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Mild Head Injury in Young Athletes

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Abstract

Mild head injury is of growing interest because of its underdiagnosed prevalence and underestimated clinical importance. Half of the patients in emergency departments report sport-related head injuries or concussions. Knowledge of symptoms and appropriate management can be improved and is a matter of practical interest for healthcare providers as well as coaches, parents and athletes in order to improve patient care and prevent future injuries. An acute concussion presents with a combination of physical, cognitive, and emotional symptoms, which are usually self-limited and resolve within a week. However, significant sequelae from even mild impacts, such as headaches, prolonged cognitive impairment, or even death, are known. A second impact before full recovery from the first may have deleterious consequences and should be avoided by observing strict rules for «return to play.» Recent research suggests that repetitive minor hits my cause delayed brain damage (dementia pugilistica or the «punch-drunk syndrome»). A link to neurodegenerative diseases such as dementia, Alzheimer's disease (AD) and parkinsonism (tauopathies) is described by amyloidβplaques in those brains. A genetic predisposition (apolipoprotein) is discussed.

This review will focus on the impact of mild head injuries in young athletes and present the current «return to play» rules to avoid the second-impact syndrome. We describe in detail standardized guidelines for appropriate diagnosis and treatment and discuss the association between repetitive minor injuries and chronic traumatic encephalopathy and neurodegenerative diseases.

Introduction

Mild head injuries in general are a serious healthcare problem contributing to 0.5% of all visits to emergency departments and resulting in 10% of all pediatric admissions. In adolescents mild head injuries are often related to sport. Recent studies[1), 2)] have shown that 20–50% of the minor head injuries treated in an emergency department are sport-related. Public knowledge about mild head injuries is astonishingly poor: only less than 10% of the population believe that a head injury could cause a brain injury, more than 25% think it is harmless and never causes long-term problems and up to 40% believe a second blow to the head may help someone remember things they forgot after a first injury[3)]. Also, many professional healthcare providers think a concussion, even in sports, is necessarily accompanied by a period of unconsciousness, which stands in contrast to the opinion of most experts[4), 5), 6)].

The lack of a consensus on the definition and terminology of mild head injury, traumatic brain injury (TBI) and concussion contributes to the misunderstanding and underestimation of this disease entity. For this reason, mild head injuries or concussions are often misjudged as minor and not recognized by the children who sustain them, their parents, coaches, or by physicians. Often only concussions with loss of consciousness are regarded as significant[7)]. To prevent athletes from incurring subsequent injuries, initial symptoms *(Table 1)* should be recognized and athletes as well as trainers encouraged to report symptoms.

It follows that patients who are not investigated according to standardized protocols are not protected from a second injury by adhering to instructions for the follow-up[4)]. The team sports with the highest risk are ice hockey and football, where 1.0 concussions occur per 1000 exposures (games or training sessions), followed by soccer (0.3 per 1000 exposures), basketball and baseball[2), 5), 8)]. In other words, 10% of all football players will sustain at least one minor head injury per season[9)] and one in 15 players with a concussion may suffer an additional concussion during the same playing season[9), 10)].

Definitions

The degree of head injury is defined according to the Glasgow Coma Scale (GCS) as mild (GCS 13–15), moderate (GCS 9–12) or severe (GCS ≤ 8) TBI. By contrast, the word «concussion» describes the pathophysio-

Physical	Cognitive	Emotional	Sleep
Headache	Feeling mentally «foggy»	Irritability	Drowsiness
Nausea	Feeling slowed down	Sadness	Sleeps more than usual
Vomiting	Difficulty concentrating	More emotional	Sleeps less than usual
Balance problems	Difficulty remembering	Nervousness	Difficulty falling asleep
Visual problems	Forgetful of recent information		
Fatigue	Confused about recent events		
Sensitivity to light	Answers questions slowly		
Sensitivity to noise	Repeats questions		
Dazed			
Stunned			

Table 1: One or more symptoms may suggest a concussion. This may or may not include loss of consciousness (LOC).

logical und functional rather than the structural aspects of the injury and is defined as a «… complex process affecting the brain, induced by traumatic biochemical forces… »[5), 6)], or as a brain injury with «… transient, traumatic disruption of brain activity …»[11)]. A concussion is described as a direct blow to the head, face or neck, which results in short-lived impairment of the neurological function of the brain.

