

An Outline of the Current Concepts of Mild Brain Injury with Special Emphasis on the Adult Population

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SUMMARY

Mild brain injury is one of the most common neurological and neurotraumatological diagnoses. The pathophysiological basis of mild brain injury is frequent diffuse axonal damage of a variable degree. In the acute phase of mild brain injury, we have to identify about 1% of patients who will undergo neurosurgery because of vital need. Analysis of the patient's personal history, screening of risk factors, neuropsychological testing and imaging methods (CT, MRI) are indispensable in the diagnostic process of mild brain injury. Although mild brain injury is currently considered an irrelevant traumatic event, approximately 10% of patients develop so-called post-concussion syndrome.

Key words: mild brain injury, cerebral concussion, epidemiology, pathophysiology, diagnosis, management.

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EPIDEMIOLOGY

Mild brain injury (MBI) includes patients with the diagnosis of cerebral concussion and accounts for 70-80% of all craniocerebral injuries. The incidence of MBI worldwide is approximately 600/100,000 pop. per year, with the incidence of MBI requiring hospitalization in the range of 100 to 300/100,000 pop. per year. MBI occurs in men twice as often as in the female population, with the age group at highest risk being those aged 15-24 years. The main causes of MBI are traffic accidents and falls (1). In the Slovak Republic, 250 MBI patients/ 100,000 pop. (70% of men and 30% of women) were hospitalized in 2003, with MBI ranking as seventh most frequent cause of hospitalization (along with strokes) (2). In the neighboring Czech Republic, the hospitalization rate for the same year was 310 MBI patients per 100,000 population (65% of men and 35% of women) (3).

TERMINOLOGY

Perhaps the first to use the term "cerebral concussion" was one of the greatest Arab physicians and philosophers Razi Abu-Bakr Mohammed ibn Zakarija, known in Europe under the name Rhazes (850-923 BC). He described cerebral concussion as abnormal brain function devoid of apparent injury, thus setting a historical milestone in its definition (4). In the 16th century, the French surgeon Ambroise Paré was already using the term *commotio cerebri* (5). Later, in the 20th century, along with the development of the Glasgow Coma Scale (GCS) by Jennett and Teasdale, the term mild

brain injury (MBI) was coined referring to cases with a GCS of 13-15 including patients with *commotio cerebri*. In the Anglo-Saxon literature other terms are used to describe cerebral concussion (mild traumatic brain injury, mild head injury, mild concussion syndrome, traumatic head syndrome) (6, 7). Given the differences of injury prognosis and resulting outcome, as well as differences in the findings from head and brain computed tomography (CT) examination, some authors reserve the term MBI for GCS 13-14 whilst the term minor brain injury refers to GCS 15 (8). States with GCS 13 are classified as moderate injury by most authors (9, 10).

The literature gives several different definitions of cerebral concussion; as a result, no consensus has to date been reached on the issue. In 2001, a task force called CISG (the Concussion in Sport Group) (11) proposed a new revised definition of cerebral concussion defined as follows:

- Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.
- Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.
- Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
- Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
- Concussion is typically associated with grossly normal structural neuroimaging studies.

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The authors of this review article define cerebral concussion as a mechanical injury to the head with subsequent short-term unconsciousness and/or disorientation and/or amnesia and ad integrum normalization within 24 hours. As related to MBI, cerebral concussion is a narrower nosological entity.

The WHO Collaborating Task Force on Mild Traumatic Brain Injury defines MBI as a state caused by the action of external mechanical energy to the region of the head associated with subsequent central nervous system dysfunction (10). The clinical picture must meet at least one of the following criteria:

1. Confusion and/or disorientation,
2. Loss of consciousness of less than 30 minutes' duration,
3. Post-traumatic amnesia of less than 24 hours' duration,
4. Any other transient neurological symptomatology (focal neurological deficit, seizure),
5. Intracranial lesion not requiring neurosurgical intervention.

