

Concussion in Sports – What Trauma/Orthopedic Surgeons Need to Know!

Otřes mozku ve sportu – co by měl traumatolog a ortoped znát

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SUMMARY

Concussion in sport is often underdiagnosed with the potential risk of long-term sequelae. This article presents the mechanisms, the underlying pathophysiology and typical primary signs and symptoms. The recognition and resulting medical measures including the present recommendations and decisions on return to play are described.

The majority of patients with concussion present with clinical and cognitive symptoms only for short time intervals. As a rule a complete subjective recovery is observed within a few hours or days. Although neurocognitive impairments can persist in individual cases, they also show a good tendency to heal. Thus, after 1 year nearly all patients no longer have any relevant disorders.

In a few cases long-term disturbances can occur in the presence of certain risk factors and/or after repetitive concussion. Unspecific symptoms and some cognitive impairments are the main reported problems; however, there is also a potential but individually unpredictable risk of developing neurodegenerative alterations and diseases.

INTRODUCTION

Mild traumatic brain injury (mTBI) is defined with a Glasgow Coma Scale (GCS) of 13 to 15 points. Brain concussion represents the predominant portion of mTBI (Fig. 1). The patient is usually conscious, being awake, with open eyes, responds adequately to speech and can perform targeted movements on request.

During the last 2 to 3 decades, intensive discussions and analyses were performed on sport-related concussions in the anglo-american scientific literature. This resulted in several consensus recommendations during the last 10 years (44). The topic of brain concussion became increasingly popular even in Germany, especially in high contact sports, e.g. ice hockey (58).

Thus, the present overview will give basic knowledge on this relevant injury regarding recognition, acute management and potential long-term problems.

Definition

Concussion is a brain injury! According to the Consensus statement on concussion in sport from 2012, a concussion is defined as „a complex pathophysiological process affecting the brain, induced by biomechanical forces“ (44). The latter can act directly or indirectly against the head. A concussion typically results in a rapid, short impairment of neurological functions that typically resolve spontaneously but can also lead to neuropathological changes.

Acute clinical symptoms indicate a more functional problem than a structural damage. Correspondingly, standard image techniques (CT/MRI) usually show no structural pathologies (44).

Injury mechanism

According to the above mentioned definition, a concussion may be caused by a direct blow or indirect force acting against the head and the brain (46).

The exact injury mechanism is unknown as several different force vectors can result in a concussion. A direct blow against the head can lead to a local brain injury at the impact site, resulting in some intracranial shaking of the whole brain. Brain parts can bounce against the bone even on the side opposite to the primary force induction. Additionally, injuries due to shearing forces and tissue deceleration along the irregularly shaped cranial base and the *falx cerebri* and *tentorium cerebelli* may occur. Many direct mechanisms are obvious (e.g. head impact against the ground etc.). The indirect injury is a result from transferred forces, not directly striking the head, which are usually transmitted to the brain via the body's trunk.

The force can be directed along an imaginary straight line (linear force effect) or it results in a rotational movement of the head and brain. In the majority a combination of both potential force mechanisms are present.

CONCUSSION RECOGNITION TOOL™
Taschenkarte
Hilfe für das Erkennen einer Gehirnerschütterung bei Kindern, Jugendlichen und Erwachsenen



ERKENNEN UND HANDELN
Eine Gehirnerschütterung sollte bei Vorliegen **eines oder mehrerer** der im Folgenden unter Punkt 1 bis 3 genannten sichtbaren Hinweise, Zeichen, Symptome oder bei falschen Antworten auf die Gedächtnisfragen vermutet werden.