Pathophysiology

Pathological studies have demonstrated that head injuries – predominantly inflicted by diffuse rotational and, to a lesser extent, linear forces – destroy the axonal and cytoskeletal structures, which causes axonal swelling and bulb formation. Failed axonal transport in the injured axons causes an accumulation of several proteins such as amyloid precursor protein (APP), presenilin-1 (PS-1), and β-APP cleaving enzyme (BACE). Condensation of APP, PS-1 and BACE causes amyloid β (Aβ) formation and accumulation. The damaged neuronal cells then release the accumulated Aβ. The enzyme neprilysin (NEP), that clears Aβ, is also increased and released by the damaged axons. This may mitigate the negative effect of increased Aβ deposition in the brain parenychma, which sometimes persists for years and results in neurodegeneration and brain atrophy seen many years after brain injury[12), 13)].

Additionally, the destruction of the cytoskeletal structure increases the amount of tau proteins in the brain. These features, Aβ plaques and abnormal tau accumulation, share many characteristics with Alzheimer's (AD) and Parkinson's disease («pugilistic parkinsonism»)[14)]. Elevation of tau proteins in the CSF is also an ominous sign in severe head injury patients[15)].

Which field are we on?
Which team are we playing today?
Which quarter (period) is it?
Which side scored the last goal?
Which team did we play last week?
Did we win last week?

Table 2: Sideline evaluation: Maddocks questions

Recently, another proteinopathy was described in former athletes[16)] that is associated with the loss of the first motor neuron and corticospinal tract degeneration: amyotrophic lateral sclerosis (ALS) has been repeatedly observed and confirms reports that (Italian) soccer players with previous repetitive head injuries have a higher risk for ALS[17)].

Symptoms

Symptoms of mild head injury or concussion are physical, cognitive, emotional, or related to balance, coordination and sleep disturbance *(Table 1)*. Symptoms may or may not involve loss of consciousness[5), 6), 18), 19), 20)]. They subside by half within 24–48 hrs and usually reach the pre-injury level by day 7. Metabolic and neurotransmitter abnormalities[21)] as well as subtle neuropsychological, cognitive symptoms and balance disturbances may last up to 30–45 days. Brain electrical activity is abnormal at the time of injury and still at day 8, despite the fact that symptoms had already disappeared by that time[22)]. Standard structural neuroimaging is normal[5), 6)].

The subsequent course is unpredictable. In general, children need a longer period for resolution of symptoms than do adults, and most children will recover unremarkably. Risk factors for a slower recovery are loss of consciousness > 1 min, prior injuries, attention deficit hyperactivity syndrome (ADHS), psychiatric disease, depression, and anxiety[6)]. In some patients recovery is complicated by the post-concussion syndrome[18)], a mix of physical, emotional, and behavioral problems similar to depression, anxiety and attention deficit disorder[5), 18), 19)]. The post-concussion syndrome is seen in more than half (58%) of concussion patients after one month, in more than 10% after three months, and still in 2.3% of patients (e. g. headache) after one year. Symptoms are more likely to persist in children older than 6 years, in those with preexisting injuries, ADHS, or psychiatric disease[6), 24)]. Recent studies report that after a mild head injury, 75% of the young patients will show endocrinological dysfunction after six months and almost 30% will suffer from thyroid hypofunction, growth hormone deficiency or premature puberty after one year[4), 25)].

Investigations

On the field, athletes should be managed according to standard emergency principles (ABC rule), giving particular attention to the cervical spine. On the sideline, a neuropsychological assessment must follow either briefly using the Maddocks questions[26)] or in more detail with the Sport Concussion Assessment (SCAT2)[5), 6)] or other tests[43)]. The Maddocks questions *(Table 2)* pertain only to orientation (time, place, person) and are not a substitute for a proper and more detailed examination in the locker room or the coach's office. The SCAT2[5), 6)] is a standardized tool that includes symptom evaluation, calculation of the Glasgow Coma Scale score, sideline assessment (Maddocks questions) and – importantly – a cognitive, balance and coordination assessment. However, the SCAT2 has never been evaluated in a prospective study. Consequently, no normative data or «cut-offs» are available for it. Nevertheless, it is a useful, practicable and standardized tool for estimating the risk for athletes following a head injury. Other neuropsychological tests are web-based (imPACT®, HeadMinder®, CogState®)[5)] and developed specifically for athletes' injuries. For all these tests a pre-injury test should ideally be obtained at the beginning of the season to assist in interpreting any post-injury result[27)].