Compared with cerebral concussion, MBI is a broader nosological entity including CT-detected, minor intracranial lesions not requiring surgery, transient focal neurological deficit and epileptic seizure. On the other hand, it should be noted that transient focal neurological deficit (e. g., hemiparesis) previously comprised part of some concepts of cerebral concussion as so-called commotio focalis. Using imaging methods, cerebral concussion is conventionally characterized by a normal finding. However, use of magnetic resonance imaging (MRI) in the diagnosis of commotio cerebri including newer types of weighing suggests a significant part of states classified as cerebral concussion includes, in the acute and/or subacute stage of injury, structural alterations of brain tissue.

The assessment is based on clinical neurological examination including determination of the GCS score and the patient's medical history (subjective and, whenever possible, objective). The clinical picture must not be confounded by penetrating craniocerebral injury, medication and addictive agents, concomitant condition (e. g., acute post-traumatic stress disorders) or other circumstances (e. g., language barrier).

PATHOPHYSIOLOGY

The cause of MBI symptomatology including cerebral concussion is mild diffuse axonal injury (DAI), due to shear mechanism on rapid acceleration or deceleration in movement of the head (12).

In head injury, external static forces (duration of action over 200 ms) or dynamic forces (duration of action below 200 ms) act on the skull (13). Static forces are represented by skull compression (e. g., injury due to closing sliding lift doors). Cranial injury caused by static forces is quite a rare event. Action of static forces in animal models generally do not result in unconsciousness, even in cases of severe skull fractures and brain contusion. The most frequent cause of craniocerebral injuries is the action of dynamic forces. Dynamic forces are divided into impact and impulsive ones (14). Impulsive forces cause the head to move without mechanical blow to the head: e. g., in a road accident; a head-on collision while restrained by seatbelts results in sudden deceleration of the driver's torso, with the head moving forward until it stops by exhausting the elasticity of cervical structures (whiplash injury). Impact forces bring the head into contact with another body (e. g., strike by a blunt object, fall on the floor) and, unlike impulsive forces, they cause superficial injuries to the scalp, skull fractures, extradural hematomas and brain contusions. Brain contusions occur as far as areas opposite to the site of impact (contre-coup). In real life, impulsive and impact forces often act at the same time (e. g., the driver's head also hits the dashboard). The result of action of dynamic forces is acceleration or deceleration of the head. The movement may be either linear (force momentum goes through the

head's center of gravity), rotational (the head turns around its own center of gravity) or angular (with the head turning around a point other than its center of gravity). In head injury in real life, all three types of movement are usually present to a different extent.

The brain is a homogeneous structure, as it is composed of parts with different physical properties (grey matter, white matter, cerebrospinal fluid spaces, etc.). Angular or rotational acceleration/deceleration is associated with a relative shift of neighboring parts with different unit weight and, consequently, momentum. Nerve fibers and tiny vessels in the borderline regions situated tangentially to the plane of shift are compressed and strained at the same time. Radially crossing fibers and vessels are also subjected to shear forces and are the most damaged ones. A similar shear mechanism between the skull bone structures and the brain results in rupture of veins and development of acute subdural hematoma.

DAI occurs by shear mechanism on rotational or angular acceleration/deceleration mainly at the coronal plane (15). In animal models, the clinical status correlates with the extent of DAI (16), with the extent of DAI being non-linearly dependent on the velocity of acceleration/deceleration (17). A factor aiding in defining the extent of DAI, in addition to the action of external forces, is the position of dural folds (13). Not all the mechanisms and biomechanism parameters related to brain tissue injury have been fully explored to date. Axonal damage by shear forces is a process evolving in the order of several hours to days. So-called primary axotomy (instantaneous severance of axon continuity at the time of injury) occurs rarely and solely in severe craniocerebral injury (18). Mechanical physical insult in MBI entails in impaired permeability of the axoplasmic membrane with impaired signal conduction, impaired axoplasmic transport, regional axonal edema and cytoskeletal degeneration. In a proportion of MBI patients, the result of this is secondary axotomy. In this period, cell metabolism works at top rate and the partially damaged nerve cell is extremely sensitive to any other insult (18, 19). This period may be as long as several weeks. The intricate cascade of metabolic processes, the primary early sequel of which is dysfunction of damaged structures, may cross the limit of reversibility and induce delayed death, i. e., apoptosis of the nervous cells involved. On the contrary, necrosis is involved as a mechanism of nervous cell death in severe craniocerebral injury.