1. Sichtbare Hinweise für den Verdacht auf eine Gehirnerschütterung
Jeder der folgenden sichtbaren Hinweise kann auf eine Gehirnerschütterung hinweisen:

Bewusstseinsverlust oder verlangsamte Reaktion
Liegt bewegungslos auf dem Boden/ verlangsamt beim Aufstehen
Unsicherheit auf den Füßen/ Gleichgewichtsprobleme oder Fallen/Koordinationsstörungen
Sich an den Kopf fassen/den Kopf mit den Händen stützen
Benommener, leerer oder ausdrucksloser Blick
Verwirrtheit/ nicht des Spiels oder der Geschehnisse bewusst

2. Zeichen und Symptome für eine mögliche Gehirnerschütterung
Jedes der folgenden Zeichen und Symptome kann eine Gehirnerschütterung anzeigen:

- Bewusstseinsverlust
- Krampfanfall
- Gleichgewichtsprobleme
- Übelkeit oder Erbrechen
- Benommenheit/Schläfrigkeit
- Emotionaler als gewohnt
- Reizbarkeit
- Traurigkeit
- Erschöpfung/Ermüdung oder wenig Energie
- Nervös oder ängstlich
- „Etwas stimmt nicht mit mir“
- Schwierigkeiten, sich zu erinnern
- Kopfschmerzen
- Schwindelgefühl
- Verwirrtheit
- Gefühl „verlangsamt/ langsam zu sein“
- „Druck im Kopf“
- Verschwommenes Sehen
- Lichtempfindlichkeit
- Gedächtnislücke
- Gefühl „wie im Nebel/benebelt zu sein“
- Nackenschmerzen
- Geräuschempfindlichkeit
- Schwierigkeiten, sich zu konzentrieren

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3. Gedächtnisfunktion

Wenn eine Frage nicht richtig beantwortet wird, besteht der Verdacht auf eine Gehirnerschütterung.

- „An welchem Spielort sind wir heute?“
„Welche Halbzeit ist jetzt?“
„Wer hat in diesem Spiel zuletzt ein Tor erzielt?“
„Gegen welches Team haben Sie letzte Woche im letzten Spiel gespielt?“
„Hat Ihre Mannschaft das letzte Spiel gewonnen?“

Jede(r) Sportlerin/Sportler mit Verdacht auf eine Gehirnerschütterung ist UMGEHEND AUS DEM SPIEL ZU NEHMEN und darf nicht zur Aktivität zurückkehren, bevor sie/er medizinisch untersucht worden ist. Sportlerinnen/Sportler mit Verdacht auf eine Gehirnerschütterung dürfen nicht allein gelassen werden und kein Fahrzeug führen.

Bei Verdacht auf eine Gehirnerschütterung ist empfohlen, dass die Spielerin/der Spieler an eine medizinische Fachperson zur Diagnosedstellung, Betreuung und Rückkehr zum Spiel-Entscheid überwiesen wird, auch wenn die Symptome abgeklungen sind.

WARNSZEICHEN

Bei JEDEM der unten aufgeführten Warnzeichen ist die Spielerin/der Spieler sicher und umgehend aus dem Spiel zu nehmen. Wenn keine medizinische Fachperson verfügbar ist, sollte ein Notfalltransport in das nächstliegende Krankenhaus zur umgehenden ärztlichen Untersuchung erwogen werden.

- Sportler(in) klagt über Nackenschmerzen
- Verschlechterung des Bewusstseinszustandes
- Zunehmende Verwirrtheit/Reizbarkeit
- Schwere oder zunehmende Kopfschmerzen
- Wiederholtes Erbrechen
- Ungewöhnliche Verhaltensänderung
- Krampfanfälle
- Doppelbilder
- Schwäche oder Brennen/Kribbeln in Armen oder Beinen

Wichtig:

- In allen Fällen müssen die grundlegenden Prinzipien der Ersten Hilfe (Gefahr erkennen, Reaktion prüfen, stabile Seitenlage, Atmung und Kreislauf stabilisieren) befolgt werden
- Nicht versuchen, die Sportlerin/den Sportler zu bewegen (ausser, um sie/ihn in die stabile Seitenlage zu bringen), wenn keine spezifische Ausbildung besteht
- Einen (evtl. vorhandenen) Helm nicht abnehmen, wenn keine spezifische Ausbildung besteht.

aus: McCrory et. al, Consensus Statement on Concussion in Sport, Br J Sports Med 47 (5), 2013

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Fig. 1. Concussion as the major part of mTBI.

In particular, the accelerating torsional forces seem to be the major cause of neurological disturbances.