Return to Play Rule

The main message is that any player with a diagnosed concussion should never be allowed to return to play on the same day, no matter how brief the symptoms are or how well the player looks («when in doubt, sit them out»). They should never be left alone in the hours subsequent to sustaining a hit to the head in order to not miss any deterioration, and they are not allowed to drive a vehicle until medically cleared[5), 6), 43)].

The return to play protocol[5), 6)] is a six step process *(Table 3)*, where each step takes 24 hours. If any symptoms recur, the athlete is put back to the previous level until he/she is finally symptom-free. This protocol is to be applied in all athletes, regardless of whether the concussion is simple or complex, or the athletes are professionals or amateurs. The concept is based on physical/mental and cognitive rest. This means

Symptom-free at rest (daily progression to next step; if symptomatic, go back one step)
Advance to full cognitive activities
Stationary biking, jogging to sprinting (light aerobic exercise)
Non-contact, sport-specific exercise
Non-contact training
Full-contact practice
Return to play

Table 3: Return to Play Rule

no physical (work, sport) or cognitive (school, noise, television, internet or text messaging, bright light, sleep shortage) activities should be undertaken. Light aerobic (stationary biking, jogging to sprinting) is followed by sport-specific exercise, then non-contact, contact training and practice. Any medication prescribed to reduce symptoms must be discontinued before training starts. There is no cure for a concussion, but treatment can help. For prolonged symptoms antidepressants or amantadine (NMDA receptor antagonist)[28] may be an option.

An exception from the rule «no return to play the same day» can be made if a player was consistently free of symptoms for 20 minutes following the concussion, had a negative test and the team physician has sufficient experience and adequate resources (neuropsychological assessment, neuroimaging) at his disposal[29]. This approach is adequate for adult athletes, but children and adolescents should be treated more conservatively[6), 23].

These recommendations are reflected in various laws enacted in recent years in the United States. In 2007 Texas passed the Will's Bill, which requires high school coaches, athletics trainers and even players to undergo safety training such as in cardiopulmonary resuscitation. The Zackery Lystedt Law (May 2009) urged the development of guidelines and educational materials for athletes, parents and coaches. All athletes suspected of having a concussion must be removed from play and may not return before they have been cleared by a licensed healthcare professional. In Switzerland guidelines for athletes, coaches and parents are published by the Swiss Ice Hockey League[43], but no obligatory/mandatory law has passed the legislature.

Repetitive Mild Head Injury – Chronic Traumatic Encephalopathy, CTE

Recently, concerns about repetitive minor traumatic head injuries came to public attention in the media. National Geographic (February 2011 issue) reported the history of a 21-year-old football defensive back, who took 537 (!) hits to the head in games and practice during one season. Several (> 20) had an impact equal to that sustained when hitting a windshield in a car crash at approx. 20 mph. After two hits he showed symptoms of a concussion without loss of consciousness.

Repeated concussions have a long-lasting effect on cognitive and motor function, even decades after the last injury[30), 31]. Already in the 1920ies, in boxers (prize fighters) with recurrent head injuries a syndrome called the «punch-drunk syndrome» or «dementia pugilistica» was described. It is characterized by dysarthria, pyramidal and cognitive signs as well as other neuropsychiatric symptoms like long-term cognitive impairment[8], anxiety or depression[24]. This disorder, currently defined as chronic traumatic encephalopathy (CTE)[30], is a rare event, given the high number of concussions occurring each year. Case studies demonstrate that a significant percentage (15–20%) of former and retired athletes in any contact sport will develop symptoms of CTE[32], with a higher prevalence in those with three or more concussions in their history and those with an apolipoprotein E4 (ApoE4) polymorphism[33), 34].

Recent research suggests that there is a link between TBI and the development of neurodegenerative diseases like AD, parkinsonism or dementia. The more severe and the more often a TBI occurs, the greater the risk for and the earlier the onset of AD is[33]. Striking arguments for this hypothesis are found in autopsies of former athletes, who sustained multiple minor head injuries[12), 30), 35]. Aβ plaques are found even in children who died following a TBI[12]. These plaques are similar to those found in the early stages of AD; they occur rapidly within a few hours and appear not only in the white, but also in the grey matter of the brain parenchyma.