CLINICAL PICTURE

MBI and cerebral concussion may, but need not, result in short-term unconsciousness in the presence of the dysfunction of the ascending reticular activation system. Other features include impaired memory, confusion, and behavioral changes (Tab. 1), usually resolving within a space of several minutes. Retrograde and/or anterograde amnesia remains to involve events associated with the injury. Retrograde (pre-trauma) amnesia evolves over time. Following restoration of continuous memory, retrograde amnesia may prolong within several minutes with subsequent repeat shortening of the interval of minutes of memory loss, with a permanent residuum remaining in most cases (20, 21). Post-traumatic (anterograde) amnesia covers the period from injury to restoration of continuous memory and, unlike retrograde amnesia, is not a disorder of recall but a disorder of storing and encoding information. Consequently, anterograde amnesia does not alter over time and is permanent and invariable. In the period prior to continuous memory restoration, there may be islands of recollection to ongoing events. From the perspective of an individual sustaining cerebral concussion, the length of unconsciousness is identical with that of anterograde amnesia (22).

Tab. 1. Acute neurobehavioral presentations of MBI

Unconsciousness
Memory impairment (repeats questions asked, unable to remember words, objects)
Vacant stare (befuddled facial expression)
Slowed verbal and motor response (responds to and executes commands with latency)
Inability to focus attention (easy to disturb, unable to perform everyday activities)
Disorientation (goes in wrong directions, unable to say what the time/date is, or where he/she is)
Incoherent speech (makes pauses, speaks incoherently)
Impaired motor coordination (tripping, unable to walk a line)
Inadequate emotionality (restlessness, proneness to cry)

Tab. 2. Most frequent symptoms of the post-commotion syndrome

Somatic: headache, dizziness, blurred vision, diplopia, nausea, vomiting, sleep disorders, easy to get tired, hypersensitivity to light and noise, tinnitus
Cognitive: impaired attention, memory, speech, slowed thinking, disorders of executive functions
Emotional: emotional instability, sadness, anxiety, apathy

In the ensuing period, the features predominant in the clinical picture include several somatic, cognitive and emotional symptoms (Tab. 2). Some of these, such as impaired attention, disorders of selected executive functions as well as difficult sleeping and mood swings, occur 1-2 weeks after the injury, while the patient makes every effort to reassume full responsibility about themselves and get back to their personal and professional lives.

The psychological picture is dominated by impaired memory, and the patient has difficulty learning new information. Likewise, attention is reduced and the patient's flexibility of thinking, speed of information processing, and planning are impaired. Some authors have reported disorders of visuospatial constructional ability as well as disturbed verbal and sensorimotor functions. Psychological examination in patients sustaining head injury plays a major role in top sports, whereby evaluation of the effect of injury on brain function of the sportsman is the element in the decision-making regarding future procedures, specifically the sportsman's return to play. In addition to psychological assessment, an important role in this process is played by adjunct imaging and electrophysiological techniques of examination (e. g., MR and EEG). On first contact with an individual sustaining cerebral concussion, it is appropriate to use selected standardized screening neuropsychological tests (e. g., Standardized Assessment of Concussion, Maddock's questions) serving as guidance to determine, within a couple of minutes, impairment of selected cognitive functions (23, 24). Psychological assessment may be biased by previous head injury,

degree of cognitive function prior to injury, education, age, anxiety, impaired ability to concentrate, sleep deprivation, medication and addictive agents, concomitant health problems, language barrier, and previous psychological assessment (23). The above factors should be taken into account in the final evaluation of the psychological assessment.