As a result of these effects, different brain movements can occur within the bony skull, leading to involvement/injury of different brain regions, which can explain the wide range of different symptoms to be observed. The mechanism can be subtle and clinically not obvious. Therefore, a positive correlation between the force extend and the resulting symptoms is often unusual (40).

Despite standard imaging techniques fail to show structural injury, „nonvisible injuries“ to the neurons and the smallest blood vessels must always be assumed. These cell injuries lead to complex changes in the cells, which can trigger inflammatory reactions in the tissues, restrict chemical changes and cell nutrition.

Pathophysiology

The mechanical deformation of the brain tissue during trauma can result in shear injuries to the brain cells (38). In consequence, local cellular injury can be present, axons can be damaged or even disrupted from the cell bodies or even apoptosis can occur.

According to the primary injury mechanism, focal/direct and diffuse brain injuries can be distinguished.

The local direct (focal) damage to the brain tissue leads to excessive excitation of the cells. As a result, substances enter the surrounding tissue, which negatively influence the energy supply of the neurons. The adjacent supporting cells in the brain are also damaged resulting

in functional impairment. As a result of cell membrane changes, mitochondrial dysfunction and cellular swelling occurs, leading to an energy crisis, which ultimately can result in complete loss of neuronal function. The extent of mitochondrial damage correlates with the long-term (persistent consequences) of the brain damage. Additionally, the local cerebral blood flow is diminished, supporting the energy crisis. This pathophysiological neurometabolic cascade is supposed to be a main reason of functional neurological impairment (25).

Diffuse brain tissue injury leads to axonal injury, which can sometimes be visualized with new imaging techniques. A stretching of 10% within 100 ms can lead to permanent axonal damage. Myelination of the

Table 1. Typical symptoms after concussions

Physical	Cognitive	Emotional	Sleep
headache	feeling foggy	sadness	trouble falling asleep
dizziness	slowed down	irritability	impaired sleep duration
balance problems	impaired concentration	emotional	less sleep than usual
voimiting	difficulty remembering	nervousness	more sleep than usual
sensitivity to light/noise	repeating questions		
visual problems			
fatigue			

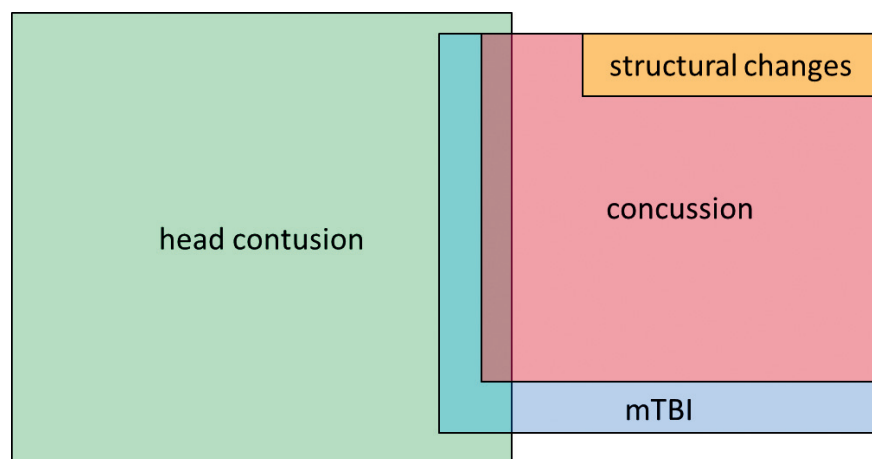


Fig. 2. Pocket Concussion Recognition Tool.

axons can be a protective mechanism. Myelination of axons usually provides faster information transport between the cells. After injury, this information transport is disturbed (38). In children, during brain development, myelination is incomplete, a possible reason that the children's brain is more vulnerable, potentially explaining the prolonged recovery phase. Histologically, comparable changes like in Alzheimer's disease could be detected.

In addition to these neuronal consequences, local circulatory disturbances in the injured brain causes increased oxygen demand, which is present for several days. The cardiovascular system can show heart rate variability during physical and mental stress.

Signs and symptoms

Signs (objective detection, observation) of concussion can include confusion, loss of consciousness, gait balance disturbances and swaying, pupil difference, a dazed, blank or vacant look and/or grabbing/clutching of head.