It is unclear and the subject of discussion whether all athletes with recurrent injuries have the same risk for developing CTE, or only those with a genetic predisposition. Susceptibility depends on (epi)genetic factors, such as age, sex and ApoE4 and other unknown genetic factors. In the non-sporting population, ApoE4 polymorphism is a risk factor for AD[33] and is associated with a significantly poorer outcome for all degrees of head injury[34), 36), 37], especially in children aged less than 15 years[38]. ApoE4 is also associated with earlier onset of Aß deposition[33), 39] and a decreased volume of the entorhinal cortex and hippocampus as compared to those without ApoE4[33]. Consequently, an ApoE4 polymorphism is a highly suspicious risk factor for the development of CTE in athletes with repetitive mild head injuries.

Second-Impact Syndrome (SIS)

Sustaining a second head trauma before full recovery from the previous one may bring on the second-impact syndrome[7), 40), 41]. The proposed pathophysiological pathway is not known exactly[41]. It is a rare condition seen only in young athletes (boxers, football, baseball but also soccer players) under the age of 21 years[21]. It is often fatal (1–2 deaths per year in the US) and may occur within up to two weeks after taking the first hit, and can even be triggered by the mildest degree of second head injury[7]. For this reason, all recent guidelines strongly advocate adherence to the return to play rule as outlined above[7].

Prevention

In order to reduce and minimize the risk for mild head injury in athletes, an attempt must be made to improve several aspects: firstly, injuries should be prevented by changing the athletes' attitude: respect the

bodies and heads of the players on your and your opponent's team. Wear an intact and fixed helmet and a mouth guard and play «heads-up»[20), 43)]. Secondly, laws must be passed to ensure mandatory facilities (require emergency and safety training for sports officials in high schools, emergency-trained physicians on the field) and procedures (return to play rule). Thirdly, trainers must be educated to recognize symptoms and adhere to safety guidelines[5), 6), 26)]. Lastly, written information must be provided for the athletes and their parents similar to the information supplied for non-sport related mild head injuries[42)], and the competence of our profession as medical doctors must also be improved.

