrand F., Lücking C.H.,1970, Die akuten traumatischen Himstammschäden, Arch. Psychiatr. Nervenhk., 213, 264-281.
4. Gerstenbrand F., Rumpl E., 1995, Rehabilitation nach Hirnverletzung, Intensiv. Med.,7,832-842.
5. Grcevic N., 1988, The concept of inner cerebral trauma, Scand.J.Rehab.Med.Supl.,17,25-31.
6. Lindenberg R., Freytag E., 1960, The mechanism. of cerebral concutions, Arch.Path. 69,440-469.
7. Vos P.E., et al., 2002, Europ.J. Neurol., EFNS Guideline on MTBI, 9, 207-219.

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