Classical symptoms such as unconsciousness and amnesia are reported in athletes in about 20% (4) and < 10% in recreational sports (47).

The subjective symptom complex is broad and includes clinical, neurocognitive symptoms and behavioral and sleep changes (40, 44, Table 1). The most common primary symptoms include headache (70–80%), dizziness (34–70%), nausea/vomiting (20–40%), weakness and fatigue (20–50%), visual disturbances (approx. 20%) and sensitivity to light and noise (10–60%) (4, 35).

In the acute assessment, the focus is generally not yet focused on neurocognitive symptoms, behavioral and sleep changes.

The frequency problem

Concussion are underreported and misunderstood by all involved persons as they are underestimated in its consequences (17, 42). Athletes perform a return-to-play often too early, despite adequate knowledge about the injury, due to „external pressure" by coaches, athletic trainers and even team physicians (11, 31, 34).

The overall rate of overlooked or unreported concussions is high with an average of 40% (30.5–81.5%) (18, 22, 36, 40, 48). In recreational sports a significantly higher rate of unrecognized injuries is reported (69).

Immediate recognition = sideline evaluation

It is generally accepted, that athletes with suspected concussion have to immediately stop their participation in sports and should be judged by a physician on the same day (44).

Several tools for sideline evaluation are available including GCS, Maddocks questions, Standardized Assessment of Concussion (SAC), Balance-Error-Scoring-System (BESS), King-Devick-Test, Sport Concussion Assessment Tool (SCAT3-Testing for children and adults), and computer-based neurocognitive approaches, e.g. ImPACT® etc. (Overview in: (19)). For orientational assessment directly at the field, the Pocket Concussion Recognition Tool (PCRT) is an easy tool, which can be used by either of physicians and non-medical personnel (Fig. 2).

The PCRT includes an assessment of objective signs, subjective symptoms and basic orientation. The maximum time to follow these steps is 1 minute (19).

An immediate medical evaluation of patients is necessary, when red-flag symptoms are present: youth age, confusion > 30 min, loss of consciousness > 5 min, focal neurological deficit, pupil difference and deterioration of symptoms or consciousness.

The athlete should not be left alone, regular monitoring within the next hour is clearly recommended and the general rule "When in doubt, take them out!" should be applied.

Acute in-hospital evaluation

The clinical and neurological assessment is often insufficient, as analyses of neurocognitive consequences are often not integrated in primary evaluation concepts.

The primary aim of in-hospital evaluation is to exclude significant intracranial structural lesions, especially in the presence of risk factors (54). Therefore, standard radiological evaluation consists of cerebral computer tomography (CCT) and in some centers magnetic resonance tomography (MRI).

Using the New Orleans Criteria or the Canadian CT Head Rule, validated criteria for implementation of CCT in the primary diagnostic algorithm in mTBI are available (30, 64).

MRI has an increased sensitivity compared to CCT, identifying up to 30% structural lesion while having a negative CCT (2). In a meta-analysis of patients with mTBI and GCS of 15, which were primarily unconscious or suffered from amnesia, CCT confirmed 7.8% (6.1–9.5%) structural lesions (1). As in typical concussions,

the presence of these clinical signs is low (10–20%), indicating a minor risk in concussions.

Clinically, a detailed head-neck evaluation is recommended including an orientated neurological evaluation with testing the mental status, cognitive functions and balance and a re-analysis of present symptoms (44). Early assessment of cognitive functions seems to be an essential component of primary evaluation (44).

A useful tool can be the SCAT3 which is available for children aged 5–12 years and older persons > 12 years (44). The SCAT3 screening includes analysis of clinical symptoms, the GCS, Maddock s-Questions, SAC-Testing and modified BESS-Testing. Testing balance is supposed of relevant value in the primary and secondary evaluation of these patients (16).

Due to its prognostic relevance, a detailed medical history has to be analyzed including number of pre-existing concussions/TBI, duration of symptoms, presence and type of amnesia (retrograde/antegrade) (44).

Blood biomarkers (e.g. S100B) are presently not recommended for general use, as they fail to clearly distinguish between concussed and non-concussed patients, especially in sports (40, 61). S100B was able to identify patients "not at risk" (21, 40).