In the differential diagnosis, it is critical to bear in mind that the so-called postconcussion symptoms are not specific for cerebral concussion and are also present in depressive, anxious and somatoform disorders (6, 25, 26). A proportion of these symptoms also occur in the general population who have not so far sought medical attention (27). In most cases, the symptoms resolve within 3 to 12 months after head injury (28). One or several symptoms will persist as late as one year after the injury in about 10% of patients. These symptoms most often include headache, dizziness (predominantly positional), fatigue, impaired attention, impaired memory and selected executive functions (6). The total of several persisting postconcussion symptoms is referred to as the postconcussion syndrome. According to the International Classification of Diseases (ICD-10), it is defined as a group of at least 3 selected symptoms developing within the first 4 weeks following head injury associated with unconsciousness. According to DSM-IV (Diagnostic and Statistical Manual), a postconcussion disorder refers to a group of at least 3 of the following symptoms (fatigue, sleep disorders, headache, dizziness, irritability, personality changes, spontaneity disorders) persisting for 3 months and longer since head injury along with impaired memory or attention present during neuropsychological assessment. Cerebral concussion is defined as injury-caused unconsciousness of duration longer than 5 minutes and/or post-traumatic amnesia of duration longer than 12 hours and/or development of epileptic seizures (or clear deterioration of a pre-existing epileptic syndrome) within 6 months of the injury (28). Revision of both definitions is most likely to be unavoidable because of the lack of a rationale for the criteria (e. g., confirmed unconsciousness, epileptic seizures within 6 months of the injury). The presence and persistence of unconsciousness in MBI and cerebral concussion are not prognostic factors affecting the persistence of postconcussion symptomatology (29, 30). In addition, McAllister and Arciniegas report that, in most cases, development of one postconcussion symptom is not related to the development of another postconcussion symptom, challenging the concept of a uniform postconcussion syndrome (31). Although the term "postconcussion syndrome" was coined 130 years ago, there has not been unanimity regarding its etiopathogenesis. It is currently recognized that, in addition to the degree of organic damage to brain tissue, psychogenic factors also play a role in the development of postconcussion symptomatology, with the proportion (not strictly defined yet) of the structural and functional components varying from one case to another. In his paper, King (32) provides a list of organic and psychological factors affecting the outcome of MBI patients. An important prognostically adverse factor of the postconcussion syndrome is the patient's effort to seek financial compensation or another benefit (20, 28). A critical consideration in these cases is a thorough differential diagnosis. The literature identifies other, less important prognostically adverse factors for long-term persistence of symptoms of the postconcussion syndrome (age over 40 years, female sex, previous brain damage, stress, psychiatric disorders, and premorbid personality structure, dizziness and excruciating headache on baseline assessment after injury, increased serum levels of protein S100b, skull fracture) (28, 33, 34). Future research in this area is warranted.

Tab. 3. Classification of MBI severity (5)

Group of patients	Clinical characteristics
0 (head injury, no brain injury)	GCS 15, without unconsciousness, amnesia, and risk factors
1	GCS 15, unconsciousness <30 mins, anterograde amnesia <1 hr, no risk factors
2	GCS 15, unconsciousness <30 mins, anterograde amnesia <1 hr, risk factors present
3	GCS 13-14, unconsciousness <30 mins, anterograde amnesia <1 hr, with/without risk factors

Tab. 4. Stratification of MBI patients by their risk for developing complications and by management of the state (37)

	Low risk	Medium risk	High risk
GCS	15	15 with clinical findings	14–15 and neurodeficits or skull fracture or risk factors with/without clinical findings
Clinical findings	No	1. Amnesia 2. Diffuse headache 3. Vomiting 4. Loss of consciousness	1. Amnesia 2. Diffuse headache 3. Vomiting 4. Loss of consciousness
Neurodeficits	No	No	Yes
Skull fracture	No	No	Yes
Risk factors	No	No	1. Coagulopathy 2. Age >60 years 3. Previous neurosurgery 4. Pre-trauma epilepsy 5. Alcohol and/or drug misuse
Imaging methods	No	CT scan or x-ray	CT scan
Management	Discharged	In-hospital observation (3–6 hrs post-CT or 24 hrs post x-ray) followed by observation at home	In-hospital observation for 24-48 hrs followed by observation at home