References

1) Callahan JM. Pediatric Concussions in United States Emergency Departments: The Tip of the Iceberg. J. Pediatr 2010; 157: 873–5.
2) Bakhos LL, Lockhart GR, Myers R, Linakis JG. Emergency department visits for concussion in young child athletes. Pediatrics 2010; 126: e550–6.
3) Guilmette TJ, Paglia MF. The public's misconception about traumatic brain injury: a follow-up survey. Arch Clin Neuropsychol 2004; 19: 183–9.
4) Meehan WP. Mannix R. Pediatric concussions in United States emergency departments in the years 2002 to 2006. J Pediatr 2010; 157: 889–93.
5) Halsted ME, Walter KD. Council on Sports Medicine and Fitness. American Academy of Pediatrics. Clinical report: Sport-related concussion in children and adolescents. Pediatrics 2010; 126: 597–615.
6) McCrory P, Meeuwisse W, Johnston K, et al. Consensus statement on concussion in sport: The 3rd International Conference on Concussion in Sport held in Zurich, November 2008. Br J Sports Med 2009; 43 (Suppl I): i76–i90.
7) Smith BW. Head injuries. In SJ Anderson, SS Harris, eds., Care of the Young Athlete, 2nd. ed., pp. 185–191. Elk Grove Village, IL: American Academy of Pediatrics.
8) Guskiewicz KM, Weaver NL, Padua DA, et al. Epidemiology of concussion in collegiate and high school football players. Am J Sports Med 2000; 28: 643–50.
9) Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players. The NCAA Concussion Study. JAMA 2003; 290: 2549–55.
10) Collins MW, Lovell MR, Iverson GL, et al. Cumulative effects of concussion in high school athletes. Neurosurgery 2002; 51: 1175–81.
11) Piatt JH. Traumatic brain injury In: Clinical Pediatric Neurosciences. First Edition. Legido A, Piatt JH (Eds), Elk Grove Village, American Academy of Pediatrics, 2009; 171–215.
12) Johnson VE, Stewart W, Smith DH. Traumatic brain injury and amyloid-ß pathology: a link to Alzheimer's disease? Nat Rev Neurosci 2010; 11: 361.
13) Miller G. A Late Hit for Pro Football Players. Science 2009; 325: 670–2.
14) DeKosky ST, Ikonomovic MD, Gandy S. Traumatic Brain Injury-Football, Warfare, and Long-Term Effects. N Eng J Med 2010; 363: 1293–6.
15) Liliang PC, Liang CL, Weng HC,et al. Tau proteins in serum predict outcome after severe traumatic brain injury. J Surg Res. 2010; 160: 302–7.
16) McKee AC, Gavett BE, Stern RA, et al. TDP-43 proteinopathy and motor neuron disease in chronic traumatic encephalopathy. J Neuropathol Exp Neurol 2010; 69: 918–29.
17) Chio A, Benzi G, Dossena M, et al. Severely increased risk of amyotrophic lateral sclerosis among Italian professional football players. Brain 2005; 138: 472–6.
18) Barlow KM, Crawford S, Stevenson A et al. Epidemiology of post-concussion syndrome in pediatric mild traumatic brain injury. Pediatrics 2010; 126: 373–81.
19) McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA 2003; 290: 2556–63.
20) American Academy of Neurology. Practice parameters: the management of concussion in sports: Centers for Disease Control and Prevention. http://www.cdc.gov/ncipe/pub-res/tbi_tollkit/physician/concussions_sports.pdf (accessed August 23rd 2012).
21) Zanfonte R. Diagnosis and management of sports-related concussion: a 15-year-old athlete with a concussion. JAMA. 2011; 306: 79–86.
22) Barr WB, Prichep LS, Chabot R, et al. Measuring brain electrical activity to track recovery from sport-related concussion. Brain Inj 2012; 26: 58–66.
23) Zuckerman SL, Odom M, Lee YM, et al. Sport-related concussion and age: number of days to neurocognitive baseline. Neurosurgery 2012; 71: 558.
24) Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. Med Sci Sports Exerc 2007; 39: 903–9.
25) Kaulfers A, Backeljauw PF, Reifschneider K, et al. Endocrine dysfunction following traumatic brain injury in children. J Pediatr 2010; 157: 894–9.
26) Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. Clin J Sports Med 1995; 5: 32–5.
27) Jinguji TM, Bompadre V, Harmon KG, et al. Sport Concussion Assessment Tool 2: baseline values for high school athletes. Br J Sports Med 2012; 46: 365–70.
28) Giacino JT, Whyte J, Bagliella E. Placebo-controlled trial of amantadine for severe traumatic brain injury. N Engl J Med 2012; 366: 819–26.
29) Makdissi M, McCrory P, Ugoni A, et al. A prospective study of post-concussive outcomes after return to play in Australian football. Am J Sports Med 2009; 37: 877–83.
30) McCrory P. Sports concussion and the risk of chronic neurological impairment. Clin J Sport Med 2011; 21: 6–12.
31) De Beaumont L, Théoret H, Mongeon D et al. Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood. Brain 2009; 132: 695–708.
32) Guskiewicz KM, Marshall SW, Bailes J, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. Neurosurgery 2005; 57: 719–26.
33) Verghese PB, Castello JM, Holtzman DM. Apolipoprotein E in Alzheimer's Disease and other neurological disorders. Lancet Neurol 2011; 10: 241–52.
34) Liberman J, Stewart W, Wesnes K, et al. Apolipoprotein E epsilon 4 and short-term recovery from predominantly mild head injury. Neurology 2002; 58: 1038–44.
35) McKee AC, Cantu RC, Nowinski CJ, et al. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. J Neuropathol Exp Neurol 2009; 68: 709–35.
36) Friedmann G, Froom P, Sazbon L, et al. Apolipoprotein E-epsilon 4 genotype predicts a poor outcome after head injury. Neurology 1999; 52: 244–8.
37) Zhou W et al. Meta-analysis of APOE E4 allele and outcome after traumatic brain injury J Neurotrauma 2008; 25: 279–90.
38) Teasdale G, Murray G, Nicoll JA. The association between APOE β4, age and outcome after head injury: a prospective cohort study. Brain 2005; 128: 2556–61.
39) Nicoll JA, Roberts GW, Graham DI. Apolipoprotein E β4 allele is associated with deposition of amyloid ß protein following head injury. Nature Med 1995; 1: 135–7.
40) McCrory P, Berkovic SF. Second-impact syndrome. Neurology 1998; 50: 677–8.
41) McCrory P, Davis G, Makdissi M. Second-impact syndrome or cerebral swelling after sporting head injury. Curr Sports Med Rep 2012; 11: 21–3.
42) Vernet O, Lutz N, Rilliet B. Die Betreuung des kindlichen Schädelhirntraumas. Paediatrica 2004; 15: 47.
43) http://www.swiss-icehockey.ch/SIH/medical/de/medkomm_concussion.php (accessed Agust 23rd 2012).

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