Acute treatment

Based on the knowledge of the pathophysiological changes at the cellular level, primary physical and mental rest was recommended (44) in order not to stress the disturbed cells with cognitive work, thus extending the recovery process. Cognitive rest involves the reduction of reading, computer use, texting, television or films, video games and other mental activities. The complete elimination of cognitive stimuli is not recommended (56).

Present knowledge indicates, that complete mental and physical rest does not appear to be meaningful but rather an intellectual and physical activity can improve the recovery phase at an early stage (24, 67). A prolonged complete resting phase can lead to other problems, e.g. depressive mood and fatigue (5). The primary goal is to modify these stimuli not to trigger or worsen symptoms.

All other treatment modalities in this initial phase are more or less symptomatic treatments of special symptoms.

Standard recovery

Complete subjective recovery of clinical symptoms is usually present within one week after trauma in approximately 85% of cases. 97% fully recover within 1 month. Complete symptom recovery is typically within 3–12 months (40, 41). It has to be noticed, that clinical symptoms (Table 1) typically show faster recovery than neurocognitive problems (39). Despite this positive prognosis, one year after trauma, about 15% of patients report on relevant symptoms, mostly headaches and motion disturbances (55). Recent studies show that, especially in children and younger people, symptom duration can be prolonged. In children, an average symptom duration of 4 to 6 weeks was reported (7, 27, 53).

Various investigations could detect relevant neurocognitive disorders for a longer period of time. Using com-

puter-based neuropsychological testing, the sensitivity and specificity of the recovery assessment could be optimized, in contrast to single clinical symptom analysis (33, 59).

Therefore, additive neuropsychological testing, especially with baseline examination is supposed to be an important part of follow-up examination getting a return-to-play decision. It should always be used in combination with other evaluation tools and not as an isolated decision-making tool (44).

Risk factors of prolonged recovery

Several risk factors affecting the healing process are identified. Primary presence of significant headaches, weakness/fatigue and the presence of amnesia as well as a pathological neurological examination may result in a prolonged recovery phase.

Other factors associated with prolongation of symptoms were female sex, the initial presence of a retrograde or antegrade amnesia, preexisting brain function disorders, anxiety, depression, learning disabilities and/or migraine.

In young and middle-aged children a statistically significant extended rehabilitation phase was observed compared to adolescents and adults (40).

Return-to-work/school and return-to-play

Consideration of the pathophysiology and the natural recovery process is important in deciding return-to-play (44). An athlete should be at rest and after exercise clinically and cognitively symptom-free before competitiveness consists!

Prerequisite for return-to-play is the complete recovery during school or professional working. Therefore, a return-to-play at the day of trauma is the absolute exception.

Accordingly, a step-by-step recovery concept is recommended, integrating the return-to school/work and return-to-sports (Fig. 3).

At least 6 to 10 days are normally necessary before full return-to sport is possible, which corresponds to the minimum recovery time of the affected neurons.

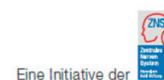
Post-concussion syndrome

In some patients, symptoms remain observable over a longer time period (> 3 months, post-concussion syndrome – PCS). Overall, these symptoms are considered non-specific as many of these symptoms are present after other injuries or diseases. Accordingly, a high prevalence of PCS symptoms is observed in the normal population (26).

Because many of these symptoms are of different origin, specialized disciplines, e.g. neuropsychology, neurology, physiotherapy, otorhinolaryngology etc.) should be integrated in an interdisciplinary management concept, if symptom prolongation is extended > 4–6 weeks after primary injury (3, 15).