DIAGNOSIS AND MANAGEMENT

The issue of craniocerebral injury is typically an interdisciplinary one involving neurology, traumatology, neurosurgery and psychology. Every patient sustaining head injury should undergo neurological assessment. Of particular importance in MBI is a thorough both direct (subjective, data from the injured person) and indirect (objective, data from any accident witnesses) history, making it possible to track exactly the course of the accident and to determine the presence and duration of confusion, unconsciousness or amnesia. The differential diagnosis of MBI including cerebral concussion is indeed very broad, virtually encompassing the entire range of neurological and internal disorders, potentially producing short-term consciousness impairment associated with any fall and head injury. The main reason for the critical need for professional MBI assessment is to identify the 1% of patients who will later have to undergo vitally indicated neurosurgery. These cases include mainly extracerebral hematomas (epidural, subdural), skull fractures with dislocation or, possibly, impression of a bone fragment, brain contusion, intracerebral traumatic hemorrhage and brain edema. Emergency neurosurgical management is usually not required with minor intracerebral hemorrhage, traumatic subarachnoidal hemorrhage, pneumocephalus and DAI (5). The prevalence of intracranial focal (multifocal) damage, need for and number of neurosurgical

intervention, and mortality in head injury closely correlate with the baseline GCS value. With GCS 15, 14, and 13, the prevalence of CT detection of intracranial focal damage is 8%, 20%, and 30%, respectively (35).

Vos et al. propose to divide patients into four groups by the degree of risk for developing severe intracranial lesions (Tab. 3) and define the risk factors of grim development of status following head injury: unclear or ambiguous accident history, continued anterograde amnesia, retrograde amnesia of more than 30 minutes' duration, signs of injury in the region above the clavicle, suspected skull fracture, post-traumatic severe headache, vomiting, focal neurological deficit, seizures, age over 60 years and below 2 years, coagulation disorders, intoxication by alcohol and/or illegal drugs, and a high-energy accident (according to the Advanced Trauma Life Support Principles, a high-energy accident is characterized by the following: vehicle collision at a speed over 64 km/h, extensive deformation of the car body or its impression of more than 30 cm into the inside passenger space, time to accident victim removal over 20 minutes, fall from a height of 6 m and higher, overturning of the car, hitting a person by car, motorcycle crash at a speed over 32 km/h, separation of the driver and motorcycle during accident) (5). Ibanez et al. extend the above risk factors to include loss of consciousness and hydrocephalus treated with a shunt (9). By contrast, Borg et al. do not consider unconsciousness a risk factor for adverse

development of MBI (35). Patients with GCS 15 but without unconsciousness, amnesia, and presence of risk factors (group 0) may be discharged without head and brain CT scanning to receive home care, unless there is another reason for hospitalization. Patients with GCS 15, unconsciousness, anterograde amnesia and presence of one or several risk factors should undergo CT scanning (group 2 on a mandatory basis and group 1 as an individual option). In the event of a normal CT findings, patients may be discharged to receive home care (group 2 as an individual option). Patients with a pathological result of head and brain CT scanning should be hospitalized. In-hospital observation should last 24 hours as a minimum. Needless to say, discharging the patient can also be considered in cases with clinically non-significant CT findings and findings definitely unrelated to the injury. In cases where CT assessment is not feasible, an acceptable alternative is hospitalization of the patient with thorough observation. All patients with GCS 13–14 should undergo CT scanning with subsequent hospitalization (5, 35). Vos et al. have noted that, since it usually takes up to 6 hours for epidural hematoma to evolve, the baseline CT finding may be normal. It is for this reason that close monitoring of the patient is imperative (e. g., initially every 15–30 minutes prolonging the interval to 1–2 hours if there is no deterioration in the finding) (5). The above proposed standard for MBI management (Vos et al.) (5), adopted by the European Federation of Neurological Societies (EFNS), is subscribed to and approximated by the standard of the Czech Neurological Society (CNS) (36). The most recently published stratification of MBI patients by the risk for developing intracranial complications and their proposed management is the outcome of a prospective study by Fabbri et al. using the proposed procedure in a series of 5,578 patients (Tab. 4) (37).

The value of head and brain MRI in craniocerebral injury has recently increased. Studies conducted over the past 15 years have shown MRI is a method with higher sensitivity and specificity compared with CT scanning also in the acute stage of craniocerebral injury. Specifically, MRI allows more detailed visualization of tiny and non-hemorrhagic foci. In MRI, DAI is visualized as multiple tiny non-hemorrhagic foci located deep in the hemisphere (80%) or those with central petechial bleeding (20%). The predilective areas for these foci include the posterior region of the corpus callosum and in the splenium corporis callosi, as well as dorsolaterally in the mesencephalon and in the superior region of the pons Varoli as well as the area of junction of brain grey and white matters (38, 39). Using MRI, Mittl et al. identify DAI in up to 30% of MBI patients with a normal head and brain CT scan (40). The standard MRI protocol employed for assessing head injury includes T1 and T2 weighted spin-echo, FLAIR sequences and T2* weighted gradient-echo sequence (38). Addition of diffusion weighted imaging (DWI) and DTI (diffusor tension imaging) sequence to the standard protocol will further enhance the sensitivity of MRI in MBI (41, 42).

Compared with the past, the value and rationale for classical native skull radiography has clearly diminished. Skull radiography is unable to diagnose intracranial focal damage. Its value is confined to diagnosis of skull fractures as a risk-related radiologic factor in craniocerebral injury (5).

The basis of medical care in MBI continues to be close monitoring of the patient's status and symptomatic therapy (e. g., analgesics in headache). Causal MBI therapy meeting conditions of evidence-based medicine has not yet been proposed. In his review article, McCrory draws attention to the potential beneficial clinical effect of corticosteroids, opiate receptor antagonists, calcium-channel antagonists and arachidonic acid metabolism inhibitors in the management of MBI (43). De Kruijk et al. have not confirmed the relevance and inevitability of strict bed-rest for the outcome of MBI patients (44). Early brief education and activation of the patients (single cognitive-behavioral psychotherapeutic interview) after

MBI demonstrably reduces the rate of later complications (45). It is critical to advise the patient about the nature of MBI, the most frequent complications and appropriate adequate algorithms for the management. The patient should be informed about the favorable prognosis of MBI and encouraged to resume social and professional activity as early as possible.

PREVENTION AND CONCLUSION

MBI, including cerebral concussion, is generally considered a rather minor injury carrying a low risk for development of intracranial complications and associated with low mortality rates (being 0.01% and 1.10% with GCS 15 and GCS 13, respectively). Particular attention should be given to the issue of the so-called postconcussion syndrome. Still, given the high incidence (with MBI being the seventh most frequent cause of hospitalization in the Slovak Republic), MBI is a significant contributor to overall morbidity of the population. Of special importance for society is general prevention of craniocerebral injury. The mandatory use of helmets by two-wheel vehicle riders (bicycles, motorcycles) has helped reduce the risk of craniocerebral injury by more than 50% (1). Adequate education of the pediatric and adult populations, modification of risk-related operating procedures in various occupations, changes of rules in selected sports, and individual efforts at caution and not overestimating one's abilities may further reduce the incidence of MBI in the future.

Abbreviations

CISG	- Concussion in Sport Group
CT	- computed tomography
DAI	- diffusion axonal injury
DSM	- Diagnostic and Statistical Manual
DTI	- diffusion tensor imaging
DWI	- diffusion-weighted imaging
EEG	- electroencephalography
EFNS	- European Federation of Neurological Societies
GCS	- Glasgow Coma Scale
ICD	- International Classification of Diseases
MBI	- mild brain injury
MRI	- magnetic resonance imaging
WHO	- World Health Organisation

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Comments on the Article by Sivák Š. et al. “An outline of the current concepts of mild brain injury with special emphasis on the adult population”

The authors have selected the highly topical issue of head injury as encountered by the traumatologist, neurologist, diagnostic radiologist and/or the neurosurgeon. As indicated by the text, this is not a simple issue. Other, not negligible, considerations to be taken into account by the physician include the cost of so-called unnecessary examinations or, alternatively, hospitalization for the purpose of observation; on the other hand, there are omnipresent lawyers with the potential threat of “maltreatment”.