Special focus should concentrate on vestibulo-ocular dysfunction, which is observed, especially in children in up to 30%, resulting in a 2-fold prolongation of recovery (20). Due to force transmission along the

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Return-to-School/Work: expected time window: 2-4 days	
stage 1	no mental activity causing complaints, no significant concentration (no homework, no reading, no SMS, no video games, no computer work, etc.), possibly lots of sleep.
stage 2	gradual, controlled increase of mental activities start mental activities for a short time (5-15 minutes)
stage 3	increase of mental endurance (in intervals) performing homework, concentrating performance in 20-30 minutes intervals
stage 4	start of school/work: possibly restricted, step by step partial completing as tolerated, 1-2 hours (cumulative) homework; increase of activity until full day
stage 5	recovery of full mental work full return to school activity; start of the return-to-sports protocol
Return-to-Sport: expected time window: at least 5 days	
stage 6	light, short aerobic training slight cardiovascular load: e.g. walking, 15 minutes cycling etc., heart rate maximum of 125/min, avoid head shaking
stage 7	sport-specific interval training start of interval training, warm up and sprint training under supervision, sport-specific training/strength-endurance
stage 8	Team training without body contact participation in normal team training, but without any body contact!
stage 9	Normal team training Participation in a normal team training
stage 10	Match test Match possible, but clearly declared as last stage in the return-to-play program

- for stage 4, no school-based monitoring option is available leading to an individual decision, ideally supervised by a doctor
- for stages 6-10: with recurrence of symptoms, stay on present stage (even on the following day); try again until stage is well tolerated; only without any complaints go to the next stage the following day!
- the total recovery phase thus comprises of 6-10 days, considering the typical recovery time of the nerve cells and the prolonged healing course in children

Fig. 3. Combined Return-to-School/Work and Return-to-Sport protocol.

irregular cranial base, a concussion can lead to hormonal dysfunctions from shearing injuries to the pituitary gland. These dysfunctions were found to be present after head injuries of all severity in 28%–69% (62).

Additionally, neuropsychological, psychiatric and psychotraumatologic consequences of concussion need further evaluation. The Brain Check Program in Germany addresses these on-going problems (60).

Second impact syndrome

In the early phase after initial trauma, there is a potential of sustaining a further, more relevant injury, due to a higher vulnerability of the (non-healed) brain cells. This can arise when the symptoms of a first concussion have not yet completely subsided, and a second concussion occurs (10, 37, 43). It was seen among athletes between 16 and 23 years of age who had no loss of consciousness as part of the second injury (50).

Clinically, dramatic symptoms are described, which can lead to even to death (8, 10). Acute imaging showed massive brain swelling, which was explained by increased brain vulnerability, resulting from a disordered autoregulation of the cerebral blood supply with resulting increased intracranial pressure (6, 29). Mortality rates of up to 50% and permanent morbidity in up to 100% have been reported (9).

Animal experiments have shown that the greatest risk for secondary effects of a second concussion is around the third day after primary trauma (65, 68).

Long-term impairments

Long-term impairments are connected to the term chronic traumatic encephalopathy (CTE), which is extensively discussed in the scientific literature, especially focusing on neurodegenerative disorders (63).

There seems to be an association between life time prevalence of depression and a history of multiple concussions. More than 3 concussions were associated with a 3-fold higher risk of development of depressive disorders in athletes compared to the normal population and sustaining 1–2 concussions showed a 1.5-fold higher risk (28, 49).

TBI is an accepted risk factor for developing Alzheimer's disease (57). Especially, the time interval between injury and development of symptoms seems to be shorter in patients with TBI (51). Moderate and severe TBI were associated with high risks with a further examination of former war veterans showed Hazard Ratio of 2.32 and 4.51, respectively (52).

A suspicion was stated in soccer players after repetitive head injuries to develop an ALS, as an unusual high the frequency was observed, especially in those < 49 years of age (13, 14). A dose relationship was suspected, as

playing > 5 years was identified as a risk factor. Comparable data were found in an US analysis, where a TBI sustained within the last 10 years increased the odds for ALS development 11-fold (12). A meta-analysis reported a 1.7-fold higher risk after TBI (12).

Whereas early results were indifferent in their conclusion on association between TBI and suicidality (32, 45, 66), a recent analysis on 235,119 patients after concussions found a 3-fold increased risk compared to the normal population (23).

CONCLUSIONS

It can be concluded from these analyses, that there is a potential risk, even after sport-related concussion, but no clear recommendations can be made to the individual patient. Recent results found a threshold dose-response relationship between concussion and later life risk for cognitive impairment, subjective executive dysfunction, depression, apathy, and behavioral dysregulation.

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