The category of “mild brain injury” includes simple cerebral concussion, which comprises the vast majority of these traumas (70–80%). Epidemiological data clearly show that those affected are predominantly males. When characterizing cerebral concussion, every effort should be made to avoid using terms such as “briefly unconscious” since, as we were told by Professor Václav Jedlička, a tumor is not the size of a tomato; he wanted to know its dimensions in centimeters. I consider most useful an older classification of brain injury developed by the Colorado Medical Association (1), recommended for sportsmen (specifically for boxing which I myself would not include among sports), i. e., Grade I – confusion without amnesia and loss of “consciousness”; the individual should be monitored every 5 minutes and, unless amnesia or other symptoms develop, return to boxing is possible within 20 minutes. Grade II – confusion with amnesia yet without loss of consciousness. Return to activity after a week free from symptoms. And, finally, Grade III – loss of consciousness with a fall, requiring careful transfer to hospital, possibly with cervical fixation. On negative neurological finding, further observation can be performed at home, with return to activity not sooner than after 14 days free from symptoms. The syndrome of MBI no doubt includes minor damage to anatomical structures, overseen on a single neuroimaging assessment; however, they do manifest themselves clinically and can be identified by neuroradiology if the insult is repeated (encephalopathia pugilistica, post-traumatic dementia in repeatedly falling alcoholics and other individuals), with deposition of amyloid, indiscernible from the amyloid in Alzheimer disease (2).

The time factor and remission of symptomatology in MBI can be compared with vascular pathology and the conventional term TIA.

An interesting experimental finding is that static (or, more exactly, slow) action of force on the skull until its fracture or formation of concussion foci in animals does not result in unconsciousness.

As regards post-commotion syndrome, it is commendable to point out the syndrome is present in as many as 10% of cases of brain commotion (provided they were truly “reversible” processes). This diagnosis can be established in the presence of three non-specific symptoms such as fatigue, sleep disorders, changes in emotivity, and personality changes. Duration of unconsciousness is not a critical factor; a mixture of organic and functional changes is sometimes difficult to distinguish from even purposeful action.

Acute states with increasing symptomatology, possibly requiring neurosurgical intervention, need not be discussed. However, what should be mentioned is the nomenclature of amnesia. Retroactive amnesia refers to the time prior to injury. Accordingly, my preferred term for anterograde amnesia would be posteroactive (post-traumatic) amnesia. It is reasonable to count its duration into the total time of unconsciousness. The authors deserve credit for referring to risk-related factors including, in addition to others, ages over 60 years and below 2 years, considering the brain’s increased vulnerability. Little attention has to date been given to lesions sustained in road-related brain injury at speeds over 64 km/h in cars and over 32 km/h in motorcycle riders and, needless to say, in cyclists. A newly arising question is that of native X-ray examination of the skull (and, one should add, also the cervical spine!), which we perform on a routine basis in our center, and which helps to determine the degree of risks potentially present in the ensuing course, and has a forensic value. It is out of the scope of an editorial to comment on the value of MRI—still, there is little doubt it may uncover, even in CT negative scans, diffuse axonal damage with (20%) or without (80%) petechias, particularly in the region of the corpus callosum or in the brainstem.

One must fully subscribe to the conclusions and suggestions regarding the need for prevention, as formulated by colleagues Sivák and his coworkers, be these improved passive safety (helmets worn by skiers, cyclists, advocated [by Henner] even in boxers, safety belts, etc.); however, special emphasis should be placed on patient education to cautiousness, proper self-assessment and avoidance of clearly risky behavior, as the phenomenon of the “human factor” is present in most accidents.

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Comments on the Article by Sivák Š. et al. “An outline of the current concepts of mild brain injury with special emphasis on the adult population”

The collective paper by authors from the Department of Neurology and Department of Orthopedics and Traumatology, Comenius University School of Medicine and Municipal University Hospital in Martin, and from the Department of Neurology, Comenius University School of Medicine and University Hospital in Bratislava, Slovakia, is a review article. It is evident from the paper that its authors have drawn from a thorough study of the relevant literature, and that their interpretation is based on their own extensive clinical experience, although the paper is not an analysis of their own group of patients. A most valuable feature is that the authors include statistical data not only for the Slovak Republic but also for the Czech Republic. The topic of the review article is one - the mildest - degree of brain injury. Still, the topic is an important one of widespread interest.

As early as 1773, Petit divided brain injury into three types, i. e., cerebral concussion (*commotio cerebri*), cerebral contusion and cerebral compression. Another banal classification is that into open or closed injury, and into primary and secondary injury (1, 2). While this classification is still useful and is used today in everyday practice, it has long been unsatisfactory. Is the criterion of open or closed injury the dura or other structures of the head, e. g., the skin and bone of the skull or the skull base? Thousands of articles and monographs have addressed the topic of brain injury both worldwide and in the Czech literature. This is due to our inadequate knowledge of pathophysiological processes and their development over time, the most varied mechanisms of injury, still inadequate diagnostic potential, and different clinical requirements on classification schemes. For example, schemes should provide an algorithm for immediate therapeutic procedure as well as long-term prognosis of the outcome. The generally known classification proposed by Teasdale and Jennett in 1974 originally only focused on consciousness impairment, with additions made in 1976, to evolve into its current form as the Glasgow Coma Scale (GCS). Prediction methods include the Glasgow Outcome Scale (GOS), the Glasgow-Liège score, and Narayan's, Choi's, or Glauber's logistic models and the classification regression tree model. At present, there are, and recognized to a different extent, at least 30 classification schemes of brain injury, based on, to a varying extent, various symptoms of injury (2, 3). Although we have gained considerable insight into the pathophysiology of brain injury (e. g., diffuse axonal injury) over the past decades, it should be noted that the criteria used to adopt classifications are those based on arbitrary consensus. For example, Kunc (1), in our classical monograph published in 1968, including its third edition appearing in 1983, defines cerebral concussion as a reversible brain injury, possibly without loss of consciousness in 5-10% of cases; retrograde amnesia may also be absent and yet the patient may die from the injury. Likewise, in the latest Czech monograph on brain injury, published in 2001, Smrčka et al. state, in their definition of cerebral concussion, duration of unconsciousness of up to 60 minutes, but note: “This division is ... arbitrary” (2).

When defining classifications of brain injury, we generally draw on results of methods of examination, particularly CT and MRI, further GCS, duration of unconsciousness, duration of amnesia, and the set of so-called risk factors. To put it very simply, injury is classified as mild (GCS 13–15), moderate (GCS 9–12), severe (GCS 8–5), and critical (GCS 4–3).

Is it necessary to write a special article about mild brain injury? Would it not be enough to refer to the latest Czech monograph on brain injury published in 2001 (2)? Unfortunately, it would not, as this predominantly neurosurgically-oriented monograph covers the term of mild brain injury in a cursory remark. Still, it is a term every physician dealing with accidents, every accident unit, intensivist, neurologist, neurosurgeon, and even every psychiatrist and psychologist should be familiar with. Is it necessary to develop a still more detailed division and classification for this mildest degree of brain injury? Current international literature and, in particular, actual management of this frequent and diagnostically serious, though a mild degree of head injury, point to the need for a sub-classification. In my view, the most informative and crucial part of the review article, with implications for everyday practice, is Tab. 3, getting the reader acquainted with the classification of mild brain injury severity proposed by Vos et al. in 2002.

The strength, but also a weakness, of the present article is that it does not disregard the issue and the biological and arbitrary relativity of the definition of the term “mild brain injury”. Though called “an outline” by the authors, the review article (including a long list of relevant references and many Tables) provides the reader with a wealth of essential information about the topic. This, however, does not make it possible to underline implications for practice. Though briefly mentioned in different parts of the review article, I would appreciate more emphasis on the implications of mild brain injury categorization in categories 0–3. All categories of mild brain injury should be assessed by the neurologist, and individuals in categories 0 and 1 with normal CT scans without risk factors can be discharged to receive home care. Patients in other categories must be hospitalized and observed for a minimum of 24 hours. Otherwise, they may miss the vital interval necessary for a decompression procedure - the only intervention to prevent irreversible secondary brain damage.

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